PROJECT REPORT

Project Title: PHARMACOKINETIC STUDIES WITH KESTRELS AND OWLS FOR VALIDATING THE CDFA/USDA RODENTICIDE PBPK MODEL

Research Agency: National Wildlife Research Center

Principal Investigator: John Johnston

Budget: $247,585.00

Background:

The goal of this research is to validate the CDFA/USDA rodenticide Physiologically Based Pharmacokinetic Model on wildlife species which may be exposed to rodenticides via secondary exposure. This will increase the scientific validity and acceptance of the PBPK model and its subsequent applications.

Rodents pose significant public health risks, and are major ecological and agricultural pests. Rodents vector diseases such as hanta virus, plague, tularemia and rat bite fever (CDC, 2006; Merck 2006). Rodents, especially rats, contribute to the extinction of native flora and flora in numerous locals (Atkinson, 1977). Burrowing rodents cause structural damage to earthen dams and irrigation ditches (Hegdal and Harbour,1991). Finally, rodents cause significant damage to a variety of crops and rangeland grasses for livestock grazing (Primus et al., 2000).

The control of rodent pests (rats, mice, ground squirrels) in both urban and rural environments relies primarily on the use of rodenticides (Johnston et al, 2005). For example, in California, application of 0.01% and 0.005% diphacinone steam rolled oat baits are essential for control of ground squirrel induced damage to crops and rangeland grasses. Unfortunately, non-target scavenging wildlife such as raptors can be exposed to the rodenticides by feeding on the carcasses of poisoned pest rodent species (Fig 1.). Regardless of the benefits of rodenticide use, non-target secondary hazards represent the greatest hurdle to the expanded use and even the continued availability of anticoagulant rodenticides in the United States. Purported incidents of anticoagulant poisoned raptors are reported in newspapers, the scientific literature and EPA adverse incidents database (6(a)(2)). These incidents effect EPA regulations regarding the continued availability of anticoagulant rodenticides.

EPA also considers potential exposure and sensitivity of non-target species when reviewing pesticide use requests. In the absence of toxicity data (LD_{50}) for threatened or endangered species, EPA typically prohibits the use of rodenticides in areas where such species may be

exposed. In the absence of toxicity data (LD_{50}) for other potentially exposed species, EPA conservatively uses the LD_{50} from the most sensitive species in the same class or phylum as the potentially exposed species of concern. Additionally, since the concentration of the rodenticide in the various organs of the poisoned rodent is unknown, EPA generally assumes that the diet of the potentially exposed species consists entirely of the liver (the tissue with the highest rodenticide residue concentration) of the rodent. EPA's approach likely overestimates the risk of non-target poisoning from anti-coagulant rodenticides.

Objectives:

1. Determine the toxicity of diphacinone to American Kestrels and Eastern Screech Owls and compare to northern bobwhite quail (avian species used to develop the PBPK model)
2. Determine diphacinone residues in kestrel and owl tissues and the relationship between tissue residues and exposure, blood clotting time and acute toxicity
3. Extrapolate PBPK model developed with northern bobwhite quail to kestrels and owls.

Progress To Date:

To Be Updated.