New Considerations for Evaluating Pesticide Impacts to Endangered/Threatened Species

Report to the Environmental Protection Agency
U.S. Fish and Wildlife Service
National Marine Fisheries Service

Case No. C00-3150 CW
Californians for Alternatives to Toxics, Environmental Protection Information Center &
Humboldt Watershed Council v. Environmental Protection Agency

Marc Lappé, PhD
Britt Bailey, MA
Center for Ethics and Toxics
Gualala, California

&

Californians for Alternatives to Toxics (CATs)
Eureka, California

Table of Contents

Executive Summary 1

The Endangered Species Act 2

General Observations on Pesticide Impacts 4

Specific Areas of Concern 8

Rationale for Recommendations 11

Overview of Risk Modeling 11

2.1 General Description of Existing Models 12

2.2 Limitations of the Tiered Approach 14

2.3 New Selection Criteria for Inclusion of Pesticides in Tiered Analyses 18

2.4 Pesticide Environmental Fate and Behavior 19

2.5 Use of Uncertainty Factors 21

2.6 Use of Surrogates 23

2.7 Accounting for Degradates, Adjuvants and Diluents 24

2.8 Reproductive Toxicity as an Example of the Consent Decree's Request for Consideration of Indirect Effects 24

2.9 Existing Reproductive and Sub-lethal Toxicity Assessments 25

3.0 Conclusions Concerning Risk Modeling 26

3.1 Compensating for Uncertainty in Pesticide Toxicity Assessment 27

3.2 Importance of Sub-lethal and Chronic Toxicity Data 27

3.3 Examples of Specific Toxicological Risks of Pesticides 28

4.0 Endangered Species Reference Dose Proposal 29

4.1 Specific Rationale for Compensating for Reproductive Risks 31

5.0 General Recommendations and Conclusions 32

5.1 Specific Recommendations for Protecting Endangered and Threatened Species 33

Appendices

Appendix A: Legal Requirements of the Endangered Species Act 35

Appendix B: Toxicity Tests Used in Risk Assessment 37

Appendix C: Ecological Models Used in Risk Assessment 38

Appendix D: Representatives of Potential High Risk Pesticides' Active Ingredients, By Class 45

Appendix E: Representative Pesticide Toxicity Profiles 47

Appendix F: Toxicity and Exposure Data for Identified Pesticides 54

References 56

1.0 Executive Summary

In order to settle litigation in Californians for Alternatives to Toxics (CATs) et al. v. EPA (N.D. Calif. No. 00-3150), the Environmental Protection Agency (EPA) entered into a Consent Decree whereby it must evaluate, with the assistance of both the U.S. Fish and Wildlife Service (FWS) and the National Marine Fisheries Service (NMFS), how best to evaluate the impacts of pesticides on all listed species as part of the EPA's affirmative duty to conserve listed species.

Pursuant to the CATs Consent Decree, EPA will consider this position paper in the course of EPA's conservation review with FWS and NMFS. Our views are intended to assist the EPA, the FWS and the NMFS in determining how to best evaluate the impacts of pesticides on species listed under the Endangered Species Act (ESA), 16 U.S.C. §1531 et seq., as endangered or threatened (hereinafter 'listed species'). This paper discusses, and the EPA has agreed to evaluate:

A) Chronic and sub-lethal effects of pesticides on all life stages of endangered and threatened species;

B) Effects of complete pesticide product formulations, effects of diluents, and adjuvants, and effects of the products of pesticide degradation;

C) How direct and indirect effects of pesticides added to the environmental baseline impact endangered and threatened species;

D) The use of systematic field monitoring in a variety of site conditions, runoff patterns, and application methods to validate transport and persistence models; and,

E) Best available scientific evidence.

Taking these five factors into consideration will require that the EPA modify its methodology for evaluating impacts to listed species. Such modifications will be necessary to meet the statutory requirement that the EPA affirmatively acts to halt and reverse the trend toward species extinction, as required by the Endangered Species Act.

We recognize that EPA uses models to assess potential impacts of pesticides. Although models can be used in risk determinations, to focus field monitoring and to act as one tool for conserving listed species, EPA's current pesticide risk assessment systems suffer from at least

the following deficiencies: 1) they inadequately consider best available scientific evidence; 2) they fail to incorporate the effects of complete pesticide formulations and multiple pesticide components including diluents, adjuvants and degradation products; 3) they exclude synergistic effects; 4) they fail to use field monitoring to validate transport and persistence models, and 5) they inadequately assess the chronic and sublethal effects of pesticides on all life stages of each species. We review and cite the resulting intrinsic uncertainties of existing risk models, especially the limitations of the acute toxicity basis for present Levels of Concern, and highlight the special protection needs of listed species.

Because EPA uses models in its risk determinations, in addition to evaluating the five Consent Decree factors, we urge the EPA to improve existing models to accommodate the complexities inherent in evaluating pesticide toxicity to listed species. To this end, we propose the incorporation of a new reference dose for measuring the impact of pesticides on listed species based on reproductive toxicity—the **Endangered Species Reference Dose** (**RfDes**).

Evaluating reproductive toxicity plays a essential role in ensuring the survival and recovery of listed species. This new reference dose should aid EPA in determining a pesticide's likely adverse effects as it impacts a given endangered species. In our view, the most pertinent reference dose will serve to further EPA's affirmative duty to conserve each listed species.

1.1 The Endangered Species Act

Congress enacted the Endangered Species Act "to provide a means whereby the ecosystems upon which endangered species and threatened species depend may be conserved [and] to provide a program for the conservation of such endangered species and threatened species.... " Through implementation of the ESA, Congress intended to achieve its dual goal of protecting and recovering endangered species and their habitat.

The Supreme Court has held that the ESA is "the most comprehensive legislation for the preservation of endangered species ever enacted by any nation" in Tennessee Valley Auth. v. Hill. Its stated purposes were "to provide a means whereby the ecosystems upon which endangered species and threatened species depend may be conserved," and "to provide a

program for the conservation of such ... species...” In furtherance of these goals, Congress expressly stated, "all Federal departments and agencies shall seek to conserve endangered species and threatened species..." The Act specifically defined ‘conserve’ as meaning "to use and the use of all methods and procedures which are necessary to bring any endangered species or threatened species to the point at which the measures provided pursuant to this chapter are no longer necessary." The Supreme Court was convinced “beyond doubt” that “Congress intended endangered species to be afforded the highest of priorities.” “The plain intent of Congress in enacting [the ESA] was to halt and reverse the trend toward species extinction, whatever the cost.” *Id.*

Section 7(a)(1) of the ESA imposes an “affirmative duty” on federal agencies, in consultation with and with the assistance of the FWS and NMFS to utilize their authorities in furtherance of the purposes of the ESA by carrying out programs for the conservation of “each” endangered and threatened species. By imposing a duty on agencies to use "all methods and procedures which are necessary to bring any endangered species or threatened species to the point at which the measures provided pursuant to this chapter are no longer necessary," Congress was clearly concerned with the conservation of “each” endangered and threatened species. (See, Sierra Club v. Glickman) As the Supreme Court recognized, the purpose of the ESA includes the conservation of the species and of the ecosystems upon which they depend, and every agency of government is committed to see that those purposes are carried out. The agencies of Government can no longer plead that they can do nothing about it. They can, and they must. The law is clear. (TVA v. Hill).

In Sierra Club v. Glickman, the trial court ordered the US Department of Agriculture (USDA) to develop, in consultation with FWS, "an organized program utilizing USDA's authorities for the conservation of the Edwards-dependent endangered and threatened species." The USDA did not challenge the scope of the district court's injunction with respect to § 7(a)(1). The Ninth Circuit rejected USDA’s argument that the agency’s discretion to ignore § 7(a)(1) was unreviewable, finding instead that the agency must show that it has considered the relevant ESA factors and followed the required ESA procedures.

In addition, every federal agency must insure that its actions are not likely to jeopardize a listed species or destroy or adversely modify its critical habitat.[1] All federal agencies must consult with FWS or NMFS about actions that 'may affect' a listed species. These 'actions' have been interpreted broadly by FWS to mean "all activities or programs of any kind authorized, funded, or carried out, in whole or in part, by Federal agencies," specifically including "actions directly or indirectly causing modifications to the land, water or air."

To make the determination of whether an agency action may affect a listed species, each agency, including FWS, must review its actions at the earliest possible time. During consultation, EPA must, among other things: (1) review all relevant information concerning the listed species and critical habitat; (2) evaluate the current status of the species and critical habitat; and (3) analyze the direct, indirect and cumulative effects of the action on the species and critical habitat. At the conclusion of consultation, FWS must prepare a biological opinion detailing how the federal agency action affects the listed species. The biological opinion must be based on the best available scientific information that we believe should include the most sensitive tests of toxicity for each species. A more detailed examination of the legal elements of ESA is given in **Appendix A.**

1.2 General Observations on Pesticide Impacts

Because pesticides comprise the single greatest class of chemicals that potentially adversely affect endangered and threatened species, the oversight of pesticides by the EPA is crucial to avoid deleterious impacts on species survival and recovery. Presently, registration of pesticides requires a battery of tests (summarized in **Appendix B**) to identify environmental fate and toxicity endpoints that require development of restrictions to minimize adverse effects when pesticides are used at a particular application rate. Where these tests fail to consider additive or synergistic effects of complete pesticide product formulations, diluents, adjuvants, and pesticide degradation, they are likely to underestimate the aggregate toxicity of a given pesticide application.

Pesticide effects have already been determined to be significant factors in endangering

species survival or recovery. A review of the nation's endangered and threatened species between 1993 and 2001 indicated that 17% of the 1,088 endangered and threatened species listed on average each year were listed predominantly because of potential pesticide-related impacts on their survival.[2] During that same time period approximately 14% of federal register notices for endangered and threatened species identified pesticides as a concern to designated critical habitat. Notable historic examples of pesticide-compromised species include the bald eagle and the brown pelican for which DDT and its congeners disrupted calcium deposition in eggshells through endocrine disruption.[3]

'Safe' environmental concentrations of pesticides for listed species may differ markedly from safe concentrations for non-listed species. A listed species is almost always more stressed than a secure species, i.e. one not presently in jeopardy. ***Because a listed species is by definition a species in danger of extinction throughout all or a significant portion of its range and a threatened species is likely to be so within the foreseeable future, special controls on pesticide use throughout their habitat may be essential to minimize adverse effects on survival.***[4]

Even if a pesticide did not directly contribute to the initial decline of a given species, a pesticidal formulation may further stress or delay a species' recovery by degrading or interfering with restoration of its habitat, including its supporting food chain.[5] Pesticides can also indirectly contribute to a listed species' further decline by impairing any single element critically needed for reproductive success, by making it more susceptible to predation or disease, or by reducing its food supply.[6]

Historically, pesticides have been recognized as chemicals of environmental concern because of their intrinsic toxicity to non-target species. Because of their generally broad and non-selective toxicity it is not surprising that pesticides are inimical to so many species. Under the Federal Insecticide, Fungicide and Rodenticide Act (FIFRA), as part of registration, all pesticide formulations must be tested prior to use and be demonstrated to have a reasonable margin of safety to workers or the end-consumer.[7] FIFRA pesticide registration must also consider known or anticipatable effects on non-target organisms in recommended application rates, and a pesticide label must be designed to carry all pertinent restrictions and warnings.[8]

Specifically, a registered pesticide must be shown to not unreasonably harm the environment when used in its standard mode of operation.[9]

Secondary impacts on non-target organisms are also relevant to assessing pesticide risks, especially where listed species are at issue. Pesticides are used in many settings including farms, forests, rights-of-way, industrial sites, and homes where impacts on listed species are often assumed to be non-existent. However, pesticide environmental fate and toxicity characteristics can produce unintended consequences on non-target species. Most pesticides are initially applied on land, thereby posing a potential risk for terrestrial species, including plants and animals. ***Also, a substantial portion of agricultural or structural pesticides creates substantial risks to non-target marine and freshwater aquatic organisms and their habitat because of their solubility and low soil sorption.***

Many of the more common pesticides on the market, especially the carbamates, have especially broad toxic impacts irrespective of their formulation.[10] As such they are a potential hazard to many organisms, particularly listed endangered and threatened species. Broad-spectrum pesticides can produce unintended effects on non-target listed species either by direct contact with an organism or its habitat, indirectly through contamination of ingested food sources (seeds, prey, etc.), or through contaminated water sources.[11] For example, birds, mammals, amphibians, and invertebrates may use contaminated puddles in a farm field for drinking, bathing, or breeding. Aquatic organisms can be exposed to pesticide contaminated run-off or groundwater infiltration. Herbicide drift can cause harm to plants or enter water where it can damage aquatic habitat or species. These indirect effects can have significant implications for listed species and their habitat.

To minimize such unintended, secondary adverse impacts, the EPA should give special consideration to restricting the use of broad-spectrum pesticides in habitat used or frequented by endangered or threatened species.

As of the year 2001, 517 animals and 744 species of plants have been listed under the Endangered Species Act as endangered or threatened within the United States. One hundred and fifty five of these species have designated critical habitat.[12] Critical habitat is defined in

reference to listed species as specific geographic areas, whether occupied by that species or not, that are determined to be essential for their conservation and management.[13]

Pesticide impacts to listed species are almost always related to unintended exposures of non-target organisms within the species' habitat.[14] When the non-target organism is a listed species, a pesticide is likely to be adding to background exposure from related control agents or toxicants. Standard toxicity endpoints established in robust, laboratory populations may therefore underestimate the impact of pesticides under field conditions. Therefore, baseline environmental stressors must be considered as part of the overall assessment. For instance, if a river ecosystem is already degraded by sediment, the effects of any given pesticide could be amplified (or dampened) to the extent the sediment served as a repository of particle-bound pesticide degradation products.

An example is the herbicide Roundup and its active ingredient glyphosate that binds tightly to soil particles. Pesticide use in headwater regions can create especially serious indirect risks to downstream habitat because of wide deposition of pesticide-containing sediments downstream.[15] Sedimentation itself, particularly when it is the result of excessive deforestation and brush control, can compromise the reproductive success of any anadromous fish using the river for spawning or rearing. The presence of reduced gravel beds could aggravate any reproductive toxicity of the same chemical or related chemicals for a listed spawning species. An example would be Garlon and its breakdown product 3,5,6 trichloropyridinol which is highly water soluble. These chemicals have particular toxicity for marine and estuarine aquatic species, including juvenile salmonids.[16] Hence, habitat degradation such as increased sedimentation can lower the threshold for adverse reproductive toxicity caused by pyridinol from Garlon or other pesticide degradation products.

These examples underscore the importance of considering the secondary effects of pesticides on habitat in any assessment on listed species.

1.3 Specific Areas of Concern: Summary and Initial Explanation

Under the consent decree, the five elements EPA must evaluate as part of its affirmative duty to conserve each listed species, and our rationales, are shown in **Table 1** below. Recommendations for each item are outlined sequentially below in **Table 1** and discussed more broadly in the text that follows.

Table 1: Summary of Consent Decree Areas of Concern

Element Under Consideration	Rationale for Inclusion	Recommendations: Examples
1. Chronic and sublethal effects of pesticides on all life stages of endangered and threatened species	Absent or deficient measures of toxicity, life stage effects	Expand testing to embrace reproductive toxicity/life stages
2. Effects of complete pesticide product formulations, diluents, & adjuvants, and effects of the products of pesticide degradation	Possible enhanced toxicity from non active ingredients	Estimate additive toxicity, identify most toxic effects, look for synergism
3. How direct and indirect effects of pesticides, added to the environmental baseline, impact endangered and threatened species	Direct, indirect and cumulative pesticide impacts on listed species and habitat ignored especially in relation to enhanced effects due to habitat condition	Identify baseline conditions then consider amplification of pesticide effect due to existing stressors
4. Use of systematic field monitoring in variety of site conditions, runoff patterns, and application methods to validate transport and persistence models	Models used are limited by use of surrogates	Sort pesticides by physio-chemical properties, followed by validation through actual field testing and measurements
5. Use of best available scientific evidence	Scientific estimation of risk to listed species flawed	Develop a new index for risk for listed species tied to survivability and recovery; incorporate most recent peer-reviewed science

1. Expanding Testing:

We recommend expanding toxicity testing to embrace pesticide formulations as they are found in the field to assure that chronic and sub-lethal effects are evaluated for all life stages of listed and endangered species. This testing would include measuring field concentrations of the parent compound and its degradates whenever a threatened or

endangered species is in the exposed area. As a new and important category of risk assessment, we propose investigating the impact of soil fumigants and endocrine disrupting chemicals of such species routinely. We note that classes of chemicals have been shown to have specific adverse effects on reproductive capacity. As shown by the recent experience with atrazine [23], pesticides may produce genetic damage at doses at or near typical environmental concentrations after application.

2. Measure additive toxicity

Many pesticides with common active ingredients may be formulated differently by adding adjuvants and diluents. Such additives may cause the formulations to have greater toxicity not shared by its registered active ingredient. Adjuvants, dispersal or wetting agents, or carriers (such as diesel oil) are intended to increase the biological availability, persistence and/or toxicity of the active ingredients.

Risk assessors currently grapple with risks from multiple sources by using simple measurements of combined toxicity. In such models, adopted from occupational health determinations, a pesticide's relative toxicity is represented as a fraction of the permissible exposure. The fraction of permissible risk contributed by each chemical in a given mixture is then added to give an estimate of arithmetically combined additive toxicity. Inevitably, such an assessment fails to accurately represent risk because it misses interactive effects that may be either synergistic or inhibitory.

Theoretically, a first chemical exposure will activate host defenses to subsequent exposures. However, activating one or more of the P-450 cytochrome series of detoxifying enzymes will often increase rather than decrease subsequent toxicity, depending on the degree to which metabolites are more toxic than the parent compound, thereby intensifying the toxic effect of any given exposure.

3. Assessing Direct and Indirect Pesticide Impacts

Where habitat disturbance adversely affects a listed species, it is often because the chemical impacts key organisms in the food chain in question. Where a herbicide decreases fodder for a grazing animal, or a pesticide reduces the number of nymphal stages of an

aquatic organism that is part of the food chain of a salmonid fish, the net damage to survivability or recovery needs to be factored into the risk assessment. To this end, a list of food chain components that make up the critical habitat for a listed species and the expected impact of pesticide use on those components should be part of the risk assessment process. A further and essential assessment must include the bioaccumulation factor that applies to each pesticide to assure that animals or fish that are higher on the food chain are protected. Where listed species are exposed to multiple pesticides, their cumulative impact of the chemicals must be assessed.

4. Validate All Pesticide Applications for Field Concentrations

Pesticides should be evaluated according to their 1) estimated environmental movement and persistence; 2) the presence of possible factors that enhance toxicity, including diluents, adjuvants and degradates; and 3) their hazard index for a given listed species. In particular, wherever and whenever models identify that a given pesticide may affect a listed or endangered species, expanded evaluation, including but not limited to field monitoring, would be desirable. In this way, any necessary field monitoring can be focused on a particular vulnerability of the endangered species to the chemical in question.

5. Use of Best Available Science

The use of best available science mandates that contemporary sources in peer-reviewed journals be consulted, and that special consideration be given to certain forms of toxicity that reach a high level of relevance to species survival. An example is reproductive toxicity in assessing risk because successful reproduction is the ultimate arbiter of species survivability. We recommend measuring the resulting modeled and field data against the best estimate of a 'safe' environmental concentration for a given listed species at risk in the impacted ecosystem. This new benchmark would be based on a tolerable dose based on a no-observed-effect-level for reproductive harm (the RfDes) consistent with ESA's mandate to conserve each listed species. The proposed benchmark is not intended to be a substitute for the continued use of peer reviewed, contemporary scientific methodology and techniques to assure the most sensitive assessment of risk.

1.4 Rationale for Recommendations

Endangered and threatened species are already in danger of extinction, or are likely to be so within the foreseeable future. Exposure to pesticides and other toxic substances may be responsible for and exacerbate the plight of these species. In spite of the mandate to conserve endangered and threatened species, current EPA ecotoxicity testing omits essential protective elements from its risk assessment methodology. ***By failing to conduct sufficient consultations and by failing to adequately incorporate the above five factors in its modeling, the EPA fails to afford listed species the "highest of priorities." Such omissions make it likely that the Environmental Protection Agency will fail to halt and reverse the trend toward species extinction as required by the Endangered Species Act.***

This position paper argues that risk assessment for listed species must differ from conventional ecological risk assessment. Risk assessment for endangered and threatened species should focus on *all of the factors* necessary to ensure adequate offspring are successfully produced and raised to sexual maturity. Producing and (where appropriate) rearing sufficient offspring in a suitable, uncontaminated habitat is critical to the survival and recovery of a listed species. In any given habitat where pesticides are present, they may have adverse impacts on both the survival and recovery of listed species through their immunological, neurobehavioral, or reproductive toxicity at levels below their immediate acutely toxic effects. Hence, ecotoxicity testing of pesticides needs to take into account the chronic and sublethal risks posed by persistent, mobile and toxic pesticides, their diluents and adjuvants and any degradation products resulting from their metabolism or breakdown in the environment on successful reproduction.

2.0 Overview of Risk Modeling

The current state of the art in ecological modeling of pesticides in the environment is summarized in three documents: one white paper that offered guidelines for ecological risk assessment,[17] and two 1992 publications, Ecological Risk Estimation,[18] and Ecological Risk Assessment.[19] As of 1998, the first of these, the Framework for Ecological Assessment, is the product of the Environmental Protection Agency and represents the culmination of expert

opinions, meetings, and a variety of workshops and peer review processes. It is not intended as a regulation, rather a structure and guideline for risk assessors looking at the effects of ecosystem change on species. The Framework provides the basis for characterizing environmental pesticide exposures and their effects by emphasizing the need to measure the space and time distribution of 'stressors' and their impact on the ecological components of concern.[20]

The Framework for Ecological Risk Assessment identified some of the major uncertainties involved in extrapolating chemical effects from laboratory ecotoxicological test results to natural ecosystems while keeping risk assessment as cost-effective as possible. To establish the most efficient modeling methodology, the EPA has generally adopted the so-called 'tiered' approach. This tiered or phased approach has been used by many different regulatory agencies, such as Environment Canada, the European Union as well as the EPA. Based on the models presented in the Framework, the EPA currently relies on these standardized ecological assessment models that use a tiered structure for approximating actual environmental risk.

The tiered process is intended to provide a progressively more detailed and logical approach to risk assessment of the potential for any individual toxicant to reach significant levels in a given environment, usually an aquatic ecosystem. In a tiered approach, risk assessors commonly approximate environmental concentrations by modeling in a first tier, and then make a determination based on the resulting risk quotient as to whether or not additional field-testing is needed as a second tier. Additional tiers are then added that make more realistic measurements to approximate direct toxic effects on a target species. Higher tiers tend to require more resources to reach the most realistic evaluation of risk for a given chemical.

2.1 General Description of Existing Models

To some degree, all models include inherent and recognized limitations to predict toxic impacts of a given pesticide on a non-target species and their habitat. In part to compensate for these limitations, Tier I screening uses generally conservative assumptions and identifies potential risk based on the intensity of active ingredient usage. If a pesticide is found to exceed established levels of concern, a higher tiered assessment ensues. Tier II analyses a larger battery

of toxicity test data. It is intended to provide a more detailed examination of the estimates of environmental concentration and to begin to offer management options for limiting the environmental concentration of high -risk pesticides.

The limitation of the first two Tiers is that they minimize the amount of analysis required to evaluate any given chemical. By handling only one pesticide at a time and limiting its parameters to easily derived data such as an index of the lethal toxic dose (e.g. an LD50 or an LC10), each tiered model runs the risk of overlooking a less overtly acute but serious survival limiting factor, such as reproductive toxicity. The Tier III approach is intended to refine the estimates of risk and to provide entry data that correct for any residual uncertainties in the modeling. It expressly uses both acute and chronic toxicity data (but not necessarily reproductive toxicity estimates), and requires more laboratory and actual field data in realistic exposure scenarios. The Tier III systems also estimate the consequences of mitigation measures (such as requiring a lower application rate in a given field condition), as well as model the impact of other management options on the targeted species. Tier IV systems are usually highly sophisticated modeling or validation studies which build on the predictions and options offered by Tier III assessments. Descriptions of the rationale for each Tier and their respective models are presented in the Draft EcoFram Reports of 1999.[21] We provide specific examples of each model and its critique in **Appendix C**.

The models are most useful in providing an estimate of likely or 'effective' environmental concentrations of a pesticide. If a pesticide does not reach an aquatic or terrestrial system in sufficient concentration to cause toxicity, the EPA's current methodology excludes the pesticide from the next tier of analysis. The model makes no adjustment for the presence of mixtures, adjuvants, diluents or inert ingredients and limits the assessment of risk to the principal active ingredient of each formulation. Unfortunately, these omissions may result in serious miscalculation of risk in the case of pesticide formulations that change the physiochemical environment of the pesticide. For instance, as we have expressed in our areas of concern, the estimation of environmental concentration does not include interactive effects, or the contribution of dispersants, wetting agents or diluents on the actual, field values for soil mobility

or solubility.

The use of a so-called 'tiered' approach is common to all of the models. Depending on the tier at issue, each model uses empirical formulae to estimate environmental concentrations from standard pesticide application rates, and then projects likely toxic impacts on potentially affected species.[22] EPA's reliance on a tiered model approach relying on acute toxicity may cause it to ignore more pertinent chronic and indirect effects that jeopardize species survival and recovery. For instance, atrazine apparently disrupts normal sexual maturation in the frog[23] at effective aquatic concentrations of 0.1-1.0 ppb, considerably below those used for the triazines (100 ppm, shown in **Appendix F)**. In theory, estimating risks to listed species should obviate this problem. However, most models only use surrogate species and ecological simulation of pesticide behavior in the environment in their first phase risk assessments. As in the case of atrazine, the models may fail to consider adverse sub-lethal effects that impede the reproductive success of listed species.

2.2 Limitations of the Tiered Approach

Risk assessors generally rely on one or more of these ecological models to approximate the presence and amount of pesticide used. The ability of any given ecological model to evaluate pesticide impacts on non-target organisms is limited by 1) lack of toxicity information for a pesticide's complete formulation, 2) inability to predict the consequences of uneven applications or spills, and, 3) assumptions about the constancy of degradation under various climatic conditions. In the case of listed species, the ecological modeling approach is also hampered by the extrapolation of data from surrogate laboratory organisms to the species in question. In the sections that follow, we review ecological modeling generally and recommend modifications to EPA's methodology for evaluating pesticide impacts to listed species in light of the five factors of the Consent Decree.

The tiered approach generally follows three basic steps:

Step 1. Gather basic data and information for a complete and transparent exposure assessment commonly using laboratory test conditions and estimates of field exposures.

Step 2. Develop a screening level exposure assessment from actual field measurements.

Step 3. Develop advanced exposure assessment if necessary

To generate an initial assessment of risk, the first tier evaluation (Tier I) typically involves pesticide application rates and other information based on physicochemical characteristics like soil carbon adsorption coefficients, water solubility, persistence and octanol water coefficients that estimate the ultimate concentrations of the pesticide in water or on land. Airborne exposure is assumed to be more immediate and transient compared to waterborne exposure. The risk assessments ostensibly make worst-case assumptions, but spills, misapplication or spotty coverage, and heterogeneous factors in the environment confound the analyses and reduce their power to predict the actual exposure to a non-target species.

If an initial assessment finds no likelihood of direct risk to non-target organisms from a given pesticide and its standard application rate, no further evaluations are usually done. If, however, the initial assessment indicates concern, a more refined assessment is performed. In the second-level research (Tier II), field data are gathered. At this juncture tests are conducted to measure chronic effects as well as to estimate sub-lethal effects, such as impairment of reproduction and/or growth. However, none of the existing models include formulae or risk estimation procedures that quantify the harm caused by indirect pesticide effects that may degrade food sources or environmental quality generally. We advocate inclusion of such procedures.

To determine risk to a non-target organism, researchers would ideally measure survivability by culturing the organism of concern or suitable surrogates under controlled conditions of exposure; testing the organism's sensitivity to a given chemical under its likely habitat conditions; simulating soil and climate conditions after chemical exposure, and measuring each pesticide’s effect on all life stages of each species in question. While this approach would provide a more accurate measurement of the effects of active ingredients of pesticides to listed species, risk assessors have generally not adopted this method. Among the reasons are, 1) the difficulties in culturing or providing laboratory conditions for an actual listed species, 2) the intrinsic problem of sacrificing individuals of an already listed species, and 3) the

problems inherent in estimating all the variables needed to measure the 'true' impact of chemicals on the listed species and their habitat.

To partially correct for these deficiencies, all of the risk assessment models used in the last seven years rely on some variation of the method of calculating a risk quotient that is then compared to a Level of Concern appropriate for a listed species. Some risk assessors have advocated the use of additional uncertainty factors to provide appropriately protective measures for listed species. Some movement towards this recommendation was apparent in the Framework for Ecological Risk Assessment discussed above, for instance where listed species were given an 'extra' uncertainty factor (e.g., 1/30) to lower the threshold for concern from a particular environmental concentration of a pesticide. We believe that the approach of adding safety factors is appropriate and necessary, but requires a more rational approach in the context of listed species. Our view is that a reproductive safety factor is the most appropriate element to add to the risk assessment models presently in use.

Manufacturers and risk assessors have used different models to estimate environmental fate and toxicity for target organisms and ascertain suitable labeling application rates that avoid ecological harm. For instance, the Generic Estimated Environmental Concentration (GENEEC) model (reviewed below) allows for the calculation of estimated environmental concentrations of a given chemical for aquatic organisms. The resulting value then can be divided by known acute concentrations of chemicals (such as its lethal concentration) to determine whether or not a given application rate exceeds a set level of concern (LOC) for the environmental impact of a chemical. Where a listed species is likely to be present, the level of concern is set to require lower pesticide environmental concentrations than would normally be used in order to cap pesticide application rates for non-target organisms.

In the GENEEC model, for instance, the risk to a species is characterized by simulating environmental concentrations of a pesticide and comparing them to projected species sensitivity to the toxic effects of the active ingredient. Risk characterization integrates the results of the exposure and ecotoxicity data to evaluate the likelihood of adverse ecological effects. The means of this integration is termed the Quotient Method.

The EPA's current process calculates risk quotients (RQs) by dividing exposure estimates of a given pesticide by a toxicity endpoint, usually an acute and/or chronic lethal concentration for 10 or 50 percent of exposed test organisms. The resulting quotient is then expressed as the fraction of anticipated exposure divided by an index of toxicity, such as the chronic lethal concentration of the chemical. The risk quotient (RQ) can be expressed as: RQ = Exposure/Toxicity, where 'exposure' is an index of the estimated environmental concentration (EEC) of the pesticide and 'toxicity' is usually set as a lethal dose or lethal concentration LD10, LD50, LC10 or LC50).

The EPA's Office of Pesticide Programs uses these risk quotients to approximate risk by comparing them to established levels of concern (LOCs). These LOCs are used to examine the potential risk to non-target organisms as well as to consider possible regulatory action based on potential risk. These criteria are intended to indicate when a pesticide used as directed has the potential to cause adverse effects on non-target organisms such as an endangered or threatened species.

Levels of Concern currently include the following risk presumption categories: 1) **acute high** -- potential for acute risk is high; regulatory action may be warranted in addition to restricted use classification, 2) use that is a limited set of circumstances and application rates for a given pesticide; 3) **acute endangered species** - listed species may be adversely affected, and 4) **chronic risk** - the potential for chronic risk is high, regulatory action may be warranted.

The ecotoxicity test values (measurement endpoints) used to generate the acute and chronic risk quotients are usually derived from required laboratory studies. Examples of ecotoxicity values derived from short-term laboratory studies that assess acute effects are lethal concentration endpoints for fish and birds, and effected concentrations for aquatic plants and aquatic invertebrates. Examples of toxicity test endpoints derived from the results of long-term laboratory studies that assess chronic effects are: 1) Lowest Observable Adverse Effect Concentration (fish; aquatic invertebrates) and 2) No Observed Adverse Effect Concentration (NOAEC) (fish; aquatic invertebrates). For fish and aquatic invertebrates, the NOAEC generally is used as the ecotoxicity test value in assessing chronic effects. The presumptions commonly

used in risk modeling are shown in **Table 2** which shows existing Levels of Concern based on Estimated Environmental Concentrations of pesticides.

Table 2: Risk Presumptions, Risk Quotients and Levels of Concern for Aquatic Species

Risk Presumption	RQ	LOC
Acute Risk	EEC/LC50 or EC50	0.5
Acute Restricted Use	EEC/LC50 or EC50	0.1
Acute Endangered Species	EEC/LC50 or EC50	0.05
Chronic Risk	EEC/ NOAEC	1

[1] Estimated Environmental Concentration (EEC) or Environmental Concentration (EC) measured in ppm or ppb in water and corresponding toxicity levels in mg/ or μg/L, respectively.

Risk Presumptions for Plants

Risk Presumption	RQ	LOC
Acute Risk	EEC/EC50	1
Acute Endangered Species	EEC/EC05 or NOAEC	1

Note: Levels of Concern are generated within the context of the risk assessment models shown in **Appendix C.**

2.3 New Selection Criteria for Inclusion of Pesticides in Tiered Analyses

If EPA is to continue to use models to discharge its affirmative duty to protect endangered species, we propose the following criteria to ensure adequate treatment of pesticide toxicity. Some of these criteria have been adopted from EPA's Hazard Evaluation Division Guidelines for Ecological Risk Assessment. Table 3 presents known and recognized toxicological criteria for evaluating the toxicity of pesticides to various habitats and organisms. Part A and Part B provide basic exposure endpoint information. Exposure information and hazard information could be included in evaluations of whether or not a pesticide 'may affect' listed species and facilitate EPA's proactive conservation review.

Should a pesticide or its components fall outside any acceptance criteria for characteristics shown in Parts A and B, we would recommend exclusion from use in areas where a listed species is present. A Tier II (or its equivalent) analysis would follow as a tool for focusing environmental monitoring in ascertaining actual contamination levels.

Table 3: Established 'Conservative' Toxicity Endpoints

A. Environmental Exposure Endpoints

	Mobility	**Persistence**	**Solubility**	**Bioaccumulation Factor:Log P_{ow}***
Acceptable	**>500 cm/hr**	**<50 days**	**<500 mg/L**	**<3**
Unacceptable	**<500 cm/hr**	**>50 days**	**>500 mg/L**	**>3**

*P_{ow} or K_{ow}: The octanol-water partition coefficient is the ratio of the concentration of a chemical between octanol and water phases. This parameter has been correlated to water solubility, and is used to help determine the fate of chemicals in the environment.

B. Acute Hazard Endpoints

	Terrestrial Vertebrates mg/kg	**Terrestrial Invertebrates mg/kg**	**Aquatic Vertebrates mg/L**	**Aquatic Invertebrates mg/L**
Acceptable	LD50 >500	LD50 > 500	LC50 >10	LC50 >10
Unacceptable	LD50 < 500	LD50 < 500	LC50 <50	LC50 < 10

2.4 Pesticide Environmental Fate and Behavior

Assessments of the qualitative characteristics of a pesticide may be used to evaluate the potential adverse effects of a given pesticide on a listed species. In some circumstances, the environmental fate information, coupled with basic toxicity data, provide sufficient information to trigger a 'may affect' determination. For instance, the endocrine disruptive effects, persistence, and high K_{ow} of DDT and DDE would have flagged these chemicals for immediate consultation and possible exclusion had this system been in place in the 1960s.

An itemized example of toxicity information (not intended to be exhaustive) is shown for some representative chemicals in **Appendix F**. These data include some hallmark fate information. That information helps the risk assessor determine whether a chemical generally remains where it was applied, degrades into potentially more toxic byproducts, or if it

bioaccumulates in the ecosystem. These variables are partially described by knowing the mobility and persistence of a chemical. In many circumstances, an aquatic organism may be affected by a chemical that is highly mobile, while a terrestrial organism or mammal may be affected by an immobile chemical.

If persistence and/or mobility exceed established parameters, they create indirect risks to those organisms present in the relevant ecosystem. For example, pre-emergent herbicides require special consideration to terrestrial invertebrates as they are designed to remain in the soil for long periods of time. Knowing a chemical's mobility provides insight into whether it is likely to leach into groundwater or remain where it was applied. The K_{oc} (soil/carbon partition coefficient) provides mobility data. Pesticides that have K_{oc} values above 500 will, by definition, largely stay in the soil. Such pesticides are likely to have their major toxicological effects (if any) on terrestrial organisms. Conversely, as shown in Table 4, those with K_{oc}s under 500 readily dissolve in organic matter and are likely to impact aquatic organisms proportional to their solubility.

Table 4: Environmental Characteristics of Pesticides

K_{oc}: Organic Carbon Binding Constant	Characterization
> 4000	(non mobile)
4000-500	(slightly mobile)
499-75	(moderately mobile)
74-15	(mobile)
< 15	(very mobile)

Persistence is measured by the length of time required for half of the chemical residue to dissipate, decompose, or be metabolically altered. The half-life can be applied to soil, water, tissues, etc. It is measured in days [t ½]. As shown in Table 5, chemicals with half-lives over 5.0 warrant attention, while those with half-lives over 22 days retain their toxicity for a moderate period. Those with half-lives over 60 days are highly persistent with extended opportunity for toxicity.

Table 5: Persistence Characteristics of Pesticides

Half Life	Characterization
<5 days	(non persistent)
5-21 days	(slightly persistent)
22-60 days	(moderately persistent)
>60 days	(very persistent)

Toxicological considerations also include whether or not a chemical is likely to bioaccumulate, adsorb to sediment or suspended organic matter, or contaminate ground or surface water. Sediment-associated toxicants are almost always the most abundant and persistent contaminants in estuarine and marine ecosystems.[24 25]

The types of pesticides being used in various ecosystems should also be considered. For example, volatile pesticide may appear to be inconsequential to organisms due to their inherent volatility. However, some highly volatile pesticides with high persistence levels can affect honeybees, birds and mammals. With this view in mind, the safeguard of predators which tend to be more vulnerable to pesticides than their prey, especially after bioaccumulation, may require that insecticide use be restricted, e.g. during nesting seasons when young birds require a high protein diet of insects that might carry pesticide residues.

Pesticides that are known to bioaccumulate should be given special attention since by virtue of increased concentration they have an increased ability to disrupt organisms at higher trophic levels. Chemicals that persist and have high fat solubility readily bioaccumulate in the tissues of fish, shellfish and waterfowl. As a result, these chemicals require special monitoring. The *n*-octanol/water partition coefficient is one measure of this solubility and hence portends a chemical's ability to bioaccumulate.[26] When this coefficient is greater than 3, implying a thousand (1000)-fold differential octanol/water partitioning, a risk of accumulation exists.

2.5 Use of Uncertainty Factors

Uncertainty factors are protections added when extrapolating toxicity data compiled from a simulated ecological model or toxicity test in order to derive an exposure concentration below the point where adverse effects on complex ecological systems are unlikely to occur. For

example, if there are no appropriate species-specific data, an interspecies uncertainty factor of 10 is used. However, this does not account for the substantial variation between chemicals and classes of chemicals where the known no observed effect levels (NOEL) can be low yet the acute lethal concentrations can be high. Many factors should be considered when deciding on a specific uncertainty value. These include 1) the species tested (type, appropriateness, and range); 2) the toxicological endpoint observed and the likely mechanism of action; 3) the range of response in the species tested; 4) the variability of response among the species tested; and 5) pharmacokinetic differences among the species tested.

As applied to listed species, uncertainty factors by nature should be protective. The present recommendations for acute pesticide measures call for a 'trigger' set for non-target species at 1/5th of an LD50 or LC50. From a policy position, this trigger is set unacceptably high. ***When a pesticide's environmental concentration is at 1/5th the LC50, an 'acceptable' death rate of 1 in a 1000 can still be expected.***[27] ***Such a rate would not be acceptable.*** Because habitat modeling is too complex to accurately depict actual risk, it is often simulated. It is also axiomatic that toxicity modeling does not utilize the endangered species itself. As a result, an additional uncertainty factor for endangered species such as our recommended Reference Dose can be justified.

Frequently the EPA lacks complete chronic and sublethal testing data for pesticidal active ingredients and their formulations. The chronic testing that has been completed often fails to include data on reproductive endpoints, even where no effect is noticed. In the case where a reproductive No Observed Effect Level (NOEL) is absent, we also argue an additional uncertainty factor of at least 3 should be used.

The uncertainty factors applied to chemical related ecological risk assessments should also protect ecosystem functions. To best protect the structure and function of ecosystems some have argued it is appropriate and sufficient to protect keystone species as the best endpoint for risk assessment.[28] Additional uncertainty factors placed upon keystone species could thus alternatively be used to protect habitats where endangered species live. However, surrogates for keystone species do not generally exist. The general issue of using surrogates is discussed below.

2.6 Use of Surrogates

To make a risk determination, it is necessary to use a surrogate or model species for the endangered one, e.g. a trout for an endangered salmonid species generally. We review the toxicity assessment procedure and results for representative surrogates (such as rainbow trout for Coho salmon) in **Appendix F**. In each instance of a toxicity test on a surrogate species, most risk assessors define the applicable species as the most susceptible species for a particular pesticide in a given phylum. We provide a listing of the surrogate species in Table 6.

Table 6: Frequently Used Surrogates and Trophic Levels

Vertebrates		**Surrogate**	**Trophic Level**
Terrestrial	Mammals	Rat Sheep Rabbits Cattle	3,4
Terrestrial	Birds	Northern bobwhite	2,3
		Mallard duck	2,3
Aquatic	Amphibians	Boreal Toad Bullfrog	2
	Fish	Rainbow Trout Sheepshead Minnow Bluegill	2,3

Invertebrates		**Surrogate**	**Trophic Level**
Terrestrial		Earthworms Honeybees	2
	Insects	Stone Fly Midge	2
Terrestrial	Arthropods	Mites	2
Aquatic	Molluscs	Eastern Oyster Pacific Oyster Mussels	2
Aquatic	Crustaceans	Daphnia magna Crayfish Dungeness Crab	2

We note that ideally the impact on the actual species at risk is preferable in all circumstances to the use of a surrogate, while recognizing the limitations of performing tests on a listed species.

2.7 Accounting for Degradates, Adjuvants & Diluents

Data compiled for the on-site toxicity of a given pesticide ultimately depends on its final formulation. We believe it is reasonable to ascertain the most toxic species-specific component of the pesticide formulation and to thereafter use it as a baseline for species sensitivity. We recognize that in most cases, this complete pesticide formulation toxicity profile is unknown. In such circumstances, the most highly toxic additional or degradate component can be used.

In some instances, the final product of concern may be one or more of the adjuvants, diluents, and degradates which can pose harm to non-target organisms. One example is Roundup herbicide. While its active ingredient glyphosate is relatively benign, an added surfactant polyethyloxylated tallow amine (POEA) is known to be three times as toxic as the active ingredient.[29] An example of a formulation with a toxic degradate is Garlon (3A or 4) which has pyridinol as a toxic degradate. While the EPA requires data on some complete formulations, where adjuvants are added to the formulation, the complete toxicity usually is unknown.

In such circumstances (as shown in the examples above), a separate risk estimation should be done on the additive in question, and that factor included in the overall risk assessment.

2.8 'Reproductive Toxicity' as an Example of the Consent Decree's Request for Consideration of Indirect Effects

Several studies have revealed that exposure to certain endocrine disruptors results in adverse effects to wildlife species and populations. These effects can vary from subtle changes in the physiology and sexual behavior of species to permanently altered sexual differentiation.[30] For example, exposure to organochlorines has been shown to adversely impact the reproductive and immune function in Baltic seals (as measured by viral susceptibility), resulting in marked population declines. There is also extensive evidence that chemical constituents present in paper and pulp mill effluents and sewage treatment effluents can affect reproductive endocrine function and contribute to alterations in reproductive development. [31]

Our focus on reproductive toxicity is premised on the Endangered Species Act requirement that species both survive and recover. Reproductive hazards from some pesticides create an intrinsic risk to survivability, depending on exposure conditions. An example would be DBCP (dibromochloropropane), which causes both direct and indirect toxicity to gonadal function, notably sperm production. DBCP is capable of suppressing testicular function in mammals at low environmental concentrations.

By definition, endocrine disruptors affect the pituitary-adrenal-end organ axis that is critical to the successful development of a functional, mature reproductive system. Where endocrine disruptors are present, an organism's overall fitness may be diminished even where reproductive success may remain 'high'. This effect was seen in the American alligator where endocrine disruptors produced a distorted sex ratio of offspring in certain Florida lakes. Had this contamination been more widespread, intergenerational survivability of this species may have been drastically affected as a result of the potentially impaired reproduction of the next generation.

This discussion of endocrine disruptors is of necessity incomplete, but is intended to point to the overall need to include a special assessment on reproductive harms as part of any risk evaluation of hormonally active pesticides which contact listed species, impair reproduction, and as a result potentially compromise species survivability.

2.9 Existing Reproductive and Sub-lethal Toxicity Assessments

The reproductive toxicity tests presently employed by the EPA in pesticide registration requirements tend to measure the teratogenicity or general fertility- damaging effects of toxic exposures before and during pregnancy. Such tests do not necessarily test *reproductive fitness,* as they would affect listed species, for instance in terms of survivability of gametes or larval stages of the species in question. This is true for two reasons: 1) a reproductive toxicity test does not measure population fitness, such as the overall survival of a litter or clutch of eggs; and 2) the toxicity tests do not measure the *continued opportunity for survival* or recovery of the potentially impacted species.

For instance, recovery of a species may be dependent on genetic diversity, lack of serious deleterious mutations (i.e. the genetic load), and/or fertility of its members. These endpoints are knowable with sufficient attention and expanded research on endocrine function, mutational ability, fertility generally, and survival characteristics of embryonic stages of a given species or surrogate under various exposure conditions and are in accordance with ESA's 'best available science' requirement.

The Consent Decree also requires consideration of sub-lethal toxicity measurements. Traditional methods of determining risk to endangered non-target organisms use a measure of exposure and acute risk criteria such as a lethal dose or concentration – typically an LD_{50} or LC/EC_{50}. These measures, however, may fail to account for habitat disruption effects, and provide only limited insight into effects of toxicants at lower concentrations. As presently designed, the models leave a wide margin of uncertainty regarding non-lethal, chronic, or sub-chronic effects and various models may miss these more subtle consequences of exposure.

From a toxicological perspective, a complete understanding of a pesticide's potential impact on listed species requires the incorporation of chronic and sub-lethal risks to survivability, the use of suitable safety factors, and the measurement of environmental fate of metabolites or breakdown products on all life stages of an organism. Existing risk assessment modeling approximates, but does not satisfy this requirement.

3.0 Conclusions Concerning Risk Modeling

The core assumption of each current EPA model is that a pesticide acts singly, and that its physical and chemical characteristics permit a determination of the likelihood that it will contact and cause harm to a non-target organism. This assumption is intrinsically flawed because it neglects indirect or synergistic effects, uses lethality in lieu of sublethal effects in the first tier of analysis, and ignores the environmental baseline including the presence of background levels of toxicants that may already be stressing the targeted species. It is also flawed because it neglects impacts on all life stages of listed species, impacts of complete pesticide product formulations, including diluents, adjuvants, and degradation, and because it

fails to use systematic field monitoring to validate transport and persistence models.

The resulting risk quotient could be considered imperfect because it relies solely on an acute measure of toxicity, notably lethality. We believe the most pertinent measure for listed species are those that relate to the toxic impact of pesticides on the central elements of survival and/or recovery for each species. Such adverse impacts are likely to occur before any lethal effects are observed. ***In addition, the present modeling used to assess pesticide impacts on listed species does not use 'best available science', such as current knowledge about safety factors and reproductive toxicity (e.g., endocrine mimicry in pesticides)***.

3.1 Compensating for Uncertainty in Pesticide Toxicity Assessment

We conclude that virtually all risk assessment models are limited in their ability to predict adverse impacts, particularly to listed species. By definition, standardizing risk assessment underestimates the complexity of factors that ultimately determine how a given toxicant impinges on a given organism. ***The EPA must go beyond existing models because it may not jeopardize listed species and, in fact, must affirmatively act to halt and reverse the species' extinction. We believe that it is difficult, if not impossible, to discharge this duty under the current testing or modeling regime used to estimate the likely impacts of pesticides on listed species.*** In the initial stages of ascertaining the risk to from a pesticide to a listed species, NMFS and FWS are largely dependent on the EPA for a toxicological evaluation. However, as we have indicated, the models used to evaluate impacts are simplifications of actual environmental conditions and can underestimate actual risk to species survivability. ***We argue that prudence requires that reasonable endpoints and safety factors be used to compensate for the inadequacies of the current risk assessment models.***

3.2 Importance of Sub-lethal and Chronic Toxicity Data

Presently, scientists have only approximated the goal of estimating chronic effects and compensating for the resulting uncertainty by improving and harmonizing test methods for toxicological and environmental endpoints.[32] The toxicological properties of a formulated

pesticide's active ingredients provide an initial understanding of the chemical's effect on an organism. Standard toxic endpoints include the LC50, EC25 & EC50, LD50, and the NOEL. ***It is widely recognized that the NOEL is an important component for establishing acceptable limits of pesticides in the presence of listed species.***

Chronic endpoints, such as a chronic toxicity NOEL can provide a more sensitive index of the potential outcome created by exposure over a long period of time than can have a lethal effect measurement or an acute NOEL. However, a NOEL based on a surrogate species or linked to a single route of exposure may have insufficient predictability for assuring a 'safe dose' for an endangered species. For this reason, addition of an uncertainty factor may be necessary. As we have argued, we believe the most relevant NOEL is that for reproductive toxicity.

Chronic and sublethal effects that EPA fails to consider can create lasting effects on the recoverability and ongoing survivability of a species. Chronic endpoints that reflect toxicity to the reproductive systems, immune systems, and genetic integrity of an organism would be ideal, but are rarely if ever calculated. For this reason, making wide adjustments for toxicity of pesticides that impact listed species could allow for greater likelihood of species recovery and habitat restoration.

3.3 Examples of Specific Toxicological Risks of Pesticides

At low doses triazine herbicides and organophosphate insecticides can adversely affect the immune systems of non-target animals,[33] putting them at risk for infectious or parasitic disease. Low levels of pesticides can also reduce survivability by affecting behaviors essential to breeding. Minimal exposure concentrations of the organophosphate diazinon (0.1 µg/L) significantly inhibited olfactory-mediated alarm responses for salmonids. At diazinon dose levels of 10 µg/L homing behavior was impaired.[34] Sublethal exposures to this or related organophosphate insecticides may cause significant behavioral deficits such as alterations to swimming patterns including decreases in speeds and turning rates.[35]

Other pesticides may impede development to sexual maturity and thereby reduce survivability of an endangered species. Herbicides such as the urea based diuron can reduce

normal growth patterns at concentrations as low as 1.0mg/L although a 'no effect' concentration has been established for diuron.[36] Other vital functions may be impaired in a manner inimical to listed species, while non-endangered (more numerous) populations remain unaffected. For instance, physical and chemical properties of blood in tropical fish species have been found to be sensitive to environmental changes. Exposure for 7-90 days to 25% of the 96h LC50 (.55 mg/L) of diuron persistently decreased erythopoietic activity, while exposure to 10% 96h LC50 resulted in initial decreases and then recovery. [37]

Low doses of certain carbamates may be of concern to organisms at one trophic level, but not another. Carbaryl has demonstrated effects of concentrations in the 1 to 5 ppb range on Daphnia development, growth rate, and swimming behavior. Low concentrations inhibit growth and reproduction and delay maturation, whereas survivorship was not affected. When considered in the context of the habitat of an endangered species that relies on Daphnia as a food source, this impact may be cumulative and adverse. For instance, sublethal exposures to carbaryl reduced Daphnia population growth rate by about 15% (at 5 ppb), enough to have significant ecological effects on the rest of the lake community.[38]

In fish*, sublethal* exposure levels of carbaryl and methyl parathion individually and in combination has been demonstrated to significantly reduce the rates of feeding, absorption, and conversion while increasing oxygen consumption and surfacing frequency.[39] Sublethal effects of carbaryl on surfacing behavior and food utilization were seen in the obligatory air-breathing fish as exhibited by increased surfacing with the increased concentration of carbaryl. Enhanced rate of surfacing may have been the result of stress exerted by the toxicant. [40]

4.0 Endangered Species Reference Dose Proposal

To partially compensate for the inadequacies of existing models, and to further EPA's affirmative duty to conserve species, we recommend development and use of an RfDes. The RfDes provides the maximum concentration that can be tolerated on a day-to-day basis over a lifetime, without harm to reproductive function in all of its parameters. As such the RfDes presents an endpoint whereby the survivability of that species will not be compromised. This

new measure should be incorporated into any ecological assessment for safeguarding listed species.

Our primary suggestion is to use the *reproductive* NOEL plus corresponding safety factors to produce the RfDes. The estimated environmental concentration divided by the RfDes would then be compared to the EPA established LOC. Appendix C provides a roster of NOELs for selected pesticides. We note that NOELs are primarily derived from acute toxicity data endpoints, although reproductive NOELs would be more pertinent for an endangered species and would almost certainly be less than typical acute toxicity endpoints. This latter category would include consideration of impairment of survival of gametes, embryonic or larval stages, and toxic effects that impair sexual maturation or fertility.

Currently, species survival depends on factors not considered by EPA's present ecological models. For example, Tier I analyses rely primarily on lethality endpoints, and not one or more of these sub-lethal effects. ***Recovery-specific needs of listed species include a critical mass of reproductively fit organisms, an uninterrupted breeding process, and survival of sufficient numbers of organisms at the various stages needed to assure a breeding population reaches sexual maturity.***

To compensate for these needs, any pesticide application rate determined by risk assessment modeling should account for listed species by having an identified reference dose with a suitable uncertainty factor. As applied to reproductive toxicity, the resulting reference dose could adjust existing NOELs for reproductive toxicity by an applied 10-fold uncertainty factor for species variation. If ovulation and release of viable eggs in the rainbow trout is found to be impaired at a concentration of 1 ppb of a given pesticide (e.g., Garlon-derived pyridinol) in the water, for example, a safety margin of at least ***10*** may be used for extrapolation from trout to Coho salmon. In keeping with other toxicity assessments, if the ***No Observed Effect Level*** is known for a sensitive toxic endpoint (e.g., renal toxicity) but has not been established for reproductive toxicity, an additional ***3-fold*** safety factor would be necessary, viz NOEL/ ***30***. Where several toxicity assessments exist, reducing the exposure proportional to the most sensitive index relevant to species survival is recommended in keeping with the requirement of

ESA to ensure species survivability.

The uncertainty factors should incorporate 1) the closeness of the listed species to the surrogate test species used in one or more toxicity assays (e.g. Coho salmon to rainbow trout), 2) the availability of chronic, life-stage, and reproductive data on a given chemical's toxicity and 3) the known sub-lethal toxicity of the pesticide to endocrine function. The resulting Tier I analysis would create a Risk Quotient more sensitive by a factor of some significant unit, e.g., 10, 30 or 100.

4.1 Specific Rationale for Compensating for Reproductive Risks

To make up for deficiencies in risk assessments that presently overlook or underestimate critical, survival-based factors, we recommend limiting environmental levels of pesticides below those likely to have reproductive harm. Our definition of such harm includes limiting fitness of the at-risk species at any life stage. We would expect that all those pesticides that are potentially toxic to reproductive success, notably endocrine disrupting pesticides, germ-line damaging chemicals or known teratogens are ipso facto those which 'may affect' an endangered species, and hence the EPA must submit them for formal consultation. ***For listed species, the RfDes risk factor would replace the present acute toxicity endpoints such as the LD10 and LD50 currently used to calculate LOC.***

For those pesticides whose application rates generate Risk Quotients exceeding the Level of Concern, we recommend EPA require pesticide restrictions including reduction of use and lower application rates (reflected in re-labeling), and de-registration if necessary to conserve species. We propose that ***to meet its obligation to halt and reverse species decline for each listed species, EPA must prove that a pesticide can consistently be used according to labeled application rates without generating risk quotients that violate a Level of Concern of 1.0 based on the Endangered Species Reference Dose.*** (Recall that the Risk Quotient is the ratio between observed or estimated environmental concentrations and the RfDes.)

To this end, we recommend RfDes values be developed for each pesticide. We note that one goal of a Tier I analysis is already intended to focus risk assessment on those use patterns of

pesticides most likely to impact 'sensitive taxa' (e.g., invertebrates, fish or aquatic plants). We recommend that EPA identify pesticides used in each listed species habitat. Once identified, the assessor should adopt the RfDes as a toxicological endpoint to protect all life stages of an endangered or threatened organism, with a special focus on the development and survival of gametes and early embryonic and larval stages.

5.0 General Recommendations and Conclusions

We recommend a new approach to assessing risks to listed species. Specifically, we urge that pesticide risk assessment embrace the spectrum of effects that may be anticipated from a pesticide's actual formulation and its components, as well as degradation products and metabolites. Specifically, we propose incorporating more thorough protective toxicity endpoints with built-in uncertainty factors adjusted for the simulations inherent in the currently employed risk models.

Toxicity assessment should focus on survivability and recovery of each listed species. As it applies to animal phyla, this requires consideration of the adverse impacts of pesticides to gametes, embryos, larva and other sub-adults, as well as impacts to adults. This focus should include consideration of the likelihood a pesticide or multiple synergistic pesticides will adversely affect neurobehavioral function necessary for successful reproduction. To this end, we urge the EPA to 1) model reproductive effects of pesticides on embryonic and larval development and survival of gametes, and 2) evaluate potential DNA damage (genotoxicity) resulting from pesticide exposure that increases the genetic load and thereby compromises the long-term genetic stability of a reduced species. Together these factors are likely to most seriously affect future generations of the species at risk.

5.1 Specific Recommendations for Protecting Endangered & Threatened Species

- **5.1.1** Systematically review all ecological models in the context of listed species. Specifically, modify existing endpoints to account for chronic and sublethal effects of pesticides on all life stages. Assess complete pesticide product a formulation, including degradates, diluents and adjuvants, and their respective toxicity. Consider how direct and indirect effects of pesticides added to the environmental baseline impact listed species. Once assessed, if their toxicity is greater than that of the parent compound, the most toxic endpoint should be used in assessing impacts for each listed species. Use the best available science.
- **5.1.2** Focus field monitoring on vulnerabilities identified by models and to test a portion of assumptions regarding safety. Use systematic field monitoring in a variety of site conditions, runoff patterns, and application methods to validate pesticide transport and persistence models.
- **5.1.3** Establish an Endangered Species Reference Dose (RfDes) based on the no observed effect level for reproductive toxicity. Use this dose to establish risk quotients for endangered species and resulting LOCs.
- **5.1.4** Apply additional uncertainty factors to the reproductive endpoint where data gaps exist to create the most appropriate risk quotients.
- **5.1.5** Conduct a formal 7(a)(2) Consultation whenever a pesticide may affect a listed species. The Consultation must evaluate a pesticide's environmental fate and the degree to which they have contact with endangered species.
- **5.1.6** Exclude, limit or label the use of pesticides wherever listed species exist, or where their habitat has any reasonable chance of being adversely affected as determined by the modified modeling or risk assessments we have proposed.
- **5.1.7** Proscribe or limit the registered uses of any product for which the proposed application rate exceeds the appropriate Level of Concern as calculated from the RfDes.
- **5.1.8** If a given endocrine-disrupting chemical or other reproductive toxin has characteristics such that exposure is or will be ubiquitous and at a sufficient level to

disrupt reproductive processes in an at-risk endangered species, the chemical's use should be discontinued.

- **5.1.9** Identify early in the assessment process high-risk pesticides, such as pesticides that bioaccumulate, for prohibition or special labeling requirements.
- **5.1.10** Monitor the habitat of threatened and endangered species on an ongoing basis for pesticide contamination to ensure that use restrictions are adequate.

APPENDICES

Appendix A: Legal Requirements of the Endangered Species Act

Congress enacted the Endangered Species Act, 16 U.S.C. §§ 1531-1544, "to provide a means whereby the ecosystems upon which endangered species and threatened species depend may be conserved [and] to provide a program for the conservation of such endangered species and threatened species"16 U.S.C. § 1531(b). Through implementation of the ESA, Congress intended to achieve its dual goal of protecting and recovering endangered species and their habitat.

The Supreme Court has held that the ESA is "the most comprehensive legislation for the preservation of endangered species ever enacted by any nation." Tennessee Valley Auth. v. Hill, 437 U.S. 153, 180 (1978) ("TVA"). Its stated purposes were "to provide a means whereby the ecosystems upon which endangered species and threatened species depend may be conserved," and "to provide a program for the conservation of such . . . species ...." 16 U.S.C. § 1531(b) In furtherance of these goals, Congress expressly stated in §2(c) that "all Federal departments and agencies shall seek to conserve endangered species and threatened species . . . ." 16 U.S.C. § 1531(c) ... [T] he Act specifically defined “conserve” as meaning "to use and the use of all methods and procedures which are necessary to bring any endangered species or threatened species to the point at which the measures provided pursuant to this chapter are no longer necessary." §1532(2). TVA v. Hill, 437 U.S. 153, 180 (1978) The Supreme Court was convinced "beyond doubt" that "Congress intended endangered species to be afforded the highest of priorities." TVA at 174 (emphasis added). "The plain intent of Congress in enacting [the ESA] was to halt and reverse the trend toward species extinction, whatever the cost." Id. at 184. Section 7(a)(1) of the ESA imposes an "affirmative duty" on federal agencies, in consultation and with the assistance of the FWS and NMFS to utilize their authorities in furtherance of the purposes of the ESA by carrying out programs for the conservation of "each" endangered and threatened species. 16 U.S.C. §1536(a)(1). By imposing a duty on agencies to use "all methods and procedures which are necessary to bring any endangered species or

threatened species to the point at which the measures provided pursuant to this chapter are no longer necessary", Congress was clearly concerned with the conservation of "each" endangered and threatened species. Sierra Club v. Glickman, (1998) 156 F.3d 606. As the Supreme Court recognized, the purpose of the ESA includes the conservation of the species and of the ecosystems upon which they depend, and every agency of government is committed to see that those purposes are carried out ... .[T]he agencies of Government can no longer plead that they can do nothing about it. They can, and they must. The law is clear. TVA v. Hill, 437 U.S. at 183-84, (citing 119 Cong. Rec. 42913 (1973)) (emphasis in original)

In Sierra Club v. Glickman, 156 F.3d 606 (1998), the trial court ordered the USDA to develop, in consultation with FWS, "an organized program utilizing USDA's authorities for the conservation of the Edwards-dependent endangered and threatened species." The USDA did not challenge the scope of the district court's injunction with respect to § 7(a)(1). The Ninth Circuit rejected USDA's argument that the agency's discretion to ignore § 7(a)(1) was unreviewable, finding instead that the agency must show that it has considered the relevant ESA factors and followed the required ESA procedures. In addition, every federal agency must insure that its actions are not likely to jeopardize a listed species or destroy or adversely modify its critical habitat. 16 U.S.C. §1536(a)(2) (emphasis added). All federal agencies must consult with FWS or NMFS about actions that 'may affect' a listed species. 16 U.S.C. § 1536(a)(2); 50 C.F.R. § 402.14(a). These 'actions' have been interpreted broadly by FWS to mean "all activities or programs of any kind authorized, funded, or carried out, in whole or in part, by Federal agencies," specifically including "actions directly or indirectly causing modifications to the land, water or air." 50 C.F.R. § 402.02.

To make the determination of whether an agency action may affect a listed species, each agency must review its actions at the earliest possible time. Id. During consultation, EPA must, among other things: (1) review all relevant information concerning the listed species and critical habitat; (2) evaluate the current status of the species and critical habitat; and (3) analyze the direct, indirect and cumulative effects of the federal action on the species and critical habitat. 50 C.F.R. § 402.14(g). At the conclusion of consultation, FWS/NMFS must prepare a biological

opinion detailing how the federal agency action affects the listed species. 16 U.S.C. § 1536(b)(3)(A); 50 CFR § 402.14(h). The biological opinion must be based on the best available scientific information. 16 U.S.C. § 1536(a)(2); 50 CFR § 402.14(d).

Appendix B: Toxicity Tests Commonly Used in Risk Assessment[41]

850.1010 Aquatic invertebrate acute toxicity, test, freshwater daphnids
850.1020 Gammarid acute toxicity test
850.1025 Oyster acute toxicity test (shell deposition)
850.1035 Mysid acute toxicity test
850.1045 Penaeid acute toxicity test
850.1055 Bivalve acute toxicity test (embryo larval)
850.1075 Fish acute toxicity test, freshwater and marine
850.1085 Fish acute toxicity mitigated by humic acid
850.1300 Daphnid chronic toxicity test
850.1350 Mysid chronic toxicity
850.1400 Fish early-life stage toxicity test
850.1500 Fish life cycle toxicity
850.1710 Oyster BCF
850.1730 Fish BCF
850.1735 Whole sediment acute toxicity invertebrates, freshwater
850.1740 Whole sediment acute toxicity invertebrates, marine
850.1790 Chironomid sediment toxicity test
850.1800 Tadpole/sediment subchronic toxicity test
850.1850 Aquatic food chain transfer
850.1900 Generic freshwater microcosm test, laboratory
850.1925 Site-specific aquatic microcosm test, laboratory
850.1950 Field testing for aquatic organisms
850.2100 Avian acute oral toxicity test
850.2200 Avian dietary toxicity test
850.2300 Avian reproduction test
850.2400 Wild mammal acute toxicity
850.2450 Terrestrial (soil-core) microcosm test
850.2500 Field testing for terrestrial wildlife
850.3020 Honey bee acute contact toxicity
850.3030 Honey bee toxicity of residues on foliage
850.3040 Field testing for pollinators
850.4000 Background-Nontarget plant testing
850.4025 Target area phytotoxicity
850.4100 Terrestrial plant toxicity, Tier I (seedling emergence)
850.4150 Terrestrial plant toxicity, Tier I (vegetative vigor)
850.4200 Seed germination/root elongation toxicity test
850.4225 Seedling emergence, Tier II
850.4230 Early seedling growth toxicity test
850.4250 Vegetative vigor, Tier II

850.4300 Terrestrial plants field study, Tier III
850.4400 Aquatic plant toxicity test using Lemna spp. Tiers I and II
850.4450 Aquatic plants field study, Tier III
850.4600 Rhizobium-legume toxicity
850.4800 Plant uptake and translocation test
850.5100 Soil microbial community toxicity test
850.5400 Algal toxicity, Tiers I and II
850.6200 Earthworm subchronic toxicity test

Appendix C: Ecological Models Used in Risk Assessment. [42]

1. GENEEC

The Generic Estimated Environmental Concentration aquatic Tier I risk assessment model is used to determine the need for a Tier II assessment.

Assumptions:

1. The runoff event transports a maximum of 10% of the pesticide remaining and available in the top 2.5 cm at the time specified above. This value is based on a review of empirical data.
2. Pesticides enter the pond in solution or attached to eroded sediment or via spray drift.
3. Applications are simulated (broadcast, disked in after broadcast, chisel plowed after broadcast, surface banded, banded-incorporated, T-banded, in-furrow, and by aerial or ground spray).
4. Application efficiency is 95% for aerial and spray blast, 99% for ground sprays and 100% for granular applications with the remainder drifting off site.
5. Five per cent of the application rate is loaded to the pond for each aerial or spray blast application and 1% of each ground spray application.
6. Degradation of pesticides entering the pond via drift begins immediately upon entry.
7. Pesticide degrades in soil via aerobic metabolic processes following a first-order decay and starts at the first application and occurs between multiple applications.
8. Runoff to the pond occurs two days after a single application or on the day of the last application of a multiple application series.
9. For pesticides, which are watered in, the pesticide runoff into the pond occurs on the same day it is applied.
10. Adsorption in the pond occurs simultaneously with chemical and biological degradation until binding equilibrium is reached (as a function of K_{oc}).
11. Degradation occurs separately in water and sediment phases after initial partitioning.

Limitations:

1. The meta model provides pesticide concentration values as a function of K_{oc}.
2. The screening mechanism is coarse in nature and therefore is subject to criticism.
3. The screen only considers a few basic chemical properties: application rate, number, and methods.
4. The scenario is designed to represent a high exposure condition and is thus deliberately conservative.

5. If the level of concern is not exceeded, it is assumed that the pesticide has low risk and therefore no additional assessment is conducted.
6. Risk may be overestimated because of the conservative assumptions rather than that the actual risk is above the level of concern.
7. Refined estimates with less uncertainty must be used as follow-up.

Notes:

In the current regulatory-tiered process for aquatic risk assessment, the GENEEC model is used as the regulatory touchstone to estimate environmental concentrations (EEC) for a pesticide in an edge-of-field water body for comparison with aquatic toxicity benchmarks to determine whether further risk characterization effort is warranted. GENEEC, developed by the EPA's Environmental Fate and Effects Division by Parker et al. (1995; 1997), provides screening level EECs for pesticides in an aquatic environment. Thus, it was intended to provide an upper-bound concentration value that might be found in ecologically sensitive areas because of pesticide use.

GENEEC is a single runoff event model, which can account for spray drift from multiple applications. Outputs presently provide 1) a maximum peak, 2) a 4-day average, 3) a 21- day, and 4) a 56-day mean EECs, using a few readily available environmental fate properties: soil/water partition coefficient and degradation half-lives to estimate runoff from a 10 hectare field into a one hectare by two meter deep farm pond with no outlet. GENEEC is generic with respect that it does not consider differences in climate, soils, topography, and crop. Currently, it only is capable of simulating crops that can be grown in something similar to a typical agricultural field: row crops, orchards, and turf.

GENEEC was specifically designed to mimic PRZM[43] and EXAMS[44] programs (discussed below), which are more complex computer models that are used in current Tier II assessments. GENEEC's primary virtues are its ease and speed of use, and the minimal data requirements. Thirty or forty GENEEC runs can be done in an hour that allow the screening of a large number of uses for a chemical. All the data used in GENEEC is available early in the registration process. This allows for preliminary assessment even at the Experimental Use Permit stage in many cases.

The inherent limitations of GENEEC in estimating aquatic exposures relate largely to the assumptions inherent in the meta-model and include the following:
1) Degradates, adjuvants, and diluents are not considered;
2) Only edge of field risks are considered;
3) Foliar interception and dissipation are not considered;
4) Volatilization is not considered;
5) Only considers a single rainfall event occurring after a specified time, depending upon number of pesticide applications and method;
6) Spray drift is only a function of application rate;
7) Maximum rates and applications and shortest application intervals are considered;
8) Assumes first-order degradation (aerobic soil metabolism rate) after each application;
9) The model does not account for chronic effects, but uses only acute endpoints.

2. PRZM: Tier II

The Pesticide Root Zone Model provides a means of estimating the effective concentrations of pesticide in the soil.

Assumptions:

1. High runoff soils are considered (Hydrologic C and D).
2. Assumes 100% of the watershed is treated with pesticide.
3. Maximum rates and applications and shortest application intervals considered.
4. Scenario represents 'edge of field' exposure.
5. Does not consider hydrology at watershed or basin scale.
6. Degradation of a pesticide in or on soil may be due to such processes as hydrolysis, photolysis, and microbial decay.
7. The foliarly applied pesticide is subjected to degradation (as a lumped first-order foliar degradation rate).
8. Transformation to metabolites occur as do losses through volatilization.
9. Adsorption and desorption are treated as instantaneous, linear, and reversible processes.
10. Foliar interception/dissipation is considered, but rate data are rarely available.
11. First-order degradation in soil pore water and on the soil surface.
12. Assumes that 1% and 5% of each application for ground and aerial spray applications, respectively, are directly deposited into the pond.

Limitations:

1. PRZM does not consider subsurface lateral water flow that could contribute to pesticide loads reaching surface water bodies.
2. PRZM simulates only downward movement of water and does not account for diffusive movement due to soil water gradients.
3. PRZM is dependant upon site-specific properties (e.g., curve numbers and precipitation event relative to pesticide application).
4. PRZM cannot address spatial and temporal variability.

Notes:

The Pesticide Root Zone Model (PRZM) was developed through the U.S. Environmental Research Laboratory in Athens, Georgia by EPA staff members and subcontractors who contributed to the development of various portions of the model's code. The first version of PRZM (PRZM-1) model was released in 1984, with the accompanying user's manual written by Carsel et al. 1984. PRZM-1 was upgraded to PRZM-2. The second version (PRZM-2) linked two models together: PRZM and VADOFT (Vadose Zone Flow and Transport Model).[45] The VADOFT model simulates one-dimensional water flow in the unsaturated zone using Richards' equation. Release number 2.0 (PRZM-2) became the official version of PRZM in the early 1990s.

PRZM-2 includes a non-uniform extraction algorithm for estimating pesticide runoff; bi-phase transformation of parent compound and metabolites; the ability to transform a parent compound from a sorbed phase to metabolites; metabolite loading transfer into EXAMS-2.97; enhanced flexibility in chemical applications and improved output features.

PRZM-2 became PRZM-3 when a septic system module and algorithms were added for modeling the fate and transport of soil nitrogen. Ultimately, the third release of PRZM (PRZM-3) resulted from the efforts of fifteen years of focused model development.[46]

The PRZM manual describes PRZM as a one-dimensional, dynamic, compartmental model that can be used to simulate chemical movement in unsaturated soil systems within and immediately below the plant root zone. PRZM was designed to provide a deterministic simulation of the fate of pesticides, applied for agricultural purposes, both in the crop root zone and the underlying vadose zone. The model is capable of simulating multiple pesticides or parent/daughter relationships and is also capable of estimating probabilities of concentrations or fluxes in or from various media for the purpose of performing exposure assessments. Predictions are made on a daily basis. Output can be summarized for a daily, monthly, or annual period.

3. EXAMS: Tier II

The Exposure Analysis Modeling System provides analyses of exposure, fate and persistence of pesticides.

Definitions:

1. Exposure: the expected environmental concentrations (EEC) resulting from a particular pattern of chemical loadings;
2. Fate: the distribution of the chemical in the system and the fraction of the loadings consumed by each transport and transformation process; and
3. Persistence: the time required for purification of the system (via export/transformation processes) should the chemical loadings cease .

Assumptions:

1. The pesticide is assumed not to radically change the environmental variables that drive its transformations.
2. Bacterial populations do not significantly increase (or decline) in response to the presence of the chemical.
3. EXAMS controls its computational range to ensure that the assumption of trace-level concentrations is not grossly violated.
4. EXAMS aborts any analysis generating EECs that exceed (the lesser of) 50% of the compound's aqueous solubility or 10 micromolar (10 -5 M) concentrations of a dissolved unionized molecular species.
5. The compartments are assumed to be 'well-mixed', that is, the reaction processes are not slowed by delays in transporting the compound from less reactive to more reactive zones in the volume element.
6. The transport of a chemical from a loading point into the bulk of the system takes place by advected flows and by turbulent dispersion.
7. The physical space of the system is broken down into a series of physically homogeneous elements connected by advective and dispersive fluxes.
8. Each compartment is a particular volume element of the system, containing water, sediments, biota, dissolved and sorbed chemicals, etc.
9. Pesticide loadings and exports are represented as mass fluxes across the boundaries of the volume elements.
10. Reactive properties are treated as point processes within each compartment.

Limitations:

1. EECs can be violated by spills.
2. A model with dynamic hydrology would more accurately reflect concentration changes due to pond overflow and evaporation.
3. Estimates derived from the current model simulate a closed-system, because the pond has no outlets, flowing water, or turnover.
4. It is assumed the inflow from runoff is exactly balanced by evaporative losses.
5. Fate data for degradates are often not available.

Notes:

EXAMS estimates exposure, fate, and persistence following release of an organic chemical into an aquatic ecosystem under certain constant environmental conditions. EXAMS consists of a number process modules that link fundamental chemical properties to the limnological parameters that control the kinetics of fate and transport in aquatic systems.[47] The chemical properties are measured by conventional laboratory methods, which are required under various regulatory data requirements. For the estimation of pesticide concentrations in surface water for aquatic risk assessments, the pesticide is loaded into the water body in response to precipitation events and spray drift from files created by PRZM.

EXAMS provides facilities for long-term (steady-state) analysis of chronic chemical discharges, initial-value approaches for study of short-term chemical releases, and full kinetic simulations that allow for monthly variation in mean climatological parameters and alteration of chemical loadings on daily time scales. Since EXAMS is a 'steady-state' model it does not accurately characterize the transient nature of water flow and pesticide influx. EXAMS was written in a generalized (N-dimensional) form in its implementation of the algorithms representing spatial detail and chemical degradation pathways.

The environment in EXAMS is represented through long-term average values of the forcing functions that control the behavior of chemicals. EXAMS is capable of considering steady state input loadings, pulse loads, and coupling to the output of the PRZM model, which can provide a lengthy time-series of contamination events due to runoff and erosion of sediments from agricultural lands.

EXAMS assumes that when a pesticide reaches an aquatic system, an entire array of transport and transformation processes begins at once to act on the chemical. The processes are combined into mathematical descriptions of their total effect on the rate of change of chemical concentration in the system. To use numerical techniques, the system is divided into a grid of spatially discrete elements, which are continuously varying in space and time. These elements are also referred to as 'grid points' or as 'compartments'.

Currently, Office Pesticide Programs, Environmental Fate and Effects Division (EFED) is using a standard water body that is maintained at a constant volume (20,000 L). The contributing area is also always assumed to be the same (10 ha). The physicochemical properties of this small water body are characteristic of a farm pond in Georgia. Daily edge-of-field loadings of pesticides dissolved in runoff waters and sorbed to entrained sediment, as predicted by PRZM, are discharged into the standard small water body simulated by the EXAMS model. Pesticide loading, through spray drift, is assumed to be a fixed percentage of the pesticide application rate, which is dependent upon method of application. The EXAMS can account for volatilization, sorption, hydrolysis, biodegradation, and photolysis of the pesticide within the water body. Although EXAMS considers many possible routes of dissipation, the environmental fate

properties such as aerobic and anaerobic aquatic half-lives (or degradation rates) are often lacking for inclusion as inputs into EXAMS.

4. MUSCRAT: Tier III

The Multiple Scenario Risk Assessment Tool, MUSCRAT, is a Windows application program developed to standardize and automate Tier III ecological risk assessments under FIFRA.

Limitations:

1. Risk characterization only reflects the variability associated with soil and weather for an index pond scenario.
2. Chemical applications are assumed to occur on the same day of the month for every year of simulation and do not reflect weather variability or other stochastic components.
3. Chemical degradation is neither climate nor soil dependent.
4. Constant drift factors do not reflect the variability that occurs with weather, application equipment, formulations, and other factors.

Strengths:

1. Automates and standardizes the Tier III ecological risk assessment process and is a tool built around the primary models used for pesticide evaluations under FIFRA.
2. Uses object-oriented (modular) technology so that future versions of PRZM, EXAMS, AgDrift or alternate environmental fate models could be substituted runoff maps.
3. Produces a distribution of exposure values across entire regions or use areas and thus embodies a great deal more data of value to the risk assessment process than do other models.

Notes:

A Tier III analysis involves selecting a crop and a region of interest from which a subset of scenarios is determined. For example, selecting cotton for all regions would result in 102 scenarios in 6 regions. Selecting a minor crop would result in substantially smaller number of regions and scenarios. MUSCRAT lists 23 crops for which 19 are currently active. A scenario represents a 10-hectare field draining to a 1-ha pond, as patterned after EPA's standard pond scenario for Tier II ecological risk assessments. MUSCRAT links chemical, crop, soil, and climate data bases and facilitates the creation of PRZM-3 and EXAMSII input files. In MUSCRAT, each region is subdivided into 25 runoff/erosion categories or 'bins' yielding 275 bins for the entire U.S. All agricultural soils within a region are allocated to a bin based on their respective runoff/erosion potential using a data set generated on the San Diego supercomputer for each soil polygon using local weather station data. Acreage within each bin is tabulated by crop based on 'crop suitability' as determined by the U.S. Department of Agriculture for those soils associated with the respective bin. Runoff/erosion potential is determined from annual water runoff and sediment yield predicted 30-year model simulations for each soil/weather combination.

The breadth and depth of this dataset offers potential for other approaches to scenario definition. Presently, chemical loadings to the pond are simulated for 36 consecutive years of pesticide application with a corresponding 36-year climate record. Chemical loadings include dissolved residues in runoff water, sorbed residues in eroded sediment, and drift. Dissolved and

eroded loads result from storm events and are predicted using PRZM-3. Drift loads are based on USEPA drift figures for the day of application as a function of the application method and the user can change these values based on data available to them.

Future versions of MUSCRAT may link AgDrift to calculate drift loads to the pond. EXAMS II as described above simulates dissipation in the pond. For each scenario, a normal probability analysis is performed on the annual maximum series of predicted exposure concentrations for a given exposure duration. The annual maximum series represents the maximum concentrations for each year of simulation determined from a rolling average evaluation for the year for a given exposure duration. Analyses are performed on the instantaneous maximum, 96-hour, 21-day, 60-day, 90-day, and 'long-term' durations. Other exposure durations may eventually be included in the analyses.

Appendix D: Representatives of Potential High-Risk Pesticides' Active Ingredients, by Class*

Chemical Class	Pesticide	Screening Discussion
Carbamate (insecticide, nematicide, acaricide, molluscicide)	Carbaryl	Direct exposures of carbaryl in or near waterways would be of concern as the chemical is highly toxic to aquatic organisms. Monitoring to assure carbaryl does not leach into waterways would be vital to the protection of endangered aquatic organisms.
Organophosphate (insecticide)	Diazinon	The risks associated with this chemical would predominantly occur at the time of application. This consultation would consist of identifying the presence of endangered species at the time of application. For example, given its high acute toxicity to birds, application methods may need scrutiny—diazinon is used as a seed dressing. Given birds' affinity for seed, this use may need to be discontinued. Perhaps cloth or netting need to be used over field crops directly after application. The no observed effect level for birds needs to be determined.
Organochlorine (fungicide)	Chorothalonil	Chlorothalonil is highly toxic to aquatic vertebrates and invertebrates, though if not applied near water, it is not likely to be found in waterways. It does suspend to organic matter once in water and is slow to biodegrade in still waters, therefore monitoring of fish and their habitat would be important. It is not likely to settle on the bottom of waterways. Therefore the benthic community may not be impacted in any way.
Dinitrophenyl (fungicide)	Dinocap	Dinocap should be monitored for its direct effects on terrestrial organisms. It is also highly toxic to aquatic invertebrates and vertebrates though it is unlikely to find its way into water unless directly applied. Care should be given to this chemical as it is suspected of being a developmental toxin. Therefore, direct exposure to endangered species could impact the recovery of the species.

Pyrethroid (insecticide)	Permethrin	Permethrin would be of primary concern to endangered terrestrial invertebrates. The chemical degrades rapidly and does not tend to persist in the environment. Therefore, direct application in areas where terrestrial invertebrates live would be the primary concern.
Phenoxy Compound (Herbicide)	2,4-D	2,4-D is of primary concern due to its high toxicity to aquatic vertebrates. It has a low soil – carbon coefficient. Coupled with its low soil persistence, this means it could readily move into waterways. Special emphasis should be placed on monitoring this chemical's use near waterways.
Triazine (herbicide)	Atrazine	Atrazine is of concern from an environmental exposure standpoint as well as for its toxicological profile. It is highly toxic to aquatic plants and is known to have sublethal and indirect effects on aquatic vertebrates and invertebrates. Due to its high mobility and solubility, it is likely to find its way into groundwater.
Substituted Urea (herbicide)	Diuron	Diuron is a highly persistent herbicide with a long half-life (greater than 6 months). Its effectiveness as a pre-emergent chemical makes its toxicity to endangered plants a great concern.
Halogenated Hydrocarbon (fungicide, insecticide, nematicide)	1,3-Dichloropropene	1,3-D is acutely toxic to terrestrial and aquatic invertebrates, as well as fish. This chemical is unlikely to have an acute exposure effect on endangered species. However, while it does volatilize readily in air, once in water it no longer volatilizes. Any effect would likely be the result of chronic exposure of terrestrial invertebrates at the time of application. Its contamination with the more highly toxic and persistent chemical 1,2-dichloropropane continues to put it in the forefront of chemicals of concern.

∗ Toxicological information for each pesticide is provided in **Appendix E**.

Appendix E: Representative Pesticide Toxicity Profiles

CARBARYL

Overview: Carbaryl has been used for about 30 years as a contact and ingestion insecticide with some systemic properties and controls a wide range of pests. The principal production plant is in the USA. Carbaryl is processed by more than 290 formulators into over 1500 different products.[48] The major degradation product is 1-naphthol.

Aquatic Invertebrates

Carbaryl is very toxic to aquatic invertebrates. The LC50 values for crustacea vary from .005 .009 mg/L (water fleas, mysid shrimps), .008 to .025 mg/L (scud), and .5 to 2.5mg/L (crayfish). Aquatic insects have a similar range of sensitivity. The toxicity of carbaryl on the early development of the sea urchin has been studied.[49] Developmental stages with active cleavage and cellular mobilization (blastula and gastrula) turned out to be the most sensitive endpoints. Daphnia magna were exposed to technical carbaryl for 21 days; reproductive performance was the most sensitive indicator; the maximum acceptable toxicant concentration was determined to be .0024 mg/L.[50]

Aquatic Vertebrates

For fish, most LC50 values are between 1 and 30 mg/L. Salmonids are the most sensitive group. A study conducted on the full life-cycle of fathead minnows (*Pimephales promelas*) exposed the fish to varying concentrations (0.008-0.68 mg/L) for 9 months, beginning with the larvae. Survival of fatheads after 6 months at 0.68 mg/L was lower than that in the controls. After 9 months at 0.68 mg/L, the mean number of eggs produced per female was reduced, the mean number eggs per spawning were also affected and no hatching occurred.[51] No other demonstrable effects were noted at 0.017, 0.062, and 0.21 mg/L concentrations; thus, the maximum acceptable toxicant concentration (MATC) for fathead minnows, exposed to carbaryl in water, was between 0.21 and 0.68 mg/L. The lethal threshold concentration for 2-month-old minnows was 9 mg/L. Another study exposed different stages of the embryo of carp (*Cyprinus carpio*) to carbaryl through the hatching stage. There was 100% mortality of carp eggs and embryos at 2.5 mg/L. There appeared to be no effect of carbaryl on hatching at 0.01-0.75 mg/L. However, there was decreased hatching at 1.0 mg/L and deformed larvae (3.3%) with enlargement of the pericardial sac and coiling of the posterior region of the embryo.[52]

Terrestrial Invertebrates

Carbaryl is toxic to honeybees and earthworms. Oral LD50 for honeybees is 1.2 mg/kg. Due to the highly toxic nature of the chemical to honeybees it should not be applied to crops during flowering stages. Stone flies and mayflies are also highly sensitive terrestrial invertebrates to the effects of carbaryl.

Terrestrial Vertebrates

The most susceptible bird tested is the red-winged blackbird (LD50= 56 mg/kg).

Plants
The rate of decomposition of carbaryl in plants depends on the climatic conditions. It is more rapidly decomposed in hot climates, at high temperatures, and by intensive ultraviolet radiation. Thus, residual levels in feed plants were lower in regions with a hot climate than in other regions.[53]

Environmental Exposure
Carbaryl has a low level of persistence in soil. The chemical breaks down primarily by sunlight and bacterial action. It binds to soil and can find its way into water in run-off.

DIAZINON
Overview: Diazinon is a contact organophosphate insecticide with a wide range of insecticidal activity. It is effective against adult and juvenile forms of flying insects, crawling insects, and spiders. It has been used since the 1950s. Diazinon is mainly formulated as wettable powders and emulsifiable concentrate. It is also available in mixed formulations with other insecticides. Metabolites include diethylphosphoric acid, diethylthiophosphoric acid and the derivates of the pyrimidinyl ring.

Aquatic Invertebrates
Diazinon has been found in large rivers and major aquifers where it is highly toxic to aquatic invertebrates. It has been detected in the surface water of 24 states and in river systems including the Mississippi and the Rio Grande. Sublethal effects on aquatic invertebrate behavior have been reported at .1 and .01 mg/L. Acute LC50 values for aquatic invertebrates range from 0.2 µg/L for *Gammarus fasciatus* to 4.0 µg/L for the shrimp *Hyallela azteca* in 96-h tests. Molluscs are substantially less sensitive according to a single test on the snail *Gillia attilis*.

Aquatic Vertebrates
Diazinon is highly toxic to freshwater, estuarine, and marine animals. Acute LC50 values for fish range from 0.09 mg/L for rainbow trout (*Oncorhynchus mykiss*) to 3.1 mg/L for the catfish (*Channa punctatus*). Growth of early life stages of fish was inhibited at concentrations between 0.01 and 0.2 mg/L. Brain acetylcholinesterase activity is suppressed following acute exposure to diazinon. As for the metabolites, pyrimidine analogue was found in all fish species, but diazinon and related compounds were found only in carp and rainbow trout.[54] Field kills of waterfowl have been reported following use of the compound on amenity turf. It has been suggested that precautions should be taken to minimize exposure of non-target aquatic vertebrates, such as do not spray over water bodies, minimize exposure by spray drift, and avoid areas where wildfowl are likely to graze.

Terrestrial Invertebrates
Diazinon is highly toxic to honeybees. The LD50 for earthworms is 130 mg/kg.

Terrestrial Vertebrates
Diazinon is highly toxic to birds. In 1988 the EPA concluded that the use of diazinon in open areas poses a widespread and continuous hazard to birds. Bird kills associated with the chemical have been reported in every area of the country.

Environmental Exposure
Based on information available in the USEPA Ecological Incident Information System (EIIS), diazinon has caused the second largest number of total known incidents of bird mortality of any pesticide, exceeded only by carbofuran (a widely used agricultural pesticide with many of its granular uses phased out). The highest number of incidents (58) caused by diazinon occurred in the years 1994-1998.

CHLOROTHALONIL
Overview: Chlorothalonil is a broad-spectrum organochlorine fungicide. This chemical can contaminate the air traveling beyond the field and has been found in nearby residential neighborhoods. It is persistent in soils and acutely toxic to fish, crab, frogs, and water fleas.

Aquatic Vertebrates and Invertebrates
Chlorothalonil does not break down in aqueous solutions between pH 5 or 7 in the dark. In sunlight, it degrades slowly to a 4-hydroxy metabolite in water, more quickly in soil, having a half-life of 26-45 days, producing five different metabolites. It is highly toxic to fish and aquatic invertebrates. In laboratory studies, the LC50 concentrations for a range of fish and invertebrates are similar and below 0.5 mg/L. A single study indicated reproductive effects in aquatic invertebrates following continuous exposure. When two generations of *Daphnia magna* were exposed to technical chlorothalonil at levels of 6.2, 12, 25, 50 and 100 µg/L for 21 consecutive days during each generation, adverse effects on adult survival and reproduction were observed at a nominal concentration of .1 ppm.

When exposed to chlorothalonil, Rainbow trout (*Oncorhynchus mykiss)* and *Galaxias sp* showed marked lethargy, the degree increasing with time and concentration of exposure. In *O. mykiss*, loss of startle reaction was followed by reduction of activity, and permanent lethargy was followed by loss of righting ability and death. In *Galaxias sp*, the onset of lethargy was accompanied by varying degrees of fin collapse.[55]

Terrestrial Invertebrates
Chlorothalonil, at dose levels up to 5000 mg/L in bacterial suspensions, inhibited the growth of three strains of *Rhizobiumjaponicum*.[56]

Terrestrial Vertebrates
Chlorothalonil is of low toxicity to birds with a reported acute oral LD50 of 4640 mg/kg diet in the mallard duck. No significant reproductive effects have been reported. The acute oral toxicity of the 4-hydroxy metabolite is greater than that of chlorothalonil itself (acute oral LD50 of 332 mg/kg).

Plants
In plants, chlorothalonil is metabolized only to a limited extent to the 4-hydroxy metabolite. The majority of the residue remains as the parent compound. Generally less than 5% of the total residue is present as the 4-hydroxy metabolite. A review of plant residues worldwide showed that the 4-hydroxy metabolite level was <0.1 mg/kg in most of the crops analyzed.

Environmental Exposure
The compound both adsorbs to suspended material and is degraded rapidly in the environment. Chlorothalonil is removed from water by strong adsorption to suspended matter. Modeled data suggest little or no partition to bottom sediment. Chlorothalonil is rapidly degraded in soil, and degradation may occur in water with the production of the 4-hydroxy metabolite, 4-hydroxy-2,5,6-trichloroisophthalonitrile. Studies with river water from two sources have shown that loss of chlorothalonil is slow in still water. However, biodegradation is unlikely to play a major role in the fate of chlorothalonil in moderate to fast flowing streams, where volatilization and adsorption are liable to be dominant factors.[57] Because chlorothalonil adsorbs strongly to organic matter in soil and suspended material in water, it does not, therefore, leach readily from soil to groundwater.

DINOCAP
Overview: Dinocap is the common name for a mixture of 2,4-dinitro-6-octylphenol crotonate (70-72%) and 2,6-dinitro-6-octylphenol crotonate (28-30%). Dinocap is most commonly sold under the trade names Karathane, Mildex, and Dikar, the latter of which is a mixture of dinocap and mancozeb. Dinocap is registered to eradicate powdery mildew diseases on agricultural crops (apples, apricots, cantaloupes, cucumbers, grapes, melons, peaches, pears, pumpkins, raspberries, and squash), nursery stock, ornamentals, and home gardens.

Aquatic Invertebrates and Vertebrates
Dinocap is highly toxic to invertebrates and vertebrates. The LC50 for rainbow trout is .015 mg/L and .02 mg/L for bluegill. The LC50 for the sideswimmer (an aquatic invertebrate) is reported as .075 mg/L.

Terrestrial Vertebrates
A special review was initiated on the basis of two oral teratology studies showing the chemical caused developmental malformations. The developmental toxicity of dinocap was studied in laboratory animals. Pregnant CD-1-mice, Sprague-Dawley-rats, and Syrian-hamsters were administered dinocap orally. Developmental toxicity was evaluated in each species by using an in-vivo teratology screen that involved determining postnatal viability and body weight gain, and observing morphological and behavioral development. Maternal toxicity was also observed. In mice, dinocap showed no maternal toxicity. Dinocap at 25mg/kg/day caused increased postnatal mortality. Many of the offspring that died were ballooned and had cleft palates.[58]

Plants
Dinocap is absorbed and broken down by treated plants. It tends to readily penetrate foliage and is not likely to be washed off by rain. The residual period of dinocap in plants is 1-2 weeks.

Environmental Exposure
Dinocap does not tend to persist in the soil environment. It is highly photosensitive breaking down by sunlight as well as by microorganisms. Because it is also slightly soluble in water, it is unlikely to contaminate groundwater. If the chemical does find its way into water sources it will adsorb to suspended matter or attach to sediment.

PERMETHRIN
Overview: Permethrin is a broad-spectrum synthetic pyrethroid insecticide. Its metabolites show higher acute (oral or intraperitoneal) toxicity than permethrin itself. It possesses a high level of activity against Leptidoptera and is also effective against Hemiptera, Diptera, and Coleoptera.

Terrestrial Vertebrates
Permethrin has very low toxicity to birds when given orally or fed in the diet. The LD50 is greater than 3000 mg/kg body weight for acute single oral dosage. The chemical has not shown an effect on reproduction in the hen at 40 mg/kg diet.

Terrestrial Invertebrates
Permethrin is highly toxic to honey bees. The topical LD50 is 0.00011 mg/bee, but there is a strong repellent effect of permethrin to bees that reduced the toxic effect in practice. There is no evidence for significant kills of honeybees under normal use. Permethrin is more toxic to predator mites than to the target pest species.

Aquatic Invertebrates and Vertebrates
In laboratory tests, permethrin has been shown to be highly toxic to aquatic arthropods, LC50 values ranging from 0.018 ppb for larval stone crabs to 1.26 µg/L for a cladoceran. Permethrin is less toxic to aquatic molluscs and amphibia, 96-h LC50 values being greater than 1 mg/L and 7 mg/L, respectively. Permethrin is highly toxic for fish, with 96-h LC50 values ranging from .00062 mg/L for larval rainbow trout to .314 mg/L for adult rainbow trout. The NOEL for early life stages of the sheepshead minnow over 28 days is .01 mg/L and the chronic no-effect level for fathead minnow is .0014 mg/L.

Environmental Exposure
Permethrin strongly adsorbs to sediment and rapidly degrades. Sediment-bound permethrin could be toxic to burrowing organisms though its rapid degradation would make the effect temporary.

2,4-D
Overview: 2,4-D is a selective herbicide that kills broad-leaved plants but not grasses or conifers. Its chemical structure is a modification of a naturally occurring plant hormone. 2,4-D is available as the free acid but is used, in agriculture and forestry, in formulations as a salt or ester.

Aquatic Invertebrates and Vertebrates
Generally, 2,4-D is relatively non-toxic aquatic invertebrates. The short-term toxicity data on the effects of 2,4-D free acid, its salts, and esters on aquatic invertebrates are

extensive. Ester formulations are more toxic than the free acids or salts. Sensitivity variations exist among species in response to the same formulation. Organisms become more sensitive to 2,4-D when the water temperature increases. Reproductive impairment occurred at concentrations below 0.1 of the short-term toxic levels determined for these formulations.

2,4-D is clearly toxic to fish at early life-stages. Studies on fish eggs and larvae immediately after hatching have been conducted on few species and mainly with simple salts of 2,4-D. There is little information on the effects of the more toxic esters. Free-swimming larvae were more sensitive to 2,4-D than eggs; the rate of survival of embryos in tests lasting between 12 and 48 h was higher than for larvae. In tests lasting between 24 and 48 h, at concentrations of 2,4-D above 400 mg/L, no larvae survived. Embryos showed malformation and reduced mobility at concentrations above 100 mg/L, and at concentrations of 800 mg/L or more, embryos were immobile (Biro, 1979).[59] Although the free acid is the physiologically toxic entity, the ester formulations represent a major hazard to fish.

Aquatic Plants

Nitrogen fixation is affected at high concentrations (400 mg/L) of 2,4-D acid. An effect of 2,4-D esters on nitrogen fixation occurs from a concentration of 36 mg/L upwards. This is significant in that cyanobacteria (blue-green algae) are major sources of nitrogen in waterways.

Terrestrial Invertebrates

Based on the widespread use of 2,4-D and its formulations, insects of many kinds could be exposed to the material. Although the compounds are generally classified as non-toxic to beneficial insects, such as honeybees and natural enemies of pests, some adverse effects have been reported on the early life-stages and adults of some insects. At moderate levels of exposure, honeybee's brood production can be seriously impaired.

Terrestrial Vertebrates

2,4-D is moderately toxic to birds. The LD50 is 1000 mg/kg in mallards and 272 mg/kg in pheasants. There is some disagreement in the literature about the toxicity of 2,4-D to birds' eggs. The low uptake of the material through the eggshell suggests that exposure would not affect hatching in normal use of the compound. Adult birds are not affected by short-term exposure to 2,4-D. The likelihood of prolonged exposure of either adult birds or eggs to high levels of 2,4-D is small.

Environmental Exposure

2,4-D is rapidly degraded in the environment predominantly by microorganisms and has low soil persistence.

ATRAZINE

Overview: Atrazine is used extensively in agriculture. The toxic effects attributed to the hydroxy-metabolites are not equivalent to atrazine; therefore, risks associated with exposure to these metabolites need to be assessed separately: desethyl atrazine (DEA), desisopropyl atrazine (DIA), diaminochloro triazine (DACT), hydroxyatrazine, desethylhydroxyatrazine, desisopropylhydroxyatrazine, and diaminohydroxyatrazine.

Aquatic Vertebrates and Invertebrates

Considering atrazine's heavy and extensive use especially in agriculture, its widespread presence in surface and ground water, its adverse toxicological effects on freshwater and estuarine plants as well as its indirect effects on aquatic invertebrate and fish populations, continued atrazine use is likely to pose a risk to the health and integrity of aquatic communities.

Reported sub-lethal effects of atrazine include endocrine effects in bass and frogs, and olfactory effects in salmon.

Environmental Exposure

If released into water, atrazine may adsorb somewhat to suspended solids and sediment. Atrazine may persist in salt marsh sediments. Atrazine in surface water may kill aquatic plants and the decaying process of dead plants may lower dissolved oxygen to levels too low for fish survival. It is known to increase the toxicity of organophosphate insecticides, such as chlorpyrifos, and a number of other pesticides that may have been applied earlier to atrazine-treated crops or applied in other fields upstream in the watershed.

Terrestrial Concerns

Atrazine can contaminate nearby non-target plants, soil and surface water via spray drift during application. Atrazine is applied directly to target plants during foliar application, but pre-plant and pre-emergent applications are generally far more prevalent.[60]
Damage to amphibia is evident in recent studies.

Appendix F: Toxicological and Exposure Data for Identified Pesticides

Chemical Class	Pesticide	Acute Endpoints				Chronic Endpoints	Water Solubility	½ Life (persistence)	K_{oc} (Mobility)	Log K_{ow}
		LD50 Mg/kg	LC50 Mg/L	EC25 Terres-trial Plants	EC50 Aquatic Plants	NOEL/ NOEC ppm	Mg/L			
Carbamate	Carbaryl	56 *f* 1‡ 150 ¤	1 † .005*		.19 ppm	1.8¤ (Repro) .09 †	40	20 days	350	1.59
Organophos-phate	Diazinon	1250¤ 130 ‡ 1.1*f*	.0002 * .01†			10¤ (repro)	40	21 days	500	3.4
Organo-chlorine	Chloro-thalonil (4 Hydroxy Meta-bolite)	332 ¤ 4640 *f*	.5*† .07*	>16 ai/acre NOEL	50 ppb NOEC	15 ¤ 10¤ (Repro) 830 *	.6	60 days	1380	2.8
Dinitrophenyl	Dinocap	980¤ 790*f*	.015†			200¤ (Repro)	<.1	5 days	550	4.5
Pyrethroid	Perme-thrin	.00011‡ 3000*f* 2000¤	.314† .0078 *		2.8	100 ¤ 1000¤ (repro) 1* 10 †	.2	28 days	100,000	6.5
Phenoxy Compound (WHO-EHC)	2,4-D	272*f* .104‡ 1200¤	3.1* .315†		.55	1¤	900	5 days	20	2.6
Triazine	Atrazine	750¤ 2000*f*	5.3† .72*		.001	50¤ .062* (fish repro) 100 (frogs)	28	100 days	100	2.7

Substituted Urea	Diuron	3400¤	2.5* 3.5†			125¤ (repro)	42	180 days	480	2.7
Halogenated Hydrocarbon	1,3-D	>10,000*f* 215¤ .044‡	.79* 3.9†		4.95	60¤ (repro)	2.0	32	1380	1.98

***f* Most Sensitive Terrestrial Avian Species**
‡ Most Sensitive Terrestrial Invertebrates
¤ Most Sensitive Terrestrial Vertebrate Species
† Most Sensitive Aquatic Vertebrates/Fish Species
*** Most Sensitive Aquatic Invertebrates Crustaceans**

References:

[1] To "jeopardize" is to "engage in an action that reasonably would be expected, directly or indirectly, to reduce appreciably the likelihood of both the survival and recovery of a listed species in the wild by reducing the reproduction, numbers or distribution of that species." 50 C.F.R. § 402.02.

[2] Center for Biological Diversity, Letter to Christine Todd-Whitman, Secretary of EPA regarding 60-day intent to sue, dated 3 June 2002.

[3] McKay, Paul. The Pilgrim and the Cowboy, McGraw-Hill, New York, 1989.

[4] The Endangered Species Act of 1973.

[5] Whitford, Fred et al. Pesticides and Wildlife. Purdue Cooperative Extension Service, 2001.

[6] Muirhead-Thomson, R.C. Pesticide Impact on Stream Fauna with Special Reference to Macroinvertebrates (Cambridge University Press: Cambridge, UK, 1987).

[7] Federal Insecticide, Fungicide and Rodenticide Act of 1947, Chapter 125, Section 136 et seq.

[8] Fite, Ed. The Environmental Protection Agency's Avian Pesticide Assessment Model. Wildlife Toxicology and Population Modeling 9.48 519-530.

[9] FIFRA, 3 USC 136 et seq.

[10] Glase, Linda. Wildlife Mortality Attributed to Organophosphate and Carbamate Pesticides, National Biological Service, 2001.

[11] Lyon, Linda. Endangered Species Bulletin 21.2, 1996.

[12] United States Fish and Wildlife Service, "General Statistics for Endangered Species" available electronically at http://ecos.fws.gov/servlet/TessStatReport 24 July, 2002.

[13] Endangered Species Act. Section 7(a)(2).

[14] Whitford, Fred et al. Pesticides and Wildlife. Purdue Cooperative Extension Service, 2001.

[15] See Gomi, T., R.C. Disle and J.S. Richardson. Understanding Processes and Downstream Linkages of Headwater Systems. BioScience 2002; 52: 9905-915.

[16] See Johansen, J.A. and G.H. Green. Archives of Environmental Contamination and Toxicity 1990; 19: 610-616.

[17] EPA, Guidelines for Ecological Risk Assessment. EPA/630/R-95/002F 1998.

[18] Gardner R. et al. Ecological Risk Estimation. (Lewis Publishers: Chelsea MI, 1992), 252.

[19] Suter, G.W., II Editor. Ecological Risk Assessment (Lewis Publishers: Chelsea MI, 1993), 538.

[20] Landis, W.G. and Y. Ming-Ho. Introduction to Environmental Toxicology: Impacts of Chemicals Upon Ecological Systems. (Lewis Publishers: Chelsea, MI, 2000, 285.

[21] www.Aqex.ecofram_Peer01_May499.doc

[22] Van Leeuwen, C.J., J.M. Hermens. Risk Assessment of Chemicals: An Introduction (Dordrecht, Netherlands: Kluwer, 1995).

[23] Haynes, Tyrone. Atrazine-Induced hermaphroditism at 1 ppb in American Leopard Frogs: Laboratory and Field Evidence. Environmental Health Perspectives 2002; 895-896.

[24] Reynoldson, T. Interactions between sediment contaminants and benthic organisms. Hydrobiologia 1987; 149:53-66.

[25] Baker, R.A. Contaminants and Sediments (Ann Arbor Science: Ann Arbor MI, 1980) 445.

[26] Branson, D.R. Predicting the fate of Chemicals in the Aquatic Environment from laboratory Data. ASTM-STP, American Society for Testing Materials 1978:55-70.

[27] Urban, D.J. and N. Cook. Hazard Evaluation Division Standard Evaluation Procedure Ecological Risk Assessment. United States Environmental Protection Agency 1986:5.

[28] Forbes, V.E. and P. Calow. Extrapolation in Ecological Risk Assessment: Balancing pragmatism and Precaution in Chemical Controls Legislation. BioScience 2002; 52.3: 249- 257.

[29] Sawada Y., Y. Nagai, I. Yamamoto, Probable toxicity of surface-active agent in commercial herbicide containing glyphosate. Lancet 1988; 1:299.

[30] International Program on Chemical Safety, Global Assessment of the State of the Science of endocrine disruptors, World Health Organization, 2002.

[31] International Program on Chemical Safety, Global Assessment of the State of the Science of endocrine disruptors, World Health Organization, 2002

[32] Office of Prevention, Pesticides, and Toxic Substances. Harmonized Test

Guidelines, Series 850 Ecological Effects Test Guidelines. EPA 2001.

[33] Hall, Carl. Pesticides and Flawed Frogs: Researchers Reveal First signs Linking Land Runoff to Deformities. San Francisco Chronicle 9 July, 2002.

[34] Scholz, N.L., N.K. Truelove, B.L. French, B.A.Berejikian, et al. Diazinon Disrupts Antopredator and Homing Behaviors in Chinook Salmon (*Oncorynchus tshawytscha*). Canadian Journal of Fish and Aquatic Science 2000; 57: 1911-1918.

[35] Brewer, S.K., et al. Behavioral dysfunctions correlate to altered physiology in rainbow trout exposed to cholinesterase-inhibiting chemicals. Arch Environ Contam Toxicol 2001; 40(1): 70-76.

[36] Call, D.J. et al. Bromacil and Diuron herbicides: toxicity, uptake, and elimination in freshwater fish. Arch Environ Contam Toxicol 1987; 16: 067-613.

[37] Reddy, D. et al. Changes in Erythropoietic activity of *sarotherodon mossambicus* exposed to sublethal concentrations of the herbicide Diuron. Bull Environ Contam Toxicol 1992 49: 730-737.

[38] Dodson S., T. Hanazato. Commentary on Effects of Anthropogenic and natural organic chemicals on development, swimming behavior, and reproduction of Daphnia, a key member of aquatic ecosystems. Env Hlth Perspectives 1995; 103. suppl. 4: 103-107.

[39] James R, K. Sampath. Combined toxic effects of carbaryl and methyl parathion on survival, growth, and respiratory metabolism in Heteropneustes fossilis (Bloch). ACTA Hydrobiol 1994; 36.3: 399-408.

[40] Arunachalam S., S. Palanichamy. Sublethal effects of carbaryl on surfacing behavior and food utilization in the air-breathing fish, *Macropodus cupanus*. Physiol Behav 1982; 29.1: 23-28.

[41] Office of Prevention, Pesticides and Toxic Substances. Harmonized Test Guidelines Series 850 Ecological Effects Test Guidelines.

[42] Pesticide Ecological Model Discussion obtained from EPA's Center for Exposure Assessment Modeling (CEAM), http://www.epa.gov/ceampubl/ Note that the listed Limitations in each table are those recognized by the EPA.

[43] Carsel, R.F., C.N. Smith, L.A. Mulkey, J.D. Dean, and P. Jowse. 1984. User's manual for pesticide root zone model (PRZM): Release 1, Report EPA-600/3-84-109, Environmental Research Laboratory, Office of Research and Development, U.S. Environmental Protection Agency. Athens, GA.

[44] Burns, L.A. 1991. Exposure analysis modeling system: user's guide for EXAMS II, version 2.94. Environmental Research Laboratory, Office of Research and Development, U.S. Environmental Protection Agency. Athens, GA.

[45] Mullins, J.A., R.F. Carsel, J.E. Scarbrough, and A.M. Ivery. 1993. PRZM2 User's manual, version 1.0. Environmental Research Laboratory, Office of Research and Development, U.S. Environmental Protection Agency. Athens, GA.

[46] Carsel, R.F., J.C. Imhoff, P.R. Hummel, J.M. Cheplick, and A.S. Donigian Jr. 1998. PRZM3, A Model for Predicting Pesticide and Nitrongen Fate in the Crop Rot and Unsaturated Soil Zones: Users Manual for Release 3.0. National Exposure Research Laboratory, Office of Research and Development, U.S. Environmental Protection Agency, Athens, GA (draft).

[47] Burns, L.A. 1997. Exposure analysis modeling system: user's guide for EXAMS II, version 2.97.5. . Ecosystems Research Division, Environmental Research Laboratory, Office of Research and Development, U.S. Environmental Protection Agency. Athens, GA.

[48] International Program on Chemical Safety, Environmental Health Criteria #153, Carbaryl, World health Organization, 1994.

[49] Hernandez, D.A., R.L. Lombardo, L. Ferrari, and M.C. Tortorelli. Toxicity of ethyl-parathion and carbaryl on early development of sea urchin. Bull Environ Contam Toxicology 1990; 45: 734-741.

[50] Surprenant, D.C. The chronic toxicity of carbaryl technical to *Daphnia magna* under flow-through conditions. Wareham, Massachusetts, Springborn Bionomics, Inc. (Report No. BW-85-7-1813) (Unpublished proprietary information submitted to WHO by Rhône-Poulenc Agro, Lyon) 1985.

[51] Carlson, A.R. Effects of long-term exposure to carbaryl (Sevin) on survival, growth and reproduction of the fathead minnow (*Pimephales promelas*). J Fish Res Board Can 1972; 29(5): 583-587.

[52] Kaur, K. and H.S. Toor. Toxicity of pesticides to embryonic stages of *Cyprinus carpio communis Linn*. Indian J Exp Biol 1977; 15: 193-196.

[53] Atabaev, S.T. Carbamate compounds. In, Pesticides and hygiene of the environment in the conditions of warm climate. 1972 Tashkent, Medizina, pp 114-121 (in Russian).

[54] Seguchi, K. and S. Asaka. Intake and excretion of diazinon in freshwater fishes. Bull Environ Contam Toxicol 1981; 27: 244-249.

[55] Davies, P.E. and R.W.G. White. The toxicology and metabolism of chlorothalonil in fish I. Lethal levels for *Salmo gairdneri*, *Galaxias maculatus*, *G. truttaceus* and *G. auratus* and the fate of 14C-TCIN in *S. gairdneri*. Aquat Toxicol 1985; 7: 93-105.

[56] Tu, C.M. Effect of fungicides on growth of *Rhizobium japonicum* in vitro. Bull Environ Contam Toxicol 1980; 25: 364-368.

[57] Davies, P.E. Disappearance rates of chlorothalonil (TCIN) in the aquatic environment. Bull Environ Contam Toxicol 1988; 40: 405-409.

[58] Gray, L.E. et al. Prenatal Exposure To The Fungicide Dinocap Causes Behavioral Torticollis, Ballooning And Cleft Palate In Mice, But Not Rats Or Hamsters. Teratogenesis, Carcinogenesis, and Mutagenesis 1986; 6.1: 33-43.

[59] Biro, P. Acute effects of the sodium salt of 2,4-D on the early developmental stages of bleak, *Alburnus alburnus*. J. Fish Biol. 1979; 14: 101-109.

[60] Rabert, William, J. Lin, M. Frankenberry and D. Urban, US Environmental Protection Agency, Environmental Fate & Effects Division, Reregistration Eligibility Science Chapter for Atrazine, 22 April, 2002.