# LETTERS TO THE EDITOR

## EFFECTS OF 1080 ON NON-TARGET SPECIES

During a study of the carrion-feeding behaviour of the Australasian harrier *(Circus approximans gouldi)* I became interested in the secondary exposure of harriers to pesticides, and came across "Contamination of forest ecosystems by sodium fluoroacetate (Compound 1080)" by J. A. Peters (1975), in volume 22 of the *Proceedings of the New Zealand Ecological Society*.

In Model II (page 36) "Toxicity of carrot baits and lethal dosage rates" I was surprised to see that the author gave the average live weight of a "hawk" as 2.5 kg. The live weights of Australasian harriers measured in New Zealand are less than one-third of this: males 604.9 g (n = 60), females 800.0 g (n = 69) (Carroll, 1970).

On consulting the references given by Peters, I found that Atzert (1971) gave the weight of "hawks", referring to the American rough-legged hawk *(Buteo lagopus sancti-johannis),* ferruginous rough-legged hawk *(Buteo regalis)* and the marsh hawk *(Circus cyaneus hudsonicus)* as 2.51b (1.14 kg). The misreading of pounds as kilograms is perhaps understandable, but should have been detected before publication. Of greater concern, however, is Peters' use of data on North American species and genera that do not occur in New Zealand. Most readers would have assumed, lacking a contrary statement, that the "hawk" in Model II referred to the Australasian harrier. As Tucker and Haegele (1971), one of Peters' references, pointed out, ". . . predicting species LD50 to one of the chemicals on the basis of the LD50 of another species could lead to considerable errors. . . . The pairing of species also failed to show any correlation between phylogenetic relationships and toxilogic susceptibility"; they concluded that "there was generally a large variability between species in their susceptibility to any given compound".

Even if the Australasian harrier had the same lethal dose level (LD100) as the 12 mg/kg for "hawks" quoted by Peters, the recalculated level of toxin required for a lethal dose for a harrier weighing 0.7 kg is 8.4 mg rather than 30 mg, and 2.1 carrot baits are required for the lethal amount, rather than 7. Therefore, an Australasian harrier would receive a lethal dose from eating the gut of an opossum containing approximately two lethal opossum doses (= two carrot baits).

Other data listed in Model II are equally misleading:

1. The "magpie" quoted is the American magpie *(Pica pica hudsonia:* Corvidae) not *Gymnorhina tibicen:* Cractididae which occurs in New Zealand. Atzert (1971) gave the weight of the "magpie" as 0.51b = 227 g or 0.2 kg not 0.3 kg. Following the calculations through, the number of baits should be 0.05 not 0.1.
2. The "blackbird" referred to is Brewer's blackbird *(Euphagus cyanocephalis:* Icteridae) not our familiar *Turdus merula:* Muscicapidae, introduced to New Zealand. The average live weights of 92.0 g (male) and 91.4 g (female) for *Turdus merula* (Gurr, 1954) are about half that quoted for Brewer's blackbird.
3. "Sparrow" refers to the house sparrow *(Passer domesticus),* which occurs in New Zealand, but the average live weight is 27.9 g for males (n = 120) and 27.5 g for females (n = 120) (P. C. Bull, pers. comm.) rather than the 0.1 kg (= 100 g) used by Peters. When these weights are substituted into the model, the toxin required for a lethal dose becomes 0.08 mg instead of 0.30 mg, and the number of 4 g carrot baits required for a lethal amount is 0.02 baits instead of 0.1 baits.
4. The "pheasant" quoted is the ring-necked pheasant *(Phasianus colchicus),* which is the species found in New Zealand. However, the weight of an adult male is 1.34 kg (n = 15) and an adult female 1.18 kg (n = 10) (Westerskov, 1956), not 2.5 kg as quoted by Peters. Therefore the toxin required for a lethal dose and the number of carrot baits required for a lethal amount must be halved.
5. The "quail" refers to the Japanese or coturnix quail *(Coturnix coturnix japonica)* a genus now absent from - New Zealand. The only quail belonging to the genus *Coturnix* in New Zealand was the New Zealand quail *(C. novaezealandiae),* which became extinct about 1870 (Kinsky, 1970).

The absence of scientific names, especially in view of the fact that all the vernacular names used in Model II are the same as those in common usage for animals occurring in New Zealand, is unscientific and very misleading. By contrast, the approach adopted on page 23 of Rammel and Fleming (1978) is both scientific and clear.

Of equal concern is Peters' use of the lethal dose values or LD 100 (an "estimate". of the dosage

* Because of the asymptotic nature of lethal dose curves, an LD100 value can never be determined.

level that would be lethal to *all* individuals in a population) rather than the median dose level or LD50 (the dosage that would be lethal to 50% of a large population); as Peters says, "The usual way of expressing acute toxicity is by means of an LD50 value (Hayes, 1963)". I can understand the need to "know" LD 100 levels for target species, but for non-target species the LD50 or even LD5 levels are more appropriate. Peters says (p. 35) that "The lethal dose values (LD 100) presented in Model II are conservative estimates determined (Thompson and Weil, 1952; Weil, 1952) from the LD50 of various species of animals. . ." However, on examining these two references it is apparent that the method of Thompson and Weil (1952) (upon which the tables of Weil (1952) are based) is a moving average method for determining LD50 values-it does not attempt to progress from LD50 to LD 100. How the lethal dose (LD 100) values shown in Model II were derived is not clear, as some are the same as LD50 values in the references, some lower and some higher. The use of LD 100 levels rather than LD50 levels would have the effect of raising the number of carrot baits required for a lethal amount and hence make the dose rates of 1080 for non-target species look much safer than they really are.

These errors do not help to allay the public apprehension, that Peters refers to, about the effects of 1080 on non-target species in New Zealand.

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## EFFECTS OF 1080 ON NON-TARGET SPECIES: A REPLY

There is a truism that states that the half-life value of a scientific finding equals the time for its publication. I presume this to mean that the results are out of date by the time they appear in print. Certainly, in a toxicological discipline as dynamic as vertebrate pest populations themselves, contradictions and redundancies occur with almost seasonal regularity. Be that as it may in retrospect, I am rather delighted that an early piece of work of mine gets taken down for a dusting.

In those early days I had the overwhelming conclusion that we, both here and overseas, really knew very little about Compound 1080. This ignorance hampered rational discussion of the options, and I set about to rectify this, initially by constructing models. I went to considerable lengths to define what purposes the models were to serve. Their structures were built around existing knowledge at that time. That I cautioned in several ways for leniency in the interpretation of the models in no way invalidates the structure of the models. Inadequacies lie in the raw data.

Before formal publication, the models were circulated rather widely for comment, both here and overseas. In response to it and an earlier acquaintance (Peters, 1972), a *verbatum* re-publication was requested to the Editor of this journal by the California Vertebrate Pest Committee (Peters, 1976).

Robertson criticises the absence of avian scientific nomenclature, but I find it curious that he doesn't chastise me on the mammalian types as well. As for their identities, their sources were copiously documented in the reference section for all, including Robertson, to take note of. Despite wide-ranging responses to the various models I

have received no indication of confusion over nomenclature.

I share Robertson's distress about lethal dose values (as does the majority of the toxicological fraternity when LD values are at issue), but not quite for the reasons he may think I should. An LD value, of whatever magnitude, tends to imbue the determination with a degree of certainty that is, a best, suspect, and, at its worst, meaningless. Every single lethal dose value, listed from its published source, is suspect on one or several counts. One example will suffice. The weka *(Gallirallus australis grey* i) LD50 value comes from 14 birds; hardly of robustly statistical confidence. It comes from one sub-species; but what about the other sub-species. It comes from a single diurnal determination; but wekas eat and live at night also. So what is the nocturnal value? Since an organism is a different biological entity at different points in time, toxic susceptibilities change at least in a circadian fashion. Also, an experimental oral dose is something quite different from a dose in a feed. So, one measurement, at one point in time, tells us nothing of maximum, minimum, or even mean values. As for other specifically New Zealand avian species, we draw a useless blank.

Robertson's assertion is that conservative assumptions are made, and that is true. But I am unhappy enough to state I didn't know whether the assumptions I was making were conservative or not because I didn't know what was occurring physically. Also, a moving-average interpolation is no more than a simplified regression analysis adjusted to small sample sizes. As a statistical tool, its flaws are well understood by those who know how to use it. As a biological tool, interpolation is invariably grossly inaccurate. I don't know how to calculate confidence limits around a sloppy or non-existent mean value.

By his charge of dose rates that "look much safer than they really are", Robertson accuses me of something I explicitly did not say. That would indeed be "unscientific", and also rather foolish. His accusation carries an assumption that I reject, and he displays an unkind and mischievous misconception of the purpose the models were to serve. It seems important to stress again that I was constructing models. I was not constructing dose-response curves. The nature of modelling, as stated explicitly, is conception and prediction. In the light of available information, conception is easy, but prediction can be wide off the mark as we know, or suspect we know, it now.

The available information is changing as a result not only of more sophisticated analytical methodology (Okuno and Meeker, 1980) and a better understanding of metabolic fates (Egekeze and Oehme, 1979), but also of insights into toxic susceptibilities (Peters, unpublished) and non-target species responses (Spurr, 1979). As stated explicitly, my models were used not only to show that a certain line of reasoning was incorrect or incomplete, but also, to act as an incentive to get something done on Compound 1080, particularly on its environmental behaviour. That something has been done on this (Bong *et al.,* 1979; Bong, Walker and Peters, 1980; Bong, 1979) and other aspects (see above) is really for Robertson to find out before he makes his charge of "misleading" data. He has a sharp pen but a blunt sense of historical perspective. By analogy, our ancestors "knew" that the earth was flat, but we "know" now it isn't.

Robertson's story is marred by the fact that our understanding of intoxication has improved since the early 1970s. Nonetheless, Robertson's contribution to the Compound 1080 debate is refreshing indeed in the sense that he has done me, and readers of this journal, a service by recalling attention to my early work. With his undoubted expertise in the field of secondary intoxication he brings into the debate aspects that we were aware of in our list of research assignments. I welcome his contributions.

Finally, Robertson refers to allaying public apprehension. Whilst in my long and varied association with Compound 1080 my own apprehension has taken priority, my observations, here and overseas, continue to lead me to the recurring conclusion that, as presently used and legislated, this toxin does not pose a significant threat to most wildlife or beneficial species, or the environment. But it has also been my experience that, when experts differ, the plain man is bound to conclude that they can't all be right, but they may all be wrong. And he is right.

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## EVOLUTION OF DIVARICATING PLANTS IN NEW ZEALAND IN RELATION TO MOA BROWSING

In a stimulating and thoughtful paper, Greenwood and Atkinson (1977) have argued that the peculiarly high incidence of divaricating shrubs in New Zealand is an evolutionary response to continued browsing by moas (Dinornithiformes) over a considerable period of the Tertiary and Pleistocene. These authors establish quite convincingly, from several lines af evidence, an empirical link between a unique floristic feature and a major aspect of the faunal history of New Zealand.

However, the suggested explanations as to why exactly the divaricating habit should be an effective defence against moa browsing seem rather less satisfactory .

Briefly, Greenwood and Atkinson relate this to limitations in the biting mode of the moa, equipped with beak instead of mammalian mouth. The divaricating habit would be less protective against present day ungulates. The authors do not distinguish between the plant materials taken by moa or contemporary browsers.

I suggest that a more direct explanation would lie in the biochemistry of foliage and the digestive capabilities of herbivores.

The net nutritive return from mature leaves of woody plants will be small unless the cellulose itself, a major component of mature leaf tissues, can be digested. No vertebrates produce cellulases directly. Those digesting cellulose do so via symbiotic bacteria in specialised regions of the gut.

Although the size of the largest moas makes it probable some degree of fermentative digestion could occur, most of the species were comparable in size with the larger contemporary ratite birds. These have a mixed diet and are apparently not dependent on a specialised cellulose-digesting ability. Thus it would be remarkable if the moas had an efficient system for utilising mature forest foliage. Certainly it would be less so than that of contemporary ruminants. Thus the moa would probably select material that gave a greater yield of easily available nutrients. With woody plants this could only be (as well as fruiting parts) the shoot tips where higher levels of proteins and soluble carbohydrates are found in developing leaves, apex and outer stem tissues. Moa browsing would therefore be concentrated on the stem tips. The small head, long neck and good vision of the moa would also favour the required behaviour.

Selection pressure would thus favour those plants capable of prompt lateral shoot development when the shoot apex was removed and those capable of profuse branching. In short it would favour the divaricating habit. Cryptic appearance of young shoots (an interesting feature noted by the authors) would also have direct protective value against this peculiar browsing behaviour.

Given that moas would preferentially browse growing tips, dispersal of the plant's extension growth effort into a large number of small and spatially separated units (precisely the divaricating habit) would make browsing less rewarding energetically. It would minimise the effect of that browsing on the plant.

I suggest this basic biochemical assumption can be applied fruitfully to various other aspects of plant-moa coevolution.

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## DIVARICATING PLANTS AND MOA BROWSING: A REPLY

In his interesting letter Dr Lowry has emphasized the importance of considering the nutritional properties of those plants likely to have been eaten by

moas (Dinornithiformes), an aspect which has lately also received attention from Mitchell (1980).

In our 1977 paper we argued that severa1 features of divaricating plants were likely to have made the browsing of these plants by moas more difficult (pp. 24, 25). We did not go as far as suggesting that these features would be an "effective defence against moa browsing". Rather we were reasoning that moa browsing had been a selective force which resulted in the evolutionary appearance of these features. Clearly, to have been selected at all, such features must have had some effect in increasing the reproductive success of divaricating plants, perhaps by increasing survival, but this does not mean that browsing of these plants was either prevented or even greatly reduced. Observations made since 1977 have confirmed that a large number of divaricating species, although browsed to varying degrees, can survive along forest margins or in grasslands where considerable browsing by cattle or sheep on grasses and non-divaricate woody plants is occurring. The reasons for this are not altogether clear and we cannot assume at present that the same would have occurred in the presence of moas.

If, as Dr Lowry suggests, moas browsed preferentially on shoot tips and developing leaves to gain greater nutritive yield, then features such as the reduced leaf size, increased internode length and cryptic coloration of young shoots can be seen more easily as possible adaptations to moa browsing. The right-angled branching can also be seen, as both we and Dr Lowry have suggested, as an adaptation towards spatially separating the growing points and thus minimizing the effect of browsing.

We should, however, be cautious about placing too much emphasis on the young growth. There are substantial periods of the year, particularly in the cooler parts of the country, when no young growth is available. Another point is that the young growth of some plants contain higher levels of toxins than does the mature foliage (McKey, 1974; Rhoades and Cates, 1975), although this has yet to be demonstrated in New Zealand. Furthermore, there are other ways (apart from ruminant digestion) of obtaining an adequate diet from cellulose-rich foods. One is that of the non-ruminant "hind-gut digesters" such as zebras *(Equus* spp.) which overcome the difficulty by eating greater amounts of plant material than ruminants of equivalent weight (Janis, 1976). If moas had evolved a similar digestion mechanism to that of the "hind-gut digesters" we can speculate that the impact on vegetation through browsing by the larger moas would have been considerable.

Some extant New Zealand birds rely rather heavily for their nutrition on cellulose-rich foods at least at certain times of the year. Examples are the pigeon *(Hemiphaga novaeseelandiae),* kokako *(Callaeas cinerea),* kakapo *(Strigops habroptilus),* parakeets *(Cyanoramphus* spp.) and the takahe *(Notornis mantelli).* The pigeon sometimes eats mature leaves including such fibrous leaves as those of silver beech *(Nothofagus menziesii),* hard beech *(N. truncata)* and phylloclades of tanekaha *(Phyllocladus trichomanoides)* (McEwan, 1978). Mature leaves of tawa *(Beilschmiedia tawa),* toro *(Myrsine salicina),* pigeonwood *(Hedycarya arborea)* and raurekau *(Comprosma grandifolia)* are included with young leaves in the diet of the kokako (St. Paul, 1966). Kakapo eat a wide range of plant leaves including many mature leaves (Grey, unpub. 1977) although they discard much of the fibrous material as chewed pellets. Red-crowned parakeets sometimes eat mature leaves of woody plants, e.g., taupata *(Coprosma repens)* (Atkinson, unpub.). Takahe sometimes feed on the basal parts of older leaves of tussock grasses although these parts include both meristematic and mature tissue (Williams *et al.* 1976; Mills and Mark, 1977; Dr J. A. Mills, pers. comm.). An understanding of the mechanisms involved in cellulose digestion in each of these birds might throw some light on how the moas could have obtained an adequate diet when feeding on mature foliage.

Each of the birds discussed has rather distinct modes of feeding as might be expected from the differences in their beaks. This should remind us that the moas were probably not a homogeneous group so far as their feeding behaviour was concerned. There are for example distinct differences between the beaks of *Megalapteryx, Euryapteryx* and *Dinornis* suggesting that some species of moa probably made greater use of certain kinds of plants than others and may have employed differing modes of feeding.

Notwithstanding the above comments, we welcome Dr Lowry's letter as a further contribution to our thinking about moa-plant relationships.

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