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Animal Welfare 2007, 16: 449-458
ISSN 0962-7286

Is sodium fluoroacetate (1080) a humane poison?

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Abstract

Sodium fluoroacetate (1080) is widely used for the control of vertebrate pests in Australia. While the ecological impact of 1080 baiting on non-target species has been the subject of ongoing research, the animal welfare implications of this practice have received little attention. Literature relevant to the humaneness of 1080 as a vertebrate pest control agent is reviewed in this paper. Previous authors have largely concentrated on the perception of pain during 1080 toxicosis, giving limited attention to other forms of distress in their assessments. Authors who suggest that 1080 is a humane poison largely base their conclusions on the argument that convulsive seizures seen in the final stages of 1080 toxicosis indicate that affected animals are in an unconscious state and unable to perceive pain. Other authors describe awareness during seizures or periodic lucidity that suggests central nervous system (CNS) disruption cannot be assumed to produce a constant pain-free state. Some literature report that 1080 poisoning in humans is painless and free of distress, but this is contradicted by other clinical studies. Using available data an attempt is made to reassess the humaneness of 1080 using the following criteria: speed and mode of action, appearance and behaviour of affected animals, experiences of human victims, long-term effect on survivors, and welfare risk to non-target animals. It is concluded that sodium fluoroacetate should not be considered a humane poison, and there is an urgent need for research into improving the humaneness of vertebrate control methods in Australia.

Keywords: *1080, animal welfare, bait, poison, sodium fluoroacetate, vertebrate pest*

Introduction

Compound 1080 is a proprietary name for the chemical compound sodium fluoroacetate (Peters 1952). Following ingestion, 1080 is metabolised to fluorocitrate which inhibits cellular energy production by inhibiting enzymes responsible for the conversion of citrate and succinate in the tricarboxylic acid cycle (Peters 1952; Fanshier *et al* 1964). A range of other cellular enzymes are also affected by 1080, but the relevance of these to toxicosis is not well understood (Mehlman 1967; Godoy & del Carmen Villarruel 1974; Taylor *et al* 1977; Kirsten *et al* 1978).

In Australia 1080 is widely used in baiting programmes for the control of vertebrate pest species (Table 1) and as such has both agricultural and wildlife-management applications (Biodiversity Group Environment Australia 1999; Government of Western Australia 2002; Williamson & Bloomfield 2003). The popularity of 1080 as a vertebrate pest control agent is based on a range of factors including its low cost, potency, relative ease of use (particularly in pre-prepared baits), low risk of persistence in the food chain, and low risk of wider environmental contamination (McIlroy 1996). It is widely regarded by users as efficient, target specific and humane (Government of Western Australia 2002; Williamson & Bloomfeld 2003). Nevertheless, the use of 1080 baits is the subject of vigorous debate in Australia, with concerns over the impact on non-target animals including rare or endangered native Australian species, domestic animals (livestock and working dogs) and companion animals. More recently, public concerns regarding the humaneness of 1080 baiting have also been raised. While non-target impacts have been the ongoing subject of research (McIlroy *et al* 1986; King 1989; McIlroy & Gifford 1991; Dexter & Meek 1998; Fairbridge *et al* 2000, 2003; Glen & Dickman 2003 a,b; Kortner *et al* 2003), the animal welfare impacts of 1080 baiting are largely unknown. Only a few publications have previously considered the humaneness of 1080 and the welfare of target or non-target animals as a wildlife management issue (Bell 1972; Morgan 1990; Saunders *et al* 1995; Gregory 1996; McIlroy 1996; Williams 1996; Marks *et al* 2000; Potter *et al* 2006).

Publications that have briefly addressed 1080 humaneness

In his review of brushtail possum (*Trichosurus vulpecula*) control in New Zealand, Bell (1972) contrasts 1080 with other poisons, stating that arsenic trioxide and strychnine alkaloid "appear to bring about exceedingly painful physiological reactions in the animal, more so than does sodium cyanide or sodium fluoroacetate". No details are given regarding the signs of poisoning during the toxicosis resulting from these compounds and pain was the only aspect of welfare considered.

Table 1 The use of 1080 for vertebrate pest control in Australian states and territories.

State or territory	Target animals
Australian Capital Territory	Foxes, rabbits
New South Wales	Foxes, rabbits, pigs, dogs, dingoes
Northern Territory	Foxes, dingoes
Queensland	Foxes, rabbits, pigs, dogs, dingoes, rats
South Australia	Foxes, rabbits, dogs, dingoes
Tasmania	Rabbits, wallabies*, brushtail possums, cats
Victoria	Foxes, rabbits, pigs, dogs, dingoes
Western Australia**	Foxes, rabbits, dogs, dingoes

* Bennett's and rufous wallabies. ** Also used experimentally for the control of pigs, cats, goats, agile wallabies and sulphur-crested cockatoos.
Data collected from the Report to the Vertebrate Pests Committee on 1080 Policies, Practices and Procedures in Australia and New Zealand from the 1080 Working Group of the Vertebrate Pests Committee, August 2001.

Morgan (1990) argues that 1080 is a relatively humane method of control for possums, based on the finding that possums were found dead in the morning at the location where they were observed the previous night. However, this provides little insight into the progression and nature of toxicosis in this species, and the author provides no explanation of how this observation contributes to a robust assessment of humaneness. A range of signs potentially indicative of pain or distress, including behavioural changes, deteriorating co-ordination, retching, vomiting, rapid breathing, lack of response to disturbances and shivering, were all described in this study. As observations were limited largely to the first few hours after the bait was eaten, later signs of poisoning may also have been overlooked.

Saunders *et al* (1995) suggest that the evidence regarding the humaneness of 1080 in the red fox (*Vulpes vulpes*) is ambiguous given that anaesthetised dogs (*Canis lupus familiaris*) poisoned with 1080 show evidence of extreme CNS stimulation (Chenoweth & Gilman 1946). It is implied that the behaviour of unanaesthetised dogs poisoned with 1080 does not, therefore, necessarily indicate pain or suffering. Assuming that the comparison between fluoroacetate poisoning in dogs and foxes is valid (and there is some support for this [Marks *et al* 2000]) there are still some problems with this line of reasoning. Chenoweth and Gilman (1946) do not clearly define the nature of the CNS stimulation they observed, nor do they make any attempt to correlate the signs observed in anaesthetised dogs (pentobarbital, 35 mg kg^{-1}) with behavioural patterns in unanaesthetised dogs during 1080 toxicosis. The relevance of findings in anaesthetised animals is unclear given that pentobarbital anaesthesia substantially alters both the signs of fluoroacetate poisoning and the mechanism of death in cats (*Felis catus*; Chenoweth & Gilman 1946) and the EEG formations recorded in dogs (Chenoweth & St John 1947a). In the absence of a study where the effects and interactions of pentobarbital in 1080 toxicosis are controlled, it is not possible to draw conclusions about the behaviour of unanaesthetised dogs from findings in anaesthetised ones.

McIlroy (1996) attempts to assess the humaneness of 1080 relative to other lethal agents used in vertebrate pest control. He states that death from cyanide poisoning is painless, that death from phosphorus is not, but that the evidence regarding 1080 is equivocal. He considers aspects relevant to welfare assessment other than pain, including the speed of action, the presence or absence of cumulative effects and the risk to non-target species (in terms of relative toxicity, the availability of antidotes, degradation in corpses and degradation in the external environment). Unfortunately, there is a lack of detail on how these categories are defined and the categorisation is largely subjective.

Marks *et al* (2000) differ from earlier authors in that their research is based on the premise that the humaneness of 1080 baits can be improved. They propose that because CNS disturbances confuse the objective assessment of the perception of pain in foxes poisoned with fluoroacetate, baits should be supplemented with anxiolytic and/or analgesic compounds in order to minimise the potential for pain and distress. They note that the early stages of 1080 toxicosis may be associated with suffering, even if pain and distress are absent in the late stages of toxicosis once severe CNS dysfunction has developed. Signs of poisoning that were commonly observed in Marks *et al*'s (2000) study of foxes that could be associated with pain or distress include retching, manic running, tail twitching, clonospasm, tetanic spasms, and collapse and paddling of all four limbs. Overall, the period during which there were overt signs of poisoning in foxes dosed with 1080 typically lasted from one to two hours. The supplementation of 1080 with diazepam (10 mg kg^{-1}) significantly extended the duration of this period, but also markedly reduced the intensity of the signs of poisoning. Marks *et al* (2000) also cite the experiments of Chenoweth and Gilman (1946) as evidence that dogs are unlikely to be conscious during the convulsions that occur in the latter stages of 1080 toxicosis. As discussed above, it is not possible to draw such a conclusion from these experiments.

Recently, Potter *et al* (2006) identify the humaneness of vertebrate pesticides as an area of current concern. They describe three discrete stages of 1080 poisoning in stoats (*Mustela erminea*); a latent phase lasting on average 1 h, 1 min (range 29 min–2 h 7 min) during which behaviour appears normal, a period characterised by ataxia and hyperactivity lasting on average 26 min (range 2 min–1 h 40 min), and a period of recumbency with muscular spasms and non-responsiveness to stimuli lasting on average 58 min (range 16 min–2 h). Of these, the ataxic/hyperactive stage is identified as most likely to be associated with pain or distress, while it is argued that the latent period is likely to be associated with minimal pain or distress given the lack of

behavioural indicators at this time (although it is noted that pain and anxiety have been described in humans at the equivalent stage of fluoroacetate poisoning [Chi *et al* 1996, cited in Potter *et al* 2006]). Potter *et al* report that para-aminopropiophenone (PAPP) kills stoats within 1 h (Fischer *et al* 2005, cited in Potter *et al* 2006) but they provide no further detail or comparison with 1080 toxicosis.

Previous reviews of 1080 humaneness

Two publications focus primarily on a consideration of the humaneness of 1080 as a vertebrate control agent (Gregory 1996; Williams 1996) and both were published as part of the proceedings of a meeting on humaneness and vertebrate pest control.

Williams (1996) considers the effects of 1080 on European rabbits (*Oryctolagus cuniculus*), providing a detailed description of the signs of poisoning and the proposed mechanism of toxicity in this species on the basis of earlier literature. A "lethargic" but conscious state lasting from 2–12 h (depending on dose) is described, during which poisoned rabbits stop feeding, develop obvious weakness and lie with the head to one side. Similar findings were reported by Chenoweth and Gilman (1946), Foss (1948) and Meldrum (1957). Williams (1996) concludes that this period is not associated with pain as indicators of pain in rabbits described by other authors (hypermotility of the gut and teeth grinding) are absent. However, given the extensive motor disturbance described, and the possibility of other neurological involvement, the relevance of these behavioural indicators to 1080 toxicosis is unclear. There is no discussion of potential distress or other welfare concerns associated with the prolonged period of weakness and immobility described (eg issues relating to increased risk of predation, decreased access to food and water, or reduced ability to thermoregulate).

Williams (1996) also describes a convulsive period occurring later during toxicosis and concludes that convulsions are not associated with pain, as laboratory data suggests that these occur during a state of unconsciousness, and reflect rapid cerebral anoxia following immediately after ventricular fibrillation. Periods of consciousness following convulsions are described, where some rabbits were observed to re-commence feeding, and this was argued by Williams (1996) to be evidence that significant suffering was not experienced at this time. The observation that rabbits that survived a sub-lethal dose of 1080 were anorexic for up to 24 h post exposure (Hutchens *et al* 1949, cited in Williams 1996) suggests that the possible existence of adverse animal welfare impacts should not be discounted.

Gregory (1996) discusses the humaneness of 1080 poisoning in a range of species on the basis of reports in earlier literature. He has been widely cited in government publications in Australia (eg Biodiversity Group Environment Australia 1999; Government of Western Australia 2002; Williamson & Bloomfield 2003). Gregory (1996) breaks down his discussion according to animal groups, first discussing the effects of fluoroacetate on herbivores, then carnivores. This division is based on a broad generalisation that herbivorous animals die of ventricular fibrillation while carnivores show extensive involvement of the central nervous system, dying as a result of respiratory depression, and omnivores exhibit a combination of signs (Egekeze & Oehme 1979a). This argument grew out of early research indicating that animals can be placed into four categories on the basis of the signs they display during fluoroacetate poisoning (Chenoweth & Gilman 1946). However, a more recent review demonstrates that there are substantial similarities in the signs of fluoroacetate poisoning across a wide range of vertebrate species (Sherley 2004).

Herbivores

Gregory (1996) describes the main signs of 1080 toxicosis in herbivores as lethargy and ataxia (inco-ordination), with some developing generalised convulsive seizures. He implies that these seizures reflect cerebral anoxia resulting from the loss of blood supply to the brain during ventricular fibrillation, and this is supported by Chenoweth and Gilman (1946). However, little attention is given to what may be prolonged periods of signs and symptoms before the onset of ventricular fibrillation. Neither does he consider the possible long-term consequences of a period of cerebral anoxia for animals that recover from a sub-lethal toxicosis (Pridmore 1978). Gregory (1996) briefly considers the issue of distress, describing anecdotal evidence that convulsing rabbits "did not unduly disturb nearby rabbits" and that these "did not seem to associate (convulsions) with fear or pain", however, this appears speculative and subjective. Potentially painful or distressing signs of fluoroacetate poisoning in herbivores have been described in other literature, including; tremor, hypersensitivity to nervous stimuli, muscular spasms, myotonic convulsions, muscular weakness, partial paralysis and respiratory distress (Chenoweth & Gilman 1946; Quin & Clark 1947; Foss 1948; Robison 1970; McIlroy 1982a; Schultz *et al* 1982).

Carnivores

Gregory (1996) describes dogs poisoned with 1080 as hyperexcitable, with abrupt bouts of barking preceding repeated convulsions interspersed with periods of normality. In contrast, he argues that all dogs experiencing pain will appear quiet and less alert, possibly lying still and adopting an abnormal posture. It is purely speculative to suggest that the signs of 1080 toxicosis rule out the possibility of pain. Other authors (Marks *et al* 2000) argue that disruption of the CNS resulting from 1080 poisoning is likely to alter behavioural patterns normally used in assessing pain, therefore making such observations difficult to interpret.

Dogs affected by fluoroacetate have been described by other authors as running uncontrollably (sometimes into rigid objects), retching and vomiting, and experiencing a range of nervous disruptions including twitching of the legs, tail, eyelids, and eyes, and prolonged involuntary contractions of their muscles (Chenoweth & Gilman 1946; Chenoweth & St

John 1947b; Foss 1948; Harris 1975). People who have ingested 1080 frequently report abdominal pain at the same time as experiencing signs of poisoning commonly attributed to poisoned dogs including verbosity (unusual vocalisation), agitation, and vomiting (Chi *et al* 1996) and have also reported pain in association with muscular spasms. In addition to any pain resulting directly from 1080 toxicosis, any injuries sustained by running into rigid objects, or during fits, have the potential to cause pain either at the time or during the lucid intervals between fits that are often described (Chenoweth & Gilman 1946; Chenoweth & St John 1947b; Foss 1948; Gajdusek & Luther 1950; Reigart 1975; McIlroy 1982a; Schultz *et al* 1982; Chung 1984; Robinson *et al* 2002).

Gregory (1996) argues that the early signs of 1080 poisoning in dogs (barking and hyperactivity) are associated with a lack of awareness of their predicament or surroundings, and are not, therefore, distressing. This interpretation is subjective and does not agree with signs and symptoms reported in human cases (Chi *et al* 1996). Moreover, the EEG data that Gregory uses to support a potential lack of awareness during this period (Chenoweth & St John 1947b) was obtained from dogs that had been paralysed with curare before being poisoned, and could not therefore be directly correlated with physical signs. *Petit mal* seizures that Gregory (1996) cites as indicative of loss of contact with the environment were a rare outcome of Chenoweth and St John's (1947b) work and followed direct injection of fluoroacetate into the lateral ventricles of the brain. Ward (1947) obtained occasional *petit mal*-like EEG recordings following intravenous administration of fluoroacetate, but again this was an uncommon finding. Chenoweth and St John's (1947b) finding that EEG disturbances similar to *grand mal* convulsions of epilepsy were common in poisoned dogs could be interpreted as supportive for periods of unconsciousness occurring during fitting, yet another author indicates that in his experiments a dog poisoned with fluoroacetate was conscious during a period of clonic-tonic convulsions (Foss 1948). In quoting Kun's (1982) statement that convulsive seizures are always associated with unconsciousness, Gregory (1996) ignores the fact that focal convulsions (those affecting only part of the brain and body) are not typically associated with unconsciousness. Even generalised convulsions are not always associated with loss of consciousness; patients remain fully conscious during the generalised tonic spasms associated with strychnine poisoning (Aggarwal & Prakash Wali 1997). Both localised muscle spasms and tonic convulsions are frequently described in 1080 toxicosis prior to the onset of clonic-tonic convulsions (reviewed in Sherley 2004). It is therefore inappropriate to suggest that all convulsive episodes observed in dogs poisoned with 1080 are generalised seizures, and that they are reliably associated with unconsciousness and a pain and distress free state.

Gregory (1996) argues that 1080 toxicosis may also be likened to hyperinsulinism because in each case there is a depletion of cellular energy. Conditions affecting aspects of cellular energy metabolism do not necessarily have the same symptoms. Cyanide inhibits cellular energy metabolism by blocking the electron transport chain, and arsenite (like fluoroacetate) inhibits energy metabolism by blocking the citric acid cycle (at multiple sites), yet the human symptoms of arsenite poisoning (violent gastroenteritis, burning oesophageal pain, vomiting, and watery or bloody diarrhoea containing shreds of mucus) differ substantially from those of cyanide (dizziness, rapid respiration, vomiting, flushing, headache, drowsiness, circulatory collapse, and unconsciousness) or 1080 poisoning (vomiting, excitability, tonic-clonic convulsions, irregularity of the heartbeat and respiration, exhaustion, coma, and respiratory depression) (Dreisbach 1983). The cause of fluoroacetate's toxic effect is probably a combination of the depletion of energy at a cellular level, an accumulation of citrate in the affected tissues (Foss 1948), and other resulting electrolyte disturbances including hypocalcaemia and hypokalaemia (Chi *et al* 1996).

How should humaneness be defined?

A major problem with previous considerations of the humaneness of 1080 poisoning, has been the absence of agreed criteria for assessing humaneness. Previous authors have primarily focused on the perception of pain, particularly during the late stages of poisoning, with limited consideration of other aspects of welfare such as distress. Mason and Littin (2003) assessed humaneness of rodent control by: 1) speed of action; 2) mode of action; 3) the appearance and behaviour of affected animals; 4) the experiences of human victims; 5) the long-term effect on survivors, and, 6) the welfare risk to non-target animals. Using available data, these categories are applied in an attempt to investigate the humaneness of 1080 toxicosis.

Speed of action

There is a lag-time between the ingestion of 1080 and the onset of obvious signs of toxicosis. The duration of this lag period, and the time from exposure until death, vary significantly between vertebrate species (McIlroy 1986; Table 2), with some animals dying within minutes and others surviving for several days. Data regarding the time from exposure until signs of toxicosis or death has typically been published as part of studies designed to determine the median lethal dose of 1080. If the duration of the toxicosis is dependent upon the dose rate of 1080, the lower doses used in many of these studies may not accurately reflect the outcome that can be expected from baits that deliver lethal doses. Evidence regarding the relationship between dose and effect in 1080 toxicosis is variable. Human poisoning cases appear to vary in the length of time elapsed before the onset of neurological or cardiac involvement, and in the severity of signs and symptoms, depending on dosage (Gajdusek & Luther 1950; Brockman 1955; Reigart 1975; Peters *et al* 1981; Chung 1984; Robinson *et al* 2002). Chenoweth and St John (1947b) also reported a strong effect of dose size on the signs of poisoning in dogs. Another study on the effects of 1080 on herbivores demonstrated that the time from exposure until death is positively

Table 2 The median lethal dose and progression of fluoroacetate toxicosis in a range of vertebrate species.

Animal	LD50 (mg kg^{-1})	Time until signs (h)	Time until death (h)
Mammals			
Marsupuial herbivores			
Brushtail possum (*Trichosurus vulpecula*)*	0.47–0.79	1.0–19.8	5.0–97.0
Bennett's wallaby (*Macropus rufogriseus*)*	> 0.21	< 16.9–23.2	8.9–38.9
Southern hairy-nosed wombat (*Lasiorhinus latifrons*)	0.21	5.1–39.4	16.2–59.3
Eastern grey kangaroo (*Macropus giganteus*)	~ 0.1–0.35	< 13.2–23.9	20.9–62.1
Eutherian herbivores			
Horse (*Equus caballus*)	1.0	~ 1.5–2.0	6.0–10.5
Sheep (*Ovis aries*)	0.5	6.2–37.6	9.6–61.6
Rabbit (*Oryctolagus cuniculus*)*	0.34–0.50	1.1–10.1	3.0–44.3
Cattle (*Bos taurus*)	0.39	1.5–29.0	1.5–29.3
Marsupial omnivores/carnivores			
Northern quoll (*Dasyurus hallucatus*)	5.66	3.0–361.9	10.0–450.7
Tasmanian devil (*Sarcophilus harrisii*)	4.24	0.3–1.6	2.6–22.3
Eastern quoll (*Dasyurus viverrinus*)	3.73	0.2–2.4	< 2.0–63.2
Stripe-faced dunnart (*Sminthopsis macroura*)	0.95	1.7–4.0	3.4–13.1
Eutherian omnivores/carnivores			
Feral pig (*Sus scrofa*)*	1.0	1.9–47.3	2.8–80
Cat (*Felis catus*)*	0.40	1.0–5.6	20.7–21.0
Dingo (*Canis lupus dingo*)*	0.11	4.8–14.6	5.3–10.8
Red fox (*Vulpes vulpes*)*	0.12	4.1[1]	5.5[1]
Rodents			
House mouse (*Mus musculus*)	8.33	1.3–2.8	2.2–68.3
Grassland melomys (*Melomys burtoni*)	2.65	0.6–1.9	14.1–205.8
Bush rat (*Rattus fuscipes*)	1.13	0.6–5.1	0.7–24.8
Black rat (*Rattus rattus*)*	0.76	0.8–27.8	2.4–36.5
Reptiles and Amphibians			
Blotched blue-tongued lizard (*Tiliqua nigrolutea*)	336.4	13.3–160.9	14.4–522.5
Bearded dragon (*Pogona vitticeps*)	< 110	15.2	14.9–24.2
Spotted grass frog (*Limnodynastes tasmaniensis*)	~ 60	12.9–77.5	36.8–98.3
Gould's monitor (*Varanus gouldii*)	43.6	24.2–141.2	66.5–292.5
Birds			
Emu (*Dromaius novaehollandae*)	~ 278	1.5–5.8	124
Sulphur-crested cockatoo (*Cacatus galerita*)	3.46	9.9–17.7	9.0–73.7
Australian magpie (*Gymnorhina tibicen*)	9.93	3.6–10.7	5.7–59.5
Wedge-tailed eagle (*Aquila audax*)	9.49	1.0–60.0	8.0–158.5

Only a few of the species for which toxicology data are available have been included (McIlroy 1981; 1982a, b; 1983; 1984; 1985; Meldrum 1957; Marks *et al* 2000; Robison 1970). The median lethal dose is expressed as milligrams of fluoroacetate per kilogram bodyweight (mg kg^{-1}). Time until signs of poisoning and time from exposure until death are given in hours. * Australian target species. [1] Data for *V. vulpes* were obtained using a known lethal dose (0.5 mg kg^{-1}) of fluoroacetate. All other values were obtained during toxicology studies, hence a range of doses were used.

related to dose for some species but not for others (McIlroy 1982a). There may be considerable variation between individuals of the same species given equal doses of poison, in the time from exposure to first signs of toxicosis, the time from exposure until death, and in the signs of toxicosis (Schultz *et al* 1982).

Despite efforts to ensure that baits contain a lethal dose, in the field there is limited control over the intake of poison. The initial concentration of poison in baits can be controlled, but environmental conditions affect the rate of degradation and loss of 1080 over time (McIlroy *et al* 1988; King *et al* 1994; Bowen *et al* 1995). The number of baits, or

amount of bait material, taken by individuals cannot be completely controlled, hence the speed of onset and time to death may be variable. Some baits may be taken by non-target animals for which the median lethal dose differs from that of the target or with substantially different body-weights. In the primary target species in Australia, the time from 1080 exposure until death ranges from 2.4–80 h, given a range of doses (Table 2) and may indicate that time to death may be highly variable in the field, given the constraints on delivering accurate, lethal doses with predictable lethal outcomes. In a highly susceptible species (eg the red fox) animals given a known lethal dose showed no overt signs of poisoning for approximately 4.05 (± 0.86) h; $P < 0.05$ with 1.57 (± 0.46) h; $P < 0.05$) from first visible signs until death (Marks *et al* 2000).

Mode of action

In addition to energy depletion (Peters 1952; Fanshier *et al* 1964) and resulting cellular damage, 1080, by blocking the conversion of citrate in the tricarboxylic acid cycle, also results in the build-up of citric acid in the tissues and blood, which leads to a metabolic acidosis (Peters 1952). This build-up of citrate, along with associated electrolyte disturbances (Chi *et al* 1996), is the likely cause of some of the signs and symptoms of 1080 toxicosis (DuBose 2005; Singer & Brenner 2005). It may be possible to adapt baits in order to ameliorate some of the signs and symptoms resulting from 1080 poisoning, without affecting lethality, but to date there has been little published research in this area (Marks *et al* 2000).

Appearance and behaviour of affected animals

Poisoned animals are sometimes divided into the four categories proposed by Chenoweth and Gilman (1946): class I, where the main effects are on the heart; class II, where both the heart and central nervous system are involved; class III, where the main effect is on the nervous system; and class IV, where there is an atypical response typified by slow, shallow breathing and a slow heart-rate. As noted above, this system of categorisation belies substantial similarities in the signs of fluoroacetate poisoning across a wide-range of vertebrate species (Sherley 2004). It also fails to take into account the sometimes substantial variation observed in individuals of a single species (eg Schultz *et al* 1982). In general, animals that have been poisoned with fluoroacetate initially display a range of signs including lethargy, retching and vomiting, trembling, faecal and urinary incontinence, unusual vocalisations, hyperactivity, excessive salivation, muscular weakness, unco-ordination, hypersensitivity to nervous stimuli, and respiratory distress. Localised nervous signs including tail twitching, twitching or jerking of limbs, twitching of facial muscles, nystagmus, and tetanic seizures, are common, and may progress to generalised convusions. These begin typically as tetanic convulsions before taking on a clonic-tonic form (reviewed in Sherley 2004). Generalised seizures typically occur cyclically, with periods of lucidity in-between (Chenoweth & St John 1947b; Foss 1948; Gajdusek & Luther 1950; McIlroy 1982a; Schultz *et al* 1982). Death may occur either during convulsions or during these lucid periods (Foss 1948). Several of the signs of toxicosis listed above are potentially painful and/or distressing.

Experiences of human victims

Several cases of 1080 poisoning in humans have been reported, although few address patient perceptions, most focusing on the overt signs of poisoning. This partly reflects a rapid deterioration in the condition of most patients by the time they have obtained medical assistance. Anxiety, irritability, verbosity, agitation, confusion, nausea, vomiting, faecal incontinence, tetanic spasms, cardiac irregularity, gradual loss of alertness culminating in coma, epileptiform convulsions with periods of lucidity between convulsions, and partial paralysis are all commonly described in human fluoroacetate-poisoning cases (reviewed in Sherley 2004). Gregory (1996) argues that people do not experience pain, referring to three case studies (Williams 1948; Gadjusek & Luther 1950; Reigart 1975). Of these, Gadjusek and Luther (1950) report on a toddler who was brought to hospital already in a comatose state and hence unable to communicate any pain he may previously have experienced. Reigart (1975) describes an unusually mild case of poisoning in an eight month old girl who, given her age, was similarly unable to verbally report pain (she was described by the author as anxious, agitated and irritable, but not distressed). Williams (1946) reported that after accidentally inhaling 1080 powder he experienced immediate sensations of tingling and numbness at the site of exposure but did not notice any pain during the period of onset. He experienced what he described as a "sour stomach", and suffered from headache for a period of five days following exposure. Other human case studies have reported pain, including epigastric pain, headache, and localised pain associated with muscular spasms (Brockman *et al* 1955; Peters *et al* 1981; Chung 1984). A more recent survey by Chi *et al* (1996) found that 26% of patients reported abdominal pain while 74% experienced nausea and vomiting, 29% experienced diarrhoea, 29% reported feelings of agitation and 21% reported respiratory distress.

Long-term effect on survivors

Sub-lethal amounts of fluoroacetate are rapidly metabolised and excreted by affected animals and there is little evidence of harm resulting from long-term, low-level exposure to fluoroacetate (Eason & Turck 2002). Complete recovery in survivors of sub-lethal 1080 toxicosis may take from a few hours to several days (Chenoweth & Gilman 1946; McIlroy 1981, 1982a, 1983). Neurological complications including weakness, convulsions and partial paralysis (especially of the hind limbs) are common and may persist for prolonged periods (Gajdusek & Luther 1950; Chenoweth & Gilman 1964; McTaggart 1970; McIlroy 1981, 1982a, 1983; Schultz 1982). For example, both feral pigs (*Sus scrofa*) and frogs have been described as remaining partially paralysed 24 hours after exposure to fluoroacetate (Chenoweth & Gilman 1964; McIlroy 1983). Animals may have an extruded tongue and/or penis during generalised convulsions, and have been described as moving significant

distances while convulsing (McIlroy 1981; Marks *et al* 2000; Potter *et al* 2006), thus there may be an opportunity for serious injuries to occur during fitting. Convulsions may occasionally be severe enough to cause physical injury themselves. For example, in a study of fluoroacteate poisoning in rats (*Rattus rattus*; Egekeze & Oehme 1979b), one rat convulsed so severely that blood began to drip from its eyes.

One of the most detailed descriptions of recovery from 1080 poisoning is McTaggart's (1970) report of a child who ingested rabbit bait: Ten days after ingesting 1080, the boy began to recover during hospitalisation. He was able to keep his eyes open, and to appreciate some movement, but had marked hypertonicity of all limbs with frequent spasms of his arms and legs. At this time he was incapable of spontaneous movement, and remained unable to feed himself for a full two weeks after regaining consciousness. Twenty-four days after he ingested 1080, the boy had regained some range of movement in his arms and was able to recognise familiar people and objects. Ten years later there was evidence of mental retardation with a verbal IQ of 65, he was still unable to walk without crutches, and suffered from tetraplegia, hypertonicity of all limbs, cogwheel rigidity of the wrists, moderate to severe cortical blindness, divergent squint, and epilepsy. It is likely that the mental retardation was the result of brain damage caused by anoxia during periods of fitting (Pridmore 1978), although there is evidence of brain damage resulting directly from fluoroacetate poisoning (Trabes *et al* 1983). Other patients have experienced a similar prolonged recovery period (5–6 days) (Gajdusek & Luther 1950; Robinson *et al* 2002) with complete eventual recovery. Pneumonia is a frequent complication of human 1080 poisoning cases (Williams 1948; Brockman *et al* 1955; Pridmore 1978; Ramirez 1986). While these infections may reflect hospital intervention, it is also possible that infection is promoted because of the increased respiratory secretions typical of 1080 exposure (Chenoweth & Gilman 1946; Quin & Clark 1947; Brockman *et al* 1955; McIlroy 1981, 1982a, 1983, 1984, 1985).

Welfare risk to non-target animals

A wide range of vertebrate species are susceptible to fluoroacetate poisoning, including eutherian and marsupial mammals, birds, reptiles and amphibians (reviewed in Sherley 2004) however, the median lethal dose varies substantially between species, with canids generally the most sensitive, followed by other carnivores, then herbivores, birds, and finally reptiles and amphibians (McIlroy 1986; Table 2). In some areas of northern and western Australia fluoroacetate is produced naturally by native vegetation and some local populations of native animals have co-evolved a tolerance to this poison that sometimes greatly exceeds that found in conspecifics in eastern Australia (McIlroy 1982a; Twigg 1994).

Given that all vertebrate species are potentially susceptible to 1080 poisoning in a dose-dependent manner, the risk to non-target species during 1080 baiting is determined by risk of exposure to 1080 during baiting and the hazard that 1080 poses after exposure. These factors are influenced by the target specificity of the method used to deliver the poison, the dose of 1080 used in the bait and the amount consumed, the comparative median lethal dose for the target species compared to likely non-target species and the relative bodyweight of the non-target species.

In Australia a wide range of strategies are used to limit the exposure of non-target species of this poison applied to baits including (from Sharp and Saunders undated; Biodiversity Group Environment Australia 1999; Government of Western Australia 2002; Williamson & Bloomfield 2003):

- Consideration of the size, colour, material and placement of baits so that non-target species are less prone to discover them.
- The use of tough, dried meat baits for canids.
- The use of large baits containing a precisely determined, known lethal dose for canids.
- Wide dispersal of baits to minimise caching by canids.
- The burying of baits intended for canids.
- Timing of baiting campaigns to avoid periods when food sources for most non-target species are scarce.
- Pre-feeding to maximise bait uptake in the target species.
- The use of dyes to reduce attractiveness of baits to birds.
- The use of bait refuges that allow bait consumption by rabbits but exclude many non-target species.
- The regular removal of uneaten baits, and removal of the carcasses of dead rabbits during rabbit baiting campaigns.

Saunders *et al* (1995) argues that the relative sensitivity of foxes to 1080 is an important advantage of 1080 as it improves the target-specificity of lethal baiting. Yet many other vertebrate pests against which 1080 baits are used in Australia have similar sensitivities to 1080 as other non-target species (Table 2). Furthermore, as foxes are relatively large in comparison with some non-target species, those species for which the median lethal dose is comparatively high may still be at risk if they have a substantially lower bodyweight (McIlroy *et al* 1986). Although the risk of death may be lower for species with a high tolerance to 1080 and/or a large bodyweight, a sub-lethal dose can have impacts upon the welfare of animals that survive, as previously discussed.

Buried predator baits can be excavated and consumed by native rats, southern brown bandicoots (*Isoodon obesulus*; Fairbridge *et al* 2000), brush-tailed phascogales (*Phascogale tapoatafa*; Fairbridge *et al* 2003) and quolls (*Dasyurus* spp) (Glen & Dickman 2003a,b). A reduction in the abundance of *Antechinus* spp (small, carnivorous, mouse-like mammals native to Australia) after 1080 trail-baiting for wild dogs has been reported (McIlroy 1982c). The use of poison meat baits in urban and semi-urban areas is inappropriate because of the risk associated with bait movement and accidental poisoning of domestic animals (Meenken & Booth 1997; Van Polanen Petal *et al* 2001). A study of 1080 baiting for wild dogs in south-eastern

Australia found that approximately 45% of uncoloured, unburied, fresh-meat baits, and 20% of green baits, was taken by birds. Less than 10% of either bait type was taken by the target species over an 18-day period (McIlroy *et al* 1986). The M-44 ejector is an alternative means to deliver toxicants to foxes and dogs as only larger species capable of pulling a lure with enough force to trigger the ejection of toxicant will be exposed (Marks *et al* 2004). The device is estimated to exclude 26/31 mammals that may otherwise be exposed to conventional meat baits that have toxicants directly injected into them (Marks & Wilson 2005). Given the large concentrations of 1080 required to achieve a lethal outcome in feral pigs, McIlroy (1983) estimated that of 40 non-target species likely to consume pig baits, all but one could consume enough bait to be poisoned. In south-eastern Australia 35 endemic mammals are considered to be capable of consuming meat baits used for fox and wild dog control and being exposed to the toxicants they contain (Marks 2001).

Regardless of all possible care taken with bait preparation and deployment, there is a potential risk to a range of non-target species of using 1080 baits. It is appropriate that the welfare risks of baiting practices for non-target species are considered as a part of a general assessment of the humaneness of 1080 baiting.

Conclusions

Previous assessments of the humaneness of 1080 have failed to adequately address welfare issues such as distress. They focus on the difficulties surrounding the interpretation of pain states in the late stages of poisoning, with little regard for earlier stages of toxicosis. The extensive CNS disruption in the late stages of 1080 poisoning poses a dilemma as abnormal electrical activity in the brain makes judgements regarding consciousness and perception difficult to make, and CNS involvement in the toxicosis may alter behavioural indicators of pain and distress. As the involvement of the CNS is progressive, an assessment of humaneness thus becomes more difficult to make as poisoning progresses.

In the initial stages of 1080 poisoning, animals display a range of signs that potentially cause them distress, or are indicative of distress. Conscious human patients who have ingested 1080 frequently report pain and anxiety at this time. A majority of species develop nervous involvement including inco-ordination, partial paralysis, and tetanic convulsions (rigid contractions of the muscles). The potential for suffering is probably greatest during this period of toxicosis. In some species neurological involvement may further progress to generalised convulsions that are typically cyclic with periods of awareness between fits. The degree of awareness during fits is difficult to assess but at least one author indicates that some animals remain conscious during fitting (Foss 1948). Overall, the period from ingestion of 1080 to death can range from less than an hour to several days, with a similarly wide-ranging symptomatic period. Survivors may suffer from partial paralysis or other nervous conditions for a period of several days before they fully recover and permanent neurological damage is a possible outcome. The extent of sub-lethal debilitation that results from 1080 baiting programmes is unknown. Apart from the possibility of pain and distress occurring in the initial stages of poisoning, or convulsive episodes, there are important welfare concerns associated with prolonged periods of repeated convulsions. Animals may experience confusion and distress during the onset of generalised convulsions before the entire cortex has become involved (Chenoweth & St John 1947b; Ward 1947) and in periods of lucidity between convulsions. Given the severity of convulsions and potential for movement during convulsions that has been observed (Egekeze & Oehme 1979a,b; McIlroy 1981; Marks *et al* 2000; Potter *et al* 2006), there is a potential for injury to occur during fitting, and affected animals experiencing periods of conscious awareness between convulsions or eventually recovering from a sub-lethal dose of 1080 may be capable of suffering as a result of any injuries sustained. A poison that caused death more rapidly, or with less opportunity for injury, would clearly be more desirable from a welfare perspective. Para-aminopropiophenone (PAPP) has been shown to produce a much more rapid death in red foxes that is not associated with many of the signs that may be indicative of distress during 1080 toxicosis (Marks *et al* 2004). There is some scope for reducing the severity of signs and symptoms associated with 1080 toxicosis by combination with other pharmacologic agents that could be co-administered in a bait and mitigate distress experienced by poisoned animals (Marks *et al* 2000).

Mason and Littin (2003) argue that the most desirable poisons have a minimum number of symptoms before rapid loss of consciousness and death, with no lasting ill-effects on the survivors. Sodium fluoroacetate does not clearly meet these criteria and it is inappropriate to claim that 1080 is a humane poison based upon prior reviews that fail to consider wider welfare impacts and do not use a consistent framework for assessing humaneness. Given the widespread use of this poison in countries such as Australia and New Zealand, research into alternative control methods and/or improving the humaneness of 1080 baits should be made a priority.

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