

Project ID: **60037**

Project Title: **Estimation of Potential Population Level Effects of Contaminants on Wildlife**

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June 4, 1999

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DOE Report No.:

Publication Date:

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¹ managed by Lockheed Martin Energy Research Corp. for the U.S. Department of Energy under contract number DE-AC05-96OR22464

Research Objective: The objective of this project is to provide DOE with improved methods to assess risks from contaminants to wildlife populations. The current approach for wildlife risk assessment consists of comparison of contaminant exposure estimates for individual animals to literature-derived toxicity test endpoints. These test endpoints are assumed to estimate thresholds for population-level effects. For several reasons, uncertainties associated with this approach are considerable. First, because toxicity data are not available for most potential wildlife endpoint species, extrapolation of toxicity data from test species to the species of interest is required. There is no consensus on the most appropriate extrapolation method. Second, toxicity data are represented as statistical measures (e.g., NOAELs or LOAELs) that provide no information on the nature or magnitude of effects. The level of effect is an artifact of the replication and dosing regime employed, and does not indicate how effects might increase with increasing exposure. Consequently, slight exceedance of a LOAEL is not distinguished from greatly exceeding it. Third, the relationship of toxic effects on individuals to effects on populations is poorly estimated by existing methods. It is assumed that if the exposure of individuals exceeds levels associated with impaired reproduction, then population level effects are likely. Uncertainty associated with this assumption is large because depending on the reproductive strategy of a given species, comparable levels of reproductive impairment may result in dramatically different population-level responses. We are working on several tasks to address these problems: 1) investigation of the validity of the current allometric scaling approach for interspecies extrapolation and development of new scaling models; 2) development of dose-response models for toxicity data presented in the literature; and 3) development of matrix-based population models that, coupled with dose-response models, will allow for realistic estimation of population-level effects for individual responses.

Uncertainties associated with the current approach to wildlife risk assessment may have direct impacts on DOE EM satisfactorily fulfilling its mission in two ways. First, risk estimates may be too conservative and therefore remediation may be recommended when it is not needed. Limited remediation funds may be spent for insignificant or non-existent risks and possibly cause a net increase in environmental damage due to unnecessary habitat destruction. Second, risk estimates may not be adequately protective and therefore remedial actions may not be recommended when they are needed. The consequences of this uncertainty is environmental damage and potential NRDA liability. Either of these alternatives results in inefficient use of limited EM funds. This project will provide the tools to better estimate population-level effects and therefore reduce uncertainty associated with wildlife risk assessments.

Research Progress and Implications: This project is being performed in four, interrelated tasks. Progress on each task is outlined below.

1. Development of a database of avian and mammalian toxicity data. Literature search and acquisition of acute toxicity data for birds and mammals is essentially complete. Data sources include the extensive

wildlife toxicity database from the Denver Wildlife Research Center, and the National Institute of Occupational Safety and Health (NIOSH) Registry of Toxic Effects of Chemical Substances (RTECS) database. Additional literature search and acquisition of chronic toxicity data for applications of the dose-response models in Task 2a (see below) is ongoing.

2a. Development of Dose-Response models. We conducted a literature search to define modeling approaches currently being applied for human health risk assessment. Based on the results of this search, we adopted a modeling approach that is comparable to that employed by the USEPA for human health risk assessment. A two to four parameter logistic model (number of parameters is determined by the attributes of the dose-response data) is fit to literature-derived toxicity data. The resulting model was then used to define the dose level (and 95% confidence limits) that corresponds with selected effect levels. The preliminary dose-response model development for a limited number of chemicals was completed in FY 1998. This work is being expanded to include a greater number of chemicals, representative of the range of dose-response relationships.

2b. Development of improved methods for interspecies toxicity extrapolation. As reported in the 1998 Annual Report, avian and mammalian acute toxicity data were used to test linear regression models for lethal toxicity of over 200 chemicals. In our previously reported preliminary work, body weight scaling factors (e.g., regression slopes) were found to vary widely across chemicals for both birds and mammals. Our subsequent work has determined that, for most chemicals, body weight accounted for much variability in acute toxicity. We found that many chemical-specific scaling factors did not differ significantly from conventional scaling factors of 0.66, 0.75, and 1 and most did not differ from a factor of 1. We also found that among chemicals for which adequate data were available to compare models for both birds and mammals, approximately 2/3 showed no significant difference between models.

Studies which included alkaloids, inorganics, organochlorines, and drugs, were used to derive our mean avian scaling factor of 1.2. This value is similar to scaling factors previously reported for organophosphates, carbamates, and several miscellaneous pesticides, suggesting that a scaling factor of 1.2 may be appropriate for avian inter-species extrapolations for many chemicals. We found no papers with scaling information specific to mammalian wildlife, however our results suggest that scaling factors of 0.66 or 0.75, while suitable for anti-cancer drugs, are not broadly applicable to all chemical classes. Our results indicate that on average, a scaling factor of 0.94 is most appropriate for mammals.

Despite preliminary results which indicate that birds and mammals have similar scaling factors for a majority of the chemicals evaluated, we found no clear pattern for differences, either on the basis of chemical categories, taxonomic group, or mode of action. Our results, therefore, do not support extrapolations between birds and mammals. In the absence of a chemical-specific scaling factor, scaling factors of 1.2 and 0.94 should be used for birds and mammals, respectively. These scaling factors are most appropriate for acute toxicity data. The work on allometric scaling factors derived from acute effects is essentially complete and has been published in the 1999 Bulletin of Environmental Contamination and Toxicology 62:653-663.

Acute toxicity data residuals from the allometric scaling regression models are being evaluated further to characterize differences in sensitivity among taxonomic groups of birds and mammals. In addition to analyses involving acute toxicity data, evaluation of chronic toxicity data is continuing.

3. Development of population models for wildlife endpoint species of interest. The initial model development is complete. Previous risk-assessment methods for relating toxic effects on individuals to population responses have assumed that relevant toxicity test endpoints (e.g., mortality and reproduction)

are equivalent to population-level effects. These studies have ignored habitat and life-history strategies. Our models estimate relative population effects by explicitly including the potential effects of contaminants on fecundity and mortality. We developed age-structured matrix models configured for hypothetical r- and K- strategy bird species using best available estimates of parameters from the literature. Interannual stochasticity was incorporated by randomly varying age-specific fecundity and mortality rates. Density dependence was incorporated by making age 0 survival and fecundity functions of adult abundance. Applications of the initial model are described in Task 4 (see below). The model is currently being parameterized for selected bird species and guilds.

4. Integration of dose-response, interspecies extrapolation, and population models to provide estimates of population responses associated with varying exposures experienced by wildlife species. We performed 100-year simulations with our population models developed in Task 3. These simulations were designed to evaluate potential differences in effects of contaminant exposure on bird species with very different life-history strategies. Percent reduction in the long-term average population abundance relative to baseline was compared between life-history strategies and among contaminant exposure scenarios. Model results suggest that birds with r-strategy may be more sensitive to contaminant effects in early life stages, while K-strategists may be more sensitive to decreased adult survival. Decreasing stochasticity of contaminant exposure in our model applications resulted in larger predicted population responses. Population effects inferred from contaminant effects on individuals were often less than population effects predicted by model simulations. A manuscript describing this research for publication in a peer reviewed book has been completed.

Planned Activities: Several activities will continue in FY99:

- 1) An abstract based on the taxonomic sensitivity of birds and mammals to environmental contaminants has been submitted for the 1999 SETAC meeting, which we will attend in November.
- 2) A book chapter on wildlife population models is expected to be completed by end of FY 1999.
- 3) Work will continue on Task 2b, including continued development and evaluation of scaling factors from chronic toxicity data and evaluation the potential for application of scaling factors based on broad groupings (e.g., chemical classes, mode of action, and taxonomic groupings).
- 4) Work will continue on Task 3, including continued development of population models for additional bird species and guilds.

Information Access: None available at this time.