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PROBABILISTIC BIOENERGETIC/ TOXICITY MODELING APPROACH FOR ESTIMATING TOXICANT INDUCED MORTALITY TO TARGET INVASIVE SPECIES AND NON-TARGET WILDLIFE

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PROBABILISTIC BIOENERGETIC/TOXICITY MODELING APPROACH FOR ESTIMATING TOXICANT INDUCED MORTALITY TO TARGET INVASIVE SPECIES AND NON-TARGET WILDLIFE

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Abstract: Non-target species may be exposed to rodenticides via feeding on rodenticide baits or the carcasses of poisoned target species. As invasive species frequently negatively impact threatened or endangered (T and E) species, there is frequently spatial and temporal overlap of invasive species and T and E species. Risk assessments provide a means to estimate the probability of rodenticide associated adverse effects to non-target species (including T and E species). Quantification of risk provides critical information for decision-makers to weigh the benefits and risks of proposed rodenticide uses and to compare the risks of management with risks associated with no management (e.g. invasive species induced extinction of native species).

Key Words: bioenergetic, invasive species, modeling, non-target species, probabilistic, starling, target, toxicity.

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INTRODUCTION

Invasive species are non-native species whose introduction causes harm to the economy, environment and/or human health. In the United States, invasive species cause an estimated \$120 billion in annual damage to agriculture and the environment. More than forty percent of threatened or endangered species are at risk because of invasive species (Pimentel et al. 2005). Lack of an effective invasive species management plan may lead to increased rates of extinction for native species. Chemical control is a highly effective means of reducing invasive species populations. However, the risks associated with chemical control of invasive species include potential contamination of the environment and/or potential mortality of non-target plant and animal species. Due to the temporal and/or spatial overlap of threatened or endangered species with targeted invasive species, it is imperative that the probability of adverse effects to non-target species (especially threatened or endangered species) be determined and minimized prior to the application of the toxicant. In response to this invasive species management need, we developed a quantitative approach to estimate the magnitude of toxicant

induced effects (e.g., mortality) to wildlife populations of interest.

MORTALITY ESTIMATION

Mortality is a function of exposure and a species' (or individual's) sensitivity to a toxicant. We developed probabilistic approaches to estimate exposure and sensitivity. After characterizing these two attributes, we can estimate the probability of mortality induced by a magnitude of exposure (Figure 1) (Johnston et al. 2005a, Johnston et al. 2005b, Homan et al. 2006).

Exposure

Wildlife exposure to toxicants is often characterized as primary or secondary exposure. Primary exposure results from an animal consuming the toxicant formulation (bait). Secondary exposure results from an animal (typically a scavenger or predator) consuming another animal (or carcass) that contains residues of the toxicant. In either case, exposure is estimated by multiplying the quantity of food (or bait) consumed by the concentration of the toxicant in the food. The concentration of the toxicant in the food or bait can be determined by analytical

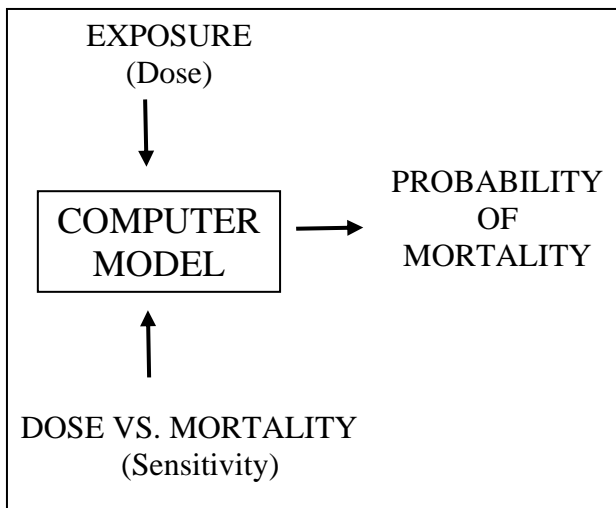


Figure 1. Computer modeling approach to estimate the probability of mortality based on exposure and toxicant sensitivity.

chemistry analyses. Such analyses are routinely conducted by the staff at the National Wildlife Research Center’s Analytical Chemistry Laboratory in Fort Collins, Colorado.

We use a bioenergetic approach for estimating the quantity of toxicant consumed by deriving metabolic energy requirements of the target/nontarget species then dividing by the energy content of the food or bait. This approach estimates the amount of food or bait needed to satisfy the animal’s daily metabolic needs, which are a function of species, activity level, body weight and environmental conditions, such as air temperature, humidity and wind velocity. The bioenergetic approach is applicable to a wide variety of species and geographic areas. It is often combined with or validated by empirical observations. Such observations may include bait consumption rates, bait feeding intervals, necropsy analyses of birds to determine the percentage or amount of a particular

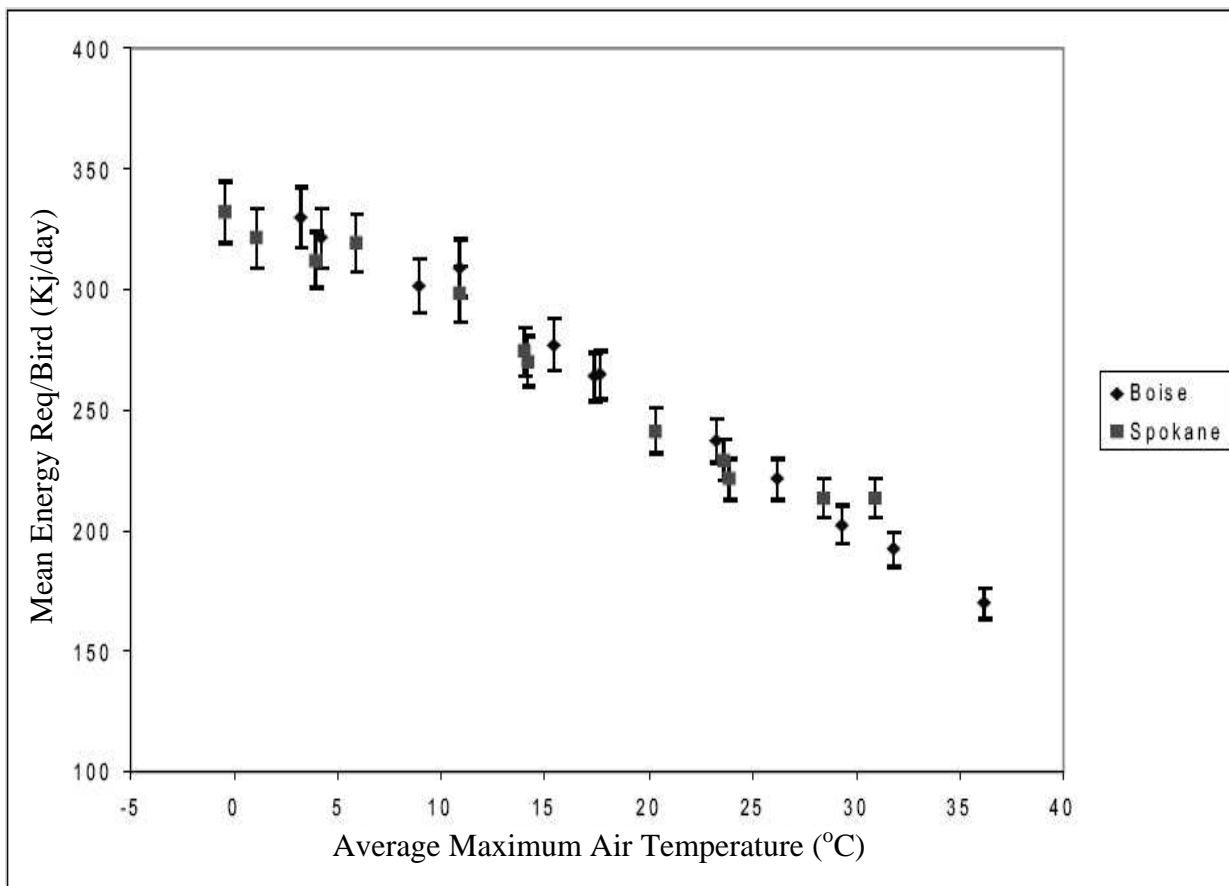


Figure 2. The effect of temperature on starlings’ energy requirements in Boise, ID and Spokane, WA.

food source consumed and/or the amount of bait left on a plot after a feeding interval. The effect of temperature on starlings' energy requirements is summarized in Figure 2 (Stahl, unpublished data).

Sensitivity Estimation

The sensitivity of a species or individual animal can be characterized with a dose versus mortality relationship. This relationship is typically generated with animal experiments where groups of animals are dosed with the toxicant of interest. Each group receives the same dose and the resulting percentage mortality for each group is subsequently determined. The best fitting curve is then estimated by plotting dose versus percent mortality (Figure 3).

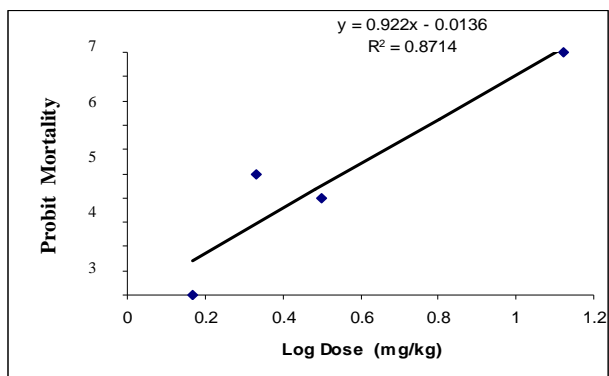


Figure 3. Dose versus mortality relationship is indicative of a species sensitivity to a toxicant.

Mortality Estimation

The probability of mortality associated with any subsequent exposure can be estimated by regressing exposure (dose) against the dose versus mortality curve. The resulting estimate will be the predicted mean mortality for the dose of interest (Figure 1).

PROBABILISTIC APPROACH

Individuals vary with respect to their sensitivity to a toxicant and their metabolic energy needs. Such variation can be attributed to a variety of factors including inter-individual variation in absorption and metabolism. The inter-individual variation (standard deviation) associated with the mean energy needs for a species can be estimated by the approach of Nagy et al. (1999). With respect to toxicant sensitivity, the result of this variation is illustrated in the magnitude of the residuals, the difference between the data points and the best fitting curve, in the dose versus mortality curve (Figure 3). The standard deviation associated with

the mean LD50 and slope of the dose versus mortality curve can be estimated using the classic probit analysis approach of Finney (1971).

To capture the individual variation in exposure and sensitivity, we utilized the mean and standard deviation values to construct normal distributions of energy requirements, LD50s and dose versus mortality slopes. Probabilistic sampling (the probability of selecting individual values from these distributions are based on the frequency of each value in the distribution) for each individual in the population of interest, permitted us to construct a theoretical population which encompasses the range of exposures and toxicant sensitivities anticipated in the exposed population. Each iteration of our model represented an individual in the potentially exposed population (Figure 4). Based on the unique exposure and sensitivity assigned to each individual, the probability of mortality for each individual was estimated. The frequency of each mortality value is summarized in a reverse cumulative frequency plot (Figure 5).

RISK MANAGEMENT

From the reverse cumulative plot, we can estimate mean mortality as well as confidence intervals associated with the estimates. These values are extremely valuable to risk managers. For example, a risk manager might use the lower 95th percentile mortality estimate to conservatively estimate the predicted treatment impact on the target species, while using upper 95th or 99th percentile mortality estimates to conservatively estimate the potential impact on non-target species. Linking these estimates to population models can produce a scientifically based approach for identifying chemical based invasive species management strategies with acceptable, minimal impacts on non-target species.

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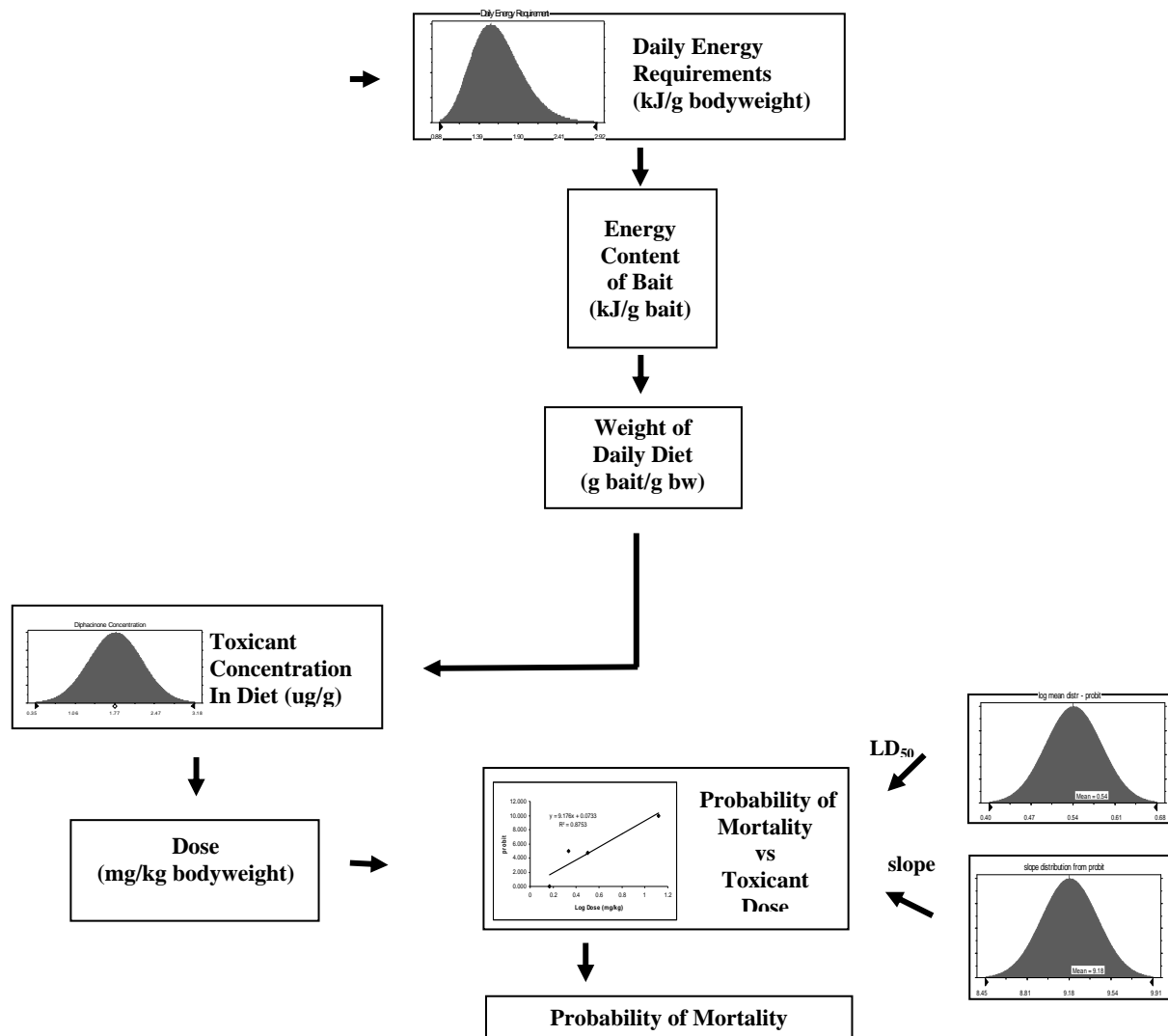
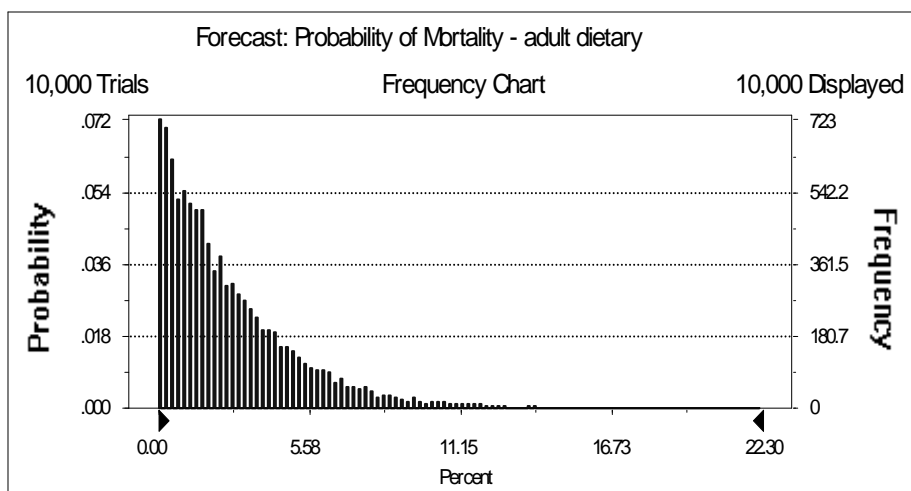


Figure 4. Computer model flow chart.



Mean Mortality = 2.8%; 95% Confidence Interval = 0.24 – 11.9%

Figure 5. Model output: reverse cumulative probability of mortality frequency plot.

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