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C. Raymond Record

Summit Laboratories, Cedaredge, Colorado

Rex E. Marsh

Department of Wildlife and Fisheries Biology, University of California, Davis

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RODENTICIDE RESIDUES IN ANIMAL CARCASSES AND THEIR RELEVANCE TO SECONDARY HAZARDS

C. RAYMOND RECORD, Summit Laboratories, Cedaredge, Colorado 81413.

REX E. MARSH, Department of Wildlife and Fisheries Biology, University of California, Davis, California 95616.

ABSTRACT: Some complexities and limitations of using carcass residue data to determine secondary hazard to nontarget species are discussed. The roles of chemical and toxicological properties of the rodenticide such as metabolism, excretion, organs of retention, site of absorption and latent period in secondary hazard are reviewed and examples given. The possible effects of bait composition and application methods, the behavioral response of the nontarget species, and local environmental factors upon secondary hazard are outlined.

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The determination of secondary hazard is an important issue to those involved in vertebrate pest control today. Registrants of vertebrate pesticides are increasingly interested in and being required to provide data which permit some effective measurement, evaluation or quantification of such hazard. It is incumbent upon those of us involved in the process of collecting, analyzing and applying such data to take an active part in determining the most appropriate systems for the collection, interpretation and application of such data.

When the potential secondary hazard of rodenticide residues in the carcasses of dead target animals is being considered, a very simplistic model is often fashioned. Such a model typically involves two values: the amount of toxicant consumed by the target species and the LD₅₀ of that toxicant to the nontarget species of concern. From these two pieces of data, extrapolations are made to represent the relative hazard to the nontarget species. While it is to a certain extent understandable that one might wish to keep the model simple because it is frequently difficult to obtain even those two pieces of data, there are so many mitigating and complicating factors that a model thus constructed, without qualifications, is often of very little practical value in determining the potential nontarget species hazard of carcass residues.

Another method of determining secondary hazard is to conduct a field experiment under the circumstances one would expect under normal (natural) conditions. The philosophy here is that one does not need to fully understand all the ecological processes involved, just measure the results and extrapolate them to the existing universe of field situations. While such "real life" research is quite useful and highly regarded by the authors, it is still necessary to have some understanding of the nature and extent of the complicating factors to avoid over extrapolating the results to situations to which they may not be applicable. Quantification of secondary hazard from field data is a difficult task and attempts to do so may even be misleading. The mammalian or avian species suspected to be at risk of secondary hazard

are generally not present in large numbers since they are predators or scavengers and at or close to the top of the ecological pyramids. Therefore, extrapolations must generally be made on the basis of extremely small sample sizes, thus greatly increasing the possibility of drawing improper conclusions on the basis of what may be atypical results. This means that it becomes more important to understand some of the more significant complexities of the determining factors of secondary hazard so that such can be properly qualified and quantified. A cognizance of these complicating factors will result in more realistic extrapolations of data from one situation to another.

The major objective of this paper is to identify and elucidate some of the biological and ecological issues involved in the determination of secondary hazards. It is hoped that this will result in a greater appreciation of the complexities of the issues and will promote better and more realistic interpretations of data when assessing potential secondary hazards. Another value of such an exercise is that understanding the mechanisms of secondary hazard gives those involved in the development and implementation of control projects more insight as to the appropriate management methods, reducing potential hazards to nontarget species through the proper selection of toxicant, bait formulation, and application rates and methods. The paper is not intended to be an exhaustive review of the literature on the subject of carcass residue, but it will draw upon a number of research articles, mostly relating to rodents as the primary species, to illustrate the various major points.

There are four primary elements involved in determining whether rodenticide residues in the bodies of target species will pose a secondary hazard or potential secondary hazard to nontarget species. These are: 1) The chemical and toxicological properties of the toxicant; 2) The composition of the toxic bait and how it is applied; 3) The behavior of the nontarget species at risk; 4) Local environmental factors. These four elements, each of which may be complex unto itself, and the manner in which they interrelate, will deter-

mine the existence and extent of secondary hazard.

CHEMICAL AND TOXICOLOGICAL PROPERTIES

Chemical and toxicological properties of the toxicant are of major importance in ascertaining the likelihood of secondary hazard. Questions that must be answered in addition to the toxicity of the compound and how much has been consumed include: 1) What are the breakdown products, and are they toxic? 2) How rapidly is the toxicant broken down and/or excreted? 3) What are the organs or tissues of retention? 4) What is the latent period (time between ingestion and onset of symptoms and ingestion and death)? 5) What is the site and speed of absorption at different toxic loads?

Metabolism and Excretion

It is valuable to know the rate of metabolism of a compound and whether the breakdown products are toxic. If a toxicant is detoxified or rapidly excreted from the target animal once its damage is done, then hazard will only exist in the form of unmetabolized material. The rate of metabolism and/or excretion then becomes an important determining factor. A poison that is largely metabolized to nontoxic by products or excreted before death obviously poses little secondary hazard. Zinc phosphide is a good example of a rodenticide which breaks down relatively rapidly in the intestinal tract and presents little secondary hazard to predators (Hill and Carpenter 1982).

A poison that acts very quickly (short latent period), perhaps even killing the target species before all the bait material consumed has been assimilated and metabolized, might be potentially hazardous even if the metabolites are nontoxic.

Warfarin is a good example of a rodenticide that is largely metabolized or excreted before the death of the target animal. First-generation anticoagulants such as warfarin (Anderson 1967) and Fumarin have a relatively short half-life and are not usually present at high levels in the bodies of animals killed by them. This is because death is relatively slow and there is ample time for much of the toxicant to be metabolized and/or excreted.

Strychnine provides a contrasting example of a compound that is rapidly excreted when sublethal doses are ingested (Schwartz 1922) but still could pose a potential secondary hazard because of the rapidity of its toxic effects. Strychnine alkaloid is rapidly excreted from the animal's body, but it also causes death very rapidly. Therefore, depending upon the amount consumed and how fast the animal consumed the toxicant, a significant portion of toxicant ingested may remain in the gastrointestinal tract at the time of death. Anthony et al. (1984) reported that 99 percent of strychnine noted in poisoned ground squirrels was found in the gut.

Research by Laas et al. (1985) on the retention of brodifacoum in sheep tissues provides a type of data that can prove very useful in understanding the time-related attributes of metabolism and excretion. The researchers dosed a number of sheep with brodifacoum, then killed animals at

each of 2, 4, 8, 15, 32, 64 and 128 days posttreatment and analyzed selected tissues for residue. In addition, feces were collected from animals for 10 days after dosing to determine the rate of excretion. These data provide very useful information on the speed with which the residue left the body. If similar half-life type research was conducted with various rodenticides using the target species as test animals and a method of dosing comparable to real life situations, the resulting data would be very pertinent in the effort to determine potential secondary hazard.

Organs or Tissues of Retention

The organs or tissues of retention of the toxicant material in the bodies of the target species can be of great significance in determining some secondary hazards, especially with the larger primary species such as ground squirrels, prairie dogs and coyotes. There is a variety of retention patterns depending on the toxicant. Such differences must be considered when assessing potential secondary hazard, and it may have the prospect of being useful in the management of risk to nontarget predators and scavengers under some circumstances of exposure.

It has long been established that anticoagulant rodenticide residues tend to be found in the greatest amounts in the rodent's liver. Hoogenboom and Rammel (1983) reported that a great deal more (approximately ten times as much) brodifacoum residue was found in the liver as in the muscle tissue of sheep orally dosed with brodifacoum. The ratio between fat tissue and liver was of the same magnitude with slightly more residue in fat than in muscle. Research by Williams et al. (1986) also showed a high divergence between residues in liver tissue and the muscle and fat tissues of field-poisoned rabbits. Clearly scavenging or predatory animals would be in greater or lesser risk depending upon whether they feed on the livers of poisoned animals. This was early pointed out by Evans and Ward (1967) in their studies of secondary poisoning of mink and dogs from consuming anticoagulant-killed nutria where they fed nutria carcasses with and without the livers.

Chemical analysis of strychnine-poisoned animals also yields a wide disparity between the amount of strychnine residue in various body tissues, though for different biochemical reasons than those influencing the distribution of anticoagulants. The rapid toxicological action of strychnine (short time between ingestion and death) usually dictates that much of the toxicant ingested is concentrated in the gastrointestinal tract of the poisoned animals. For example, Anthony et al. (1984) reported that almost all of the strychnine found in field-killed golden-mantled ground squirrels (Spermophilus lateralis) was found in the gut.

The lack of strychnine dispersion into other tissues is of particular significance because there is other evidence that some predatory and scavenger species select against the gastrointestinal tract when feeding. Marsh et al. (1987) noted that captive coyotes (Canis latrans) rejected the stomachs or intestines of strychnine-poisoned ground squirrels significantly more frequently than those of squirrels poisoned with

Compound 1080. Those tissues and contents of strychnine-poisoned squirrels were rejected 65.6 percent of the time, and in 1080-poisoned squirrels these tissues were rejected only 25.5 percent of the time. This is thought to be related to the more detectable taste of strychnine, but may have involved some aversive conditioning. In another instance golden eagles (*Aquila chrysaetos*), after a field baiting project for Richardson ground squirrels (*Spermophilus richardsoni*) in Montana, were observed as they fed upon the squirrels killed by strychnine grain bait (Graham 1977). The eagles eviscerated numerous ground squirrels and fed on muscle tissue while leaving the stomach and entrails. No ill effects were seen in the eagles, and an intensive air and ground posttreatment search disclosed no dead or ill eagles.

The combination of the localization of the toxicant in the stomach and intestines, combined with the rejection of those tissues by some scavenging species, may provide a degree of protection against secondary hazard in strychnine baiting programs. If one were not aware of the feeding habits of the nontarget species potentially at risk and the distribution pattern of strychnine residue in the carcass, an overestimation of secondary hazard would almost certainly result. This has frequently been the case in the past.

Compound 1080 (sodium fluoroacetate) behaves somewhat differently than the aforementioned toxicants in terms of differential affinity for body tissues. Unlike the anticoagulants, there does not seem to be a tendency for it to concentrate to any great degree in a particular organ or tissue. It is able to disperse comparatively evenly throughout body tissues, although some concentration probably occurs in the stomach because of death occurring prior to total assimilation. Tissue levels of fluoroacetate noted in Ward (1985) were rather evenly distributed throughout eight types of black-tailed prairie dog (*Cynomys ludovicianus*) tissue analyzed. Muscle, stomach, liver, kidney, lung, caecum, brain, and spleen were included in the analysis. The toxicant was administered via oral gavage to animals that had been starved overnight. Interestingly, the lowest concentration of fluoroacetate of every animal was found in the liver, just the opposite of the case with anticoagulants.

Results that were different in one respect were given by Casper et al. (1986) in the analysis of tissues from California ground squirrels (*Spermophilus beecheyi*) poisoned with 1080. The same eight tissues were analyzed as in the Ward study. The chemical analysis was conducted by the same laboratory. In this study, the stomachs of the squirrels showed a much higher concentration of fluoroacetate than the other tissues.

The wide disparity between the results of the stomach analysis in the two studies may be explained by the difference in the way the toxicant was administered and the amount administered. Ward introduced the 1080 via oral gavage to prairie dogs which had been starved overnight. The researchers in the Casper experiment fed the squirrels 1080-treated oat groats. The amount of 1080 consumed by the squirrels was much greater than the amount with which the prairie dogs were gavaged. It appears that the gavaged animals were able

to assimilate the toxic load from the stomach more thoroughly (and possibly more quickly) than the animals which were fed the toxicant in grain bait form. This demonstrates a situation in which perfectly valid test results (e.g., the gavage data) might lead to an incorrect conclusion about nontarget species hazard of various tissues if one were not aware of possible complicating factors needed to correctly interpret the data. It would appear from these two tests that data from oral gavage testing should not be used exclusively to make determinations about relative organ or tissue retention properties for toxicants. This is especially true if these data were going to be used to extrapolate to field rodent control. Data from the test animals that were fed the toxic bait more closely mimic an actual field baiting situation and should prove more valuable in determinations of potential secondary hazard.

There may be a further complication in higher animals with a tendency and/or ability to vomit, thus evacuating their stomachs of much of the contents including the unabsorbed toxicant. In that case the tissue distribution of fluoroacetate or other residues may be more even because of the animal's self-evacuation of the stomach contents.

Site of Absorption

The site of absorption seems to vary for different toxicants. Strychnine, for example, is known as a "pouch poison" (Gabrielson 1932) because it is rapidly absorbed from the mucosal lining of the cheek pouches of species such as ground squirrels. Older references indicate that one-fifth of the amount of strychnine needed to produce death through stomach absorption will kill a squirrel if it is absorbed through the cheek pouches. Record (1978) cites field evidence that the tendency to carry grain in cheek pouches may be an important factor in the relative success of strychnine grain baits for Richardson ground squirrel control. It is also generally known that in some areas strychnine grain baits are most effective on the California ground squirrel during the time of year when the squirrels are pouching grain.

Latent Period

The latent period or time between ingestion and onset of symptoms (also sometimes defined as time between ingestion and death) has potentially important implications for nontarget species hazard. The influence of short latent period (time to death) upon stomach residues was already mentioned in the discussions of organ retention and metabolism and excretion. Another important factor to be considered is that the longer the latent period, the more time will be available for the target species to continue consuming bait, thus potentially increasing the residue. The consumption could possibly be limited, however, by reducing the concentration of toxicant in the bait and or limiting the amount of bait offered.

Time to death is important for several reasons. It may lower residue levels and it permits time for animals to seek cover when they feel ill. Another implication of a long latent period is that animals which have consumed rodenticides and

contain the toxic residues will remain alive longer, making them available to predatory species which may prefer killing live prey rather than eating carrion. This possible hazard must, however, be balanced against the fact that this long delay also allows time for rodenticides to be metabolized and excreted, thus decreasing the eventual residues in the dead animal's carcass.

It is clear that short or long latent periods each can have beneficial or detrimental effects on potential secondary hazard depending upon other biological and environmental aspects of the toxicant.

BAIT COMPOSITION AND APPLICATION

The composition of the bait can have a major influence on the toxic load of the bodies of the target species. Obviously the concentration of toxicant in the bait may have an influence. There is now a major data requirement for registrants of strychnine and 1080 to develop data demonstrating the lowest effective dose for field rodent baits in order to reduce the hazard, both primary and secondary, to non-target species.

The expected effect of decreasing the amount of toxicant in the bait would be to decrease the amount of toxicant in the bodies of the target species killed. A study was conducted using caged California ground squirrels (*Spermophilus beecheyi*) that were fed either 0.8 or 4.8 mg/kg of Compound 1080 baits. The fluoroacetate concentration in the tissues of the squirrels varied from 182 to 11,765 ppb in the ones given the 0.8 mg/kg dose and from 535 to 55,864 ppb in those given the higher dose (Casper et al. 1986). In other research, reducing the concentration of brodifacoum bait from 50 to 10 ppm reduced the residue in laboratory-killed voles from 5.21 and 2.17 ppm to 0.53 and 0.40 ppm in males and females, respectively (Kaukeinen 1982). In a study with birds, Schaefer (1986) found that a reduction from 0.6 to 0.4 percent active ingredients in the concentration of strychnine baits led to a 51 to 63 percent reduction of residues in the bodies of pigeons killed by the bait.

The application method could have a significant influence on the amount of residue present in the bodies of the target animals killed. "Spot" or "hole" baiting involves putting bait in concentrated placements at or near the animals' burrow entrance. Typically between one teaspoon and one tablespoon is applied at each placement for ground squirrels or prairie dogs. Each of these placements must have sufficient bait to provide a lethal dose for more than one target animal because several may be living in that burrow.

Contrasted with this is the broadcast method of bait application where bait is scattered mechanically over the immediate area where the rodent burrows are found. Such broadcasting may be accomplished by the use of seed spreaders (e.g., Cyclone Seeder) or by aircraft.

The theoretical advantage of broadcast application in reducing residue in the dead target animals is that multiple doses are not available in a single placement as in spot baiting. This might potentially limit the consumption of bait by target animals to about a single lethal dose with less chance

of overkill. The theoretical disadvantage of broadcast application is that somewhat more bait is applied overall (on a per acre or hectare basis). Furthermore, if the target species' foraging ability is so great that scattering the bait does not significantly slow its consumption, then there would be no nontarget safeguard advantage to broadcasting. This is an area where further research is needed to determine which method of application yields the lower amount of residue in the bodies of target species.

The rate of application (pounds per acre or amount per spot placement) also is likely to have an influence on the residue in the target animals. This again assumes that the amount consumed is physically limited by the amount of bait the animal can find. If, however, there were some other overriding limiting factor on consumption (e.g., stomach capacity, alternative food source, etc.), then application rate would become less important in determining secondary hazard.

Prebaiting, offering untreated bait material to the target population to accustom them to the new "food source" prior to applying toxic bait, will probably increase the potential for secondary hazard. The intent of prebaiting is to increase the amount and/or speed of bait consumption by the target species, thereby improving control. An increase in either of these factors has the potential for increasing the body residues of the toxicant. This possibility should be weighed, considered, and perhaps researched when contemplating the requirement of prebaiting. There may be no advantage, for example, in reducing the concentration of a bait in an attempt to reduce secondary hazard if prebaiting is then required to achieve adequate control of the target species.

BEHAVIORAL RESPONSE OF THE NONTARGET SPECIES

The hazard of a rodenticide is determined by two factors: toxicity and exposure. Regardless of how toxic a carcass might be, if a particular nontarget species does not frequent the area where the carcasses are, no hazard exists. For example, if the target rodents occur in cultivated valleys and the nontarget scavenger of concern lives only in heavily wooded mountains, then the animal is not at risk.

A variation of this exposure limitation on hazard occurs on a smaller scale as well. If the target species die underground and the potential scavenger does not or cannot dig for its food, then no hazard exists to that species. One of the primary determinants of whether the target species dies above or below ground is probably the speed with which the toxicant acts.

The food habits and behavioral response of the nontarget species presumably at risk are of paramount importance in determining that risk potential. Questions that must be asked include: 1) What nontarget species normally feed on the target species? 2) Is the target species consumed whole or are certain parts of the body selected for or against? 3) Where are the residues concentrated in the body of the target species? 4) How many (much) of the target species is the nontarget species likely to consume? 5) Is the residue present in that

amount of target carcass(es) sufficient to produce lethal or debilitating nonlethal effects in the nontarget animals?

If the nontarget species does not consume carrion but prefers live prey, then the secondary hazard is limited to the predation that might occur after the bait is consumed and before the animals die.

There appears to be a significant amount of tissue selectivity demonstrated by some predatory/scavenging species when feeding on carcasses the size of ground squirrels or larger. Two instances of this phenomenon were noted in the previous discussion on chemical and toxicological properties (See Graham (1977) and Marsh (1987)).

The likelihood of a carcass being fed upon by predatory or scavenging species present is a critical determinant in the matter of secondary hazard. More research of the type conducted by Sullivan et al. (1986) is needed to help quantify that likelihood. In his research, Sullivan attached radio transmitters to Columbian ground squirrel (Spermophilus Columbianus) carcasses, placed them in situations simulating those expected in field rodent control operations and monitored their fate. This provided useful data on the identity of scavenging species and their habits. By weighing carcasses and noting their condition at intervals during the test, the researchers were able to judge the length of time during which the carcasses of the squirrels were desirable to those scavenging species under that set of environmental conditions. The combination of these sorts of data with carcass residue data could provide a reasonable quantification of secondary hazard in many instances.

Research by Hegdal et al. (1984) provides an example of a situation in which an increased understanding of the nontarget species' behavior was necessary in order to properly determine the nontarget species hazard. The tracking of radioed barn owls (Tyto alba) and analysis of their regurgitated pellets showed that their diets included very little of the commensal rodent species that were being controlled with toxic bait even though the owls were closely associated with the areas of human activity in which commensal rodents lived. The owls apparently rejected feeding upon the commensal species in favor of meadow voles even though some of the owls nested in and around buildings where the house mice and rats lived. Behavioral information such as this has dispelled the assumption that poisoned commensal rodents would pose a major hazard to barn owls.

LOCAL ENVIRONMENTAL FACTORS

Local environmental factors could have major influence on the consumption of bait by the target animals. The amount and desirability of natural foods present is a major determinant in the acceptance of toxic bait by rodents. Frequently, availability of fresh green forage so limits the consumption of bait that it is impossible to achieve adequate control of the target rodents. Therefore an abundance of locally available foods would tend to decrease the intake of baits; and even if death resulted in the target animals, the toxic residues would probably be lower than if there had been no competing food source.

The weather also has the potential to affect the consumption of baits by target species. Adverse weather (e.g., rain, snow or wind) that interrupts feeding on a bait will almost certainly reduce the total intake of bait. Some toxicants are dependant upon rapid consumption of a lethal dose (e.g., strychnine) and any interruption may allow time for the onset of toxicosis to occur and then feeding to cease. This will likely result in bait shyness (refusal to eat more bait) in the animals. Therefore residue in the carcasses of those dying would be limited.

CONCLUSION

The need for viable data on nontarget species secondary hazard is increasing. There is also a continued need for information that will assist professionals in the design of baits and baiting programs that will achieve the desired efficacy while mitigating nontarget species hazard. Some data are beginning to be developed on the residues of rodenticides in the bodies of target species. Proper determination and management of secondary hazard requires a more complete understanding of the multiplicity of factors that influence hazard.

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