

University of Nebraska - Lincoln

DigitalCommons@University of Nebraska - Lincoln

Proceedings of the Tenth Vertebrate Pest
Conference (1982)

Vertebrate Pest Conference Proceedings
collection

February 1982

A REVIEW OF THE SECONDARY POISONING HAZARD POTENTIAL TO WILDLIFE FROM THE USE OF ANTICOAGULANT RODENTICIDES

Dale Kaukeinen

Biological Research Center, ICI Americas Inc., Goldsboro, North Carolina 27530

Follow this and additional works at: <https://digitalcommons.unl.edu/vpc10>



Part of the [Environmental Health and Protection Commons](#)

Kaukeinen, Dale, "A REVIEW OF THE SECONDARY POISONING HAZARD POTENTIAL TO WILDLIFE FROM THE USE OF ANTICOAGULANT RODENTICIDES" (1982). *Proceedings of the Tenth Vertebrate Pest Conference (1982)*. 27.

<https://digitalcommons.unl.edu/vpc10/27>

This Article is brought to you for free and open access by the Vertebrate Pest Conference Proceedings collection at DigitalCommons@University of Nebraska - Lincoln. It has been accepted for inclusion in Proceedings of the Tenth Vertebrate Pest Conference (1982) by an authorized administrator of DigitalCommons@University of Nebraska - Lincoln.

A REVIEW OF THE SECONDARY POISONING HAZARD POTENTIAL TO WILDLIFE FROM THE USE OF ANTICOAGULANT RODENTICIDES

DALE KAUKAINEN, Biological Research Center, ICI Americas Inc., Goldsboro, North Carolina 27530

ABSTRACT: The utility and characteristics of the family of anticoagulant rodenticides are reviewed, including the new members difenacoum, bromadiolone and brodifacoum. General considerations are given in investigating the likelihood of nontarget poisoning with rodenticides. The literature dealing with secondary poisoning studies and concerns with the use of anticoagulant rodenticides is reviewed. The utility of laboratory toxicity data versus field-generated exposure data is compared. Considerations of secondary poisoning by anticoagulants are reviewed as regards parameters such as specific predator-prey systems, biotopes, rodenticide use patterns, and risk-benefit assessments. Finally, examples of appropriate field studies proposed to assess specific secondary poisoning risk situations associated with particular anticoagulant usage patterns are exemplified by reference to studies conducted by ICI and outside researchers with brodifacoum rodenticide bait formulations.

INTRODUCTION

Since their introduction in the 1950s, anticoagulants have revolutionized rodent control, particularly commensal rodent control. They have also been applied with some success against rodent pests of agriculture. However, the need for multiple feedings and reapplications of typical anticoagulants has limited agricultural utility and has necessitated the dependence in all or part (depending on rodent species involved) on several acute rodenticide materials, notably 1080, zinc phosphide and strychnine. These acute materials, while effective in limited feedings, do have disadvantages in their general lack of selectivity and antidote, and the generally accepted perception of bait shyness occurring from initial ingestion of sublethal quantities of bait, thus limiting their rebaiting potential.

These acute materials are also not to be considered as fully interchangeable or comparable alternatives in different use situations, and their specific rodent acceptance and nontarget hazard aspects have correspondingly relegated each to fairly specific current use patterns in the U.S.

SECOND-GENERATION ANTICOAGULANTS

It was of great general interest that the new "second-generation" anticoagulants have been developed during the past five to six years. These materials combine the advantage of the older anticoagulants with the additional and novel characteristics of single or limited lethal feeding potential and efficacy against rodents resistant to, or generally more tolerant of, other older anticoagulant materials. These new materials have the common ISO chemical names of brodifacoum, difenacoum, and bromadiolone.

Difenacoum, an ICI compound, has been the subject of a previous paper (Bull 1976) and is currently marketed under the trade name RATAK in parts of Europe and some other countries. Bromadiolone has also been previously characterized (Marsh 1977, Meehan 1978). It was developed by Lipha Lionnaise of France and formulations have been registered in the U.S. for commensal rodent control under the trade names MAKI® and CONTRAC®.

BRODIFACOUM

Brodifacoum has been the subject of many publications by ICI and independent researchers describing its efficacy and potential as an "all-purpose" rodent control material (e.g., Dubock and Kaukeinen 1978). With an LD₅₀ to most rodents of less than 0.5 mg/kg, brodifacoum provides for a single-feeding action at low active concentrations which offers novel rodent control applications. In contrast with acute rodenticides, symptoms are delayed and no bait shyness is observed. Brodifacoum has also been shown capable of controlling rodents resistant to other anticoagulants (Dubock and Kaukeinen 1978, Apperson, Sanders and Kaukeinen 1981). Brodifacoum first received U.S. registration for commensal rodent control in 1979 under the trade name TALON® and is also known and registered in many other countries as TALON, or HAVOC, RATAK-PLUS, RATAK-SUPER, KLERAT, or MATIKUS.

ANTICOAGULANT HAZARD CONCERNS

Rodenticides have not figured in most major conferences, reviews, handbooks, or other materials or forums dealing with pesticide-wildlife interactions (for example, see: Moore 1966, Tucker and Crabtree 1970, Pimentel 1971, and NAS 1979). For anticoagulant rodenticides, no significant nontarget incidents of nonrodent, wildlife mortality have been the subject of any known scientific publication during the 30 years of their usage throughout the world.

For example, extensive monitoring of the 400 to 500 tons of anticoagulant bait applied yearly in California for the control of agricultural rodent pests has noted very few primary and secondary poisoning effects over the past 15 years (Clark 1978)

The current interests in elucidating pesticide-wildlife interaction stem, of course, from the unfortunate incidents and effects of compounds such as persistent hydrocarbon insecticides, for example, DDT. The resulting public furor and the tremendous mass of scientific, political, regulatory, and other activities have sought to elucidate and preclude these complex phenomena resulting from major use pesticides such as insecticides, herbicides, and fungicides. These materials may impact very low on the food chain and thus ultimately may involve a multitude of organisms and environments and potentially produce widespread effects. Rodenticides, by contrast, are minor use pesticides, acting higher in the biologic community, in specific, localized habitats and interfaces. They could thus be theorized to have the greatest potential impact on those local mammalian or avian predators or scavengers which might consume poisoned rodent prey in specific rodenticide use situations (secondary poisoning).

PRIOR LITERATURE ON HAZARD

Acute rodenticides such as 1080, zinc phosphide, and strychnine have been the subjects of previous extensive field hazard evaluations by the U.S. Fish and Wildlife Service, funded by the government (e.g., Hegdal and Gatz 1976). The new second-generation anticoagulant rodenticides have been demonstrated (Park et al. 1980) to share mode of action and the same antidote (Vitamin K₁). They have the same low active content (or even lower) of the earlier anticoagulants. They are also envisioned as being effective at lower amounts of bait applied than first-generation anticoagulants and even some acute materials. However, the second-generation anticoagulants have been subject to the new U.S. regulatory requirements and environmental concerns which earlier anticoagulants largely escaped. Therefore, largely through industry funding, it has been necessary to address these concerns by the most appropriate studies that consensus opinion could devise to evaluate brodifacoum's potential hazards.

EVALUATING CHEMICAL RISK

Some secondary-toxicity laboratory studies with wildlife have shown that captive predators could be intoxicated by sufficient no-choice feeding of anticoagulant-poisoned or dosed prey. For example, Evans and Ward (1967) fed poisoned nutria to mink. Another study dealt with diphacinone for canine predator control with poisoned carcasses (Savarie et al. 1979). Golden eagles exhibited toxic symptoms after feeding on the poisoned tissue. Neither study involved a use pattern making it particularly relevant to most current field rodent control uses and related hazard concerns.

A more recent study (Mendenhall and Pank 1980) determined the effect of feeding anticoagulant-poisoned prey to captive barn owls, and for some anticoagulants, to yet other owl species. All of the three second-generation anticoagulants caused some secondary toxic effect within groups of barn owls from multiple no-choice feeding on toxic prey, as did diphacinone in two other owl species. Different effects of the same anticoagulants on the same and on different owl species after comparable toxic diet intake could not be readily explained as variations and limitations of the protocols used made dose-response effects and comparisons difficult to determine. Parallel but yet-unpublished secondary-toxicity studies by Pank (personal communication) with mongooses similarly fed poisoned rats showed no apparent effect with some treated mongooses, even with the ICI anticoagulants difenacoum and brodifacoum, while more extensive group mortality was seen with diphacinone, chlorophacinone, and bromadiolone.

STRESS AND OTHER EFFECTS

Variations observed among these and similar studies may relate, in part, to artificial test conditions. Diet changes (Colvin and Wang 1974) and increased activity (Oliver and Wheeler 1978) may lower susceptibility to anticoagulants. As Jaques and Hiebert (1972) have demonstrated, spontaneous hemorrhage from anticoagulant ingestion is a multi-causative phenomenon and is greatly influenced and triggered by stress and other variables. This suggests captive testing with some wild predators may be of limited value, as the activity and stress variables could not easily be standardized and only prohibitively large test groups might overcome the problem. Even slight injury in such captive predators may predispose them to hemorrhage in affected areas, as for example, the site of blood collection (e.g., Mendenhall and Pank 1980), or bruising or wounds from efforts to escape confinement.

SECONDARY TOXICITY PROTOCOL DEVELOPMENT

To overcome potential stress effects, secondary-toxicity studies with domestic animals such as dogs or cats fed poisoned rodents may be viewed as a model for wild canines or felines. For example, the research by Prier and Derse (1962) with fox terriers indicated warfarin did not pose a significant secondary hazard to dogs. However, domestic dogs show considerable variation in response to anticoagulants depending on breed. Beagles, for example, may be unusually sensitive to anticoagulants (McKelvie and Anderson 1963), although they are the most common laboratory research canine. Extrapolation to wild canines and felines from domesticated breeds must therefore be done with caution, and the use of large groups of outbred, mixed-breed animals may be indicated (Godfrey, Reid and McAllum 1981). For raptors, an alternate model has been suggested (Fink and Jaber 1981) which may allow for useful comparisons. For the endangered black-footed ferret, and as indicators for mustelids in general, mink or domestic ferrets are often used. For example, Ringer has determined the LD₅₀ of brodifacoum to the mink as 9.2 mg/kg (personnel communication). This relatively high figure may have been due to the rapid digestion time of approximately two hours in the mink and suggests limited time for anticoagulant absorption in the gastrointestinal tract.

Many additional laboratory testing variables and limitations make most existing lab-derived secondary-toxicity data and proposed testing protocols of limited utility in the sense of interpreting and extrapolating to a field hazard situation. Parameters to be addressed and selected include: prey selection, intoxication, preparation, and presentation; predator selection, health, handling, and captive conditions;

predator acclimation, intoxication, and observation. There may be clear preferences for the study design of specific predator-prey models and such studies or protocols (e.g., Holler and LeFebvre 1981) may reveal a degree of absolute and relative toxicity (as for example, when compared with toxicants tested under "identical" conditions). Especially, such studies may be of great value in determining diagnostic characteristics for investigating causes of suspected wildlife poisonings. However, they are seldom able by themselves to determine the significance of the particular toxicity or lack of toxicity observed. Dietary toxicant levels given to predators, if known, are seldom correlated to residues of that toxicant in poisoned prey under actual use conditions. Repeated toxic prey administration does not allow for the decreasing toxic body burdens expected in rodents in the field as survivors metabolize or excrete the toxicant. Further and most importantly, such laboratory studies cannot assess the probability that poisoned prey and predators will interact in the field to cause a particular exposure level.

In a recent study, potential tawny owl secondary poisoning from the baiting of squirrels with warfarin in the UK was assessed (Townsend et al. 1981). A lab secondary-toxicity study involved dietary levels comparable to rodent residues in the field. Given the results and the unlikelihood that the diet of tawny owls would consist solely of contaminated prey, it was concluded that this usage of warfarin did not pose a significant threat to the local owl population. Such studies with known dietary levels can be of greater predictive value in assessing field hazard.

HAZARD ASSESSMENT

In assessing in the field the impact of toxic chemical use on nontarget animal risk, both chemical, biological, and ecological components must be investigated in the field to assess actual exposure, as pharmacological susceptibility bears little relationship to ecological vulnerability (Moore 1966). Proposed toxic chemical use, such as for anticoagulants, can involve considerations of basic toxic properties, the formulation persistence and mobility, amount and frequency of application, method and timing of application, external pattern of use, site and locality of use, and effect on target animals in the use area (behavior, accessibility, and place of death, etc.). Nontarget animal risk, such as with predators, is related to exposure and toxicity factors such as chemical sensitivity and effect; site and locality of chemical use; animal or population health, status, and behavior; periodicity; breeding and feeding habits; habitat requirements; stage of development; and isolated or combined effects of other potentially deleterious factors in the environment.

Primary poisoning of most nontarget animals with rodenticides such as anticoagulants can frequently be overcome by modification of toxic chemical formulations or application techniques. For example, it has been shown that certain coloring agents can reduce the acceptability of rodenticide baits to some birds (Pank 1976). Modified application techniques may significantly reduce hazard, such as by the use of burrow builders for subsoil rodenticide application (e.g., for gopher bait; Hegdal and Gatz 1976) or bait boxes, bait packs, or other bait enclosures or protective applications.

The use of bait stations or protected, hand-applied bait may have some practical limitations for large acreages (Brooks and Schwarzkopf 1981). The mechanical broadcast application of small, dispersed bait particles in some agricultural situations and the use of pulsed baiting (Dubock 1982) in others both can act to reduce the amount and availability of bait to larger-bodied primary feeders and also reduce consumption and resulting residues in rodents potentially at risk of predation. For some specific applications, it is possible to reduce the concentration of anticoagulants, particularly brodifacoum, in baits for some species without any corresponding need to significantly increase rates or frequency of applications. Thus, we have shown with an experimental brodifacoum bait, VOLID, for vole control in orchards, that reducing the active content of the bait from 50 to 10 ppm caused an even greater proportional decrease in vole residues (Table 1). Efficacy in the field was maintained at low application

Table 1. Results of no-choice lab feeding for 3 days of *Microtus pinetorum* with 10 vs 50 ppm bait (groups of 15 voles, killed on 4th day).

Brodifacoum concentration group	Sex	Mean tissue residue in voles (ppm)
10 ppm	M	0.53 (S.D. ± 0.24)
	F	0.40 (S.D. ± 0.20)
50 ppm	M	5.21 (S.D. ± 2.06)
	F	2.17 (S.D. ± 1.17)

rates (e.g., 30 pellets per square meter). In an unpublished lab study, Savarie (personal communication) observed no deaths in captive kestrels fed on voles poisoned with brodifacoum at doses below a residue of 6 ppm in vole tissue fed at 30 grams/day for five days. Many rodent predators such as most raptors do not prefer dead carcasses as prey items. Rodents that have died from anticoagulants, therefore, generally pose less hazard than do those active individuals with toxic residues for those few days before either death or recovery.

However, it is impossible by formulation and application modifications or good use practices to preclude all potential predator-prey interactions or the potential for secondary poisoning in habitats where some predatory scavenging animals may exist.

POPULATION EFFECTS

It is recognized in such considerations that all the variables in actual rodenticide use, geographic differences, and different degrees of predator exposure and sensitivity may offer some individual effect. Therefore, with the possible exception of work with endangered species, field studies in which actual hazard is being investigated must be of a scale to monitor population effects. Such studies are thus necessarily large and time-consuming. Their execution is beyond the capabilities of all but a small group of specialists, and large teams of skilled personnel must usually be involved. Study planning must involve selection of a greatest-risk predator(s) or alternate indicator species based on intended rodenticide use; selection of site based on presence of target rodents and potential secondary feeders; consideration of timing of study and study impact; determining a meaningful number of predators to monitor and the type, incidence, and extent of monitoring; verifying prey presence, estimate of abundance, verifying intoxication, and determining chemical effects in prey rodents; determining the specific application of the chemical, in particular, the quantity applied and whether one or several rates are considered; and finally, determining overall predator-prey interactions and effects. With these data in conjunction with other laboratory and field results, and with the published literature and consideration of not using chemical control, an optimum and realistic assessment of rodenticide impact on wildlife can be facilitated.

BRODIFACOU M SECONDARY HAZARD STUDIES

The largest study of potential anticoagulant rodenticide secondary hazard to raptors to date was supported by ICI and completed in 1980 by staff from the Denver Wildlife Research Center of the U.S. Fish and Wildlife Service. Working with barn owls within a 1100 km² area of southwestern New Jersey, the study sought to determine the effect of brodifacoum farm baiting with the 50-ppm pelletized TALON formulation on the raptor--the barn owl--most closely associated with this rodenticide use and the type of treated sites. The sites involved typical farms with commensal rodent infestations which were verified by census techniques. Also, sites were monitored during baiting to verify that brodifacoum bait was being consumed. Some treated sites with nesting owls received stocked rodents to create a "worst case" situation.

Valuable movement and feeding data were obtained from the 34 radio-equipped owls and other non-radioed birds. Owls moved farther than expected and hunted away from farmsteads, consuming very low levels of commensal rodents. At least 9 and possibly 12 of the radioed birds during the 6-month study were shown to have frequented TALON-treated sites for at least 5 and up to 62 days posttreatment. Young owls were fledged from at least 8 sites where poisoned rodents were demonstrated to be available on the farmstead for at least a portion of the nesting and feeding period.

Although radioed and other owls were observed killed by vehicles, predation, human disturbance, and electrocution on power lines, no owl mortality could be attributed to TALON baiting. Significant habitat loss was observed even during the course of the study. It was additionally noted that erection of simple nest boxes in this or similar areas of the country could have a very significant effect in sustaining and increasing local barn owl populations which are generally suffering from the loss of suitable nesting habitat (e.g., Marti, Wagner, and Denne 1976). This enhancement would undoubtedly be such to counter any but the most serious insult to local owl populations.

Based on a review of the final report (Hegdal and Blaskiewicz 1981) of this barn owl hazard study and in consideration of other relevant submitted data, the U.S. Environmental Protection Agency, during January and February of 1982, lifted previous restrictions for 50-ppm brodifacoum commensal rodent control formulations (TALON, WEATHER BLOKS, HAVOC), and allowed unrestricted usage of these formulations in and around structures including agricultural buildings. (These findings have not been applied to other second-generation anticoagulants, and additional studies with these materials in the laboratory or in specific field use situations may be needed.)

CURRENT AND FUTURE HAZARD RESEARCH

In considering the second-generation anticoagulants for noncommensal agricultural rodent control, somewhat different predator-prey complexes are perceived. Currently, large-scale field hazard evaluations of the effects on raptors in noncommensal, agricultural applications of brodifacoum are underway. These will be the subject of future publications by the principal investigators. Results are incomplete, but, again, deleterious effects such as vehicle kills and apparent raptor kills from other predators caused a significant part of the total mortality observed. Such nonchemical effects are for the most part continuous as opposed to any impact from seasonal rodenticide baiting in which poisoned prey might be available to predators for only a few days prior to death. Raptor hunting ranges as determined from telemetry generally extend considerably to nontreated habitat, providing a dilution effect in the consumption of any poisoned prey.

DISCUSSION

The new anticoagulants share the same mode of action and the same antidote as the older anticoagulants so well known and well studied by the medical, biochemical, and veterinary community. Conversely, attempts to develop new acute rodenticides with largely unknown modes of action and for which effective

antidotes may be unavailable have resulted in unforeseen complications (Prosser and Karam 1978). However, with anticoagulants, liver accumulation, for example, has been shown not to affect liver function (Jaques 1959). Further, the apparently complete recovery of animals sublethally dosed in the laboratory (or antidoted after greater toxicant administration) suggests the condition and effects of hypocoagulability in the field would be completely reversible. Thus, sublethal effects should be of limited duration and significance to the individual. Long-term feeding (90 days) with 0.03 to 0.5 ppm diphacinone to Norway rats failed to show any mortalities or abnormalities at the conclusion of the study (Elias and Johns 1981). The lack of adverse effect from low doses of anticoagulants in mammalian systems is evidenced by the fact that thousands of people daily consume medicinal doses of hydroxycoumarins and other anticoagulants as antithrombic agents.

REGISTRATION REQUIREMENTS

All rodenticides now being registered in the U.S., as for brodifacoum, must have provided regulatory authorities with studies such as mode of action, therapy, handling and disposal, basic LD₅₀ and LC₅₀ screening on a number of mammal and bird species, and environmental fate as regards microbial action, soil mobility and dissipation and other data including potential wildlife hazard.

Building on existing rodenticide registrations and use patterns in the pursuit of additional uses is an optimum strategy, both for the registrant and for animal control and wildlife specialists, since it provides for a measure of foresight and assurance and is the most economical use for dwindling industry and government research funds, with the rapidly escalating costs associated with such research and registrations. A joint FAO/IAEA Committee said "not to find new chemical pesticides but to evaluate existing ones in terms of acceptable limits, potentiality for improved formulation and more prudent application, potentiality for causing resistance in pests and potential for incorporating into IPM at national and international levels" (Winterinham 1975).

ALTERNATIVES TO RODENTICIDES

Our knowledge of the general population dynamics of pest rodents is perhaps best exemplified in the work of Davis (e.g., Davis 1972) in which the significance of food, water, and shelter on the rodent population level is well understood. Toxic chemicals are properly only one part of integrated pest management (IPM). Rodent killing without associated environmental improvement provides only temporary control. While such principles apply generally to all pest rodents, it is unfortunate that in the one environment (the urban environment) where environmental control (e.g., reduced food and habitat) is most possible, this is seldom practiced to the extent necessary on an area-wide basis. Chemical control pressure must therefore be sustained.

Rodenticides are commonly used against agricultural rodent species in the recognition that, unlike the urban commensal rodent's environment, control by environmental manipulation is difficult. In high-value crops, a very low economic threshold generally negates effective environmental management, for such management, if possible, still leaves the few rodents necessary to produce unacceptable losses and to restore the population. While it is desirable to eradicate rodents from our cities, it is neither possible nor desirable to completely exterminate agricultural pest rodent species in most situations.

Field agricultural rodent control needs may also be quite limited in time, unlike the urban rodent situation. This may be to protect specific and vulnerable crop stages. Thus, field rodent rodenticides such as effective anticoagulants are appropriate to produce the drastic but admittedly temporary and localized population declines providing low level suppression such as can be achieved seasonally, or, for example, with ground squirrels by bait application every two to three years with truly effective control materials.

Biological (Wodzicki 1973), mechanical, and environmental rodent control have been shown to be of little or limited value as pest rodent control methods and some techniques, such as introduced predators, may pose a significant threat to native fauna. Bacterial preparations have been strongly discouraged by the World Health Organization because of potential dangers to man and other animals (WHO 1967). Virtually no significant practical application of these alternative methods has been verified or adopted in actual large-scale or widespread use—neither chemosterilants for control of ground squirrels (e.g., Alsager 1972) nor introduced predators for control of voles (e.g., Sullivan and Sullivan 1980), for example.

WILDLIFE MANAGEMENT

Predators have many values requiring their conservation. Aesthetics may be the strongest aspect in any rating system (Landry, Hirsch and McCaffrey 1979). Predators have often been shown to be generally of little direct value in controlling pest rodent populations (e.g., Pearson 1964, Voight and Glenn-Lewin 1978) and to provide little ecological stability to the agricultural lands and systems that are necessary to maintain our agricultural production and which currently suffer such serious rodent depredations in the U.S. and elsewhere (for example, see reviews by Jackson 1976 and Hopf, Morley and Humphries 1976). Most healthy predator populations adapt to prey declines (e.g., vultures in England after rabbit declines; Moore 1970) as might be locally caused by rodenticide treatments. Continued use of effective rodenticides may thus shift raptors and other rodent predators to alternate areas where they will be in less contact with man and potentially less affected by man's agricultural or control practices.

Wildlife management and conservation concerns such as for valued predatory birds and mammals should first include a determination of population health and status (e.g., Kennedy 1980) before consideration of the effects of both major use and selected minor use pesticides. However, such concerns and public

awareness must also extend to the generally far greater impact of changing land use on predator-prey complexes (e.g., Craighead and Mindell 1981, Geier and Best 1980). Also to be considered are the often considerable influence of vehicles, prey cycles, and declines; direct human disturbances, disease, weather and other physical or environmental effects. During a 3-year period, for example, shootings constituted a much greater cause of death in bald eagles from 29 states than other known factors, including pesticides (Kaiser et al. 1980). Public education by wildlife specialists can reduce direct human disturbance to valued predators, particularly those considered threatened or endangered. Optimum habitat identification (Bednarz and Dinsmore 1981) or existing habitat enhancement in depopulated portions of a predator's range can be an optimum use of resources by significantly improving the predator population's chances of withstanding the increased pressures brought upon it by all man's activities.

CONCLUSION

In summary, no significant nontarget effects on nonrodent wildlife from the registered field use of anticoagulant rodenticides have yet been reported. While individual predator effects may be occasionally expected, no significant mass effects to predator populations are anticipated from available information. Mass nontarget mortality from any rodenticides, when present, is usually readily observable (e.g., Clausen and Karlog 1977, Mendelssohn and Paz 1977). The cost of registering just a basic commensal rodenticide in the U.S. has risen 5-fold in less than 10 years to presently over \$5 million. The significantly smaller markets and the greater data requirements for registration that noncommensal agricultural rodent use situations present to registrants in developing rodenticides preclude an indefinite exhaustive excursion into complex wildlife impact considerations for each new use pattern of the same minor-use material. Knowledge that previous field hazard studies provide concerning specific predator-prey relationships in or near habitats where chemical rodent control is applied, in conjunction with the already extensive scientific literature, may preclude the need for such extensive studies for each additional rodenticide pattern or different geographic use area. Alternatives to the great expense and expertise requirements of radiotelemetry have been proposed (e.g., Edwards et al. 1979, Przygodna in Moore 1966) and may allow for more practical though less-exhaustive field hazard evaluations of both potential primary and secondary field effects of toxicants.

While environmental concerns should properly occupy the attention of us all, a realistic risk/benefit climate must prevail. To the agriculturalist, rodent damage and lack of effective control materials over several years have created emergency conditions in many areas (e.g., vole damage in the Northeast; Byers, Young and Neely 1976). To the forester, rodent damage and depredation have called a virtual halt to reforestation efforts in many areas (Radvanyi 1974). To the public health official, the potential of a rodent-borne disease outbreak is all too real (e.g., plague in western U.S. field rodents; Nelson 1980). Numerical information on potential rodent damage or disease threats in the absence of effective control is sorely needed in risk/benefit considerations to provide a realistic decision-making perspective for all involved. Through existing state and federal governments, wildlife biologists, conservationists, conscientious and skilled rodenticide applicators plus the power regulatory authorities now have, sufficient checks would already seem to exist to note and rapidly curtail any significant effects to wildlife from rodenticides under conditions of experimental or actual registered use. Such continual feedback allows industry, researchers, applicators and others, in a concerted effort, to continually and usefully increase their sophistication in devising effective low-hazard chemical rodent control.

The newer anticoagulant materials such as brodifacoum offer effective and practical alternatives to the continued use of the older anticoagulants and the currently used acute rodenticides. Vigorous pursuit and general support in all areas are needed to obtain the additional chemical control tools and the potential worldwide utility that the second-generation anticoagulants represent.

LITERATURE CITED

- ALSAGER, D.E. 1972. Experimental population suppression of Richardson's ground squirrels (*Spermophilus richardsonii*) in Alberta. Proc. 5th Vertebrate Pest Conf., Fresno, California, pp. 93-100.
- APPERSON, C.S., O.T. SANDERS and D.E. KAUKKINEN. 1981. Laboratory and field trials of the rodenticide brodifacoum against warfarin-resistant Norway rats. Pest. Sci. 12:622-668.
- BEDNARZ, J.C. and J.J. DINSMORE. 1981. Status, habitat use, and management of red-shouldered hawks in Iowa. J. Wildl. Manage. 45(1):236-241.
- BROOKS, R.J. and L. SCHWARZKOPF. 1981. Vole damage and control methods in Ontario, Canada, orchards. In: Byers, R.E., ed., Proc. 5th Pine and Meadow Vole Symp., Gettysburg, Pennsylvania. March 1981. pp. 2-6.
- BULL, J.O. 1976. Laboratory and field investigations with difenacoum, a promising new rodenticide. Proc. 7th Vertebrate Pest Conf., Monterey, California, pp. 72-84.
- BYERS, R.E., R.S. YOUNG and R.D. NEELY. 1976. Review of cultural and other control methods for reducing pine vole populations in apple orchards. Proc. 7th Vertebrate Pest Conf., Monterey, California. pp. 242-253.
- CLARK, D.O. 1978. Control of ground squirrels in California using anticoagulant treated baits. Proc. 8th Vertebrate Pest Conf., Sacramento, California, pp. 98-111.
- CLAUSEN, B. and O. KARLOG. 1977. Thallium loading in owls and other birds of prey in Denmark. Nord. Veterinarmed. 29:227-231.
- COLVIN, H.W. JR. and W.L. WANG. 1974. Toxic effects of warfarin in rats fed different diets. Toxicol. Appl. Pharmacol. 28:337-348.
- CRAIGHEAD, F.C. and D.P. MINDELL. 1981. Nesting raptors in western Wyoming, 1947 and 1975. J. Wildl. Manage. 45(4):865-872.

- DAVIS, D.E. 1972. Rodent control strategy. In: Pest Control Strategies for the Future. National Acad. Sci., Washington, pp. 157-171.
- DUBOCK, A.C. 1982. Pulsed baiting--a new technique for using high potency, slow-acting rodenticides. Proc. 10th Vertebrate Pest Conf., Monterey, California, pp. 123-136.
- and D.E. KAUKKINEN, 1978. Brodifacoum (TALON Rodenticide), a novel concept. Proc. 8th Vertebrate Pest Conf., Sacramento, California pp. 127-137.
- EDWARDS, P.J., S.M. BROWN, M.R. FLETCHER, and P.I. STANLEY. 1979. The use of a bird territory mapping method for detecting mortality following pesticide application. *Agro-Ecosystems* 5:271-282.
- ELIAS, D.J. and B.E. JOHNS. 1981. Response of rats to chronic ingestion of diphacinone. *Bull. Environm. Contam. Toxicol.* 27:559-567.
- EVANS, J., and J.L. WARD. 1967. Secondary poisoning associated with anticoagulant-treated nutria. *J. Amer. Vet. Med. Assoc.* 151:856-861.
- FINK, R.J. and M.J. JABER. 1981. The laughing gull (*Larus atricilia*) as a model for assessment of secondary poisoning. In: Lamb, D.W. and Kenaga, E.E., eds., *Avian and Mammalian Wildlife Toxicology: 2nd Conf. Proc.* Louisville, Kentucky. Amer. Soc. Test. Mater. Spec. Tech. Publ. 757, pp. 66-71.
- GEIER, A.R. and L.B. BEST. 1980. Habitat selection by small mammals of riparian communities: evaluating effects of habitat alterations. *J. Wildl. Manage.* 44(1):16-24.
- GODFREY, M.E.R., T.C. REID and H.J.F. MCALLUM. 1981. The acute oral toxicity of the anticoagulant brodifacoum to dogs. *N.Z.J. Exp. Agric.* 9:147-149.
- HEGDAL, P.L. and R.W. BLASKIEWICZ. 1981. Hazards to barn owls associated with the use of TALON (Brodifacoum bait) for controlling rats and house mice. Unpublished report to ICI, 81 pp. (Submitted to SETAC Journal).
- HEGDAL, P.E. and T.A. GATZ. 1976. Hazards to wildlife associated with underground strychnine baiting for pocket gophers. Proc. 7th Vertebrate Pest Conf., Monterey, California, pp. 258-266.
- _____, T.A. GATZ and E.C. FITE. 1981. Secondary effects of rodenticides on mammalian predators. In: Chapman, J.A. and Pursley, D., eds., *Worldwide Furbearer Conference Proc.* Frostburg, MD, Vol. III, pp. 1781-1793.
- HOLLER, N.R. and P.W. LEFEBVRE, 1981. Laboratory method for evaluating the secondary hazard potential of avicides and rodenticides to nontarget birds and mammals. In: E.W. Schafer and C.R. Walker, eds., *Vertebrate Pest Control and Management Materials*, Amer. Soc. Test. Mater. Spec. Tech. Publ. 752:46-51.
- HOPF, H.S., G.E.J. MORLEY and J.R.O. HUMPHRIES, eds. 1976. Rodent damage to growing crops and to farm and village storage in tropical and subtropical regions. *UK Cent. Overseas Pest Res. and Trop. Prod. Inst.*, HMSO, 115 pp.
- JACKSON, W.B. 1976. Evaluation of rodent depredations to crops and stored products. Joint FAO/WHO/EPPO Conf. Rodents of Agricultural and Public Health Concern, Geneva, 75 pp. mimeo.
- JAQUES, L.B. 1959. Dicoumarol drugs and the problem of hemorrhage. *Canadian Med. Assoc. J.* 81:848-854.
- and L.M. HIEBERT. 1972. Relation of stress to hemorrhage in laboratory animals. *Proc. Animal Care Tech. CALAS*, Edmonton, Canada, pp. 37-52.
- KAISER, T.E. et al. 1980. Organochlorine pesticide, PCB, and PBB residues and necropsy data for bald eagles from 29 states--1975-77. *Pesticides Monitor. J.* 13(4):145-149.
- KENNEDY, P.L. 1980. Raptor baseline studies in energy development. *Wildl. Soc. Bull.* 8(2):129-135.
- LANDRY, J.L., L.P. HIRSCH and E.R. MC CAFFREY. 1979. A rating system for threatened and endangered species of wildlife. *New York Fish Game J.* 26(1):11-21.
- MARSH, R.E. 1977. Bromadiolone, a new anticoagulant rodenticide. *EPPO Bulletin* 7(2):495-502.
- MARTI, CD., P.W. WAGNER and K.W. DENNE. 1979. Nest boxes for the management of barn owls. *Wildl. Soc. Bull.* 7(3):145-148.
- MCKELVIE, D.H. and A.C. ANDERSON. 1963. Neonatal deaths in relation to the total number production of experimental beagles to the weaning age. *Lab. Animal Care* 13:725.
- MEEHAN, A.P. 1978. Rodenticidal activity of bromadiolone--a new anticoagulant. Proc. 8th Vertebrate Pest Conf., Sacramento, California, pp. 122-126.
- MENDELSSOHN, H. and U. PAZ. 1977. Mass mortality of birds of prey caused by Azodrin, an organo phosphorus insecticide. *Biol. Conserv.* 11(3):163-170.
- MENDENHALL, V.M. and L.F. PANK. 1980. Secondary poisoning of owls by anticoagulant rodenticides. *Wildlife Soc. Bull.* 8(4):311-315.
- MOORE, N.W., ed. 1966. Pesticides in the environment and their effect on wildlife. *J. Appl. Ecol.* 3(Suppl.), 311 pp.
- MOORE, N.W. 1970. New factors controlling populations of land vertebrates in Great Britain. Proc. 4th Vertebrate Pest Conf., West Sacramento, California, pp. 9-14.
- NAS (NAT. ACAD. SCI. U.S.). 1979. Animals as monitors of environmental pollutants. NAS, Washington, 421 pp.
- NELSON, B.C. 1980. Plague studies in California--the roles of various species of sylvatic rodents in plague ecology in California. Proc. 9th Vertebrate Pest Conf., Fresno, California, pp. 89-96.
- OLIVER, A.J. and S.H. WHEELER. 1978. The toxicity of the anticoagulant pindone to the European rabbit, *Oryctolagus cuniculus* and the sheep, *Ovis aries*. *Aust. Wildl. Res.* 5:135-142.
- PANK, L.F. 1976. Effects of seed and background colors on seed acceptance by birds. *J. Wildl. Manage.* 40(4):769-774.
- PARK, B.K., J.B. LECK, A. WILSON and A.M. BRECKENRIDGE. 1980. Investigation of anticoagulants and vitamin K₁ in the rabbit. In: Suttie, J.W., ed., *Vitamin K Metabolism and Vitamin K-dependent Proteins*. Univ. Park Press, Baltimore, pp. 348-353.
- PEARSON, O.P. 1964. The prey of carnivores during one cycle of mouse abundance. *J. Animal Ecol.* 35:217-233.
- PIMENTEL, D. 1971. Ecological effects of pesticides on nontarget species. U.S. Pres. Offic Sci. Tech., Super. Doc, Washington, D.C., 220 pp.
- PRIER, R.F. and P.H. DERSE. 1962. Evaluation of the hazard of secondary poisoning by warfarin-poisoned rodents. *J. Amer. Vet. Med. Assoc.* 140(4):351-354.

- PROSSER, P.R. and J.H. KARAM. 1978. Diabetes mellitus following rodenticide [Vacor] ingestion in man. J. Amer. Med. Assoc. 239(12):1148-1150.
- RADVANYI, A. 1974. Small mammal census and control on a hardwood plantation. Proc. 6th Vertebrate Pest Conf., Anaheim, California, pp. 9-19.
- SAVARIE, P.J., D.J. HAYES, R.T. MCBRIDE and J.D. ROBERTS. 1979. Efficacy and safety of diphacinone as a predacide. In: E.E. Kenaga, ed., Avian and Mammalian Wildlife Toxicology, Amer. Soc. Test. Mater. Spec. Tech. Publ. 693:69-79.
- SULLIVAN, T.P. and D.S. SULLIVAN. 1980. The use of weasels for natural control of mouse and vole populations in a coastal coniferous forest. Oecologia 47:125-129.
- TOWNSEND, M.G., M.R. FLETCHER, E.M. ODAM, and P.I. STANLEY. 1981. An assessment of the secondary poisoning hazard of warfarin to Tawny Owls. J. Wildl. Manage. 45(1):242-248.
- TUCKER, R.K. and D.G. CRABTREE. 1970. Handbook of toxicity of pesticides to wildlife. USDI, Fish and Wildl. Serv. Res. Publ. 84, Super. Doc, Washington, 131 pp.
- VOIGHT, J. and D.C. GLENN-LEWIN. 1978. Prey availability and prey taken by long-eared owls in Iowa. Amer. Midl. Natur. 99(1): 162-171.
- WALKER, C.R. 1981. Reducing potential hazards of pesticides to fish, wildlife, and habitat. In: Schafer, E.W. Jr. and C.R. Walker, eds., Proc. 3rd Conf. Vertebrate Pest Control and Management materials, Amer. Soc. Test. Mater. Spec. Tech. Publ. 752:174-187.
- WHO. 1967. Joint FAO/WHO Expert Committee on Zoonoses, 3rd Symp. WHO Tech. Rept. Ser. 378, Geneva, 127 pp.
- WINTERINHAM, F.P.W. 1975. Fate and significance of chemical pesticides; an appraisal in the context of integrated control. EPP0 Bull. 5(2):65-71.
- WODZICKI, K. 1973. Prospects for biological control of rodent populations. Bull. World Health Organ. 48:461-467.