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SODIUM MONOFLUOROACETATE (1080) HAZARDS TO FISH, WILDLIFE, AND INVERTEBRATES: A SYNOPTIC REVIEW

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TABLES

SODIUM MONOFLUOROACETATE (1080) HAZARDS TO FISH, WILDLIFE, AND INVERTEBRATES: A SYNOPTIC REVIEW

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Abstract. Sodium monofluoroacetate ($CH_2FCOONa$), also known as 1080, was first used in the United States to control gophers (*Geomys* spp.), squirrels (*Sciurus* spp., *Spermophilus* spp.), prairie dogs (*Cynomys* spp.), other rodents (Rodentia), and coyotes (*Canis latrans*); 1080 domestic use is currently restricted to livestock-protection collars on sheep and goats to selectively kill depredating coyotes. However, Australia, New Zealand, and some other nations continue to use 1080 to control rabbits, possums, deer, foxes, feral pigs and cats, wild dogs, wallabies, rodents, and other mammals. The chemical is readily absorbed by ingestion or inhalation. At lethal doses, metabolic conversion of fluoroacetate to fluorocitrate results in the accumulation of citrate in the tissues and death within 24 h from ventricular fibrillation or from respiratory failure; no antidote is available. At sublethal doses, the toxic effects of 1080 are reversible. Primary and secondary poisoning of nontarget vertebrates may accompany the use of 1080. Sensitive mammals including representative species of livestock, marsupials, felids, rodents, and canids died after receiving single doses of 0.05-0.2 mg 1080/kg body weight (BW); most tested species died after a single dose of 1-3 mg/kg BW. High residues were measured in some poisoned target mammals, and this contributed to secondary poisoning of carnivores that ingested poisoned prey. Sublethal effects occurred in sensitive mammals at greater than 2.2 mg 1080/L in drinking water or at 0.8-1.1 mg 1080/kg in the diet. Sensitive species of birds died after a single 1080 dose of 0.6-2.5 mg/kg BW, after daily doses of 0.5 mg/kg BW for 30 days, after 47 mg/kg in diets for 5 days, or after 18 mg/L in drinking water for 5 days. Adverse effects occurred in birds at dietary loadings as low as 10-13 mg 1080/kg ration. Amphibians and reptiles were more resistant to 1080 than birds and mammals. LD50 values were greater than 44 mg/kg BW in tested amphibians and greater than 54 mg/kg BW in tested reptiles; resistance to 1080 was attributed to their reduced ability to convert fluoroacetate to fluorocitrate and their increased ability to detoxify fluoroacetate by defluorination. Mosquito larvae reportedly died at 0.025-0.05 mg 1080/L, but fishes seemed unaffected at 13 mg/L; however, data on 1080 in aquatic ecosystems are incomplete. Acute LD50 values in terrestrial insects ranged from 1.1-3.9 mg/kg BW to 130.0 mg/kg BW in larvae feeding on fluoroacetate-bearing vegetation. Residues of 1080 in exposed insects were usually low (<4 mg 1080/kg fresh weight) or negligible and were usually eliminated completely within 6 days, suggesting low risk to insectivorous birds. Loss of 1080 from baits occurs primarily from microbial defluorination and secondarily from leaching by rainfall and consumption by insect larvae; leachates from 1080 baits are probably held in the upper soil layers. The use of 1080 seems warranted in the absence of suitable alternative control methods.

Key words: Sodium monofluoroacetate, 1080, organofluorines, pesticide, predacide, toxicity, wildlife, livestock, invertebrates.

Sodium monofluoroacetate ($CH_2FCOONa$), also known as 1080 or Compound 1080, belongs to the class of chemicals known as fluoroacetates (Pattison 1959). It is a tasteless and odorless, water-soluble poison of extraordinary potency that has been used widely against rodents and other mammalian pests (Anonymous 1946; Negherbon 1959; Rammell and Fleming 1978; McIlroy 1981a; Hornshaw et al. 1986; Aulerich et al. 1987; Connoly and Bums 1990). The widespread use of 1080 in pest control has caused accidental deaths of livestock, wildlife, pets (cats and dogs), and humans (Anonymous 1946; Chenoweth 1949; Sayama and Brunetti 1952; Negherbon 1959; U.S. Environmental Protection Agency [EPA] 1976; McIlroy 1982a), and several suicides in Asia from drinking 1080 rat poison solutions (Howard 1983). There is no effective antidote to 1080 (Mead et al. 1991). When consumed, fluoroacetate is converted to fluorocitrate that inhibits the enzymes aconitase and succinate dehydrogenase; the accumulated citrate interferes with energy production and cellular function (Aulerich et al. 1987).

Monofluoroacetic acid (CH_2FCOOH) was first synthesized in Belgium in 1896 but attracted little attention from chemists and pharmacologists at that time (Chenoweth 1949; Atzert 1971). In 1927, sodium monofluoroacetate was patented as a preservative against moths (Sayama and Brunetti 1952). The toxic nature of monofluoroacetate compounds was first noted in Germany in 1934 (Atzert 1971). In the late 1930's and early 1940's, Polish scientists conducted additional research on the toxic properties of fluoroacetate compounds, especially the methyl ester of fluoroacetic acid that they had synthesized (Anonymous 1946; Chenoweth 1949). In 1942, British scientists further refined this compound to the sodium salt that became known as 1080 (Anonymous 1946). In 1944, potassium monofluoroacetate (CH_2FCOOK) was isolated from *Dichapetalum cymosium*, a South African plant, and was the first known example of a naturally occurring organic fluoride; the plant, known locally as Gifblaar, caused many deaths of livestock (Chenoweth 1949) and was recognized by Europeans as poisonous as early as 1890 (Peacock 1964). Fluoroacetate compounds have since been isolated from poisonous plants in Australia (*Acacia georginae*, *Gastrolobium* spp.), in Brazil (rat weed, *Palicourea marcgravii*), and in Africa (*Dichapetalum* spp; Atzert 1971). Ratsbane (*Dichapetalum toxicarium*), an African plant, was known to contain a poison--subsequently identified as a fluoroacetate--that was lethal to rats, livestock, and humans and reportedly was used by African natives during the 1800's to poison the wells and water supplies of hostile tribes (Anonymous 1946).

During World War II (1939-45), as a result of acute domestic shortages of common rodenticides such as thallium, strychnine, and red squill, testing for alternative chemicals was initiated (Anonymous 1946). In June 1944, the U.S. Office of Scientific Research and Development supplied the Patuxent Wildlife Research Center--then a laboratory of the U.S. Fish and Wildlife Service--with sodium monofluoroacetate and other chemicals for testing as rodenticides (Atzert 1971). The center gave sodium monofluoroacetate the acquisition number 1080, which subsequently was adopted as its name by the chemical's manufacturer. Samples of 1080 were also shipped to the Denver Wildlife Research Center, another former laboratory of the U.S. Fish and Wildlife Service, for testing on additional species. Results of these tests gave evidence of the value of 1080 as an effective control of animal predators of livestock and other animal pests (Atzert 1971). During World War II, 1080 protected allied troops in the Pacific Theater against scrub typhus, also known as tsut sugamushi, a louse-borne rickettsial disease with rodents as vectors (Peacock 1964). In the United States, 1080 was first used in 1945 to control rodents, and later coyotes (*Canis latrans*) and rabbits (*Lepus* spp.; *Sylvilagus* spp.; Hornshaw et al. 1986; Aulerich et al. 1987). Between 1946 and 1949, at least 12 humans died accidentally in the United States from 1080 poisoning when used as a rodenticide; a child became ill but recovered after eating the cooked flesh of a 1080-poisoned squirrel (EPA 1976). Since 1955, 1080 has been used extensively in a variety of baits--especially in Australia and New Zealand--to control European rabbits (*Oryctolagus cuniculus*), dingoes (*Canis familiaris dingo*), feral pigs (*Sus scrofa*), brush-tailed possums (*Trichosurus vulpecula*), and various species of wallabies (McIlroy 1981a, 1981b, 1982a, 1984; Twigg and King 1991). In Australia, vegetable baits are sometimes eaten by nontarget herbivores such as sheep (*Ovis aries*), cattle (*Bos taurus*), and various species of wildlife and cause primary and secondary poisoning of nontarget animals (McIlroy 1982a). In the United States, most uses of 1080 were canceled in 1972 because, in part, of deaths of nontarget animals (Balcomb et al. 1983). At present, the use of 1080 in the United States is restricted to livestock-protection collars on sheep and goats (*Capra hircus*) against predation by coyotes (Palmateer 1989,1990).

Useful reviews of ecotoxicological aspects of 1080 include those by Chenoweth (1949), Peacock (1964), Atzert (1971), Kun (1982), Twigg and King (1991), and Seawright and Eason (1994). I prepared my account in response to requests for information on 1080 from environmental contaminant specialists of the U.S. Fish and

Wildlife Service. It is part of a continuing series of brief reviews of chemicals in the environment with an emphasis on hazards to plants and animals.

Uses of 1080

The use of 1080 in the United States is now restricted to the protection of livestock--collars on sheep and goats--from predation by coyotes. Other countries, most notably Australia and New Zealand, use 1080 extensively in a variety of baits to control many species of vertebrate pests.

Domestic Use

Compound 1080 is highly poisonous to all tested mammals and to humans (Green 1946). There is no known antidote to 1080, and it has been impossible to resuscitate any animal or human during the final stages of 1080 poisoning (Kalbach 1945; Green 1946; Connolly 1989, 1993a). In the United States, 4 suicides and at least 12 accidental human deaths occurred in 25 years of use of 1080 between 1959 and 1969 and 37 known incidents of domestic animal poisoning resulted from federal use of 1080 (Atzert 1971). Compound 1080 is not recommended for use in residential areas or for distribution where the public may be exposed (Green 1946); only licensed pest control operators can use 1080 (Green 1946; Peacock 1964; EPA 1985; Murphy 1986). Tull Chemical in Oxford, Alabama, is the sole domestic producer of 1080; none is imported (EPA 1985). Operators who handle 1080 should wear protective clothing, including gloves and a respirator; extreme caution is recommended at all times (Green 1946). During attaching, removing, or disposing of livestock-protection collars, each applicator must carry syrup of ipecac to induce vomiting in case of accidental poisoning (Connolly 1989, 1993a).

Compound 1080 was first used in the United States in the late 1940's to control gophers (*Geomys* spp.), ground squirrels (*Spermophilus* spp.), prairie dogs (*Cynomys* spp.), field mice (Muridae), commensal rodents, and coyotes (Chenoweth 1949; Fry et al. 1986). Annual damage from coyotes to livestock in California alone is an estimated $75 million (Howard 1983). Yearly amounts of 1080 used in the United States for predator control were 23 kg in the early 1960's, 7,727 kg in the late 1960's, and only 8 kg in 1971 (Connolly 1982). The total annual production of 1080 in the United States between 1968 and 1970 averaged about 1,182 kg (Atzert 1971). In 1977, 277,545 kg of 1080-containing baits (272 kg of 1080) were used to control ground squirrels (76%), prairie dogs (7%), and mice, rats, chipmunks (*Tamias* spp.), and other rodents (17%); California used 83% of all 1080 baits, Colorado 12%, and Nevada and Oregon 5% (EPA 1985). About 0.3 kg of 1080/year are used in the livestock-protection collars but only about 35 g/year is released into the environment (Connolly 1993b). In March 1972, the use of 1080 for predator control was prohibited on federal lands. Later that year, all uses of 1080 for predator control were banned in the United States because of adverse effects on nontarget organisms including endangered species (Palmateer 1989, 1990). Since 1080 was banned, the number of grazing livestock reported lost to predation in western national forests has increased. Between 1960 and 1971, 1.42% (range 1.0-1.9%) of all grazed sheep and goats were lost to predators versus 2.17% (1.7-2.5%) in 1970-78 (Lynch and Nass 1981). Until it was banned in 1972, the use of 1080 as a control agent of predators in the United States was strictly controlled. The chemical was registered under the Federal Insecticide, Fungicide and Rodenticide Act (61 Stat 163; 7 U.S.C. 135-135K) for use only by governmental agencies and experienced pest-control operators (Atzert 1971). The use of 1080 as a rodenticide was disallowed in 1985 for three reasons; (1) lack of emergency treatment, namely a viable medical antidote; (2) high acute toxicity to nontarget mammals and birds; and (3) a significant reduction in populations of nontarget organisms and fatalities to endangered species (EPA 1985). In 1985, 1080 use was conditionally permitted in livestock-protection collars and in single lethal dose baits; a registration of the livestock-protection collar was issued to the U.S. Department of the Interior on 18 July 1985 (EPA 1985). On 21 February 1989, the registration of 1080 was cancelled, prohibiting all uses. In June 1989, however, technical 1080 was conditionally approved for use only in the livestock-protection collar. The 30-mL collar is registered for use by the U.S. Department of Agriculture; by the states of Montana, Wyoming, South Dakota, and New Mexico; and by Rancher's Supply, Alpine, Texas (Palmateer 1989, 1990).

Compound 1080 was highly effective against all species of rats, prairie dogs, and ground squirrels and satisfactory for the control of mice (Peacock 1964). The chemical was formulated in grain baits or chopped greens for crop and range rodents and in water bait stations to control rats (EPA 1985). The concentration of 1080 in baits was lowered to 0.02% in the range of the California condor (*Gymnogyps californianus*) and for prairie dog control because of possible harm to the endangered black-footed ferret (*Mustela nigripes*; EPA 1985). Commercial 1080 was commonly colored with 0.5% nigrosine and sold as a compound containing

greater than 90% sodium monofluoroacetate, to be mixed with foods at 2,226 mg/kg in preparing baits or to be dissolved in water at 3,756 mg/L for poisoning drinking water in indoor control of rodents (Anonymous 1946; Green 1946; Negherbon 1959). Bait acceptance by rats was not significantly reduced by the dye (Peacock 1964). Compound 1080 was adequately accepted by rats and mice when present in water; solid food baits poisoned with 1080 were not always accepted as readily and sometimes required special preparation to insure the ingestion of lethal amounts (Green 1946). A water solution of 1080 was the most effective tested rodenticide for rat control in southern states, and 1080-grain baits were the most effective field rodenticides against ground squirrels, prairie dogs, and mice in California, South Dakota, and Colorado (Kalmach 1945). Seeds and cereal grains were the most effective baits for small rodents: 1 kg of 1080 was sufficient to kill 3.96 million squirrels (Peacock 1964). Grain baits were colored brilliantly yellow or green to heighten the repellency of birds; coloring did not affect the acceptance of baits by rodents (Peacock 1964; Atzert 1971). Rats did not develop a significant tolerance to 1080 from ingestion of sublethal doses; although rats that survived poisoning may develop an aversion to 1080 (Green 1946; Peacock 1964).

To kill coyotes and gray wolves (*Canis lupus*) in the United States and in Canada, meat baits containing 35 mg 1080/kg were recommended--usually an injected water solution of 1080 into horsemeat baits; only 28-56 g of a poisoned bait was sufficient to kill (Peacock 1964). Meat baits were usually placed in fall in areas with maximum coyote use and minimum use by most nontarget carnivores (Atzert 1971). The most widely publicized technique for poisoning predators was the 1080 large bait station: a 22-45 kg livestock meat bait injected with 35 mg 1080/kg bait (Connolly 1982). The use of 1080 stations peaked in the early 1960's, at which time 15 to 16 thousand stations were placed each winter in the western United States. After 1964, the number of stations declined annually to 7,289 stations in 1971 (Connolly 1982). Against canine predators of livestock, 1080 was more selective and less hazardous than strychnine or traps to nontarget species (Peacock 1964). Meat baits for the control of coyotes were seldom fatal to hawks, owls, and eagles (Falconiformes), even when these birds gorged themselves on the poisoned baits (Peacock 1964). In addition to the large bait stations, an unknown number of U.S. Government hunters used 1080 in smaller baits at various stations (Connolly 1982).

The introduction of 1080-livestock-protection collars to protect goats and sheep against coyote depredation was initiated in 1985; its use was limited to certified applicators (Burns et al. 1991). The 1080-filled rubber collars are attached to the throats of sheep and goats; 1080 is released when coyotes attack collared livestock with characteristic bites to the throat (Walton 1990; Burns et al. 1991). The livestock-protection collars contain 30 mL of a 1% 1080 solution (Walton 1990) and tartrazine (Burns and Savarie 1989; Connolly 1993a) as a marker. The livestock-protection collar may not be used in areas known to be frequented by endangered species of wildlife, and these include various geographic areas in California, Michigan, Minnesota, Montana, Washington, Wisconsin, and Wyoming (Connolly 1989, 1993a). In livestock-protection collars, Compound 1080 is reportedly more effective and safer than sodium cyanide, diphacinone, or methomyl (Connolly 1982). Pen tests with compound 1080 in livestock-protection collars began late in 1976 and field tests in 1978 (Connolly and Burns 1990). In the field, 1080 livestock-protection collars on sheep seem to protect selectively against predation by coyotes; no adverse effects on humans, domestic animals, and nontarget wildlife were recorded (Connolly and Burns 1990). The decision to permit limited use of 1080 in livestock-protection collars is now being contested by at least 14 conservation groups because of its alleged hazard to nontarget organisms (bears, *Ursus* spp.; badgers, *Taxidea taxus*; dogs, *Canis* spp.; eagles, *Aquila chrysaetos*, *Haliaeetus leucocephalus*) and to human health, and to the availability of alternate and more successful methods of coyote control (Sibbison 1984). In Texas, for example, the annual cost from losses of sheep and goats to coyotes are an estimated $5 million. But few Texas ranchers have taken advantage of the opportunity to use livestock-protection collars, and only 23 coyotes were killed in 1989 by the collars versus 473 by cyanide, snares, aerial gunning, and other measures (Walton 1990). Toxic livestock-protection collars in full operation would probably kill fewer than 1,000 coyotes annually versus 1 million coyotes killed annually from hunting and other measures (Sibbison 1984).

Compound 1080 was also effective against jackrabbits, foxes, and moles. Baits containing 0.05-0.1% of 1080 on vegetables were used in California to kill jackrabbits (*Lepus* spp.) and various rodents (Schitoskey 1975). The Arctic fox (*Alopex lagopus*), intentionally introduced onto the Aleutian Islands in 1835 (Bailey 1993), almost eliminated the Aleutian Canada goose (*Branta canadensis leucopariea*) by 1967; 1080-tallow baits were successfully used to control fox populations (Byrd et al. 1988; Tietjen et al. 1988; Bailey 1993). Earthworm baits are used to kill moles (Talpidae). The earthworms are soaked for 45 min in a 2.5% solution of 1080 and placed

in mole burrows. The solution can be used several times for additional lots of worms; however, the use of the manure worm (*Eisenia foetida*) should be avoided because it is seldom eaten by moles (Peacock 1964).

Secondary poisoning of domestic cats and dogs from consumption of 1080-poisoned rodents was frequently noted (Anonymous 1946). Cats and dogs are highly susceptible to 1080 and may die after eating freshly poisoned rodents, dried carcasses, or 1080-baits or after drinking 1080-poisoned water (Green 1946). All pets should be confined or removed from the area to be poisoned and released after the entire control is completed. Pigs and carnivorous wildlife are also at risk from consumption of 1080-poisoned rodents (Peacock 1964). Secondary poisoning of kit foxes (*Vulpes macrotis*) is theoretically possible after eating a single kangaroo rat (*Dipodomys* sp.) that swallowed or stuffed its cheeks with 1 g of a 0.1% vegetable/cereal bait and contained a total whole-body burden of about 1 mg of 1080/rat (Schitoskey 1975). To prevent secondary poisoning, all uneaten baits and carcasses of poisoned rodents should be recovered and incinerated (Green 1946), and no 1080-contaminated animal should be eaten by humans or be fed to animals (Connolly 1989, 1993a).

Nondomestic Use

Compound 1080 has had limited use as a vertebrate pesticide in Canada, India, Mexico, and South Africa and extensive use in Australia (Calver et al. 1989b) and New Zealand (Rammell and Fleming 1978). In Canada, 1080 was first used in 1950-51 in British Columbia to control wolves and coyotes preying on livestock (Peacock 1964). Poisoned 1080 baits were used in India to control (67-100% effective) populations of the Indian crested porcupine (*Hystrix indica*) throughout its range because of porcupine-caused damage and losses to agricultural crops; however, control of this species with 1080 baits was not as effective as fumigants (Khan et al. 1992). In Mexico, 1080 was used against rabid coyotes, although many domestic dogs were also killed (Peacock 1964). Beginning in 1961 in South Africa, 1080 was used to control the black-backed jackal (*Canis mesomelas*) that preyed on livestock, and baboons (*Papio anubis*) and moles that consumed agricultural crops (Peacock 1964). Livestock-protection collars containing 30 mL of a 1% solution of 1080 are now used in South Africa to combat predation by the Asiatic jackal (*Canis aureus*; Walton 1990).

Compound 1080 was first used in Australia in the 1950's to kill the introduced European rabbit (*Oryctolagus cuniculus*). Principal target species in Australia now include other introductions such as dingoes, red foxes (*Vulpes vulpes*), feral pigs (*Sus scrofa*), feral cats (*Felis cattus*) as well as native brush-tailed possums (*Trichosurus vulpecula*), red-necked wallabies (*Macropus rufogriseus*), and pademelons (*Thylogale billardierii*; McIlroy 1981a, 1981b, 1982,1984; Calver et al. 1989a, 1989b; Wong et al. 1991). In Australia, different baits contained different concentrations of 1080; meat baits contained 144 mg/kg, grain baits 288-300 mg/kg, fruits and vegetables 330 mg/kg, and pellets 500 mg/kg (McIlroy 1983a).

One method of killing rabbits in many areas of Australia is to apply 1080-poisoned bait (carrots, oat grains, pellets of bran or pollard) to furrows made in the earth or to broadcast baits across the area from the air or on the ground (McIlroy 1984; McIlroy and Gifford 1991). Aerial dropping of diced carrots treated with 1080 was almost 100% effective against rabbits (Anonymous 1964). In Victoria, more than 6.5 million ha were treated with 1080 poisoned carrots. To attract rabbits to the kill area, nonpoisoned carrots were applied to rabbit trails at more than 8.3 kg/km; nonpoisoned baits were offered twice, 3 days apart, before 1080-poisoned carrots were offered 1 week later (Woodfield et al. 1964). Bait avoidance is reported in some populations of European rabbits exposed repeatedly to 1080 baits during sustained control. Behavioral resistance may reduce the effectiveness of sustained control and should be considered in pest management (Hickling 1994). Individuals--but not populations--of some native species of Australian animals and birds face a greater risk of poisoning by 1080 during rabbit-poisoning campaigns than rabbits, particularly herbivorous macropodids, rodents, and bird species with no prior exposure to naturally occurring fluoroacetates (McIlroy 1992). Foxes, dingoes, dogs, and cats seem to be at greater risk of secondary poisoning than native birds and mammals, particularly from eating muscle from poisoned rabbits that contained as much as 5 mg of 1080/rabbit (McIlroy 1992).

The injection of fresh meat baits for the control of dingoes produced more uniform amounts of 1080 in the baits than tumbling mixed baits in 1080 solutions. Both techniques, however, produced baits containing variable quantities of 1080 (Kramer et al. 1987). Use of 1080-poisoned baits to control wild dogs (*Canis familiaris familiaris*) and dingoes were not as successful as traps: 22% control from 1080 versus 56% control with traps. Factors that reduced the success of poisoned baits included rapid loss in toxicity after distribution of the baits; the rapid rate at which they were removed by other animals, particularly foxes and birds; and the dogs' apparent preference for natural prey (McIlroy et al. 1986a).

Feral pigs in Australia damage crops, degrade pastures, kill and eat lambs, and are potential vectors and reservoirs of exotic pathogens (O'Brien et al. 1986; O'Brien 1988). Control of feral pigs with poisoned baits, including 1080 bait, is difficult because most pigs regurgitate these baits shortly after ingestion (O'Brien et al. 1986). The vomitus may cause secondary poisoning of nontarget species, and pigs surviving sublethal exposure to 1080 from vomiting may develop an aversion to 1080 and thus decrease their susceptibility to subsequent poisoning programs. The incorporation of antiemetics into 1080 baits should reduce or prevent vomiting, but those tested were not completely successful (O'Brien et al. 1986). Feral cats altered ecosystems and depleted populations of indigenous lizards and birds in Australia and in New Zealand and in numerous island habitats throughout the world. Fresh fish baits injected with 2 mg of 1080 per bait are used as a humane and lethal poison for feral cats (Eason and Frampton 1991).

The use of 1080 in New Zealand is restricted to licensed operators of pest-destruction boards and government departments (Temple and Edwards 1985). In Australia and in other locations, the addition of dye to identify toxic baits is standard practice (Temple and Edwards 1985; Statham 1989). The main purpose of such addition is to reduce the unintentional poisoning of birds; birds eat significantly less blue- or green-dyed feed than undyed feed (Statham 1989). Although birds prefer undyed baits to those dyed green, Canada geese (*Branta canadensis*) when feeding at night are unable to distinguish between dyed and undyed baits and consume both with equal frequency (Temple and Edwards 1985). Carrots used as wallaby baits in New Zealand are dyed with special green or blue pigments: however, the red-necked wallaby (*Macropus rufogriseus*) equally accepted dyed and undyed carrots (Statham 1989). Mice (*Mus* spp.) readily consumed dyed wheat (Twigg and Kay 1992). Compound 1080 is used in jam-type baits to control brush-tailed possums. These baits contained 1080 at concentrations as high as 1,500 mg 1080/kg FW bait and were dyed green to protect birds. Cinnamon was added to mask the flavor of the 1080 poison, and 800 mg potassium sorbate/kg was added as an antifungal preservative (Goodwin and Ten Houten 1991).

The Norway rat (*Rattus norvegicus*) had a severe effect on island populations of birds, reptiles, and invertebrates in New Zealand (Moors 1985). In one case, rats on the Big South Island exterminated five species of native forest birds within 3 years, including the last known population of the bush wren (*Xenicus longipes*). A paste containing petroleum jelly, soya oil, sugar, green dye, and 800 mg 1080/kg remained toxic for 6 to 9 months to rats that prey on grey-faced petrels (*Pterodroma macroptera*) and on other birds. Because 1080 produces a poison-shyness in any Norway rat that eats a sublethal dose, complete eradication of this species by 1080 is improbable (Moors 1985). The use of anticoagulants--such as warfarin (multiple doses needed), brodifacoum. (single dose) or coumatetralyl--seems more promising than 1080 for the control of rats (Moors 1985), although secondary poisoning of owls and hawks may occur (Hegdal and Colvin 1988).

In New Zealand, compound 1080 in a gel carrier is sometimes applied to the leaves of broadleaf (*Griselinia littoralis*) to poison red deer (*Cervus elephus*), feral goats, white-tailed deer (*Odocoileus virginianus*) and red-necked wallabies (Batcheler and Challies 1988). Use of 1080-gel baits reduced feral goat populations by 90% (Parkes 1983). Wallaby populations were reduced 87-91% with a 1080 gel applied to the foliage of palatable plants, and this compares favorably to reductions with aerially sown baits (Warburton 1990). The gel carrier was an effective phytotoxin, causing withering, death, or loss of chlorophyll from leaves within 10 days and sometimes within 24 h (Parkes 1983).

Feral pigs are sometimes poisoned by inserting as many as 10 gelatin capsules (each containing 100 mg of 1080) into carcasses or offal baits. Poisoned carcasses may remain edible for more than 2 months during fall and winter when poisoning campaigns are conducted; the 1080 is leached out when the carcass disintegrated (McIlroy 1983a). Other techniques to control feral pigs include injection of 1080 gel into beef-lung baits or insertion of capsules containing 1080 into apple, potato, or other fruit and vegetable baits. However, these techniques are potentially the most dangerous to applicators because 1080 powder, rather than a diluted solution, is used; also, the baits are lethal to nontarget scavengers (McIlroy 1983a).

Environmental Chemistry

General

Sodium monofluoroacetate is a whitish powder that is soluble in water to at least 263 mg/L but relatively insoluble in organic solvents. Some aqueous solutions of 1080 retain their rodenticidal properties for at least 12 months, but others lose as much as 54% of their toxicity after 24 days. Compound 1080 is unstable at more than 110° C and decomposes at more than 200° C, although 1080 in baits or poisoned carcasses is comparatively stable. Losses of 1080 from meat baits are due primarily to microbial defluorination and also to leaching from rainfall and consumption by maggots. Leachates from 1080 baits are probably not transported long distances by groundwater because they tend to be held in the upper soil layers. Compound 1080 can be measured in water at concentrations as low as 0.6 m/L and in biological samples at 10-15 m/kg. As discussed later, 1080 is readily absorbed through the gastrointestinal tract, mucous membranes, and pulmonary epithelia; once absorbed, it is uniformly distributed in the tissues. Metabolic conversion of high concentrations of fluoroacetate to fluorocitrate results in large accumulations of citrate in the tissues and eventual death from ventricular fibrillation or respiratory failure. Regardless of dose and tested species, no signs or symptoms of 1080 poisoning were evident during a latent period of 30 min to 2 h; however, death usually occurred within 24 h of exposure. Repeated sublethal doses of 1080 have increased the tolerance of some species of tested birds and mammals to lethal 1080 doses. Because of their low facility to convert fluoroacetate to fluorocitrate and their high defluorination capability, reptiles are more resistant to 1080 than mammals. No effective antidote is now available to treat advanced cases of fluoroacetate poisoning; accidental poisoning of livestock and dogs by 1080 is probably fatal. Partial protection against 1080 poisoning in mammals has been demonstrated with glycerol monoacetate, a sodium acetate-ethanol mixture, and with a calcium glutonate-sodium succinate mixture.

Chemical Properties

Compound 1080 is relatively soluble in water but not in organic solvents (Table 1). In water, trace amounts (0.6 m/L) of 1080 were detected during gas chromatography (GC) with electron capture detection; recoveries from environmental water spiked at 5-10 mg/L ranged from 93 to 97% (Ozawa and Tsukioka 1987). Recent advances make it possible to measure 1080 in solutions at concentrations as low as 0.2 mg/L (Kimball and Mishalanie 1993). In biological tissues, various methods have been used to determine fluoroacetic acid, including colorimetry, fluoride-ion electrodes, gas-liquid chromatography, and high-pressure chromatography; however, these methods involve lengthy extraction procedures, have low recoveries, or show lack of selectivity (Allender 1990). A sensitive gas chromatographic technique was developed and used successfully to determine fluoroacetate levels in organs from a magpie (*Gymnorhina tibicen*) that had ingested a bait containing 1080 poison. The procedure involved extraction of 1080 with acetone:water (8:1) followed by derivatization with pentafluorobenzyl bromide. Bait samples were initially screened by thin-layer chromatography, and identification of derivatized extracts was confirmed by gas chromatography/mass spectrometry = GC/MS (Allender 1990). A new method for fluoroacetate determination in biological samples requires the isolation of fluoroacetate as its potassium salt by ion-exchange chromatography and conversion to its dodecyl ester. The ester is quantified by capillary GC with a flame ionization detector for the range 1-10 mg/kg and by selected ion monitoring with GC/MS for the range 0.01-1.00 mg/kg (Burke et al. 1989). The detection limit for 1080 in tissues and baits is 15 mg/kg by a reaction-capillary GC procedure with photo-ionization detection; the detection limit is 100 mg/kg with flame-ionization procedures. The detection limit with these procedures is less sensitive than GC/MS; however, GC/MS is not normally available in veterinary diagnostic laboratories (Hoogenboom and Rammell 1987).

Table 1. Some properties of sodium monofluoroacetate.[a]

Variable	Datum
Alternate names	1080; Compound 1080; fratol; monosodium fluoroacetate; sodium fluoacetate; sodium fluoroacetate; ten-eighty
Chemical formula	$CH_2FCOONa$
Molecular weight	100.03
Physical state	White, odorless, almost tasteless, hygroscopic powdery salt, resembling powdered sugar or baking powder
Primary use	Rodenticide; mammal control agent
Purity	96.0-98.6%
Solubility	
Water	263 mg/L
Acetone, alcohol, animal and vegetable fats, kerosene, oils	Relatively insoluble
Stability	Unstable at > 110 and decomposes at >200° C. Hydrogen fluoride (20% by weight) is a decomposition product that readily reacts with metals or metal compounds to form stable inorganic fluoride compounds

[a]Chenoweth 1949; Negherbon 1959; Peacock 1964; Tucker and Crabtree 1970; Atzert 1971; Hudson et al. 1984.

Persistence

Significant water contamination is unlikely after aerial distribution of 1080 baits (Eason et al. 1993a). In one field trial in New Zealand in which more than 20 metric tons of 1080 baits were aerially sown over a 2,300-ha island to control brushtail possums (*Trichosurus vulpecula*) and rock wallabies (*Petrogale penicillata*), no 1080 was detected in surface or ground water of the island for at least 6 months after baits were dropped. A similar case was made for streams and rivers after 100 metric tons of 1080 baits were sown by airplane over 17,000 ha of forest (Eason et al. 1992, 1993b). Laboratory studies on 1080 persistence in solutions suggested that degradation to nontoxic metabolites is most rapid at elevated temperatures and in biologically conditioned media but is highly variable. In general, aqueous solutions of the salt or esters decrease in toxicity over time through spontaneous decarboxylation to sodium bicarbonate and to the highly volatile, relatively nontoxic methyl fluoride. Solutions refrigerated at 5° C lost about 54% of their initial toxicity to laboratory rats after 24 days and about 40% after 7 days at room temperature, but 1080 solutions remained toxic to yeast for at least 1 month after storage at 3-5° C (Chenoweth 1949). In an aquarium with plants and invertebrates and 0.1 mg 1080/L, the water concentration of 1080 declined 70% in 24 h and was not detectable after 100 h; residues in plants were not detectable after 330 h (Eason et al. 1993b). In a distilled water aquarium without biota, 1080 residues declined only 16% in 170 h (Eason et al. 1993b). In another study, 1080 solutions prepared in distilled water and stored at room temperature for 10 years showed no significant breakdown; moreover, solutions of 1080 prepared in stagnant algal-laden water did not lose biocidal properties during 12 months (McIlroy 1981a). More research on 1080 persistence in aquatic environments seems needed.

In soils, 1080 is degraded to nontoxic metabolites by soil bacteria and fungi, usually through cleavage of the carbon-fluoride bond (Eason et al. 1991, 1993a). Soil microorganisms capable of defluorinating 1080 include *Aspergillus fumigatus*, *Fusarium oxysporum*, at least 3 species of *Pseudomonas*, *Nocardia* spp., and 2 species of *Penicillium* (Wong et al. 1992a). When grown in solution with 1080 as the sole carbon source and in autoclaved soil, these microorganisms can defluorinate 1080; the amount of defluorination ranged from 2 to 89% in soils and from 2 to 85% in 1080 solutions. Some indigenous soil microflora were able to defluorinate 50-87% of the 1080 within 5-9 days in soil at 10% moisture and 15-28° C. The most effective defluorinaters in solution and in soils were certain strains of *Pseudomonas*, *Fusarium*, and *Penicillium* (Wong et al. 1991, 1992a; Walker 1994). *Pseudomonas cepacia*, for example, isolated from the seeds of various fluoroacetate-accumulating plants can grow and degrade fluoroacetate in fluoroacetate concentrations as high as 10,000

mg/kg (Meyer 1994). Biodefluorination of 1080 by soil bacteria was maximal under conditions of neutral to alkaline pH, at fluctuating temperatures between 11 and 24° C, and at soil moisture contents of 8-15%; biodefluorination of 1080 by soil fungi was maximal at pH 5 (Wong et al. 1992b).

Losses of 1080 from meat baits were probably due to consumption of the bait by blowfly maggots, leaching by rainfall, defluorination by microorganisms, and leakage from baits during decomposition (McIlroy et al. 1988), The 1080 in baits will persist under hot and dry conditions where leaching from rain is unlikely (Wong et al. 1992a). Baits remained toxic to dogs for more than 32 days during winter when maggots were absent and for 6-31 days during summer when maggots were present. Baits contained an average LD50 dose to tiger quolls (*Dasyurus maculatus*)—a raccoon-like marsupial--for 4-15 days in winter and for 2-4 days in summer (McIlroy et al. 1988). Meat baits that initially contained 4.6 mg of 1080 retained 62% after 3 days, 29% after 6 days, and 28% after 8 days (McIlroy et al. 1986a). The persistence of 1080 in fatty meat baits for control of wild dogs in Australia was measured during 226 days (Fleming and Parker 1991). Baits that initially contained 5.4 mg of 1080 retained 73% at day 7, 64% at day 20, 25% at day 48, and 15% at day 226. These baits retained LD50 kill values after 52 days to wild dogs, 93 days to cattle dogs, and 171 days to sheep dogs. In that study, loss of 1080 from the baits did not correlate with rainfall, temperature, or humidity. Losses were attributed to metabolism of 1080 bound to the fatty meat bait, leaching, consumption by maggots, and bacterial defluorination (Fleming and Parker 1991). When it is desirable for baits to remain toxic for long periods, the defluorination activity and microbial growth can be reduced significantly by incorporating bacteriostats and fungistats; conversely, inoculations with suitable defluorinating microbes rapidly detoxify 1080-poisoned baits (Wong et al. 1991).

Compound 1080 was highly persistent in diets formulated for the mink (*Mustela vison*). Mink diets analyzed 30 months after formulation lost 19-29% of the 1080 when the initial concentration was between 0.9 and 5.25 mg 1080/kg; loss was negligible at 0.5 mg 1080/kg ration (Hornshaw et al. 1986). A paste containing 0.08% 1080 and petroleum jelly, soya oil, sugar, and green dye retained its rodenticidal properties for 6 to 9 months. But a rolled oats-cat food 1080 bait, because of its moistness, became fly-infested in warm weather, tended to rot, and lost its rodenticidal properties in a few days (Moors 1985). Gel baits set to kill deer were sampled after 45 days of weathering; only 10% of the 1080-treated leaves retained toxic gel after 45 days (Batcheler and Challies 1988). About 1.4% of 1080/mm rainfall was lost from the leaves; about 90% was lost in 2 trials in which 81 and 207 mm of rainfall were recorded. Compound 1080 decreased from 604 mg/bait at the start to 76 mg/bait after 30 days and to 5 mg/bait after 45 days. Significant losses of compound 1080 also resulted from biodegradation in storage. *Penicillium* spp. from broadleaf samples degraded 1080 at pH 5.4 and 23° C and grew vigorously on 1080-poisoned gels; other species of microorganisms can also degrade 1080 (Batcheler and Challies 1988).

Leachates from 1080-poisoned baits are probably not transported long distances by the leaching water because they are held in the upper soil layers (Atzert 1971). This statement is predicated on the facts that (1) salts of monofluoroacetic acid rapidly adsorb to plant tissues and other cellulosic materials; (2) some plants can decompose 29% of the adsorbed 1080 in 48 h; and (3) 1080 in soils is decomposed by soil microorganisms (Atzert 1971). The percent of 1080 defluorinated from various bait materials after 30 days as a result of microbial action ranged from 0.0 to 7.2% in cereals, eggs, horse meat, and beef and was 14% in kangaroo meat and 71% in oats (Wong et al. 1991). The defluorinating ability of fungi and bacteria was low when 1080 was the sole carbon source and high when alternative carbon sources such as peptone-meat extracts were present. The extent of defluorination varied among the different types of organisms in the baits. Microorganisms isolated from oats and kangaroo meat had the highest defluorinating activity and those from cereals and eggs, the lowest (Wong et al. 1991).

Metabolism

Sodium monofluoroacetate is absorbed through the gastrointestinal tract, open wounds, mucous membranes, and the pulmonary epithelium; it is not readily absorbed through intact skin (Negherbon 1959; Atzert 1971). Once absorbed, it seems to be uniformly distributed in the tissues including the brain, heart, liver, and kidney (Peacock 1964). All tested routes of 1080 administration are equally toxic; there is no noteworthy difference in the acute toxicity of 1080 when administered orally, subcutaneously, intramuscularly, intraperitoneally, or intravenously (Chenoweth 1949; Peacock 1964; Atzert 1971). Moreover, the oral toxicity of

1080 is independent of the carrier, including water, meat, grain, oil, gum acacia suspension, or gelatin capsule carriers (Atzert 1971).

All students of the action of fluoroacetate have been impressed with the unusually long and variable latent period between administration and response. This latent period occurred in all studied species regardless of route of administration (Chenoweth 1949; Negherbon 1959; Peacock 1964; Tucker and Crabtree 1970; Atzert 1971; Hudson et al. 1984). With few exceptions, the latent period ranges from 30 min to 2 h and massive doses—such as 50 times an LD95 dose—do not elicit immediate responses. The time between 1080 treatment and death was relatively constant in all tested species and usually ranged from 1 h to 1 day. The latent period associated with 1080 may result from 3 major factors: (1) the time required for hydrolysis of monofluoroacetate to monofluoroacetic acid and its subsequent translocation and cell penetration; (2) the time required for biochemical synthesis of a lethal quantity of fluorocitrate; and (3) the time required for the fluorocitrate to disrupt intracellular functions on a large enough scale to induce gross signs of poisoning (Chenoweth 1949; Atzert 1971).

Many authorities agree that the toxicity of 1080 to mammals is due to its conversion to fluorocitrate, a fluorotricarboxylic acid (Gal et al. 1961; Atzert 1971; Roy et al. 1980; McIlroy 1981b; Kun 1982; Mead et al. 1985a, 1985b; Hornshaw et al. 1986; Twigg et al. 1986, 1988a, 1988b; Murphy 1986). These authorities concur that enzymatic conversion of fluoroacetate via fluoroacetyl coenzyme A plus oxalacetate in mitochondria is the metabolic pathway that converts the nontoxic fluoroacetate to fluorocitrate. Fluorocitrate blocks the Krebs cycle, also known as the tricarboxylic acid cycle, which is the major mechanism for realizing energy from food. Fluorocitrate inhibits the enzyme aconitase and thereby inhibits the conversion of citrate to isocitrate. Fluorocitrate also inhibits succinate dehydrogenase, which plays a key role in succinate metabolism. The inhibition of these two enzymes results in large accumulations of citrate in the tissues, blocking glucose metabolism through phosphofructokinase inhibition, and eventually destroys cellular permeability, cell function, and finally the cell itself. The classical explanation of fluorocitrate toxicity through aconitase inhibition has been questioned (Kun 1982; Savarie 1984). A more recent explanation is that fluorocitrate binds with mitochondrial protein, thereby preventing citrate transport and its utilization by cells for energy production, although the underlying biochemical mechanisms are not completely understood (Kun 1982). Based on calculated metabolic rates of fluorocarboxylic acids, secondary poisoning of animals that have consumed 1080-poisoned prey is probably due to unmetabolized fluoroacetate rather than to fluorocitric acid (Kun 1982).

Dogs, rats, and rabbits metabolize fluoroacetate compounds to nontoxic metabolites and excrete fluoroacetate and fluorocitrate compounds; the rate of excretion peaks during the first day after dosing and drops shortly thereafter. Rats dosed with radiolabeled 1080 at 5 mg/kg BW had 7 different radioactive compounds in their urine. Monofluoroacetate comprised only 13% of the urinary radioactive material, fluorocitrate only 11%, and an unidentified toxic metabolite, 3%; 2 nontoxic metabolites accounted for almost 73% of the urinary radioactivity (Atzert 1971). Animal muscle usually contained nondetectable residues of any 1080 component within 1 to 5 days of treatment (Marsh et al. 1987; Eason et al. 1993c). Defluorination occurred in the liver by way of an enzymic glutathione-dependent mechanism, which in the brush-tailed opossum resulted in the formation of S-carboxymethylcysteine and free fluoride ion (Twigg et al. 1986). A rapid rate of defluorination together with a low reliance on aerobic respiration favored detoxification of fluoroacetate rather than its conversion into fluorocitrate and may account for the greater resistance of 1080 by reptiles than by mammals (Twigg et al. 1986).

Sublethal doses of 1080 led to a tolerance to subsequent challenging doses in certain animals; in other species, however, lethal concentrations accumulated after repeated sublethal doses (Atzert 1971). Repeated sublethal doses of 1080 increased the tolerance of some eagles, rats, mice, and monkeys but not of dogs. Conversely, repeated sublethal doses of 1080 accumulated to lethal levels in dogs, guinea pigs, rabbits, and mallards. Continued sublethal doses of 1080 to rats caused regressive changes in the germinal epithelium of the seminiferous tubules (Atzert 1971). Altered behavior in mice after high sublethal doses of 1080 were probably caused by neuronal damage from concurrent energy deficiency that was further accentuated by the CNS stimulant action of fluoroacetate/fluorocitrate and the brain anoxia that occurred during 1080-induced intermittent convulsions; a similar pattern was observed in two human patients (Omara and Sisodia 1990). Anuria in some 1080-dosed mice were probably caused by renal shutdown from hypocalcemic tension (Omara and Sisodia 1990). Tolerance to 1080 is a time-related phenomenon (Atzert 1971). Laboratory rats that were pretreated with 0.5 mg 1080/kg BW 4-24 h prior to a dose of 5.0 mg/kg BW were more resistant than rats that

were not similarly pretreated (Atzert 1971). Accumulation of 1080 is also a time-related phenomenon (Chenoweth 1949; Atzert 1971). Domestic dogs given 25 mg 1080/kg BW daily were unaffected until the fifth dose, when they went into convulsions and died. Also, larger sublethal doses could be administered to dogs on alternate days without adverse effects (Atzert 1971).

Fishes, amphibians, and reptiles are usually less sensitive to 1080 than warm-blooded animals (Atzert 1971). Reptiles, for example, are more resistant to 1080 than mammals (Twigg et al. 1986). The relatively small elevation of plasma citrate levels in skinks (*Tiliqua rugosa*) given 100 mg 1080/kg BW reflects the exceptional tolerance of this lizard species. The minimal effect of fluoroacetate on aerobic respiration in *T*. *rugosa* could be explained by a low conversion of fluoroacetate into fluorocitrate or by a low susceptibility of aconitase to the fluorocitrate formed. Although defluorination in skinks helped to minimize the immediate effects of fluoroacetate in aerobic respiration, it resulted in rapid depletion of liver glutathione levels (Twigg et al. 1986).

The breakdown in intracellular processes from fluorocitrate or from decreased energy production may cause death from gradual cardiac failure or ventricular fibrillation, death from progressive depression of the CNS with either cardiac or respiratory failure, or death from respiratory arrest after severe convulsions; signs of 1080 intoxication included labored breathing, vomiting, lethargy, muscular incoordination, weakness, and tremors (Chenoweth 1949; Negherbon 1959; Tucker and Crabtree 1970; Atzert 1971; Hudson et al. 1984; Murphy 1986; Eason and Frampton 1991). Among herbivores, 1080-induced deaths were due primarily to cardiac disorders; among carnivores, deaths were from CNS disorders; and among omnivores, deaths were from both cardiac and CNS disorders (Atzert 1971). Other signs of 1080 intoxication included kidney and testicular damage (Savarie 1984) and altered blood chemistry, specifically, elevated concentrations of citrate (Twigg et al. 1986), glucose, lactic acid, pyruvic acid, acetate, inorganic phosphate, potassium, and fluorine (Negherbon 1959). Some mammals also displayed parasympathetic nervous system effects including increased salivation, urination, and defecation and eventual cardiac failure (Hudson et al. 1984).

Vomiting probably evolved among carrion eaters as a natural protective mechanism, but it does not necessarily ensure survival from 1080 poisoning (McIlroy 1981b). For example, even though 90% of eastern native quolls (*Dasyurus viverrinus*) and 95% of tasmanian devils (*Sarcophilus harrisii*) vomited within 26-55 min after ingesting 1080, this time was still sufficient for many to absorb a lethal dose. Loud sounds, sudden movements of an observer, or convulsions by another animal nearby sometimes stimulated convulsions; however, intraspecific and interspecific variability was great. Signs preceding convulsions usually included restlessness; hyperexcitability or increased response to stimuli; trembling; rapid, shallow breathing; incontinence or diarrhea; excessive salivation; twitching of facial muscles; abnormal eye movements; incoordination; vocalization; and sudden bursts of violent activity. All affected animals subsequently fall to the ground in a tetanic seizure; their hind limbs or all four limbs and sometimes the tail extend rigidly from their arched bodies. This tonic phase is followed by a clonic phase in which the animals kick with the front legs and eventually begin to relax. After this phase, animals either recover gradually, die shortly afterwards, experience additional seizures and then die or recover, or remain comatose until death as many as 6 days later (McIlroy 1981b).

Antidotes

No highly effective treatment of well established fluoroacetate poisoning is available (Chenoweth 1949; Peacock 1964; Atzert 1971), and accidental poisoning of livestock and domestic dogs is probably fatal (Mead et al. 1991). The following compounds were tested and had no effect on ameliorating 1080 intoxication: salts of fatty acids, anticonvulsants, vitamins, metabolic intermediates (Chenoweth 1949), and nonphysiological sulfhydryl compounds such as N-acetylcysteine and cysteamine (Mead et al. 1985a). As discussed later, sodium-acetate/ethanol mixtures, barbituates, glycerol monoacetate, calcium glutonate/sodium succinate mixtures, and 4-methylpyrazole offer partial protection to 1080-poisoned mammals, possibly because they compete with fluoroacetate in the Krebs cycle.

Sodium acetate and ethanol partially protect mice against 1080. Ethanol and sodium acetate administered together are twice as effective as either alone, suggesting a synergistic effect (Chenoweth 1949). Mixtures of acetate and ethanol reduced mortality of 1080-poisoned mice (given 2 times an LD50 dose) from 80% to 30% (Tourtellotte and Coon 1950). Mortality was reduced by 90% in mice given 170 mg 1080/kg BW (about 10 times an LD50 dose) and an intraperitoneal injection of sodium acetate (2-3 g/kg BW) dissolved in ethanol (1.6 g/kg BW). But the beneficial effect of the acetate-ethanol treatment to mice decreased rapidly with increasing time after the administration of 1080. Ethanol-acetate mixtures had some antidotal effect on 1080-poisoned dogs

provided that treatment was administered within 30 min of poisoning (Tourtellette and Coon 1950). A mixture of 2 g sodium acetate/kg BW and 2 g ethanol/kg BW is recommended for treatment of 1080-poisoned monkeys (Peacock 1964).

Barbituates were marginally effective in protecting domestic dogs but not laboratory mice against fluoroacetate poisoning (Chenoweth 1949; Peacock 1964). Barbituates administered to dogs within 30 min of 1080 poisoning (4 times an LD50 dose) resulted in 80% survival; when therapy was given 3 h after poisoning, survival was 17% (Tourtellette and Coon 1950). At higher 1080 doses (i.e., 6 times the LD50 value), barbiturates were ineffective. Repeated intravenous injections of 20 mg pentobarbital/kg BW to a 1080-poisoned dog (0.3 mg 1080/kg BW) prevented death when administered within 8.5 h of poisoning (Tourtellette and Coon 1950).

Glycerol monoacetate at 2 to 4 g/kg BW partially protects 1080-poisoned rats, rabbits, dogs, and rhesus monkeys (Chenoweth 1949; Peacock 1964; Murphy 1986). But its effectiveness is apparent only when administered intramuscularly in large amounts immediately after 1080-ingestion (Mead et al. 1991). A single dose of magnesium sulphate at 800 mg/kg BW given intramuscularly as a 50% solution shortly after 1080 exposure prevented death of rats dosed with marginally lethal amounts of 1080 (Peacock 1964).

A reduced level of blood calcium is one explanation for the toxic effects of fluoroacetate and may account for the gap between chemical manifestations and the biochemistry of 1080 poisoning (Roy et al. 1980). Cats poisoned with 1080 showed a 27% drop in blood calcium levels within 40 min; intravenous administration of calcium chloride prolonged the life of treated cats from 94 min to 167 min (Roy et al. 1980). In a search for effective antidotes to fluoroacetate poisoning, calcium gluconate was chosen to antagonize hypocalcemia, and sodium alpha ketoglutarate and sodium succinate were selected to revive the TCA cycle (Omara and Sisodia 1990). Effectiveness of each of these antidotes individually and in certain combinations was tested in laboratory mice exposed to lethal doses (15 mg/kg BW, intraperitoneal injection) of 1080. Antidotal treatments were administered from 15 min to 36 h after dosing. All three of the antidotes alone, and a combination of calcium glutonate with sodium alpha ketoglutarate were ineffective in reducing mortality in treated mice. However, a combination of calcium glutonate (130 mg/kg BW) and sodium succinate (240 mg/kg BW) was effective if the two solutions were either injected at separate sites or mixed in the same syringe just prior to injection. Increasing the dose of sodium succinate to 360 or 480 mg/kg BW with calcium glutonate (130 mg/kg BW) was unrewarding. Additional studies are needed to confirm the efficacy and mechanisms of action of this combination (Omara and Sisodia 1990).

Intraperitoneal injection of 4-methylpyrazole to rats at 90 mg/kg BW, given 2 h prior to 1080 administration, offered partial protection against accumulations of citrate or fluorocitrate in the kidney (Feldwick et al. 1994). The antidotal effects of 4-methylpyrazole are attributed to its inhibition of NAD^+-dependent alcohol dehydrogenase that converts 1,3-difluoro-2-propanol to difluoroacetone, an intermediate in the pathway of erythrofluorocitrate metabolism (Feldwick et al. 1994). A disadvantage of 4-methylpyrazole is that it needs to be administered before significant exposure to fluoroacetate.

First-aid treatment of humans accidentally poisoned with 1080 includes immediate emesis and gastric lavage followed by an oral dose of magnesium sulfate or sodium sulfate to remove the poison from the alimentary tract before absorption of lethal quantities can occur (Peacock 1964; Atzert 1971). When the stomach is emptied, oral administration of ethanol may be beneficial (Temple and Edwards 1985). The patient should be put at complete rest and given barbituates with moderate duration of action, such as sodium amytol, to control convulsions (Anonymous 1964; Atzert 1971). Intramuscular injections of undiluted glycerol monoacetate at 0.5 mg/kg BW are recommended every 30 min for several hours and then at a reduced level for at least 12 h (Atzert 1971; Temple and Edwards 1985). If intramuscular administration is not feasible, a mixture of 100 mL of undiluted glycerol monoacetate in 500 mL of water can be given orally and repeated in an hour (Atzert 1971). If glycerol monoacetate is not available, acetamide or a combination of sodium acetate and ethanol may be given in the same dose (Atzert 1971). If ventricular fibrillation occurs, the heroic treatment of 5 mL of a 1% procaine hydrochloride by intracardiac puncture is justified (Anonymous 1964). Intravenous administration of procainamide is also effective in the restoration of normal rhythm in ventricular fibrillations (Atzert 1971). Symptoms of 1080 poisoning usually subside in 12 to 24 h, but the patient should be kept in bed for at least 3 days (Anonymous 1946).

Lethal and Sublethal Effects

General

Mammals were the least resistant tested group against 1080; individuals of sensitive species died after receiving a single dose of 0.05-0.2 mg/kg BW. As discussed later, adverse sublethal effects included testicular damage in rats (*Rattus* spp.) after drinking water containing 2.2-20.0 mg 1080/L for 7 days (0.07-0.71 mg/kg BW daily), impaired reproduction in the mink on diets containing 0.8 mg 1080/kg ration for 60 days, and altered blood chemistry in European ferrets on diets containing 1.1 mg 1080/kg feed for 28 days. Elevated fluoroacetate residues of 34 mg/kg DW muscle and 423 mg/kg DW liver were measured in some 1080-poisoned mammals, notably in European rabbits. Sensitive species of birds died after a single 1080 dose of 0.6-2.5 mg/kg BW, daily doses of 0.5 mg/kg BW for 30 days, 47 mg/kg diet for 5 days, or 18 mg/L drinking water for 5 days.

Accumulation and adverse sublethal effects in birds occurred at dietary loadings of 10-13 mg 1080/kg ration. The risk to human consumers of cooked meat from 1080-poisoned waterfowl seems negligible. Amphibians and reptiles were more resistant to 1080 than mammals and birds because of their greater ability to detoxify fluoroacetate by defluorination, a reduced ability to convert fluoroacetate to fluorocitrate, and an aconitase hydratase enzyme that is comparatively insensitive to fluorocitrate inhibition. LD50 values in amphibians were greater than 44 mg 1080/kg BW; in reptiles, this value was greater than 54 mg 1080/kg BW. Other studies with 1080 and sensitive species showed death of mosquito larvae at water concentrations of 0.025-0.05 mg/L, death of terrestrial beetle and lepidopteran larvae at 1.1-3.9 mg/kg BW, no phytotoxicity to terrestrial flora at water concentrations of 10 mg/L, and--based on limited data--no adverse effects on freshwater fishes at 370 mg/L.

Terrestrial Plants and Invertebrates

Fluoroacetate was first isolated in South Africa in 1944 from the gifblaar plant (*Dichapetalum cymosum*; Negherbon 1959). Seeds of the South African *D*. *braunii* may contain as much as 8,000 mg fluoroacetate/kg DW (Meyer 1994). Several other species of *Dichapetalum* and *Palicourea marcgravii*, a South American poisonous species, produce fluoroacetate (Twigg et al. 1986; Twigg and King 1991). In Australia, fluoroacetate occurs naturally in the leaves, flowers, and seeds of more than 35 species of leguminous plants of the genera *Gastrolobium* and *Acacia* (Mead et al. 1985; Twigg et al. 1986, 1988, 1990; Twigg and King 1991; McIlroy 1992). All but two of these species are confined to the southwestern corner of Western Australia; the other two species occur in northern and central Australia. Fluoroacetate concentrations varied regionally, seasonally, among species, and among parts of the plants. The fluoroacetate content of these plants is usually greatest in flowers, seeds, and young leaves, and this is consistent with chemically mediated defense strategies in which plants use poisonous compounds to protect their most essential parts (Twigg and King 1991). In Australia, the highest measured fluoroacetate concentrations were in air-dried leaves and seeds of two species from Western Australia: concentrations reached 2,650 mg/kg in leaves and 6,500 mg/kg in seeds of *Gastrolobium* spp. Air-dried samples of the two species from northern and central Australia, *Acacia georginae* and *Gastrolobium grandiflorum*, contained as much as 25 mg fluoroacetate/kg leaf and 185 mg/kg seed (Twigg and King 1991).

Economic losses of domestic livestock were significant in Africa and Australia after ingestion of fluoroacetate-bearing vegetation (Twigg and King 1991). Herbivores that have had evolutionary exposure to this vegetation are much less susceptible to fluoroacetate intoxication than geographically separate, unchallenged species (Mead et al. 1985; Twigg et al. 1986). The development of tolerance to fluoroacetate by insects, reptiles, birds, and mammals has evolved on at least three continents where indigenous plants produce fluoroacetate that protects them against herbivory (Twigg and King 1991). In Australia, for example, animal populations that have coexisted with fluoroacetate-bearing vegetation for at least several thousands of years have developed varying degrees of tolerance to this potent toxin. Tolerance depends on their diets and habitats, sizes of their home ranges, mobility, and length of evolutionary exposure to fluoroacetate-bearing vegetation. Once developed, this tolerance is retained by animal populations even after isolation from the toxic vegetation for 70 to 100 centuries. Biochemical mechanisms responsible for the large toxicity differential between conspecifics with and without exposure to fluoroacetate-bearing vegetation are poorly understood (Twigg and King 1991).

Fluoroacetate and fluorocitrate have also been isolated from forage crops grown in an environment that is rich in atmospheric or inorganic fluoride (Lovelace et al. 1968; Ward and Huskisson 1969; Atzert 1971; Savarie 1984; Twigg and King 1991). For example, soybeans (*Glycine max*) can synthesize fluoroacetic acid when

grown in an atmosphere with elevated levels of hydrogen fluoride or in media containing high levels of sodium fluoride. Forage crops, including alfalfa (*Medicago sativa*) and crested wheat grass (*Agropyron cristatum*) grew near a phosphate plant that discharged inorganic fluoride with as much as 179 mg fluoroacetate/kg DW, 896 mg fluorocitrate/kg DW, and 1,000 mg total fluoride/kg. The plants were not adversely affected, but horses (*Equus caballus*) that grazed on these crops showed signs of fluoride poisoning, suggesting that the toxic effect of inorganic fluoride adsorbed or absorbed by plants and not incorporated into monofluoroacetic acid was greater than the toxic effect of monofluoroacetic acid synthesized by the plants (Lovelace et al. 1968; Atzert 1971). Lettuce (*Lactuca sativa*) that absorbed radiolabeled 1080 through its roots or leaves had elevated citrate concentrations and retained radioactivity (Ward and Huskisson 1969). Plants can degrade 1080 by cleaving the carbon-fluorine bond, as judged by studies with germinating seeds of the peanut *Arachis hypogea* (Atzert 1971).

Compound 1080 in gels, pastes, or grease carriers and smeared on leaves of palatable plants has been used to control ungulate and marsupial pests in New Zealand, including feral goats (*Capra* sp.), red deer (*Cervus elephus*), and white-tailed deer (*Odocoileus virginianus*; Parkes 1991). The effectiveness of 1080 in carbopol gel or in petrolatum grease on leaves of the mahoe (*Melicytus ramiflorus*) was significantly modified by the phytotoxicity of these carriers. Both carriers caused baited leaves to absciss, and the rate of abscission increased when 1080 was included. Petrolatum was one-third as phytotoxic as carbopol and retained 1080 for longer periods--at least 1 year. Carbopol lost about 95% of its 1080 after 64 days of exposure and 100 mm of rain but only 22% in petrolatum under similar conditions. Carbopol with 1080 is recommended for use where its distribution is sufficient to place goats and other target species at immediate risk; petrolatum can be used where a long-lasting bait is needed (Parkes 1991).

Compound 1080 has systemic insecticidal properties against insects feeding on treated plants. Cabbage (*Brassica oleracea capitata*) that had accumulated 1080 through its roots from solution or from soil cultures after application on leaves was toxic by contact to eggs and larvae of the large white butterfly (*Pieris brassicae*) and to various species of aphids (Negherbon 1959). Compound 1080 was not phytotoxic at 10 mg/L or at several times the concentration necessary for insecticidal action, but its use as an insecticide is not recommended because of its high toxicity in mammals (Negherbon 1959; Spurr 1991).

At least nine groups of terrestrial invertebrates are adversely affected by 1080-poisoned baits, by contaminated habitats with residues that leach from 1080 baits, or by the consumption of animal byproducts and carcasses contaminated with 1080 (Chenoweth 1949; Notman 1989). Lethal effects are reported in houseflies, moths, aphids, ants, bees, and mites that ate 1080-poisoned baits and in fleas that ingested 1080-poisoned rats (Notman 1989). Cockroaches, collembolids, and slugs that ate poisoned baits experienced adverse effects. Egg production in wasps was disrupted after a single sublethal dose of 1080, and mortality of larvae from butterfly eggs treated with 1080 was 98% (Notman 1989). Harvester ants (*Pogonomyrtnex* sp.) and darkling ground beetles (Tentyridae) removed and consumed 1080 bait, leaving bait and dead ants concentrated on the ground near the nest (Hegdal et al. 1986). In a control program, German wasps (*Vespula germanica*) and common wasps (*Vespula vulgaris*) fed 1080-poisoned canned sardines in aspic jelly were not affected at concentrations of less than 100 mg 1080/kg bait (Spurr 1991). At 1,000 mg/kg, however, wasp traffic at nest entrances was reduced by 17%; at 5,000-10,000 mg/kg, traffic was reduced by 78-89%, and almost all wasps died within 100 m of bait stations after 6 h (Spurr 1991). Honeybees (*Apis mellifera*) feed readily on 1080-jam baits that are used to control opossums (*Trichosurus vulpecula*) in New Zealand (Goodwin and Ten Houten 1991). Bee kills have been documented in the vicinity of jam baits, and dead bees contained 3.1 to 10.0 mg 1080/kg whole bee. The oral LD50 in the honey bee is 0.8 mg/bee. Because no deaths occur within 2 h after feeding, poisoned bees may make several foraging trips before dying. Molasses or oxalic acid is now added to 1080-jam baits to repel bees (Goodwin and Ten Houten 1991). Poisoned insects may cause secondary poisoning of insectivores. Accordingly, 1080 should not be used in the vicinities of susceptible nontarget invertebrates or endangered insectivores (Notman 1989).

Variability in sensitivity to 1080 after abdominal injection in tested insect larvae was great (Twigg 1990). The LD50 value in mg 1080/kg BW--administered by fluoroacetate-bearing vegetation--was 1.05 in *Perga dorsalis* (Hymenoptera); in Lepidoptera, these values were 3.9 in *Mnesampla privata*, 42.7 in *Spilosoma* sp., and about 130.0 in *Ochrogaster lunifer*. In all tested species, death occurred within 2 to 48 h after injection, and total body citrate concentrations were significantly higher in the poisoned than in the unpoisoned conspecifics. Tolerance to 1080 was enhanced in larvae of Western Australian insects that consumed fluoroacetate-bearing vegetation (Twigg 1990).

Populations of terrestrial invertebrates including populations of amphipods, ants, beetles, collembolids, millipedes, mites, weevils, slugs, spiders, and snails were not adversely affected by 1080 poisoning in operations to control brushtail possums in New Zealand (Spurr 1994). Residues of 1080 in nontarget terrestrial invertebrates were low or negligible after aerial applications (Eason et al. 1993b). Residues of 1080 were measured in various species of terrestrial invertebrates in New Zealand before and after aerial application of possum baits with 800 mg 1080/kg and sown at 5 kg/ha. No residues of 1080 were found in spiders, beetles, millipedes, centipedes, or earthworms at any stage. Residues of 1080 were detectable in some orthopteran insects (2 mg/kg FW) and in some cockroaches (4 mg/kg FW). Laboratory studies indicated that 90% of all 1080 was eliminated from insects within 4-6 days after dosing, suggesting a low risk to insectivorous birds (Eason et al. 1993b).

Aquatic Organisms

Despite an intensive literature search, very little data were found on the toxicity of 1080 to aquatic life. King and Penfound (1946) reported that fingerling bream and bass (species unidentified) tolerated 370 mg of 1080/L without apparent discomfort for an indefinite period. Deonier et al. (1946) aver that fourth-instar larvae of the mosquito *Anopheles quadrimaculatas* were comparatively sensitive to 1080 and that 1080 was among the most toxic 3% of 6,000 organic compounds screened against this life stage. In 48 h, concentrations of 0.025 mg 1080/L were fatal to 15% of these larvae, 0.05 mg 1080/L to 40%, and 0.1 mg 1080/L to 65%. The common duckweed (*Spirodela oligorrhiza*) seems to be unusually sensitive to 1080. Growth of duckweed was inhibited at 0.5 mg/L (Walker 1994), but this needs verification.

Recent unpublished data (as quoted in Fagerstone et al. 1994) on the acute toxicity of 1080 to rainbow trout (*Oncorhynchus mykiss*), bluegills (*Lepomis macrochirus*), and daphnids (*Daphnia magna*) suggested that these organisms are comparatively tolerant to 1080. For example, bluegills exposed to 970 mg 1080/L for 96 h showed no observable adverse effects; in rainbow trout, the no-observable-effect concentration during a 96-h exposure was 13 mg 1080/L, and the LC50 (96 h) value was 54 mg/L with a 95% confidence interval of 39-74 mg/L; in *Daphnia*, no adverse effects were noted at 130 mg 1080/L during an exposure for 48 h, although 50% were immobilized at 350 mg/L in 48 h (Fagerstone et al. 1994). No data were available on effects of 1080 to aquatic biota during a life cycle or during long-term exposures. The effects of chronic exposure to 1080 on nontarget species of aquatic arthropods and macrophytes require study.

Amphibians and Reptiles

In general, the onset of action and time to death or to recovery was slowest in amphibians and reptiles, which were among the most resistant to 1080 of all tested vertebrate animals (McIlroy et al. 1985; McIlroy 1986). LD50 values in representative species of amphibians ranged from 54 to 2,000 mg 1080/kg BW and in reptiles from 44 to 800 mg/kg BW (Table 2). Frogs and lizards given a lethal oral dose of 1080 did not show signs of poisoning for 22 to 56 h and survived for 78 to 131 h (McIlroy et al. 1985). Frogs seem to be more sensitive to 1080 in summer than in winter (Chenowith 1949). Unlike mammals, amphibians and reptiles possess an innate tolerance to 1080 because of their greater ability to detoxify fluoroacetate by defluorination, a reduced ability to convert fluoroacetate to fluorocitrate, and an aconitase hydratase enzyme system that is less sensitive to inhibition by fluorocitrate (Twigg and Mead 1990).

One of the most tolerant tested reptiles was the shingle-back lizard (*Tiliqua rugosa*; McIlroy 1986), but populations of *T. rugosa* from Western Australia that coexist with fluoroacetate-bearing vegetation were much less sensitive to 1080 intoxication than conspecifics from South Australia not exposed to the toxic plants (Table 2; McIlroy et al. 1985; Twigg et al. 1988a; Twigg and Mead 1990). The shingle-back lizard is an omnivore that feeds on flowers, leaves, and seeds and probably evolved an increased tolerance to fluoroacetate through feeding on toxic plants such as *Gastrolobium* and *Oxylobium* that are abundant in southern Western Australia (McIlroy et al. 1985).

Reptiles are probably not affected by either primary or secondary poisoning during 1080-poisoning campaigns (McIlroy 1992). In Australia, 1080-poisoned baits contained 330 mg 1080/kg in carrot baits for rabbits and in oat baits for pigs, 400 mg 1080/kg in oat baits for rabbits, 500 mg 1080/kg in pellet baits for rabbits and pigs, 14 mg 1080/kg in meat baits for dingoes, and 144 mg/kg in meat baits for pigs (McIlroy et al. 1985). These data indicate that most species of tested reptiles must ingest unrealistic quantities of bait to be adversely affected by 1080. Most lizards, for example, must eat 43-172% of their body weight of poisoned rabbit

baits and 143 to 393% of their body weight of meat baits intended for pigs. However, Gould's monitor (*Varanus gouldi*) may ingest lethal amounts of meat baits intended for pigs after eating 31% of its body weight of poisoned baits. By comparison, a large pig (130 kg) must eat about 2 kg of meat baits (1.6% of its body weight) for an LD99 dose (McIlroy et al. 1985).

Birds

Laboratory studies with birds (Table 3) revealed several trends: (1) death occurred in orally dosed sensitive species after a single dose of 0.6-2.5 mg 1080/kg BW, after daily doses of 0.5 mg 1080/kg BW for 30 days, after 47 mg/kg diet for 5 days, or after 18 mg/L drinking water for 5 days; (2) single doses of more than 10 mg/kg BW were usually fatal; (3) 1080 toxicity was enhanced at lower temperatures; (4) younger birds were more sensitive than older birds; (5) birds tended to avoid diets and drinking water with high sublethal concentrations of 1080; (6) accumulations and adverse effects were noted at dietary concentrations of 10-13 mg 1080/kg feed; and (7) birds with prior or continuing exposures to naturally occurring fluoroacetates were more resistant to 1080 than conspecifics without such exposures. Drinking-water-LC50 values were about 10 times higher (i.e., 10 times less toxic) than dietary LC50s in mallards (*Anas platyrhynchos*) and in northern bobwhites (*Colinus virginianus*); however, both species of birds consumed 5 to 10 times more water than food on a daily mg/kg BW basis (Kononen et al. 1991). The minimum repeated daily oral dosage that was lethal to mallards in 30-day tests was 0.5 mg/kg BW, suggesting a high degree of cumulative action by this species (Tucker and Crabtree 1970). But European starlings (*Sturnus vulgaris*) tolerated 13.5 mg 1080/kg diet for extended periods without significant adverse effects (Balcomb et al. 1983). Studies with the galah (*Cacatua roseicapilla*) showed that 1080 lethality was not affected by the age or sex of the bird or by the route of administration (McIlroy 1981a). But breeding adult female Pacific black ducks (*Anas superciliosa*) were more sensitive to 1080 than either males or nonbreeding females (McIlroy 1984).

The most common external signs of 1080 poisoning in birds included depression, fluffed feathers, a reluctance to move, and convulsions (McIlroy 1984). Signs of 1080 poisoning first appeared 1 to 60 h after dosing, and deaths occurred 1 h to almost 11 days after dosing (McIlroy 1984). Death of 1080-poisoned California quail (*Callipepla californica*) usually occurred within 3 h, although birds were inactive within 2 h of dosing and comatose until death (Sayama and Brunetti 1952). The most common internal sign of 1080 poisoning was a dose-related increase in plasma citrate concentration, and this was a useful indicator of fluoroacetate sensitivity among birds of similar metabolic rates and phylogenetic affinities (Twigg and King 1989).

Table 2. Effects of 1080 on representative amphibians and reptiles.

Table 2. Group, species, dose, And other variables	Effects	Reference[a]
Amphibians		
Spotted grass frog, *Limnodynastes tasmaniensis*; 60 mg/kg body weight (BW); single dose	LD50, adults	1
Bullfrog, *Rana catesbeiana*; 54.4 (95% confidence interval [=CI] of 25.6-115.0) mg/kg BW; single dose	LD50	2, 6
Frogs, various; 1,000-2,000 mg/kg BW; single dose	LD50	7, 8
Leopard frog, *Rana pipiens*; 150 mg/kg BW; single dose	LD50	2, 9
South African clawed frog, *Xenopus laevis*; >500 mg/kg BW; single dose	LD50	2, 9

Table 2.

Group, species, dose, And other variables	Effects	Reference[a]
Reptiles		
Australian reptiles 163 (44-336) mg/kg BW	LD50 mean and range for five species with no previous exposure to naturally occurring fluoroacetates	10
250 and 800 mg/kg BW	LD50 for two species with prior or continuing exposure to naturally occuring fluoroacetates	10
Gopher snake, *Pituophis catenifer*; fed dead or moribund rodents poisoned with high concentrations of 1080	In 21 separate trials, 14 snakes regurgitated rodents and 7 had no significant effects within 5 days of ingestion	11
Bearded dragon, *Pogona barbatus*; <110 mg/kg BW; single dose	LD50	1
Blotched blue-tongued lizard, *Tiliqua nigrolutea*; 336 (95% CI of 232-487) mg/kg BW; single dose	LD50	1, 5
Shingle-back lizard, *Tiliqua rugosa*; single dose 25 mg/kg BW	No effect on plasma testosterone concentration	3
100 mg/kg BW	Plasma testosterone concentration decreased 52%	3
100 mg/kg BW	Plasma citrate levels increased 3.4 times after 48 h	4
206 (95% CI of 147-289) mg/kg BW	LD50; nontolerant populations from South Australia	1,5
300 mg/kg BW	Oxygen consumption reduced 2.5-11.0% over a 22-h postdosing observation period	4
525 (95% CI of 487-589 mg/kg BW)	LD50; tolerant populations from Western Australia	1
Gould's monitor, *Varanus gouldi* 43.6 (95% CI of 27.5-69.2) mg/kg BW; single dose	LD50	1,5
Lace monitor, *Varanus varius*; <119 mg/kg BW; single dose	LD50	1

[a]1, McIlroy et al. 1985; 2, Atzert 1971; 3, Twigg et al. 1988a; 4, Twigg et al. 1986; 5, McIlroy and Gifford 1992; 6, Tucker and Crabtree 1970; 7, Negherbon 1959; 8, Anonymous 1946; 9, Chenoweth 1949; 10, McIlroy 1992; 11, Brock 1965.

Table 3. Effects of 1080 on representative birds.

Table 3. Species, dose, and other variables	Effects	Reference[a]
Chukar, *Alectoris graeca*; 3.5 (95% confidence interval [=CI] of 2.6-4.8) mg/kg body weight (BW); single dose	LD50	1, 2, 3, 4
Northern pintail, *Anas acuta*; 8-10 mg/kg BW; single dose	50-100% dead	2, 5
American wigeon, *Anas americana*; single dose		
4.0 mg/kg BW; males	LD100	5
11.0 mg/kg BW; females	LD100	5
Mallard, *Anas platyrhynchos*		
0.5 mg/kg BW; daily oral dose for 30 days	Some deaths in 30 days, but less than 50%	3, 4
3.7 (95% CI of 21.5-5.5) mg/kg BW; single dose; age 7 days	LD50	6
4.8 (95% CI of 2.6-9.0) mg/kg BW; single dose; age 6 months	LD50	6
6.0 (95% CI of 4.2-8.4) mg/kg BW; single dose; ducklings	LD50	3, 4
7.0-7.5 mg/kg BW; single dose	LD75-LD100	5
8.0 mg/kg BW; adult females; single dose	LD50	2
9.1 (95% CI of 5.6-14.6) mg/kg BW; single dose; adults	LD50	1, 3, 4
10.0 mg/kg BW; adult males; single dose	LD50	2
13-24 mg/L drinking water for 5 days plus 3-day observation period; age 10 days	Avoidance of water containing 1080 when given choice	7
18-24 mg/L drinking water for 5 days plus 3-day observation period; age 10 days	50-90% dead	7
≥236 mg/kg diet fresh weight (FW) for 5 days plus 3-day observation period; age 10 days	Avoidance of diets containing 1080 when given choice	7
527 mg/kg diet FW for 5 days plus 3-day observation period; age 10 days	50% dead	7
Pacific black duck, *Anas superciliosa*; single dose		
10.0 (95% CI of 7.4-13.5) mg/kg BW;	LD50	8

Table 3. Species, dose, and other variables	Effects	Reference[a]
adult breeding females		
18.9 (95% CI of 16.3-219) mg/kg BW; adult males	LD50	8, 9
23.8 (95% CI of 15.3- 37.0) mg/kg BW; adult nonbreeding females	LD50	8
Wedge-tailed eagle *Aquila audax*; 9.5 (95% CI of 7.2-12.5) mg/kg BW; single dose	LD50	8, 10
Golden eagle, *Aquila chrysaetos*; single dose		
1.25-5.00 mg/kg BW	LD50	2, 3, 5, 26
3.5 (95% CI of 0.5-25.1) mg/kg BW	LD50	4, 11
Australian birds; various species; single dose		
7.8 (0.6-25.0) mg/kg BW	LD50 mean and range for 45 species with no known past exposure to naturally occurring fluoroacetates	27
28.4 (1.8-102.0) mg/kg BW	LD50 mean and range for 14 species with prior or continuing exposure to naturally occurring fluoroacetates	27
Australian birds, 41 species; single dose		
0.6-0.99 mg/kg BW	LD50, 2 species	8
1.0-9.9 mg/kg BW	LD50, 27 species	8
20.0-49.9 mg/kg BW	LD50, 11 species	8
>200 mg/kg BW	LD50, 1 species	8
Port Lincoln parrot, *Barnardius zonarius*; 11.5 (95% CI of 9.6-13.7) mg/kg BW; single dose	LD50	9, 12
Great horned owl, *Bubo virginianus*; 20 mg/kg BW; single dose	LD50	5
Rough-legged hawk, *Buteo lagopus*; 10 mg/kg BW; single dose	LD50	5
Ferruginous hawk, *Buteo regalis*; 10 mg/kg BW; single dose	LD50	5
Sulphur-crested cockatoo, *Cacatua galerita*; 3.5 (95% CI of 2.9-4. 1) mg/kg BW; single dose	LD50	8, 13
Galah, *Cacatua roseicapilla*; ~5.6 (95% CI of 3.1-10.5) mg/kg BW; single dose	LD50	8, 14
California quail, *Callipepla californica*		
0.5 or 1.0 mg/kg BW; single dose	No deaths	15
0.5 or 1.0 mg/kg BW on day 1; 2.5 mg/kg BW on days 2, 3, and 4	All dead	15

Table 3. Species, dose, and other variables	Effects	Reference[a]
4.6 (95% CI of 2.7-8.1) mg/kg BW; single dose	LD50	4
>5.0 mg/kg BW; single dose	All dead	15
Turkey vulture, *Cathartes aura*; single dose		
20 mg/kg BW	Lethargy and wing-drooping at 13° C	24
30 mg/kg BW	Tremors, lethargy, ataxia, incoordination at 11-17° C	24
40 mg/kg BW	Lethal at 7-9° C; lethargy, ataxia, and incoordination at 15° C	24
60 mg/kg BW	Tremors, lethargy, and wing-droop at 15-20° C	24
80 mg/kg BW	All dead within 4 h at 20° C; no regurgitation	24
100 mg/kg BW	75% dead at 23-28° C	24
Maned duck, *Chenonetta jubatta*; 12.6 (95% CI of 10.1-15.7 mg 1080/kg BW); single dose	LD50	8, 9
Northern harrier, *Circus cyaneus*; 10 mg/kg BW; single dose	LD50	5
Northern bobwhite, *Colinus virginianus*		
>9 mg/L drinking water daily for 5 days plus 3-day observation period	Avoidance of water containing 1080 when given choice	7
31 mg/L drinking water daily for 5 days plus 3-day observation period	50% dead	7
93 mg/L drinking water daily for 5 days plus 3-day observation period	All dead	7
>95 mg/kg diet daily for 5 days plus 3-day observation period	Avoidance of 1080 diets when given choice	7
385 mg/kg diet daily for 5 days plus 3-day observation period	50% dead	7
Grey shrike thrush, *Colluricincla harmonica*; ~ 12.0 mg/kg BW; single dose	LD50	13
Rock dove, *Columba livia*; 4.2 (95% CI of 3.4-5.3) mg/kg BW; single dose	LD50	1, 2, 3
Black vulture, *Coragyps atratus*; 15 mg/kg BW; single dose	LD50	2, 5
Little crow, *Corvus bennetti*; 13.4 (95% CI of 11.7-15.2) mg/kg BW; single dose	LD50	8, 10, 13

Table 3. Species, dose, and other variables	Effects	Reference[a]
Australian raven, *Corvus coronoides*; 5.1 mg/kg BW; single dose	LD50	10, 13
Little raven, *Corvus mellori*; 3.1 (95% CI of 2.7-3.6) mg/kg BW; single dose	LD50	8
Japanese quail, *Coturnix japonica*; 16.2 (95% CI of 7.2-28.7) mg/kg BW; single dose	LD50	2, 4
Laughing kookaburra, *Dacelo novaeguineae*;~6.0 mg/kg BW; single dose	LD50	10, 13
Emu, *Dromaius novaehollandiae*; 102-278 mg/kg BW; single dose	LD50	8, 12, 13
Red-browed firetail, *Emblema temporalis*; 0.6 (95% CI of 0.4-1.0) mg/kg BW; single dose	LD50	8
Brewer's blackbird, *Euphagus cyanocephalus*; 2.5-3.0 mg/kg BW; single dose	LD33-LD50	2,5
Finches, 7 species; 2.7 (95% CI of 0.8-4.6) mg/kg BW; single dose	LD50	16
Flycatchers, 4 species; 13.2 (95% CI of 8.7-20.0) mg/kg BW; single dose	LD50	16
Domestic chicken, *Gallus* spp.; 5.0-18.0 mg/kg BW; single dose	LD50-LD100	2, 5, 15, 17, 18, 19, 25, 26
Gamebirds, 8 species; 7.3 (95% CI of 0.0-16.4) mg/kg BW; single dose	LD50	16
Australian magpie-lark, *Grallina cyanoleuca*; 8.8 (95% CI of 4.0-13.5) mg/kg BW; single dose	LD50	8, 13
Australian magpie, *Gymnorhina tibicen*; 9.9 (95% CI of 7.6 12.9) mg/kg BW; single dose	LD50	8, 10, 13
Honeyeaters, 5 species; 8.1 (95% CI of 6.9-9.5) mg/kg BW; single dose	LD50	16
Gambel's quail, *Callipepla gambeli*; 20 mg/kg BW; single dose	LD50-LD57	2, 5, 26
Turkey, *Meleagris gallopavo*; 4.8 (95% CI of 1.2-19.0) mg/kg BW; single dose	LD50	4
Black kite, *Milvus migrans*; 18.5 (95% CI of 15.0-23.2) mg/kg BW; single dose	LD50	8, 10, 13
Parrots, single dose 8 species; 4.0 (95% CI of 0.0-9.3) mg/kg BW	LD50	16

Table 3. Species, dose, and other variables	Effects	Reference[a]
5 species; 5-75 mg/kg BW	LD50	9
House sparrow, *Passer domesticus*; single dose		
2.5 mg/kg BW	LD43	5
3.0 (95% CI of 2.4-3.8) mg/kg BW	LD50-LD100	1, 2, 3, 4, 20, 26
Zebra finch, *Peophila guttata*; fed diet containing 10 mg 1080/kg; equivalent to 11-15 mg/kg BW daily	Maximum fluoroacetate concentrations, in mg/kg FW, were 12.6 in crop, 2.0 in stomach, 2.0 in liver, 6.0 in heart, 3.9 in intestine, and 1.2 in muscle; mean concentrations were about 1 mg/kg FW for all tissues except heart (2.0 mg/kg FW)	21
Ring-necked pheasant, *Phasianus colchicus*; 6.5 (95% CI of 3.9-10.8) mg/kg BW; single dose	LD50	1, 2, 3, 4
Black-billed magpie, *Pica pica*; single dose		
0.67 mg/kg BW	No deaths	5
1.3 mg/kg BW	LD100	5
1.6 mg/kg BW; survivors sacrificed at 24 h	Residues of 1080, in mg/kg FW, in survivors were 0.05-0.34 in muscle and 0.07-0.49 In stomach. Dead birds contained 0.2 mg/kg FW in muscle and 0.25 in stomach	22
2.0, 2.5, or 3.2 mg/kg BW	All dead within 24 h. Mean (max.) 1080 residue concentrations, in mg/kg FW, were 0.4 (0.6),0.7 (1.0) and 0.9 (1.4) in muscle, respectively; for stomach, these values were 0.4 (0.9), 0.7 (1.1), and 1.0 (1.5), respectively	22
Pigeons and doves, single dose		
3 species; 10.6 (6-40) mg/kg BW	LD50	9
5 species; 10.6 (95% CI of 1.9-60.9) mg/kg BW	LD50	8, 16, 26
Red-rumped parrot, *Psephotus haematonotus*; ~5.3 mg/kg BW; single dose	LD50	13
Raptors, 5 species; 9.1 (95% CI of 5.1-13. 1) mg/kg BW; single dose	LD50	16
Seed-eating birds; 4 species; single dose; from Western Australia, exposed to fluoroacetate-bearing vegetation; 25-75 mg/kg BW	LD50	12
Pied currawong, *Strepera graculina*; 13.1 (95% CI of 10.9-15.7)	LD50	8

Table 3. Species, dose, and other variables	Effects	Reference[a]
mg/kg BW; single dose		
Laughing dove, *Streptopelea senegalensis*; 5.9 (95% CI of 4.2-8.2) mg/kg BW; single dose	LD50	9
European starling, *Sturnus vulgaris*		
13.5 mg 1080/kg diet for 4 weeks	Treated birds had slightly lower body weight and testes weight than controls, but differences were not statistically significant	23
27 mg 1080/kg diet	No deaths in 5 days	23
47 (95% CI of 27-108) mg 1080/kg diet for 5 days	50% dead	23
54 mg 1080/kg diet for 5 days	67% dead	23
108 mg 1080/kg diet for 3 days	50% dead	23
198 (95% CI of 119-400) mg 1080/kg diet for 24 h	50% dead	23
432 mg 1080/kg diet for 48 h	All dead	23
Waterfowl, 7 species; 7.1 (95% CI of 1.9-25.6) mg/kg BW; single dose	LD50	16
Mourning dove, *Zenaida macroura*; 8.6-14.6 mg/kg BW; single dose	LD25-LD50	1, 2, 4, 5, 20

[a] 1, Tucker and Haegele 1971; 2, Atzert 1971; 3, Tucker and Crabtree 1970; 4, Hudson et al. 1984; 5, Peacock 1964; 6, Hudson et al. 1972; 7, Kononen et al. 1991; 8, McIlroy 1984; 9, Twigg and King 1989; 10, McIlroy and Gifford 1992; 11, Burns et al. 1991; 12, Twigg et al. 1988b; 13, McIlroy 1983a; 14, McIlroy 1981a; 15, Sayama and Brunetti 1952; 16, McIlroy 1986; 17, Anonymous 1946; 18, Kalmbach 1945; 19, Negherbon 1959; 20, Green 1946; 21, Burke et al. 1989; 22, Okuno et al. 1984; 23, Balcomb et al. 1983; 24, Fry et al. 1986; 25, Robison 1970; 26, Chenoweth 1949; 27, McIlroy 1992.

Some birds poisoned with 1080 either vomited (little crow, *Corvus bennetti*; emu, *Dromaius novaehollandiae*; wedge-tailed eagle, *Aquila audax*; sulphur-crested cockatoo, *Cacatua galerita*) or had saliva or fluid dripping from their beaks (Pacific black duck, *Anas superciliosa*; McIlroy 1984). Early signs of poisoning, such as vomiting, were seen at oral doses of 10 mg/kg BW in various raptors including the rough-legged hawk (*Buteo lagopus*), the ferruginous hawk (*Buteo regalis*), the northern harrier (*Circus cyaneus*), and the great horned owl (*Bubo virginianus*; Atzert 1971). The onset of convulsions was preceded by rapid panting, squawking, shrieking or other vocalizations and then a brief period (5-120s) of violent wing flapping, loss of balance, or paddling or running motions with the feet. Birds then fell to the ground while undergoing tetanic seizures, breathing slowly and laboriously, and having wings and tail outstretched (McIlroy 1984). Turkey vultures (*Cathartes aura*) fatally poisoned by 1080 died 4-32 h after dosing; prior to death, birds displayed tremors, ataxia, lethargy, wing drooping and emesis. Turkey vultures were more sensitive to 1080 at colder temperatures of 8-9° C than at 23-28° C; this may be due to inhibition by 1080 of mitchondrial oxidative phosphorylation at colder temperatures that make animals more sensitive at times of increased metabolic demand (Fry et al. 1986).

Some bird species probably developed a tolerance to 1080 from eating plants that contain fluoroacetate or insects and other organisms that fed on such plants (McIlroy 1984). Birds indigenous to geographic areas of Australia where fluoroacetate-bearing vegetation is abundant were more tolerant to 1080 than birds distributed outside the range of the toxic plants. Fluoroacetate tolerance in birds is postulated to increase with increasing evolutionary exposure to the toxic plants and decreasing mobility (Twigg and King 1989). In the low-nutrient environment of Western Australia, fluoroacetate-tolerant herbivores clearly have a potential advantage over nontolerant herbivores in their broadened choice of fluoroacetate-bearing vegetation in the diet (Twigg et al.

1988b). The most sensitive tested Australian bird was the red-browed firetail (*Emblema temporalis*) with an LD50 of 0.63 mg 1080/kg BW (0.007 mg/whole bird); the most resistant tested bird was the emu with an LD50 of about 250 mg 1080/kg BW or about 8,000 mg/whole bird (McIlroy 1983a, 1984, 1986). Emus in the southwestern portion of Western Australia with evolutionary exposure to fluoroacetate-bearing vegetation unusually have a high tolerance to 1080. The tolerance in emus was attributed to (1) their ability to detoxify fluoroacetate by defluorination; (2) a limited ability to convert fluoroacetate into fluorocitrate; and (3) possession of an aconitase hydratase enzyme that is relatively insensitive to fluorocitrate (Twigg et al. 1988b).

Nontarget species of birds have died after eating 1080-poisoned baits (Spurr 1979; McIlroy 1984; Fry et al. 1986; Hegdal et al. 1986; McIlroy et al. 1986a), although population effects have not yet been demonstrated. Dead birds of several species were found after 1080 baits were applied to kill California ground squirrels (*Spermophilus beecheyi*), but only Brewer's blackbirds (*Euphagus cyanocephalus*) contained measurable 1080 residues. Nontarget seed-eating birds that died after eating 1080-poisoned baits included sparrows, blackbirds, towhees (*Pipilo* spp.), horned larks (*Eremophila alpestris*), McCown's longspurs (*Calcarius mccownii*), chestnut-collared longspurs (*Calcarius ornatus*), and western meadowlarks (*Sturnella neglecta*; Hegdal et al. 1986). Individuals of at least 20 species of Australian birds are at risk from dingo and pig poisoning campaigns with meat baits that contain 14-140 mg 1080/kg bait, and 39 species are at risk from rabbit and pig poisoning campaigns with vegetable baits that contain 330-500 mg 1080/kg bait. The extent of bird mortality and possible population effects depend on several factors: bait palatability to each species; availability of other foods; the amount of ingested 1080; the number of birds in each population that consumes baits before the target species or other nontarget groups; and the rate of 1080 leaching from baits by dew or rainfall (McIlroy 1984). Birds that fed on 1080-poisoned baits for control of wild dogs included the pied currawong (*Strepera graculina*), the Australian raven (*Corvus coronoides*), the Australian magpie, (*Gymnorhina tibicen*), and the wedge-tailed eagle (*Aquila audax*; McIlroy 1981b; McIlroy et al. 1986a). Avian scavengers such as vultures, condors, hawks, and ravens probably find poisoned food items as they search for carcasses (Fry et al. 1986).

Secondary 1080 poisoning of birds is documented. Australian birds that died after eating 1080-poisoned carcasses of pigs (*Sus* sp.) included kites (whistling kite, *Haliastur sphenurus*; black kite, *Milvus migrans*), eagles (Australian little eagle, *Hieraaetus morphnoides*; wedge-tailed eagle), the brown falcon (*Falco bevigora*), the Australian kestrel (*Falco cenchroides*), the brown goshawk (*Accipiter fasciatus*), the Australian magpielark (*Grallina cyanoleuca*), the Australian raven, and crows (Australian crow, *Corvus orru*; little crow, *Corvus bennetti*; McIlroy 1983a). Insectivorous birds that may have died after eating 1080-poisoned ants (*Veromessor andrei*, *Liometopum occidentale*) in the United States included acorn woodpeckers (*Melanerpes formicivorus*), the white-breasted nuthatch (*Sitta carolinensis*), and the ash-throated flycatcher (*Myiarchus cinerascens*; Hegdal et al. 1986).

Little or no secondary hazards were evident--as judged by the absence of carcasses--to raptors (hawks, harriers, eagles, ravens, vultures, and condors) from 1080 poisoning of ground squirrels. However, some species of owls including burrowing owls (*Athene cunicularia*) and barn-owls (*Tyto alba*; Hegdal et al. 1986) were comparatively susceptible to 1080. Raptors are less susceptible to secondary poisoning from 1080 than mammalian predators because birds have higher LD50 values, refuse to eat large amounts of 1080-poisoned meats, and sometimes regurgitate poisoned baits (Hegdal et al. 1986). The reduced hazard of acute 1080 poisoning of raptors from secondary sources is illustrated by the golden eagle (*Aquila chrysaetos*), a bird that normally consumes the internal organs of its prey before consuming other portions of the carcass (Atzert 1971). All golden eagles on diets with 7.7 mg 1080/kg diet--about 3 times the highest concentration of 1080 detected in carcasses of coyotes killed by 1080 in livestock-protection collars--survived, although some eagles showed signs of 1080 intoxication such as loss of strength and coordination, lethargy, and tremors (Bums et al. 1991). A 3.2 kg golden eagle must consume the internal organs of 7 to 30 coyotes killed by 1080 to obtain an LD50 dose (1.25-5.00 mg 1080/kg BW), assuming that each coyote ingested 0.1 mg 1080/kg BW and did not excrete, detoxify, or regurgitate any of the toxicant and that, as in rats, about 40% of the dose is present in the internal organs at death (Atzert 1971). Because the internal organs of a coyote account for 20-25% of its live weight or 2.7-3.2 kg/coyote, and a golden eagle's daily consumption of food is about 30% of its live weight or 0.9 kg (Atzert 1971), raptors are probably not at great risk from consuming coyotes killed by 1080 in livestock-protection collars (Burns et al. 1991).

Human consumers of meat from 1080-killed ducks would probably not be adversely affected after eating an average cooked portion (Temple and Edwards 1985). Moreover, oven-baking or grilling at temperatures of

greater than 200° C causes breakdown of 1080. For example, if a mallard received a triple lethal dose of 1080, then a 1-kg mallard would contain an estimated 14.4 mg of 1080. A 70-kg human must consume 25.4 kg of poisoned duck flesh to receive a lethal dose, as judged by LD50 values of 4.8 mg/kg BW in mallards and 5 mg/kg BW in humans; theoretically, consumption of only two whole ducks poisoned by 1080 may cause transient toxicity (Temple and Edwards 1985).

Bird populations that were reduced in numbers during 1080 poisoning of possums usually recovered quickly if they had a high potential for reproduction and dispersal (Spurr 1979). Birds from Australia or New Zealand with poor reproductive potential and poor dispersal had a high risk of nonrecovery; this group includes the three species of kiwi (*Apteryx* spp.), the takake (*Notornis mantelli*), kakapo (*Strigops habroptilus*), laughing owl (*Sceloglaux albifacies*), bush wren (*Xenicus longipes*), rock wren (*Xenicus gilviventris*), fernbird (*Bowdleria punctata*), yellowhead (*Mohoua ochrocephala*), stitchbird (*Notiomystis cincta*), saddleback (*Philesturnus carunculatus*), kokako (*Callaeas cinera*), and New Zealand thrush (*Turnagra capensis*; Spurr 1979, 1993). Control of wild dogs, dingoes, and their hybrids with 1080 meat baits did not significantly affect nontarget populations of birds in the treated areas (McIlroy et al, 1986b). Baiting with 1080 to control rabbits and foxes in Australia usually had no significant permanent adverse effects on nontarget birds, although the abundances of 15 of the 30 bird species in the treated areas tended to decline during the poisoning campaign, especially welcome swallows (*Hirundo neoxena*), tree martins (*Hirundo nigricans*), and crimson rosellas (*Platycercus elegans*; McIlroy and Gifford 1991). Aerial drops of 1080-laced pellets (11.8 kg/ha) to control brushtail possums and rock wallabies (*Petrogale penicillata*) on Rangitoto Island, New Zealand, had no observed effect on island bird populations during the next 12 months (Miller and Anderson 1992). No species of bird showed a population decline and several showed significant increases in numbers, including the greenfinch (*Carduelis chloris*), Australian harrier hawk (*Circus approximans*), and tui (*Prosthemadera novae-seelandiae*). Increases were attributed to the reduction in numbers of mammalian browsers that increased vegetation and improved habitat for nontarget bird species (Miller and Anderson 1992).

The mortalities of nontarget birds from 1080 poisonings may be underreported because many die in their nests or roosts and are never found (Koenig and Reynolds 1987). Dead raptors of several species were found shortly after application of 1080 baits; however, no 1080 residues were detected in any of these birds and the causes of death were not established (Hegdal et al. 1986). Application of 1080 baits to control California ground squirrels was associated with deaths of yellow-billed magpies (*Pica nuttalli*) that contained about 1.02 mg 1080/kg FW of internal organs (Koenig and Reynolds 1987) in contrast to 0.6-0.7 mg 1080/kg FW in stomachs of black-billed magpies (*Pica pica*) that were treated with lethal doses of 1.6-3.2 mg 1080/kg BW (Okuno et al. 1984). Whether *P. nutalli* ingested the 1080 bait directly, ate other poisoned animals, or both is not known (Koenig and Reynolds 1987). Risks of 1080 poisoning to birds can be reduced by (1) setting meat baits out just before sunset and removing them early next morning; (2) burying baits for pigs below ground; (3) using baits that only the target animals prefer; (4) reducing the number of available small bait fragments; and (5) masking the appearance of baits and enhancing their specificity with dyes--although some birds in Australia seem to prefer green-dyed meat baits (McIlroy 1984; McIlroy et al. 1986a).

Mammals

Studies with mammals (Table 4) showed several trends: (1) individuals of sensitive species including species of livestock, marsupials, felids, rodents, and canids died after receiving a single dose between 0.05 and 0.2 mg/kg BW; (2) most individuals of tested species died after a single dose between 1 and 3 mg/kg BW; (3) a latent period was evident between exposure and signs of intoxication; (4) mortality patterns usually stabilized within 24 h after exposure; (5) species from fluoroacetate-bearing vegetation areas were more resistant than conspecifics from nonfluoroacetate vegetation areas; (6) the route of administration had little effect on survival patterns; (7) younger animals were more sensitive than adults; (8) high residues were in some 1080-poisoned animals, notably rabbits with 34 mg/kg DW muscle and 423 mg/kg DW liver; (9) secondary poisoning was evident among carnivores after eating 1080-poisoned mammals; and (10) sublethal effects included testicular damage in rats after drinking water containing 2.2-20.0 mg 1080/L for 7 days (0.07-0.71 mg/kg BW daily), impaired reproduction in mink on diets containing 0.8 mg 1080/kg ration for 60 days, and altered blood chemistry in ferrets on diets containing 1.1 mg 1080/kg ration for 28 days.

Table 4. Effects of 1080 on representative mammals.

Table 4. Species, dose, and other variables	Effects	Reference[a]
Arctic fox, *Alopex lagopus*		
Fed a single bait containing 4 mg of 1080	Muscle contained 0.39 (0.24-0.65) mg 1080/kg fresh weight (FW)	1
Muscle from 66 foxes found dead on Kiska Island, Alaska, after 1080 poisoning. Analysis 60 days after collection	Muscle from males contained 0.7 (0.12-2.2) mg 1080/kg FW; for females, it was 0.81 (0.09-2.8) mg/kg FW	1
Brown antechinus, *Antechinus stuartii*		
1.1-3.5 mg/kg body weight (BW)	LD50, from nonfluoroacetate-bearing vegetation area	2, 3, 4
11.0 mg/kg BW	LD50, from fluoroacetate vegetation area	4
Dusky antechinus, *Antechinus swainsonii*; 3.2 (95% confidence interval [=CI] of 2.4-4.2) mg/kg BW	LD50	3
Black-handed spider monkey, *Ateles geoffroyi*; 10.0-15.0 mg/kg BW	LD50	5, 6, 7, 8
Australian mammals, various; 1.6-20.0 mg/kg BW	Lethal to 8 species of marsupials and 5 species of rodents	9
Australian rodents		
3.1 (0.7-9.0) mg/kg BW	LD50 mean (range) for 10 species with no known exposure to naturally occurring fluoroacetates	10
21.6 (3.5-80.0) mg/kg BW	LD50 mean (range) for 10 species with known past or continuing exposure to naturally occurring fluoroacetates	10
Burrowing bettong, *Bettongia*. *leseuer*; 10-20 mg/kg BW	LD50	11
Cow, *Bos* spp; single dose		
0.078 mg/kg BW	Not fatal to calves and adults	12
0.156 mg/kg BW	Not fatal to cows; LD20 for calves	12
0.22 (95% CI of 0.15-0.33) mg/kg BW	LD50 for steers and calves	5, 11, 12
0.312 mg/kg BW	LD67 for cows; LD80 for calves	12
0.39 (95% CI of 0.25-0.63) mg/kg BW	LD50 for Hereford cows	5, 11
0.624 mg/kg BW	LD100 for cows and calves	12
Canids, 6 species; 0.15 (95% CI of <0.1-0.3) mg/kg BW	LD50	13
Dog, *Canis familiaris*		
0.06 mg/kg BW, single oral dose	LD50; death in 5-9 h	6, 14
0.1-0.35 mg/kg BW, single oral dose	LD100; death in 4-6 h	6, 7, 14, 15, 16
Ingested about 1.96 mg of 1080 (56 g of a 1080-poisoned bait containing 35 mg 1080/kg horse meat)	Vomiting at 1.75 h postingestion; seizure and a short yip 20 min later; seizures and exhaustion for the next 50 min; death at about	16

Table 4. Species, dose, and other variables	Effects	Reference[a]
	3 h after ingestion	
Dingo, *Canis familiaris dingo*		
0.11 (95% CI of 0.09-0.15) mg/kg BW	LD50	3
0.123 (95% CI of 0.110-0.137) mg/kg BW	LD99	13
Coyote, *Canis latrans*		
Fed 1080-poisoned ground squirrels (*Spermophilus* sp.); that contained 0.01-0.09 mg fluoroacetate/kg FW	Maximum residues in dead coyotes, in mg 1080/kg FW, were 0.14 in large intestine, 0.09 in kidney, 0.07 in brain, 0.05 in stomach, and 0.03 in liver	17
0.1-0.2 mg/kg BW	LD50	5, 6, 7, 8, 16
0.13-0.16 mg/kg BW by gavage	Muscle residues were 0.10-0.11 mg/kg FW	18
0.23-0.5 mg/kg BW; poisoned bait	Muscle residues of 0.08-0. 15 mg/kg FW	18
0.5-1.0 mg/kg BW by gavage	Muscle residue of 0.08-0.15 mg/kg FW	18
1 mg/kg BW; poisoned bait	Muscle residue of 0.21 mg/kg FW	18
Ingestion of bait containing 5 mg 1080 (about 2.28 mg 1080/kg BW)	Signs of poisoning noted in 17-18 min after bait ingestion; death in 243 to 313 min after ingestion	19
Single lethal oral dose of 5 mg/kg BW		
Nonrefrigerated muscle tissue	Muscle contained 2.3 mg/kg FW <3 h after death; 1.5 mg/kg FW at 7 days	18
Frozen muscle tissue	Residue of 2.3 mg/kg FW after 30 days, 2.1 mg/kg FW after 60 days	18
Room temperature, muscle tissue	Residues ranged from 1.8-2.0 mg/kg FW between <3 h and 28 days	18
<3 h after death	Residues, in mg/kg FW, were 11.0 in stomach; 2.1-2.4 in heart, muscle, kidney and intestine; and 1.2 in liver	18
30.0 mg/kg BW by gavage	19.5 mg/kg FW in muscle	18
In pen tests, 25 coyotes were offered lambs with collars containing 5 or 10 mg 1080/mL	A total of 23 coyotes attacked and 21 died after collars were punctured in their first (n = 17), second (n = 3), or fifth (n = 1) tests. The average time to death was 217 min (range 115-436 min)	20
Goat, *Capra* sp.		
0.1 mg/kg BW; single oral dose	Half-time persistence of 5.4 h in plasma	21
0.3-0.7 mg/kg BW	LD50	5, 6, 7, 8, 11, 16
Guinea pig, *Cavia* spp.		
0.18 mg/kg BW	LD50 at 4° C	22

Table 4. Species, dose, and other variables	Effects	Reference[a]
0.23 mg/kg BW	LD50 at 32° C	22
0.38 mg/kg BW	LD50 at 17° C	22
0.39 mg/kg BW	LD50 at 24° C	22
Ground squirrels, *Citellus* spp.; 0.3-0.9 mg/kg BW	LD50	5, 6, 7, 15, 16
Hamsters, *Cricetus* spp.; 3.0 mg/kg BW	LD50	6
Black-tailed prairie dog, *Cynomys ludovicianus*		
0.125 mg/kg BW	No deaths	7, 23
0.17-0.3 mg/kg BW	LD50	5, 8, 23, 50
0.4-2.5 mg/kg BW	LD50-LD100	15, 16, 50
Dasyurids, 11 species; 3-12 mg/kg BW	LD50	24
Kowari, *Dasyuroides byrnei*; ~2.8 mg/kg BW	LD50	3
Northern native cat, *Dasyurus hallucatus*; 5.7 (95% CI of 3.9- 8.2) mg/kg BW	LD50	3
Tiger cat, *Dasyurus* *maculatus*; 1.8 (95% CI of 1.3-2.7) mg/kg BW	LD50	3
Quolls, *Dasyurus* spp.		
1.5 mg/kg BW	LD50, nontolerant population from southeastern Australia	4
7.5 mg/kg BW	LD50, tolerant populations from Western Australia	4
Eastern native cat, *Dasyurus viverrinus*; 3.7 (95% CI of 3.2- 4.4) mg/kg BW	LD50	3
Opposum, *Didelphis* *virginana*; 60.0 mg/kg BW	LD50	5
Kangaroo rats, *Dipodomys* spp.		
0.1-0.2 mg/kg BW	LD47-LD85	5, 6, 16
0.2-1.0 mg/kg BW	LD100	16
Mule, *Equus asinus* X *E. caballus*; 0.22-0.44 mg/kg BW	LD50	5, 11, 25
Horse, *Equus caballus*; 0.32-1.00 mg/kg BW	LD50	5, 6, 7, 8, 11, 12, 16, 25
North American porcupine, *Erethizon dorsatum*; ~1.0 mg/kg BW	LD50	5
Eutherian mammals, Australia		
0.36 (95% CI of 0.04- 3.5) mg/kg BW	LD50, 13 species of carnivores	13
0.44 (95% CI of 0.21- 0.60) mg/kg BW	LD50, 7 species of herbivores	13
Feral cat, *Felis cattus*		
0.1-0.19 mg/kg BW	No deaths using poisoned fish baits	26

Table 4. Species, dose, and other variables	Effects	Reference[a]
0.2-0.3 mg/kg BW	LD50, intravenous injection	26
0.28 (95% CI of 0.07-0.49) mg/kg BW	LD50, poisoned fish baits	26
0.35 mg/kg BW	LD50, acute oral route	26
0.35 mg/kg BW	LD90, poisoned fish baits	26
0.4 (95% CI of 0.31-0.52) mg/kg BW	LD50, oral intubation	3
1.3 mg/kg BW	All dead within 24 h when ingested as a fish bait	26
Domestic cat, *Felis cattus*		
0.2-0.3 mg/kg BW	LD50	5, 6, 7, 8, 16
0.5 mg/kg BW	LD100	16
Breviceps pocket gopher, *Geomys breviceps*		
<0.05 mg/kg BW	LD50	5, 6
0.05 mg/kg BW	LD100	16
Tuza pocket gopher, *Geomys floridanus*		
0.2 mg/kg BW	LD50	5, 6
0.25-0.5 mg/kg BW	LD60-LD100	16
Human, *Homo sapiens*		
0.7-2.1 mg/kg BW	LD50 (estimated)	5
2.0 mg/kg BW	LD100 (estimated) for children	16
2.0-10.0 mg/kg BW	LD50 (estimated) for adults	6, 27
Water-rat, *Hydromys chrysogaster*, 2.9 mg/kg BW	LD50	28, 29
Golden bandicoot, *Isoodon auratus barrowensis*; 8.9 (95% CI of 7.2-11.0) mg/kg BW	LD50	30
Northern brown bandicoot, *Isoodon macrourus*; 3.5 mg/kg BW	LD50	30, 31
Southern brown bandicoot, *Isoodon obesulus*		
7.0-8.0 mg/kg BW	LD50; maximum latent period, 183 h; time until death, 7-206 h; time for survivors to recover, 27 h	30, 31
20.0 mg/kg BW; from area of fluoroacetate-bearing vegetation	Tolerated	30
Southern hairy-nosed wombat, *Lasiorhinus latifrons*; 0.21 (95% CI of 0.15-0.29) mg/kg BW	LD50	11
Black-tailed jack rabbit, *Lepus californicus*; 5.6 mg/kg BW	LD50	5, 11
Bobcat, *Lynx rufus*; 0.67 mg/kg BW	LD50-LD100	7, 8, 16
Rhesus monkey, *Macaca mulatta*; 4-12 mg/kg BW	LD50	5, 6, 7, 16
Macropodids, 7 species; 0.23 (95% CI of 0.1-0.6) mg/kg BW	LD50	13
Agile wallaby, *Macropus agilis*; 0.22 mg/kg BW	LD50	11

Table 4. Species, dose, and other variables	Effects	Reference[a]
Tammar wallaby, *Macropus eugenii*		
0.15 mg/kg BW	LD50, pouch young	11
0.27 mg/kg BW vs. 2.0-10.0 mg/kg BW	LD50, adults from nonfluoroacetate- vs. fluoroacetate-vegetation areas	11
Gel containing 12.5 mg 1080 applied to a single leaf of edible foliage	Population reduced 91% in North Island, New Zealand field trial	32
Marsupials		
Various species; fatally poisoned with 1080 under laboratory conditions	Mean residue concentrations, in mg 1080/kg FW, were 0.2 in muscle, 6.1 in viscera, and 29.7 in stomach and contents	33
0.25 (95% CI of 0.1-0.7) mg/kg BW; 10 species of herbivores	LD50, eastern Australia (nonfluoroacetate-vegetation area)	10, 13
2.6 (95% CI of 0.9-7.6) mg/kg BW; 9 species of carnivores	LD50	13
24.2-42.0 (95% CI of 1.5-389.4) mg/kg BW; 10 species of herbivores	LD50, Western Australia (fluoroacetate-vegetation area)	10, 13
From area of fluoroacetate-bearing vegetation		
Red kangaroo, *Macropus rufus*; 2.0-4.4 mg/kg BW	LD50	11
Western brush wallaby, *Macropus irma*; 5-10 mg/kg BW	LD50	11
Western gray kangaroo, *Macropus fuliginosus*; 40-60 mg/kg BW	LD50	11
Brush-tailed bettong, *Bettongia penicillata* and banded hare-wallaby, *Lagostrophus fasciatus*; 100-200 mg/kg BW	LD50	11
Eastern gray kangaroo, *Macropus giganteus*; 0.1-0.4 mg/kg BW	LD50	11
Bennett's wallaby, *Macropus rufogriseus*		
0.2 mg/kg BW	LD50	11
Gel containing about 25 mg 1080 applied to single leaf of edible foliage	Population reduced 87% in South Island, New Zealand, field trial	32
Greater bilby (bandicoot), *Macrotus lagotis*; 15 mg/kg BW; from area of fluoroacetate-bearing vegetation	Tolerated	30
Marten, *Martes martes*; ~1.0 mg/kg BW	LD50	5
Grassland melomys rat, *Melomys burtoni*; 2.6 (95% CI of 2.2-3.1) mg/kg BW	LD50	28

Table 4. Species, dose, and other variables	Effects	Reference[a]
Striped skunk, *Mephitis mephitis*		
Diet		
Fed diet containing 4.1 mg 1080 kg/ration for 5 days (about 2 times level found in 1080-poisoned coyotes)	No deaths or signs of poisoning other than reduced feeding and loss in body weight	35
Fed coyote muscle for 14 to 35 days; coyote had been poisoned with massive (400 mg) dose of 1080	Fatal to all 3 skunks tested	19
Single dose		
0.125 mg/kg BW	No deaths in 7 days	34
0.25 mg/kg BW	LD40	34
0.35 (95% CI of 0.21-0.54) mg/kg BW	LD50	34
0.75 mg/kg BW	LD100	34
Tristram jird, *Meriones tristrami*; fed wheat grain baits		
0.38-0.47 mg/kg BW	50% dead in 3 days	36
1.7-2.5 mg/kg BW	All dead within 24 h	36
Levant vole, *Microtus guentheri*; fed wheat grain baits		
0.24-0.43 mg/kg BW	LD50	36
0.44 mg/kg BW	LD73	36
2.0-2.5 mg/kg BW	All dead within 24 h	36
Meadow mouse, *Microtus haydeni*; 0.3-0.5 mg/kg BW	LD33-LD100	16
Meadow vole, *Microtus pennsylvanicus*; 0.92 mg/kg BW	LD50	5
House mouse, *Mus musculus*		
2.6 mg/kg BW	LD50 at 12.2° C	22
4.5 mg/kg BW	LD50 at 33° C	22
5.8 mg/kg BW	LD50 at 17.9° C	22
7.4 mg/kg BW	LD50 at 30° C	22
8.3 (95% CI of 6.3-11.0) mg/kg BW	LD50	28
10.0 mg/kg BW	LD66	16
12.8 mg/kg BW	LD50 at 24° C	22
Mice, *Mus* spp.		
5.0-19.3 mg/kg BW	LD50	6, 14
13.5 (95% CI of 11.0-16.6) mg/kg BW	LD50; survivors exhibited persistent abnormal behavior ranging from circling to resting with their heads tucked under the abdomen or brisket	37
15 mg 1080/kg BW alone, or followed by intraperitoneal injection of mixture of 130 mg calcium glutonate/kg BW plus 240 mg sodium succinate/kg BW	Alone, 1080 resulted in 80% dead in 48 h and 100% in 120 h. If antidote is administered within 15 min of 1080 exposure, survival increased to 70% at 48 h and 50% at 120 h after 1080 treatment; antidote survivors	37

Table 4. Species, dose, and other variables	Effects	Reference[a]
	recovered much earlier and resumed feeding within 3 days of 1080 injection	
150 mg 1080/kg bait	In pen tests, population numbers were reduced 88% in 20 days	38
Domestic ferret, *Mustela putorius*		
1.41 (95% CI of 1.00-2.00) mg/kg BW	LD50	39
Fed one 1080-poisoned white-footed mouse (*Peromyscus leucopus*) equivalent to 1, 2, 4, or 8 mg/kg BW ferret	All died at all doses except 1 ferret at 2 mg/kg BW	39
European ferret, *Mustela putorius furo*		
Fed internal organs for 3 days of 1080-killed black-tailed prairie dogs	1 of 10 ferrets died and 5 others showed signs of 1080 poisoning; all affected ferrets recovered 24-48 h after exposure	50
Fed ground whole carcasses (less skin, skull, and feet) of black-tailed prairie dogs that died of 1080 poisoning. Carcasses contained 0.05-0.1 mg fluoroacetate/kg FW and composed 90% of diet	No adverse effects after 28 days	23
1.1 mg/kg diet for 28 days	Reduction in red and white blood cell numbers	40
1.2-1.4 mg/kg BW, single dose	LD50	5, 25, 40, 50
9.4 mg/kg diet for 28 days	LD50	40
Mink, *Mustela vison*		
0.1 mg/kg BW	No deaths in 3 days	40
0.25 mg/kg BW	50% dead in 3 days	40
0.5 mg/kg BW	2.5 to 2.8 h to death	40
0.8 mg/kg diet for 2 months prior to breeding	Impaired reproduction	40
1.0 mg/kg BW	1.5 h to death	40
2.9 mg/kg diet for 28 days	40% dead	40
3.2 (95% CI of 2.4-4.5) mg/kg diet for 28 days	50% dead	40
5.25 mg/kg diet for 28 days	Partial paralysis of hind limbs and reduced feed intake by day 5; 90% dead at 28 days	40
Nutria, *Myocastor coypus*; 0.6 mg/kg BW	LD50	5
White-throated wood rat, *Neotoma albigula*		
<0.8 mg/kg BW	LD50	5
0.8 mg/kg BW	LD100	16
Wood rat, *Neotoma intermedia*		
1.0 mg/kg BW	LD20	16
1.5 mg/kg BW	LD50	5
2.0 mg/kg BW	LD100	16

Table 4. Species, dose, and other variables	Effects	Reference[a]
Spinifex hopping mouse, *Notomys alexis*; 32.7 (95% CI of 27.4-39.3) mg/kg BW	LD50	28
Mitchell's hopping mouse, *Notomys mitchelli*; 19.4 (95% CI of 15.8-23.9) mg/kg BW	LD50	28
Mule deer, *Odocoileus hemionus hemionus*; 0.3-1.0 mg/kg BW	LD50, 8-11 months of age	5, 11, 25, 39
European rabbit, *Oryctolagus cuniculus*		
Found dead after consuming 1080-treated carrots; New South Wales, Australia; February 1986	Maximum concentrations of 1080, in mg/kg DW, were 263 in kidney, 423 in liver, 151 in heart, 34 in muscle, 136 in stomach, and 243 in stomach contents. Total 1080 content was 7.04 mg whole body and 4.87 mg in whole body less stomach and contents	33
0.36 (95% CI of 0.30-0.42) mg/kg BW	LD50, immatures	
0.42 (95% CI of 0.26-0.58) mg/kg BW	LD50, adults	
0.51 (95% CI of 0.44-0.58) mg/kg BW	LD90	
Fed pellets containing 10 mg 1080/kg pellet	All dead within 6 h	41
Sheep, *Ovis aries*		
0.1 mg/kg BW; single oral dose	Residues after 2.5 h, in mg/kg, were 0.1 in plasma and 0.02-0.06 in other tissues; after 96 h the maximum value in any tissue was 0.003 mg/kg. Half-time persistence of 1080 in plasma was 10.8 h	21
0.25-0.64 mg/kg BW	LD50	5, 11
2.0 mg/kg BW	LD50	6, 12
Gunn's bandicoot, *Perameles gunni*; 5.4 mg/kg BW	LD50; latent period, 2-6 h; time until death, 4-86 h	31
Long-nosed bandicoot, *Perameles nasuta*; 7.7 (95% CI of 5.3-11.2) mg/kg BW	LD50; maximum latent period, 6.4 h; time until death 4-56 h; time for survivors to recover, 26-42 h	3, 31
Pocket mouse, *Perognathus inornatus*; 1.0 mg/kg BW	LD100	16
Deer mouse, *Peromyscus sp*.		
2.0-4.0 mg/kg BW	LD39-LD50	16
4.0-5.0 mg/kg BW	LD50	5, 6, 15
Raccoon, *Procyon lotor*; single oral dose		
Ambient air temperature of 23-27° C vs. 13-23° C		
0.5 mg/kg BW	LD40 vs. none dead	42

Table 4. Species, dose, and other variables	Effects	Reference[a]
1.0-1.85 mg/kg BW	LD60 vs. LD20	42
2.45 mg/kg BW	LD80 vs. LD60	42
2.82 mg/kg BW	LD100 vs. LD75	42
3.24 mg/kg BW	All dead	42
Plains mouse, *Pseudomys australis*; 1.2 (95% CI of 1.1-1.4) mg/kg BW	LD50	28
Sandy inland mouse, *Pseudomys hermannsburgensis*		
1.6 (95% CI of 1.3-2.0) mg/kg BW	LD50, New Zealand	9
39.3 (95% CI of 23.6-65.4) mg/kg BW	LD50, Australia	28
Long-tailed mouse, *Pseudomys higginsi*; 9.0 (95% CI of 6.2-13.1) mg/kg BW	LD50	28
Western chestnut mouse, *Pseudomys nanus*; 14.7 (95% CI of 13.7-15.9) mg/kg BW	LD50	28
Alexandrine rat, *Rattus alexandricus*		
0.5 mg/kg BW	LD50	5, 6
1.0-2.0 mg/kg BW	LD92-LD100	16
Bush rat, *Rattus fuscipes*; 1.1 (95% CI of 0.9-1.5) mg/kg BW	LD50	28, 29
Swamp rat, *Rattus lutreolus*; 1.7 (95% CI of 1.4-2.1) mg/kg BW	LD50	28
Norway rat, *Rattus norvegicus*		
0.22-3.0 mg/kg BW	LD50, wild strains	6, 43
2.0 mg/kg BW	Oxygen consumption reduced 28-57% in 24 h	43
2.1-2.2 mg/kg BW	LD50, laboratory strains	5
3.0 mg/kg BW	5-fold increase in plasma citrate levels in 4 h	43
4.0-8.0 mg/kg BW	LD72-LD100	16
Black rat, *Rattus rattus*; 1.7 (95% CI of 1.2-2.4) mg/kg BW	LD50	28
Canefield rat, *Rattus sordidus*; 1.3 (95% CI of 1.0-1.6) mg/kg BW	LD50	28, 29
Laboratory white rat, *Rattus* sp.		
Single dose		
0.2 mg/kg BW	LD50	27
4.0 mg/kg BW	LD60	16
7.5 mg/kg BW	LD100	16
10.53 mg radiolabelled 1080/kg BW	After 4 h, radioactivity was highest in carcass (60%), liver (12%), intestine and stomach (10%) and brain, kidney, testes, and spleen (2-3% each)	5
Multiple doses		
Males given drinking water containing 2.2, 6.6, or 20 mg	No overt signs of acute toxicity in any group. However, all groups	44

Table 4. Species, dose, and other variables	Effects	Reference[a]
1080/L for 7 days then observed for 21 days. Daily dose rates, in mg/kg BW, were 0.07 (2.2 mg/L), 0.18 and 0.71 (20 mg/L), respectively	had testes damage (altered appearance, decreased number of spermatids, formation of spermatid and spermatocyte giant cells). The two high dose groups had reduction in testicular weight and seminiferious tubule atrophy; regeneration of tubules was incomplete at day 21 postexposure	
Tunney's rat, *Rattus tunneyi*; 2.6 (95% CI of 2.2-2.9) mg/kg BW	LD50	9
Rodents, 32 species		
<0.1 mg/kg BW	LD50, 4 species	28
0.1-0.25 mg/kg BW	LD50, 6 species	28
0.26-1.0 mg/kg BW	LD50, 15 species	28
>1.0 mg/kg BW	LD50, 7 species	28
Rodents, various, single dose		
0.83 (95% CI of 0.1-6.3) mg/kg BW	LD50, 11 species of cricetids	13
1.05 (95% CI 0.02-2.1) mg/kg BW	LD50, 5 species of *Rattus*	13
2.0-20.0 mg/kg BW	Lethal, 8 species	24
19.4 (95% CI <0.05-48.1) mg/kg BW	LD50, 6 species of pseudo-mice	13
Tasmanian devil, *Sarcophilus harrisii*; 4.2 (95% CI of 2.8-6.6) mg/kg BW	LD50	3
Quokka (kangaroo), *Setonix brachyurus*		
3.5 mg/kg BW	Nontolerant populations had significantly increased plasma citrate levels in 12 h, but none died	45
10-40 mg/kg BW	10 mg/kg BW killed 50% of a nontolerant population; tolerant populations survived	45
60 mg/kg BW	All populations dead within 12 h; plasma citrate levels elevated	45
Cotton rat, *Sigmodon hispidus*		
0.1 mg/kg BW	LD50	5, 6, 16
5.0 mg/kg BW	LD100	16
Fat-tailed dunnart, *Sminthopsis crassicaudata*; 2.1 (95% CI of 1.6-2.7) mg/kg BW	LD50	3
Stripe-faced dunnart, *Sminthopsis macroura*; 0.9 (95% CI of 0.6-1.6) mg/kg BW	LD50	3
Ground squirrel, *Spermophilus beecheyi*; fatally poisoned with 1080		
0.8 mg/kg BW	Tissue residues, in mg fluoroacetate/kg FW, were	17

Table 4. Species, dose, and other variables	Effects	Reference[a]
	0.2-0.7 in brain, kidney, liver, muscle, and lung; 1.0 in caecum; 1.3 in spleen; and 11.8 in stomach	
4.8 mg/kg BW	Tissue residues, in mg fluoroacetate/kg FW, were 0.5-0.7 in brain and muscle; 1.1-1.8 in caecum, kidney, liver, and lung; 9.7 in spleen; and 55.9 in stomach	17
Feral pig, *Sus scrofa*		
0.4 mg/kg BW	LD50, juveniles	46
<1.0 mg/kg BW	LD50, adults	46
1.0 (95% CI of 0.8-1.3) mg/kg BW	LD50	29
1.8 (95% CI of 1.3-185.9) mg/kg BW	LD95	29
2.3 (95% CI of 1.6-3,381) mg/kg BW	LD99	29
4.3 mg/kg BW	LD28 with pellet baits; LD60 with wheat baits	47
21.3 mg/kg BW	LD100; median time to death of 244 min (range 131-7,200 min)	47
Domestic pig, *Sus* sp.		
0.3-0.4 mg/kg BW	LD50, juveniles	5, 7, 8, 16
<1.0 mg/kg BW	LD50, adults	5
Badger, *Taxidea taxus*; 1.0-1.5 mg/kg BW	LD50-LD100	5, 16
Red-bellied pademelon, *Thylogale billardierii*; 0.13 (95% CI of 0.09-0.19) mg/kg BW	LD50	11, 31
Brush-tailed possum, *Trichosurus vulpecula*		
0.3-1.0 mg/kg BW	LD50, from nonfluoroacetate vegetation area	2, 11
16.9 (95% CI of 11.6-24.7) mg/kg BW	LD50 at 10.5° C	22
41.2 (95% CI of 30.2-56.1) mg/kg BW	LD50 at 23.5° C	22
>100->125 mg/kg BW	LD50, from fluoroacetate-bearing vegetation area	11, 45
Gray fox, *Urocyon cinereoargenteus*; 0.3 mg/kg BW	Lethal	5, 16
Bears, *Ursus* spp.; 0.5-1.0 mg/kg BW	LD50	5
Common wombat, *Vombatus ursinus*		
0.15 (95% CI of 0.12-0.19) mg/kg BW	LD50, free-ranging	11
0.22 (95% CI of 0.18-0.27) mg/kg BW	LD50, captive wombats	11
Desert kit fox, *Vulpes*		

Table 4. Species, dose, and other variables	Effects	Reference[a]
macrotis arsipus		
0.22 (95% CI of 0.15-0.34) mg/kg BW; single dose	LD50	48
Fed a 1080-poisoned kangaroo rat (*Dipodomys* sp.). Approximate dose to fox of 0.434 mg/kg BW	Death within 12 h	48
Red fox, *Vulpes vulpes*		
0.08-0.10 mg/kg BW	No deaths	49
0.125-0.15 mg/kg BW	All dead	49
Thick-tailed rat, *Zyzomys argurus*; 3.2-5.8 mg/kg BW	LD50	9

[a] 1,Tietjen et al. 1988; 2, McIlroy 1981a; 3, McIlroy 1981b; 4, King et al. 1989; 5, Atzert 1971; 6, Chenoweth 1949; 7, Anonymous 1946; 8, Negherbon 1959; 9, Calver et al. 1989b; 10, McIlroy 1992; 11, McIlroy 1982a; 12, Robison 1970; 13, McIlroy 1986; 14, Tourtellotte and Coon 1950; 15, Kalmbach 1945; 16, Peacock 1964; 17, Casper et al. 1986; 18, Okuno et al. 1984; 19, Burns et al. 1986; 20, Connolly and Bums 1990; 21, Eason et al. 1994; 22, Oliver and King 1983; 23, Hugghins et al. 1988; 24, Calver et al. 1989a; 25, Tucker and Crabtree 1970; 26, Eason and Frampton 1991; 27, Murphy 1986; 28, McIlroy 1982b; 29, McIlroy 1983a; 30, Twigg et al. 1990; 31, McIlroy 1983b; 32, Warburton 1990; 33, McIlroy and Gifford 1992; 34, Eastland and Beasom 1987; 35, Burns et al. 1991; 36, Moran 1991; 37, Omara and Sisodia 1990; 38, Twigg and Kay 1992; 39, Hudson et al. 1984; 40, Hornshaw et al. 1986; 41, Aulerich et al. 1987; 42, Eastland and Beasom 1986b; 43, Twigg et al. 1986; 44, Sullivan et al. 1979; 45, Mead et al. 1985b; 46, Rathore 1985; 47, O'Brien et al. 1988; 48, Schitoskey 1975; 49, McIlroy and King 1990; 50, Savarie et al. 1994.

The most sensitive tested mammal was the Texas pocket gopher (*Geomys personatus*) with an LD50 of less than 0.05 mg 1080/kg BW (McIlroy 1986). In general, carnivorous eutherian mammals were most sensitive to 1080 and amphibians were most resistant; intermediate in sensitivity were (in that order) herbivorous eutherian mammals and marsupials, carnivorous marsupials, herbivorous-granivorous rodents, omnivorous mammals, and birds (McIlroy 1992). Very young mammals seemed more sensitive to 1080 than other members of their populations (McIlroy 1981a); no other differences in sensitivity to 1080 were found that could be related to sex, age, or nutritional status (McIlroy 1981a, 1981b; O'Brien 1988; O'Brien and Lukins 1988). Route of administration had little effect on 1080 toxicity. Oral dosages were as toxic as subcutaneous, intramuscular, intravenous, and intraperitoneal dosages (Negherbon 1959; McIlroy 1981a, 1983a). There are as yet unexplained species differences in fatal 1080 poisonings: dogs died of convulsions or respiratory paralysis, but monkeys, horses, rabbits, and humans died of ventricular fibrillations (Murphy 1986). Individuals of most species dosed with 1080 died within 7 days, but feral pigs and wedge-tailed eagles took longer (McIlroy 1981a). Ambient air temperatures in the range of 4-33° C modified the sensitivity of small mammals to 1080. In mice (*Mus* spp.) and guinea pigs (*Cavia* spp.), sensitivity was greatest at the extremes of the tested thermal regimes and not at intermediate temperatures (McIlroy 1981b; Oliver and King 1983). Raccoons (*Procyon lotor*) and feral pigs were more sensitive at elevated ambient temperatures (Eastland and Beasom 1986b; O'Brien 1988), but possums and domestic sheep were more sensitive at low temperatures (McIlroy 1982a; Eastland and Beasom 1986b). At elevated temperatures, 1080 was more toxic to feral pigs when administered in drinking water than in oat baits and, in wheat baits than in pellet baits (O'Brien 1988).

Warm-blooded species varied considerably in response to sodium fluoroacetate; primates were more resistant, and rodents and carnivores were more susceptible. Based on fatal or near-fatal cases of human poisonings, the dangerous dose for humans is 0.5-2.0 mg/kg BW (Negherbon 1959). Among the 171 species of tested mammals, for which there are data, variability was considerable in the time until signs of poisoning became apparent (0.1 h to greater than 7 days), the time to death (0.1 h to greater than 21 days), and the time until animals began to show signs of recovery (2 h to 18 days; McIlroy 1986). Signs of poisoning among herbivorous species of marsupials first appeared 1-39 h after dosing; death followed 3-156 h after dosing

(McIlroy 1982a). Australian carnivores did not show signs of 1080 poisoning for 0.6-4.8 h; first deaths occurred between 1.6 and 21 h and recovery in 0.4 to longer than 26 h (McIlroy 1981b). Marsupial carnivores generally showed signs of 1080 poisoning earlier and died or recovered quicker than marsupial herbivores and placental mammals (McIlroy 1986). After the latent period, common signs of 1080 poisoning in caged mammals included hyperexcitation, rapid breathing, and trembling. Some animals then recovered, whereas others began to vomit, convulse, or both (McIlroy 1981b). The most common signs of 1080 poisoning in 14 species of Australian rodents were depression, hypersensitivity to stimuli, respiratory distress, and convulsions; signs usually appeared 0.4-38.1 h after dosing; deaths occurred 0.7-206 h after dosing. Some species were more tolerant, perhaps because of evolutionary exposure to indigenous plants that contained fluoroacetate (McIlroy 1982b). Rabbits (*Oryctolagus* sp.) poisoned by 1080 showed increased sensitivity to noise or disturbance; those surviving high sublethal doses began recovering 5-23 h after dosing (McIlroy 1982a). Cows (*Bos* spp.) showed no signs of fatal 1080 poisoning until shortly before death; signs appeared in the following sequence: urination, staggering, falling down, slight spasms, and death 1.5-29 h after treatment (Robison 1970). Prairie dogs showed no signs of 1080 poisoning for several hours after consuming a fatal dose; death occurred 8-13 h after dosing and was preceded by a rapid respiratory rate, hyperactivity, and convulsions (Hugghins et al. 1988). In feral pigs, signs of poisoning such as vomiting, increasing lethargy, and labored breathing appeared about 6.2 h after dosing (range 1.9-47.3 h) and death, after 16.1 h (range 2.8-80 h) of dosing (McIlroy 1983a). Vomiting occurred in 98% of poisoned pigs but was unrelated to dose (O'Brien 1988) or bait type (O'Brien et al. 1988). In some animals, particularly the eastern native cat (*Dasyurus viverrinus*), the tiger cat (*Dasyurus maculatus*), and the tasmanian devil, the first sign of 1080 poisoning is the sudden onset of vomiting. Vomiting was independent of the ingested dose or mode of administration. Thereafter, animals may either recover or experience hyperexcitation, convulsions, and death (McIlroy 1981b).

Many 1080 control programs had high effectiveness without significant effects on nontarget species. Australian baits for the control of various mammalian pests usually contain 15-110 mg 1080/kg bait, although concentrations as high as 1,200 mg/kg bait are documented (McIlroy 1981b). Baiting with 1080 to control European rabbits and red foxes (*Vulpes vulpes*) in New South Wales, Australia, caused a 90% reduction in numbers of rabbits and 75%, of foxes; populations of both species began to recover soon after the campaign ended, indicating the need for continued control. Populations of nontarget birds and mammals did not seem to be affected, and no dead birds or nontarget mammals were found (McIlroy and Gifford 1991). Reports of 1080 control in Australia of wild dogs, dingoes, and their hybrids are similar (McIlroy et al. 1986b). In Tasmania, deliberate poisoning of forest-browsing pests with carrot baits containing 0.014% of 1080—the same concentration used elsewhere in Tasmania for rabbit control—resulted in 94% mortality of brushtail possum populations, 96% mortality of red-bellied pademelons, and 86% mortality of Bennett's wallabies (McIlroy 1982a). McIlroy (1982a) suggested the use of 1080 to protect island-dwelling rare or endangered species of herbivorous marsupials--a comparatively tolerant group--during control of more sensitive introduced competitors or predators such as rabbits, foxes, and feral cats.

Compound 1080 is highly toxic to some species of nontarget mammals, including domestic cats and dogs (Kalmbach 1945). Baiting of California ground squirrels with 1080 reduced squirrel populations by 85% but also killed individuals of the Heermann's kangaroo rat (*Dipodomys heermanni*), the little pocket mouse (*Perognathus longimembris*), the desert woodrat (*Neotoma lepida*), the deer mouse (*Peromyscus* sp.), and the western harvest mouse (*Reithrodontomys megalotis*); poisoned rodents contained between 5.2 and 23.1 mg 1080/kg BW and 1080-poisoned desert cottontails (*Sylvilagus audubonii*) contained 8.2 mg 1080/kg stomach content (Hegdal et al. 1986). Dead nontarget animals found in New South Wales State forests after 22 rabbit-poisoning operations between 1971 and 1975 included in decreasing order of frequency foxes, wallabies, possums, gray kangaroos, wombats, rats, hares, birds, cats, sheep, and dogs. This pattern may reflect each species' relative abundance in the target areas, access to and acceptance of baits, and ease of detectability after death by forestry personnel (McIlroy 1982a). In Australia, the animals alleged to be most at risk during rabbit or pig-poisoning campaigns with pellet, grain, or carrot baits are the kangaroos, wallabies, and wombats. For example, common wombats (*Vombatus ursinus*) and hairy-nosed wombats (*Lasiorhinus latifrons*) must consume only 10-16 g of pellet, grain or carrot baits containing 0.33 to 0.5 mg of 1080 to receive an LD50. Hairy-nosed wombats eat 120-570 g of food daily, and common wombats can eat more than 500 g of unpoisoned carrots daily, indicating that both species could easily consume lethal quantities of bait. Next at risk in descending order are livestock, brushtail possums, pigs, and various rodents and birds (McIlroy 1986). More data are needed on bait consumption rates by nontarget mammals to assess the risk from 1080 poisoning campaigns.

Laboratory studies may overestimate the risk to nontarget species from 1080 baiting. The northern quoll (*Dasyurus hallucatus*), for example, was at highest theoretical risk from aerial baiting as judged by LD50 laboratory studies with 15 species of rodents and dasyurids. But no dead quolls were found during aerial baiting to control dingoes, and all radio-tagged quolls seemed to have normal movements (King 1989). Alternatives to LD50 testing now include tissue culture, monitoring of metabolite levels in blood or tissues, and estimation of the lowest lethal dose (Calver et al. 1989a). Monitoring the level of citrate in blood plasma of animals that received a sublethal dose of 1080 has been successful with species that are large enough to provide adequate samples of blood plasma during a 24-h period but have not been attempted on Australian fauna (Calver et al. 1989a).

Because 1080 acts as an emetic, especially in coyotes and feral pigs, nontarget animals are at risk of primary poisoning from eating the vomitus (Atzert 1971; McIlroy 1983a; Rathmore 1985; O'Brien et al. 1986, 1988). Wild pigs poisoned by carrot baits for European rabbits left trails of vomitus with carrots and other ingested foods (Rathore 1985). The antiemetic compound metoclopramide (Maxolon) prevents vomiting in pigs by blocking dopamine receptors in the chemoreceptor trigger zones. The addition of metoclopramide to 1080 poison baits for wild pigs reduces vomiting and thereby reduces the poisoning risk to nontarget species. The addition of metoclopramide improves the efficiency and percentage of the kill of wild pigs because they do not develop a taste aversion to the baits. Similarly, baits containing this antiemetic at an effective concentration of 1 mg/kg BW shortened the median time for death in dogs from 151 min postdose in 1080 baits without metoclopramide to 132 min (Rathore 1985). At tested doses (1-16 mg/kg BW), metoclopramide did not decrease the frequency of vomiting by dogs but decreased the amount of vomitus (O'Brien et al. 1986).

Secondary poisoning is probable among carrion eaters feeding on rabbits and other herbivores poisoned with 1080-treated carrots, especially foxes and dingoes (secondary target species) and dogs and cats (McIlroy 1981b; McIlroy and Gifford 1992). Secondary poisoning was experienced by dogs that fed on 1080-treated rodents and prairie dogs and by cats that consumed treated rats and mice (Anonymous 1946). Some dead domestic dogs and cats were found within 450 m of a 1080-treatment area; signs of 1080 poisoning were evident, but no 1080 residues were detected by chemical analyses (Hegdal et al. 1986). Ground squirrel control with 1080 baits caused secondary poisoning of dogs, cats, coyotes, bobcats (*Lynx rufus*), skunks, and kit foxes (Hegdal et al. 1986). The high susceptibility of threatened and endangered populations of the kit fox to 1080 rodenticides, as judged by studies with nonthreatened populations of the kit fox, suggested that 1080 is a factor in their population decline (Schitoskey 1975). Sodium monofluoroacetate has a high degree of secondary toxicity in mammals, as evidenced by deaths of domestic ferrets that ate 1080-poisoned white-footed mice (*Peromyscus leucopus*; Hudson et al. 1984). Similarly, coyotes died after ingestion of 1080-poisoned ground squirrels that contained 3-6 mg of 1080 equivalent to 0.24-0.63 mg/kg BW coyote (Casper et al. 1986; Marsh et al. 1987). Coyotes that ate a single 1080-poisoned squirrel daily for 5 days or an estimated total dose of 0.12-0.27 mg/kg BW usually survived, suggesting that there is little secondary hazard from multiple small doses (Marsh et al. 1987). Carcasses and viscera from coyotes that died after ingesting 5-15 mg of 1080 were fed for 14-35 days to other coyotes, domestic dogs, striped skunks (*Mephitis mephitis*), and black-billed magpies; no evidence of secondary poisoning was seen in any tested species. Maximum residues of 1080 in dead coyote tissues in mg/kg FW were 0.66 in muscle, 0.79 in the small intestines, and 0.76 in stomach tissue (Burns et al. 1986). Tissues of 1080-poisoned coyotes did not produce secondary poisoning in Virginia opossums (*Didelphis virginiana*; Eastland and Beasom 1986a), striped skunks (Eastland and Beasom 1986a; Burns et al. 1991), raccoons (Eastland and Beasom 1986a; Hegdal et al. 1986), or badgers (*Taxidea taxus*; Hegdal et al. 1986). The hazard of secondary poisoning to predators is minimal after consuming tissues of 1080-killed black-tailed prairie dogs (*Cynomys ludovicianus*) because their tissues contained less than 0.1 mg fluoroacetate/kg FW (Hugghins et al. 1988). No mink died when fed 1080-poisoned rabbits at 40% of the total diet if the rabbit gastrointestinal tract had been removed from the carcass. This suggests that secondary toxicity from 1080 is due primarily to consumption of the unmetabolized compound from the gut of prey species (Aulerich et al. 1987). The risk to different individuals or populations depends on the species' sensitivity to 1080, the number of consumed poisoned animals, and the amounts of different consumed tissues or organs (McIlroy and Gifford 1992).

The sensitivity to 1080 poison by animals in Australia varies greatly; known LD50 values range from 0.11 to more than 800 mg/kg BW. Many native species, particularly in Western Australia, have evolved tolerances to 1080 through ingestion of native plants that contain fluoroacetate or prey that consume these plants (McIlroy 1982a; McIlroy 1992). The degree to which this tolerance is developed depends on the extent of the toxic plants in the microhabitat, the need of each species to include food species that contain fluoroacetate in its diet, and

the length of evolutionary exposure to the toxic plants (Twigg et al. 1988b; King et al. 1989; Twigg and Mead 1990). This naturally occurring resistance to the toxins allows control with 1080 to be more specific for introduced test species (Mead et al. 1985). Tolerance to fluoroacetate is present in insects, reptiles, mammals, and birds and descends in magnitude from herbivores to carnivores (Twigg and King 1991). Mammals with lower metabolic rates--such as marsupial carnivores--seem to be more tolerant than mammals with a higher metabolism--such as eutherian carnivores--to a poison such as 1080 that interferes with the metabolism (McIlroy 1981a, 1981b). Tolerance to gradually increasing doses of fluoroacetate can be induced in the mouse, rat, and rhesus monkey but not in the dog or rabbit; however, the protective effect of prior exposure to 1080 seldom persisted for more than 48 h (Chenoweth 1949). Laboratory white rats may acquire tolerances to 1080 by the ingestion of sublethal doses for a period of 5-14 days; cessation of dosing for 7 days caused a loss of tolerance (Kalmbach 1945). Some species acquired tolerances to 1080 after repeated sublethal doses, and others accumulated the chemical until a lethal threshold was reached (McIlroy 1981a). Both phenomena were unpredictable if 1080 residues in the tissues remained between doses. The required time for complete elimination of 1080 from tissues varied among species: dogs required 2-3 days, rats 36 h, and sheep as long as 1 month (McIlroy 1981a).

Sublethal concentrations of 1080 may adversely affect reproduction, growth, and behavior. In rats (*Rattus* sp.), the most vulnerable organ to 1080 poisoning is the testes and this is consistent with 1080-impaired energy production from blockage of the Krebs cycle and subsequent impairment of the carbohydrate metabolism (Sullivan et al. 1979). Subacute dietary exposure to 1080 caused dose-dependent decreases in body weights and feed consumption in mink and in European ferrets (Hornshaw et al. 1986). Toxic 1080 meat baits were usually avoided by tested, nontarget dasyurids and rodents when alternative foods were available; 12 of the 24 tested groups did not sample meat baits under these conditions (Calver et al. 1989a). Adult wild pigs given a sublethal dose of 1080 (0.5 mg/kg BW) in apple baits vomited within 30 min after eating the treated bait and avoided apple baits in future tests (Rathore 1985). Caged wild Norway rats (*Rattus norvegicus*) and black rats (*Rattus rattus*) developed a gradually increasing aversion to drinking water solutions of 1080, although this aversion was not sufficient to disrupt growth and reproduction (Kalmbach 1945).

Recommendations

It is emphasized that 1080 is a restricted pesticide that can only be used by certified applicators who received special training (Green 1946; Negherbon 1959; EPA 1985; Connolly 1993a) and that all organisms that died from 1080 poisoning must be buried or incinerated (EPA 1985). Some authorities aver that continued use of 1080 is justified and desirable and that risk is minimal to nontarget organisms. As discussed earlier, 1080 is a natural plant product, is generally highly toxic to most pests at low concentrations, is readily lost from baits after heavy dews or rainfall, is biodegraded by fungi and bacteria, and does not persist in soil or water. In New Zealand, 1080 has been used since 1954 and is still considered an essential pesticide for limiting forest and crop damage and for containing the spread of tuberculosis to livestock by brush-tailed possums (Eason et al. 1993b). It has been used to control isolated island populations of mammals that prey on endangered or threatened species of birds, for example, Arctic foxes that preyed on Aleutian Canada geese in the Aleutian Islands (Tietjen et al. 1988; Bailey 1993). In Australia and New Zealand, results of field studies suggested that 1080 poisoning campaigns had no significant effect on almost all populations of common nontarget species (McIlroy 1982a, 1992; McIlroy et al. 1986b; McIlroy and Gifford 1991; Spurr 1994), although more studies of vulnerable, rare, endangered, or uncommon species are recommended (McIlroy 1992).

However, a growing body of information questions the usefulness of 1080 in the United States. This database includes adverse effects on some nontarget organisms and endangered species; the confounding effects of the latent period, behavioral alterations, and application routes; and the development of suitable alternative chemicals. On the basis of acute oral toxicity tests, sensitive nontarget mammals and birds may consume lethal quantities of 1080 from poisoned baits or from consumption of organisms fatally poisoned with 1080 (EPA 1985). Field studies showed deaths among sensitive nontarget species that ate 1080 baits, including bees (Goodwin and Ten Houten 1991), insectivorous birds, (McIlroy 1982a; Hegdal et al. 1986), rabbits, rodents (Hegdal et al. 1986), cats, dogs (Kalmbach 1945; Green 1946; Hegdal et al. 1986), and live-stock (McIlroy 1982a, 1986). Carrion eaters and mammalian predators--especially canids and felines—experience secondary poisoning after feeding on 1080-poisoned prey (Hegdal et al. 1986; McIlroy and Gifford 1992). Sublethal effects of 1080 on growth in ferrets and on reproduction in mink are reported (Hudson et al. 1984; Hornshaw et al. 1986). Some endangered species are at risk from direct consumption of the 1080 baits or from secondary

poisoning (EPA 1985). In general, the use of 1080 in the geographic range of any endangered species is discouraged or disallowed outright in the ranges of the California condor, the San Joaquin kit fox (*Vulpes macrotis mutica*), the Aleutian Canada goose; the Morro Bay kangaroo rat (*Dipodomys heermanni morroensis*), and the salt marsh harvest mouse (*Reithrodontomys raviventris*). When exceptions are made or when 1080 use is permitted in an area known to be frequented by an endangered species, restrictions are placed on the maximum concentration of 1080 in the baits (EPA 1985).

Human consumers of meat from 1080-killed ducks are probably not adversely affected after eating an average cooked portion (Temple and Edwards 1985). The risk to humans from eating meat of domestic animals accidentally poisoned with high sublethal concentrations is minimal to low because 1080 is cleared rapidly from domestic animals--usually within a few days (Eason et al. 1994). In the absence of additional data, a minimal 3-week postponement of the slaughter or marketing of livestock that survived 1080 exposure seems prudent. No livestock in the United States contaminated with 1080 are marketed (Connolly 1993a).

No effective antidote to 1080 is currently available, and accidental poisoning of livestock and dogs is probably fatal (Green 1946; Chenoweth 1949; Peacock 1964; Atzert 1971; Mead et al. 1991). The lack of emergency treatment of 1080-poisoned humans and the unavailability of monoacetin--potentially the most effective medication for compound 1080 poisoning--in a pharmaceutical grade (EPA 1985) strongly indicate the need for a viable 1080 antidote. The search for an effective 1080 antidote is ongoing, and some candidate compounds that offer partial protection are mixtures of sodium acetate and ethanol, barbituates (Tourtellotte and Coon 1950; Peacock 1964), glycerol monoacetate (Peacock 1964; Murphy 1986), a mixture of calcium glutonate and sodium succinate (Roy et al. 1980; Omara and Sisodia 1990), and 4-methylpyrazole (Feldwick et al. 1994). The development and availability of an effective 1080 antidote should be of high priority. Until this antidote is distributed, it seems reasonable to use 1080 in the United States only after other alternatives were considered.

The interval between 1080 dosage and signs of intoxication is at least 30 min, regardless of dose or tested species, and must be considered in the evaluation of the efficacy of 1080. Coyotes, for example, may continue to kill livestock after receiving a lethal dose (Connolly and Burns 1990). And coyotes may travel some distance from their prey prior to incapacitation, making carcass recovery and program evaluation difficult--as was the case of 1080-poisoned quolls in Australia (King 1989). Similarly, many 1080-poisoned nontarget animals may have left the treated area before succumbing, thus leading to underestimation of mortality among this group (Collins 1965). Tolerance to fluoroacetates and avoidance of 1080 baits should also be considered in future 1080 poisoning campaigns by wildlife managers and by animal damage-control operators. Avoidance of 1080 toxic baits by target mammals is documented when alternative foods are available (Calver et al. 1989a) and by pigs and rats surviving sublethal exposures (Kalmbach 1945; Rathore 1985). Indigenous populations of mammals, birds, and reptiles that coexist with fluoroacetate-bearing vegetation are much less sensitive to 1080 poisoning, perhaps by as much as 2 orders of magnitude, than conspecifics lacking such exposure (Twigg et al. 1988; King et al. 1989; Twigg and Mean 1990).

The timing of application of 1080 baits is critical. In one mishap, baits were dropped from aircraft while many ground squirrels--the targeted species--were still in hibernation underground for the winter and had not emerged (Collins 1965). Aerial application of 1080 baits in a ground squirrel control program in California, although effective in controlling the squirrels, resulted in great overuse of the baits. As many as 70-77% of the poisoned baits were not eaten by the squirrels and were not recovered. Also, the yellow dye to color the baits--as a deterrent to birds--faded rapidly (Collins 1964, 1965). To protect migratory waterfowl, 1080 baits should not be applied immediately preceding or during the main waterfowl hunting season or whenever birds are abundant (Temple and Edwards 1985). To protect honeybees, 1080-poisoned jam baits should be deposited more than 400 m from apiary sites. If 1080 baits are dispersed less than 400 m from apiary sites, beekeepers should remove their hives to a more distant site (Goodwin and Ten Houten 1991). The 1080 toxicity database on aquatic organisms is insufficient for practicable formulation of criteria to protect this ecosystem. This seems to be a high-priority research need in geographic areas of intensive 1080 application.

Potential replacement chemicals for 1080 include PAPP (*para*-aminopropiophenome), DFP (1,3-difluoro-2-propanol), and various anticoagulant and nonanticoagulant toxins. PAPP is highly toxic to coyotes and domestic cats (each with LD50s of 5.6 mg/kg BW) and lethal to rats (LD50 of 177 mg/kg BW) and mice (LD50 of 233 mg/kg BW); intermediate in toxicity to bobcats (10.0) and to kit foxes (14.1 mg/kg BW; Savarie et al. 1983). DFP

is under investigation in Australia as an alternative to 1080 for management of fauna because it has a mode of action similar to that of 1080 and has an antidote in pyrazole (Mead et al. 1991). DFP is the major ingredient of the pesticide gliftor used in Russia to control rodents, particularly voles of the genus *Microtus*. Also deserving of evaluation are 4-methylpyrazole and related compounds to function as antidotes to DFP intoxication (Mead et al, 1991). In New Zealand, alternatives to 1080 under evaluation include several nonanticoagulant toxins (gliftor, cholecalciferol, calciferol, alpha-chloralase, nicotine, malathion) and anticoagulants including brodifacoum and pindone (Eason et al. 1993a).

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