Australian
VETERINARY
JOURNAL

CLINICAL SECTION

REVIEW

Sodium monofluoroacetate (Compound 1080) poisoning in dogs

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Sodium monofluoroacetate (Compound 1080) is a widely used pesticide for control of feral animals such as the fox. Accidental poisoning of domestic animals occurs despite strict regulations on 1080 usage. Dogs are particularly susceptible to the toxin.

The mechanism of 1080 toxicity, susceptibility of target and non-target species, persistence of 1080 in the environment and risk of accidental poisoning are discussed. Particular emphasis is placed on 1080 toxicity in the dog. Early recognition of intoxication is most important for prognosis and relies upon characteristic clinical signs and diagnostic findings.

The treatment of 1080 intoxication remains a challenge with no proven antidotes. However, there are possible benefits from monoacetin, acetamide, calcium salts, colestipol, activated charcoal, peritoneal dialysis, sodium bicarbonate, neurotransmitter modulators and four-methylpyrazole. A recommended treatment protocol for 1080 toxicosis in dogs is included.

Safety measures such as the use of wire dog muzzles and investigating alternatives to 1080 in pest control programs may be the key to reducing the incidence of future accidental poisonings.

Aust Vet J 2005;83:474-479

In Australia, sodium monofluoroacetate (Compound 1080) has been regarded as the most efficient and target specific pesticide available for pest animal control since the early 1960s. Today, 1080 continues to be widely used in baiting programs worldwide to protect agricultural production and native flora and fauna from the impact of introduced and feral animals such as the fox.[1] Community concerns over the abundant evidence that predation by foxes is a major threat to native fauna has recently led to baiting programs in the Sydney region.[2] The deliberate introduction of foxes into Tasmania has also initiated recent action to eradicate this species before the start of their breeding season.[3] Farmers, biologists and conservationists agree that use of 1080 baits in conjunction with shooting and trapping is likely to be the best mode of eradication.[3]

Compound 1080 can only be supplied by accredited local government officers, and its use is restricted to trained operators adhering to strict guidelines set down in the Health (Drugs and Poisons) Regulations 1996.[1] Unfortunately, despite such guidelines, accidental poisoning of domestic animals does occur. Dogs are particularly vulnerable as they are the most susceptible species to the toxin,[4,5] and often live in or near areas where baiting occurs. Poisoning can occur directly from an animal ingesting a bait, or following the ingestion of a poisoned carcase.[6] However, the frequency of 1080 poisoning in dogs in Australia is not known.

1080	Compound 1080/Sodium monofluoroacetate/fluoroacetate
CNS	Central nervous system
ECG	Electrocardiogram
GABA	Gamma-aminobutyric acid
GIT	Gastrointestinal tract
LD50	Median lethal dose
LD80	Dose that will kill 80% of the tested group
TCA	Tricarboxylic acid cycle/Krebs cycle

The treatment of 1080 intoxication remains a challenge to emergency physicians and veterinarians alike. The rapid onset and progression of clinical signs leads to a poor prognosis, and mortality rates of 40% in humans and 75% in dogs have been reported.[7] Various techniques such as induction of emesis, gastric and peritoneal lavage, and administration of adsorbents, including activated charcoal, colestipol, and anion-exchange resins, have been recommended after 1080 ingestion,[7-10] however limited controlled data exists on their efficacy. A variety of potential antidotes has also been investigated, primarily in rats and mice, including glycerol monoacetate,[11-13] actetamide,[14] calcium salts (alone[15-17] and in combination with sodium α-ketoglutarate and sodium succinate[18]), sodium bicarbonate,[19] neurotransmitter modulating agents,[8-10,20] and 4-methyl pyrazole.[21] Unfortunately, no single therapy has proven to be an effective treatment for 1080 toxicosis in the dog.

This review discusses the use of 1080 in Australia with particular emphasis on the risks to domestic companion and working dogs, the importance of early recognition of toxicity, and the clinical syndrome of 1080 toxicity. Past and current therapeutic regimens are also discussed.

The toxin

Source

Compound 1080 has been manufactured as a pesticide since the 1940s. It is a white, odourless, tasteless, water soluble toxin, with a high degree of adsorption to cellulose. For these reasons it has been incorporated into many different forms of bait and livestock protection collars worldwide.[1,4]

Monofluoroacetate is found naturally in close to 40 species of Australian plants including *Acacia, Gastrolobium* and *Oxylobium* spp.[22] Monofluoroacetate also occurs in very low concentrations in common food constituents such as tea leaves and guar gum.[1]

Mechanism of toxicity

The mechanism of action of 1080 is thought to be related to disruption of cellular energy metabolism.[4] Compound 1080 can be absorbed from the gastrointestinal and respiratory tracts as well as across mucous membranes and abraded skin. Once absorbed, fluoroacetate combines with acetyl CoA and is metabolised to fluorocitrate. Fluorocitrate is subsequently converted to 4-hydroxy-*trans*-aconitate (HTn). The latter enzyme binds and inactivates aconitase resulting in inhibition of citrate oxidation.[23] This inhibits the TCA (Krebs) cycle, resulting in cellular energy depletion, citric acid and lactic acid accumulation, and interference with cellular respiration and metabolism of carbohydrates, fats and proteins[4] (Figure 1). Organs with cells with a high metabolic rate, such as the heart, brain and kidneys, are most susceptible to malfunction.[24]

In addition to blockade of the TCA cycle, citrate accumulates within blood to toxic concentrations and binds to serum calcium.[4] This binding of calcium results in a significant decrease in serum ionised calcium.[17] The extent of this decrease is correlated with prolongation of the Q-T interval on ECG trace recordings.[15,17] A prolonged Q-T interval is known to be associated with life threatening ventricular arrhythmias, and is reported as the major cause of death in experimental animals and humans with fluoroacetate poisoning.[12]

One final mechanism of 1080 toxicity may be that high citrate concentrations can inhibit the glycolytic enzyme phosphofructokinase, which further impairs cellular energy metabolism.[25]

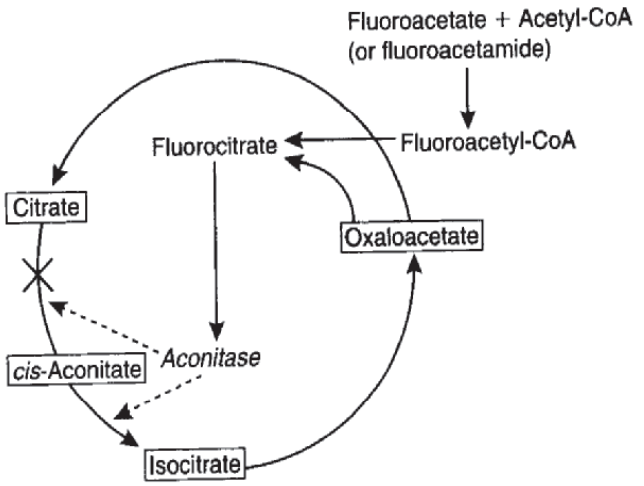

Figure 1. Mechanism of action of 1080 involves disruption of the TCA cycle and accumulation of citrate (reproduced from figure 53-1).[4] Reprinted with permission.

The end result of these widespread cellular events once sufficient fluoroacetate has been metabolised to fluorocitrate is multi-organ energy depletion and dysfunction, particularly of the cardiovascular, central nervous and gastrointestinal systems, which eventually results in death of the animal.

Susceptibility of target and non-target species to 1080

There is much variation in species susceptibility to 1080 poisoning (Table 1). The exact reason for this variation is not fully understood. Carnivores are particularly susceptible with oral lethal doses of 0.05 mg/kg reported in the dog, whereas birds are very tolerant of 1080 ingestion.[4]

An animal's sensitivity to 1080 is a key factor in susceptibility and is usually expressed as the median lethal dose (LD50), or dose per individual that will kill 50% of a population.[5] Sensitivity can, however, vary according to the animal's age or health, or whether its ancestors developed tolerance to 1080 through feeding on plants containing fluoroacetate.[5] This consideration is particularly important in regions of Australia where fluoroacetate occurs naturally.[26] Body size and ambient temperature also appear to be important factors. Smaller animals often are more tolerant to 1080, while high or low ambient temperatures affect metabolic rate, resulting in variably high and low sensitivity to 1080 respectively.[5,27]

Persistence of 1080 in the environment and risk of accidental poisoning

As 1080 is one of the most toxic pesticides known, there is concern about its fate in the living and non-living environment, its toxic effects on non-target species, and the risks of secondary poisoning.[25]

The persistence of 1080 in baits and in the environment is highly dependent on environmental factors. For example, 1080 toxicity and longevity is maximised in cool dry conditions. The compound is highly water-soluble and thus readily leaches from baits. The liberated 1080 is then trapped by cellulose material in the soil and subsequently becomes degraded by at least 20 different species of soil micro-organisms such as *Pseudomonas* spp. However, results of several independent studies conclude that there is no evidence of 1080 persisting in or contaminating soil or

Table 1. Compound 1080 toxicity for various species.[4]

Species	Oral Lethal Dose (mg/kg)	LD50 (mg/kg)
Dog	0.06 - 0.20	0.05 - 1.00
Cat	0.30 - 0.50	0.20
Cattle/Sheep/Goats	0.15 - 0.70	0.25 - 0.50
Possum	0.30	N/A
Human	2.00 - 5.00	0.70 - 2.10
Rodents	N/A	2.00 - 8.00
Pigs	0.30	0.40 - 1.00
Horse	0.50 - 2.75	0.35 - 0.55
Rabbit	0.80	N/A

LD50 = Median lethal dose

waterways in Australia or New Zealand following the completion of baiting campaigns.[25,28]

Secondary poisoning of non-target species through ingestion of carcases has been investigated through study of 1080 residues in rabbits following lethal and sub-lethal dosing.[29] When rabbits ingest a sub-lethal dose of 1080, complete elimination from tissues within approximately 6 hours is reported, posing a limited risk to non-target species. However, plasma 1080 concentration in rabbits dosed with a lethal dose may be significantly higher than the LD50 for a dog. It should be noted that this experimental study was conducted under favourable conditions for 1080 breakdown. It is also possible that lethal baits may remain partly undigested in the stomach of animals after death. This suggests that secondary poisoning from the consumption of a recently poisoned animal may be a significant risk, especially for non-target carnivores such as the dog.[29]

There is no specific data on the frequency of accidental poisonings in Australia. However, a summary of the number of reported cases of accidental 1080 poisonings as recorded by the Animal Health Laboratory Network in New Zealand is given in Table 2. It is important to note that the number of tests requested and the number of confirmed poisoning cases were higher for dogs than other species. The majority of poisonings occur when livestock or pets wander into poisoned areas, when stocked paddocks are accidentally poisoned after an aerial drop, or when stock are returned to poisoned areas within 6 to 8 weeks. However, some cases have been reported to occur after the elapse of the withholding period, especially in areas with frequent extended dry weather conditions.[6] These results should be interpreted in light of the level of 1080 usage in Australia and New Zealand. Compound 1080 baits have been used in New Zealand at levels up to 2 to 4 tonnes of active ingredient per year compared with 200 kg active ingredient per year in Australia. Therefore, sowing rates of 1080 for possum control in New Zealand is over 1000 times higher than rates in Australia for the control of foxes, wild dogs, feral pigs and rabbits (4500 to 7500 mg/ha vs 0.375 mg/ha respectively).[28] It is difficult to postulate whether the sowing rates of 1080 in the two countries would reflect the occurrence of accidental poisoning due to differences in bait formulation and distribution. This highlights the need for further studies into the prevalence of 1080 intoxication in Australia.

Table 2. Confirmed 1080 poisonings reported in New Zealand between 1980-1992.[6]

Year	Dogs	Cattle[a]	Sheep[a]	Others[b]
1980	3	0	0	0
1981	6	0	1	1
1982	11	3	4	0
1983	6	1	2	0
1984	7	5	4	2
1985	15	8	0	1
1986	8	1	1	3
1987	9	4	2	2
1988	10	4	6	6
1989	8	3	9	0
1990	4	2	9	0
1991	16	3	5	1
1992	17	12	3	5
Total	120	46	46	21

[a]Refers to outbreaks, not numbers of animals
[b]Pigs, cats, goats, horses, deer and birds

Table 3. Progression of clinical signs in canine 1080 poisoning.

Stage of disease	Clinical signs[4,24,30-32]
Initial	Anxiety/frenzied activity; running; howling; hyperaesthesia and non-responsive to external stimuli
Mid/progressive	Excessive salivation; vomiting; inappropriate urination/defaecation; tenesmus and hyperthermia
Late/terminal	Tonic/clonic convulsions; collapse/obtunded; dyspnoea and cardio-respiratory arrest

Clinical signs

Clinical signs of 1080 toxicity in the dog are characterised by CNS excitation and GIT hypermotility.[4] Early signs include anxiety, running, howling or other frenzied activities, and hyperaesthesia. Dogs are often non-responsive to external stimuli during these agitated episodes. Convulsions of a tonic and clonic nature soon ensue and can occur between periods of abnormal or normal behaviour.[4,30] Gastrointestinal signs of excessive salivation, vomiting, and defaecation are often observed, and tenesmus has been reported.[4,30-32] Inappropriate urination and hyperthermia may also occur.[32]

The onset of clinical signs may occur between 30 minutes and 2 hours following ingestion and is dose dependent. The rate limiting step that determines the latent period is probably the conversion of fluoroacetate to fluorocitrate and its accumulation to toxic plasma concentrations.[4,24,31,32]

Progression of signs is usually rapid with the animal becoming weaker as cellular energy levels decline and anoxia from convulsions leads to respiratory distress. Death may result from ventricular tachycardia and fibrillation and/or respiratory failure secondary to pulmonary oedema or bronchopneumonia, and usually occurs from 2 to 12 hours after the onset of clinical signs (Table 3).[4,24]

Diagnosis

Diagnosis of 1080 poisoning is usually made on the basis of characteristic clinical signs in conjunction with known access to the poison.

Changes in serum biochemical parameters are non-specific, and include elevations in the concentrations of blood glucose (two fold increase has been reported[33]), urea, creatinine, and the activity of glutamic pyruvic transaminase. Metabolic acidosis and hypokalaemia have also been reported.[24] Increases in serum citrate concentrations over two to three times greater than the reference range may occur.[33] High serum citrate concentrations result in increased calcium binding and therefore decreased serum calcium. In cats, ionised calcium rather than total calcium is decreased.[17,34] The magnitude of hypocalcaemia is correlated with prolongation of the Q-T interval on ECG tracings.[17] Additionally, non-specific abnormalities in the S-T and T wave as well as arrhythmias, ranging from various atrio-ventricular blocks to intractable ventricular fibrillation, have been found in confirmed cases of 1080 toxicity.[17,24,34]

Samples of suspected bait, vomitus, stomach contents or gastric lavage fluid from affected animals can be analysed for fluoroacetate with a minimum of 50g material required.[30,31] Detection of any fluoroacetate in the latter samples is indicative of exposure. However, the analysis is complicated, time consuming, only available from selected laboratories,[a] and requires rapid sample processing because 1080 is metabolised rapidly by bacteria.

Animals that have ingested a minimum lethal dose of 1080 are unlikely to have detectable concentrations in most body tissues.[31] Although minute amounts of sodium monofluoroacetate in blood plasma and serum, urine and adipose tissue, can be detected using high-performance liquid chromatography, such analysis is not clinically useful due to the rapid progression of clinical signs, expense of testing, and time for analysis.[35,36]

Differential diagnoses

Conditions associated with sudden onset of convulsions, CNS excitation and signs of GIT dysfunction must be considered as differential diagnoses for 1080 intoxication. These include intoxication with strychnine, chlorinated hydrocarbons, lead, cardiac glycosides, taxine (Japenese Yew) and methylxanthines, as well as conditions resulting in hypomagnesaemia or hypocalcaemia. Whilst these conditions may cause varying degrees of CNS stimulation, tremors, GIT signs or sudden death, it would be uncharacteristic to see the frenzied, vocalised excitation usually associated with cases of 1080 toxicity.[31]

Necropsy findings

Gross post mortem changes in carnivores poisoned with 1080 are non-specific, with a rapid onset of rigor mortis. Reported findings include generalised cyanosis, congestion of visceral organs, myocardial petechiae, and pulmonary congestion.[4,30] An empty stomach, signs of enteritis, and a flaccid, pale heart in diastole have also been reported.[4] Histopathological changes in brain parenchyma include oedema and lymphocytic infiltration of perivascular tissue.[32]

Due to the non-specific changes found, necropsy examinations are usually of little value in determining the cause of death, unless they can be associated with 1080 baits or toxic plants in gastric contents, or fluoroacetate residues in blood or tissue.[25]

[a]IDEXX Laboratories can provide this service in Australia with an approximate turn around time of 3 weeks.

Treatment

Many studies have been conducted on treatment regimens for 1080 toxicosis. Although most of the studies are experimental or based on human poisoning cases, key findings will be discussed in view of possible extrapolation to the dog.

Monoacetin and acetamide

Initially, monoacetin (glycerol monoacetate) was primarily used as an antidote in fluoroacetate poisoning[11,12] because it was believed that acetate exerted a protective effect via competition with fluoroacetate for binding with coenzyme A.[13] Monoacetin has since been superceded by acetamide therapy, which is thought to have a similar mechanism of action.[14]

Administration of acetamide is an effective treatment in the early stages of 1080 poisoning in rats.[14] A suggested therapeutic regimen in dogs is outlined later in this review.

Calcium salts

As previously discussed, hypocalcaemia may play an important role in manifestation of clinical signs and the potentiation of cardiac arrhythmias. Supplementing calcium to poisoned animals may prolong the mean survival time of 1080 poisoned animals,[11,17] although in mice, calcium gluconate alone does not improve survival time.[18] Similarly, sodium succinate and sodium 2-ketoglutarate, which may act as substrates for the TCA cycle, are individually ineffective therapies. However, a significant antidotal effect is found with combined calcium gluconate (130 mg/kg) and sodium succinate (240 mg/kg) therapy. It is postulated that increased urinary excretion of citrate due to combined calcium gluconate and sodium succinate administration may contribute to their beneficial effects, although this was not measured in the study.[18] Clinical trials are yet to be undertaken in dogs, although monitoring of serum calcium concentrations and appropriate supplementation is currently warranted.

Reducing 1080 absorption-colestipol, activated charcoal and peritoneal dialysis

Both activated charcoal and colestipol are effective at binding 1080 in vitro and colestipol reduces 1080 serum concentrations by 50% during the first 4 hours after 1080 exposure in rats.[8] This result, although supported by another study,[7] was not reproducible in dogs. Colestipol reduces mortality in rats when administered 30 minutes after 1080 dosing,[7] however, neither compound is able to reduce absorption sufficiently to affect survival in rats given a high dose of 1080.[8]

Peritoneal dialysis is not effective at reducing 1080 blood concentrations in dogs dosed orally with 1080 even though substantial toxin is recovered in the dialysate.[8]

Neurotransmitter modulators

GABA agonists alone control seizure activity but have no effect on survival of rats given 1080.[9] Delivery of a combination of neurotransmitter modulating agents directly into relevant brain regions significantly improves survival, probably mediated through amelioration of the toxic effects of 1080 on neuronal viability.[9,10] In support of the latter finding, a mixture of neurotransmitter modulating agents (including glutamate and nitric oxide inhibitors, calcium and sodium channel blockers, GABA agonists, kappa opioid agonists, dopaminergic and serotonergic agonists, various neurotrophic growth factors and non-steroidal anti-inflammatory drugs) when administered orally in rats, sheep and chickens, provides significant protection from the lethal

Figure 2. Treatment Protocol for 1080 toxicity.[31]

- Induce emesis if the animal is fully conscious
- Gastric lavage and oral administration of activated charcoal or colestipol
- Control seizures and convulsions with pentobarbital sodium or gaseous anaesthetic agents
- Gain vascular access and commence fluid therapy with 0.9% NaCl.
- Infuse 300mg/kg sodium bicarbonate (8.4%w/v) as a continuous rate intravenous infusion over 15-30mins OR give ½ the calculated dose as a bolus and the remainder infused slowly.
- Monitor serum calcium and potassium concentrations and supplement as required
- Maintain fluid therapy and seizure control until clinical signs have resolved (12-18 hrs). Animals can usually be discharged within 48h of presentation.

If ACETAMIDE is available, instead of the sodium bicarbonate infusion

- Dissolve 15g acetamide granules in 1 L warmed 5% glucose solution. Administer 10mL/kg intravenously over 15min then reduce rate to 8mL/kg/h until the first litre is infused. Additional acetamide should be formulated and administered at 5mL/kg/h until resolution of clinical signs is achieved.

Note- Higher doses of acetamide (10% acetamide in 5% dextrose) have been used in experimental work in rats and in humans, however, this is more than double the dose found to be 85% effective in the treatment of dogs [31]

effects of 1080.[8,20] This beneficial effect can be augmented in rats and sheep, but not in chickens, with the addition of pyrrolopyrimidines to speed transfer of the antidote across the blood brain barrier.[20] This antidote is aimed at controlling the cascade of secondary effects that can occur during intoxication, potentially allowing a longer period between intoxication and therapeutic success.[20] The margin of safety with use of the mixture is small, as treatment at two to four times the therapeutic dose increases mortality in 1080 exposed rats.[8] The individual agents and their potential as 1080 antidotes have not been investigated in dogs and require further study.

Sodium bicarbonate

Anecdotally, use of large doses of sodium bicarbonate, as a continuous rate infusion of 300 mg/kg over 15 to 30 minutes, may aid survival of dogs with a history of access to the poison and advanced clinical signs of toxicosis.[19] Although the latter report referred to only five of six dogs, such promising results warrant further investigation with clinical trials.

Four-methylpyrazole and Gliftor®

In Australia and New Zealand, research is underway investigating the potential benefits of 4-methylpyrazole (indicated for treatment of ethylene glycol poisoning) in the treatment of 1080 toxicity. Administration of 4-methylpyrazole in rats reduces oxaloacetate production via inhibition of malate dehydrogenase, which then reduces (-) erythrofluorocitrate production from fluoroacetate.[21] Although this reduction does not significantly decrease serum citrate concentrations in rats dosed with combined 1080 and 4-methylpyrazole versus control rats, dosed with 1080 alone, the clinical manifestations of toxicity are decreased. In addition, 4-methylpyrazole can act as an antidote for another pesticide, 1,3-difluoro-2-propanol (Gliftor®), which has an analogous mechanism of action to fluoroacetate.[21] Four-methylpyrazole is effective in eliminating fluoride and citrate accumulation when administered as a prophylactic in rats given two times a lethal dose of Gliftor® and also when given 2 hours after poisoning.[21] Gliftor® has therefore been suggested as a potential pesticide replacement for 1080, with the added advantage that 4-methylpyrazole is available as an antidote in case of accidental poisoning.[21,37]

Current recommended treatment protocol for 1080 toxicosis

As for any poisoning, basic principles of patient stabilisation, decontamination and supportive care apply to 1080 toxicity. A current recommended treatment protocol is outlined in Figure 2.

Prognosis

The prognosis for the majority of 1080 poisonings is poor to grave depending on the amount of toxin ingested and severity of clinical signs at presentation.[4] In humans, hypotension, metabolic acidosis and increased serum creatinine concentration are poor prognostic indicators,[34] although no such indicators have been proven in animals. Early recognition of 1080 poisoning and appropriate aggressive treatment has been reported to improve the survival rate in 75 to 85% of dogs that are poisoned.[31] No residual effects have been reported in surviving dogs.[31]

Conclusion

Compound 1080 is currently an essential tool in fauna protection programs throughout Australia and the world. Extensive experimental and environmental studies have shown that it is biodegradable in all living systems and does not accumulate in the environment or food chain.

The use of 1080 in Australia is highly regulated by government agencies. Provision of 1080 is allowed on the basis of demonstrated need and strict guidelines are provided on appropriate use. These precautions include detailed staff training, correct bait formulation, restricted use of 1080 to licensed operators, protective clothing, separation of livestock from poisoned areas, and appropriate public notification and awareness of baiting operations.

Despite such stringent regulations accidental poisonings do occur and companion and working dogs are particularly susceptible. Increasing public awareness of the risks of poisoning and recommending the use of wire dog muzzles, supervision of dogs off the lead, and prompt removal and proper disposal of poisoned carcases is most important to prevent accidental exposure. This issue has been addressed with programs coordinated by the Australian Government Pest Animal Control such as the distribution of information booklets for local farmers where extensive 1080 baiting programs are to be carried out. [38]

Over-reliance of baiting programs on 1080 alone warrants more research into adjunctive or alternative pest-control methods. Such an alternative includes the use of sterilised vixens carrying oestrogen implants to keep them constantly in season in order to lure male foxes to trapping/baiting sites.[3] In addition, exploiting the fact that the toxicity of 1080 is increased at low temperatures and that species susceptibility to the toxin varies, baiting in warmer weather in Australia may also restrict toxicity to highly susceptible target animals and reduce risk to non-target fauna. However, this may not be practical and does not reduce the risk to domestic carnivores.[28]

Of all suggested alternatives to 1080 usage, 1,3-difluoro-2-propanol (Gliftor®) is perhaps the most promising. It possesses an analogous mechanism of action but has the distinct advantage that antidotal compounds such as 4-methyl-pyrazole are available to combat accidental ingestion.[21,37]

The rapid clinical progression and poor prognosis associated with 1080 toxicity emphasises the importance of early recognition of the clinical signs of poisoning and rapid implementation of appropriate therapy. Clinical signs of 1080 toxicity in dogs, including frenzied CNS excitation, vocalisation, with GIT hyperactivity, are uncharacteristic for other toxicities and diseases involving the CNS, and when seen in association with known exposure to the toxin, are almost pathognomonic. Unfortunately, the window of opportunity for decontamination is small and studies on various adsorbents have not appeared to improve survivability in dogs when used alone. Due to the complex pathophysiology of 1080 toxicity the best approach to treatment may be a combination of agents such as acetamide, calcium, sodium succinate and bicarbonate. Due to the lack of clinical trials in dogs, veterinary clinicians must currently rely on their own judgment regarding which treatment to administer. Currently, the protocol shown in Figure 2 with either sodium bicarbonate or acetamide is recommended.

In conclusion, despite many studies that have been conducted on 1080 in other species, extrapolation of information to the dog may be inappropriate and further work is needed in this species. The prevalence of 1080 intoxication and the treatment of accidental poisoning need to be clarified, and could be addressed with surveys of Australian practitioners in regions in which 1080 has been regulated for use, and controlled clinical trials. The over-dependence of many pest control programs on 1080 also warrants further investigation of alternative toxins and methods. Ideally, as developments in biological pest control emerge, reliance on such toxins will diminish. Until safer alternatives can be found, the importance of prevention of accidental poisoning, through public education and strict regulations during all 1080 baiting campaigns, is essential.

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(Accepted for publication 15 November 2004)