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Review of non-target hazards associated with rodenticide use in the USA¹

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Relatively few field studies have been conducted to evaluate hazards to wildlife from rodenticide use. In the USA, field studies have been conducted on both acute and chronic compounds, including zinc phosphide, strychnine, 1080 (sodium monofluoroacetate), and the anticoagulants diphacinone and brodifacoum. Techniques employed in these studies have included carcass counts, direct counts, indirect counts, nest site monitoring, radiotelemetry, habitat and diet evaluation, necropsy, and residue analysis. Although zinc phosphide generally is not secondarily hazardous, it can pose primary hazards, especially to seed-eating and gallinaceous birds and waterfowl. Strychnine can pose primary hazards, such as to seed-eating birds; secondary poisoning may be minimal unless predators consume stomach or cheek-pouch contents of poisoned prey. 1080 can result in primary poisoning, but it especially can pose a secondary hazard to mammalian predators; the risk to raptors is minimal. Anticoagulants are toxic both primarily and secondarily; they can pose a substantial hazard to raptors. Gallinaceous birds, however, are quite resistant to them. Hazards associated with any one rodenticide may vary significantly depending upon use pattern (e.g. commensal *vs* field). Environmental concerns over rodenticide hazards to wildlife are increasing greatly and will affect future registrations.

Introduction

In comparison with other pesticides registered in the USA, the hazards to wildlife associated with rodenticide use have received limited attention. However, concerns for non-target hazards are addressed by the US Environmental Protection Agency (EPA) and rodenticide manufacturers as part of the current registration process and are reflected in restrictions on rodenticide labels.

Although some laboratory research has been conducted to help assess primary and secondary poisoning hazards from rodenticide use, these studies usually have involved a limited number of species and highly controlled conditions. More importantly, relatively few field studies have been conducted to evaluate the specific hazards to non-target wildlife from the various use patterns, active ingredients, and formulations of rodent bait available today.

In the USA, more emphasis now is being placed on the evaluation of non-target hazards from rodenticide use. Of the field studies that have been conducted, most were directed by the US Fish and Wildlife Service. The objective of this paper is to review some of those field studies and, in

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particular, to discuss methodologies, hazards associated with various compounds and use patterns, and various aspects of hazard evaluation and interpretation.

Methods

Primary poisoning involves consumption of rodenticide bait by a non-target animal (such as a grain-eating bird or mammal), while secondary poisoning results when a predator or scavenger consumes a target or non-target animal that has consumed a rodenticide bait. Most field studies have focused on either primary or secondary hazards rather than both. Additionally, most studies have focused on a limited number of non-target species or an indicator species. Logistics, equipment, and capture limitations often preclude broader efforts to evaluate simultaneously both types of hazards or to monitor numerous species.

The approximate hazard to closely related species may be assessed through examination of an indicator species that is representative of a particular vertebrate group (e.g. Passeriformes, Strigiformes, Canidae, Mustelidae). Indicator species most often have been chosen because their diet and habitat use suggest, *a priori*, that they are species at greatest risk.

Carcass searches have been used to assess hazards to non-target wildlife (Hegdal *et al.*, 1986), but these provide limited data. Animals are found dead infrequently in the wild because of cryptic colouration, vegetative cover, or consumption by scavengers. Additionally, because of the delayed action of some rodenticides and the ranging ability of some non-target wildlife, mortality may occur at a considerable distance from a treated area. We have found that even an animal wearing a radio transmitter can be difficult to visually locate although immediately underfoot. Carcass searches also have been used to find target animals, in order to determine their residue loadings. Thereby, potential secondary hazards to predators have been extrapolated (Barnes *et al.*, 1985).

Direct counts of individuals present before and after treatment demonstrate the persistence or reduction of individuals, but this technique has not been widely used as part of rodenticide hazard evaluations. It has been used successfully in primary hazard assessment by marking birds, noting their territories, and monitoring their presence before and after treatment (Hegdal & Gatz, 1976). Without marking of individuals, it generally is not possible to quantify individual mortality, only numerical abundance of animals present. If individuals, such as passerine birds, are replaced quickly by conspecifics, then mortality may not be detected unless it is catastrophic. Most importantly, direct counts do not provide adequate data on many predatory species, especially nocturnal ones, because of their secretive nature. For this reason, direct counts may be more useful in primary hazard evaluations because of the observability of the species at potential risk and their numerical abundance.

Indirect counts also have been used as an index to numerical changes in non-target populations. These techniques can include track counts and call counts (Hegdal *et al.*, 1986). However, as with direct counts (especially of unmarked individuals), these techniques do not document the actual mortality of individuals; in addition, the results can be distorted by population changes occurring independent of rodenticide use (such as from weather effects or emigration).

Monitoring of nest or den sites and breeding success has been an important and useful component of non-target hazard evaluations (Hegdal *et al.*, 1986). Not only might adult mortality be determined, but also that of young, and thus both long-term and short-term impact on productivity. Artificial nest sites have been used in some studies to provide specific locations to look for nesting raptors and to monitor nesting success (Hegdal & Blaskiewicz, 1984).

Radiotelemetry has been the technique used most frequently in the USA to evaluate non-target poisoning hazards (Hegdal & Gatz, 1976; Merson *et al.*, 1984). This method provides specific information on the fate of individuals after treatment. It also provides critical information on habitat use by non-target animals; such data can be essential in interpreting the

presence or absence of a potential hazard. Additionally, radiotelemetry allows for carcasses to be located for necropsy and residue analysis. Although radiotelemetry is an outstanding tool for detecting hazards, it is an expensive undertaking in time and equipment and requires skilled personnel. However, it is particularly essential for evaluating hazards to predators, since they frequently are difficult to observe or locate.

Necropsy and residue analyses have provided strong supportive evidence for documenting the most probable cause of death when mortalities have been observed. However, necropsy and residue information alone may not provide conclusive evidence for establishing cause of death, simply factors symptomatic of rodenticide poisoning. Residue loads may not be closely correlated with mortality (Hegdal & Colvin, 1988). Additionally, in some situations (such as with 1080), poisoning symptoms may not be obvious or residue detection readily achievable (Hegdal *et al.*, 1981).

Field studies of rodenticide hazards typically are not exclusive in the techniques used. Information assembled from population evaluation, behavioral studies of habitat use and diet, necropsy, and residue analyses all may be essential for implicating rodenticide poisoning as a mortality factor.

Results and discussion

Field evaluations of rodenticide hazards have included studies on three acute compounds, zinc phosphide, strychnine, and 1080 (sodium monofluoroacetate), and also the anticoagulants diphacinone and brodifacoum. Additional information on the toxicity of these compounds and potential hazards has been acquired through laboratory studies.

Zinc phosphide

This compound can pose primary hazards to rabbits, gallinaceous birds, waterfowl, and seed-eating birds (Rudd & Genelly, 1956; Janda & Bosseova, 1970; Hegdal & Gatz, 1977a). In field evaluations, Hegdal & Gatz (1977a) demonstrated primary hazards, such as to *Sylvilagus floridanus* (cottontail rabbit) and *Phasianus colchicus* (pheasant), when cracked corn treated with 2% zinc phosphide was used to control microtine rodents in apple orchards. However, they considered this use to be relatively safe for non-target wildlife and that it would not adversely affect any populations.

Zinc phosphide generally is not secondarily hazardous. Hegdal & Gatz (1977a) did not observe adverse effects on mammalian or avian predators during their orchard study. In another orchard study, Hegdal & Colvin (1988) did not observe a relationship between exposure to zinc phosphide and raptor mortality.

Laboratory studies repeatedly have demonstrated a lack of secondary toxicity to both birds and mammals from zinc phosphide (Brock, 1965; Siegfried, 1968; Bell & Dimmick, 1975; Schitoskey, 1975). For example, Evans *et al.* (1970) reported that *Aquila chrysaetos* (golden eagle), *Bubo virginianus* (great horned owl), and *Canis latrans* (coyote) that received multiple feedings of zinc phosphide-killed black-tailed *Lepus californicus* (jackrabbit) showed no symptoms of secondary intoxication. Steininger (1952) concluded that zinc phosphide is so extensively decomposed in the digestive tract of poisoned rodents that it cannot subsequently injure rodent-eating birds.

Strychnine

Field studies have been conducted by Hegdal & Gatz (1976, 1977b); Fagerstone *et al.* (1980), and Anthony *et al.* (1984) to evaluate potential hazards from using strychnine baits for rodent control. Each study included grain bait containing 0.5% strychnine. However, the first study

(1976) focused on underground baiting for *Geomys bursarius* (plains pocket gopher) in old-fields using a burrow-builder; the second (1977) involved surface baiting for *Spermophilus richardsonii* (Richardson's ground squirrel) in rangeland; and the third (1980) and fourth (1984) dealt with hand-baiting of underground burrows for *Thomomys mazama* (western pocket gopher) in conifer plantations.

Hegdal & Gatz (1976) did not observe detrimental effects on seed-eating birds except for one *Zenaidura macroura* (mourning dove) mortality. They regarded underground baiting with strychnine as relatively safe for non-target wildlife. In contrast, Hegdal & Gatz (1977b) observed a significant hazard to seed-eating birds, especially *Eremophila alpestris* (horned lark), *Z. macroura*, and *Agelaius phoeniceus* and *Euphagus cyanocephalus* (blackbirds), from surface baiting.

Fagerstone et al. (1980) observed some primary poisoning of small mammals but detected no difference in population sizes before and after treatment. Anthony et al. (1984) only monitored primary hazards to *Spermophilus lateralis* (golden-mantle ground squirrel) but found that squirrel populations were reduced 50–70% after treatment.

Secondary poisoning was not observed during either of the two strychnine studies by Hegdal & Gatz (1976, 1977b), even though numerous raptors, mammalian predators, and nest sites were monitored. Hegdal et al. (1981), and also Anthony et al. (1984), described the secondary hazard from strychnine as minimal, although they believed that secondary poisoning could occur if the stomach or cheek-pouch contents of poisoned prey were consumed. Schitoskey (1975) demonstrated secondary strychnine poisoning of *Vulpes macrotis* (kit fox) in the laboratory, while Marsh et al. (1987) noted that *Canis latrans* tended to reject portions of gastrointestinal tracts of poisoned squirrels, resulting in a minimal secondary hazard.

1080

Compound 1080 was evaluated for primary and secondary hazards by Hegdal et al. (1986) when used to control *Spermophilus beecheyi* (California ground squirrel) in rangeland. They found that primary hazards existed for non-target rodents and rabbits but that there were few incidences of primary poisoning of seed-eating birds. Poisoning of rabbits and non-target rodents as a result of 1080 baiting for ground squirrels also has been noted by Marsh (1968).

Hegdal et al. (1986) found that canids and felids were most at risk from secondary 1080 poisoning but that the risk to predatory birds was low. They also reported some mortality of insectivorous birds that apparently fed on ants killed by 1080. Similarly, the secondary toxicity of 1080 to *C. latrans* was recently demonstrated by Marsh et al. (1987) in the laboratory.

Marsh et al. (1987) commented that the secondary hazards with 1080 may be reduced in the field by lowering bait concentrations and the amount of bait applied. Recent investigations (Matschke & Hegdal, 1985) indicate that 1080 efficacy can be maintained, at least for some species, at lower bait concentrations.

First-generation anticoagulants

Currently, only diphacinone and chlorophacinone are registered and used for field rodent control, such as for microtine rodents in orchards. Field data on the primary and secondary hazards associated with first-generation anticoagulants are limited. However, some secondary hazard data are available from laboratory studies (Evans & Ward, 1967; Mendenhall & Pank, 1980; Townsend et al., 1981).

Hegdal (1985) evaluated the primary hazards to game birds from diphacinone when used to control microtine rodents in apple orchards. He found the primary hazard to these gallinaceous birds to be low, even though many fed extensively on the bait. Secondary hazards were not evaluated, but observations of primary poisoning suggested a potential hazard to predators, including man.

Second-generation anticoagulants

Hazards to wildlife associated with anticoagulant rodenticides have been discussed by Kaukeinen (1982). Limited field data on primary hazards are available on second-generation compounds, and two major field studies have been conducted to evaluate secondary hazards.

In a study of *Rattus norvegicus* (Norway rat) and *Mus musculus* (house mouse) control on farmsteads (in and around buildings), Hegdal & Blaskiewicz (1984) and Colvin (1984) found that the potential hazard to *Tyto alba* (barn owl) from brodifacoum bait (50 ppm) was low. Although the owls nested and roosted on farmsteads, they demonstrated selective behavior for grassland foraging habitat and microtine rodents. Thus, neither the location of rodenticide use nor the target species were used frequently by owls as a foraging resource.

In contrast to the study on *Tyto alba*, Hegdal & Colvin (1988) found that when brodifacoum bait (10 ppm) was evaluated for control of microtine rodents in orchards, there was a substantial hazard to *Otus asio* (eastern screech-owl). This study demonstrated the poisoning hazard that can be associated with second-generation compounds, and the impact when the target species is the same for both rodenticide and predator.

No second-generation anticoagulant currently is registered in the USA for non-commensal (field) uses. Hazards to non-target wildlife probably will limit such registrations.

Field studies of rodenticide hazards provide data on non-target wildlife in settings that allow for normal display of behavioral characteristics, the effects of ecological conditions, and typical use patterns of rodenticide bait. However, field evaluations are expensive, can involve numerous personnel, are difficult to coordinate on a large scale, and can encounter problems related to weather, animal capture, and equipment maintenance in the field. Additionally, given a host of environmental factors that can affect population change, it may be difficult to distinguish rodenticide-related effects from those resulting from other environmental or behavioral factors.

Laboratory studies, in contrast, involve a highly controlled situation. However, the behavioral patterns of wildlife in captivity may be altered, and limited space and stress may contribute to poisoning symptoms (Jaques & Hiebert, 1972). Importantly, rodenticide exposure is actively and rigidly controlled by the experimenter in the laboratory, rather than being a passive encounter during normal feeding and habitat use, as occurs in the field. Because specific use patterns and formulations of bait, relative to foraging behavior of the non-target animal, cannot be taken easily into direct account, dose levels administered in the laboratory become tests of toxicity rather than an absolute determination of hazard.

Laboratory studies can generate important base-line information for determining whether a field study should be initiated and also the species that may be at greatest risk (avian or mammalian). If a rodenticide is demonstrated in the laboratory not to be hazardous to non-target wildlife or to certain non-target species either primarily or secondarily, field studies testing such questions may not be necessary or of critical importance. Conversely, if primary or secondary toxicity is demonstrated readily in the laboratory, decisions then can be made to either proceed with a field study or not to pursue rodenticide registration or certain use patterns.

Hazard studies can be viewed sequentially, starting with laboratory studies, short-term field studies, and then long-term field studies (Colvin & Hegdal, 1988). At each plateau, information obtained may signal the need to proceed with additional research, or be adequate to demonstrate or project the potential hazard. Credible decisions and interpretations must be made at each step, since it simply is not possible to test every conceivable species in the laboratory or to monitor numerous species in prolonged field studies.

The grand question of long-term population effects lies beyond the issue of individual hazard and short-term population effects. However, a study to evaluate the population dynamics of a non-target species and its ability to withstand an additional mortality factor such as rodenticide use should be strongly justified before commitments are made to such involved and expensive studies.

Field studies readily can become simply a test of survival or mortality. Of critical importance, however, are the reasons why survival or mortality were observed after treatment. For example, interpretation of results and the predictability of hazard to individuals require information on non-target species' foraging behavior and habitat use (Colvin, 1984); the acquisition of such information needs to be given greater emphasis in field studies. An ecological approach is demanded, rather than simply asking 'How many lived and how many died?'

Similarly, sound ecological information on non-target species is important for explaining changes in non-target populations. Otherwise, the mere presence of a rodenticide in the environment and a reduced non-target population can be falsely related. For example, the drastic decline of *Tyto alba* populations in the midwestern United States over the past 30 years was attributed to rodenticide use on farmsteads. However, research on population dynamics and habitat requirements showed that loss of grassland foraging habitat was the principal factor in species loss, not farmstead use of rodenticide (Colvin, 1985).

The hazards associated with a particular rodenticide may be influenced (often drastically) by the formulation (treated grain, pellets, wax blocks), concentration of active ingredient in bait, use pattern, and target species. For example, studies of brodifacoum use on farmsteads (for commensal rodent control) and in orchards (for vole control) have illustrated how the same active ingredient can pose a minimal or substantial hazard, depending upon the use pattern and target species. The study of brodifacoum use in orchards (Hegdal & Colvin, 1988) also demonstrated that even with the same use pattern, one raptor species (*Otus asio*) may be at considerable risk, while another (*Strix varia*, barred owl) may not be (apparently because of differing habitat selection).

There is growing concern in the USA regarding the hazards associated with rodenticide use. Concern over the hazard to raptors (at the apex of food pyramids) is particularly acute. Because rodenticides used to control field rodents (e.g. voles, pocket gophers, ground squirrels) pose the most direct exposure to wildlife, particularly predators, those compounds and use patterns will receive greater scrutiny. We anticipate that for rodenticides in use, but for which there are limited data available on non-target hazards, additional data will be required by EPA to maintain registrations. Also non-target hazard data will continue to be a key registration requirement for proposed rodenticides and use patterns.

Development of rodenticides with low toxicity to birds and low secondary toxicity should be given particular attention, and these will have the greatest opportunity for registration for field rodent control. Additionally, reduction in treatment rates, lower concentration of active ingredient in bait, and restricted use patterns may receive greater emphasis in the effort to avoid or reduce non-target hazards associated with rodenticide use.

Risques d'effets non intentionnels associés à l'utilisation de rodenticides aux Etats-Unis

Il y a eu relativement peu d'études sur l'évaluation des risques secondaires de rodenticides pour la faune sauvage. Aux Etats-Unis, des études de plein champ ont porté sur l'utilisation de poisons à toxicité aiguë et chronique, y compris le phosphore de zinc, la strychnine, le produit 1080 (monofluoroacétate de sodium) et les anticoagulants diphacinone et brodifacoum. Les techniques utilisées comprennent le dénombrement de cadavres, d'animaux vivants ou de signes de leur activité, la surveillance des nids, la radiotélémétrie, les études sur l'écologie et l'alimentation des espèces concernées, la nécropsie et l'étude des résidus. Si le phosphore de zinc ne présente généralement pas de risques secondaires, il occasionne des risques primaires, essentiellement aux oiseaux granivores, aux gallinacés et aux oiseaux aquatiques. La strychnine peut présenter des risques primaires (par exemple pour les oiseaux granivores) mais un faible risque secondaire à condition que les prédateurs ne consomment pas le contenu gastrique ou celui des abajoues des proies empoisonnées. Le 1080 peut donner lieu à des empoisonnements

primaires, mais pose surtout un problème de risque secondaire pour les mammifères prédateurs; le risque pour les rapaces est minime. Les anticoagulants sont toxiques aux niveaux primaire et secondaire; ils présentent un risque important pour les rapaces. Les gallinacés y sont par contre assez résistants. Les risques associés à l'utilisation d'un rodenticide peuvent varier de façon importante en fonction du schéma d'utilisation (par exemple rongeurs synanthropiques ou rongeurs aux champs). Le problème des effets secondaires des rodenticides est de plus en plus préoccupant et il en sera davantage tenu compte à l'avenir pour l'homologation de ces produits.

Обзор нецелевых вредностей, связанных с применением родентицидов в США

Сравнительно мало исследований было проведено для оценки вредностей для фауны в связи с применением родентицидов. В США полевые исследования проводились как на острых, так и на хронических соединениях, включая фосфид цинка, стрихнин, 1080 (монофтороацетат натрия), а также с такими антикоагулянтами, как дифацин и бродифакум. В этих исследованиях использовались следующие методы: подсчет трупиков, прямые и косвенные подсчеты, мониторинг гнездовой, дистанционные измерения с помощью радиопередатчиков, оценка мест обитания и диеты, аутопсия и анализ остаточных количеств. Несмотря на то, что фосфид цинка, как правило, не влечет за собой никаких побочных вредностей, им могут вызываться вредности первичного характера, в частности, для пожирающих семена и куровидных птиц, а также для водоплавающей птицы. Стрихнин может вызывать первичные вредности, в частности для птиц, пожирающих семена; вторичное отравление может быть минимальным, если хищники не потребляют содержимого желудочно-кишечного тракта или зашечных мешков отравленной добычи. Препарат 1080 может приводить ко вторичному отравлению, но, главным образом, он ставит серьезную проблему вторичных вредностей для млекопитающих хищников; опасность для хищных птиц при этом минимальная. Антикоагулянты показывают токсичность как первичную, так и вторичную. Они представляют собой существенную опасность для хищных птиц. Однако, водоплавающая птица, со своей стороны, показывает довольно высокую к ним устойчивость. Вредности, связанные с любым из родентицидов, могут быть самыми разнообразными, в зависимости от конкретной модели применения (например, для домашних грызунов, в отличие от полевых). Опасения в появлении родентицидов для фауны, в настоящее время сильно возросли и в будущем будут непременно сказываться на дальнейшем сертифицировании препаратов.

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