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CONTAMINATION OF FOREST ECOSYSTEMS BY SODIUM FLUOROACETATE (COMPOUND 1080)*

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ABSTRACT: Predictive and conceptual models are used to examine the contamination, toxicology, and residues of sodium fIuoroacetate (Compound 1080) in relation to its application in vertebrate pest control programmes on forest and pastoral lands.

As a pesticide, the toxin appears to be neither mobile nor persistent. Exceedingly slender opportunities exist therefore for significant contamination of susceptible components of the environment.

INTRODUCTION

The unintentional migration of pesticides into various components of the environment has generated considerable public apprehension. Certain pesticides applied to pastoral or forest lands do not remain on site. Instead, they are transported into the soil and, via eroded soil particles, into ground and surface waters. As a result of their persistence the use of some long-lived pesticides, such as organochlorines, has been restricted. Other pesticides that are shorter lived or are applied at lower rates can be hazardous in the period immediately following their application.

In common with other toxic pesticides, there are several reasons for making the behaviour of sodium fluoroacetate (Compound 1080 or SFA) in the environment the subject of detailed investigations. One reason concerns the effectiveness of its application to diminish vertebrate pest populations. The central question here is how the desired control effect can be achieved with the lowest possible dosage. Another reason is that the use of SFA, or its derivatives, can have undesirable side effects, such as secondary hazards to man and protected species, both during and after the intended control period. Once the toxin has been distributed, control over its effects on other environmental components has essentially been lost. Also, it must be recognized that the ultimate fate of the toxin lies in the soil. Its effects on soil microbe populations, soil absorption, structure and transport deserve consideration since these interactions contribute to possible contamination of downstream waters.

In investigations on chemical control the demand for quantitative data has acted as an incentive to the development and use of computation models. As such models will play an increasing part in the collection and predictive utilization of information on toxin behaviour it is important to realize what purpose a given model is supposed to serve. Reddingius (1971) distinguished these categories of models:

1. Models may be used to illustrate or exemplify a provisional theory, or to see whether a certain theoretical idea makes sense.
2. Models may be used as counter examples to show that a certain theory or line of reasoning is incorrect or incomplete.
3. Models may be used to summarize our knowledge and insights.

A collection of basic computation models is presented here to define the fate of SFA in the forest ecosystem from a theoretical and practical viewpoint. The models are constructed by mathematical logic of the simplest kind. More general discussions on the control of vertebrate pest populations by chemicals have been presented elsewhere (Peters, 1973; Peters, 1974).

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DENSITY OF DISTRIBUTION OF TOXIC CARROT BAITS BY AERIAL APPLICATION

Model I describes the theoretical expectations from which field evaluations can be derived. The carrot bait distribution and the load of SFA are those used by the New- Zealand Forest Service during aerial control programmes against the brush-tailed opossum (Trichosuru vulpecula) in high-rainfall indigenous forest areas. Sparser distributions and lower toxic loads of carrot baits are used in regions accessible to the public or where there is danger of exposure of farm animals. For instance, Agricultural Pest Destruction Boards in their operations against the rabbit on pastoral lands use a bait density and toxic load per unit area about 10 times smaller than that described in Model I.

Model I. Density of Distribution of Toxic Carrot Baits by Aerial Application.

Proposition: Define an expected estimate of bait density and its toxic load per unit area.

Assume:
1. Carrot bait distribution is 34 kg/ha (30 lb/acre).
2. Toxic load is 1 kg toxin/tonne carrot (2 lb/ton).
3. Average weight of carrot bait is 4 g.

Then:
1. Carrot bait distribution is 3.4 g/m^2.
2. One tonne of carrots covers $\frac{1000}{34} = 29$ ha.
3. Toxic load is $\frac{1000}{29} = 34$ g/ha = 3.4 mg/m^2.

Therefore:
1. Carrot bait distribution is about one bait/m^2.
2. Toxic load is 3.4 mg toxin/bait/m^2.

Considerable deviation from the Model has been found under field conditions. Godfrey (1973) in particular has directed attention to several factors that influence bait distribution. Of these, the wide diversity of bait sizes obtained from conventional carrot-cutting machines (Bell, unpubl. data) has contributed not only to uneven dispersion of baits, but also to considerable variation of toxic loads (Staples, 1969; Peters and Baxter, unpubl. data). Although Godfrey (1973) indicated the direction of improvements, few of these can be resolved easily with root crop baits under large-scale operations in the field.

TOXICITY OF CARROT BAITS AND LETHAL DOSAGE RATES

The relationships between acute lethal toxicities and consumption of toxic carrot bait materials is examined in Model II. Whilst acute toxicity (i.e., the single dosage required to produce death) is a conveniently measured effect in the laboratory, in the field sub-lethal amounts of the toxin may have subtle (e.g., bait-shy or poison-wise) effects on an animal population. Also, acute toxicity is not always the most sensitive measure of potential hazard. An animal with a high susceptibility to the toxin may have an aversion to the bait material. In such a situation pharmacological susceptibility bears little relationship to ecological vulnerability.

The usual way of expressing acute toxicity is by means of an LD_{50}, value (Hayes, 1963). The LD_{50} (Median Lethal Dose) is a statistical estimate of the dosage that would be lethal to 50% of a very large population of the test species. The lethal dose values (LD_{100}) presented in Model II are conservative estimates determined (Thompson and Weil, 1952; Weil, 1952) from the LD_{50} of the various species of animals (Tucker and Haegele, 1971; Atzert, 1971; Bell, 1972; Mclntosh, Bell, Poole and Staples, 1966; Robinson, 1970; Annison, Hill, Lindsay and Peters, 1960).

When the lethal dose values are used to evaluate the effectiveness, safety or hazard of the toxin in the field, several other factors must be taken into account:

1. The timing and the amount of toxin applied per unit area. Field operations against the opossum in indigenous and exotic forest areas are usually carried out during winter when natural food is scarce. Under these conditions the toxic load used per unit carrot bait should provide one lethal dose for an opossum.

2. The degree of contamination of various environmental components, such as water and vegetation. On the basis of appropriate methodology (Peters and Baxter, 1974) aspects of accumulation, persistence and translocation of the toxin will be examined in subsequent models.

3. The extent of exposure of non-target species. Seed-eating bird species can be expected to eat carrot "chaff", which are small fragments of carrot produced during the cutting process. To minimize this hazard the inclusion of a deterrent dye "Lisamine Green" is a mandatory addition to bait materials. Whilst yellow or green are the predominant deterring colours, Kalmbach (1943) concluded that the size and shape of the bait are equally important.

4. Secondary exposure hazards can occur when poisoned carcasses are consumed. Model II allows the prediction of the extent and severity of such hazards. For instance, a hawk would receive a lethal dose from the consumption of the gut of an opossum containing seven lethal opossum doses. Alternatively, any opossum carcass can be considered lethal to a dog.

Model II. Toxicity of Carrot Baits and Lethal Dosage Rates.

Proposition: Provide an estimate of species specificity and its relationship to bait consumption.

Assume: 1. 1 kg toxin/tonne carrot = 1 mg toxin/g carrot.
2. Average weight of carrot bait is 4 g.

Then: This bait contains 1 x 4 = 4 mg toxin.

Animal	Lethal Dose (mg/kg)	Average live weight (kg)	Toxin required for lethal dose (mg)	Number of carrot baits required for lethal amount
Dog	0.1	25	2.5	0.6
Sheep	0.5	50	25	6
Cattle	0.5	500	250	62
Deer	0.5	70	35	8
Goat	0.5	50	25	6
Rabbit	0.8	1.5	1.2	0.3
Pig	1	50	50	12
Opossum	1.2	3	3.6	1
Man	2	70	140	35
Magpie	1	0.3	0.3	0.1
Blackbird	3	0.2	0.6	0.2
Sparrow	3	0.1	0.3	0.1
Turkey	5	5	25	6
Hen	8	1	8	2
Pheasant	8	2.5	20	5
Weka	8	1	8	2
Hawk	12	2.5	30	7
Quail	15	0.2	3	1

CATCHMENT WATER POLLUTION

Because of the proximity of pastoral and urban occupation to forest regions that are subjected to widespread applications of SFA, there is concern that farm and domestic water supplies can be contaminated. Model III examines the extent of such hazards. In addition, the behaviour of the toxin in relation to soil and animal imposes at least three questions that need to be resolved.

1. Chemical adsorption and bioactivity

The strength of adsorption of pesticides by soil is governed by the chemical structure of the pesticide, the types of soil colloids, the pH or hydrogen ion content of the soil, the kind of saturating cations, the moisture content of the soil, and the time that the pesticide has remained in contact with the soil. As the adsorption of pesticides by soil increases, the bioactivity decreases. Amorphous clay minerals and organic matter in particular can trap, inactivate and protect pesticides in soils (for a detailed review see Adams, 1973).

In common with the phenoxyacetic acid series of herbicides (of which trichlorophenoxy acetate [2,4,5-T] is a member), sodium fluoroacetate exists as the dissociated anion in most soils. Adsorption-desorption phenomena are therefore governed particularly by the anion-exchange capacity of the soil. Cation exchange, if it does occur, would not contribute to the detoxication of the SFA molecule since Na^+ is not the toxic part of the molecule. To retain its toxicity the $CH_2.F.COO-$ group of the molecule must be preserved intact. It has been our experience that the ion-exchange capacity of the forest soils investigated greatly exceeds the amounts of SFA adsorbed during conventional vertebrate pest control programmes. Thus, there is little or no tendency for the toxin to move in the soil even if it were assumed that the integrity of the $CH_2.F.COO-$ bond remains intact. The predicted lethal amounts of catchment waters in Model III are consequently absolute minimum values.

Arising from the above considerations the exposure of aquatic organisms, in particular fish, to SFA, must be examined. Cold-blooded vertebrates, as compared to warm-blooded species, are extremely tolerant to the toxic action of SFA or its derivatives. For instance, fingerling bream and bass can survive, for an indefinite period and with no apparent discomfort, concentrations of SFA as great as 370 mg/1 (King and Penfound, 1946). This amount of toxin would be contained in about 90 carrot baits/1 water (Model I), or about 2500 times the predicted catchment water contamination (Model III). Thus, although the lethal affects of SFA on aquatic life are accentuated by increased water temperatures, in the context of vertebrate pest control programmes the actual exposure hazards become significant.

Model III. Catchment Water Pollution.

Proposition: Examine the extent of surface water contamination and its associated intoxication hazards to animals.

Assume:
1. 34 kg carrot/ha at 1 kg toxin/tonne carrot.
2. A stream draining a 1 ha catchment.
3. Rainfall 25 mm (1").
4. The entire toxic load is translocated (leached) from the carrot baits directly into the stream.

Then:
1. 25 mm rain/ha = 251311 l.
2. 1 kg toxin/tonne = 1 mg toxin/g carrot.
3. 34 kg carrot = 34000 g carrot.

Therefore:
1. Toxic load per ha is 34 g toxin.
2. If this entire amount of toxin leached into the stream by 25 mm rain, then the runoff would contain 34000/251311 = 0.14 mg toxin/l.
3. An animal drinking this water would require, <u>in one single drinking session</u>, these amounts in order to receive a lethal dose.

Rabbit	1.2/0.14 = 8 litre
Dog	2.5/0.14 = 17 litre
Opossum	3.6/0.14 = 24 litre
Sheep	25 /0.14 = 178 litre
Goat	25 /0.14 = 178 litre
Deer	35 /0.14 = 250 litre
Pig	50 /0.14 = 357 litre
Man	140 /0.14 = 1000 litre
Cattle	250 /0.14 = 1785 litre

Assume:
1. A stagnant pool of 10 x 10 x 0.1 m.
2. In this pool are 20 dead opossums, each containing 10 lethal doses.
3. The toxin is completely leached into the water without further bio-degradation.

Then:
1. This pool contains 10 x 10 x 0.1 = 10 m^3 = 10000 litre water.
2. Toxic load is $\frac{20 \times 10 \times 36}{10000}$ = 0.072 mg toxin/litre water.

Therefore: An animal drinking this water would require, <u>in one single drinking session</u>, these amounts in order to receive a lethal dose.

Rabbit	1.2/0.072 =	17 litre
Dog	2.5/0.072 =	35 litre
Opossum	3.6/0.072 =	50 litre
Sheep	25 /0.072 =	347 litre
Goat	25 /0.072 =	347 litre
Deer	50 /O.072 =	486 litre
Pig	50 /O.072 =	694 litre
Man	140 /0.072 =	1944 litre
Cattle	250 /0.072 =	3472 litre

2. Biomagnification and biological detoxication

Irrespective of whether SFA remains intact at the site of application or is translocated, its ultimate fate lies in the soil. The question can be posed whether by progressive applications, lethal accumulations of the toxin can occur in the soil.

In common with a majority of pesticides (Guyer, 1970) there is abundant evidence that SFA can be degraded into non-toxic components. The carbon-fluorine (C-F) bond of the SFA molecule can be ruptured by enzyme systems present in *Pseudomonas* and *Nocardia* species of soil micro-organisms (Goldman, 1965; Goldman and Milne, 1966; Horiuchi, 1962; Tonomura, Futai, Tanabe, and Yamaoka, 1965; Davis and Evans, 1962; Kelly, 1965). Microbiological surveys of several Westland, South Island, regions have isolated many bacterial and several fungal species that actively degrade the C-F bond in SFA on carrot substrates (Peters and Mulcock, unpubl. data).

The uptake of SFA by root systems of plants has been examined in the context of its phytotoxicity and its natural occurrence in several plant genera, e.g., *Acacia*, *Gastrolobium* and *Oxylobium* (references see Peters, 1972; Preuss and Weinstein, 1969; Hall, 1974). Stock losses have been reported in Australia where animals have access to these endemic poisonous plants (Aplin, 1969). No detailed knowledge of this phenomenon exists in the New Zealand situation, although *Oxylobium callistachys* has been declared a noxious weed by many South Island and several North Island Counties (Fitzharris, 1973).

SFA is not particularly toxic to plants, but inorganic fluorides are considerably more toxic.

Thus, sufficient circumstantial evidence exists that the predicted values enumerated in Model III underestimate actual values.

3. Mammalian metabolic responses

SFA acts predominantly on the tricarboxylic acid cycle; a fundamental source of energy conversion from foodstuffs at cell, tissue, and organ levels in living systems (Peters, 1963; Pattison, 1959; Pattison and Peters, 1966; Peters, 1972; Peters, 1973). Unlike those pesticides that possess specific affinities for accumulation in vertebrate and invertebrate tissues, SFA (and its toxic cellular metabolite fluorocitrate) has no cumulative effects in viable organs and tissues. Also, sub-lethal doses of SFA have been given to rats in their drinking water for several months without harm (Peters, 1972; Howard, Marsh and Palmateer, 1973).

Thus, the metabolic fate of the toxin must also be considered in the interpretation of the predicted values of Model III.

TOXIC RESIDUES IN MEAT

Model IV has taken intoxicated venison as the type example, and the species are regarded as flesh-eating scavengers. The secondary poisoning hazards require cautious interpretation because consideration must be given to the following factors:

1. On weight/weight basis, the toxin is confined predominantly to internal organs and viscera. Also, tissue levels are determined by the amount of toxin and the duration of its distribution within the viable organs, which in turn is related to species susceptibility. Gal, Drewes and Taylor (1961) injected radioactive C^{14}-SFA intraperitoneally into rats and examined the distribution of radioactivity at death. The level of radioactive isotope (C^{14}-SFA/gram wet tissue) decreased in the order of brain, liver, heart, kidney, intestines

and stomach, lungs, spleen, testes, carcass muscle, expired CO^2 , urine. It can be expected that the oral consumption of toxic bait materials would make the stomach of the poisoned carcass the most likely cause of secondary poisoning.

2. As with earlier investigations of toxic carcass meat (Mclntosh and Staples, 1959), we have not found compositions equal to, or in excess of those predicted in the Model. If however, despite the above, an equal distribution of toxin throughout the carcass is assumed the following types of computations can be derived from the Model.

(1) Liver and kidneys comprise about 2% of the dressed deer carcass (Coop and Lamming, 1974). Assuming that 15% of the toxic load is contained in liver and kidneys, these organs (1.3 kg) will contain 16 mg toxin (12 mg/kg tissue). This amount would provide about five lethal doses for a dog. For a man to receive one lethal dose it would be necessary that he eat these organs from about eleven deer.

(2) If about 60% of the toxic load is contained in the dressed carcass, this venison (41 kg) will contain 62 mg toxin (1.5 mg/kg meat).

3. Unlike scavengers in the wild, man prefers to cook his meat. The quantities of toxic meat in Model IV imply that the meat is consumed in its raw state.

The structural integrity of the SFA molecule becomes unstable at temperatures about 130°C, and decomposition takes place at 200°C (Sunshine, 1969). We have been unable to detect organof1uorine residues in oven-baked meat inoculated with physiological amounts of SFA prior to baking. Similarly, boiled inoculated meat contained no organof1uorine residues, but the water contained detectable traces. Grilling could conceivably allow toxic residues to be retained in the interior of the meat. Nonetheless, according to Model IV, gargantuan appetites are required.

Model IV. Toxic Residues in Meat (Venison).

Proposition: Define the possibility of acquiring lethal amounts of toxin from consumption of toxic meat.

Assume:
1. A deer killed by three lethal doses, obtained from eating about 24 toxic carrot baits.
2. The toxin is distributed evenly throughout body.
3. Total live weight is 70 kg.
4. Lethal dose is 0.5 mg/kg.

Then: Total toxic load is 70 x 0.5 x 3 = 105 mg toxin = 105/70 = 1.5 mg toxin/kg.

Therefore: An animal feeding from this carcass would require, in one single feeding, these amounts in order to receive a lethal dose.

Rabbit	1.5/1.5 = 1	kg
Dog	2.5/1.5 = 1.7	kg
Opossum	3.6/1.5 = 2.4	kg
Sheep	25 /1.5 = 16.7	kg
Pig	50 /1.5 = 33.3	kg
Man	140 /1.5 = 93.3	kg
Cattle	250 /1.5 = 167	kg

FOREST CONTAMINATION BY TOXIC RESIDUES

A common feature of pesticide applications is the overwhelming excess of material that is required to reach and control the target organism. Despite such over-abundance of available toxin, complete control is seldom achieved and repeated applications are necessary.

In common with other pesticides (Durham, 1967) the manner in which SFA becomes available to, and is assimilated by, the target species is related not only to its mammalian toxicity, but also to interactions with climatic conditions (temperature, light, rain), with formulation of bait materials, with physiological states (age, sex, nutritional status and disease), and with other chemicals interacting with the toxin itself (detoxicat ion, antagonists and antidotes). Several aspects of these interactions have been examined (Staples, 1968; Staples, 1969; Peters and Baxter, 1974; Peters, 1974; Corr and Martire, 1971).

Irrespective of the precise role of the toxin-host interactions that contribute to the success or failure of complete control, Model V allows a number of deductions:

1. It indicates that about 99% of the toxic load is not utilized.

2. A lower toxic load per bait implies the supposition that more bait must be found by the target animal.

3. A higher toxic load per bait distributed at a lower density will diminish animal access to the bait. Also, since taste is a concentration-dependent factor, aversion to the taste of SFA by the opossum may be analogous to that of other mammalian pest species (Howard and Marsh; Denver Wildlife Research Centre; pers. comm.).

4. The introduction of other toxins with higher mammalian toxicity at lower concentrations offers few benefits (with more difficulties) to the pest control operator (Clark, 1970; Oser, 1971; Worden, 1974). Whereas such investigations are in progress it must also be noted that many toxins fall short of operational and legislative expectations.

5. The search for bait materials and methods of bait application whereby animal-bait affinities can be improved requires not only a knowledge of bait formulation technology and environmental consequences of toxin dispersion, but also an understanding of the behaviour characteristics of the target and non-target animals in their preferred habitat.

Model V. Forest Contamination by Toxic Residues.

Proposition: Consider the degree of efficiency that can be expected from conventional aerial control programmes in terms of consumption of toxin by an animal population.

Assume:
1. 34 kg carrot/ha at 1 kg toxin/tonne carrot.
2. Lethal dose of toxin for opossum (3 kg) is 1.2 x 3= 3.6 mg toxin.
3. Density of opossum is 50 animals/ha -- a very high density.
4. All 50 opossums take <u>two</u> lethal doses each of toxin.

Then: Toxic load per ha is 34000 mg. Toxin requirement by opossums is 50 x 3.6 x 2 = 360 mg.

Therefore: Consumption by opossum population is $\frac{360}{34000} \times 100\% = 1.01\%$ of total load of toxin.

UTILIZATION IN ENVIRONMENTAL FORESTRY

In terms of application of the toxin as a pesticide per unit area, the deductions derived from Model V indicate an abundance of available toxin but the preceding models have found that such amounts are a tolerable contamination.

It is prudent however also to determine the upper levels of abundance that could be tolerated from widespread applications. Model VI indicates that, even at a level of about 160 times the conventional rate of application, the contamination of the various environmental components would still be expected to remain within the limits derived from the preceding models.

CONCLUSION

The models developed here indicate, and field monitoring operations confirm, that the use of SFA as a pesticide (mammalicide):

1. Imposes an exceedingly small toxic burden on the susceptible environment.

2. As a soil resident, SFA appears to be neither mobile, nor persistent.

3. The chances of significantly contaminating rural and urban water supplies are extremely remote.

4. There is no valid evidence to suggest that lethal toxic residues can accumulate in carcass meats intended for human consumption.

Model VI. Utilization in Environmental Forestry.

Proposition: Define an upper limit of abundance of toxin in comparison with conventional aerial control applications.

Assume:
1. 2500 kg of toxin is used annually for the control of noxious forest animals.
2. Area of control is 12 million ha of protection forests.
3. Even distribution of toxin over protection forests area.
4. Rate of distribution over total area equals that applied in Conventional aerial programmes.

Then: 2500 kg in 12 x 10^6 ha = 2.5 g in 12 ha = 0.21 g of toxin per ha. But: Toxic load in conventional aerial programmes is 34 kg carrot/ ha = 34 g toxin/ha.

Therefore: At a similar rate of distribution total toxin requirement for total protection forests area is

$$\frac{34}{0.21} \times 2500 \text{ kg} = 404$$

tonnes of toxin.

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