Pesticide Action Network North America
49 Powell St., Suite 500
San Francisco, CA 94102
Phone: (415) 981-1771
Fax: (415) 981-1991
www.panna.org
www.pesticideinfo.org

April 16, 2004

Mr. Gary Frazer
Assistant Director for Endangered Species
U.S. Fish and Wildlife Service
4401 North Fairfax Drive, Room 420
Arlington, VA 22203

Dear Mr. Frazer,

We write to comment on **1018-AI95**, the **Joint Counterpart Endangered Species Act Section 7 Consultation Regulations**, 69 Fed. Reg. 4465 (Jan. 30, 2004) on behalf of Pesticide Action Network North America, WaterKeepers Northern California, DeltaKeeper, Farmworker Justice Fund, World Wildlife Fund, Oregon Natural Resources Council, California Rural Legal Assistance Foundation, Northwest Environmental Defense Center, Northwest Environmental Advocates, Headwaters, Northcoast Environmental Center, Friends of Del Norte, Lane County Audubon Society, Central Coast Environmental Health Project, Kalmiopsis Audubon Society, California Indian Basketweavers Association, Center for Sierra Nevada Conservation, Sierra Nevada Alliance, Fresno Coalition Against the Misuse of Pesticides, Helping Our Peninsula's Environment, and Professor Steve Sheffield at George Mason University to submit the following comments on the Joint Counterpart Endangered Species Act Section 7 Consultation Regulations. We include by reference the substantive points of the letters from Defenders of Wildlife, *et al*., Beyond Pesticides *et al.,* Earthjustice, and Washington Toxics Coalition *et al*. commenting on these regulations (1018-AI95).

The proposed regulations would transfer authority to determine whether a pesticide "may affect" an endangered species from the U.S. Fish and Wildlife Service (USFWS) and the National Marine Fisheries Services (NMFS) ("the Services") to the U.S. EPA. We urge you not to adopt the Proposed Regulations for the following reasons:

- U.S. EPA's exposure modeling is incomplete and inadequate for predicting exposures endangered species may experience.
- U.S. EPA does not adequately assess the effects of pesticide toxicity on endangered species.
- U.S. EPA does not have the in-house biological expertise to accurately make these "may affect" determinations.

The combination of these factors results in false "no effect" determinations from U.S. EPA's ecological risk assessments and/or no actions taken to mitigate known adverse effects. Such decisions jeopardize protected species and/or adversely modify critical habitat, contrary to the mandate of the Endangered Species Act. Consultation with the Services was written into the law to ensure that a U.S. EPA action of registering a pesticide and placing restrictions on its use would involve those agencies specializing in wildlife biology in the decision-making process. The Administration's proposal to allow U.S. EPA to legally avoid consultation is a blatant violation of the Services' authority and will likely result in increasing harm to endangered species. We urge you to reject these proposed regulations, and instead work to strengthen the consultative process between U.S. EPA and the Services.

As chemicals designed to be both toxic to living organisms and intentionally released into the environment with few if any controls on their dispersal, pesticides are one of the most significant toxic threats to endangered and threatened species. Many of these pesticides have broad spectrum toxicity to a wide range of living organisms, with impacts extending beyond the target organism to non-target wildlife and plant species. Pesticides may have direct lethal or sublethal impacts on a species, or may have indirect impacts by degrading a species' habitat, reducing its food supply or impairing its ability to reproduce or survive. The consultation process plays an important role in ensuring that pesticides will not adversely affect endangered species. By transferring to U.S. EPA much of the authority for determining whether a pesticide "may affect" endangered species, the Administration's proposed changes to the regulations would result in decreased protection and increased "take" of endangered species for the following reasons.

U.S. EPA's exposure modeling is incomplete and inadequate for predicting exposures endangered species may experience

To assess risk, EPA adopts a tiered approach using modeling that estimates the expected environmental concentrations ("EEC") of a given pesticide based on normal use in the field. If the estimated environmental concentration of a given pesticide is above a certain concentration, then EPA will proceed to the next "tier," in which more refined models are applied to assess environmental risk. For pesticide impacts on wildlife, EPA applies a four-tier approach. If at any tier, EPA finds that a pesticide does not pose an environmental risk, EPA ends the risk assessment process and makes a "no effect" determination. *See e.g.,* 54 Fed. Reg. 28003-28004. U.S. EPA's methods do not accurately assess risk to endangered species because the exposure assessment is incomplete and the prediction of fate and transport of pesticides into the environment does not reflect take into account some transport pathways.

Neither dermal nor inhalation exposures are evaluated or considered in U.S. EPA's ecological risk assessment process

Endangered species may be exposed to pesticides through several different routes, including:
1) **Dietary:** Direct ingestion of contaminated materials or prey or through grooming,
2) **Dermal:** Absorption of pesticides through the skin, and
3) **Inhalation:** Absorption of pesticides through the lungs by breathing air contaminated with spray or volatilization drift.

One might depict the exposure equation as follows:

$$E_{total} = E_{dietary} + E_{dermal}\ E_{inhalation}$$

U.S. EPA's exposure assessment determines whether exposures exceed a Level of Concern (LOC) by considering only the dietary component (without evaluation of exposures from grooming) and omits all reference to exposures through dermal or inhalation routes.

$$E_{total} = E_{dietary} + E_{dermal}\ E_{inhalation}$$

Since the exposure assessment is the basis of any and all further action to protect endangered species, the fact that two substantive components of the equation are summarily ignored suggests that EPA

falsely determines (on the basis of *no* information) that many pesticides have "no effect" on endangered species because exposures are deemed insignificant.

These unquantified exposures can be significant, especially from spray drift during the application and from post-application volatilization drift:

Spray drift: Application methods such as aerial spraying, high-speed dust blowers or high pressure spray rigs can result in extensive off-site spray drift at the time of application. Spray or dust applications of any type that are conducted when windspeeds are high are likely to result in off-site transport by prevailing winds. Each of these processes offers the potential for pesticides to contaminate endangered species habitat and or endangered species directly. Grooming of fur or feathers contaminated by spray drift can lead to additional oral exposure. While the typical range for spray drift of is 100 to 300 meters, longer distances of up to 10 miles have been well documented.[1]

Post-application volatilization drift: Field studies show that post-application volatilization drift can be expected for volatile and semi-volatile pesticides.[2, 3] Pesticides with vapor pressures above 0.000001 (10^{-6}) mm Hg can move off-site through volatilization drift, with prevailing winds determining the direction of movement of the pesticide plume from a particular application site. Any endangered species in the area downwind of the application site will be exposed to this toxic plume. Analysis of air monitoring studies conducted by the California Air Resources Board shows that concentrations of pesticides in air near application sites (75–300 ft.) exceed acute Reference Concentrations (RfCs) for humans (see Figure 1 below). Small mammals and birds have much higher respiratory rates per kilogram of body mass and will be exposed proportionately more than humans through inhalation, thus concentrations of airborne pesticides are likely to be exceeding RfCs for these animals as well.

Numerous studies demonstrate the potential for pesticides to drift through volatilization, thereby contaminating non-target sites. A USGS study detected 17 pesticides in air around the Sacramento region of California.[4] Other studies, including air monitoring performed by the California Air Resources Board, demonstrate that pesticides routinely drift from their point of application.[2, 5]

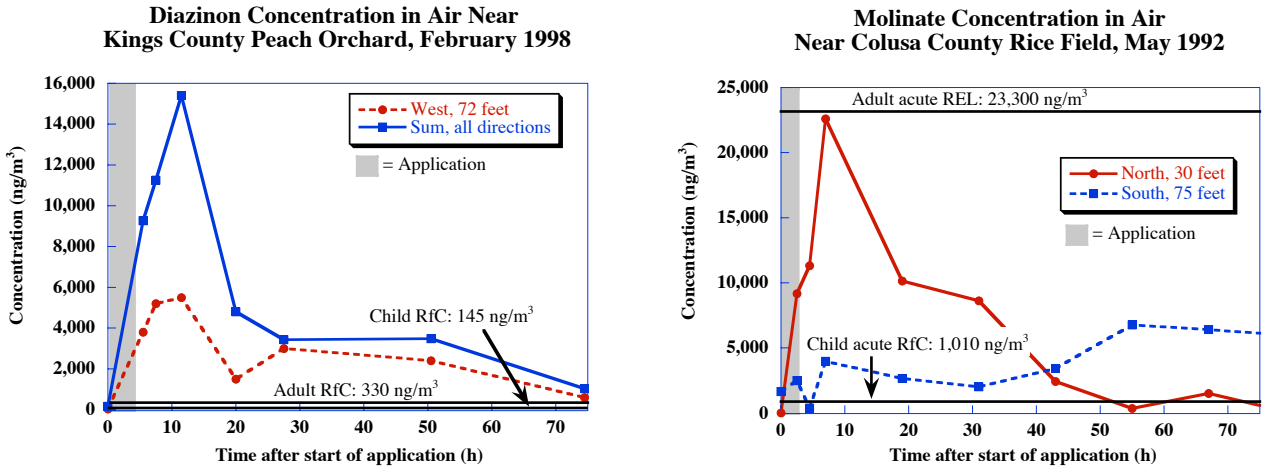

Figure 1: Pesticide concentrations in air for the semi-volatile pesticides diazinon and molinate exceed the acute Reference Concentration for a one-year-old child (molinate and diazinon) and even for adults (diazinon) for at least three days after the application takes place.

U.S. EPA's modeling of fate and transport of pesticides into the environment omits important transport pathways

The potential for a pesticide to move from the application site into endangered species habitat depends on a number of factors including: the physical properties of the pesticide being applied (longevity, water solubility, ability to bind to soil particles, and volatility), atmospheric conditions, and the application technique. Pesticides with substantial water solubility and/or ability to adsorb to sediments may move off-site with stormwater or irrigation return flows. Adsorbed pesticides may also move off-site through aerial transport of wind-blown particles. Pesticides with high to moderate volatility can move off-site via volatilization drift through evaporation and deposition with rainfall or other precipitation, or through condensation caused by cooling nighttime temperatures or increasing elevation. A number of studies have demonstrated long-distance transport for pesticides.[6]

EPA's models are likely to err in predicting the potential range of contamination that may occur from pesticide applications. For example, the GENEEC model, which EPA uses to measure water contamination potential, is described by EPA as " . . a single event model. It assumes one single large rainfall/runoff event occurs that removes a large quantity of pesticide from the field to the water all at one time."[7] The model thus does not account for repeated stormwater or irrigation applications that may result in repeated surface water runoff of pesticides.

This model also underestimates the potential for aerial transport in a number of ways. First, actual studies on the fate of pesticides after an application typically demonstrate a loss of 40% of the mass of the pesticide to spray drift,[8] which may rise as high as 80% for particular applications.[9] The GENEEC model assumes a maximum surface contamination potential for spray drift estimated at 0% of the application rate for broadcast applications, 1% for ground applications and 5% for aerial applications. While it may be *possible* for transport to be this low, prior studies indicate that it is extremely *unlikely*, given the real-world mix of spray equipment in use, applicator expertise, and applicator willingness to ignore adverse weather conditions, label instructions, and/or best operating procedures, as the data from the first set of referenced studies show.[8]

Second, the model fails to account for transport due to post-application volatilization drift (see above). Pesticides that have the potential to be aerially transported away from the point of application also have the potential to contaminate endangered habitat if they are applied in areas upwind of such habitat. A number of studies demonstrate that pesticides are transported from low to high elevations in the Sierra Nevada by prevailing westerly winds from the Central Valley in California.[10]

One study estimated an annual loading of 24-31 kg of chlorpyrifos per year to Sequoia National Park from volatilization drift from the Central Valley in California.[6b] In central Washington, 2,4-D applied to wheat fields was found to have drifted 10 to 50 miles and damaged vineyards downwind.[11] Other studies indicate that up to 95% of total drift for a single pesticide may arise from post-application volatilization and that such drift may occur for several days to several weeks after application.[2. 6a, 12] In Figure 2 below are set forth four graphs, which are plots of the aforementioned ARB data from their field monitoring of two representative pesticides. These plots demonstrate that pesticide volatilization can result in the transport of large quantities of pesticide off of the application site for many days after the application. Approximately 83% of the total measured diazinon drift and 95% of the total measured 1,3-dichloropropene drift is due to volatilization that occurs after the application period.

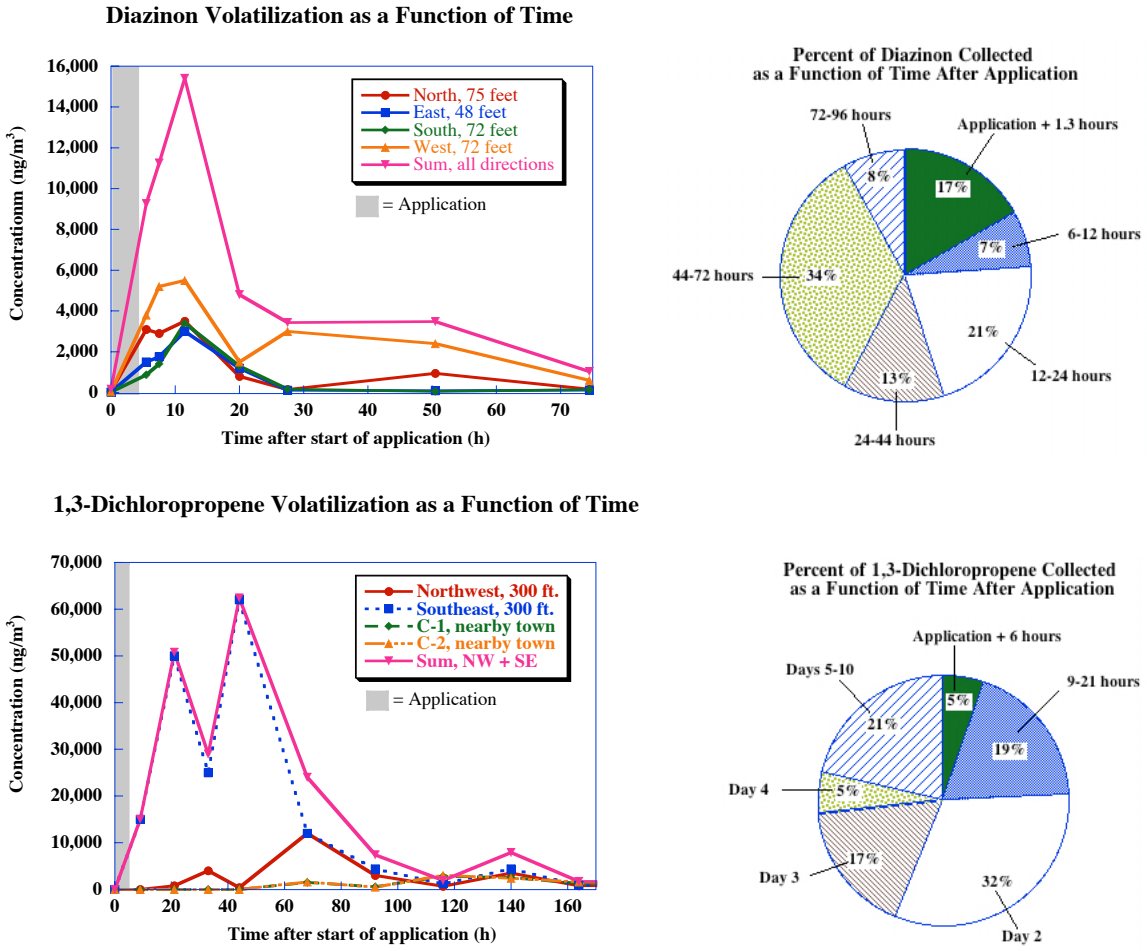

Figure 2: The graphs on the left show the concentration of pesticide in air as a function of time after the application and demonstrate that measurable volatilization drift continues for four days after application for diazinon and for seven days afterwards for 1,3-dichloropropene. The graphs on the right show the percentage of total measured drift for different time periods after the application and indicate that

The potential for pesticides to contaminate endangered species habitat through off-site transport is corroborated by the numerous government and academic studies that have detected pesticides in air and surface water. For example, a recent U.S. Geological Survey (USGS) study detected the presence of 49 pesticides (out of 83 tested for, and out of approximately 900 registered pesticides in California) in surface water rivers, ponds and streams in the Central Valley. Most samples tested contained more than one and usually more than 7 pesticides.[13] These results are consistent with other studies in which pesticides were detected in surface water, often above chronic levels currently considered safe for aquatic organisms.[14] Even these "safe" levels were not set with consideration of possible endocrine disrupting effects, which typically occur at much lower concentrations (see below). Other studies show pesticide residues in frogs and tadpoles in the Sierra Nevada at tissue concentrations high enough to result in reduced levels of the cholinesterase enzyme ChE.[6, 15] A recent USGS study shows that 60% of diazinon loading to a particular watershed is due to diazinon deposition in rainfall, i.e. more diazinon is being deposited into water bodies from rainfall than from runoff from fields.[16] The Central Valley Regional Water Quality Control Board found concentrations of diazinon in rainwater that were lethal to *Daphnia*, an aquatic invertebrate organism that is a key member of the food web for aquatic ecosystems.[17]

Finally, EPA's models do not adequately represent the potential fate of pesticide contaminants once they have entered a given ecosystem, such as whether adsorbed pesticides will settle on surface water films of dust or particulate organic matter, remain in the water column or be deposited in sediments or other areas where endangered species may take refuge.

These data indicate that EPA's first tier model (GENEEC), which determines whether EPA will even deem it necessary to conduct further risk assessment, underestimates the amount of pesticide contamination that may occur by failing to take into consideration a number of factors affecting the fate and transport of pesticide chemicals in the environment. According to the EPA, GENEEC is the most protective model, to be used in the first tier of analysis, with the other models providing further refinement. If EPA's most protective model underestimates potential pesticide concentrations in the environment, this will necessarily lead to systematic underprotection of endangered species.

U.S. EPA does not assess the cumulative effects of pesticides and "other" ingredients

EPA's modeling of the fate of a single pesticide for ecotoxicity also fails to account for the fact that wildlife may be exposed to pesticide transformation products or "other" ingredients in pesticide products that are also toxic, as well as to the cumulative presence of a variety of different pesticide contaminants in a particular environmental medium.

A number of pesticide active ingredients have common mechanisms of toxicity, based on their physiological or biochemical mode of action. Exposures to multiple pesticides with a common mechanism of toxicity results in cumulative toxic effects, a fact that EPA is now required to take into account in the reregistration process when setting tolerances for pesticide residues on foods (21 U.S.C. 346a(b)(2)). EPA has formally found a common mechanism of toxicity for the organophosphorus insecticides (which include such widely used pesticides as chlorpyrifos, diazinon and malathion), and is presently evaluating the the N-methyl carbamate insecticides, the triazine herbicides and the chloroacetanilide herbicides for such cumulative effects. [18] Synergistic or additive effects of pesticides in different classes are not presently evaluated by EPA, but are likely to contribute to additional toxicity above and beyond toxicity attributed to each active ingredient assessed separately.[19]

Pesticide formulation also plays a role in toxicity. Pesticide products are composed of the "active ingredient," which is the chemical designed to kill living organisms, and "other" ingredients, which may themselves be toxic, and are included within the formulated pesticide product to facilitate application and effectiveness. In some cases, the toxicity of pesticide products may be greater than the toxicity of the active ingredient due to presence of "other" ingredients that have been added to the product formulation. U.S. EPA fails to assess the effects of these "other" ingredients and does not even require submission of data on the identity and toxicity of these substances from manufacturers.

The potential for adverse effects from pesticide exposure is substantially increased due to the overall impacts of cumulative exposure. EPA's failure to account for these effects, or even request data that would help them evaluate risks, leads to under-protection of endangered species.

Parts of U.S. EPA's model remain unvalidated and unavailable for the public to review

All of the comments above point to the fact that U.S. EPA's models are too simplistic to be representative of actual exposures, fate and transport. In order for these models to be accurate for predicting exposure and effects on endangered species, U.S. EPA must first be more inclusive of exposure and transport pathways, and then validate the models against actual field data. At this time,

EPA has conducted little sampling to determine the extent of pesticide contamination in these environmental media to validate its models. In fact, EPA seems to be allergic to monitoring data and is reluctant to even consider such data in the risk assessment process.

In addition to lack of validation, EPA's methods for exposure assessment are not fully transparent. For spray drift analysis, EPA uses the AgDrift model, an industry-generated proprietary model that is not accessible to the public for review and comment. This fact casts additional doubt on the validity of EPA's exposure assessment methods.

U.S. EPA does not adequately assess the effects of pesticide toxicity on endangered species

Pesticides may be sub-lethally toxic at relatively low, environmentally relevant levels, affecting endangered species either directly or indirectly by altering its habitat. While the extent of such impacts may not yet be completely characterized, there is no basis for EPA to conclude that such exposures have "no effect" on endangered species.

Pesticides may have short-term "acute" impacts of relative immediacy or "chronic" long term impacts that occur over time. These impacts may be lethal or sub-lethal. Acute impacts for a particular pesticide are assessed by determining the lethal concentration (LC_{50}) or lethal dose (LD_{50}) for 50% of the test organisms. However, the LC_{50} or LD_{50} tests U.S. EPA uses for estimation of effects on endangered species do not provide a complete understanding of whether or not pesticides may be having direct impacts for several reasons.

Test data limited to a few species that may or may not have similar susceptibility to pesticides

The impacts of a pesticide on wildlife may vary substantially depending on the organism. Required tests are conducted on a limited number of species that may or may not reflect actual toxicity to an endangered species. In some cases, U.S. EPA even uses toxicity data from different taxa groups—for example, toxic effects on freshwater fish are used to assess impacts on amphibians.[20] Many studies, however, indicate that pesticides may have different and in some cases more severe impacts on amphibians than fish, up to three orders of magnitude more sensitive. The following examples from the U.S. EPA AQUIRE ecotoxicity database demonstrate this potential.[21]

Pesticide	Organism	LC_{50} (µg/L)
Diuron	Red-legged frog (*Rana aurora*)	22,200
Diuron	Carp, (*Carassius*)	63,000
Diazinon	Climbing perch (*Anabas scandens*)	37,750
Diazinon	Green frog (*Rana clamitans*)	21
Diazinon	Bog frog (*Rana limnocharis*)	7,977
Carbaryl	Walking catfish (*Clarias batrachus)*	71,350
Carbaryl	Western mosquitofish (*Gambusia affinis*)	20,377
Carbaryl	Toad (*Bufo bufo japonicus*)	7,200
Carbaryl	Gray tree frog (*Hyla versicolor*)	2,470
Carbaryl	Green frog (*Rana clamitans*)	20,372
Endosulfan	Snake-head catfish (*Channa punctata*)	4,586
Endosulfan	Zebra danio (*Danio rerio*)	750
Endosulfan	Toad (*Bufo vulgaris formosus*)	2,075
Endosulfan	Bog frog (*Rana limnocharis*)	12
Endosulfan	Tiger frog (*Rana tigrina*)	2

Further, studies also demonstrate that toxic impacts may vary even between different genera and species of organisms (see Table above for examples).

EPA has still not developed a series of standardized tests that are routinely applied to current use pesticides to determine potential impacts on some species (e.g., amphibians). EPA states its approach on this issue as follows:

> "[T]oxicity testing does not test all species of birds or fish. Only two surrogate species for both freshwater fish and birds are used to represent all freshwater fish species (2000+) and bird species (680+) in the United States. For mammals, acute studies are usually limited to the Norway rat or the house mouse. Estuarine/marine testing is limited to a crustacean, mollusk, and fish. Also, reptiles and amphibians are not tested. The assessment makes the assumption that the bird and reptilian toxicities are the same. The same assumption applies to amphibians and fish. Therefore, without definitive testing, it is assumed that the conclusions regarding hazards of iprodione to birds and fish apply to other vertebrate animals that are not currently tested."[22]

While this assumption may simplify the risk assessment process, the fact that sensitivities can vary by over three orders of magnitude within a given taxa group means that this approach gives EPA no scientific basis for making a "no effect" determination.

U.S. FWS agrees, indicating their concerns with the EPA approach:

> "In certain instances, OPP will have substantial information tending to demonstrate that the test species appropriately represents the sensitivity of another species. However, in other instances, frequently concerning the use of a surrogate species outside the class of the listed species, information demonstrating that the tested species appropriately represents the listed species is limited or inferential. The Services note that for some uses of surrogate species, the trend in data may indicate that the tested species is likely to be at least as sensitive as the untested class of species, but the analytical confidence in any conclusions drawn from these studies currently is limited. First, the confidence may be limited due simply to the small number of the studies. Second, the confidence may be limited due to the potential, and unknown, range of toxicological sensitivity among different species within the untested class, especially given the potential variability regarding most sensitive life stages or age classes among different species. Consequently, while this surrogate information is the best available toxicological data, OPP's analysis will discuss species extrapolation uncertainties to ensure that scientific judgments using this data are made in a transparent manner. The Services and EPA will work cooperatively to develop methods in the future to increase confidence in the use of surrogate species test data, such as determining whether new safety factors may be identified, or exploring opportunities for testing additional species."[23]

We acknowledge that it is neither desirable nor feasible to test every single species; however, in the absence of data, it is essential to apply an uncertainty factor large enough to accommodate the possible spread in sensitivity of different organisms. In addition, U.S. EPA needs to give more than lip service to identification of non-toxic alternatives.

Sublethal effects are not considered

Sub-lethal impacts are any adverse impact to a species that is not directly lethal, though the sub-lethal effects, such as reduced feeding or inability to evade predators, may lead to death or inability to reproduce. These sub-lethal impacts are likely to pose substantial risk to endangered species since they can occur at pesticide concentrations typically found in the environment, and have the potential to have significant impacts on species populations. The required LC_{50} tests do not address the potentially significant sub-lethal impacts that pesticides may have on endangered species from either a single

acute or long term chronic exposure and cannot reliably be used as the sole basis for making a determination that a pesticide contamination will not have adverse effects on endangered species. Chronic exposures are of particular concern because in most cases, the threshold for adverse effects due to chronic exposure is much lower than the threshold for acute impacts, yet these low-dose regimens are not a required part of toxicity testing.

EPA's cutoff point for determining whether further risk assessment is necessary in the aquatic environment (Risk Quotients greater than 0.05 for aquatic animals or 0.1 for mammals and birds) is not based on any actual assessment of the potential sub-lethal impacts that may occur and is thus likely to fail to identify potential effects of pesticide contamination.[24] For example, at this time, EPA has not developed appropriate testing methods to measure the impacts of pesticide residues on chronic endpoints that reflect toxicity to the reproductive systems, immune systems, and genetic integrity of an organism. Without such testing, EPA is not in a position to find that a particular pesticide will have "no effect" based on the fact that contamination levels do not approach the levels needed to kill 50% of a population in a limited time period. In fact, a pesticide concentration level that is 1/20 of a given LC_{50} for a particular species may be substantially *above* the levels necessary to cause significant, adverse sub-lethal impacts.

For example, studies have found a 96 hour LC_{50} for atrazine of 47,500 ppb (47,500 parts per billion).[25] Under EPA's approach, EPA would make a "no effect" determination where EECs were below 2,375 parts per billion or 2,375 ppb, based on the formula of 47,500/20 = 2,375. However, recent studies indicate that atrazine will have adverse impacts on the sexual development of male leopard frogs at levels as low as 0.1 ppb or 0.1 parts per billion.[26] This potential impact would not have been accounted for under EPA's system based on LC_{50} testing, resulting in an underestimate of adverse ecological effects.

Endocrine disruption is not considered

Over the last two decades, researchers have discovered and regulatory agencies have acknowledged that certain chemicals, including pesticides, may disrupt the normal functioning of the endocrine system, which controls the levels and flows of hormonal compounds within a living organism.[27] The endocrine system regulates a number of essential physiological functions, including development of the immature organism, reproduction, and immune system strength and response. As mentioned above, the potential impacts of endocrine disrupting pesticides on amphibians has been demonstrated by recent studies conducted at UC Berkeley, which found that extremely low levels (0.1 parts per billion) of the triazine herbicide atrazine had adverse impacts on the sexual differentiation of the African clawed frog.[25a] Subsequent surveys of atrazine-contaminated ponds in the Midwest found many native leopard frogs (*Rana pipiens*) with similar reproductive abnormalities.[25b]

Other studies have focused on the potential for pesticides to disrupt the immune system, also controlled by the endocrine system.[28] For example, sub-lethal doses of malathion, a pesticide detected in the Sierra, increased the likelihood of a fatal infection in adult Woodhouse toads (*Bufo woodhousi*) from the bacterium *Aeromonas hydrophila.*[27b] Two recent 2002 studies indicate that pesticides lower amphibian immunity, thus rendering them more susceptible to disease[29] or to trematode parasites thought to play a role in amphibian deformities.[30]

Possible mechanisms of the immuno-suppression related to pesticide exposure have recently been proposed.[27] These studies demonstrate that endocrine disrupting chemicals may elevate the levels of

certain hormones that suppress the ability of amphibians to mount an immune response to a pathogen, or may interfere with the production of natural amino acids that occur in amphibian skin and provide a first line of defense against fungi and other diseases.[31] The evidence that pesticide exposures may cause or contribute to immune suppression in amphibians, together with the studies showing potential mechanisms by which immuno-suppression may occur, is a particular cause of concern given the recent evidence that disease epidemics, often caused by formerly unknown or formerly non-lethal pathogens, are an immediate and observed contributing cause of amphibian declines in the Sierra Nevada, in the western United States and worldwide.[27, 30, 32] Specifically, in California, disease has played a role in the declines of two higher elevation Sierra species—the mountain yellow-legged frog (*Rana muscosa*) and Yosemite toad (*Bufo canorus*).[33]

EPA has recognized the threat posed by endocrine disrupting chemicals, including pesticides. In 1998, EPA issued a statement that "[t]aken collectively, the body of scientific research on human epidemiology, laboratory animals, and fish and wildlife provides a plausible scientific hypothesis that environmental contaminants can disrupt the endocrine system leading to adverse-health consequences."[26] This conclusion was based on EPA's 1997 Special Report which stated:

> Scientific evidence has been accumulating that humans, domestic animals, and fish and wildlife species have exhibited adverse health consequences from exposure to environmental chemicals that interact with the endocrine system. To date, such problems have been detected in domestic or wildlife species with relatively high exposure to organochlorine compounds (e.g., 1,1,1- trichloro-2,2-bis(p-chlorophenyl) ethane (DDT) and its metabolite dichorodiphenyldichloroethylene (DDE), polychlorinated biphenyls (PCBs), and dioxins) or to some naturally occurring plant estrogens. But effects from exposure to low levels of endocrine disruptors has been observed as well (e.g., parts per trillion levels of tributyl tin have caused masculinization of female marine molluscs such as the dog whelk and ivory shell). Adverse effects have been reported for humans exposed to relatively high concentrations of certain contaminants. However, whether such effects are occurring in the human population atlarge at concentrations present in the ambient environment, drinking water, and food remains unclear. Several conflicting reports have been published concerning declines in the quality and quantity of sperm production in humans over the last 4 decades, and there are reported increases in certain cancers (e.g., breast, prostate, testicular). Such effects may have an endocrine-related basis, which has led to speculation about the possibility that these endocrine effects may have environmental causes. However, considerable scientific uncertainty remains regarding the actual causes of such effects. Nevertheless, there is little doubt that small disturbances in endocrine function, particularly during certain highly sensitive stages of the life cycle (e.g., development, pregnancy, lactation) can lead to profound and lasting effects.[34]

EPA's risk assessment documents acknowledge the potential for pesticides to have endocrine disrupting effects. For example, EPA's risk assessment for methyl parathion, an organophosphorus insecticide used in California, states:

> Methyl parathion has been observed in the open literature to display metabolic effects which hinder successful reproduction and/or successful development in birds, mammals and fish....Based on these observed effects, EPA's risk assessment recommends that methyl parathion be subjected to more definitive testing to better characterize endocrine disrupting effects."[35]

At this time, the state of science related to endocrine disrupting chemicals is incomplete. For example, EPA's risk assessment for the herbicide diuron states:

> EPA is in the process of developing criteria for characterizing and testing endocrine disrupting chemicals and plans to implement an Endocrine Disruptor Screening Program. Diuron will be reevaluated at that time and

> additional studies may be required.[36]

The research that has been conducted continues to reveal new pesticide chemicals that have endocrine disrupting properties. For example, EPA's risk assessment for endosulfan, an organochlorine insecticide used in California and detected in endangered species habitat,[6] states the following:

> Exposure to endosulfan has resulted in both reproductive and developmental effects in nontarget animals. Endosulfan exposure resulted in impaired development in amphibians, reduced cortisol secretion in fish, impaired development of the genital tract in birds and reduced hormone levels and sperm production and produced testicular atrophy in mammals. Additionally, endosulfan has been demonstrated to bind to the human estrogen receptor and exhibit significant estrogenic activity. Whether the toxicity endpoints are a result of endocrine disruption is not known. However, it is clear that organisms treated with endosulfan did exhibit some toxic effects that have historically been associated with endocrine disrupting chemicals,e.g., developmental and reproductive effects.[37]

U.S. EPA's lack of knowledge regarding the mechanisms or extent of endocrine-disrupting abilities of pesticide chemicals suggests that they should be exceedingly cautious in concluding that an exposure to a pesticide will not likely have direct adverse impacts on endangered species. Although progress has been made in evaluating possible screening methods, EPA has not yet formalized protocols for testing the endocrine disrupting properties of chemicals including pesticides. EPA has conducted no testing for endocrine effects of pesticides on endangered species, and such testing is not required for a pesticide to be registered under FIFRA.

Until EPA better understands the subtle hormonal impacts of pesticide chemicals on sensitive wildlife, it is contrary to sound science for EPA to determine that a particular pesticide will have "no effect," to the extent there is a potential for contamination of the habitat.

Indirect impacts to endangered species are not considered

In addition to potential direct impacts, pesticides may have indirect impacts on endangered species by affecting their habitat. Pesticide contamination, particularly herbicide contamination of habitats may eliminate or greatly reduce plant communities, thereby affecting endangered species through loss of habitat.

Pesticides may also have indirect impacts by disrupting the ecological balance of the ecosytem. For example, EPA's risk assessment documents point out that the loss of freshwater invertebrates through pesticide contamination can lead to algae blooms, which may cause fish kills by depleting dissolved oxygen in ponds when excess algae dies and decays. Similarly, herbicide contamination which kills aquatic plants such as algae will lead to the decay of those plants within the aquatic ecosystem, which also results in loss of oxygen. In sum, pesticide contamination of the aquatic ecosystem has the potential to affect endangered species by altering the habitat that they need to survive and/or reducing or eliminating their food supply.

U.S. EPA does not have the in-house biological expertise to accurately make these "may affect" determinations

The proposed regulations would take the Services largely out of the loop in the determination of whether or not a pesticide "may affect" an endangered species. Yet U.S. EPA lacks the biological expertise to conduct this assessment because it does not have in-house experts with specialized knowledge of endangered species habitat needs, life cycles, feeding patterns, breeding requirements

and other information that is essential for making a "may affect" determination. The Services does have this knowledge and expertise. The interagency consultation process should not be dismantled.

No data does not equal no effect

A disturbingly unscientific theme runs throughout U.S. EPA's methods for conducting ecological risk assessments: If no data are readily available (which can mean that U.S. EPA simply isn't aware of existing data), the potential adverse effect is assumed to be negligible. This philosophy manifests itself in several ways:

- If there are no data on a particular exposure route, the exposure via that route is determined to be zero.
- If there are no data on a particular toxic endpoint, that particular type of toxicity is assumed to be nonexistent.
- If there are no data on the toxicity of "other" ingredients, these compounds are assumed to be completely non-toxic.
- If there are no data on cumulative effects of pesticides with common mechanisms of action, it is assumed that there are no cumulative effects.

The fact that EPA lacks full information on these questions, however, cannot form the basis for a conclusion that a pesticide is having “no effect” on endangered species. Indeed, in light of the compelling suite of evidence demonstrating transport of pesticides into habitat, evidence of toxicity of some pesticides at very low levels, and in some cases statistically significant declines in endangered species populations downwind of pesticide applications,[38] a reasonable scientific hypothesis is that pesticides with the potential to contaminate endangered species habitat may affect these species.

Conclusion

EPA’s policy of making “no effect” determinations based on single-media, single-pesticide EECs according to LC_{50} data derived from testing on surrogate species and without full consideration of all exposure routes is contrary to sound science and will, in all likelihood, jeopardize the survival of endangered species. Attempted legitimization of this approach by changing the regulations will not change the fact that U.S. EPA's "sound science" in support of such activities is neither sound nor is it science. We urge you to reject these proposed regulations.

Sincerely yours,

Susan Kegley, Ph.D.
Senior Scientist
Pesticide Action Network
49 Powell St., Suite 500
San Francisco, CA 94102

Cynthia Palmer-Olsen
Senior Program Officer
World Wildlife Fund
1250 24th St. NW
Washington, DC 20037

Shelley Davis
Farmworker Justice Fund
1010 Vermont Ave NW Suite 915
Washington, DC 20005

Wendell Wood
Southern Oregon Field Representative
Oregon Natural Resources Council
HC 63, Box332
Chiloquin, OR 97624

Mark Riskedahl, Executive Director
Northwest Environmental Defense Center
10015 SW Terwilliger Blvd.
Portland, OR 97219

Tonya Graham, Executive Director
Headwaters
P O Box 729
84 Fourth St.
Ashland, OR 97520

Steve Sheffield, Ph.D., Affiliate Professor
Dept. of Environmental Science and Policy
George Mason University
4400 University Dr.
Fairfax, VA 22030

Joan Poss, Organizer
Fresno Coalition Against the Misuse of Pesticides
4061 N Wilson
Fresno, CA 93704

Vivian Parker
Resource Policy Analyst/Biologist
California Indian Basketweavers Association
6221 Shoo Fly Rd.
Kelsey, CA 95667

Ann Vileisis
Kalmiopsis Audubon Society
Box 1286
Port Orford, OR 97465

Leo O'Brien, Executive Director
WaterKeepers Northern California
Presidio Building 1004
San Francisco, CA 94129-0921

Anne Katten
Worker Health and Safety Specialist
California Rural Legal Assistance Foundation
2210 K Street, 2nd Floor
Sacramento, CA 95816

Nina Bell, J.D., Executive Director
Northwest Environmental Advocates
P.O. Box 12187
Portland, OR 97212-0187

Tim McKay, Executive Director
Northcoast Environmental Center
575 H Street
Arcata, CA 95521

Greg King, Executive Director
Smith River Project
P.O. Box 157
Orleans, CA 95556

David Dilworth, Executive Director
Helping Our Peninsula's Environment
Box 1495
Carmel, CA 93921

Karen Schambach, President
Center for Sierra Nevada Conservation
P.O. Box 603
Georgetown, CA 95634

Joe Gillespie, President
Eileen Cooper, Board Member
Friends of Del Norte
PO Box 229
Gasquet, CA 95543

Eric Cárdenas
Central Coast Environmental Health Project
906 Garden St.
Santa Barbara, CA 93101

Ron Renchler, Vice President
Lane County Audubon Society
Eugene, Oregon

Joan Clayburgh, Executive Director
Sierra Nevada Alliance
P.O. Box 7989
South Lake Tahoe, CA 96158

Bill Jennings, Director
DeltaKeeper
3536 Ranier Ave.
Stockton, CA 95204

References

[1] a) Cox, C., Pesticide Drift-Indiscriminately from the Skies, *Journal of Pesticide Reform,* Spring, 15(1), (1995).

b) Rice, C., S. Chernyak, "Marine Arctic Fog: An Accumulator of Currently Used Pesticide," *Chemosphere*, 35: 867 (1997).

c) Dexter, A. G., *Herbicide Spray Drift,* A–657, North Dakota State University, 1993, http://www.ext.nodak.edu/extpubs/plantsci/weeds/a657w.htm.

d) DPR, Department of Pesticide Regulation, T. Barry and J. Walters, *Characterization of Propanil Prune Foliage Residues as related to Propanil Use Patterns in the Sacramento Valley, California* (2002).

[2] ARB, California Air Resources Board, *Toxic Air Contaminant (TAC) Monitoring Studies,* as presented on California Department of Pesticide Regulation TAC web site, 2003. http://www.cdpr.ca.gov/docs/empm/pubs/tac/tacstdys.htm.

[3] a) Majewski, M.S., Foreman, W.T., Goolsby, D.A., *et al*., Airborne pesticide residues along the Mississippi River, *Env Sci Tech,* 32: 3689–98 (1998).

b) ARB, California Air Resources Board, *Toxic Air Contaminant (TAC) Monitoring Studies,* as presented on California Department of Pesticide Regulation TAC web site, 2003. http://www.cdpr.ca.gov/docs/empm/pubs/tac/tacstdys.htm.

[4] Michael S. Majewski and David S. Baston, *Atmospheric Transport of Pesticides in the Sacramento California Metropolitan Area, 1996-1997,* USGS Water-Resources Investigations Report 02-41000 (2002)

[5] a) J. Zabik, J. Seiber, "Atmospheric Transport of Organophosphate Pesticides from California's Central Valley to the Sierra Nevada Mountains," *J. Envi. Qual.,* 22: 80–90 (1993).

b) Baker L, *et al*., "Ambient Air Concentrations of Pesticides in California," *Envi. Sci. Tech.,* 30: 1356 (1996).

[6] a) M. S. Majewski and D. S. Baston, *Atmospheric Transport of Pesticides in the Sacramento California Metropolitan Area, 1996-1997,* USGS Water-Resources Investigations Report 02-41000 (2002).

b) J. LeNoir, L. McConnell, G. Fellers, T. Cahill, and J. Seiber, "Summertime transport of current-use pesticides from California's Central Valley to the Sierra Nevada Mountain Range, USA." *Environmental Toxicology and Chemistr*y, 18(12): 2715-2722 (1999).

c) Sparling, Donald W., Gary M. Fellers and Laura L. McConnell, "Pesticides and Amphibian Population Declines in California, USA," *Environmental Toxicology and Chemistr*y, 20(7): 1591-1595 (2001).

[7] U.S. EPA, *GENEEC Description,* http://www.epa.gov/oppefed1/models/water/geneec2_description.htm (2001).

[8] a) U.S. Congress, Office of Technology Assessment, 1990, *Beneath the bottom line: Agricultural approaches to reduce agrichemical contamination of groundwater,* Report No. OTA–4–418, Washington, D.C., U.S. GPO.

b) National Research Council, Board on Agriculture, Committee on Long-Range Soil and Water Conservation, *Soil and water quality: An agenda for agriculture,* Natl Academy Press (Washington, DC, 1993), 323–324.

[9] Bennet, J., "Pesticide drift and Runoff: Considerations for the U.S. Fish and Wildlife Service, Draft Biological Opinion on Effects of 31 Pesticides on Threatened and Endangered Species," Washington Cooperative Fish and Wildlife Resource Unit, University of Washington, March 1, 1992.

[10] a) Reference 6b, LeNoir, *et al*. (1999).

b) Reference 6a, Sparling, *et al*. (2001).

c) Reference 5a, Zabik *et al*. (1993).

d) Datta, S., Hansen, L., McConnell, *et al.,* "Pesticides and PCB Contaminants in Fish and Tadpoles from the Kaweah River Basin, California," *Bull. Envi. Contam. Toxicol.*, 60: 829–836 (1998).

e) McConnell, L, J. LeNoir, S. Datta and J. Seiber, "Wet Deposition of Current-use Pesticides in the Sierra Nevada Mountain Range, California, USA," *Envi. Toxicol. Chem.,* 17(10): 1908–1916 (1998).

f) Aston, L., Seiber, J., "Fate of Summertime Airborne Organophosphate Pesticide Residues in the Sierra Nevada Mountains," *J. Envi. Qual.*, 26: 1483–1492 (1997).

[11] Robinson, E., L.L. Fox, *Air Pollution Control Association Journal,* 28:1015-1020, (1978).

[12] Kegley, S.E., Katten, A., Moses, M. *Secondhand Pesticides: Airborne Pesticide Drift in California,* Californians for Pesticide Reform (San Francisco, 2003).

[13] Sandra Panshin, Neil Dubrovsky, JoAnn Gronberg, and Joseph Domagalski, *Occurrence and Distribution of Dissolved Pesticides in the San Joaquin River Basin, California,* USGS Water-Resources Investigations Report 98-4032, 1998.

[14] a) Panshin, S.Y, Domagalski, J.L., Dubrovsky, N.M., "Pesticide Concentrations in Surface Water as a Function of Agricultural Land Use in Five Small Watersheds, Western San Joaquin Valley, California," *Eos: Trans. of the Am. Geophys. Union,* 75 (Suppl.): 246 (1994).

b) Kratzer, C.R., "Pesticides in storm runoff from agricultural and urban areas in the Tuolomne River Basin in the vicinity of Modesto, California," U.S. Geological Survey, Water Resources Investigations Reports 98–4017 (1998).

c) DPR, Department of Pesticide Regulation, *Preliminary Results of Acute and Chronic Toxicity Testing of Surface Water Monitored in the San Joaquin River Watershed, Winter 1998–1999*, July 20, 1999.

d) MacCoy, D., Crepeau, K.L., Kuivila, K.M., *Dissolved Pesticide Data for the San Joaquin River at Vernalis and the Sacramento River at Sacramento, California, 1991–1994,* USGS Open-File Report 95–110 (1995).

e) U.S. Forest Service Reports *Re* Water Quality Monitoring, Herbicide Application, dated January 1995, July 1996 and October 1996.

[15] Deborah Cowman and Laura Maznti, "Ecotoxicology of 'New Generation' Pesticides to Amphibians," Chapter 6A in *Ecotoxicology of Amphibians and Reptiles*, Sparling, Donald W., *et. al* Editors, 2000 (SETAC).

[16] USGS, Zamora, C., Kratzer, C.R., Majewski, M.S., Knifong, D.L. *Diazinon and Chlorpyrifos Loads in Precipitation and Urban and Agricultural Storm Runoff during January and February 2001 in the San Joaquin River Basin, California,* U.S. Geological Survey Water–Resources Investigations Report 03–4091 (2003).

[17] Connor, V., Central Valley Regional Water Quality Control Board, as cited in J. P. Fox and E. Archibald, *Aquatic Toxicity and Pesticides in Surface Waters of the Central Valley,* California Urban Water Agencies, p. 112 (1998).

[18] U.S. EPA, *Organophosphate Pesticides: Revised OP Cumulative Risk Assessment,* http:www.epa.gov/pesticides/cumulative/rra-op/index.html, June 2002.

[19] Porter, Warren, Jaeger, James, Carlsan, Ian, "Endocrine, immune and behavioral effects of aldicarb (carbamate), atrazine (triazine) and nitrate (fertilizer) mixtures at groundwater concentrations," *Toxicology and Industrial Health,* 15: 133-150, (1999).

[20] U.S. EPA, *OPPTS Harmonized Test Guidelines, Series 850 Ecological Effects Test Guidelines*, www.epa.gov/opptsfrs/OPPTS_Harmonized/850_Ecological_Effects_Test_Guidelines/Drafts/ (2003).

[21] U.S. EPA 2000; Pesticide Ecotoxicity Database (Formerly: Environmental Effects Database (EEDB)), Office of Pesticide Programs, Environmental Fate and Effects Division, U.S.EPA, Washington, D.C., 2000. AQUIRE Reference No: 344.

[22] U.S. EPA, *Reregistration Eligibility Decision for Iprodione,* EPA 738-R-98-019, November 1998, http://cfpub.epa.gov/oppref/rereg/status.cfm?show=rereg#I.

[23] U.S. FWS and NMFS comment to Susan B. Hazen, *Evalaution of U.S. EPA's Approach*, January 6, 2004, http://www.nmfs.noaa.gov/pr/laws/pesticides.htm.

[24] U.S. *EPA, Overview of the Ecological Risk Assessment Process in the Office of Pesticide Programs, Endangered and Threatened Species Effects Determinations,* January 23, 2004.

[25] Howe, G.E., R. Gillis, and R.C. Mowbray, "Effect of Chemical Synergy and Larval Stage on the Toxicity of Atrazine and Alachlor to Amphibian Larvae, *Envi.Toxicol.Chem.* 17(3): 519–525 (1998).

[26] a) Hayes, T., Collins, A., Lee, M. Mendoza, M., Noriega, N., Stuart, A., Vonk, A., "Hermaphroditic, Demasculinized Frogs Following Exposure to the Herbicide Atrazine at Ecologically Relevant Low Doses," *Proc. Natl. Acad. Sci. USA,* 99(8): 5476-5480 (2002).
b) Hayes, T., K. Haston, M. Tsui, A. Hoang, C. Haeffele & A. Vonk, "Atrazine Induced Hermaphroditism at 0.1 ppb in American Leopard Frogs (*Rana pipiens*): Laboratory and Field Evidence," *Envi. Health Persp.* 111: 568–575 (2003).

[27] a) U.S. EPA, Endocrine Disruptor Screening Program: Proposed Statement of Policy, *Federal Register,* Vol. 63, No. 248, December 28, 1998.

b) Colborn, T., F.S. Vom Saal and A.M. Soto, "Developmental effects of endocrine-disrupting chemicals in wildlife and humans," *Envi. Health Persp.,* 101: 378–384 (1993).

c) Colborn, T., Dumanoski, D. Myers, J.P., *Our Stolen Future,* Penguin Books (New York, 1996).

d) Danish Environmental Protection Agency, *Auxiliary Matters with Estrogenic Effects,* October 1999, http://www.mst.dk/chemi/Pesticider/02030300.doc.

e) Our Stolen Future, *Widespread pollutants with reproductive and endocrine-disrupting effects,* web site, http://www.ourstolenfuture.org/Basics/chemlist.htm.

f) European Commission, DG ENV, *Towards the establishment of a priority list of substances for further evaluation of their role in endocrine disruption,* BKH report M0355008/1786Q, Delft, June 21, 2000, http://europa.eu.int/comm/environment/docum/01262_en.htm#bkh.

g) Illinois EPA, *Report on Endocrine Disrupting Chemicals,* (February, 1997).

h) Keith, L.H., *Environmental Endocrine Disruptors: A Handbook of Property Data,* Wiley Interscience (New York, 1997).

i) Tulane University, *Environmental Estrogens and Other Hormones: Modes of Action,* http://e.hormone.tulane.edu/learning/modesofaction.html (2003).

[28] a) Carey C., and C. Bryant, "Possible Interactions among Environmental Toxicants, Amphibian Development, and Decline of Amphibian Populations," 103 *Environmental Health Perspectives,*103 (Suppl.4): 13-16 (1995).
b) Taylor, Sharon K.; Williams, Elizabeth S.; Mills, Ken W., "Effects of malathion on disease susceptibility in Woodhouse's toads," *Journal of Wildlife Diseases*, 35(3): 536-541 (1999).

[29] Dixon, B., Gilbertson, M.-K., Haffner, D.G., *et al.* "Immunosuppression in the Northern Leopard Frog (*Rana pipiens)* Induced by Pesticide Exposure," *Envi. Toxicol. Chem.*, 22(1): 100–110 (2002).

[30] Kiesecker, J.M., "Synergism between trematode infection and pesticide exposure: A link to amphibian limb deformities in nature?" *Proc. Nat. Acad. Sci.,* 99(15): 9900–9904 (2002).

[31] Rollins-Smith, L. A., "Neuroendocrine-immune system interactions in amphibians: Implications for understanding global amphibian declines." *Immunol. Res.* 23(2–3): 273–280 (2001).

[32] Crawshaw, G. J., "Diseases and Pathology of Amphibians and Reptiles," Chapter 5 in *Ecotoxicology of Amphibians and Reptiles,* Sparling, Donald W., *et. al* editors, SETAC Press, (Pensacola, FL, 2000).

[33] a) U.S. Fish and Wildlife Service (FWS) "Endangered and Threatened Wildlife and Plants; 12-Month Finding for a Petition to List the Mountain Yellow Legged Frog (*Rana muscosa*)," 68 Fed. Reg. 2283, January 16, 2003.

b) U.S. Fish and Wildlife Service (FWS) "Endangered and Threatened Wildlife and Plants; 12-Month Finding for a Petition to List the Yosemite Toad," *67 Fed. Reg.* 75834, December 10, 2002.

[34] *Federal Register,* Vol. 63, No. 248, 71542, Monday, December 28, 1998.

[35] U.S. EPA, *Reregistration Eligibility Decision for Methyl Parathion,* Case No. 0153, May, 2003, http://cfpub.epa.gov/oppref/rereg/status.cfm?show=rereg#M.

[36] U.S. EPA, *Reregistration Eligibility Decision for Diuron (TRED),* July 30, 2002, http://cfpub.epa.gov/oppref/rereg/status.cfm?show=rereg#D.

[37] U.S. EPA, Reregistration Eligibility Decision for Endosulfan (RED), EPA 738-R-02-013, November, 2002, http://cfpub.epa.gov/oppref/rereg/status.cfm?show=rereg#E.

[38] a) C. Davidson, H. B. Shaffer and M. Jennings, "Spatial tests of the pesticide drift, habitat destruction, UV-B and climate change hypotheses for California amphibian declines. *Conservation Biology* 16:1588-1601 (2002).

b) C. Davidson, Shaffer, H. B. Jennings, M. R., "Declines of the California red-legged frog: Climate, UV-B, habitat, and pesticides hypotheses." *Ecological Applications,* 11(2): 464–479 (2001).