January 2007

ASSESSING RODENTICIDE HAZARDS: IMPROVING THE ART AND SCIENCE OF RISK ASSESSMENT

John J. Johnston
USDA, APHIS, Wildlife Services, National Wildlife Research Center

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JOHN J. JOHNSTON, USDA, APHIS, Wildlife Services, National Wildlife Research Center, Fort Collins, CO, USA

Abstract: Non-target hazards represent the most significant hurdle to the continued and possibly expanded use of anticoagulant rodenticides. In addition to the possibility of non-target access to the rodenticide bait, non-target scavenger and/or predator species may be exposed to these rodenticides via feeding on the carcasses of poisoned target species. Risk assessments provide a means to estimate the probability of rodenticide associated effects to target and non-target species. Quantification of risk provides critical information for decision-makers to weigh the benefits versus the risks of proposed rodenticide uses. This manuscript reports on the development of a probabilistic risk assessment model for quantifying efficacy and/or adverse effects to target and non-target species, respectively. This risk assessment approach can also be used to identify pesticide use strategies (formulations, bating practices) which minimize non-target secondary risks yet are efficacious.

Key words: adverse effects, hazard, probabilistic, risk assessment, rodent, rodenticide

INTRODUCTION

To successfully register a pesticide for use in the United States, two criteria must be demonstrated: acceptable efficacy for the target species and the level of risks to non-target species. The level of concern for efficacy is seventy percent; for a toxicant, the proposed use must demonstrate a minimum of seventy percent reduction in the target population.

For risks to non-target species, the proposed use must demonstrate minimal adverse risk to non-target species. Pesticide induced adverse incidents to wildlife are difficult to quantify under "real world" conditions. Due to the mobility of many wildlife species (especially birds), pesticide exposed wildlife may travel significant distances from the site of exposure prior to the onset of adverse effects. Additionally, pesticide induced wildlife mortality may be difficult to observe because carcasses may be scavenged before they are "discovered" by humans. Scenarios such as these may lead to an underestimation of pesticide induced adverse effects. On the other hand, detection of pesticide residues in wildlife carcasses frequently lead to the assumption that pesticide exposure induced or contributed to mortality. This assumption can lead to an overestimation of adverse effects as it is likely that pesticide exposure does not induce or increase the probability mortality for some of these wildlife mortality incidents.

The most simple way to demonstrate non-target risk is to calculate risk quotients (dietary pesticide concentration/median lethal dietary pesticide concentration (LC_{50}) or pesticide dose/median lethal dose (LD_{50}). The level of concern for risk quotients is 0.1 for threatened or endangered species and 0.5

for all other species. Unfortunately, risk quotients are overly simplistic. Risk quotients ignore the fact that different species may share a common median lethality value, yet demonstrate significantly different lethality values for metrics other than the median because of differences in the slope of each species' dose versus mortality curve. To improve the accuracy of the risk assessments for evaluating proposed pesticide uses to control damage induced by pest wildlife species, I have developed a probabilistic computer model risk assessment approach based on the dose versus response relationship.

MORTALITY ESTIMATION

Mortality is a function of exposure and sensitivity. As exposure and/or sensitivity increases, the probability of adverse effects also increases. I quantify the probability of mortality by developing a log exposure (dose or dietary concentration) versus probit mortality relationship. Estimated exposure is subsequently regressed against these values to generate an estimate of mortality.

There are two common sources of the toxicity information required to generate the dose versus mortality curves: dietary toxicity studies and acute toxicity studies. In dietary toxicity studies, each treatment group of test animals is fed a diet containing a different concentration of the toxicant of interest. The test animals are generally fed the treatment diets for five days. This five day exposure period is followed by a five day observation period. At the end of the observation period, the percent mortality for each treatment group is calculated and used to generate the dietary concentration versus mortality relationship. Acute toxicity studies follow a similar format except that the animals in each treatment group are administered a single dose of the toxicant of interest. While the animals in each treatment group receive an identical dose, the target dose varies between treatment groups.

EXPOSURE ESTIMATION

Exposure estimates focus on primary and secondary routes of exposure (Figure 1). Primary exposure results from the consumption of the pesticide bait (formulation) by the target or non-target species of interest. Secondary exposure results from the consumption of animals (frequently the target species) which contain residues of the pesticide. Estimation of exposure for either the primary or secondary scenario requires knowledge of the pesticide concentration in the food source and the fraction of the diet constituted by that food source. To obtain exposure as a dose, the total amount of food consumed by the species of interest must also be estimated. Equation 1: Dietary concentration of pesticide ($\mu g/g$) = pesticide concentration in food source ($\mu g/g$) x percentage of food source in the diet
Equation 2: Dose (μg) = dietary concentration of pesticide ($\mu g/g$) x mass of diet (g)
The estimation of the percentage of the food source in the diet is frequently determined by necropsy experiments or observations of wild species. The mass of the diet is frequently determined by a bioenergetic approach which assumes that the mass of the diet is governed by caloric (energy) needs of the individual (Nagy 1999).
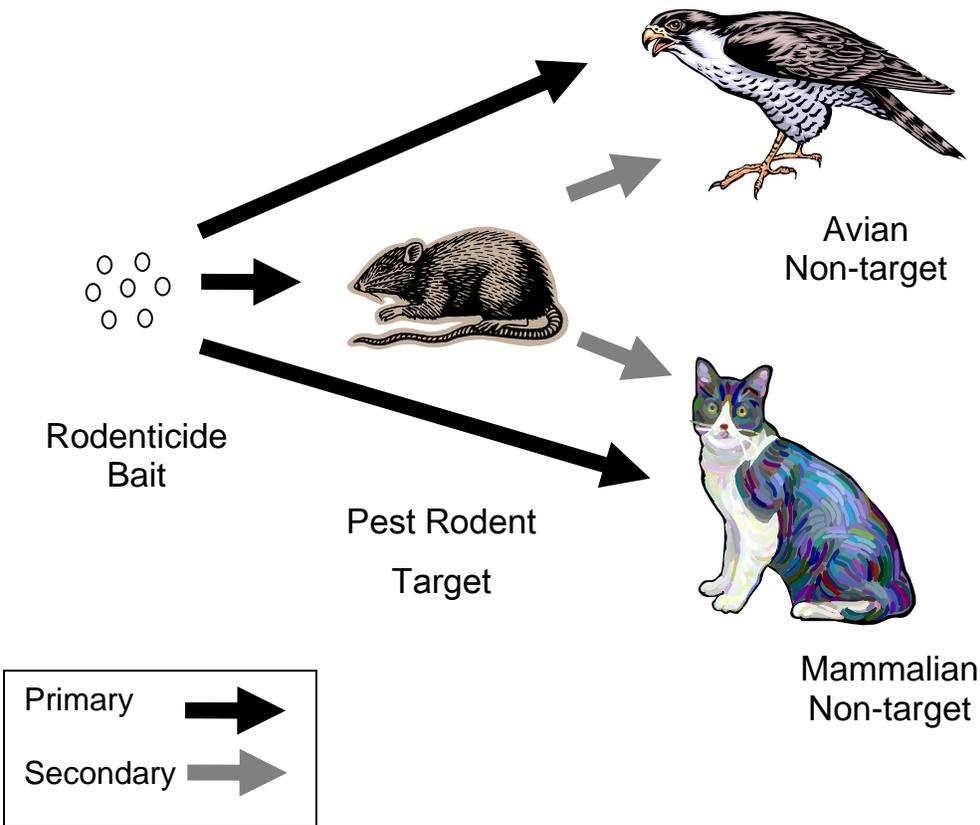
Figure 1. Primary and secondary routes of rodenticide exposure.

VARIABILITY

Regressing a single exposure estimate (e.g., mean or 90th percentile) against the best fitting (average) linear regression for the exposure versus mortality relationship yields a deterministic (single value) estimate of mortality. Obviously, such an estimate will be observed only a small percentage of time under field or even laboratory experiments. Actual results vary from the estimate due to variability in exposure and sensitivity between individuals in the study populations. I utilize a probabilistic approach to incorporate this variability into the risk assessment process (Johnston et al. 2005a, Johnston et al. 2005b, Johnston et al. 2006).

PROBABILISTIC RISK ESTIMATION

The probabilistic approach utilizes the full range of exposure and toxicity observations to yield outputs that include the probabilities associated with the entire range of percent mortality values. For toxicity estimation, distributions of slopes and LD_{50} or LC_{50} values are constructed to permit construction of dose versus response relationships for each individual in the population. Similarly, for exposure estimation, distributions of dietary pesticide concentration are constructed for the fraction of the diet that is constituted by the pesticide containing bait (primary exposure) or target species' carcasses (secondary exposure). For dietary dose estimations of the amount of food consumed, distributions

of energy requirements for the species of interest and energy content of dietary constituents are constructed (Nagy 1999, Johnston et al. 2005b).

For each individual, exposure (dietary concentration of the pesticide) is estimated by multiplying a Monte Carlo sampled value from the dietary fraction of target species (or bait) distribution and the pesticide concentration distribution. To determine dose, the estimated dietary pesticide concentration is multiplied by the mass of the diet (Figure 2). This individual exposure estimate is regressed against a unique exposure versus mortality linear relationship (constructed by Monte Carlo sampling of a value from the LD_{50} and slope distributions) to yield the probability of mortality for that individual. Each iteration of the model estimates the probability of mortality for an individual in the exposure scenario of interest. By assembling the mortality estimates for a large number of iterations (e.g., 10,000 – 100,000), a distribution of predicted mortalities and associated probabilities can be constructed for the proposed pesticide use scenario.
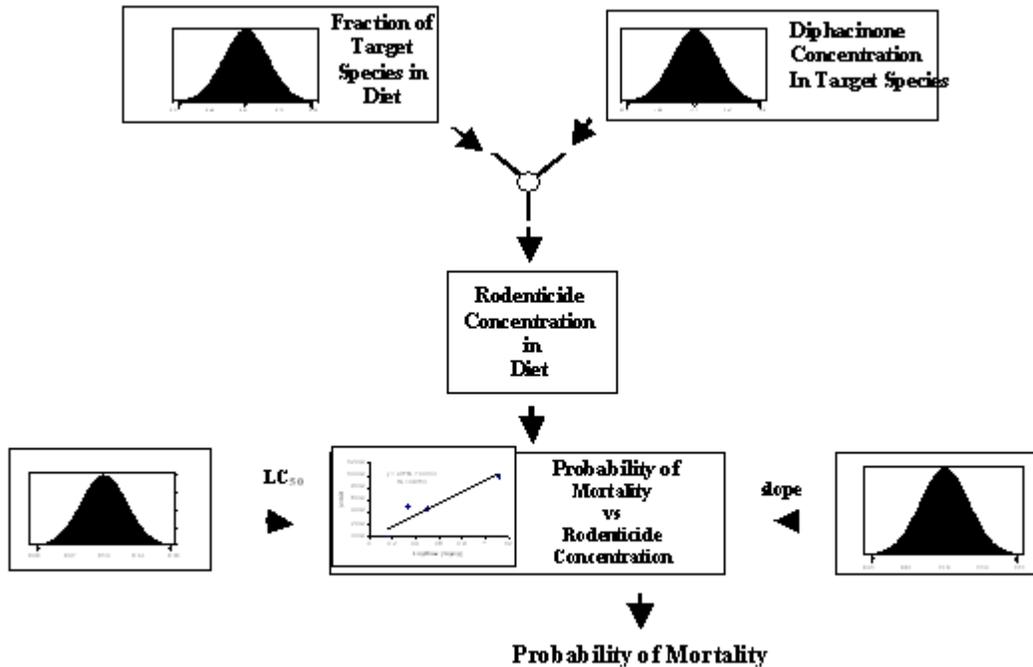

Figure 2. Probabilistic computer model for estimation of adverse effects.

CONCLUSION

Analysis of the distribution of predicted mortalities and associated probabilities can yield a variety of mortality estimates (mean, upper 95th percentile, upper 99th percentile, range) which provide useful predictions for risk managers. The utility of such estimates can be maximized by incorporating these estimates into population models for the species of interest. Sensitivity analysis can identify the input variables which have the greatest impact on mortality. Identification of these variables can prove extremely valuable in providing risk managers direction for developing acceptable mitigation procedures to develop effective pesticide use scenarios with minimal non-target impacts.

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