Policy Implications of 1080 Toxicology in New Zealand

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Abstract

Sodium monofluoroacetate (1080) is used for large-scale pest control operations in New Zealand to control the introduced marsupial brush tailed possum. Wide-scale opposition to the use of 1080 has grown in recent years with the development of a substantial "anti-1080" lobby. Concerns for public health and effects on non-target animals among critics of 1080 have prompted the Environmental Risk Management Authority to undertake an official review of this pesticide. In anticipation of this regulatory review an evaluation of the peer-reviewed scientific literature was conducted on the risks associated with the use of 1080, in order to ascertain the degree to which regulations on 1080 reflect current scientific knowledge of the toxicology of this poison. Key areas of concern revealed in the literature include evidence that 1080 could have endocrine disrupting capabilities and that it is relatively slow to break down at low temperatures when microbial activity is low. These two issues are yet to be fully resolved through further research and represent significant gaps in current knowledge. If regulations are to take full account of current science on 1080 they will need to acknowledge and reflect what is known, the gaps in this knowledge, and the risks associated with this uncertainty. Recommendations include further targeted research to fill gaps in current knowledge, regulatory precaution until such research is completed, and explorations of alternative methods to be used either in conjunction with, or instead of this toxin.

Keywords: Sodium monofluoroacetate; 1080; chronic toxicology; human health; public policy; New Zealand.

Introduction

In January 2003 a community activist was convicted of burglary and willful damage for sabotaging a bulk supply of 1080 poison (sodium monofluoroacetate) owned by the New Zealand Department of Conservation (DOC). The poison was to be used for a pest control operation to control the brush tail possum, an introduced pest marsupial native to Australia, which poses a considerable threat to native vegetation and wildlife in parks and reserves. The Department of Conservation argues that 1080 poison is among the most cost effective and efficient form of pest control for this and other mammalian pest species and that the conservation benefits of this poison are substantial. The poison is also used in agriculture to control bovine tuberculosis, which is spread by possums. The above attack cost DOC $12,000 according to its local area manager (Shuttleworth 2003). In defense the activist said that he was trying to protect his community from a "weapon of mass destruction," pointing to the threat to domestic water supplies of 1080 contamination.

This political and media drama is merely one episode in a much larger controversy in New Zealand over the widespread use of 1080 poison as a conservation management tool. A search of web-based media publications and print media archives easily reveals a broad selection of stories focusing on community concerns relating to 1080 use. They range from fire bombings of DOC vehicles to protests, petitions and conferences. In short, a substantial anti-1080 lobby has developed in New Zealand with concerns primarily focusing on acute and chronic human health risks and the non-target effects of the poison in the field. In terms of the latter, the recreational hunting fraternity has become very vocal with complaints that deer populations have been decimated as a result of aerial 1080 applications in hunting areas. Another concern for hunters is the risk to their dogs, which are particularly susceptible to secondary poisoning after eating poisoned possums. These concerns reflect growing public interest in environmental health (Leggat 2003) as well as a degree of self-interest among hunters who do not want their sport to be jeopardized by a non-specific poison.

As a consequence of the build up of community disquiet over 1080 use in recent years the Environmental Risk Management Authority (ERMA) decided in March 2002 that there were sufficient grounds for a full scientific review of the poison (ERMA 2002)[1]. This will be[2] the first official review of the poison since its introduction for pest control in 1964. The review will enable an assessment of the risks, costs, and benefits of 1080 under the provisions of the Hazardous Substances and New

[1] In Australia a review of 1080 is currently underway to ensure there is minimal non-target impact and minimal environmental impact. The results of this review are likely to be published in 2004.
[2] At the time of writing (early 2003) the ERMA review had not officially commenced but had been publicly announced.

Organisms (HSNO) Act (1996), and provide an opportunity for the wider public to raise their concerns.[3] This paper was prepared in response to this review as a contribution to the debate (i.e. as an independent public submission).[4] The central research questions for this analysis are:

- What can the peer-reviewed scientific literature tell us about the health risks associated with 1080?
- What are the policy implications of this scientific knowledge and/or gaps in this knowledge?

Science is an integral tool for the setting of regulations relating to public health and environmental protection. Scientific knowledge however, is not always complete, particularly in the complex arena of public health toxicology and the effects of chemicals on biological systems. Where it is incomplete, regulators are compelled to build regulatory bridges over these gaps until they can be filled. One such regulatory bridge is the employment of the precautionary principle which, according to Principle 15 of the 1992 Rio Declaration, invites states and regulators to implement cost-effective measures to prevent adverse environmental effects, where there are threats of serious or irreversible damage, even when there is a lack of full scientific certainty (UNEP 1992). Several issues are associated with science, its relationship with regulatory structures, and the theme of precaution:

- Scientists do not always agree with each other because there are a variety of different theoretical standpoints and degrees of understanding among scientists in any scientific discipline.
- There is commonly a lag time between scientific understanding and the incorporation of this understanding into regulations. Sometimes regulations never catch up with science.
- New scientific consensus is commonly preceded by a period where evidence of causal mechanisms build up ahead of conclusive research findings that are capable of verifying such causal mechanisms. This evidence may point to significant potential risks that are relevant to regulatory structures.
- The role of government is to protect the public good whilst also protecting private and other economic interests. For example, section 9 of the (Methodology) Order 1998 of the Hazardous Substances and New Organisms (HSNO) Act (1996) instructs ERMA to "recognize risks, costs, benefits, and other impacts" when registering pesticides. As such, this regulatory authority is responsible for weighing risks against benefits of toxic substances, rather than purely protecting the public good from toxins. As such, by definition the ERMA is not an advocate for public or environmental health per se, but instead an advocate for balancing risks and benefits of potential hazards.

The following sections explore the history of the toxin and its use, followed by a summary of scientific understanding of the acute and chronic toxicological effects of the toxin, including research on its effects on non-target organisms, and rates of degradation. Policy implications of this knowledge are then discussed, leading to a set of policy recommendations

Naturally occurring toxin

Sodium monofluoroacetate is a naturally occurring toxin found in a number of different plant species that may have developed this compound as a defense strategy against browsing (Twigg et al 1996). This led to a degree of toxin tolerance among certain herbivores and carnivores preying on tolerant herbivores (King et al 1996; Martin and Twigg 2002). The toxin was first isolated as a component of the African plant *Dichapetalum cymosum* in 1944 and was the first organofluorine compound known to occur naturally (Twigg et al 1996). Several other species of this same genus have since been discovered to contain the toxin (Meyer 1994), as with South American plant *Palicourea marcgravii* (de-Moraes-Moreau et al 1995), and forty-one species of legume in Australia (Twigg 1994).

[3] The purpose of the HSNO Act (1996) is to "protect the environment and the health and safety of people and communities, by preventing the adverse effects of hazardous substances and new organisms" (section 4).

[4] The author regards himself as a conservationist who is acutely aware of the importance of pest control in conserving functional ecosystems (as habitats) that provide for the protection of New Zealand's unique contribution to global biodiversity. He is also aware that pest control comes at a price and that this price needs to fit within the budget of agencies charged with the responsibility to undertake such management. The author is also concerned for the appropriate management of hazardous substances in the protection of public health.

The manufactured compound '1080' (first synthesized in 1896 in Belgium) is chemically identical to naturally occurring sodium monofluoroacetate and exhibits identical symptoms of poisoning in animals (Eason et al 1999). It was first recorded as toxic in the US in 1934 and thereafter patented as a rodenticide in the late 1930s (Rammell and Fleming 1978). It was developed as a pest control agent through the 1940s and 1950s and was used primarily for the control of coyotes (Fagerstone et al 1994). In 1972 the US EPA banned the use of 1080 for predator control except for its use in livestock protection collars (LPC), designed to kill coyotes when they bite the neck of a lamb or kid goat. At that time most of 1080 use was for rodent control and this was not affected by the ban. Subsequently, all rodenticide registrations for 1080 were cancelled in 1990 (Fagerstone et al 1994), leaving the LPC as the only registered form of the pesticide in the United States. The only registration for LPC use (EPA Reg. No. 56228-22) is held by the Animal and Plant Health Protection Service of the US Department of Agriculture (APHIS).

Compound 1080 was first imported into New Zealand in 1954 for the control of rabbits (Rammell and Fleming 1978). It has since become a key pest management tool for the control of brush tail possums and is commonly dispensed in the form of cereal-based pellets and carrot baits, dropped by air, or dispersed by hand. Since 1080 was de-registered in the US for rodenticides up to 90% of world production is now imported to New Zealand according to media reports (Evening Post 2002; The Timaru Herald 2002). An estimated $27 million was spent in the 1993/4 season on possum control with 1080 being the primary tool for this task, employing 5,000 tones of 1080 carrot bait and 1,200 tones of 1080 cereal bait (Livingstone 1994). In the 2001/2 year, Animal Control Products produced 3023 kg of 1080, of which 2271 kg was used by the Animal Health Board and 581.30 kg used by the Department of Conservation (Table 1.).

Table 1. 1080 Production and use (kg) by different agencies.

Year	ACP (Production)	DOC (use)	AHB (use)	Other users
1996/97		378.19		
1997/98	2532	534.08	1798	199.92
1998/99	1915	504.12	1270	140.88
1999/00	2111	658.21	1308	144.79
2000/01	2455	481.16	1777	196.84
2001/02	3023	581.30	2271	170.60

(Source: Parliamentary Questions for written answer No. 012822, 012824, 12823 November 2002).
DOC - Department of Conservation
AHB - Animal Health Board
ACP - Animal Control Products

As of 2002, $53 million was being spent annually controlling possums and other bovine tuberculosis vectors over 8.5 million ha of land. In addition to this, $21 million is being spent annually on controlling possum and other mammalian pests on 770,000 ha of publicly owned conservation land. The possum control component of regional pest management strategies costs $15 million annually.[5]

Science, evidence and policy

An objective assessment of the health risks of a toxin requires consideration of:

1. Empirical evidence of adverse health effects where there is scientific consensus.
2. Explanations of the biological mechanism associated with adverse health effects.
3. Empirical evidence of potential adverse health effects where scientific consensus is currently lacking.
4. Explanations of the biological mechanism for any potential adverse health effect.

[5] Figures from the National Science Strategy Committee for Possum Bovine Tb Control. Draft, June 2002.

Where there is substantial data and scientific consensus on points 1 and 2 above, regulations commonly, but not always, follow as a protection from known risks. Where regulations do not follow in spite of scientific consensus we simply have a case of, what is often a politically normal, lag time between scientific knowledge and the translation of such knowledge into policy and law. Points 3 and 4, on the other hand, are normal components in any potential shift in scientific consensus. If both evidence and credible scientific explanations for any potential health effects are lacking then no shift in scientific consensus is likely and no shift in regulations would be scientifically warranted.

To make a scientifically robust assessment of the established and potential health risks associated with 1080, it is also important to consider the various ways in which toxic compounds can affect living systems. These include acute and chronic health effects, exposure pathways, dose response variations, persistence in the environment, movement pathways for the chemical, variations in vulnerability to the toxin, and potential effects on non-human organisms.

Acute toxicity

The toxicity of chemical poisons can be understood in terms of short term (acute) and long-term (chronic) toxicity (Corvalan et al 2000). The severity of toxic effects in both short and long term toxicity ranges from minor irritation to death. An example of chronic toxicity leading to death can be seen in arsenic contamination of water, where low doses over a long sustained period can lead to cancer (Corvalan et al 2000). Most pollutants have a threshold below which no adverse health effect occurs or is evident. Others have no, or at least very low safety thresholds and can cause adverse health effects even at extremely low doses (e.g. some genotoxins – i.e. those that cause DNA damage, and can lead to malignant tumors, but these would tend to fall under the heading of chronic toxicity)[6] (see Bickham and Smolen 1994).

The vast majority of 1080 toxicity studies available in the published literature focus on acute toxicity in animals with an emphasis on severe reactions. Acute and severe toxicity in humans is also documented although for obvious ethical reasons there are no experimental data relating to dose response relationships. The current scientific consensus is that sodium monofluoroacetate is a deadly human poison. According to the EPA (1987) "This material is super toxic. The probable oral lethal dose in humans is less than 5 mg/kg, or a taste (less than 7 drops) for a 150-lb. person." Exposure symptoms include nausea, blurred vision, numbness, low blood pressure, hyperactivity, excessive salivation, respiratory depression or arrest, cyanosis (blue tint to the skin and mucous membranes), vomiting, diarrhea, hyperactive behavior, convulsions, coma, ventricular fibrillation and heart failure. These are normally observed within 30 min of exposure, although evidence of severe effects may not be apparent for up to 20 hr following exposure (EPA 1987). The pathway to toxicity is by ingestion inhalation, dermal absorption, eye and skin contact.

Once fluoroacetate has been absorbed or ingested it is converted to fluorocitrate in the body (Peters and Wakelin 1953), which is the toxic form of the chemical, where it accumulates in the fetus and certain organs such as the heart, lungs, kidneys, liver, and testes (McTaggart 1970; Sullivan et al 1979; Twigg et al 1988). Fluorocitrate, in turn, inhibits the tricarboxylic acid cycle in the Krebs cycle (Eason 1997; Schofl et al 2000) by competitively inhibiting the enzyme aconitate hydratase (Ataria et al 2000). Here, citrate would normally be converted to aconitate, but the blocking of this leads to toxic accumulation of citrate. As a result, energy production (a key function of the Krebs cycle) falls, which in turn leads to cellular energy deprivation and death (Rammell and Fleming 1978; Twigg 1994). The accumulation of citric acid causes violent convulsions and death from cardiac or respiratory failure (Chi et al 1999).

The few human case studies on the acute toxicity of 1080 available in the scientific literature provide little detail on its human effects. A rabbiter who was repeatedly exposed to 1080 developed kidney failure and showed evidence of other organ damage (Parkin et al 1977). Other human studies have arisen from poisoning cases in hospitals. Chi et al (1996) undertook a retrospective study of 38 cases of 1080 poisoning and found that the early onset of metabolic acidosis, and increased serum creatinine were associated with poor long term survival in humans. Chi et al (1996, 1999) found that hypotension is one of the most important predictors of mortality in 1080 poisoning. Robinson et al (2002) studied the symptomatic response in a 47-year-old male survivor of 1080 poisoning. He observed the patient to respond only to noxious stimuli after 34 hours and noted that the patient was non-responsive to painful stimuli at 48 hours following ingestion.

[6] DNA damage in itself is not sufficient to cause toxicity in all cases, because DNA is also capable of repairing itself (see Wood et al 2001).

Animal studies

Studies on the metabolism of sodium monofluoroacetate on animals have shown that unmetabolised fluoroacetate and at least seven breakdown products are excreted in the urine (Hagan et al 1950; Sykes et al 1987; Eason et al 1994a). Symptoms such as vomiting, nausea, heart and respiratory failure are usually apparent following a lag period of 0.5 to 3 hr (Eason et al 1994a; Twigg 1994). Herbivores receiving lethal doses tend to respond to intoxication with cardiac failure whereas carnivores tend to experience central nervous system dysfunction and eventually die of respiratory failure (Egeheze and Oehme 1979). The elimination half life for near lethal doses is 11 hr in sheep, 2 hr in mice, 1 hr in rabbits, and 5 hr in goats (Eason et al 1994). Animal studies in the US have shown 1080 to be absorbed through the skin, which has important implications for safety procedures, and regulations for bait handlers and manufacturers (Fagerstone et al 1994).

Toxicology studies conducted by APHIS looked at the effects of absorption of 1080 through the skin and eyes of rabbits. The results of the dermal toxicity tests demonstrated an LD_{50}[7] of 324 mg kg^{-1}, and allowed it to be classified as a Category IV skin irritant. The study of the effects of 1080 on the eyes of rabbits treated with a 1% solution showed only slight conjunctival irritation enabling it to be classified as a Category III eye irritant (Fagerstone et al 1994). Three aquatic toxicity studies were undertaken by APHIS in the early 1990s. One looked into the acute toxicity of variable 1080 concentrations for bluegill sunfish (*Lepomis macrochirus*) where no lethal or sub-lethal adverse effects were observed at any concentration. Another looked into the acute toxicity of 1080 on rainbow trout (*Oncorhynchus mykiss*) using the same test conditions, and found after 96 hr mortality ranging from 10% (at 23 mg l^{-1}) through 50% to 90% depending on concentration (at concentrations between 39 and 170 mg l^{-1}). The third study looked into the acute toxicity of 1080 on *Daphnia magna*, a freshwater invertebrate. After 48 hr, 70 and 100% immobilisation was observed in daphnids exposed to concentrations of 350 and 980 mg l^{-1} respectively (Fagerstone 1994). Eason (1997) reviewed experimental and regulatory toxicology studies on 1080 and mentions a study cited in Ramell and Fleming (1978) where fingerling trout were subjected to 1080 concentrations of 500 mg/l and 1000 mg/l without any visible effect on the fish. He also noted that force-feeding pellets containing approximately 4 mg and 8 mg of 1080 to fingerling and adult trout had no visible effect.

The effects of 1080 on non-target wildlife populations have been extensively studied in New Zealand and Australia (Booth and Wickstrom 1999; Hartley et al 1999; Lloyd and McQueen 2000, 2002; McIlroy 1981a; 1981b; 1982a; 1982b; 1983; 1984; 1986; 1994; McIlroy and Gifford 1991; McIlroy et al 1985; McIlroy et al 1986; Morgan 1999; Perfect 1996; Powlesland et al 1999; 2000; Robertson et al 1999; Spurr 1994; Spurr and Drew 1999). Each of these studies has focused on acute toxicity only, where mortality rates of non-target wildlife were assessed following 1080 control operations.

Secondary poisoning

The issue of secondary poisoning in the wake of pest control operations has raised concerns associated with:

- The risk of humans consuming domestic animals exposed to 1080
- The risk of animal carcasses being eaten by scavenging species and hunting dogs

A study by Meenken and Booth (1997) assessed the risk of secondary poisoning of 1080 in dogs and found that possum carcasses collected after a possum control operation contained concentrations of 1080 high enough to pose a serious hazard to dogs even up to 75 days after poisoning. Conversely, there are studies that observed no ill effects on non-target animals such as the one by Hugghins et al (1988), which showed that ferrets suffered no ill effects after feeding on prairie dogs that had died from 1080 poisoning.

Other studies into non-target effects have looked into the effects of 1080 in the secondary poisoning of predators, and the potential for 1080 and its breakdown products to persist in meat (e.g. Hugghins et al 1988; Allender 1990; Gooneratne et al 1994, 1995; Eason et al 1994b; Savarie et al 1994; Meenken and Booth 1997; Murphy et al 1999; Gillies and Pierce 1999). In most of these studies the findings showed no evidence for concern for acute adverse effects on non-target species, apart from the effects of secondary poisoning of predators eating carcasses of animals killed by lethal doses of 1080 (Meenken

[7] The LD_{50} is also known as the median lethal dose. It is the dose of a substance that will kill 50% of a sample population (McIlroy 1994).

and Booth 1997). For example, stoats can also suffer mortality from secondary poisoning following control operations for possums (Murphy et al 1999), and from taking baits directly (Moller et al 1996).

The location of the poison in carcasses has been shown to be higher in plasma compared with muscle and organ tissue (Gooneratne et al 1995). This tendency of 1080 concentrations to be highest in blood plasma was also shown in a study that looked at the potential for human secondary poisoning. Eason et al (1994b) administered 1080 orally to sheep and goats at a dose of 0.1 mg kg^{-1} body weight to assess the risk to humans of eating meat contaminated with 1080. Poison residues were measured in blood, muscle, kidney and liver. The plasma elimination half-life of 1080 was 10.8 hr in sheep and 5.4 hr in goats. The concentrations of the poison in plasma were significantly higher than in other tissues. Concentrations of 1080 in sheep tissues dropped to <0.002 to 0.008 mg kg^{-1} after 96 hr. They concluded that human secondary poisoning from meat contaminated with 1080 is highly unlikely due to the elimination of the toxin from tissues and the fact that livestock are usually removed from areas near 1080 applications. A minimum withholding period of 5 days is currently recommended for stock that are suspected to have come into contact with 1080 even when no deaths have been observed. Longer periods of quarantine prior to slaughter are recommended when a livestock death has been observed (Eason et al 1994b). Animals have variable sensitivity to 1080 depending on the species, which means that lethal doses will vary and some species are more vulnerable than others. Dogs for example are known to be far more sensitive to the toxin than many other mammals (Eason 1997; Meenken and Booth 1997), which is of particular concern in relation to secondary poisoning when a dog eats a carcass of a poisoned animal. Limited research on the development of an antidote for 1080 has been conducted (e.g. Omara and Sisodia 1990; Gorniak et al 1994; Cook et al 2001).

In regions where fluoroacetate occurs naturally in plants, some animal species have developed a tolerance to the compound. Where animals have developed a resistance to sodium monofluoroacetate (e.g. in parts of Australia) the degree of tolerance will tend to be a function of how long they have lived in association with such plants, the degree of reliance on these plants as a food source, their degree of mobility as a species, and the size of their home range (Twigg 1994; King et al 1996; Martin and Twigg 2002). Herbivores tend to have the highest degree of tolerance, followed by omnivores, followed by carnivores. This tolerance has also been observed in pest species targeted with 1080 poison campaigns. According to Twigg (News in Science 2002) the LD_{50} for rabbits was about 0.45 mg kg^{-1} in 1979, but had increased to 1.1 mg kg^{-1}, and that the tolerance was greatest in areas where the poison has been used intensively.

Chronic toxicity

The relationship between a toxin and observed increases in mortality within a population has tended to dominate environmental health assessments and the setting of regulations. Cancer provides a good example. Corvalan et al (2000:89) point out however, that linking mortality data with well-known toxins is not easy in population studies. The link between tobacco and cancer is a paradigm case, where, even though the source of the toxin was clear and the biological mechanism of toxicity was well understood, for many years it remained extremely difficult to "prove" that smoking caused cancer in public health regulatory debates. In most situations of environmental toxicology "only a small subset of a population experiences high levels of exposure, and the doses received by the general population are so low that only vulnerable high-risk groups are severely affected". According to an editorial in the Lancet in 1992 relatively few studies have shown clear associations between environmental pollutants and actual increases in death rates (Lancet 1992).[8] As such, mortality data are a very insensitive measure of toxicity for environmental contaminants, and yet these have formed the basis of most regulatory standards for environmental toxins.

Some studies on the chronic toxicology of 1080 have been conducted, although significant gaps in our knowledge remain. Eason et al (1999) reviewed recent research into the chronic toxicology of 1080 and organized these studies into the following categories: mutagenicity, developmental toxicity, and teratogenic potential. A teratogen is an agent that can cause malformations of an embryo or fetus. This can be a chemical substance, a virus or electromagnetic radiation that can affect parents and offspring. Symptoms of teratogenic influences tested in parents and offspring include: sperm abnormalities (decreased number/motility, abnormal morphology of sperm); sub-fecundity (abnormal gonads/ducts of external genitalia); abnormal pubertal development; infertility of male/female; delay in conception; illness during pregnancy/parturition (toxemia; hemorrhage); early fetal loss; late fetal loss (stillbirth,

[8] This is one of the great difficulties of epidemiology and its role in public health policy, particularly in a culture where an agency producing a contaminant is innocent until proven guilty in a court of law.

death in the first week); decreased birth weight; premature/ post mature births; altered sex ratio of offspring; chromosome abnormalities; multiple births; birth defects; infant death; offspring morbidity; and, offspring malignancies (Amdur et al 1991).

No mutagenicity was observed at any dose level in 3 studies into the mutagenicity (genotoxicity) of 1080 (Ames et al 1975; Blazak et al 1989; Hoddle et al 1983).

A developmental toxicity study conducted by Eason et al (1999) on female rats, showed no maternal mortality at oral 1080 doses of 0.1, 0.33 or 0.75 mg kg^{-1} day^{-1}, but did show decreased maternal body weight, decreased weight gain, and decreased food consumption at 0.75 mg kg^{-1} day^{-1}. No soft tissue abnormalities were observed in fetuses at any dose. Fetal skeletal abnormalities, including abnormalities in forelimb development and bend ribs were observed at doses of 0.33 and 0.75 mg kg^{-1} day^{-1}. The authors concluded that the No-Observable-Effects-Level (NOEL) for maternal toxicity was 0.33 mg kg^{-1} day^{-1}, and the NOEL for developmental toxicity for rats subjected to 1080 is 0.1 mg kg^{-1} day^{-1} even though they did not investigate fetuses beyond gross observable effects. They concluded that 1080 is teratogenic in rats at, but not below, 0.75 mg kg^{-1} day^{-1}. On the basis of this study, the New Zealand Ministry of Health has set the Maximum Acceptable Level (MAV) for 1080 at 0.0035 mg l^{-1}.

In the Eason et al (1999) study, birth defects were observed[9], but apart from that, none of the above teratological symptoms were tested, and for this reason it failed to determine whether or not 1080 is an endocrine disrupter. This points to a significant toxicological flaw in the rationale for establishing the MAV for 1080 in New Zealand, primarily because the teratological study it is based on was more of an introductory than a conclusive study.

There are a number of studies that do link 1080 with possible endocrine disruption. In a study on the sub-lethal effects of 1080 on rats, Mazzanti (1965) found that 1080 gave rise to lesions on the testes consisting of regressive modifications of the seminiferous tubules, causing damage to spermatogonia. Atzert (1971) also observed 1080 induced damage to seminiferous tubules in rat testes. Sullivan et al (1979) found that testicular weight decreased in rats receiving 20 or 6 ppm of fluoroacetate, and found morphological damage to the testes of all rats treated in their experiment. At higher concentrations (e.g. 20 and 6 ppm) damage progressed to marked seminiferous atrophy. Balcomb et al (1983) studied the acute and sub-lethal effects of 1080 on starlings (*Sturnus vulgaris*) with a particular emphasis on testicular morphology. They observed a 14% reduction in testicular weight development in starlings fed 1080 compared with control birds.[10] Hornshaw et al (1986) observed impaired reproduction in mink and ferrets. Twigg et al (1988) administered 1080 to skinks (*Tiliqua rigosa*) tolerant to naturally occurring fluoroacetate and observed a reduction in plasma testosterone concentration, which may affect spermatogenesis. In a study by the US EPA, Sprague-Dawley rats where administered with sub-lethal doses of 1080 (0.05, 0.20 and 0.50 mg/kg/day) for 13 wk. Findings included increased heart weight, decreased testes weight and accompanying microscopic lesions of the testes, and central nervous system disruptions (EPA 1988). Twigg (1994) asserts that fluoroacetate is known to cause a reduction in animal fertility and points out that the sub-lethal, chronic toxicity of this poison needs to be taken seriously. According to the Office of Environmental Health Hazard Assessment, of the State of California Environmental Protection Agency sodium fluoroacetate (1080) is a male reproductive toxin (EPA 2003).

Persistence and degradation

Toxic substances vary in their rates of degradation depending on their chemical structure and the availability of conditions that affect degradation rates. In the case of compounds that are biodegradable (e.g. through microbial activity), persistence may be restricted in time, but this can vary in relation to other environmental factors such as temperature. Microbial activity decreases as temperature decreases for example, which means that biodegradable substances may persist for longer periods in the winter and/or cold climates compared with the summer, and/or warmer climates.

Studies on 1080 toxicology have shown that this compound breaks down metabolically within animals that have been exposed to the toxin, involving defluorination of fluoroacetate and fluorocitrate (Schaefer and Machleidt 1971; Smith et al 1977; Twigg 1994). Defluorination (detoxification) is also known to occur in plants and bacteria (Twigg 1994). For example, in soils where the toxin does not

[9] Fetal skeletal abnormalities were observed at doses of 0.33 and 0.75 mg kg^{-1} day^{-1}. Abnormalities in forelimb development were observed in fetuses at the same dose. Bent ribs were observed at doses of 0.33 and 0.75 mg kg^{-1} day^{-1}, and unossified sternebrae were observed at the 0.75 mg kg^{-1} day^{-1} dose.
[10] They pointed out, however, that these results were not statistically significant.

occur naturally, breakdown is facilitated by several species of bacteria, fungi, and algae (Twigg op cit.). In mammals and birds defluorination normally occurs in the liver, although when concentrations of the toxin are too high for detoxification, poisoning occurs (Twigg op. cit.).

Numerous studies have also been undertaken to monitor 1080 concentrations in stream water following 1080 control operations (e.g. Eason et al 1992; Parfitt et al 1994; Hamilton and Eason 1994; Meenken and Eason 1995), in bait dust (Wright et al 2002), in soil (Walker 1994; Parfitt et al 1995), in a landfill (Bowman 1999), and uptake in plants (Olgilvie et al 1998). 1080 has shown to be highly soluble in water and is likely to leach from baits into the environment in the presence of rainfall. The high solubility will also facilitate rapid dilution of the leached toxin. Of concern from an environmental toxicology perspective is the length of time 1080 takes to degrade in streams, surface waters and soils, and the concentrations that may persist, perhaps for a limited period, in these environments. The rates of bio-degradation of 1080 in water and soil have also been subject to laboratory studies (Olgilvie et al 1996; Booth et al 1999).

Ogilvie et al (1996) examined the rates of 1080 degradation at different water temperatures, involving the inoculation of stream water with an initial dose of 0.12 μg ml^{-1} of 1080. Experiments investigated degradation rates at 21^0C and 11^0C and were designed to test the different rates likely to occur in different seasons. The research showed that the overall rate of degradation was significantly different at different water temperatures. Concentrations of 1080 declined by 25% at both temperatures during the first 24 hours. Significant differences in degradation rates became evident between 24 and 48 hours depending on the water temperature. The rate of degradation after the first 24 hours at 21^0C was significantly higher than at 11^0C. These different rates of degradation were also demonstrated over longer time periods (between 48 and 72 hours). After 141 hours no detectable 1080 was found in the warmer water, whereas approximately 30% of the initial dose of 1080 remained in the cooler water although the degradation trend continued. 1080 dissolved in deionised water was also tested for breakdown at both temperatures. The results showed that there was little or no breakdown at both temperatures in the absence of microbes. Booth et al (1999) tested 1080 degradation in stream water at 21^0C and showed 1080 to break down into fluorocitrate, as happens in the body, which is likely to be the result of stream microbial activity. Within 17 days of dosing with 1080 there was little or no 1080 or fluorocitrate remaining in the water at that temperature. Eason et al (1999) stated that the breakdown of this toxin has been found to occur more rapidly at higher temperatures, "but still occurs at $<7^0$C within 1-2 weeks." The paper cited in substantiation of this statement is Ogilvie et al (1996). Nowhere in the Ogilvie paper is there any mention of any experiment that tested the degradation of 1080 at or near 7^0C. If experimental evidence does exist that shows rapid breakdown at or below 7^0C this should be published as it would greatly assist this debate.

In New Zealand there are very few forested mountain rivers (i.e. where 1080 control operations occur) with water temperatures as high as 21^0C. This is particularly true for the winter months when poisoning operations are most commonly undertaken. Water temperatures in mountain streams are generally considerably colder and these temperatures will decrease with increasing altitude and latitude.[11] In previously mentioned research, possum carcasses still posed a serious hazard to dogs 75 days after poisoning (Meenken and Booth 1997), which suggests that 1080 or its toxic breakdown product (fluorocitrate) can persist at hazardous levels for lengthy periods in the environment. The evidence provided above suggests that 1080 may be moderately persistent at colder temperatures.

There are a variety of potential hazards associated with any partial persistence of 1080 including endocrine disruption, which can happen at very low concentrations, acute and chronic hazards to dogs, invertebrates, vertebrate wildlife, fish and other aquatic wildlife, aquatic and terrestrial food webs, and human drinking water supplies - particularly subterranean water flows. Further research is needed to test degradation rates at lower temperatures in water and other substrates and for longer time periods before comprehensive conclusions can be made on the issue of persistence.

As with most studies on the direct toxicity of 1080, secondary poisoning studies have limited their focus to acute severe toxicity and none have been found in peer-reviewed literature that looked at teratogenic effects. If 1080 were capable of causing *direct* adverse health effects at levels considerably lower than those that would cause acute symptoms (e.g. if it is an endocrine disrupter), then it is also possible that it could cause *indirect* adverse health effects at low levels through secondary poisoning. This could potentially occur through the consumption of meat contaminated by 1080. Of particular

[11] Autumn stream temperatures at mid altitudes (i.e. 600m) at Nelson Lakes in the South Island (during April, and before winter snows) were 8^0C (personal observation).

concern here is game meat, such as venison, which may be killed following exposure to 1080 by the animal. Given that 1080 has shown to break down slowly in cold conditions, it is not out of the question that deer could move into and out of areas targeted for possum control operations, be hunted and then eaten by hunters or other consumers of game meat (e.g. tourist facilities) with significant doses of 1080 in the meat (i.e. not high enough to cause severe acute reactions, but enough to contribute to teratogenic effects).[12]

Policy implications

Until the issue of possible endocrine disruption and partial persistence at cold temperatures is properly resolved, a number of public health and environmental questions will hang over 1080 use in New Zealand. It is important that proper experiments are conducted to determine if 1080 is or is not an endocrine disrupter once and for all. Until this evidence is available, the precautionary principle may give the anti-1080 lobby in New Zealand sufficient political grounds to convince lawmakers and local authorities that 1080 should not be used to the same extent in large-scale possum control operations. If 1080 is not in reality an endocrine disrupter, such political pressure could lead to the demise of a highly successful agent of possum control for the Department of Conservation and the Animal Health Board and could seriously jeopardize New Zealand's ability to protect its unique biological diversity and control bovine tuberculosis.

For science to be used with integrity in the regulatory process, it is important that regulators at least take full account of scientific consensus - otherwise we have wasted our research investment and science fails to serve society. Where consensus is not forthcoming, it is important to prudently manage uncertainty, particularly where public health or environmental risks may real, but are yet to be confirmed by conclusive scientific research.[13] Section 7 of the HSNO Act (1996) states that "all persons exercising functions, powers and duties under this Act… shall take into account the need for caution in managing adverse effects, where there is scientific and technical uncertainty about those effects." Whether regulators choose to err on the side of caution or on the side of negligence is for them and their political managers to decide. History has taught us though, that the negligent option can lead to adverse and sometimes irreversible environmental and public health impacts. Such impacts can also translate into significant financial costs. The current annual costs of possum control and possum damage[14] in New Zealand are a consequence of a lack of caution in the late 19th century, which saw the introduction of possums for the fur trade.

Conclusion and recommendations

In terms of the relationship between scientific evidence and policy mentioned at the beginning of this paper it is clear that for acute toxicity there is ample empirical evidence of adverse health effects with a high degree of scientific consensus, credible explanations of a biological mechanism to explain these effects, and regulations that reflect this consensus. For chronic toxicity, such as potential endocrine disruption, there is some empirical evidence of adverse health effects with animal models without scientific consensus. As yet, no comprehensive explanations have led to scientific consensus in Australasia on this matter although sufficient consensus exists in California for the US EPA to classify 1080 as a male reproductive toxin.

To enable the regulatory framework to be fully informed by science on this matter there is a need to fill the gaps in our current knowledge where possible and take appropriate regulatory action during an interim period in order to appropriately deal with the uncertainty associated with chronic toxicity. In terms of research there is a need to:

- Conduct experiments to determine whether 1080 is an endocrine disrupter, and determine the endocrine disrupting effects (if any) on a variety of aquatic and terrestrial organisms.
- Conduct experiments to determine the rates of 1080 degradation at temperatures equivalent to those experienced in the winter months in forested mountain areas in New Zealand.

[12] A mildly poisoned (i.e. sick) deer would move more slowly than a healthy one and could conceivably be an easier target for a hunter. Once killed, the rate of 1080 breakdown in tissues is likely to slow down considerably which may allow 1080 levels, high enough to cause endocrine disruption, to remain in the meat.

[13] For example, it may be raining but it remains an anecdote until we set up a rain gauge to record this fact.

[14] According to Eason et al (1994) possums cause $35 million in damage each year.

Until the above research has been completed there is a need to re-evaluate the regulatory status of 1080 in the light of these gaps in our knowledge. In particular, there is a need to:

- Evaluate the risk of 1080 use during the regulatory period prior to the completion of such research.
- Set interim regulations that reflect this uncertainty and its associated risks.
- Explore the practical and financial feasibility of alternative methodologies for possum control,[15] including possible use of 1080 in combination with other methods currently in use (e.g. trapping, other poisons, bounty schemes). As a precautionary measure, prior to the completion of research mentioned in points 1 and 2 above, it would be appropriate to explore the feasibility of using methods other than 1080 in human drinking water catchments and perhaps restricting 1080 use to areas at some distance from human habitation. Because of the risks associated with the build-up of resistance to 1080 among target populations, as has been reported with rabbits in Australia, it would be prudent to regulate 1080 use to minimize this possibility. As such, increasing the use of other methods for possum control would serve this end as well as a human health precaution.[16]

Once the above research has been completed it will be necessary to re-evaluate the calculation of the MAV for 1080 on the basis of the findings of this research and all of the science readily available on acute and chronic toxicity internationally.

Further research should also be conducted as part of the on-going relationship between research, regulation and management for 1080. In particular, supplementary research warrants:

- Evaluation of the effects of on-going 1080 use on the broader ecological functionality of habitats where it is used, including:
    - Potential impacts on food webs.
    - Chronic wildlife toxicity, with particular reference to long-term fertility and fecundity studies of native wildlife populations at concentrations below the known or estimated LD_{50} for these species.
    - Chronic human toxicity, with particular reference to potential adverse health effects other than endocrine disruption at concentrations at and below the current MAV.

It is recommended that the Environmental Risk Management Authority take account of all currently available science on 1080 when it forms recommendations on regulatory issues relating to this chemical. Taking account of available scientific evidence relating to 1080 use will also mean considering the risks of not continuing with the current regime for the use of 1080. Such risks include:

- The potential loss of gains made in recent years as a result of 1080 use in conservation management and bovine tuberculosis control (should 1080 use be more heavily restricted as a result of the ERMA review).
- The risks associated with the employment of any alternative methods of possum control (e.g. the risks associated with the use of other poisons).

Alternatives to current practices will always have their impacts and it is important that the impacts of such alternatives are weighed up against the risks and benefits of the status quo. This can sometimes mean that regulatory change is necessary but costly or that the status quo provides the least of a selection of evils.

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[15] Innes and Barker (1999) recommend that more research be conducted on toxin-free pest control methods in New Zealand.

[16] Research into the potential for immuno-contraception techniques to be used for possum control are well advanced and need to be given full consideration as part of this process of regulatory review for 1080 (Duckworth 2001; Harris et al 2001).

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