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Re: Vegetation Treatments Using Herbicides on BLM Lands in 17 Western States – Draft PEIS / PER

Dear Mr. Amme,

These comments are submitted by Californians for Alternatives to Toxics (CATs) regarding the Bureau of Land Management's (BLM) Draft Programmatic Environmental Impact Statement (PEIS) regarding vegetation treatments using herbicides in 17 western states. The Environmental Protection Information Center (EPIC) joins CATs in these comments.

CATs is a public interest organization that for 24 years has been concerned about activities undertaken by the BLM and other public lands management agencies that directly involve the use of pesticides, including herbicides, or create conditions that can lead to the use of pesticides. Members of CATs live near, depend for their culture and/or livelihood, or visit for study and recreation areas of BLM lands that would be affected by the proposals set forth in the PEIS. Water used by many of the members of CATs is discharged from these lands. Members observe, recreate, gather or otherwise enjoy the resources of BLM lands in the western states, or simply derive satisfaction from knowing that it is there, alive with wildlife, still beautiful and available to visit when they choose, and free of toxic chemicals. BLM lands are public land that, as such and as a part of the United States, holds immeasurable value for CATs members.

EPIC is a grassroots organization dedicated to the protection and restoration of forests, watersheds, and biodiversity in northern California. EPIC maintains its offices in Humboldt County, California. Most of EPIC's approximately 2,000 members and supporters live in Northern California. EPIC's members use, enjoy, and recreate on public lands, including those managed by the Bureau of Land Management.

The BLM's weed management plan is timely and it is important; we welcome and support well thought out efforts to prevent and control invasive plants and noxious weeds to support and sustain the natural environment. We applaud the BLM's stated commitment to protect native species biodiversity, though we question the environmental effects and efficacy of the current plans for implementing this policy.

We have identified several areas in the Draft Programmatic Environmental Impact Statement (PEIS) that describe abusive and unnecessary uses of herbicides to accomplish desired conditions. In addition, we have

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found that the PEIS fails to provide adequate means, despite the use of herbicides, for achieving the project goals.

The aerial application of herbicides over wide areas is supercilious and irresponsible. The non-specific use of herbicides, with the intention of satisfying several specific goals, consequently reduces the likelihood for success. It does however permit the herbicide to adversely impact a variety of sensitive non-target species of plants and wildlife as well as humans. Widespread and aerial applications of herbicides may also result in air, water, and soil contamination, which could potentially impact project areas and adjacent wildland systems for years. CATs members are appreciative of their wildlands and prefer to enjoy them in their natural state to the extent possible, certainly devoid of unnecessary chemically induced disturbances and contamination.

We have chosen to focus our concerns on several subject areas. Within each subject area, critical elements of excessive, inappropriate, and ineffective herbicide use are addressed. We have provided an outline below, systematically listing some of our larger concerns. This list is by no means exclusive. In the text following, we will specifically address each concern in detail.

#### Part one: Herbicide Issues

- I. Inadequate toxicology analyses
  - a. Active ingredients
  - b. Inerts, adjuvants, degradates and diluents
  - c. Endocrine effects
  - d. Nonylphenol ethoxylates
  - e. Reproductive and developmental toxicity
  - f. Non-endocrine effects
- II. Impacts to Human Health
  - a. Chemically sensitive
  - b. Pregnant women and fetuses
- III. Cumulative Impacts

#### Part two: Invasive species Issues

- I. Herbicides as a disturbance factor
  - a. Invasives thrive after herbicide use
  - b. Increased fire risk
  - c. Cheatgrass invasion
- II. Alternatives
  - a. Reasonable range
  - b. Lack of IPM
  - c. Examples
- III. Fail to treat cause, instead just treating symptoms
  - a. Grazing
  - b. Logging
  - c. Off-road vehicles
  - d. Re-vegetation
- IV. Adequate information disclosure

In its current form the PEIS is inadequate and unacceptable. It is lacking the necessary analyses to evaluate any alternatives including the use of herbicides. We do not accept its flagrant misuse of herbicides in order to achieve quick results. In addition, the proposed actions introduce extraneous problems and inflame existing problems, especially in terms increased fire danger and the spread of noxious weeds and invasive non-native pest plants. The PEIS demands further investigation of alternative methods with greater efficacy for achieving

the desired project goals. Until significant potential adverse impacts missing or inadequately disclosed and analyzed are given the required hard look at alternatives, the heart of an environmental analysis, as required National Environmental Quality Act (NEPA) and supported by the Administrative Procedures Act (APA), cannot be realized. The Endangered Species Act cannot be upheld by the current analysis. The PEIS, as currently drafted, fails to uphold the requirements of federal law and must be subject to revision on a grand scale to pass muster.

## **1. LACK OF ANALYSIS FOR AIs, INERTS , ADJUVANTS AND DEGRADATES**

### **OVERVIEW**

A glaring deficiency found in the PEIS is it's lack of analysis of potential negative effects from all toxic substances proposed for use in this program. The substances in need of analysis include all components of the final tank mixture that is then applied into the environment. All inerts, adjuvants and active ingredients. The analysis should also include any known degradates and contaminants that could cause negative impacts.

The BLM has instead chosen to give cursory analysis to certain active ingredients (AIs), while piggybacking on limited analysis used by the Forest Service for others, and refusing to perform any analysis whatsoever for potential effects from the use of the hundreds of toxic substances BLM introduces into the environment as inerts, adjuvants and degradates.

These comments will illustrate the need for adequate analysis to be performed on all components of the final mixture of pesticides, adjuvants and diluents. Anything less is in violation of the requirements of NEPA.

### **Active Ingredients**

The PEIS provides a very limited analysis of certain active ingredients, and no analysis of others. For those that receive no analysis, the rationale used for excluding them from analysis is covered in the following paragraphs;

*“In order to ensure that the agency fulfills its responsibility for protection of the public, Native American and Alaska Native subsistence practices, public land workers, and federally-listed species, species proposed for listing, and BLM special status species, a risk assessment was conducted (see appendices B and C). The assessment consisted of a comprehensive literature search, and in some cases new toxicological analyses, for (1) active ingredients currently in use to determine if there are new human health and ecological health risks that have been identified since the chemicals were last assessed (1988– 1992); and (2) active ingredients proposed for use by the BLM”. (EIS I-3)*

*“Since the late 1990s, the Forest Service has conducted ecological risk assessments (ERAs) for nine herbicide active ingredients also used by the BLM: 2,4-D, clopyralid, dicamba, glyphosate, hexazinone, imazapyr, metsulfuron methyl, picloram, and triclopyr. In addition, the Forest Service prepared interactive spreadsheets that allowed the BLM to determine exposure concentrations for plants and animals under different application rates and exposure scenarios for these herbicides. The ERAs are available at the Forest Service Pesticide Management and Coordination website <http://www.fs.fed.us/foresthealth/pesticide/risk.htm>. (PER 2-15) . (It should be noted that the url given here is incorrect, and leads to a dead page. The correct url ends with shtml, not htm).*

This rationale is false and can be upheld as such. To state that there has been no research in the last 15 years on either 2,4-D, clopyralid, dicamba, glyphosate, hexazinone, imazapyr, metsulfuron methyl, picloram, and triclopyr, that would warrant further concern, is simply wrong, and wrong by a wide margin. There are countless studies, all readily available through Toxline and PubMed, or other search engines, and many within the last few years, that show a need for great concern with the use of products containing these active

ingredients.

Case in point. The BLM risk assessment of these chemicals was 1988 or 1991. One of the above compounds, 2,4-D, is a commonly listed endocrine disruptor (ED). Alkylphenol ethoxylates, surfactants commonly used by the BLM in herbicide mixtures, are also EDs. Endocrine disruptors were unknown as a health problem until 1991, with the greatest advances in identification, and understanding, of the effects and pathways involved, occurring in the past seven years. This fact alone invalidates the assessment claims stated above in EIS 1-3.

If, as stated, there was a “comprehensive literature search” of “new human health and ecological health risks”, some very important data was overlooked. Without taking such evidence into consideration, BLM has failed in its analysis.

### **Inert Ingredients, Adjuvants, Degradates and, Omitted From Analysis, Diluents**

Even greater disregard is paid to adjuvants, inert ingredients, and known degradates; the effects of diluents have been ignored entirely. This area of discussion is particularly important in part due to the inconsistent approach to its analysis taken by BLM. Appendix C describes a list of herbicides and adjuvants to be used, but for most names only the active ingredient where, for a few, a formulation containing the active ingredient is named. Any number of inert ingredients and adjuvants can be associated with and used depending on the formulation employed. The formulations used should be identified so that the full range of inerts, diluents and degradates can be analyzed and so that it can be assured that no chemical, to the extent possible, will be used in the program, and thus no significant potential adverse impact will have been overlooked. Without a confirmed list of chemicals that may be used, the NEPA analysis cannot be accomplished.

Furthermore, BLM’s position is that new chemicals and formulations can be used if approved internally, without NEPA review (see Appendix D-2). Specifically, adoption of new formulations and new active ingredients would depend on pesticide registration of the product to stand in for the required analysis. This process cannot stand. It is in direct conflict with established law, as was recently cited in *Californians for Alternatives to Toxics et al v. California Department of Food and Agriculture*, \_\_ Cal.Rptr.3d \_\_; 2005 WL 3549483; 2006 Daily Journal D.A.R. 1204.

in which CATs argument that reliance on the registration process and labels of pesticides was not sufficient to satisfy the requirements of the California Environmental Quality Act. The state appeals court agreed, noting that

*Save Our Ecosystems v. Clark (9th Cir. 1984) 747 F.2d 1240 is instructive. There, the United States Forest Service had determined that certain herbicides could properly be used for defoliation activities, relying solely on their EPA registration under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA). The Ninth Circuit Court of Appeals held that “[t]he EPA registration process for herbicides under FIFRA is inadequate to address environmental concerns under NEPA [National Environmental Policy Act] . . . .” Instead, an agency must conduct independent research on the safety of herbicides it proposes to use.6 (Id. at p. 1248; see Northwest Coal. for Altern. to Pesticides v. Lyng (9th Cir. 1988) 844 F.2d 588, 596.) An agency can appropriately fulfill this duty of independent investigation by considering the registering agency’s data on herbicides in the specific context of the area targeted for proposed application. (Save Our Ecosystems v. Clark, supra, 747 F.2d at p. 1247.)*

BLM cannot depend on the registration of pesticides to satisfy the requirements of NEPA. All pesticides used in the program must be subject to NEPA analysis and, as an integral part of those pesticides, the so-named inerts of chemicals of the formulations, degradants and allowed diluents must be analyzed.

It has been clearly established in the scientific literature that the substances that contribute to the makeup of individual formulations, the adjuvants that increase the efficacy of specific formulations, and diluents that

may be allowed, by the registration label, can be highly toxic and often more toxic than the active ingredient. The BLM addressed these issues with the following;

Inerts,

*“Relatively little toxicity information was found. A few acute studies on aquatic or terrestrial species were reported. No chronic data, no cumulative effects data, and almost no indirect effects data (food chain species) were found for the inerts in the 10 herbicides.” (EIS C-83)*

Adjuvants,

*“In general, adjuvants compose a relatively small portion of the volume of herbicide applied; however, selection of adjuvants with limited toxicity and low volumes is recommended to reduce the potential for the adjuvant to influence the toxicity of the herbicide.” (EIS C-84)*

The Full Tank Mixture

*“Ecological Risks of Degradates, Inerts, Adjuvants, and Tank Mixtures – Only limited information is available regarding the toxicological effects of degradates, inerts, adjuvants, and tank mixtures. In general, it is unlikely that highly toxic degradates or inerts are present in approved herbicides. Also, selection of tank mixes and adjuvants is under the control of BLM land managers, and to reduce uncertainties and potential risks, products should be thoroughly reviewed and mixtures with the least potential for negative effects should be selected” (Bromacil ERA pg 7-10).*

Once again, this rationale and subsequent lack of analysis for these broad class of chemicals is unjustified, and illegal in respect to NEPA and ESA. There is a wide body of data concerning many effects from the list of BLM approved inerts and adjuvants and their degradates, readily available through internet search engines. We hope the following data we present will help you to understand this fact. Time constraints, however, force us to highlight only certain compounds, effects and pathways in this comment period.

One chemical family and associated health effects is herein highlighted for each of the individual concerns, ie; a) For AIs, 2,4-D/endocrine & reproductive; b) For inerts, POEA/multiple concerns; c) For adjuvants & degradates, NPE/endocrine disruption & acute toxicity. Endocrine disruption will be the primary health effect analyzed that has not been given analysis in the PEIS or supporting documents.

NEPA is very clear what agencies must do when there is insufficient data on a potential significant adverse effect: Describe the data gaps that need to be filled and describe either how those shall be filled or why it is not possible to do so.

*“When an agency is evaluating reasonably foreseeable significant adverse effects on the human environment in an environmental impact statement and there is incomplete or unavailable information, the agency shall always make clear that such information is lacking.*

*(a) If the incomplete information relevant to reasonably foreseeable significant adverse impacts is essential to a reasoned choice among alternatives and the overall costs of obtaining it are not exorbitant, the agency shall include the information in the environmental impact statement.*

*(b) If the information relevant to reasonably foreseeable significant adverse impacts cannot be obtained because the overall costs of obtaining it are exorbitant or the means to obtain it are not known, the agency shall include within the environmental impact statement: (1) A statement that such information is incomplete or unavailable; (2) a statement of the relevance of the incomplete or unavailable information to evaluating*

*reasonably foreseeable significant adverse impacts on the human environment; (3) a summary of existing credible scientific evidence which is relevant to evaluating the reasonably foreseeable significant adverse impacts on the human environment, and (4) the agency's evaluation of such impacts based upon theoretical approaches or research methods generally accepted in the scientific community. For the purposes of this section, "reasonably foreseeable" includes impacts which have catastrophic consequences, even if their probability of occurrence is low, provided that the analysis of the impacts is supported by credible scientific evidence, is not based on pure conjecture, and is within the rule of reason." (40 CFR 1502.22)*

## **ACTIVE INGREDIENTS**

It is important for BLM to present the analytical route and investigative tools used to arrive at the conclusion that scientific data had changed little in the toxic profiles of “2,4-D, clopyralid, dicamba, glyphosate, hexazinone, imazapyr, metsulfuron methyl, picloram, and triclopyr,” since 1992. It might open a window through which we could understand where this assessment was derived from and support claim that this analysis upholds NEPA.

In truth, as stated above, there has been a wealth of information presented by the scientific community concerning effects produced by some these pesticides in the last 14 years, as well as an entirely new toxicological paradigm (as evidenced by endocrine disruption). This new paradigm no longer lives by the classic rule, “the dose makes the poison”. The evolving science of endocrine disruption, by itself, warrants further analysis of all components in the final mix. To highlight this need, 2,4-D and endocrine/reproductive effects will be given as an example.

Currently, 2,4-D is listed as an endocrine disruptor (ED) by the European Union (Priority List), Illinois EPA and the Japanese National Institute of Health Sciences, and suspected as being a potential ED by most regulatory agencies, including the USEPA and the USGS. It is also in the process of being listed by CalEPA under Proposition 65 as a reproductive toxicant.

The science on endocrine effects produced by 2,4-D is well established, especially since 1992. That these effects were mediated via endocrine disruptor pathways is a more recent understanding. The 2,4-D supporting document, the Forest Service Risk Assessment for 2,4-D (SERA 1998), mentions some of the endocrine or reproductive effects studies prior to 1997, but never mentions endocrine disruption as a possible cause. SERA 1998 discusses Lerda and Rizzi’s (1991) sperm analyses study of pesticide applicators showing a possible link between sperm abnormalities and the use of 2,4-D (SERA 1998 p3-13). There is general discussion of thyroid effects (SERA 1998 p3-2). There is discussion of Jeffries et al (1995) with findings of high dose testicular atrophy, and a discussion of Charles et al (1996) with findings of low dose decreased testes weights (SERA 1998 p3-14). There is mention of earlier 2,4-D studies that showed endocrine effects. Nicolau (1983) found protein synthesis in the testes with more pronounced effects observed in the thyroid and adrenals. There is even mention of de Duffard et al. (1995) where it was demonstrated that the butyl ester of 2,4-D blocks the action of testosterone in the behavioral performance of castrated rats. And yet SERA 1998 makes no mention of endocrine disruption, even though 2,4-D was already suspected as an ED by regulatory agencies at that time, and the weight of the above data supports this assumption.

More recent data has established a clear link, and the USEPA has acknowledged that 2,4-D is neurotoxic and immunotoxic as well as a potential endocrine disruptor. Since the neurologic, immunologic and endocrine systems are interrelated, these neurotoxic and immunotoxic effects could be mediated via endocrine pathways.

In the recently released “Reregistration Eligibility Decision for 2,4-D” (USEPA 2005), the EPA states,

*“there is a concern for developmental neurotoxicity resulting from exposure to 2,4-D, and that a developmental neurotoxicity (DNT) study in rats is required for 2,4-D. The Agency has also concluded that a 2-generation reproduction study is required to address both the concern for thyroid effects and immunotoxicity, as well as a more thorough assessment of the gonads and reproductive/developmental endpoints” (USEPA 2005 p 81).*

*“When the appropriate screening and/or testing protocols being considered under the Agency’s Endocrine Disruption Screening Program (EDSP) have been developed, 2,4-D may be subject to additional screening and/or testing to better characterize effects related to endocrine disruption.....Based on currently available toxicity data, which demonstrate effects on the thyroid and gonads following exposure to 2,4-D, there is concern regarding its endocrine disruption potential. There have been no studies on 2,4-D that specifically assess its endocrine disruption potential” (USEPA 2005 p 21).*

More recent research has, however, assessed 2,4-D and ED potential. A new study not reviewed by the EPA was just released in Nov/2005. It is titled “Effects of 2,4-D and DCP on the DHT-Induced Androgenic Action in Human Prostate Cancer Cells” (Kim et al 2005). This study clearly shows that both 2,4-D and its DCP degradate are endocrine disruptors. This study also shows 2,4-D working in a synergistic fashion when mixed with 5{alpha}-dihydroxytestosterone (DHT).

It should also be noted that research (since 1992) showing serious adverse reproductive effects from 2,4-D is causing other regulatory agencies to express concern, or take appropriate actions, (ie CalEPA listing 2,4-D as a Proposition 65 reproductive toxicant).

The BLM, however, sees no need for concern in the body of scientific data provided since 1992 on endocrine, neurologic or reproductive effects relating to 2,4-D. When the BLM last did an ERA for 2,4-D, endocrine disruption was an unknown science. And though it was better understood by 1998, it isn’t mentioned in the SERA 1998 supporting document. Nor can it currently be found in the BLM PEIS or 2,4-D supporting documents. It has somehow missed detection during the BLM’s search for “new human health and ecological health risks” for 2,4-D.

A simple analogy. Just as a policeman wouldn’t let a speeder off because the motorist claims they never saw a speed sign, NEPA will not allow an agency to shrug their shoulders and cast a blind eye on relevant data that is available and needed for responsible decision making.

2,4-D is but one of the many herbicide AIs that could be used in this program. Endocrine disruption is but one of the many health effects that has been associated with chemicals proposed for use in this program. There are other known or suspected EDs in the list of approved BLM herbicides. The triazines, especially atrazine, are commonly listed. (And though the PER states that triazines are not to be used in this proposed program, they can be used with the current program without having an analysis of endocrine disruption ever employed.)

Other suspected EDs include bromacil (EPA-TRI, EU G2), diquat, and diuron (EU G2). Bromacil is suspected because of its ability to effect the thyroid system.

Diuron is considered to be potentially antiandrogenic because; a) of the great similarity in structure of diuron and the antiandrogen linuron and b) the common degradate, 3,4-dichloroaniline, of diuron and linuron, which was shown to bind to the androgen receptor (Cook et al., 1993). Picloram has also been suspected, and technical grades of picloram contain the contaminant hexachlorobenzene, a known ED.

There are also countless EDs found in the inerts, and adjuvants (or their degradates) currently approved for use. Alkylphenol ethoxylates, siloxanes, hexachlorobenzenes to name a few.

Other health effects that were not well understood during the writing of the BLM ERAs include effects from immunotoxicity and neurotoxicity. Great strides have been made in recent years by the scientific community, developing a body of data on these health effects. An FS RA found on the FS website listed by the BLM as the site for supporting documents, states it clearly;

*“Neurotoxicity, immunotoxicity, and endocrine disruption are three classes of effects that are important in any risk assessment” (SERA 2002)*

In truth, science has come a long way since 1991 with respect to toxicology in general. There are ample reasons why the AIs need to be reanalyzed, and new ERAs prepared. The above are only a few of them. Every AI, (as well as other components in the final mix), needs to be analyzed for the full range of known health

effects, including neurotoxicity, immunotoxicity and endocrine disruption. All data used in this analysis needs to be derived from published and peer reviewed studies and reports, to ensure the integrity of the analysis. Only then can the following statement be made;

*“In order to ensure that the agency fulfills its responsibility for protection of the public, Native American and Alaska Native subsistence practices, public land workers, and federally-listed species, species proposed for listing, and BLM special status species, a risk assessment was conducted...”. (EIS 1-3)*

The analysis must also take into account the fact that real world conditions can often increase the potency of toxic substances. Numerous studies, mostly with insecticides, have demonstrated that pesticide toxicity can increase with environmental factors, such as differences in temperature, water pH, and competition (Boone and Semlitsch 2002, Boone MD, Bridges CM 1999, Zaga et al. 1998). Predatory stress has been also shown to increase pesticide toxicity, in the case of carbaryl, making it anywhere from 4 to 46 times more lethal (Boone and Semlitsch 2001, Relyea and Mills 2001, Relyea 2003). Understanding the complexities of toxic response, and how seemingly insignificant factors can produce different results, is one of the recent advances in the science of toxicology. The following is from Relyea;

*The current study suggests that the lethal concentrations of carbaryl (and perhaps other pesticides) can be much lower than we currently appreciate because traditional toxicology studies frequently isolate animals from their natural ecology (including predator cues). When we include some of the natural ecology, even low concentrations of a pesticide can be highly lethal to amphibians. In short, ignoring the relevant ecology can cause incorrect estimates of a pesticide’s lethality in nature, yet it is the lethality of pesticides under natural conditions that is of utmost interest. The accumulating evidence strongly suggests that pesticides in nature could be playing a role in the decline of amphibians (Relyea 2003).*

Though this quote (and studies cited) concerns carbaryl, it’s implications are clear. Recent research on toxicity and stress, has also been performed with a pesticide approved for this program, glyphosate, and six species of North American amphibian larvae. The results were similar, with the LD50 for RoundUp lowering from 1.5 to 15.5 mg AI/L to 0.55 to 2.52 mg AI/L (Relyea 2005a). Relyea, concludes;

*“This discovery suggests that synergistic interactions between predatory stress and pesticides may indeed be a generalizable phenomenon in amphibians that occurs with a wide variety of pesticides” (Relyea 2005a).*

## **Confusing Citations**

There are a couple of points in need of clarification, because it is very difficult to follow the winding trail that has been provided by BLM for locating supporting documents, including the SERA 2,4-D risk assessment.

First, on page PEIS B-1 and C-1, you state that the more recent document, the invasive plant EIS, USDA Forest Service (FS) 2004, will be used as the main supporting document. This document, however, is cited in your bibliography as USDA FS 2005a, which is the correct citation date, as the FEIS was released in April 2005. To avoid confusion, this document will be cited here as FS IPEIS 2005.

Second, on page PER 2-15, as stated above, the location for reviewing the SERA supporting documents is given as <http://www.fs.fed.us/foresthealth/pesticide/risk.htm> . Once discovering that this url is a disconnect, I was finally able to link through sera-inc.com . The proper url is <http://www.fs.fed.us/foresthealth/pesticide/risk.shtml> .

Third, upon downloading the SERA 2,4-D RA, the report date of 1998 was noticed. Having already reviewed SERA 1998, we expected SERA 2003a (as cited in the BLM PEIS) to be an update. However, SERA 2003a was the same as SERA 1998, as evidenced by the same TR numbers. The proper citation, as listed in FS

IPEIS 2005, is SERA 1998 (FS IPEIS 2005 references-22). Though some typos and misprints have no effect on the material, wrong citations can lead to hours of frustrating search looking for something that doesn't exist. This is in violation of NEPA and the APA.

## **OTHER INGREDIENTS IN THE FULL TANK MIXTURE**

Another important fact that has been overlooked by BLM in your preparation of the EIS, is, quite simply, whatever comes out the end of the spray nozzle, is the material that BLM is adding to the environment, and which must be analyzed for potential adverse impacts to satisfy the requirements of NEPA. To isolate components of the final mix and pretend that their introduction into the environment is not your responsibility, is, to put it mildly, absurd, in addition to being in violation of law. To state that analysis cannot be attempted because it wouldn't fit into current modeling does not re-leave you of performing other aspects of amassing data and evaluating potential effects (PEIS p C-78).

This fact has been highlighted by regulatory agencies. Environment Canada, in CEPANP 2001, have detailed why it is important to analyze the full mixture and have analyzed effects from both NPE and it's degradates individually, and in combination as they are found in the environment. The following are quotes from the CEPANP 2001.

*“Because NPEs occur as complex mixtures in the environment and have different toxicities and estrogenic potencies, the approach used in this assessment was to first assess each chemical separately, then assess the complex mixtures found in the environment” (CEPANP p 42).*

*“In one study, NP2EO and NP1EC were only slightly less potent than NP in inducing vitellogenin in trout hepatocytes. NP, NPEs and NPECs are found as complex mixtures in effluents, and their combined estrogenic effects on aquatic organisms should be considered together..... Estrogenic responses occur at concentrations similar to those at which chronic toxicity occurs, although biochemical and histological changes have been reported at concentrations a factor of 10 lower” (CEPANP p 2).*

*“The relative estrogenic potency determined in several different in vitro systems is in the order NP > NP1EO = NP2EO > NP1EC = NP2EC > NP9EO. The estrogenic responses appear to be at least additive and should, therefore, be considered as a group” (p 28).*

*“It is important that all of the NPE metabolites, not only NP, be considered together to assess the potential for impacts in the environment” (CEPANP p 2).*

*“In addition to examining the exposure and toxicity of each metabolite individually, a toxic equivalency approach was applied, which factored in contributions from NP as well as the lower-chain-length (1,2) NPEs and NPECs to determine the overall potential risk of the group” (CEPANP p 46).*

*“As observed in field measurements, NP and NPEs occur as complex mixtures, and the toxicities of the metabolites are expected to be additive. When NP is considered alone, only three sites have predicted concentrations in receiving waters that exceed a value of 1 µg/L. When NP1EO and NP2EO are considered in addition to NP, an additional four sites exceed the ENEV” (CEPANP p 57).*

Environment Canada saw the need to analyze all components of a compound, including any degradates that might be present, after release into the environment. This is known as real world modeling, and something that is critical to an honest evaluation of potential effects. The family of chemicals they are addressing are common use surfactants that have been, and probably will continue to be, used frequently by the BLM.

NPEs and degradates are just one of the many chemicals proposed for use whose known toxicity contradicts the following BLM statement;

*“Only limited information is available regarding the toxicological effects of degradates, inerts, adjuvants, and tank mixtures. In general, it is unlikely that highly toxic degradates or inerts are present in approved herbicides”.*

Again, nothing could be further from the truth. In fact, this statement is almost beyond comprehension. The scientific body of toxicological data concerning chemicals used as inert ingredients or adjuvants, or their degradates, by the BLM, is massive. Some of these substances are 100 to 10,000 times more toxic than the AI they are mixed with. Because the BLM is having trouble locating this data, might we suggest that you start with studies referenced here. Many of these studies then reference other studies which can be then acquired, and those studies reference other studies, etc etc. This is known as following a reference trail, and very helpful for locating data about specific chemicals or health effects.

Interesting enough, the BLM is aware of the importance of viewing the application of the final mix as a singular action. In the Ecological RA you state;

*In a detailed herbicide risk assessment, it is preferable to estimate risks not just from the a.i. of an herbicide, but also from the cumulative risks of degradates, inert ingredients (inerts), and adjuvants.....However, using currently available models (e.g., GLEAMS), it is only practical to make deterministic risk calculations (i.e., exposure modeling, effects assessment, and RQ derivations) for a single a.i.” (PEIS p C-78).*

This statement raises some serious questions. First, if the NEPA analysis of an agency program calling for the treatment of a million acres a year with toxic substances isn't the right time for a detailed risk assessment, when is? Second, just because current modeling standards don't allow for cumulative analysis risk quotients of all ingredients, this does not excuse BLM from analyzing the individual components and then making assessments as to their cumulative effect. And irrespective of how you perform a cumulative analysis, the individual substances need to be analyzed and toxicologically profiled. As NEPA demands, the BLM must perform the following steps;

1) State “that such information is incomplete or unavailable.”

2) “A statement of the relevance of the incomplete or unavailable information to evaluating reasonably foreseeable significant adverse impacts”.

3) “A summary of existing credible scientific evidence which is relevant to evaluating the reasonably foreseeable significant adverse impacts”.

4) “An evaluation of such impacts based upon theoretical approaches or research methods generally accepted in the scientific community”. (40 CFR 1502.22)

BLM must analyze all components of the final mixture that pose a risk to human health or the environment. This is an unavoidable reality. NEPA demands it, for endangered species ESA demands it, and responsible decision making demands it. Claiming that there is no data available for review, when there is a wide body of data with relevant information, does not free BLM from its responsibilities.

In the Consent Decree for Californians for Alternatives to Toxics et al v. The Environmental Protection Agency, C00-3150 CW, EPA agreed to consider inert ingredients, adjuvants, degranants and diluents in ESA consultations undertaken under the CD. This approach should be applied to all analysis of pesticide use in the BLM program where listed species may be affected. The BLM has failed to make such analysis in the draft PEIS.

Each individual component must be analyzed as an individual action. Then, an analysis of the cumulative effects from all components of the final mix must be performed. Where similar modes of action are identified, this fact must be addressed. There should also be an analysis of cumulative effects both for environmental effects and as they pertain to the general body burden of an individual or species, including how this affects the immune system.

The lack of analysis performed by BLM, is a curious act when one considers that, as the PEIS states on the last page of the PEIS Appendix C;

*“ERAs will assist BLM field offices on the proper application of herbicides to ensure that impacts to plants and animals and their habitats are minimized to the extent practical”. (PEIS C-88)*

The ERAs, or other toxicological analysis, are integral to sound decision making. Without this knowledge at the program level, site specific analysis becomes a pointless exercise. Yet the BLM seems to think that, absent ERAs, field workers and land managers will still be able to arrive at informed choices. The PEIS further states;

*“selection of adjuvants with limited toxicity and low volumes is recommended to reduce the potential for the adjuvant to influence the toxicity of the herbicide”. (PEIS C-86)*

*“The composition of such mixtures is highly site-specific, and thus nearly impossible to address at the programmatic level of the EIS”. (PEIS C-84)*

The PEIS states that selection of tank mixes and adjuvants is under the control of BLM land managers, and to reduce uncertainties and potential risks, products should be thoroughly reviewed and mixtures with the least potential for negative effects should be selected. As we have noted previously, this is in violation of established law.

None of this rationale concerning analysis or use of inerts, adjuvants, and degradates, satisfies legal requirements. On one hand, BLM is saying that ERAs are important to decision making. On the other hand BLM is saying that analysis of the full mixture is a) impossible to address and b) will be addressed adequately at the site specific level because land managers and field workers will have the knowledge needed to pick the components that will produce the least toxicity.

How can land managers and field workers accomplish this task? Without documentation in support, the BLM claims that there is little data available concerning inerts, adjuvants and degradates currently used by BLM. Then BLM claims that the issues are too complex and are outside the scope of the EIS. Yet BLM expects land managers to have enough information to choose the right combinations of inerts and adjuvants to limit toxicity. If BLM does not provide this data at the program level, where is this knowledge going to come from? Does BLM think that their land managers and field workers spend their free time doing Toxline and PubMed data searches, and studying up on the latest health effects in medical journals, in order to have the knowledge needed to perform their task of choosing *“mixtures with the least potential for negative effects”*.

There needs to be an NEPA analysis of individual components at the program level and any additions made in the future so land managers will have a basic understanding of potential effects at the project level. Congress intended that NEPA would serve this purpose. Currently, the Biological Assessment for this program does not even contain the words inert and adjuvant, let alone analyze potential effects, thus illustrating that BLM’s failure to realize these mandates have created an Agency-wide incompetence.

We will demonstrate that there is ample data for analysis purposes concerning certain inerts, adjuvants and degradates, and that this data shows serious toxic effects associated with these substances. For inerts we primarily highlight the relationship between glyphosate and POEA in the RoundUp formulation. POEA is one of the more studied inerts among those currently in use by the BLM. For adjuvants and degradates, we join these two, as the surfactants in the alkylphenol ethoxylate family breakdown rapidly to degradates that are many times more toxic than the parent compound. We use nonylphenol ethoxylates, as found in the R-11 adjuvant, to highlight these concerns. We also highlight the health effect, endocrine disruption. As stated above, this health effect was unknown until recently but has been available for BLM analysis if it had been undertaken as required. There are chemicals proposed for use by BLM that are known EDs that have never been analyzed in regard to this effect.

Hopefully, this data will help BLM to see the insufficiency of the statement *“it is unlikely that highly*

*toxic degradates or inert ingredients are present in approved herbicides”.*

## **INERT INGREDIENTS**

The list of inert ingredients associated with the herbicides approved for use by BLM is extensive. Many are unknown ingredients. For the nine pesticide AIs proposed for use in this program that have received ERAs, inert ingredients in the approved products contain nine inert ingredients with unknown toxicity (EPA List 3), and nine inert ingredients that are completely unknown. 18 chemicals that could run the gamut, in terms of toxicity, from dioxin to water or anywhere in between (PEIS C-83). And the list of products associated with the above nine, is far less than the number associated with the 10 that have not received analysis, some of which have been on the commercial market for sometime, with many different formulations, (2,4-D, clopyralid, dicamba, glyphosate, hexazinone, imazapyr, metsulfuron methyl, picloram, and triclopyr). Inert ingredients are misnamed, as the EPA itself notes in attempting to change the term to “other ingredients” to better define these frequently very active and toxic chemicals.

### **RoundUp and POEA**

However, the names of many inert ingredients are known, and from this list we can assess effects. POEA (polyethoxylated tallowamine, listed as polyoxethylene-alkylamine by Monsanto) is the primary inert ingredient in the original RoundUp formulation. In every scientific study we have reviewed that deals with the differences in toxicity between glyphosate and POEA in the RoundUp formulation, it has been shown, or stated, that the surfactant POEA was the primary contributor to the toxicity of RoundUp. The following from Relyea (2005b, 2005c) summarizes some of these findings;

*“Mann and Bidwell (1999) found that LC5048-h values in the laboratory ranged from 3.9 to 15.5 mg AI/L for Roundup (glyphosate plus POEA surfactant), 108 to 161 mg AI/L for technical grade glyphosate acid, and 450 mg AI/L for glyphosate isopropylamine salt (the latter two formulations lack the POEA surfactant). Perkins et al. (2000) conducted laboratory experiments on Xenopus laevis tadpoles and found LC5096-h values of 12.4 mg AI/L for Roundup, 6.8 mg/L for the POEA surfactant alone, and 9729 mg AI/L for Rodeo (an aquatic form of glyphosate that lacks the POEA surfactant). Smith (2001) examined the impact of Kleeaway (another form of glyphosate that includes the POEA surfactant) and found that nearly half of western chorus frog tadpoles (Pseudacris triseriata) died at 0.75 mg AI/L; plains leopard frog larvae (Rana blairi) experienced 0% and 100% survival at 0.75 mg AI/L in two separate experiments. All tadpoles of both species died at higher concentrations (7.5, 750, and 7500 mg AI/L). These studies suggest that the high mortality associated with commercial forms of Roundup is actually due to the POEA surfactant and not to glyphosate itself (Relyea 2005b)”.*

*“A critical question in interpreting the results of the aquatic and terrestrial experiments is whether the high rates of mortality observed were due to the active ingredient of Roundup (glyphosate) or whether they were due to the added surfactant (POEA).....laboratory studies have shown that glyphosate alone has a low toxicity while the POEA surfactant can be highly toxic to a variety of taxa including amphibians (Mann and Bidwell 1999, Giesy et al. 2000, Perkins et al. 2000, Lajmanovich et al. 2003, Tsui and Chu 2003, Edginton et al. 2004, Howe et al. 2004). The current study did not isolate the impacts of glyphosate and the surfactant, so one cannot determine which component of Roundup caused the mortality, but it seems likely that the surfactant was the cause (Relyea 2005c)”.*

*“ecologically relevant concentrations of Roundup can cause substantial mortality in some species of amphibian larvae and that this death is primarily due to the POEA surfactant (Relyea 2005c)”.*

This evaluation of increased toxicity with increased levels of inert POEA has been recognized for

sometime. These concerns have been analyzed in SERA 2003b, where the authors state that;

*“Much of the available information on the toxicity of surfactants used with glyphosate have been summarized in SERA (1997) and more recent studies (Chang et al. 1999; Garry et al. 1999; Lin and Garry 2000; Reluso et al. 1998) reenforce the conclusion reached in SERA (1997) that some surfactants may be more toxic than the herbicides with which they are used” (SERA 2003b p 3-20).*

*“Both of these studies indicate that POEA is substantially more toxic than glyphosate and that POEA surfactant is the primary toxic agent of concern”(SERA 2003b p 4-14).*

*“As in the human health risk assessment, the formulation of glyphosate with surfactants, especially the POEA surfactant commonly used in glyphosate formulations, has a pronounced effect on the acute lethal potency of glyphosate”. (SERA 2003b p4-2).*

In the study referenced in the paragraph above, SERA 1997, as well as in SERA’s earlier 1996 RA for glyphosate, it states;

*“For aquatic organisms, the surfactant is much more toxic than glyphosate. Unlike glyphosate, POEA is more toxic in alkaline water than in acid water”.*

## **Endocrine Effects**

Relyea’s findings are consistent with the known science on POEA toxicity. Other scientists have found numerous health effects that are associated primarily with the inert POEA in glyphosate formulations. Others have shown a synergistic relationship between glyphosate and POEA. In Richards 2005, the increased toxicity of the POEA surfactant formulations was said to be a two edged sword. On one hand, the POEA increased toxicity of, and by itself. On the other, it facilitated penetration of glyphosate through cell walls (a primary function of surfactants) increasing the toxic effect of the AI.

It should be noted that Richards 2005 also raises serious questions concerning endocrine mediated effects from the full formulation. After an hour’s incubation with Roundup, estrogen synthesis in placental cells (as shown by aromatase activity) was enhanced by about 40%. After 18 hours, however, synthesis was inhibited, perhaps reflecting an effect on aromatase gene expression. This effect was not seen with glyphosate alone. Roundup also disrupted aromatase activity at concentrations 100 times lower than those used in agriculture. The following are quotes from Richards 2005;

*“Roundup is always more toxic than its active ingredient”.*

*“We conclude that endocrine and toxic effects of Roundup, not just glyphosate, can be observed in mammals. We suggest that the presence of Roundup adjuvants enhances glyphosate bioavailability and/or bioaccumulation”.*

*“glyphosate’s mechanism of action in mammals is still questioned, and it may have several enzymatic effects (Darwich et al. 2001; Williams et al. 2000). It has also been recently shown to disrupt the animal cell cycle in urchin eggs (Marc et al. 2002) and even the post-transcriptional expression of the steroidogenic acute regulatory protein (StAR) in mouse testicular Leydig cells (Walsh et al. 2000).....Walsh showed that Roundup preferentially diminished the expression of StAR mRNA by decreasing at least the rate of gene transcription”*

*“For example, the harmful effect of glyphosate on semen quality after 6 weeks of post-treatment period in rabbits (Yousef et al. 1995) may be considered an indication of its retention and conjugation in the body,*

helped by Roundup adjuvants”.

*“Glyphosate penetration through the cell membrane and subsequent intracellular action appeared in our work to be greatly facilitated by adjuvants, as in plants (Haefs et al. 2002) or in animal cells, where it can act at the level of cycle regulation (Marc et al. 2002). Indeed, in this work, minute dilutions of Roundup bringing adjuvants to cells allowed the aromatase inhibitory effect of glyphosate as well as cytotoxic effects”.*

*“Our studies show that glyphosate acts as a disruptor of mammalian cytochrome P450 aromatase activity from concentrations 100 times lower than the recommended use in agriculture; this is noticeable on human placental cells after only 18 hr, and it can also affect aromatase gene expression. It also partially disrupts the ubiquitous reductase activity but at higher concentrations. Its effects are allowed and amplified by at least 0.02% of the adjuvants present in Roundup, known to facilitate cell penetration, and this should be carefully taken into account in pesticide evaluation. The dilution of glyphosate in Roundup formulation may multiply its endocrine effect. Roundup may be thus considered as a potential endocrine disruptor. Moreover, at higher doses still below the classical agricultural dilutions, its toxicity on placental cells could induce some reproduction problems”.*

In the study cited above by Richards, Marc et al. 2002, the synergistic relationship between glyphosate and POEA was explored further. Researchers found that glyphosate exhibited the toxic response, and that the toxic response was synergistically amplified with increasing amounts of surfactant.

*“The delay is dependent on the concentration of Roundup. The delay in the cell cycle could be induced using increasing glyphosate concentrations (1-10 mM) in the presence of a subthreshold concentration of Roundup 0.2%, while glyphosate alone was ineffective, thus indicating synergy between glyphosate and Roundup formulation products”.*

*“Roundup affects cell cycle regulation by delaying activation of the CDK1/cyclin B complex, by synergic effect of glyphosate and formulation products. Considering the universality among species of the CDK1/cyclin B regulator, our results question the safety of glyphosate and Roundup on human health”.*

*“Roundup contains surfactants, which promote wetting of plant surface and rapid herbicide penetration. In previous reports, the toxicity of Roundup was ascribed to the surfactant component (Mitchell, et al 1987). We show that glyphosate and formulation products act in synergy on the cell cycle indicating an effect of glyphosate by itself. Our experiments indicate that glyphosate requires the presence of the formulation products to be effective on the embryo. It is likely that the formulation products favor the penetration of glyphosate in the embryos that were already reported to be impermeable to some compounds (Epel, D. 1990)” (Marc et al. 2002).*

This indirect effect associated with surfactants, carrying toxic substances through cell walls to cause effect, is in need of thorough evaluation. Data exists showing the ability of surfactants to carry toxic substances through plant surfaces, and a growing body of data showing similar transport through animal cell walls. This is a serious issue, and as Marc et al state, *“our results question the safety of glyphosate and Roundup on human health”*. The BLM must consider such warnings in its NEPA analysis but has failed in the draft PEIS.

A 2005 study from Marc has verified their earlier findings. POEA facilitates the toxic effects of the AI glyphosate, as well as providing it's own toxicity.

*“The adverse effect on transcription involves the commercial product and therefore is the result of a combination of the formulation products. The contribution of glyphosate to the adverse effect of Roundup was*

*investigated and demonstrated by two lines of evidence. On the one hand, four different glyphosate-based formulations provoked a delay in hatching at glyphosate concentration within similar range. On the second hand, an additional effect on hatching was observed when a threshold amount of Roundup was supplemented with pure glyphosate. However, our results do not exclude a contribution of the formulation products to the Roundup effect: first, because permeabilizing agents are required for glyphosate effect as a herbicide (Williams et al., 2000) or as a cell cycle deregulator (Marc et al., 2002), for the intracellular access of the chemical to its molecular targets. Second, because the major component of Roundup, polyoxyethylene amine (POEA), was found to be highly toxic to the embryos and led to lethality. Such higher toxicity of POEA compared to Roundup has been observed on other aquatic organisms (Tsui and Chu, 2003). Altogether, the adverse effect of Roundup on hatching is due, at least in part, to the active herbicide component glyphosate, which reaches its intracellular molecular target through the synergic effects of the formulation ingredients. Regarding the potential human health concern, it is important to note that glyphosate is never sprayed for herbicide usage with/without the formulation compounds (Williams et al., 2000)” (Marc et al 2005).*

There is a wealth of studies and information available concerning the toxicity of the inert surfactant POEA in certain glyphosate formulations. What has been presented here is only the tip of the iceberg.

Addressing the surfactant POEA, in respect to all the other inerts proposed for use with the AIs, is again only the tip of the iceberg. One need only look at the different formulations of glyphosate available. SERA 2003b stated;

*“Appendix 3c summarizes the available ecological information from all of the MSDS’s for the formulations that are labeled for forestry applications. It is apparent that these formulations fall into relatively clear groups. The most toxic formulations appear to be Credit Systemic, Credit, Glyfos, Glyphosate, Glyphosate Original, Prosecutor Plus Tracker, Razor SPI, Razor, Roundup Original, Roundup Pro Concentrate, and RoundupUltraMax. It may be presumed that these formulations contain the most toxic surfactants. Other formulations such as Aqua Neat, Aquamaster, Debit TMF, Eagre, Foresters’ Non-Selective Herbicide, Glyphosate VMF, and Roundup Custom are much less acutely toxic. Some of these, however, require or recommend the use of a surfactant and this would likely increase the toxicity of the formulation” (SERA 2003b p 4-13).*

Each of these formulations contain different inerts. As stated above, it is assumed that the surfactant is the deciding factor in gaging toxicity. This assumption is based on the wide body of scientific data concerning the toxicity of these surfactants, especially, but certainly not limited to, POEA.

There are countless inerts in use, or proposed for use, by the BLM. Some are identified through the EPA list of inerts which, (especially in terms of list 3), is dated and provides insufficient data. Some are highly toxic, some are not. The affected community (BLM employees, contract workers, anyone passing through or near the annually treated million acres, all wildlife that call these acres home, etc.) is depending on BLM to amass as much pertinent data, with help from the scientific and environmental communities, and perform an honest evaluation of the known data. It is required under NEPA.

## **ADJUVANTS AND DEGRADATES**

When dealing with additives to a tank mixture, it makes little difference whether the chemical is an inert (premixed with the AI in a commercial mixture), or an adjuvant (mixed in at the tank stage). This is especially true for surfactants, a necessary component of most herbicide mixtures. Since they are essential, their use can be expected. And this use must be analyzed at the program level, in order for there to be reasoned decision making at the project level.

In recent years, government agencies have started moving away from the use of formulated products,

leaving the mix to be performed on site. There are two basic reasons for this. One is that it gives land managers more choices at their disposal.

The other is, after certain formulations like RoundUp began to show greater toxicity than the AI alone, agencies tried to find a way to get risky projects past public scrutiny and the courts with only the AIs needing analysis. They felt they could do this by separating the mix. To explain this, we will use as an example RoundUp and Accord/Rodeo.

In *NCAP v Lyng*, 844 F.2d 598 (9<sup>th</sup> Cir. 1988), the courts sided with the Forest Service and said that the only analysis needed for inerts, adjuvants and degradates, was to compare the toxicity of commercial formulations and the AI, perform searches with the help of the EPA and the chemical companies to identify any concerns with these formulated products, and disclose if testing has been done to these products. Analysis was directed at the formulated product.

Though the level of analysis being demanded is actually quite low, the FS wanted to reduce their “analysis paralysis” wherever possible. They also wanted to avoid having to search for and disclose toxicity data, because this opened the door to public concerns and potential court challenges.

So formulated products like RoundUp were used less, and formulated products that were nothing more than the AI plus water, like Accord, took their place. This of course meant having to add more chemicals at the mix stage, adding one more wrinkle to operations. This was interpreted to mean that only the AI needed analysis. By removing the inerts from the picture with otherwise formulated products, and instead adding them at the tank stage as adjuvants, analysis, it was apparently concluded, was not required.

This, however, proved to be a poor interpretation of NEPA. The courts said that a review formulated products was enough, because at the time, there was little toxicity data other additives. The situation has changed: Today there is a wealth of information on the toxicity of many adjuvants and degradates.

The Cottonwood Fire Vegetation Management Project was proposed using this familiar tactic, delaying analysis of the additives. One of the additives, however, was R-11, (which interestingly enough, is also approved for use with the BLM and this program). R-11 contains a highly toxic surfactant, nonylphenol ethoxylate (NPE), that degraded into even more toxic and persistent substances. One of the health effects clearly associated with NPE and it’s degradates was endocrine disruption.

The scientific community and concerned citizens alerted the FS to the toxicity of R-11, and its association with endocrine disruption, during the comment period. The FS turned a blind eye to this data and claimed that a thorough analysis of adjuvants, that weren’t a part of the formulated herbicide product, wasn’t required by NEPA law. The courts strongly disagreed, and forced the FS to perform an analysis of an adjuvant because the toxicity and potential for harm from endocrine disruption was clearly demonstrated.

In *Californians for Alternatives to Toxics v. Dombeck*, NO. CIV. S-00-2016 LKK/JFM, the court found that:

*Plaintiffs argue that the risk of endocrine disruption posed by the adjuvants to the herbicides proposed for the Cottonwood Project require the Forest Service to prepare an environmental impact statement. □Endocrine disruption was not addressed in the 1988 R5 VMR FEIS. The Forest Service was notified that endocrine disruption is a serious concern even with extremely low doses of the adjuvants, R-11 and MOR-ACT, which will be used with the full formulations of the two herbicide mixes approved for this project... In light of the significance of this new information and the failure of the Forest Service to support its FONSI with sufficient data, Warm Springs Dam Task Force, 621 F.2d at 1024, the court concludes that an SEIS is required to analyze the endocrine-disrupting properties, immunotoxicity and neurotoxicity caused by the herbicides to be used.*

It should also be noted that California requires that adjuvant formulation be registered as pesticides and their use accounted for in Pesticide Use Reports submitted to County Agricultural Commissioners by the BLM.

Clearly, the BLM cannot avoid its responsibility to analyze the full range of chemicals it will use in the

current program.

Since many of the issues are similar or even identical to the current proposed program, we would respectfully ask that the public records for both of the Cottonwood Fire Vegetation Management Projects EAs, and the public record for the current Cottonwood Fire Vegetation Management Project FEIS, be incorporated by reference into the public record for this program, the BLM Vegetation Treatments Using Herbicides PEIS.

And below, we will clearly demonstrate why adjuvants, as well as degradates from the final mix, are in need of thorough and honest evaluation by the BLM before approving this program.

## **Diluents**

Diluents allowed to be mixed with certain pesticide formulations can also be toxic chemicals that can have significant environmental effects.

For example one diluent is diesel oil that, by label, can be mixed with Garlon 4, a triclopyr formulation commonly used by BLM. According to the label, up to 99 gallons of diesel oil may be mixed with each gallon of Garlon 4 to aid in the effectiveness of the formulation.

As Irwin, Stevens, and Basham note (1997. Environmental Contaminants Encyclopedia. National Park Service, Water Resources Division, <http://www.nature.nps.gov/hazardssafety/toxic/diesel.pdf>) diesel has multiple effects on the environment due to its properties as an oil and also the effects individually and cumulatively of the chemicals which constitute the compound.

BLM must analyze the effects of any diluent, including diesel, that may be used with any of the herbicides in its proposed program.

## **Nonylphenol Ethoxylates**

Appendix A (Comments to the R6 IPEIS (2005) is an updated version of comments submitted to the FS during the R6 IPEIS comment period. These comments are extensive and will be summarized here. The updating includes addition of recent studies, as well as making the comments generic, (ie. not specific to a particular program or project). All issues discussed and studies cited apply to this PEIS as well.

Once again, because the issues are identical, we would respectfully ask that the public record for the Forest Service's R6 Invasive Plant Program EIS, 2005, (which the BLM is tiering to), be incorporated by reference into the public record for this program, the BLM Vegetation Treatments Using Herbicides PEIS.

Some of the comments provided in Appendix A refer to the document USDA 2003 (or Bakke 2003). This is the current risk assessment for NPEs used by the FS. Since the BLM is piggybacking on FS RAs, it is assumed that the BLM will attempt to piggyback on the USDA 2003 as a supporting document for endocrine disruption effects from NPEs and other EDs proposed for use. This would be a mistake, as USDA 2003 is limited in its understanding of endocrine disruptor effects, poorly written, and outdated.

In truth, USDA 2003 is nothing more than white wash of issues, and could have just as easily been written by an industry sponsored NGO. It does nothing to further an understanding of ecological concerns, or assess risk in a responsible fashion. It is not peer reviewed, uses questionable data that was not peer reviewed, ignores data that disagrees with its findings, and relies on too much industry input to arrive at its assessment.

There are two types of risk assessments being performed these days. One type uses peer reviewed data, and attempts to responsibly address the issues presented. The other type is nothing more than a "yes paper". It is amassed for the sole purpose of getting a project past NEPA requirements. The analysis will usually be written in such a way as to confuse most members of the public, and hopefully fool a judge (if need be). USDA 2003

appears to be of the latter.

## Background

Nonylphenol ethoxylates (NPEs) are adjuvants approved by BLM and commonly used with glyphosate and other herbicides, performing the same function as POEA. NPE and its degradates are more toxic than POEA, and have been shown to produce a wide range of toxic effects, both acute and chronic.

NPEs are nonionic surfactants, identified numerically by their ethoxylate chain length, and are a class of a broader group of compounds known as alkylphenol ethoxylates (APEs). NP is a chemical intermediate composed of a phenol ring attached to a lipophilic straight or, more usually, branched nonyl group.

NPEs and their degradation products (e.g., nonylphenol [NP]) are not produced naturally. Their presence in the environment is solely a consequence of anthropogenic activity. The mechanism of degradation is complex, but, in general, there is an initial loss of ethoxylate (EO) groups from the original moiety. Under aerobic and anaerobic treatment conditions, biodegradation to more persistent, toxic and hormonally active degradates occurs. These products include NP, nonylphenol ethoxylate (NP1EO), nonylphenol diethoxylate (NP2EO), nonylphenoxyacetic acid (NP1EC) and nonylphenoxyethoxyacetic acid (NP2EC) (Environment Canada, 2001).

Analysis of toxic effects will be divided into two sections, endocrine system effects, and non-endocrine effects.

## Endocrine Effects

### Overview

The endocrine system consists of a set of glands, the thyroid, parathyroids, testes, ovaries, adrenal, hypothalamus, pancreas, pineal, and pituitary glands, as well as other chemical regulators; and the hormones they produce, such as thyroxine, oestrogen, testosterone and adrenaline, which coordinate and regulate internal communication in cellular organisms. Endocrine cells release chemical messengers, known as hormones, which are carried into contact with target cells in the body. Interactions between the hormone and particular recognition features (receptors) in the cell, trigger pre-existing cellular responses that may result in effects on growth, behavior, development, or reproduction, as well as numerous other critical biological functions.

The currently recognized definition of endocrine disrupting chemicals is;

*“An endocrine disruptor is an exogenous substance or mixture that alters function(s) of the endocrine system and consequently causes adverse health effects in an intact organism, or its progeny, or (sub)populations. A potential endocrine disruptor is an exogenous substance or mixture that possesses properties that might be expected to lead to endocrine disruption in an intact organism, or its progeny, or (sub)populations”* (WHO, 2002).

### Modes of Action

Endocrine disruptors interfere with the functioning of the endocrine system, in at least four possible ways:

1) mimic or partly mimic the sex steroid hormones estrogens and androgens (the male sex hormone) by binding to hormone receptors or influencing cell signaling pathways. Those that act like estrogen are called environmental estrogens.

2) block, prevent and alter hormonal binding to hormone receptors or influencing cell signaling pathways. Chemicals that block or antagonize hormones are labeled anti-estrogens or anti-androgens.

3) affect the synthesis, transport, metabolism and excretion of hormones, thus altering the concentrations of natural hormones.

4) modify the making and function of hormone receptors.

However, an understanding of endocrine disrupting processes gets more and more complicated as new research findings are revealed. New compounds, modes of action, LOECs or endpoints are being uncovered on a regular basis. And for every question that is answered, new questions arise. One of the biggest, and probably most complex, mysteries is how substances with different shapes and structures produce similar physiological results.

An example of this is the recent work by Bulayeva and Watson, 2004, where the authors found dissimilar chemicals producing similar effects, at dosing levels never before recorded. The following is from Bulayeva 2004;

*“Compounds from different classes of endocrine disruptors with dissimilar chemical structures (e.g., endosulfan as an organochlorine compound vs. nonylphenol as a simple phenolic detergent) can produce the same time-dependent activation pattern for ERKs.”*

*“There are likely to be specific pathways within the nongenomic signaling network that individual compounds will trigger, leading to different functional end points. Therefore, each xenoestrogenic compound must be tested for an array of possible mechanistic routes of action”.*

*“Possible reasons for these potent effects not being noted previously are that little testing of the nongenomic pathway has been done, many tests did not examine such low concentrations, and some test conditions probably did not adequately remove endogenous estrogen levels (as we have done by use of low quantities of extensively charcoalstripped serum) to reveal effects of these low concentrations. The potent effects we see on nongenomic signaling mechanisms could explain why concentrations previously determined to be inactive via genomic mechanisms still have toxic and teratogenic effects on wildlife (Brucker-Davis et al. 2001). Therefore, the threat levels of these compounds to wildlife, and probably humans, need to be reconsidered.”*

The science on endocrine disruption is relatively new. There will always be a need for a better understanding of the mechanisms involved and identification of new endpoints and toxic sources. But it will be many years before we fully understand the scope of the problem. The following is from the World Health Organization;

*“Research has clearly shown that EDCs can act at multiple sites via multiple mechanisms of action. Receptor-mediated mechanisms have received the most attention, but other mechanisms (e.g., hormone synthesis, transport, and metabolism) have been shown to be equally important. For most associations reported between exposure to EDCs and a variety of biologic outcomes, the mechanism(s) of action are poorly understood. This makes it difficult to distinguish between direct and indirect effects and primary versus secondary effects of exposure to EDCs. It also indicates that considerable caution is necessary in extrapolating from in vitro data to in vivo effects, in predicting effects from limited in vivo data, and in extrapolating from experimental data to the human situation. A collective weight of evidence is essential in determining under what conditions observed effects resulting from exposure to EDCs occur via endocrine mediated mechanisms.”*

*“Despite an overall lack of knowledge of mechanisms of action of EDCs, there are several examples where the mechanism of action is clearly related to direct perturbations of endocrine function and ultimately to adverse in vivo effects. These examples also illustrate the following important issues:*

*a) Exposure to EDCs during the period when “programming” of the endocrine system is in progress may result in a permanent change of function or sensitivity to stimulatory/inhibitory signals.*

*b) Exposure in adulthood may be compensated for by normal homeostatic mechanisms and may therefore not result in any significant or detectable effects.*

*c) Exposure to the same level of an endocrine signal during different life history stages or during different seasons may produce different effects.*

*d) Because of cross talk between different components of the endocrine systems, effects may occur unpredictably in endocrine target tissues other than the system predicted to be affected” (WHO 2002, Executive Summary). (A more thorough discussion of mechanisms of action can be found in Chaps 2 and 3 of WHO 2002).*

Though the original scope of inquiry related to endocrine disruption only considered effects produced through an estrogen receptor mechanism, this has now been expanded to include the blocking, synthesis, transport, metabolism and excretion of all hormones generated by all organs in the endocrine system. Recent research suggests chemicals that alter hormone production and metabolism may be more harmful and pose greater risk than those that bind hormone receptors (Sharpe 2004).

As research continues to expose the range of chemical-signaling systems vulnerable to disruption, it is becoming apparent that endocrine disruption is most likely but one example of a broader class of contamination effects, termed "signal disruption" (Fox et al, 2001; McLachlan JA, 2001). All biotic systems use some form of signaling in their reproduction, growth, or other life functions. These chemical signals are important at all levels of organization of life; within cells, among cells, between organs, even between organisms, including from one species to another. Any of these chemical signals, in principle, are vulnerable to disruption.

Scientists, for example, have just begun to look at the chemical signals that mediate communication between symbiotic organisms, such as nitrogen-fixing bacteria and the roots of the plants in which they live, and are examining how synthetic chemicals might interfere with these signals. It is through this system of communication that cells talk to each other and produce the results needed to keep a living organism functioning properly. Disrupting these 'signals of life' could have important and far reaching ecosystem impacts.

A simplistic analogy of all this is our information highway, the flow of data through microwave transmissions, the internet, satellites, or phone lines. We know all too well what can happen when these breakdown, when the flow of data is blocked. But what if instead of being blocked, communication was altered to send the wrong signal. What if all the lights turn green at the same time. What if the train is told to continue on the track it's on even though another train is coming from the other direction.

This is the danger that organisms face through signal disruption. The end result is potentially disastrous, biota thrown into chaos. It is with this understanding that risk assessors must view potential effects from EDs. It is not something trivial that can be cast aside with the wave of a few supporting documents. The core of any life form is supported through it's ability to communicate with it's parts, to form a whole. This is adversely affected through "signal disruptors", the full extent of which may not be known for many years to come, if ever. It has been only 15 years since endocrine disruption was first identified as a toxicological concern. In that time, though great progress has been made, it is painfully obvious to the scientific community that much more lies in the realm of the unknown than that which is safely tucked away as scientific fact.

Due to these, and other confounding factors, results from every research project and data review needs to be thoroughly analyzed, through peer review and independent analysis, unlike USDA 2003. There has never been a time when the need for critical, objective analysis has been more important than with the issues surrounding endocrine effects. This is especially true when one considers that new pathways of communication and functional overlap between the various endocrine systems are still being discovered (WHO 2002).

## **NPE Modes of Action**

It is well established that NPE and degradates are estrogen modulators, and suspected as acting as both a mimic and blocking agent. Recent research has also shown that it is an anti-androgen as well.

NPs effect on androgens, steroid hormones such as testosterone that mainly control male traits, is an area of intense interest. They bind to androgen receptors (AR) in a cell, move into the cell's nucleus, and combine with DNA to initiate genetic transcription that leads to androgens bodily effects.

Although weak androgenic NP activity was identified by Sohoni and Sumpter (1998), a more recent study using a yeast two-hybrid system revealed the antiandrogenic effects of NP (Lee et al. 2003). NP may thus have different effects according to the experimental conditions of each assay system. It is therefore also possible

that NP exerts a variety of activities under in vivo conditions (Negishi et al 2004).

These findings of antiandrogenic activity were further confirmed in See (2003). In this study, researchers used yeast systems and other laboratory assays to show that nonylphenol (NP) adversely affected the androgen receptor (AR) at many levels including blocking androgen binding, interfering with AR movement into a cell's nucleus, and choking genetic communication (See et al 2003).

In an earlier study, Baldwin demonstrated, after definitive analyses, that both 25 and 100 µg/L 4nonylphenol disrupted components of the testosterone metabolic pathway that would lead to a decrease in the metabolic elimination of testosterone and an increase in the accumulation of androgenic derivatives (Baldwin 1997).

Verslycke found that Mysids exposed to nonylphenol at 10 µg/L had a significantly higher metabolic androgenization ratio. This study indicates that energy and testosterone metabolism of mysids, as endpoints, are able to detect endocrine-disruptive activity of chemicals after short-term exposure to environmentally realistic levels of NP (Verslycke et al, 2004).

As these studies show, NP is an antiandrogen, with effects produced at dosing levels 1000 times (or more) lower than stated in USDA 2003.

Other research has also identified concerns with the synthesis, transport, metabolism and excretion of hormones (Teles et al, 2004, Kullman et al, 2004, Khan et al, 2003; Verslycke et al, 2004; Kleinow et al, 2004).

There have also been numerous studies that have shown endocrine effects without being able to identify the mode of action.

### **Toxic Effects and Endpoints Associated with ED and NPEs**

The toxic effects and endpoints from endocrine disruption of NPE degradates is a long list. It is also one that is growing longer as the scope of inquiry related to endocrine disruption is broadened to include non-estrogen mimic related effects. These now include androgen blocking, interference with thyroid hormones and progesterone, and many other endocrine related functions. As stated above, as research continues to mount about the range of chemical-signaling systems vulnerable to disruption, it is becoming apparent that endocrine disruption is most likely but one example of a broader class of contamination effects, termed "signal disruption."

Toxic effects from NPE degradates associated with endocrine disruption include reproductive and developmental toxicity, including embryotoxicity and teratogenicity, genotoxicity, immunotoxicity, neurotoxicity, and cytotoxicity. Research has also shown the potential for these degradates to be mutagenic and carcinogenic, via endocrine pathways, though linkage with these effects is not thoroughly defined.

### **Reproductive and Developmental Toxicity**

Given the central role that hormones play in guiding the development of the reproductive system and then in controlling its activities once developed, it is not surprising that a major focus of endocrine disruption research has been on reproductive health. There are many studies, especially experimental work with laboratory animals, that document endocrine disruption of the reproductive system. These include reductions in fertility, alterations in sexual behavior, deformations of the reproductive tract and reproductive diseases (reviews of research up to 1999 can be found in WHO, 2002; Environment Canada 2001; Servos, 1999; EU RA 2002).

There is a wide body of data showing developmental and reproductive effects, including embryotoxic effects associated with NP and NPE degradates exposure (Scott-Fordsmand & Krogh. 2004, Marcial et al, 2003, Kinnberg et al, 2000, Fan et al 2001, Zhang et al 2003a, Kwak et al, 2001, King et al, 2003, Kang et al 2003, Bevan et al 2001, Bevan et al 2003, Bettinetti & Provini 2002, WHO 2002).

### **Recent Research**

Kyselova tested the effect of p-nonylphenol on the body weight, reproductive organ weight and

histology, and in vivo fertility of the CD1 outbred mouse strain. The damage to the reproductive organs increased in the F1 generation, when NP influenced the animals during gestation, lactation and the pubertal period. In the group treated by the lower dose of NP, the prostate weight was decreased, and in the group treated by the higher dose, a lower body weight was found. Parental generation males treated with the lower dose of NP had normal spermatogenesis when compared with controls. Interestingly, researchers detected damage to the acrosome in spermatozoa already in the P generation. The acrosomal status (% of acrosome-intact cells) of spermatozoa decreased (compared to control) in the P generation by about 14% (50 µg NP) or 10% (500 µg NP). A marked decrease was observed in the F1 generation: 26% (50 µg NP) and 26% (500 µg NP). Both NP doses had a similar effect on acrosomal damage in the P and F1 generations. The data indicate that we did not find any dose-dependence effect. We can conclude that although spermatogenesis was established in the P generation, NP had an effect on sperm quality. In contrast to the quality, the number of spermatozoa was similar to the control group (in the P and F1 generations). Depending on duration of exposure, there were progressive degenerative changes in the reproductive organs (Kyselova et al, 2003).

Leblanc found a dose dependent increase in the proportion of developmentally compromised neonates, raising the possibility that 4-nonylphenol stimulated egg production without increasing some critical developmental component provided to the eggs by the maternal organisms, such as ecdysteroids, essential fatty acids, or triglycerides. As a result, more offspring were produced, but a significant percentage of the offspring were developmentally compromised, concluding that 4NP's mechanism of embryotoxicity is distinct from that associated with testosterone (LeBlanc et al 2000).

In Mackenzie 2003, gonadal differentiation was observed in leopard frogs (*Rana pipiens*) and wood frogs (*Rana sylvatica*) exposed as tadpoles to nonylphenol. Exposure at micrograms/L concentrations altered gonadal differentiation in some animals by inducing either complete feminization or an intersex condition, and altered testicular tubule morphology, increased germ cell maturation (vitellogenesis), and oocyte atresia. Comparisons between the two species indicate that *R. pipiens* are more susceptible to sex reversal and development of intersex gonads. However, *R. sylvatica* also showed alterations to testicular morphology, germ cell maturation, and oocyte atresia. (Mackenzie et al, 2003)

## **Immunotoxicity and Neurotoxicity**

Since the immune and nervous systems are intricately connected to the endocrine system, there has been a good deal of interest concerning immunotoxic and neurotoxic effects via endocrine pathways.

The greatest level of current research is focusing on endocrine mediated effects to the developing neural system. During the nine months between conception and birth, the fetal brain is transformed from instructions in genes to a complex, highly differentiated mass of organized cells capable of interacting with the outside world and prepared for learning.

Like virtually all development, the transformation is guided by natural chemical signals instructing cells to differentiate, form brain structures, forge links of immense complexity, and even to die (in a process that is thought to carefully prune unnecessary connections). Normal brain development is heavily influenced by a host of hormonal signaling systems. Thyroid hormones play a major role. The sex steroids (testosterone, estrogen, etc.) contribute to, among other things, sexual differentiation of brain centers, and thereby, to the development of sexual identity and sexual behaviors.

Dependent upon natural hormone signals, neural development is very sensitive to endocrine disruption. What is emerging from research is that brain and behavior are likely to be the most sensitive endpoints vulnerable to endocrine disruption. An important aspect of this research is the realization that small losses in intelligence might have large consequences for a society if they are experienced in a broad swath of the population (Colburn 2005).

NPEs and degradates have been shown to produce a wide range of effects to both the neural and immune systems (Negishi et al. 2003; Negishi et al, 2004; Bevan et al 2001; Iwata et al, 2004; Karrow ET AL, 2004; Scallet AC 2001; Scallet et al, 2001; Funabashi et al, 2004; Ohtani-Kaneko 2002; Masuo et al, 2004; Canesi et al, 2004).

## Cytotoxicity and Genotoxicity

Cytotoxic and genotoxic research of NPE degradates via an endocrine pathway is well documented. Recent research includes Bevan 2001, Bevan 2003, Balasubramanian et al, 2001, Aoki et al, 2004; Teles et al, 2004; Masuno et al, 2003; Kudo et al, 2004; Atienzar et al 2002; Ohtani-Kaneko 2002; Ferguson 2000; Sato 2002.

## NON-ENDOCRINE EFFECTS

Acute and chronic toxicity from non-endocrine pathways is just as pronounced as those effects from endocrine mediated pathways. Once again, most researched has focused on nonylphenol, though there is a growing trend of using other NPE degradates in testing.

### Acute and Chronic Toxicity

Once again, USDA 2003 is flawed in it's assessment of risk from non-endocrine disruption mediated toxicity, similar to failings found concerning EDCs. Scientifically unrealistic threshold levels are used to establish levels of risk. The following addresses those issues not covered in the endocrine disruption section. Though some of the toxic effects below could be manifested through an endocrine disruption pathway, at this time the mode of action is unknown. Other data used below was also incorporated in the endocrine section (as well as here) because both endocrine as well as non-endocrine effects were reported.

Acute and chronic non-endocrine effects have been shown to occur at similar dose levels as endocrine disrupting effects (Environment Canada 2001, Lussier 1999, Hecht 2002, Zhang 2003, EU RA 2002). There is a large body of data that shows LC50s to occur in the parts per billion range, and acute and chronic adverse toxic effects to occur in the low parts per billion range for NPE degradates, with recent research showing effects produced in the low parts per trillion range. As with endocrine effects, a review of research up to 1999 can be found in WHO, 2002; Environment Canada 2001; Servos, 1999; EU RA 2002. The following brief summaries of findings are from Environment Canada's CEPA toxic substances assessment for NPE and metabolites and the European Unions Nonylphenol Risk Assessment.

*“There are a large number of studies reporting acute and chronic effects of NP in aquatic biota. There are, however, fewer studies reporting the toxicity of NPEs, and only a few studies that included the PNECs. Although studies described in the literature have used many species, different test methods and different chemicals, there is a consistent pattern in the toxicity reported. The range of acute toxicity for NP is similar for different organisms: for example, fish (17–1400  $\Phi$ g/L), invertebrates (20–3000  $\Phi$ g/L) and algae (27–2500  $\Phi$ g/L). Chronic toxicity values (No-Observed-Effect Concentrations, or NOECs) for NP are as low as 6  $\Phi$ g/L in fish and 3.9  $\Phi$ g/L in invertebrates. An acute to chronic toxicity ratio of 4:1 was determined based on the available literature.”* (Environment Canada 2001)

The EU RA 2002 has found similar results, though slightly more toxic.

*The PNEC (water) is calculated using the assessment factors detailed in the TGD. For nonylphenol short-term and long-term data are available for both freshwater and seawater species for three trophic levels.*

*Short-term studies are available for fish, aquatic invertebrates and algae. The most sensitive species appears to be the freshwater invertebrate *Hyaella azteca* with a 96-hour EC of 0.0207 mg/l. Long-term studies are also reported for fish, aquatic invertebrates and algae. The most sensitive species in long-term studies appears to be the freshwater algae *Scenedesmus subspicatus* with a 72-hour EC of 3.3  $\Phi$ g/l. As long-term NOECs from at least three species representing three trophic levels are available an assessment factor of 10 may be used. Applying this to the long-term NOEC for algae gives a PNEC (water) of 0.33  $\Phi$ g/l.*

*For nonylphenol a mesocosm study is available which studied the effects on species from several trophic levels. Generally the effect levels determined in the study for various organisms agree reasonably well with the laboratory data. However, there are several aspects of the experiment design that suggest that the system used, while suitable for detecting gross changes in populations, is not sufficiently sensitive to detect small changes in populations that could become significant with continued exposure. The field study is therefore taken as supporting data in generating the PNEC, but cannot be used as the basis for deriving a PNEC to protect the aquatic compartment.*

*The PNEC (water) is calculated using all the aquatic toxicity data present on nonylphenol. Data exist indicating toxicity at lower concentrations than the concentrations at which oestrogenic effects are observed. Therefore, the calculated PNEC (water) should be protective for oestrogenic effects in fish as well (EU RA 2002).*

As with the risk assessment for endocrine effects in the USDA 2003, the NOAELs incorporated into the risk assessment for acute and chronic non-endocrine toxicity are also unrealistic, do not reflect current scientific understanding of expected effects and misrepresent potential risk from NPE based surfactants. If the USDA 2003 were to incorporate the data used to establish the PNEC in EU RA 2002 into its risk assessment, the MOS would be 33 parts per trillion (ppt) for chronic toxicity and 170 to 200 ppt for acute toxicity, which would be only slightly higher than those the risk assessors should use for endocrine related effects from NPE based surfactants, (though for endocrine disruption there may be no threshold and therefore no way to establish potential risk).

### **Carcinogenic or Mutagenic Effects**

Though not listed as carcinogens, NPE and degradates have shown the potential to cause mutations and deformities and are suspected of producing cancer effects through both endocrine and non-endocrine mediated pathways.

Seiki et al found that the total incidences of adenomas and carcinomas in the lungs of animals treated with nonylphenol and genistein were significantly higher than in the control group. 5-Bromo-2'-deoxyuridine labeling indices, reflecting cell proliferation, were also significantly elevated in the lungs of rats given 250 and 25 ppm nonylphenol.....These results indicate that nonylphenol and genistein have the potential to promote rat lung carcinogenesis, possibly via a mechanism involving stimulation of cell proliferation and DNA damage caused by oxygen radicals. (Seiki 2003).

Other effects reported are DNA mutations (Atienzar 2002), embryo lethality (arrested egg development) and deformities (curved or unextended shell spines and undeveloped second antennae) (Zhang 2003), cell proliferation and spindle disturbances (Zumbado 2002), proliferation of T47D cell and the metaphase of cell division (Yu 2003), increased adenocarcinoma and total mammary tumor multiplicity (Fukamachi et al, 2004), increased both tumor incidence and latency (Villanueva et al, 2004).

Other research includes Vivacqua et al, 2003 and Wu F, Safe S, 2004.

### **Dose Response Curves and Low Dose Effects**

The key to risk assessment is that the dose makes the poison. However, in the case of NPE degradates and other EDs, finding the threshold dose has proven to be a challenge. In fact, some substances have no threshold level currently established. The reasons for this are generally accepted to be as;

A) Initially, dosing levels were kept high because no effects were identified. Then scientists realized that EDs often show effects only at extremely low doses, and therefore dosing regimes needed to be lowered. This response curve is known as a non-monotonic dose response curve, and is now well documented and accepted as scientific fact. The presence of non-monotonic dose response curves in endocrine disruption also means that many earlier toxicological tests may have led to erroneous conclusions about safety.

B) Once the dosing regimes were lowered, effects were often seen to occur at all levels tested, therefore

no NOEL or NOEC can be established. This can be attributed, in some cases, to the fact that EDs are often times acting as hormone mimics, and are therefore impacting a system that is already at the threshold of effects, i.e. hormones are already present and producing effects.

C) The science of endocrine disruption is new, and the endocrine system and related functions are so complex, that it will probably take many years of detailed research to form a clear picture of all the different types of effects, modes of action and different endpoints involved.

D) Timing of exposure can be a more important factor than rate and duration.

### **Nonmonotonic Dose Response Curve**

A significant factor in analyzing research findings and associating these findings to appropriate risk assessment protocol, is the fact that nonylphenol does not follow the typical linear dose response curve currently used by risk assessors. As stated, the non-monotonic dose response curve often seen in nonylphenol endocrine mediated effects forces the re-evaluation of many older high dose studies that found no effects generated. It also questions the validity of current risk assessment protocol which adheres to the belief that the greater the dose, the greater the effects, and below which exists a threshold of no effects. With NPE degradates and other EDs, even when there appears to be a threshold, until lower doses are tested, there is no certainty that a threshold has been met.

That NPE degradates exhibit non monotonic dose responses with endocrine mediated effects is now accepted as scientific fact (WHO, 2002; Environment Canada 2001; EU RA 2002).

*“A key outcome of the (NTP Low Dose Peer Review) was verification that some endocrine disruptors exhibit dose-response relationships described as nonmonotonic, meaning that within a certain dose range, a chemical's effects on a given end point actually become greater as the dose is reduced. The dose-response curves can be shaped like a U, with a high response at both low and high levels of exposure, or like an inverted U, with the greatest response at intermediate dose levels. According to Frederick vom Saal, a professor in the Division of Biological Sciences at the University of Missouri in Columbia, nonmonotonic curves challenge the EPA's standard assumption of linear or threshold dose responses, which holds that toxic effects always lessen as the dose is reduced toward zero” (NIEHS 2001).*

The following is from the Commission on Life Sciences, 2000; “Hormonally Active Agents in the Environment”;

*“Knowing the shape of the dose-response curve for environmental contaminants is critical for understanding how such contaminants...act on organs and organisms. Understanding the dose-response relationship is also critical for the design of studies to test the effects of contaminants.*

*If an underlying monotonic dose-response function (i.e., a function where response increases as dose increases or at least does not decrease) and a dose below which there is no effect (a threshold dose) are assumed when designing a toxicologic study, there is a risk of failing to understand or properly test a contaminant that does not display a monotonic dose-response function or a threshold dose.*

*It is well known that some compounds produce nonlinear and even nonmonotonic dose-response functions in some organisms over certain ranges of dose. Furthermore, some compounds can produce different dose-response functions depending on the target organ and the species exposed” (CLS 2000 p82).*

*“There are numerous examples of nonmonotonic inverted U-shaped dose-response curves from in vitro studies. These studies involve a variety of natural and anthropogenic estrogens (e.g., estradiol, estriol, nonylphenol, and DES), end points (e.g., cell proliferation, prolactin synthesis, and induction of specific mRNAs), and cell lines (e.g., Jordan et al. 1985; Soto et al. 1991; Bigazzi et al. 1992; Pilat et al. 1993; Truss and Beato 1993; Tzukerman et al. 1994; Olea et al. 1996). Sonnenschein et al. (1989) also observed a nonmonotonic response curve for androgen-induced cell proliferation in LNCAP cells by using a diverse group of steroidal and nonsteroidal compounds”. (CLS 2000 p110)*

The reasons for the nonmonotonic response curve findings are poorly understood at present. One recognized theory is expressed by Fred vom Saal, the first to document this response in association with EDCs;

*"Any endocrinologist will tell you that hormone receptors are up-regulated [stimulated] at low doses and down-regulated at high doses," he says. "In fact, in clinical therapy you can shut down a hormonal system simply by treating with high levels of hormone" (NIEHS 2001).*

Recent research highlighting nonmonotonic dose response includes the following, Duft et al 2003, Hense et al, 2003, Jobling et al 2004, Negishi et al, 2004; Ohtani-Kaneko 2002.

In the study cited previously by Bulayeva and Watson, the dose response was an inverted U shape. For NP this showed as effects generated in the low parts per billion range, no effects produced in the middle dosing regime, and then effects again at the low parts per trillion dosing range.

*"The reason for this gap in dose responsiveness at intermediate concentrations is still not understood, but it is interesting that other estrogens in the present study demonstrate the same phenomenon. These very low effective doses for xenoestrogens demonstrate that many environmental contamination levels previously thought to be subtoxic may very well exert significant signal and endocrine-disruptive effects, discernable only when the appropriate mechanism is assayed. Possible reasons for these potent effects not being noted previously are that little testing of the nongenomic pathway has been done, many tests did not examine such low concentrations, and some test conditions probably did not adequately remove endogenous estrogen levels (as we have done by use of low quantities of extensively charcoalstripped serum) to reveal effects of these low concentrations. The potent effects we see on nongenomic signaling mechanisms could explain why concentrations previously determined to be inactive via genomic mechanisms still have toxic and teratogenic effects on wildlife (Brucker-Davis et al. 2001). Therefore, the threat levels of these compounds to wildlife, and probably humans, need to be reconsidered."* (Bulayeva and Watson, 2004)

## **Low Dose Effects**

Currently, most research is using dosing regimes in the ppb (parts per billion) range, though some research is now beginning to show that both nonylphenols and octylphenols produce effects in the ppt (parts per trillion) range (Nice 2003, Fent, 2000, Christian and Gillies 1999, Ohtani-Kaneko 2002, Dreze V 2000, Bulayeva 2004, Kwack et al. 2002, Hahn et al. 2002, Uguz 2003, Hemmer MJ, et al. 2002, Ackermann et al. 2002, Pickford KA, et al. 2003, Huang RK, Wang CH, 2001, Czech et al. 2001, Burkhardt-Holm, 2000, Yokota 2001, Matozo 2003, Tanaka 2001, Tanaka 2002, Zhang 2001, Zhang 2003, Weber 2003, Hill 2003, Chitra 2002, Schwaiger 2002, Hecht 2002, Servos 1999, Seki 2003, Meregalli, 2001, Bevan 2003, Negishi 2004).

This is a very important fact that has been ignored in current risk assessment protocols used by the Forest Service. As will be analyzed in detail below, USDA 2003 does not use low dose research from NP1EO, NP2EO or NP, justifying this with the assumption that the less toxic carboxylate derivatives are all that would be experienced in an open environment. This is a false and very dangerous assumption. It is also the main reason why any documentation used for establishing risk from NPE degradates needs to go through a thorough independent peer review.

Once again, reviews of research up to 1999 can be found in WHO, 2002; Environment Canada 2001; Servos, 1999; EU RA 2002. Of special importance to the risk assessment analysis is the EU RA 2002, where the NOEL is 3.3 ppb and the PNEC (Predicted No Effects Concentration [EU risk assessment protocol]) for aquatic species is 0.33 ppb (330 ppt). The PNEC differs from the safety margins used to establish hazard quotients in USDA. The equivalent MOS would be 33 ppt. However, the findings of Bulayeva 2004 would lower the MOS even further, approaching parts per quadrillion range.

In Bulayeva and Watson, 2004, the authors of this study found that xenoestrogens produced time dependent endocrine effects, within 30 minutes of exposure, through pathways that had never been explored

before. By analyzing effects to extracellular-regulated kinases (ERKs) in the pituitary tumor cell line, they found that nonylphenols produced effects in the parts per trillion dose range, at the same potency of E2. For sometime, it was assumed that xenoestrogens like NPEs were 1,000 to 10,000 times less potent than E2. However, Bulayeva 2004, and other recent studies have shown that NPE degradates are as potent as E2, and have also shown that they produce negative effects through pathways that E2 does not cause effect through. These new studies have opened up new areas of research for NPEs and endocrine effects. The following is from Bulayeva 2004;

*“An important and surprising conclusion from our studies was that all tested estrogenic compounds, except bisphenol A, elicited rapid membrane-initiated actions at very low concentrations compared with their reported potencies in classical genomic pathways (Gutendorf and Westendorf 2001; Hodges et al. 2000; Inoue et al. 2002). All active compounds were able to produce rapid (3–30 min) ERK phosphorylations in the nanomolar concentration range, and some (E2, coumestrol, nonylphenol, and endosulfan) were also active in the subpicomolar range.”* (Bulayeva and Watson, 2004)

### **Timing of Exposure**

Another important aspect to consider when addressing documentation for risk assessment is the fact that NPE degradates and other EDs have shown a pronounced tendency to exert influence on the endocrine system at different times in the life cycle of all living things. It is imperative that this be incorporated into any BLM risk assessment.

The importance of understanding timing as a risk assessment parameter is that it once again dispels the risk assessment methodology incorporated in USDA 2003. The concept of acceptable dose levels (those below the threshold NOAEL x 100) are only appropriate if a) the most sensitive time for exposure is the tested exposure period and b) these studies are long term chronic or multi-generational studies to identify “later in life” or trans-generational effects. Since nonylphenol has shown itself to produce effects in the very low ppb range during the developmental stage of most organisms that have been tested, this would place the risk quotient multiplier in the ppt, which in turn would place all species at serious risk from exposure.

The following are quotes from government agencies that describe the importance of acknowledging timing of exposure as a risk factor for acute, chronic and multi-generational effects.

*“Exposure to EDCs during the period when “programming” of the endocrine system is in progress may result in a permanent change of function or sensitivity to stimulatory/inhibitory signals”* (WHO 2002).

*“Normal endocrine function is often dependent on cyclical events, rather than steady-state. Timing is everything, as evidenced by significant differences in adverse outcome as a function of age and stage of development”* (USEPA).

*“Experts suggest that endocrine disruptors pose the greatest risk during fetal development, which is regulated by hormones at specific levels. Hormonal alterations due to maternal exposure in pregnancy could lead to effects such as reduced cognitive function or cancer that might not be evident for months, even years”* (NIEHS 2001).

Other research with similar findings include Sone et al 2004, Burkhardt-Holm, 2000, vom Saal 1993, Thibaut et al, 2002, Lee 1998, Colborn et al, 1995, Colborn et al 1993.

Of special note in recent studies exploring the role of timing are the findings of Nice et al. 2003. The authors provide evidence clearly demonstrating that when larvae are exposed to environmentally relevant concentrations of nonylphenol for a single 48 hour exposure at a key stage in their development, long-term sexual developmental effects are induced. Data provided by this study suggest that exposure to 1 ppb and 100 ppb nonylphenol at days 7 to 8 post-fertilization results in a change in the sex ratio towards females and an

increase in the incidence of hermaphroditism (10 mo later, up to 30% of the resulting adults were fully functional hermaphrodites). Gamete viability is also affected, resulting in poor embryonic and larval development (up to 100% mortality) of the subsequent generation (Nice et al 2003). This study is important because it is one of the first to identify serious adverse effects from a single “pulse” exposure of extreme low doses with no NOAEL identified.

The USDA 2003 puts much weight in the concept that quick degradation of NPE will limit effects, itself a flawed assumption. However, when one low dose of a substance can produce serious long term effects to both individuals and to populations, it matters not how long something persists, or which degradate is potentially going to be the most common in the environment.

## No Threshold

When risk assessors look at potential effects from different dose levels of a toxic substance, they are assuming that the system these chemicals might impact is not carrying a body burden of this substance. If this substance (or other substances that share a common mechanism) is already present in the system, then that is taken into account in an additive fashion. With EDs however, the equation is completely different. Hormone active substances (that is, hormones themselves) are already present in quantities sufficient to cause effect.

In WHO 2002 it was defined as;

*“The issue of dose–response relationships is perhaps the most controversial issue regarding EDCs. One of the reasons is that EDCs often act by mimicking or antagonizing the actions of naturally occurring hormones. These hormones (often more potent than exogenous EDCs) are present at physiologically functional concentrations, so the dose–response considerations for EDCs are often different than for other environmental chemicals, which are not acting directly on the endocrine system” (WHO 2002).*

These principles were first described in Sheehan 1999.

*“Risk assessments for nongenotoxic chemicals assume a threshold below which no adverse outcomes are seen. However, when an endogenous chemical, such as 17 $\beta$ -estradiol (E2), occurs at a concentration sufficient to cause an effect, the threshold is already exceeded. Under these circumstances, exogenous estradiol is not expected to provide a threshold dose”.*

*“There was no apparent threshold dose for E2. A smaller replication confirmed these results. These results provide a simple biologically based dose-response model and suggest that chemicals which act mechanistically like E2 may also show no threshold dose. If so, even low environmental concentrations of such chemicals may carry risk for sex reversal” (Sheehan et al 1999).*

Sheehan et al. worked experimentally with sex control in the red-eared slider, a turtle in which sex determination is normally controlled by temperature (via a mechanism in which the hormonal processes involved in sex determination are temperature dependent). They exposed a series of turtle eggs at 28.6E°C to a range of doses of 17 $\beta$ -estradiol. The temperature they chose normally would have resulted in mostly males but some females. They then determined the sex of each egg at hatching. They analyzed the results using a theoretical construct based on the Michaelis-Menten equation, which has been developed in basic chemistry to model enzyme kinetic studies. The data from the large experiment fit the M-M model exceptionally well. The combination of both experimentation and theoretical analysis is very powerful. Their analyses showed that any addition of exogenous estrogen caused a change in the sex ratio of pool of eggs and "that no exogenous estrogen is without risk." This is because in their experimental system, endogenous estrogen is already at a high enough level to exceed the threshold for causing an effect. Endogenous estrogen is already activating the system. A contaminant doesn't have to exceed the threshold because endogenous estrogen already does.

This is an important concept to understand as a risk assessor. Organisms contain substances that put

them already past the point of producing effects. The difference from EDs is that the natural hormones are sending the right messages, in the right order, and of the right magnitude to put the proper message into effect. Then they disappear so new messages can be brought forth. An EDC acts on a part of that message stream. It changes the message in a way that makes no sense. Whether or not that message will produce or add to an adverse reaction is dependent on many factors. The fact remains however, that these marauding hormone mimics are causing adverse effects at extreme low doses, often times at all levels tested, with no NOAEL being defined, because they are entering an active system.

## **Dose Response Summary**

In essence, the “dose-response and threshold” assumptions are the core of any risk assessment. Its use in regulatory science has been a pragmatic step, not something based on theory or on fact. This assumption is a key part of the way that safety standards are set. All risk assessment must first start by identifying a threshold for effects, or a "no observed adverse effect level" or NOAEL. Then the NOAEL is divided, often by 100. The assumption is that an exposure level calculated in this fashion is safe, and it is used to determine acceptable per day exposure levels.

These fundamental assumptions used to guide current risk assessment are no longer applicable when assessing EDs. Since the issues surrounding dose response to environmental EDs are pivotal to exposure risk assessment and consequently to regulatory considerations, numerous research projects are attempting to come to grips with this need for a new risk assessment model. A review of the state of the science of these concerns was recently published in *Environmental Health Perspectives*. Welshons et al. review the issues associated with the underestimation of true bioactivity when only high doses are used in toxicologic studies. The major points considered include low-dose biological activity not observed by traditional testing, nonlinear dose extrapolation, complex receptor responses, and the effects of exogenous exposure on an already active biological pathway. This was their conclusion;

*“Information concerning the fundamental mechanisms of action of both natural and environmental hormones, combined with information concerning endogenous hormone concentrations, reveals how endocrine-disrupting chemicals with estrogenic activity (EEDCs) can be active at concentrations far below those currently being tested in toxicological studies. Using only very high doses in toxicological studies of EEDCs thus can dramatically underestimate bioactivity. Specifically: a) The hormonal action mechanisms and the physiology of delivery of EEDCs predict with accuracy the low-dose ranges of biological activity, which have been missed by traditional toxicological testing. b) Toxicology assumes that it is valid to extrapolate linearly from high doses over a very wide dose range to predict responses at doses within the physiological range of receptor occupancy for an EEDC; however, because receptor-mediated responses saturate, this assumption is invalid. c) Furthermore, receptor-mediated responses can first increase and then decrease as dose increases, contradicting the assumption that dose-response relationships are monotonic. d) Exogenous estrogens modulate a system that is physiologically active and thus is already above threshold, contradicting the traditional toxicological assumption of thresholds for endocrine responses to EEDCs. These four fundamental issues are problematic for risk assessment methods used by regulatory agencies, because they challenge the traditional use of extrapolation from high-dose testing to predict responses at the much lower environmentally relevant doses. These doses are within the range of current exposures to numerous chemicals in wildlife and humans. These problems are exacerbated by the fact that the type of positive and negative controls appropriate to the study of endocrine responses are not part of traditional toxicological testing and are frequently omitted, or when present, have been misinterpreted” (Welshons et al, 2003).*

## **USDA 2003**

Currently, USDA 2003 uses the acute toxicity values for NP9E for establishing risk of acute toxicity, and chronic toxicity values for NPnECs for establishing risk of chronic toxicity. In essence, USDA 2003 has

chosen to turn a blind eye on the more serious effects caused by NPEO1, NPEO2, and NP, common degradates of NPE9. This is an inappropriate use of toxicity data for risk assessment purposes. (Appendix A contains a more thorough critique of why this is inappropriate).

It is basic sound science to incorporate the full mixture of NPE degradates as a single concern. The endpoints and mechanisms of action for toxic effects are basically the same for all the primary intermediaries. It is also basic sound science to use the acute, chronic and endocrine toxicity values for NP(1-2)EO, and where these are not available, NP, as the references for establishing risk.

## **The BLM PEIS and Endocrine Disruption**

There is no discussion of endocrine disruption in the PEIS. The only reference to ED effects that could be found were in the FS ERAs. Concerning ED and glyphosate, the FS supporting document, SERA 2003b, is one of the few places within the extended body of the PEIS where one can find a discussion of endocrine disruption. Unfortunately, it is a very limited and poorly written analysis. This is its entirety;

*3.1.8. Effects on Endocrine Function. In terms of functional effects that have important public health implications, effects on endocrine function would be expressed as diminished or abnormal reproductive performance. This issue is addressed specifically in the following section (Section 3.1.9). This section is limited to direct and largely mechanistic assays that can be used to assess potential direct action on the endocrine system.*

*Only three specific tests on the potential effects of glyphosate on the endocrine system have been conducted and all of these tests reported no effects. Glyphosate was inactive as an estrogen receptor agonist (estrogenic activity) in MCF-7 human breast cancer cells (Lin and Garry, 2000) as well as in yeast and trout hepatocyte assays (Petit et al., 1997). In a third assay, glyphosate did not inhibit steroid synthesis in MA-10 mouse Leydig tumor cells by disrupting expression of the steroidogenic acute regulatory (StAR) protein (Walsh et al., 2000). This protein mediates the rate-limiting step in the mitochondrial synthesis of steroid hormones (the transfer of cholesterol to the inner mitochondrial membrane). In the Walsh et al. (2000) study, however, Roundup did inhibit steroid synthesis, probably due to the effects of the surfactant on membrane function. All of these assays are in vitro – i.e., not conducted in whole animals. Thus, such studies are used qualitatively in the hazard identification to assess whether there is a plausible biologic mechanism for asserting that endocrine disruption is plausible. Because they are in vitro assays, measures of dose and quantitative use of the information in dose/response assessment is not appropriate. For glyphosate, these studies do not indicate a basis for suggesting that glyphosate is an endocrine disruptor.*

This brief endocrine analysis raises many questions. The first part concerning “*important public health implications*”, is not understood. Endocrine disruptors affect the endocrine system. The endocrine system is responsible for far more than just reproductive performance. It is the key communication and control link between the nervous system and bodily functions such as reproduction, immunity, metabolism, and behavior. The nervous system works in tandem with the endocrine system to control all bodily functions and processes. Endpoints associated with endocrine effects or endocrine disruption is almost limitless. This is from Weiss 1998;

*"Agents that alter the functional properties of endocrine systems pervade the environment....The primary distinction between cancer and endocrine disruptors and neurotoxicants is the plethora of possible endpoints by which toxicity can be expressed. Cancer is a unitary index. Adverse consequences flowing from exposure to endocrine disruptors can take an almost infinite variety of forms, including neurobehavioral outcomes. In their most troubling manifestations, these emerge as disorders of early development. They can range from deviant patterns of male copulatory behavior to impaired cognitive function. Each of these indices, in turn, exhibits multiple dimensions.*

*Moreover, some aftermaths, as with cancer, might emerge only after long latencies. Different stages of the life cycle following developmental exposure will manifest different outcomes as a consequence. Some adverse effects may arise for the first time in advanced age because it is a period of declining compensatory margins. These multiple facets of neurobehavioral toxicity, and, by extension, their coupling to endocrine disruptors, imply a risk assessment process that corresponds, in many ways, to the global views adopted by ecotoxicologists" (Weiss 1998).*

Why SERA attempts to limit the scope of concern from endocrine effects to “*diminished or abnormal reproductive performance*” is unclear. As Weiss explains, adverse consequences can “take an almost infinite variety of forms”. Also, since ED research is an evolving and new science, it is important to re-analyze known effects and endpoints whose cause is unknown, but may, or could, have been mediated via endocrine disruption.

It is true that the body of data relating to glyphosate and endocrine effects, without additives included in the test, is not large. Even still, this SERA analysis is nothing more than a cursory glance at endocrine effects, and does not take into account a wide range of effects that could be mediated via endocrine pathways. Nor does it take into account the findings of Marc 2002, of a synergistic relationship between additives and glyphosate that could facilitate potential endocrine effects. It does look at Yousef et al. 1995, but never attempts to relate the findings in Yousef to effects produced through endocrine disruption, while stating;

*“The mechanism of the this effect is not clear but it may be related to the ability of glyphosate to inhibit oxidative phosphorylation” (SERA 2003b p 3-15).*

This limited ED analysis of glyphosate performed by SERA is, for most extent and purposes, the entire body of analysis relating to endocrine disruption in the whole of the PEIS, PER and supporting documents. Though some of the SERA ERA’s (supporting documents found on the FS website) contain this brief analysis of ED, it is interesting to note that the SERA ERA for 2,4-D, the most well known ED among the herbicides that are relying on FS ERAs, has no discussion whatsoever of endocrine disruption. A further discussion of endocrine disruption will be provided below in a later section.

## **CONCLUSION**

BLM has proposed one of the largest herbicide projects in history, not only in acreage, but also for the number of active ingredients, inerts and adjuvants at their disposal. Unfortunately, the level of analysis performed is a throwback to the late 1980's. The excuses used for foregoing analysis are;

- a) Research since 1988 has shown no need to re-analyze 11 of the herbicides in question. As such, it is appropriate to piggyback on analysis provided by the FS.
- b) There isn't enough data available to do proper analysis of inerts, adjuvants and degradates, and besides, they're probably harmless anyway.
- c) The issues are too complex.

We have demonstrated, with overwhelming support from scientific data, that;

- a) Research since 1988 shows a pressing need for re-analysis of all AIs. The example given is 2,4-D, shown to be an endocrine disruptor, and recently listed as a Proposition 65 reproductive toxicant.
- b) For many of the inerts, adjuvants and degradates that are known, there is a wealth of information available, for both acute and chronic effects. In some cases, these additives can be 1000 to 10,000 times more toxic than the AI. The examples given, inerts in glyphosate products and NPE adjuvants and degradates, have had countless studies performed that are readily available. Endocrine disruption, a family of health effects that was not known in 1988, is now being extensively researched. There are numerous ED's, both known and suspected, among the additives.

c) Yes, the issues are very complex. If BLM is not up to the task, then it would be best to abandon the herbicide component of this program, until such time that BLM can effectively carry out it's mandate. When that time comes, all ingredients (and their degradates) proposed and approved for use in this project, will need thorough analysis, updated profiles, and with RA's for any substance that shows a clear threat to human health or the environment. Cumulative effects analysis is important with or without GLEAMS modeling. If certain aspects of risk assessment modeling cannot be applied, NEPA still demands the following;

*(3) a summary of existing credible scientific evidence which is relevant to evaluating the reasonably foreseeable significant adverse impacts on the human environment." (40 CFR 1502.22)*

We have attempted to show you how to perform (3) above, by highlighting direct effects from 2,4-D, glyphosate formulations, and NPE surfactants. Indirect and cumulative effects for these components, and analysis of direct, indirect and cumulative effects for all other compounds, we leave up to you. And remember SERA's advice;

*"Neurotoxicity, immunotoxicity, and endocrine disruption are three classes of effects that are important in any risk assessment" (SERA 2002)*

## 2. Public Health Risks

### Chemically sensitive people

Concern is especially magnified for sensitive populations including those with compromised immune systems, children and the elderly. Dr. Robert Kreutzer of the California Department of Health Services reported in the results of the Department's annual survey of California health indicators that 16.9%, or five million Californians, are sensitive to chemicals and that 6.4%, or close to two million Californians, have been medically diagnosed as having chemical sensitivity that affects their health status. (Kreutzer, Neutra, Lashuay. 1999. Prevalence of People Reporting Sensitivities to Chemicals in a Population-based Survey. American Journal of Epidemiology) In a personal communication Dr. Kreutzer assured us that the findings of his 1999 report corresponded with other studies of chemical sensitivity in the general population and field experiences of his office in chemical release accidents.

### Impacts to pregnant women, fetuses and infants

(Much of the following is from Having Faith: An Ecologist's Journey To Motherhood by Sandra Steingraber).

Pesticides with low molecular weights cross the placenta without restriction. Even heavier pesticide molecules pass through, but sometimes they are partly metabolized by the placenta's enzymes before they pass through, which can make them *more* toxic than they were to begin with. Pesticides do not have to pass through the placenta to cause harm. Some chemicals will lodge in the placenta and do damage there. (U.S. Department of Health and Human Services. Agency for Toxic Substances and Disease Registry. 2002).

The brain of a fetus and of a child under six months is more vulnerable to toxins because it lacks a blood-brain barrier, which in older children and adults prevents many blood-borne toxins from entering the brain's gray matter. Fetal brains are even more vulnerable because of the lack of fat in the fetal body. Thus, the fetal brain attracts most of the fat-soluble toxic chemicals resulting in a disproportionately greater effect on the brain. (ibid)

There has only been one study of environmental contaminants in amniotic fluid ever conducted, and it found detectable levels of organochlorine pesticides in one third of the thirty samples of amniotic fluid tested.

Research conducted using information from the California Birth Defects Monitoring Program has found elevated risks of particular kinds of birth defects among women using pesticides for gardening and for those living within a quarter mile of agricultural crops. Of 2000 mothers of children with birth defects, who were interviewed, 75 percent of them had at least one source of exposure to pesticides while pregnant.

Pesticide applicators have a higher risk of having children with birth defects or fetuses with anencephaly. In a study from the Netherlands found an increased risk of spina bifida when fathers are exposed to certain chemicals, including pesticides. Some chemicals are known to injure the DNA strands carried in the heads of sperm cells, while others affect the testicles ability to produce sperm.

In Finland, a birth defect registry shows that children born to women employed during their first trimester of pregnancy in agricultural occupations involving pesticides had twice the risk of cleft lips and palates. In Spain, the rate of oral clefts in similar children was three times that of other children. Spain also shows that undescended testicles were more common in areas of high pesticide use. These findings were mirrored in Denmark. Norwegian researchers found strong associations between spina bifida and hydrocephaly and pregnant workers in orchards or greenhouses. In the U.S., a study of 700 women in California showed an

increased risk of fetal death from birth defects among babies whose mothers lived near agricultural crops where certain pesticides were sprayed. Women in their first trimester who live in a square mile of pesticide use are most vulnerable to impacts.

One of the most thorough studies linking pesticides to birth defects was conducted in Minnesota by Dr. Vincent Garry at the University of Minnesota medical school. He found elevated levels of birth defects among children of registered pesticide applicators (like farmers) in western Minnesota. He also found a clear geographical pattern of birth defects among the general population. Non-farming families living in the western half of the state were 85 percent more likely to have a baby with birth defects than nonfarming families living in the eastern half because of the increased use of pesticides in the agricultural portion of the state. Further, in western Minnesota, children conceived in the spring when pesticide use is at its highest were significantly more likely to have birth defects than those conceived at other times of the year. No seasonal pattern for birth defects was present in the eastern part of the state.

In 1997, the U.S. Toxics Release Inventory included 47 different chemicals classified as known or suspected fetal toxicants, which amounted to 989,700,000 pounds of these chemicals released in that one year.

Of particular interest in regards to the current analysis, there appears to be a direct relationship between birth-defect rates and the application volumes of chlorophenoxy herbicides, according to an EPA researcher.

Dina Schreinemachers, a statistician with the agency's National Health and Environmental Effects Research Laboratory based her conclusions on birth records from nearly 150 wheat-producing counties in four Northern Tier states.

In the published report, Schreinemachers says her findings should be viewed with caution because, among other reasons, wheat production acreage was used as a surrogate for actual exposures to 2,4-dichlorophenoxyacetic acid (2,4-D) and 4-chloro-2-methylphenoxyacetic acid (MCPA).

However, she also notes that her findings are similar to those from a number of previous studies. Consequently, she says, her results "are especially of concern because of [the] widespread use of chlorophenoxy herbicides."

Richard et al found (Differential effects of glyphosate and Roundup on human placental cells and aromatase. Environmental Health Perspectives. Vol 113, Number 6, June 2005) that some agricultural workers using glyphosate have pregnancy problems, but its mechanism of action in mammals is questioned. They show that glyphosate is toxic to human placental JEG3 cells within 18 hr with concentrations lower than the agricultural use, and this effect increases with concentration and time, or in the presence of Roundup adjuvants. Surprisingly, Roundup is always more toxic than its active ingredient. The herbicide acts as an endocrine disruptor on aromatase activity and mRNA levels, and glyphosate interacts within the active site of the purified enzyme, but its effect is facilitated by Roundup formulation in microsomes or in cell culture. We conclude that endocrine and toxic effects of Roundup and not only glyphosate can be observed in mammals, suggesting that the presence of Roundup adjuvants enhances glyphosate bioavailability and / or bioaccumulation.

Further, Farr et al found (Pesticide Use and Menstrual Cycle Characteristics among Premenopausal Women in the Agricultural Health Study. Am J Epidemiol 2004;160:1194-1204) that women who used pesticides experienced longer menstrual cycles and increased odds of missed periods compared with women who never used pesticides. Women who used probable hormonally active pesticides had a 60-100% increased odds of experiencing long cycles, missed periods, and intermenstrual bleeding compared with women who had never used pesticides. These abnormalities are linked to increased risk to reproductive success.

The precautionary principle should be invoked by a federal agency and if in ignorance, it should abstain. To the extent the agency chooses to proceed, it has a duty to engage in as thorough a study as possible on the impacts to pregnant women, fetuses and infants in order to disclose the true cumulative effects of pesticide use, including this program, to the public.

## **SCIENTIFIC HONESTY AND RATIONALITY**

The agency must use high quality information and accurate scientific analysis, 40 C.F.R. 1500.1(b), and must disclose "any responsible opposing view." Id. 1502.9(b). The EIS must disclose and analyze opposing opinions. Center For Biological Diversity v. United States Forest Service, 349 F.3d 1157 (9th Cir. 2003). Thus far the BLM has failed to disclose opposing scientific opinion regarding toxicity of herbicide formulations and potential impacts of herbicide applications, thus violating this NEPA requirement

### **III. Cumulative Effects**

The PEIS fails across the board to identify cumulative impacts to human health and the environment that may arise from the proposed program. The example we use is the impact of pesticide applications that may be undertaken in the same watershed or which in some other manner may interact with herbicide applications undertaken by BLM. Such considerations must be part of the analysis so that the relevant agencies may be alerted to potential impacts on endangered species, under the ESA and to satisfy cumulative impacts analysis required under NEPA.

We attach maps of sections of Riverside County (Attachment C) and Monterey County (Attachment D) in California compiled by CATs from data recorded by California Department of Pesticide Regulation (CDPR) that indicate BLM managed land adjacent to or within the water or air shed of pesticide applications reported to the CDPR. Also attached are descriptions of the pesticides reported to be used within these areas (Table 1 - Monterey County and Table 2 - Riverside County; both given as attachments). Table 1 indicates that 1,395,407 pounds of pesticide active ingredients and 2,468,769 pounds of pesticide products were used in near BLM-managed lands in Monterey County; Table 2 indicates 1,908,760 pounds of active ingredient and 3,601,818 pounds of product were reported used in Riverside County near BLM-managed lands. Given that pesticide use is demonstrated by this data to be occurring, sometimes at significant rates, near BLM-managed lands which may be subject to pesticide application under the program, analysis of cumulative is required.

## **CATs' comments – Part II: Invasive species and herbicides issues**

### **1. Herbicides as a disturbance factor not analyzed.**

The Draft PEIS / PER fails in its analysis of the effects of the treatments that may be used to combat invasive plants and conduct vegetation management. Evidence exists, for example, that herbicides use may create conditions more hospitable to invasive species than were present before the chemicals were applied. CATs is concerned that by spraying herbicides on almost a million acres (more than tripling current application acreage), the BLM will be increasing potential invasive species infestations, rather than reducing them. This is contrary to and exactly the opposite of the BLM's stated project objectives for the PEIS. This evidence and indirect / cumulative effects of the proposed actions must be analyzed by the BLM in the PEIS.

### **Invasive species may thrive due to herbicide disturbances.**

Several studies have confirmed that increased nutrient availability, in the form of excessive dead organic matter, can favor non-indigenous annual species where natural nutrient levels may be insufficient. For example, increases in nitrogen (i.e. the widening of the C:N ratio) have shown to provide a competitive advantage to annuals such as cheatgrass that germinate much earlier in the season than native grasses (personal communication USGS, Corvallis, Oregon). If the BLM follows through with the proposed large scale increase of herbicide spraying, lots of plants will die, leaving an unnatural amount of dead organic matter on the ground, changing natural nutrient levels, and thus creating an unnatural advantage for unwanted exotic species.

Cheatgrass, for example, is not necessarily only encouraged by soil disturbance. It is more sensitive to light availability (Zouhar 2003). Increased spraying, especially wide spread aerial spraying, will kill large swaths of vegetation and drastically increase light availability. Decreases in adjacent canopy cover will introduce the invasion promoter of light that would provide suitable habitat for cheatgrass establishment (Zouhar 2003). The BLM cannot allow any treatment method (like herbicide spraying) that will just increase invasive species infestations. The BLM is proposing to spray invasives, but the spraying may actually create conditions more favorable to invasives rather than native species. This has the potential to become a continuous spraying loop.

Use of herbicides where non-native weed plants already occur frequently results in a reproductive advantage for non-native species, which then expand rapidly due to the lack of competition. In a short period of time, this can result in an exponential increase in non-native plants (Wooten and Renwyck 2001). The BLM fails to provide analysis of such information in the Draft PEIS, and it is found in the literature and very relevant to the issue at hand.

McDonald and Everest (1996) of the USFS Pacific Southwest Research Station, found that cheatgrass populations, not observed in the study plots at the beginning of a study, exploded in herbicide-treated plots in a vegetation management study comparing herbicides and non-chemical means of reducing unwanted shrubs. Herbicide plots ended the four year study with 743,667 cheatgrass plants per acre with 22% foliar cover, where cheatgrass was 6 times greater in number of plants and more than 7 times greater in foliar cover than in the non-herbicide control plots (130,300 plants per acre, 3% foliar cover).

McDonald and Everest (1996) found that the cheatgrass was colonizing ground cleared by herbicides. Harper and Whitehead, of the Canadian Forest Service, found similar dynamics in comparable ecological conditions in British Columbia. As he notes in his Brush River Brushing Trial site project report (1994) "Total number of species are higher in glyphosate than in other treatments due to the ability of invading plant species to colonize on exposed sites. The initial reduction of shrub and herb cover of naturally occurring species following herbicide application probably allows for the establishment of such 'invaders.'" That herbicides appear to be a disturbance factor that actually encourages invasive species to colonize and spread in herbicide-treated areas clearly must be analyzed in the PEIS.

A study done by the British Columbia Ministry of Forests Research Program in the Upper McKay Creek near Lillooet, B.C. found that the choice of herbicides can have a profound effect on the plant species content and diversity many years after treatment (Simar, Heineman and Youwe 1998). "The abundance of several low shrub species (black twinberry, black gooseberry, thimbleberry, trailing raspberry, red raspberry, birch-leaved spirea, and black huckleberry) was reduced for nine years following application of glyphosate. Hexazinone tended to have a longer-lasting effect than glyphosate on the abundance of grasses and forbs." As this report observes, "Plant communities naturally change over time, but sudden shifts in structure and composition may negatively affect the availability of food for wildlife." Lacking an analysis of the impacts over the long-term that may be expected from the use of various herbicides on non-target plant species composition and abundance, and lacking adequate guidance for which herbicides and other treatment options are suited or not suited for various ecological conditions common on BLM lands covered by the PEIS, the PEIS cannot serve as an appropriate tiering document for future decisions regarding invasive species as it is currently written.

## **Fire danger may be increased, not lessened due to herbicides.**

CATs contends that the proposed action of widespread spraying of herbicides to kill unwanted vegetation will result in increased fire dangers from standing dead biomass and exotic species invasions post spraying. Killing large amounts of the brush and other weeds with herbicides will undoubtedly increase light availability in heavy brush areas and thus increase potential noxious weed and invasive species habitat.

The BLM has failed to discuss in the PEIS cheatgrass' ability to indirectly benefit from herbicides and proliferate in disturbed herbicide sprayed areas, and then to create an additional major fire threats.

The BLM needs to take a long and honest look at the potential for creating that which they say they are trying to avoid, tinder dry forests and grasslands, thick with both living and dead ladder fuels. In essence that is exactly what will be created by the preferred alternative. In truth the only way to avoid this is to cut unwanted brush, either mechanically, or by hand, leave it on the ground to discourage new brush growth and noxious weed invasion, and restock the area the following planting season. This would provide jobs, give greater protection to wildlife, provide erosion protection, and create a healthier soil profile. The brush would decompose faster than dead brush left standing. A selective re-cut 2 to 3 years later would allow for release. The beneficial aspects of brush (soil and nitrogen production, wildlife feed and habitat) would allow for a faster growing and healthier forest.

Cheatgrass will not be suppressed from control of brush and wildfire fuels, and in fact will flourish wherever herbicide treatments happen. Cheatgrass lives for disturbed ground, be it from fire or herbicide application. Cutting and leaving the brush component on the ground will control cheatgrass spread. This in turn will help ensure that the fire threat cheatgrass produces will not exist, nor will the home it would provide for gophers and grasshoppers exist.

With cheatgrass spread comes the lengthening of the fire season and increase in numbers of fires, the very same fires that the BLM's PEIS is suppose to avoid. This invasive greatly impacts ecosystem functioning causing changes in fire regimes including increased fire frequency and extent, often to the point where native species cannot recover (D'Antonio et al 2002, Brooks et al 2004, Young and Clements 2005). The Tahoe National Forest wrote in the Cottonwood FEIS (2005) "The biggest threat the project area faces from cheatgrass is repeated stand replacing fires...Cheatgrass dominated communities tend to burn more frequently and can shorten the fire return interval, thus effectively hampering the recovery of native vegetation (Personal communication, Young, 2002)."

The probability of increased fires due to cheatgrass proliferation is not part of the effects or alternative analysis and thus fails to fully inform the decision maker. The frequent fires associated with cheatgrass infestations, as well as all the dead brush left standing from the herbicide spraying has the likely potential to wipe out a vast areas of public lands, and render this project a huge waste of BLM resources and tax payer money.

These expected frequent fires will burn up the dead brush and leaf litter left from herbicide spraying and thus open up the soils and provide a medium where cheatgrass populations will expand and thrive, thus compounding the problem.

The factors contributing to the proliferation of existing weed populations and the establishment of new populations have been described above. The advent of increased light availability and soil disturbance to sprayed areas sets the stage for this advancement of invasive weeds. The use of herbicides is unwarranted as its

current proposed application would not accomplish the desired goals. In addition, it is feared that the proposed fuels reduction treatments would promote further weed establishment and increase the area of existing weed populations, resulting in future proposals for even more widespread herbicide treatment to control an escalating weed problem. Until further exploration of intensive weed management is considered, the use of herbicide, as described in the PEIS, is futile, insufficient and exposes the public lands to vast amounts of toxics unnecessarily

## **2. Alternative (non-herbicide) treatment methods**

### **Failure to evaluate a reasonable range of alternatives**

#### Restore Native Ecosystems Alternative

The BLM has failed to include an alternative based on ecological healing and prevention. The current PEIS is focused purely on treatment of symptoms, rather than prevention of the conditions that lead to the problem. CATs supports an alternative with a focus on restoring native ecosystems. In such an alternative the BLM would view vegetation management in the context of first, prevention of conditions that have led to introduction, colonization, proliferation, and spread of invasive species and fuels hazards; and then second, restoration of healthy public lands (including forests, grasslands, etc) to strong native ecosystems; thereby third, reducing the need for continued treatments (passive restoration). An alternative suggested to the BLM, yet without reason excluded from the PEIS / PER analyses, that CATs endorses is the Restore Native Ecosystems Alternative (see Appendix B for details).

#### Non-Herbicide Treatments for Noxious Weed Species

The BLM has neglected to consider the use of non-toxic organic herbicides and other weed control methods utilized by organic farming practices. For example St. Gabriel Laboratories produces an organic herbicide called Burn Out. It is advertised to work faster than Roundup (the glyphosate the BLM is proposing to liberally apply) and by meeting NOP Organic Farming Requirements is less likely to have adverse impacts to the environment or human health. If the BLM insists on using herbicides, why not use ones that are least likely to have adverse environmental impacts? What about hot foam or other non-herbicide methods the BLM has used in other projects before? What about mulching/covers and solarization? What about organizing volunteer weed pulling days? What about flaming or torching? Goats? Bio-control agents?

The BLM has failed to include an IPM alternative. Weed control scientists regularly point to the necessity of integrating multiple methods for effective long term weed control.

### **Lack of IPM**

The only viable alternative for control of invasive plant species is Integrated Pest Management (IPM). IPM involves combining elements of the various treatment methods with preventative measures, increased knowledge of the target species biology and ecology, and restoration of the biotic and abiotic components of a habitat before or concomitant with the removal of the invasive exotic (Achuff et al., 1990; Thomas, 1986; Thomas, 1991). Invasion of a community by an alien plant usually occurs because that community has been disturbed, either in terms of its vegetation structure, composition, or its topography (Thomas, 1986). For an exotic to be successfully removed from a community, the disturbance factor that allowed the alien to invade in the first place must be removed and the habitat restored to as near to its original condition as possible (Thomas, 1986). This habitat restoration can involve restoring the native dominants, filling vacant niches with natives, restoring natural densities, restoring age and class structures, and correcting any disturbed physical conditions (Thomas, 1986). If these steps are not taken, the removal of an exotic species may be followed by either reinvasion or establishment of another exotic (Thomas, 1986) Without adequate analysis, IPM cannot be

utilized,

## Defining IPM and IWM

Integrated Weed Management (IWM) is a subset of integrated pest management (IPM), and frequently referred to by the BLM in regards to invasive weed management plans. The California Department of Pesticide Regulation calls IPM a widely accepted approach to pest management that results in effective suppression of pest populations while minimizing human health and environmental hazards. Yet the BLM is disregarding public health, instead proposing actions focused on killing as much unwanted vegetation as quickly as possible, while incorrectly claiming it uses IPM to deal with unwanted vegetation.

IPM is defined as: "...a pest management strategy that focuses on long-term prevention or suppression of pest problems through a combination of techniques such as monitoring for pest presence and establishing treatment threshold levels, using non-chemical practices to make the habitat less conducive to pest development, improving sanitation, and employing mechanical and physical controls. Pesticides that pose the least possible hazard and are effective in a manner that minimizes risks to people, property, and the environment, are used only after careful monitoring indicates they are needed according to pre-established guidelines and treatment thresholds" ([www.cdpr.ca.gov](http://www.cdpr.ca.gov)).

CATs is concerned that the BLM has failed to discuss and disclose established weed treatment threshold levels for this project. The BLM needs to establish that current treatments are failing to control weed infestations at pre-established threshold levels before considering the use of toxic chemicals. CATs also expects the BLM to quantify any weed increases above threshold levels. How much are populations increasing? How big were infestations when treatments began and how big are they now? Where are the monitoring results to determine whether past treatments have been effective or if new treatments are needed?

Published scientific literature provides overwhelming evidence that one-time herbicide applications for the purpose of weed eradication will only deliver short-term results. Over time, the seed bank of existing populations will allow weed populations to re-establish in project areas, often in greater density, abundance, and extent (Zouhar 2003, CDFA Encycloweed website, Huckins and Soll 2004, Raj 2002, Hoshovsky 1986). Herbicide use for noxious weed management has been shown to be neither the lowest impact nor the most environmentally or economically effective control treatment method (CDFA Encycloweed website, Kedzie-Web et al. 1996, Huckins and Stoll 2004, Hoshovsky 1986).

The key to any IPM strategy is to know the ecology, biology, and life cycle of the invasive species. "Integrated pest management is a proven approach to managing pest problems, including invasive nonnative plants. Integrated pest management is based on a sound understanding of the ecology and biology of a pest and its environment" (Andrascik et al. 1996). This is something the BLM must do and include within NEPA documentation prior to evaluating control plans. The BLM has failed to even identify the primary species targeted for herbicide spraying. How the species reproduces, spreads, and colonizes are all essential information. Some species are known for being prolific seed producers and maintaining extensive seed banks, while others reproduce vegetatively and can clone themselves.

Effective weed control has shown to be dependent on the integrating combinations of treatment techniques (Archer 2001 and <http://tncweeds.ucdavis.edu/esadocs.html> and [http://www.cdfa.ca.gov/phpps/ipc/encycloweed/encycloweed\\_hp.htm](http://www.cdfa.ca.gov/phpps/ipc/encycloweed/encycloweed_hp.htm)). Weed management used in any other fashion is not likely to successfully reduce, suppress, and/or eradicate weed populations.

Successful weed management is a direct result of proper timing (DiTomaso 2001, Kedzie-Webb et al. 1996, CDFA website, Pitcher 1986, WA Noxious Weed Control Board). Timing for treatments must be included in

the analysis of how the project will achieve the overall goal of returning the area to the desired status of a “biologically and structurally diverse forest”, but it is not. Significant effects must be analyzed, and improper timing of weed treatment is recognized as a contributor to a significant effect, the spread and establishment, and eventual dominance and contribution to severe hazardous fire frequency of invasive plants, particularly the dreaded cheatgrass (Zouhar 2003).

### **Examples of non-herbicide treatment methods for major prevalent invasive weeds**

Below, CATs will provide examples of non-herbicide treatment methods that should be part of the BLM’s IPM alternative for some of the more common invasive species on BLM lands in the western US.

#### **Yellow Starthistle**

There is an abundance of literature regarding the control and management of yellow starthistle (YST). The BLM has failed to disclose this information and has thus skewed the evaluation of feasible alternatives. California governmental sources often rely on the expertise of Dr. Joseph DiTomaso of the University of California, Davis, in regards to YST management and control. DiTomaso states in UC Davis’s Weed Research and Information web site that viable treatment options include grazing, mowing, manual removal, perennial grass reseeding, burning, and biological control. Yet the BLM has failed to evaluate most of these methods. With a myriad of low-impact effective and commonly used treatment options available, why is the BLM so focused on spraying? Hand pulling, hoeing, and other manual removal methods are most effective for smaller infestations. They are an “important tool in steep or uneven terrain” and “typically cause minimal environmental impact” (DiTomaso 2001).

In areas where the starthistles are working against other competitive vegetation, hand pulling is particularly easy and effective. Why is there no discussion or analysis of integrating manual and cultural treatments? Using the “Bradley method”, it is possible to control large starthistle infestations at low costs while risking low impacts (DiTomaso 2001).

The following excerpt was taken from the Integrated Vegetation Management's Technical Bulletin, Bio-Integral Resource Center, Berkeley, CA. (Drlik et al 1998): “The Bradley method is an approach that was developed by the Bradley sisters in Sydney, Australia. It combines the strategies of containment and reduction and can be used most successfully in natural areas where weed stands are close to or intermingled with native vegetation. This approach uses carefully planned hand weeding to tip the ecological balance in favor of the native vegetation, which is then allowed to regenerate and fill the area where the weeds have been removed. The weeding is always done outward from the edge of the best stands of natives. The Bradley’s recommend choosing an area you can visit easily and often, where the native vegetation meets a mixture of natives and weeds not worse than 1 weed to 2 natives. Using this method, the two Bradley sisters (both over fifty) cleared a 40-acre woodland reserve so successfully that the area needed only slight attention once or twice a year (mainly in vulnerable spots such as roadsides and creek banks) to be maintained weed-free. To do this they expended only a minimum amount of time: an average of 45 minutes per day between the two of them. This low-cost, low-impact approach enables restoration to occur with minimal labor or equipment.”

Other management methods recommended by experts and ignored by the EA for evaluation and analysis are tilling, mowing, grazing, and prescribed burning. Mowing, a cost effective late season tool, is also a popular treatment method (DiTomaso 2001). Properly timed mowing (or weed whacking) can limit YST ability to produce seeds, provide excellent control, and reduce seed banks and populations. The BLM should at the least be considering an integrated method alternative that combines mowing, grazing and hand pulling with revegetation efforts.

Grazing has been shown to be effective controlling young yellow starthistle plants (DiTomaso 2001). If integrated with mowing, burning, bio-controls, or even as a treatment for re-growth after hand pulling, grazing could be efficiently and effectively utilized for controlling yellow starthistle. Yet the BLM has failed to mention or even consider grazing.

Properly timed grazing (early season) or prescribed burning (late season) have both been used with success as controls (DiTomaso 2001). These are feasible options. DiTomaso says that grazing can help competitive vegetation and that it is good for use in both the first year of control program and as maintenance in later years (2001). He also highly recommends prescribed burning, calling it very effective, especially when used in conjunction with re-seeding methods. Burning is recommended for use in the first, second, and third years of long-term management strategies (2001). Is the BLM planning to wage war on weeds for the long term? Why were these feasible options not included as potential alternatives or part of an integrated management strategy? The current EA is unacceptable and in violation of NEPA due to its failure to include analysis of long term, viable IPM options such as these.

The EA fails to consider the option of using bio-control agents on yellow starthistle even though the literature shows that it has proven effective. Six different insects have become established in California for controlling Yellow starthistle. Two in particular, the false peacock fly (*Chaetorellia succinea*) and the hairy weevil (*Eustenopus villosus*), have been shown to have significant impact on seed production (DiTomaso 2001). DiTomaso (2001) also states that several plant pathogens have shown promise as bio-control tools, and in particular the naturally-occurring and host-specific *Ascophyta spp.* DiTomaso states that bio-control is recommended to be part of any integrated management strategy and that they provide the possibility of long-term and sustainable management (2001). Isn't that the ideal goal of noxious weed management? Bio-controls should at least be mentioned and evaluated?

DiTomaso mentions problems with using herbicides as part of an integrated, long-term management strategy for YST. DiTomaso reports that herbicides are not effective in the early years of a long-term strategy and do not provide control of seeds germinating after treatment. Yet this is exactly what the BLM is proposing. Is the BLM looking for a long-term solution to invasive plants or a short-term fix? Why has the BLM failed to disclose this information within the EA? While glyphosate is reported by DiTomaso to be effective on YST seedlings, so are hand pulling and other methods, which have lower adverse impact potential. The BLM has failed to objectively discuss the potential problems and disadvantages of their herbicide solution, again failing to comply with NEPA requirements and thus the PEIS is unacceptable.

#### Tamarisk (Salt cedar)

Many options exist within the scientific literature for control and removal of tamarisk without the use of herbicides. CATs expects the Forest to create, include, analyze, and utilize a complete long term non-herbicide IPM alternative using several treatment methods at the appropriate stages of the project. Below is a compilation of non-toxic methods CATs advocates for found in the scientific literature.

No one technique alone will usually work. The Nature Conservancy has compiled much information on management of Tamarix species. Experts report high effectiveness using a combination of cutting and burning or just digging up tamarisk at Joshua Tree National Park, CA, Organ Pipe Cactus National Monument, AZ, and Picacho State Recreation Area, CA. It is possible to cut or burn and then control sprout regrowth with cattle or goat grazers. It has also been suggested that repeated cutting or burning may kill the root system. Hand pulling of tamarisk is effective for removal where the plants are small or where access is difficult (Carpenter 1998).

Also a fair amount of research has been compiled by the Rocky Mountain Research Station regarding tamarisk management. Whatever the techniques that are used, it is essential that the entire root system is killed

eventually. Restoration with desirable natives is the key to long term success. An integrated approach which focuses on the invaded system and managing factors that facilitated invasion are advocated for by scientists. Manipulating dam releases and flow regimes, causing flooding, inundation and scouring are recommended by several experts and work best in areas with native species already in place (Zouhar 2003).

Most managers and researchers recommend combining physical, biological, and cultural control methods. Cutting and pulling is the most effective in small newer infestations. Root plowing is reported to be one of the most successful mechanical control methods for tamarisk, especially in relatively dry soils. Cutting, burning and mowing are other effective ways for initial clearings. Grazing can control regrowth. The tamarisk leaf beetle may be another excellent biological control method. Shading is especially effective on mature saltcedar (Zouhar 2003).

Mechanical (bulldozing and rootplowing) treatments have been shown to provide better control than herbicides. Experts conclude that tamarisk is very difficult to kill with herbicides alone. One study found that bulldozing was just as effective as imazapyr applications. This is important as imazapyr is highly mobile and persistent and effects a wide variety of plant species. Recent studies have reported that imazapyr can leak out of the roots of treated plants and adversely affect surrounding native vegetation (Zouhar 2003).

Knapweeds (Spotted, Diffuse, and Squarose, *Centaurea sp.*)

Hand pulling and digging of knapweed infestations has been shown to be an effective treatment method, especially for small infestations and all Diffuse knapweed infestations (CDFA Encycloweedia website, Carpinelli 2003, Dirlik et al 1998, Engeland 1988, Waldo 2001).

Engeland (1988) states that areas of severe knapweed infestation can be best treated using a group of people working a few hours daily over a period of several weeks. This strategy has been effectively used in many areas, but perhaps nowhere as successfully as by the Salmon River Restoration Council (SRRC) and their partnership with the Klamath National Forest. The SRRC is an excellent model to follow for using volunteer labor to effectively remove and eliminate invasive species without herbicides. The SRRC has been winning the war against knapweeds using manual efforts. The SRRC is happy to not only share their model, but also to help teach recruitment and training of workers. SRRC information can be found on the web at <http://www.srrc.org/>.

Using native perennial grasses and forbs is effective for providing competition for knapweed seedlings and prevent spread and reinvasion (Beck 2005, Dirlik et al 1998, Waldo 2001). “A two-year study of four grasses – Paiute orchardgrass, Covar sheep fescue, Critana thickspike wheatgrass, and Ephriam crested wheatgrass – found that the greater the biomass produced by the grass, the more it reduced the number of diffuse knapweed seedlings” (Mauer et al 1987).

Prescribed burns, followed by aggressive re-seeding efforts, can reduce knapweed infestations (CDFA Encycloweedia website, Waldo 2001). Timely mowing is also a feasible control method for knapweeds, as it will reduce seed production (CDFA Encycloweedia website, Mauer et al 1987, Waldo 2001). The same can be said regarding grazing, as goats and sheep have shown to control spotted knapweed (Beck 2005, Carpinelli 2003, Dirlik et al 1998, Waldo 2001).

Many experts recommend the use of biological controls for managing knapweeds. It is feasible for use on larger infestation sites. Several biological control agents have been established in the U.S. to attack knapweeds. Only two, the bronze knapweed root borer (*Spinoptera jugoslavica*) and the banded gall fly (*Urophora affinis*), are currently established, effective, and used for control of diffuse and spotted knapweeds, and both are compatible for dual release (Beck 2005, CDFa Encycloweedia website, Carpinelli 2003, Dirlik et al 1998, Mauer et al 1987, USDA-APHIS 1994). In drier climates, the knapweed flower weevil (*Larinus minutus*) has

shown itself to be a very effective control of diffuse and spotted knapweeds in Oregon and Washington (Waldo 2001).

Some experts warn against herbicide use on knapweeds as it only provides temporary control, not preventing germination from seeds in soil, needing long-term re-treatments, and due to the cost being prohibitive for larger infestations (CDFA Encyclopedica website, Mauer et al 1987, USDA-APHIS 1994).

### Brooms (Scotch and French)

The literature on scotch and french brooms states that they both grow best in dry, disturbed soils with plenty of sunlight, such as those created with new partial cutting timber harvest techniques (Raj 2002). The literature continues on that line saying that the brooms rapidly invade following logging and land clearing (CDFA Encyclopedica website, Huckins and Soll 2004, Hoshovsky 1986). The brooms do not do well in heavily forested areas and don't tolerate heavy shade (CDFA Encyclopedica website, Huckins and Soll 2004, Hoshovsky 1986).

The literature states that because of extremely long-lived seeds broom control requires long-term management to exhaust the seed bank and prevent rapid recolonization of treated areas (CDFA Encyclopedica website, Huckins and Soll 2004, Raj 2002, Hoshovsky 1986). Many non-herbicide methods are recommended in the scientific literature for effective scotch and french broom control/removal. An integrated approach, requiring several years of treatments is accepted as the most effective, economical, and environmentally sound strategy (Huckins and Soll 2004, Hoshovsky 1986). Established broom infestations will require persistence and retreatments each year for ten years or more, regardless of methods chosen (Huckins and Soll 2004, Parker, Miller and Burrill 1998).

Manual hand methods are highly selective and can remove broom without impacts to desirable vegetation (Huckins and Soll 2004, Raj 2002, Hoshovsky 1986). Experts suggest hand pulling as a good and preferable manual broom removal method. It should be done in moist soils (Huckins and Soll 2004, Hoshovsky 1986). Also there are several hand tools for pulling broom plants including: weed wrenches, root jacks, pulaskis, and more (Huckins and Soll 2004). The weed wrench is mentioned as one of the most effective techniques for complete broom removal (CDFA Encyclopedica website). Hand hoeing and grubbing out crowns effectively control large plants (Parker, Miller and Burrill 1998, Hoshovsky 1986). Hand digging is a sure way of removing broom plants (Hoshovsky 1986). Well timed (before seeds mature) and executed cutting, especially effective manual cutting methods (at ground surface level) which can nearly eliminate re-sprouting, are recommended as an important first step in an integrated broom management plan (CDFA Encyclopedica website, Huckins and Soll 2004, Raj 2002). Experts conclude that the key to long-term broom control is prevention of seed set after the initial clearing takes place (Huckins and Soll 2004).

Mechanical control has shown to be practical in some instances, using tractor mounted mowers or scythes, depending on terrain (CDFA Encyclopedica website, Huckins and Soll 2004, Raj 2002, Hoshovsky 1986). Repeated mowing/cutting can exhaust broom plant food supplies (Hoshovsky 1986). The literature contains examples of grazing, using goats (Angora and Spanish) as shown to be effective in controlling broom (CDFA Encyclopedica website, Parker, Miller and Burrill 1998, Hoshovsky 1986). Some expert sources suggest that goats are the most effective for controlling regrowth as a follow up control method after burning or cutting (CDFA Encyclopedica website, Huckins and Soll 2004). Goats can be less costly, can negotiate steeper slopes, and don't pose the environmental dangers inherent with herbicides (Huckins and Soll 2004, Hoshovsky 1986).

Experts support the use of torching (flame thrower or weed burner) to heat-girdle the lower stems as spot treatments (Huckins and Soll 2004, Hoshovsky 1986). This technique is reported to be less costly than

herbicide treatments (Hoshovsky 1986). Large infestations can be removed using prescribed fire, but follow up methods are needed as fire can stimulate broom seed germination (CDFA Encycloweedia website, Huckins and Stoll 2004, Raj 2002, Hoshovsky 1986). This can be preferable for reducing the remaining seed bank far more quickly (Huckins and Stoll 2004). The literature states that burning of broom should be followed by re-burning, manual seedling removal, and re-vegetation with competitive native species (Huckins and Stoll 2004). Re-vegetating with, at first, native perennial grasses and forbes, and later with native broadleaf plants will be necessary for long-term control (CDFA Encycloweedia website, Huckins and Stoll 2004, Hoshovsky 1986).

For large infestations, the Bradley method is recommended as a sensible approach for manually controlling weeds (Fuller and Barbe 1985). This method consists of hand weeding small areas of the infestation, starting with the best stands of native vegetation (those with the least weeds) and working towards those stands with the worst weeds. Initially, single and small groups of weeds should be removed from the edges of the infestation. Next, work on areas with at least two natives to every weed. The native populations will be stabilized in each cleared area, and then one should progressively work deeper into the center of the most dense weed patches. This method has great promise for sensitive natural areas with low budgets.

### Harding grass

Harding grass likes to grow in open sites, such as grasslands and rangelands, watercourses, and disturbed sites, like roadsides and trails (Holloran et al 2004). It produces a high number of seeds between May and September and can also spread vegetatively (Holloran et al 2004). Seeds remain viable for one to three years (Holloran et al 2004).

Experts report that there is no evidence to suggest that Harding grass will threaten areas with healthy native vegetation (CDFG 2000, Peterson 1988). Thus localized occurrences of Harding grass infestations are not a problem in grasslands or other plant communities of otherwise high quality (Peterson 1988).

Keys to management includes minimum of three years of treatment (due to seed longevity), retreating resprouts from roots left in the soil, and beginning removal efforts before infestations get large and are fully established (seedlings are less aggressive and do not compete well with other species) (Holloran et al 2004).

As with management efforts of most invasive species, experts recommend an integrated multi-technique approach (Holloran et al 2004).

Mulching can be effective for smothering smaller infestations. The trick is to cover the infestation with some sort of weed barrier – landscape fabric, nylon, plastic, or cardboard – and then place three to six inches of rice straw or wood chips on top of that. Once the Harding grass underneath is dead, remove the weed barrier and revegetate with native plants (Holloran et al 2004).

Digging and pulling can effectively remove Harding grasses (Peterson 1988). Smaller clumps can be hand-pulled but larger ones need to be dug up (Huselid-Glass and Hernandez 2004). Cut around the base of each clump (with a Pulaski or similar tool) and then dig the roots out (Holloran et al 2004). It is important to remove all roots greater than two inches to prevent resprouting and reestablishment. Then follow up the digging efforts with a thick layer of mulch (about six inches) of straw (rice) to discourage resprouts (Holloran et al 2004).

Mowing and clipping can be an effective method for controlling Harding grass infestations (Holloran et al 2004, CDFG 2000, Peterson 1988). Mowing repeatedly during the growing season (at least 3 times) will ensure that plants won't flower and suppresses above ground growth (Holloran et al 2004, CDFG 2000, Peterson 1988). Experts also recommend mowing close to the ground, generally late spring, when it is later in the growing

season (Holloran et al 2004). Research shows greatest success with repeated mowing as it weakens the grass and reduces the seedbank (Holloran et al 2004, Peterson 1988).

Prescribed fire is an effective tool to use on Harding grass. Burning in the winter will reduce growth for about two years and allow more competitive native fire-adapted plants to increase and compete more effectively (CDFG 2000, Peterson 1988). Experts recommend burning at two-year intervals as part of management strategies (CDFG 2000, Peterson 1988). There is some evidence that older plants lose their vigor, have less above ground shoots, and roots decline. This provides annual grasses and shrub species the ability to out compete Harding grass when it is in this declining stage (Peterson 1988).

Experts also recommend brush cutting small patches and then covering with landscape fabric. The other, yet very similar option, is after brush cutting, to mulch with a 6-inch layer of straw, and pull any emerging plants the following year (Holloran et al 2004). Pulling will likely be the most successful after rains, when the soils are moist and loose and less roots will be broken off and left in the ground. It is recommended that after mowing and covering, to plant native shrubs and trees to shade out any resprouts (Holloran et al 2004).

Since seedlings don't compete well with other species it is important to establish competitive native species (perennial grasses and forbs) after treatment efforts. Revegetation is also important to prevent recolonization of treatment sites by new invasive species.

#### Dalmatian toadflax

As an aggressive, adaptable, deep-rooted perennial, prevention is the key with Dalmatian toadflax management. Since seedlings are at a competitive disadvantage, cultural control methods focusing on maintaining well adapted native perennial grasses can produce competition problems for the toadflax seedlings and help prevent reinfestation (CDFA Encycloweediea website, Moser and Crisp 2001, Lajeunesse 2004, WA Noxious Weed Control Board 2003).

It is recommended that all treatment programs are initiated during June when root carbohydrate reserves are at their lowest, making root system recover the most difficult (Carpenter and Murray 1998).

Hand pulling or grubbing has been found to be effective treating small infestations (CDFA Encycloweediea website, Carpenter and Murray 1998, Lajeunesse 2004, Moser and Crisp 2001). Hand treatments that have been found to be extremely effective if done repeatedly for 5 – 10 years. At the Magnusson Butte Preserve, in Washington, a decade long hand-pulling experiment proved how effect non-chemical treatment could be over a 28-acre preserve. Flowering stems were reduced 90-90% over the preserve over the first couple of years and by the third year that the few remaining flowering stems were significantly smaller. An increase in native and non-native grasses and perennial forbs was also notice post hand pulling treatments (Carpenter and Murray 1998).

Cutting stands of Dalmatian toadflax in spring and early summer can help lower reproduction in larger stands (Carpenter and Murray 1998). While grazing is not normally used to manage Dalmatian toadflax, preliminary results of field trials in Montana show that sheep can be used to help suppress stands and limit seed production (Lajeunesse 2004).

An abundance of information exists regarding the potential and successful usage of biological controls for treating toadflax infestations. The most common bio-control agent is the toadflax moth (*Calophasia lunula*), which is highly active in the larval stage and has been shown to dramatically defoliate plants, reduce seed production, and lower root carbohydrate levels. Several other bio-control agents have been shown to be effective for controlling Dalmatian toadflax including: a shoot, flower, and ovary feeding beetle, *Brachypterolus pulicarius*; two seed capsule feeding weevils, *Gymnaetron antirrhini* and *Gymnaetron netum*;

and a particularly effective stem boring weevil, *Mecinus janthinius* (CDFA Encyclopedea website, Carpenter and Murray 1998, Harris and DeClerck-Floate 2003, Hansen, Lajeunesse 2004, Moser and Crisp 2001, WA Noxious Weed Control Board 2003).

There are several problems related to herbicides and effective treatment of toadflax infestations mentioned in the literature due to the plants' high genetic variability. Herbicides have been shown to have mixed results, tending to run off of the waxy toadflax leaves and are considered to be impractical for large sized infestation sites due to economic, logistic, and environmental constraints (CDFA Encyclopedea website, Harris and DeClerck-Floate 2003, Hansen, Lajeunesse 2004, Moser and Crisp 2001).

### **3. BLM failing to prevent cause of weed infestations, instead just treating symptoms.**

CATs is concerned that the BLM has failed to discuss, analyze, or evaluate weed vectors as part of the PEIS. The BLM should determine the major sources of weed spread (waterways, vehicles, area visitors, livestock grazing, wind and/or wildlife) and include a plan to prevent the cause of weed spread, not just treat the symptoms. Including preventative measures as part of any treatment strategy is critical for long-term control of invasive species and noxious weeds. The BLM's PEIS is doomed to be unsuccessful without first focusing on the cause of the weed infestations and utilizing a holistic native species ecosystem health approach to combating exotic species. For that reason CATs supports the Restore Native Ecosystems Alternative (which we didn't see evaluated as one of the alternatives, even though we know a coalition of groups presented it to the BLM during the scoping phase of the PEIS). We will attach a copy as Appendix B as part of our comments.

Focusing non-chemical control efforts along the river corridors, at trail heads and recreation locations, and along side roads would be an obvious starting point for reducing weed vectors. If prevention actions aren't part of the proposed project, after a few years, following project completion, a new problem may arise, with possibly worse conditions. CATs questions the wisdom of the proposed herbicide related actions without a long-term game plan to manage invasive species in the project area, and hopes the BLM provides this as part of project NEPA documentation.

The standard Region 5 Forest Service prevention weed methods of washing heavy equipment and vehicles, weed free straw, and education of area users are a good start (USDA Forest Service 2000). While CATs applauds these efforts, we feel that more can, and should be done. The BLM must include those and additional methods as part of the proposed actions for this project to be successful. Immediate action, digging or pulling new infestations, post and pre project monitoring, and flagging and avoiding large infestations can all be effective. These are basic prevention methods commonly referred to by weed experts and utilized with success by many public land managers.

The BLM needs to develop a plan to deal with prevention, and eliminate disturbance factors that led to past, and will lead to future, invasive species distribution and establishment. Re-vegetation with desirable and competitive natives is essential, but timing and reduction of the seed bank first is essential to rehabilitation success. What specific activities on BLM public lands have facilitated invasive species infestations? What can the BLM do to limit future invasions?

Seed banks exist and one-time (or short term) herbicide spraying treatments will not prevent the weeds from returning and proliferating, most likely in greater numbers, as herbicide residues in the soil will kill any competitive natives. Each noxious or exotic weed species must be analyzed to determine the most effective treatment strategy. It appears that the BLM accepts the presence and proliferation of noxious weeds and cheatgrass, as the PEIS fails to disclose adequate prevention measures.

Disturbances are likely to occur beyond what is described in PEIS or PER. The PEIS fails to even outline efforts to keep vehicles, machinery, or workers (shoe treads, clothing) clean of exotic seeds, the very least that can be expected. Unfortunately, while cleaning efforts will reduce the likelihood of seed dispersal, this approach is not fail-safe and in most cases avoidance is not feasible. It is possible however to set strict guidelines that weed infestations exceeding specific magnitudes of density or area will be avoided. Recent land management policy (USDA Forest Service) has suggested buffers established around weed populations are necessary to ensure their isolation (Lassen National Forest 2005, Clark 2003). Such mitigation will reduce the extent of future herbicide treatments deemed necessary for weed suppression. For this reason, among others described below, a more thorough analysis is required so that mitigations can be formed.

CATs will briefly discuss four activities, grazing, logging, off-road vehicles, and re-vegetation, that the BLM should be looking at and analyzing in the PEIS and evaluate how they can be dealt with to reduce weed spread in our public lands. This is by no means an exclusive list of weed vectors that must be part of the PEIS herbicide application analyses.

### **Grazing**

CATs is concerned that grazing has been suggested as being a significant factor contributing to changes in forest structure leading to both high fuel levels and invasive plant species (Belsky and Blumenthal 1997). Livestock typically prefer to graze perennial native grasses and forbs thus reducing their biomass, density, diversity, and reproductive capabilities. This culmination of adversity eliminates native competition from exotic annuals. Additionally, the soil disturbance resulting from livestock trampling and the bare ground produced by grazing of grasses not adapted to such pressure provides ample opportunity for noxious and invasive weed seed germination. Livestock are also responsible for weed seed dispersal by carrying seeds stuck in their fur and hooves and by ingesting seed and later excreting the seed in new locations, often scarified and prepared to germinate. Finally, through soil compaction from trampling, livestock are responsible for reducing infiltration rates in soil thus reducing soil moisture levels. Exotic annuals have been observed out-competing natives for soil moisture (Weed Research Information Center of the University of California Cooperative Extension). This is critical when soil moisture may be limited as a result of soil compaction or already limited due to interspecific competition. The exclusion of grazers from sensitive areas where weeds exist already or may spread to in order to facilitate the restructuring of soil, provide a competitive advantage to native perennials, and eliminate an additional vector of seed dispersal, is necessary to achieve the desired goals of the PEIS. The exclusion of grazers from existing infestations is most crucial and should be the bare minimum expected.

Conversely, grazing could be considered as a tool for weed suppression and vegetation management. Such a technique is usually most successful when used in combination with other weed control techniques and employed over several seasons with cautious and restrictive rotational grazing practices (CDFA Encycloweedia website, Pitcher 1986, WA Noxious Weed Control Board). However, as previously mentioned, the use of grazers in weed management is a delicate tool that must be applied with great responsibility and commitment, not without careful planning, full analysis and monitored implementation. There is no hint of this level of awareness in the FEIS.

### **Logging**

Logging, whether part of fuel reduction thinning efforts, or timber harvesting, changes canopy levels, causes disturbances to soil and vegetation, and opens lands to possible invasive species infestations. For example, the scotch and french brooms both grow best in dry, disturbed soils with plenty of sunlight, such as those created with new partial cutting timber harvest techniques (Raj 2002). The literature also says that brooms rapidly invade following logging and land clearing and coversly don't do well in heavily forested, heavy shade areas

(CDFA Encyclopedica website, Huckins and Soll 2004, Hoshovsky 1986). Logging equipment, vehicles, and workers also facilitate the movement of exotic weed seeds. The BLM should analyze the impacts of logging and fuel reduction activities will have on the spread of invasive species and noxious weeds both on and near BLM lands.

The implementation of fuel reduction efforts will inherently increase light availability and disturb the soil surface. These actions will create optimal conditions for the invasion of noxious and invasive plant species as well as undesired natives. In such an instance, undesired vegetation, or early seral species, are typically represented by annual grasses and weeds, and woody shrubs (Merriam et al. 2005, Zouhar 2003, Raj 2002, CDFa Encyclopedica website, Huckins and Soll 2004, Hoshovsky 1986). The successful establishment of such a stratum could result in high fuel levels in as little as three to five years, or as long as five to eleven, depending on vegetation types, but all would require maintenance in the next two to five years. CATs fears such an outcome will prompt the BLM to adopt a chemical dependent maintenance strategy. CATs is opposed to and will not support any forest management actions that will result in the potential for future herbicide use.

Thinning can have both positive and negative effects depending on the forest type and its existing structure and age (Graham et al. 1999). Pre-fire fuel reduction projects have been shown to facilitate invasive species infestations both in fuel breaks and in adjacent wildlands (Merriam et al. 2005).

In addition to the proliferation of vegetation, the fuels treatment areas will experience reduced surface fuel moisture and increased flammability (Countryman 1955 as cited in Weatherspoon 1996). The greater the stand opening, the more pronounced the change in microclimate is likely to be. Increased ladder fuels and decreased surface fuel moisture can be a catastrophic combination. These effects must be analyzed within the PEIS (or PER).

There is no questioning that the existing conditions and fuel levels need to be mechanically treated prior to the implementation of any fire and/or fire surrogate maintenance strategies. Abundant surface and ladder fuels, and dense stands pose a high risk for any prescribed burning efforts. However, studies have shown that following the mechanical treatment, underburning every 5-8 years is required to stabilize the system in order to reintroduce any type of natural fire regime (Stephens 1998).

Studies have shown that fuel breaks alone will not halt the spread of wildfire. Consistent prescribed burning has shown to be the most effective treatment for reducing a fire's rate of spread, fireline intensity, flame length, and heat per unit of area (van Wagendonk 1996). The implementation of frequently designed DFPZs, as done by the USFS, will ultimately fail due to their excessive harvesting prescription promoting the proliferation of surface and ladder fuels, the reduction in surface fuel moisture resulting from increased insolation, and the lack of landscape scale prescribed burning, or alternative fire surrogate strategies, used in combination with the fuel breaks.

Although the proposed actions are rehabilitative and preventative in nature, plans for consistent long-term maintenance need to be implemented for proactive management. The proposed actions intend to return the BLM lands to pre-historical natural conditions. But without changes in future management, the existing conditions will likely return with more severity. Specifically, the Forest should include consistent prescribed burning as an element of their typical management practices. We hope that the BLM evaluate and incorporate a maintenance strategy, founded on prescribed burning, into the proposed action plan. The restoration of a site to pre-historically natural conditions is unlikely to be achieved with the omission of a reoccurring fire regime. We fear that negligence of future maintenance could lead to circumstances where the BLM incorrectly feels that chemical treatment of vegetation would be the only viable solution. We are opposed to any land management actions that will likely lead to future vegetation management strategies dependent upon herbicides.

## **Off-road vehicles**

Vehicles are well accepted as a major contributor to movement of invasive species. Seeds and reproductive parts can be picked up by tires, attach themselves to vehicles, and be relocated many miles away. Off-road vehicles are even more of a problem as a invasive weed vector. Off-road vehicles may be traveling over public lands, thru or near weed infestations and frequently provide transportation necessary for weed migration and movement into previously undisturbed uninfested natural areas. Off-road vehicles can disturb natural plant communities, creating open spaces where exotic weeds can proliferate. The following is an excerpt from the Nature Trails and Water Coalition (<http://www.naturaltrails.org/issues/>): Weeds are carried across the landscape by the wind, water, wildlife, people and vehicles. While most vehicles disperse weeds along well-established transportation routes, dirt bikes, all-terrain vehicles (ATVs) and other off-road vehicles traveling cross country can spread invasive weeds over a wide area in only a few hours. A study in Montana demonstrated that a single ATV can disperse more than 2,000 invasive knapweed seeds over a 10-mile radius. The research also found that these seeds are more likely to germinate and crowd out native plants in areas where soil has been compacted by off-road vehicles. (Montana State University Extension Service, 1992). Research in Wisconsin in 2002 found that ATVs commonly transport a variety of weed seeds. This study concludes that ATVs could spread nearly 200 million seeds, many of them noxious weeds, statewide over the next 20 years. (Tom Rooney, University of Wisconsin). The BLM needs to include steps to prevent the spread of weeds by both vehicles and especially off-road vehicles as part of its weed management strategy. The PEIS must analyze the impacts that off-road vehicles are having on the spread of invasive weed and thus the potential success of the proposed actions.

## **Re-vegetation**

CATs promotes the re-vegetation of project areas with native forbs and grasses. Such an action will increase wildlife habitat values, promote soil stability, and provide competition against noxious weeds and undesirable woody species. Native forbs and grasses are often absent from the understory of intensively managed forests. The establishment of this vegetative component is critical in restoring our public lands to their historical conditions and preventing the occurrence and continued spread of noxious weeds.

CATs comments that establishment of native plants (specifically native perennials) will be crucial to the suppression of exotic plants and fuel reductions. Native perennial grasses have been observed as being capable of successfully competing against exotic annuals and “pest” shrubs, as well as being considered integral in achieving pre-European low intensity fire regimes (Belsky and Blumenthal 1997). The omission of re-vegetation, in the forms of grasses and forbs, from the proposed action will not achieve the desired conditions of a “biologically and structurally diverse forest” as described in the FEIS. The herbal layer will most likely consist of significant areas dominated by annual exotic grasses and weeds, which contribute to catastrophic fire, soil destabilization, and increased soil moisture loss. This will defeat the BLM’s very justification for the proposed actions. If the PEIS is not altered to avoid these consequences, an analysis of their effects must be undertaken due to the significant impacts that may be anticipated. This concern was not addressed in the PEIS.

## **Realistic goals (control vs. eradication)**

The goal of a control program could be to eradicate completely a plant everywhere, it could be to eradicate it only in a specific area, or it could be to reduce its population to a level that does not significantly displace native flora and fauna (Dahlsten et al. 1989). The Draft PEIS does not make this analysis. Furthermore, it does not provide an adequate system for making decisions for each site. How can BLM staff prepare NEPA documents in the future when they have no decision making guidance in the programmatic EIS?

Also missing is even a rudimentary analysis of how differences in climate, soil, topography and other factors will impact what treatments may be used and how efficacious they will be.

## **5. Cumulative Impacts**

The BLM has failed to analyze the cumulative impacts of annually spraying 932,000 acres with herbicides in the Draft PEIS. This vast amount of repeated herbicides spraying has the potential to cause significant harm to the natural environment, soils, water quality, native vegetation, wildlife, fish, and human health as it works its way through the food chain and web of life.

It is essential that the PEIS include analyses of the cumulative impacts, including not only those of the active ingredients, but also breakdown products, surfactants, inerts, adjuvants, additives, and everything else that will be entering our ecosystems as a result of herbicide applications.

Cumulative impacts analysis must include analysis of past, present, and future herbicide impacts.

## **6. Adequate information disclosure**

The Draft PEIS fails to inform the decision maker by making several very serious lapses in the description of the environmental consequences of the proposed program. The primary assumption is that for a programmatic EIS the BLM is absolved from analysis of the relative need for any of the means of control. The BLM can and must come up with, at the very least, pie charts and graphs that illustrate the proportion of each treatment option that may be anticipated to be used. This is not an impossible task, or if it is, why it is impossible should be described in the EIS. How can a decision maker be informed without this basic information?

The BLM fails to provide fundamental information for project analysis related to noxious weed and invasive weed treatments. How much extra spraying will be involved to treat noxious weeds? What portion of the annual BLM budget will go towards which weed treatment methods? In what areas will which herbicides be considered? Are there areas or situations where certain chemicals will not be considered acceptable? Where is the analysis of the extra spraying proposed for weeds regarding impacts and effects to watershed, vegetation, and wildlife?

Because the surveys and evaluations for the presence of invasive plants on BLM lands in the western US provided for the current analysis varies from district to district -- with some doing a good or even exemplary job, some barely getting by and others in-between -- no adequate determination of a range of values for presence of invasive plants can be made. Indeed, it isn't made, with only lists of invasive plants provided to guide the decision maker and no informative description of where and particularly HOW MUCH of the plants are currently present and how they are expected to spread given current knowledge. By not taking on the challenge of giving broad brush descriptions of this status, the DEIS leaves the future under any of the alternatives subject to uninformed speculation.

See Invasive Plants of Natural Habitats in Canada for examples of describing plant distribution and control methods ([http://www.cws-scf.ec.gc.ca/publications/inv/cont\\_e.cfm](http://www.cws-scf.ec.gc.ca/publications/inv/cont_e.cfm)).

A ranking system was developed for alien plants in Indiana. This system was used to set control and management priorities, and it evaluated alien plants on their: significance of impact (highly ranked species occur in high quality natural areas or have large populations that invade and replace natural communities), innate ability to be a pest (highly ranked species are highly fecund, have specialized dispersal abilities, and germinate in a wide range of environmental conditions), and feasibility of control (highly ranked species are

widely distributed, have extensive seed banks, and require high levels of mechanical or chemical control) (Hiebert and Klick 1988). Point Pelee, in southern Ontario, Canada, has been invaded by a number of invasive aliens. Dunster (1990) developed a set of criteria to assess the priority for removal of these invasive plants. The criteria included: aggressiveness, reproductive success, ability to hybridize with native plants, showiness, extent of populations, and location in sensitive habitats.

To compound the situation, the Draft PEIS does not describe the various methods for controlling the most problematic plant species, whether they are currently or anticipated to become the most problematic. Not all plant groups respond the same to herbicide application, for example. Some may be knocked back only to resurrect in a year or two. Others require considerably more herbicide and stronger adjuvants than "usual" before a dose is lethal.

An analysis at the programmatic level, for example, could describe plants by the characteristics that most influence the ability and the means to control it by providing guidance such as the following: "Herbicides will not be the control method of choice for invasive plants that spread by profuse seed production such as purple loosestrife, et cetera (listing other species with this characteristic on western BLM lands). These seeds are relatively long-lived and germinate sporadically, therefore the seed bank of an established population of such a plant is at little risk since it is not affected by a control program that removes only the current year's standing crop of growing plants. Plants should be removed before they go to seed and the entire plant including all roots and root tips must be removed. Plant locations should be flagged and rechecked every year."

Or, for example: "Fire is an effective option for the control of garlic mustard (*Alliaria petiolata*) in wooded areas (Nuzzo et al., 1991)."

Or, for example: "A prescribed burn is the recommended method for removing reed canary grass (Hutchinson 1999). Repeated late spring or late autumn burning of the reed canary grass is necessary because seeds of native plant species are present in the soil in and around it. Fire will allow native, fire-adapted plants to compete successfully. Hand removal is not feasible, herbicides are not selective enough, and heavy machinery would not destroy the hearty underground rhizomes. Annual burns may be necessary for 5-6 years. Seeding with native grasses and forbs after reed canary grass has died or gone dormant can also hasten the recovery of native plant species."

The type of control treatment for an invasive plant may involve special considerations for species that are now using the invasive plant as habitat. This has occurred in the case of endangered subspecies of the Southwestern Willow Flycatcher, which is now nesting in the highly invasive salt cedar in some areas of Arizona and New Mexico. See the University of California's website <http://groups.ucanr.org/saltcedar/Grant/Introduction.htm> for further discussion of how decisions were made regarding the use of biological controls in this situation. How will future decision makers take into account adaptations of native species to invasive plants, and how are they to deal with these the possible displacement of these other species since guidance is not provided in the programmatic DEIS?

## **SCIENTIFIC HONESTY AND RATIONALITY**

The agency must use high quality information and accurate scientific analysis, 40 C.F.R. 1500.1(b), and must disclose "any responsible opposing view." Id. 1502.9(b). The EIS must disclose and analyze opposing opinions. *Center For Biological Diversity v. United States Forest Service*, 349 F.3d 1157 (9th Cir. 2003). Thus far the BLM has failed to disclose opposing scientific opinion regarding toxicity of herbicide formulations, potential impacts of herbicide applications, and potential alternatives, thus violating this NEPA requirement.

## Conclusion

Without a description of which species are most problematic, what their response is to various treatment options, what their current and anticipated scope of invasion is or what are the various regional influences, the DEIS fails to provide the evaluation necessary for informing the public and making an informed decision. For all the DEIS tells us regarding the influences of the environment on the vegetation management program, we could guess that the problem is occurring on Mars, or in the sands of Saudi Arabia, or perhaps in Florida or Nova Scotia. NEPA requires more than this of a programmatic EIS. Without adequate description of the problem and the RANGE of responses that may be taken, and with constant assurance that all that's necessary will be described at the more site-specific level in an EA or EIS, it is not possible to gain an adequate vision of what is in store under the various alternatives with the current DEIS. That is not consistent with the demands of NEPA. The analysis cannot be delayed to the future because those future NEPA documents must tier to this one, and without a solid basis in the programmatic EIS, those documents will fail. To put it plainly, such piece-mealing is patently illegal under NEPA. The decision maker and the public would have to scramble IN THE FUTURE to read every project proposal and NEPA document that flows from the current EIS to piece together a picture of the extent of the program, the priority given to particular species, the treatment options most likely to be employed, and what effects regional differences may make in the approach to controlling invasive plants. This is one of the primary failures of the DEIS as it is currently written.

As it currently stands the BLM's PEIS is doomed to be unsuccessful without first focusing on the cause of the weed infestations and utilizing a holistic native species ecosystem health approach to combating exotic species. For that reason CATs supports the Restore Native Ecosystems Alternative (which should be added to the Final PEIS for consideration). Of the alternatives included in the Draft PEIS, we can only support Alternative C, the no pesticides alternative. Yet no alternative can be adopted until a fully informative NEPA document is prepared. As described above, this Draft PEIS does not achieve the informational standard required by NEPA. We urge you to correct these deficiencies in the Final PEIS.

CATs supports reduction of hazardous fuels and work to protect native biodiversity from exotic species. Yet we do not support unnecessary and excessive applications of pesticides in our public forest lands. The BLM has a responsibility to answer the questions raised in these comments and provide adequate and complete analysis of herbicide formulation toxicity and impacts, including evaluation of a full range of alternative management methods.

CATs would like to thank the BLM in advance for regarding our comments. Please consider the issues discussed above prudently. We look forward to your responses to the public comment period.

Sincerely,

/s/ Patty Clary  
Executive Director  
Californians for Alternatives to Toxics

/s/ Pete Harrison  
Public Lands Associate  
Californians for Alternatives to Toxics

/s/ Julia Olson  
Staff Attorney  
Californians for Alternatives to Toxics

/s/ Dan Zimmerman

Environmental Investigator

/s/ Scott Greacen  
National Forests Coordinator  
Environmental Protection Information Center

## **BIBLIOGRAPHY and REFERENCES**

### **Part One: Herbicide Issues**

#### Inadequate toxicological analyses

Ackermann et al 2002. Effects of long-term nonylphenol exposure on gonadal development and biomarkers of estrogenicity in juvenile rainbow trout *Oncorhynchus mykiss*. *Aquat Toxicol.* 2002 Oct 30;60(3-4):203-21.

Ahmed, SA 2000; The immune system as a potential target for environmental estrogens (endocrine disrupters): a new emerging field; *Toxicology*, vol. 150, no. 1-3, pp. 191-206, 7 Sep 2000

Aoki et al, 2004. Nonylphenol enhances apoptosis induced by serum deprivation in PC12 cells. *Life Sci.* 2004 Mar 19;74(18):2301-12.

Atienzar et al 2002, 4-n-Nonylphenol and 17-beta estradiol may induce common DNA effects in developing barnacle larvae. *Environ Pollut.* 2002;120(3):735-8.

Balasubramanian et al, 2001, Inhibition of cytochrome P450 reductase expression in testes of nonylphenol (NP)-exposed rats. *Toxicologist* 2001 Mar;60(1):73

Baldwin et al, 1997; Metabolic androgenization of female *Daphnia magna* by the xenoestrogen 4-nonylphenol; *Environmental Toxicology and Chemistry*; 16 (9). 1997. 1905-1911.

Bettinetti & Provini 2002; Toxicity of 4-nonylphenol to *Tubifex tubifex* and *Chironomus riparius* in 28-day whole-sediment tests. *Ecotoxicol Environ Saf.* 2002 Sep;53(1):113-21.

Bevan et al 2001, Defects in embryogenesis and growth factor responsiveness induced by exposure to endocrine disrupting compounds. [/cgi-bin/sis/search/r?./temp/\u126~8zP9Jr:@and+@au+@term+Porter+DAbstr Soc Neurosci 2001;27\(Pt 1\):658](#)

Bevan et al 2003; Environmental estrogens alter early development in *Xenopus laevis*. *Environ Health Perspect.* 2003 Apr;111(4):488-96.

Boone MD, Semlitsch RD, 2001. Interactions of an insecticide with larval density and predation in experimental amphibian communities. *Conserv Biol* 15:228–238

Boone MD, Semlitsch RD, 2002. Interactions of an insecticide with competition and pond drying in amphibian communities. *Ecol Appl* 12:307–316

Boone MD, Bridges CM 1999. The effect of temperature on the potency of carbaryl for survival of tadpoles of the green frog (*Rana clamitans*). *Environ Toxicol Chem* 18:1482–1484

Burkhardt-Holm, et al 2000. Nonylphenol Affects the Granulation Pattern of Epidermal Mucous Cells in Rainbow Trout, *Oncorhynchus mykiss*. *Ecotoxicol. Environ. Saf.*, vol. 46, no. 1, pp. 34-40, May 2000,

Bulayeva 2004; Xenoestrogen-Induced ERK-1 and ERK-2 Activation via Multiple Membrane-Initiated Signaling Pathways, *Environ Health Perspect* 112:1481–1487 (2004).

Canesi et al, 2004. Environmental estrogens can affect the function of mussel hemocytes through rapid modulation of kinase pathways. *Gen Comp Endocrinol*. 2004 Aug;138(1):58-69.

CEPANP, 2001. Environment Canada, Canadian Environmental Protection Act, 1999, Priority Substances List Assessment Report, Nonylphenol and its Ethoxylates.

Chitra KC, et al. 2002; Effect of nonylphenol on the antioxidant system in epididymal sperm of rats. *Arch Toxicol*. 2002 Sep;76(9):545-51. Epub 2002 Jun 25.

Christian, M and G Gillies. 1999. Developing hypothalamic dopaminergic neurones as potential targets for environmental estrogens. *Journal of Endocrinology* 160:R1-R6

Colborn et al, 1993. Developmental Effects of Endocrine-disrupting Chemicals in Wildlife and Humans. *Environmental Health Perspectives*. Vol 101 No. 5

Colborn T. 1995. How Research has Succeeded and Failed to Translate Science into Policy: Endocrinological Effects on Wildlife. *Environmental Health Perspectives*. Vol 103 No. 6

Colborn T 2005. Neurodevelopment and Endocrine Disruption. *Environ Health Perspect*. 112(9):1670–1682.

Coldham 1998, Biotransformation, tissue distribution, and persistence of 4-nonylphenol residues in juvenile rainbow trout (*Oncorhynchus mykiss*). *Drug Metabolism and Disposition* Vol. 26, No. 4.

Cook et al 1993. Investigation of a mechanism for Leydig cell tumorigenesis by linuron in rats. *Toxicol. Appl. Pharmacol* 1993; 119: 195-204.

Czech P, Weber K, Dietrich DR. 2001: Effects of endocrine modulating substances on reproduction in the hermaphroditic snail *Lymnaea stagnalis* L. *Aquat Toxicol*. 2001 Jul;53(2):103-14.

Daruich J, Zirulnik F, Gimenez MS. 2001. Effect of the herbicide glyphosate on enzymatic activity in pregnant rats and their fetuses. *Environ Res* 85:226–231.

DiCorcia, et al. 2000. Occurrence and Abundance of Dicarboxylated Metabolites of Nonylphenol Polyethoxylate Surfactants in Treated Sewages. *Environ. Sci. Technol*. 34, 3914-3919.

Dreze V et al 2000; Effects of 4-Nonylphenol on Sex Differentiation and Puberty in Mosquitofish (*Gambusia holbrooki*); *Ecotoxicology*, 9 (1-2): 93-103, April 2000

Edginton, et al 2004. Comparative effects of pH and Vision herbicide on two life stages of four anuran amphibian species. *Environmental Toxicology and Chemistry* 23:815–822.

Epel, D. 1990. The initiation of development at fertilization. *Cell Differ. Dev*. 29, 1-12.

EU G2. European Union Priority listing of Endocrine Disruptors. Group 2, Evidence of potential endocrine disruption.

EU RA 2002. European Union Risk Assessment Report. 4-Nonylphenol (Branched) and Nonylphenol. Office for Official Publications of the European Communities; L – 2985 Luxembourg. Risk-Assessment Report Vol.10, 2002 on: 4-nonylphenol (branched), CAS#: 84852-15-3, EINECS#: 284-325-5. and: nonylphenol, CAS#: 25154-52-3, EINECS#: 246-672-0. Publication: EUR 20387 [http://ecb.jrc.it/DOCUMENTS/Existing-Chemicals/RISK\\_ASSESSMENT/REPORT/4-nonylphenol\\_nonylphenolreport017.pdf](http://ecb.jrc.it/DOCUMENTS/Existing-Chemicals/RISK_ASSESSMENT/REPORT/4-nonylphenol_nonylphenolreport017.pdf)

Fan et al. 1995; Risk assessment of environmental chemicals. *Ca EPA. Annu Rev Pharmacol Toxicol* 1995;35:341-68

Fent et al, 2000, Analysis of vitellogenin mRNA by quantitative reverse transcription polymerase chain reaction (RT-PCR) in juvenile fish exposed for 12 months to nonylphenol, *Marine Environmental Research [Mar. Environ. Res.]*, vol. 50, no. 1-5, p. 193, Dec 2000 ISSN 0141-1136

Ferguson, SA; Scallet, AC; Flynn, KM; Meredith, JM; Schwetz, BA, 2000, Developmental Neurotoxicity of Endocrine Disrupters: Focus on Estrogens, *Neurotoxicology*, vol. 21, no. 6, pp. 947-956, Dec 2000, ISSN 0161-813X

Fox, JE, et al, 2001; Nitrogen fixation: Endocrine disrupters and flavonoid signalling. *Nature*, 2001, 413(13 Sept 01):128-129.

Fukamachi et al, 2004. Possible enhancing effects of atrazine and nonylphenol on 7,12-dimethylbenz[a]anthracene-induced mammary tumor development in human c-Ha-ras proto-oncogene transgenic rats. *Cancer Sci.* 2004 May;95(5):404-10.

Funabashi et al, 2004. p-Nonylphenol, 4-tert-octylphenol and bisphenol A increase the expression of progesterone receptor mRNA in the frontal cortex of adult ovariectomized rats. *J Neuroendocrinol.* 2004 Feb;16(2):99-104.

Giesy, et al 2000. Ecotoxicological risk assessment for Roundup herbicide. *Review of Contamination and Toxicology* 167:35–120.

Hahn T, Schenk K, Schulz R 2002; Environmental chemicals with known endocrine potential affect yolk protein content in the aquatic insect *Chironomus riparius*. *Environ Pollut.* 2002;120(3):525-8.

Hecht S, Boese BL. 2002; Sensitivity of an infaunal amphipod, *Eohaustorius estuarius*, to acute waterborne exposures of 4-nonylphenol: evidence of a toxic hangover. *Environ Toxicol Chem.* 2002 Apr;21(4):816-9.

Hemmer et al, 2001; Effects of p-nonylphenol, methoxychlor, and endosulfan on vitellogenin induction and expression in sheepshead minnow (*Cyprinodon variegatus*); *Environmental Toxicology and Chemistry [Environ. Toxicol. Chem.]*, vol. 20, no. 2, pp. 336-343, Feb 2001 ISSN 0730-7268

Hill RL Jr, Janz DM. 2003; Developmental estrogenic exposure in zebrafish (*Danio rerio*): I. Effects on sex ratio and breeding success. *Aquat Toxicol.* 2003 May 29;63(4):417-29.

Howe, et al 2004. Toxicity of glyphosate-based pesticides to four North American frog species. *Environmental Toxicology and Chemistry* 23:1928–1938.

Huang RK, Wang CH, 2001; The effect of two alkylphenols on vitellogenin levels in male carp; *Proc Natl Sci Counc Repub China B* 2001 Oct;25(4):248-52

Iwata et al, 2004. The endocrine disruptors nonylphenol and octylphenol exert direct effects on T cells to suppress Th1 development and enhance Th2 development. *Immunol Lett.* 2004 Jun 15;94(1-2):135-9.

Kang et al 2003. Effects of 4-nonylphenol on reproduction of Japanese medaka, *Oryzias latipes*. *Environ Toxicol Chem.* 2003 Oct;22(10):2438-45.

Karrow et al, 2004. Nonylphenol alters the activity of splenic NK cells and the numbers of leukocyte subpopulations in Sprague-Dawley rats: a two-generation feeding study. *Toxicology.* 2004 Mar 15;196(3):237-45.

Khan et al 2003; Alkylphenol endocrine disrupters inhibit IP3-sensitive Ca<sup>2+</sup> channels. *Biochem Biophys Res Commun.* 2003 Oct 17;310(2):261-6.

- Kim et al 2005. Effects of 2,4-D and DCP on the DHT-Induced Androgenic Action in Human Prostate Cancer Cells. *Toxicological Sciences* 2005 88(1):52-59;
- King et al, 2003. Effects of intraperitoneal and aqueous exposures of nonylphenol on smoltification in juvenile Pacific salmon. Proceedings from the recent Society of Environmental Toxicology and Chemistry's 24th Annual Meeting. 11 November 2003
- Kinnberg et al, 2000. Concentration-dependent effects of nonylphenol on testis structure in adult platyfish *Xiphophorus maculatus*. *Mar Environ Res.* 2000 Jul-Dec;50(1-5):169-73.
- Kleinow et al, 2004. Inhibition of P-glycoprotein transport: a mechanism for endocrine disruption in the channel catfish? *Mar Environ Res.* 2004 Aug-Dec;58(2-5):205-8.
- Kudo et al, 2004. Nonylphenol induces the death of neural stem cells due to activation of the caspase cascade and regulation of the cell cycle. *Journal of Neurochemistry*, Volume 88 Issue 6 Page 1416 - March 2004
- Kullman et al, 2004. Analysis of medaka cytochrome P450 3A homotropic and heterotropic cooperativity. *Mar Environ Res.* 2004 Aug-Dec;58(2-5):469-73.
- Kwack SJ, et al. 2002; Comparative evaluation of alkylphenolic compounds on estrogenic activity in vitro and in vivo. *J Toxicol Environ Health A.* 2002 Mar;65(5-6):419-31.
- Kwak et al, 2001. Effects of nonylphenol, bisphenol A, and their mixture on the viviparous swordtail fish (*Xiphophorus helleri*). *Environ Toxicol Chem.* 2001 Apr;20(4):787-95.
- Kyselova et al, 2003. Effects of p-nonylphenol and resveratrol on body and organ weight and in vivo fertility of outbred CD-1 mice. *Reproductive Biology and Endocrinology* 2003, 1:30; 24 March 2003
- Lajmanovich, et al 2003. Induction of mortality and malformation in *Scinax nasicus* tadpoles exposed to glyphosate formulations. *Bulletin of Environmental Contamination and Toxicology* 70:612– 618.
- LeBlanc et al 2000, Embryotoxicity of the Alkylphenol Degradation Product 4-Nonylphenol to the Crustacean *Daphnia magna* *Environ Health Perspect*, 108(12): 1133-1138; Dec 2000
- Lee PC. 1998; Disruption of male reproductive tract development by administration of the xenoestrogen, nonylphenol, to male newborn rats. *Endocrine* 1998 Aug;9(1):105-11
- Lee et al. 2003. Antiandrogenic effects of bisphenol A and nonylphenol on the function of androgen receptor. *Toxicol Sci* 75:40–46.
- Lussier et al, 1999; Acute Toxicity OF Para-nonylphenol TO Saltwater Animals. *Environmental Toxicology and Chemistry*: Vol. 19, No. 3, pp. 617-621.
- Mackenzie et al, 2003. Gonadal differentiation in frogs exposed to estrogenic and antiestrogenic compounds. *Environ Toxicol Chem.* 2003 Oct;22(10):2466-75.
- Mann, R. M., and J. R. Bidwell. 1999. The toxicity of glyphosate and several glyphosate formulations to four species of southwestern Australian frogs. *Archives of Environmental Contamination and Toxicology* 26:193–199.
- Marc et al. 2002. Pesticide Roundup provokes cell division dysfunction at the level of CDK1/cyclin B activation. *Chem Res Toxicol* 15:326–331.
- Marc et al 2005. A glyphosate-based pesticide impinges on transcription. *Toxicology and Applied Pharmacology* 203 (2005) 1 – 8

- Marcial et al, 2003. Estrogenic compounds affect development of harpacticoid copepod *Tigriopus japonicus*. *Environ Toxicol Chem.* 2003 Dec;22(12):3025-30.
- Masuno et al, 2003. Effect of 4-nonylphenol on cell proliferation and adipocyte formation in cultures of fully differentiated 3T3-L1 cells. *Toxicol Sci.* 2003 Oct;75(2):314-20. Epub 2003 Jul 25.
- Masuo et al, 2004. Effects of neonatal treatment with 6-hydroxydopamine and endocrine disruptors on motor activity and gene expression in rats. *Neural Plast.* 2004;11(1-2):59-76.
- Matozzo et al 2003, Evaluation of 4-nonylphenol toxicity in the clam *Tapes philippinarum*. *Environ Res.* 2003 Mar;91(3):179-85.
- Meregalli, G; Pluymers, L; Ollevier, F; 2001; Induction of mouthpart deformities in *Chironomus riparius* larvae exposed to 4-n-nonylphenol. *Environmental Pollution [Environ. Pollut.]*, vol. 111, no. 2, pp. 241-246, 2001
- McLachlan JA.2001; Environmental signaling: what embryos and evolution teach us about endocrine disrupting chemicals. *Endocr Rev* 2001 Jun;22(3):319-41 □McLachlan JA.
- Mitchell et al 1987. Acute toxicity of Roundup and Rodeo herbicides to rainbow trout, chinook, and coho salmon. *Bull. Environ. Contam. Toxicol.* 39, 1028
- Negishi et al. 2003. Inhibition of staurosporine-induced neuronal cell death by bisphenol A and nonylphenol in primary cultured rat hippocampal and cortical neurons. *Neurosci Lett* 353:99–102.
- Negishi et al, 2004; Behavioral Alterations in Response to Fear-Provoking Stimuli and Tranlycypromine Induced by Perinatal Exposure to Bisphenol A and Nonylphenol in Male Rats; *Environ Health Perspect.* 2004 Aug, 112:1159–1164.
- Nice, HE, D Morritt, M Crane and M Thorndyke. 2003. Long-term and transgenerational effects of nonylphenol exposure at a key stage in the development of *Crassostrea gigas*. Possible endocrine disruption. *Marine Ecology Progress Series* 256:293-300.
- Ohtani-Kaneko 2002, Endocrine disruptors influence synaptogenesis in primary cultures of fetal hypothalamic cells. *Environmental Sciences: an International Journal of Environmental Physiology and Toxicology* 2002;9(2-3):204-5
- Perkins, P. J., H. J. Boermans, and G. R. Stephenson. 2000. Toxicity of glyphosate and triclopyr using the frog embryo teratogenesis assay—*Xenopus*. *Environmental Toxicology and Chemistry* 19:940–945.
- Pickford KA, et al. 2003; Route of exposure affects the oestrogenic response of fish to 4-tert-nonylphenol. *Aquat Toxicol.* 2003 Nov 19;65(3):
- Porter, A. and Hayden, N. 2001. Processes Affecting the Fate of Nonylphenol During Wastewater Treatment: In Proceedings of 2nd International Conference on Pharmaceuticals and Endocrine Disrupting Chemicals in Water, The National Groundwater Association, October 9-11, 2001, Minneapolis, MN.
- Relyea and Mills, 2001. Predator-induced stress makes the pesticide carbaryl more deadly to grey treefrog tadpoles (*Hyla versicolor*). *Proceedings of the National Academy of Sciences, USA* 98:2491–2496.
- Relyea 2003. Predator Cues and Pesticides: a Double Dose of Danger for Amphibians. *Ecological Applications*, 13(6), 2003, pp. 1515–1521
- Relyea 2005a. The Lethal Impacts of Roundup and Predatory Stress on Six Species of North American Tadpoles. *Arch. Environ. Contam. Toxicol.* 48, 351–357 (2005)
- Relyea 2005b. The Impact of Insecticides and Herbicides on the Biodiversity and Productivity of Aquatic

Communities. *Ecological Applications*, 15(2), 2005, pp. 618–627

Relyea 2005c. The lethal impact of roundup on aquatic and terrestrial amphibians. *Ecological Applications*, 15(4), 2005, pp. 1118–1124

Richards et al 2005. Differential Effects of Glyphosate and Roundup on Human Placental Cells and Aromatase. *Environ Health Perspect* 113:716–720 (2005).

Sato K, Matsuki N, Ohno Y, Nakazawa K. 2002; Effects of 17beta-estradiol and xenoestrogens on the neuronal survival in an organotypic hippocampal culture. *Neuroendocrinology*. 2002 Oct;76(4):223-34.

Scallet AC 2001. Endocrine influences on developmental neuroanatomy. *Neurotoxicology* 2001 Feb;22(1):130-1

Scallet et al, 2001; Estrogenic disruption of hypothalamic development in rats. *Neurotoxicol Teratol* 2001 May-Jun;23(3):292

Schwaiger J, et al. 2002; How estrogenic is nonylphenol? A transgenerational study using rainbow trout (*Oncorhynchus mykiss*) as a test organism. *Aquat Toxicol*. 2002 Sep 24;59(3-4):177-89.

Scott-Fordsmand JJ, Krogh PH. 2004. The influence of application form on the toxicity of nonylphenol to *Folsomia fimetaria* (Collembola: Isotomidae). *Ecotoxicol Environ Saf*. 2004 Jul;58(3):294-9.

See, HJ, S Chattopadhyay, E-Y Gong, RS Ahn, and K Lee. 2003. Antiandrogenic Effects of Bisphenol A and Nonylphenol on the Function of Androgen Receptor. *Toxicological Sciences*. 75(September):40-46.

Seike et al 2003, Enhancement of lung carcinogenesis by nonylphenol and genistein in a F344 rat multiorgan carcinogenesis model. *Cancer Lett*. 2003 Mar 20;192(1):25-36.

Seki M, et al. 2003; Effects of 4-nonylphenol and 4-tert-octylphenol on sex differentiation and vitellogenin induction in medaka (*Oryzias latipes*). *Environ Toxicol Chem*. 2003 Jul;22(7):1507-16.

SERA 2002. Neurotoxic, Immunotoxic, and Endocrine Disruption with Specific Commentary on Glyphosate, Triclopyr, and Hexazinone: Final Report. Syracuse Environmental Research Associates, Inc. Unpublished Data

Servos MR, 1999; Review of the Aquatic Toxicity, Estrogenic Responses and Bioaccumulation of Alkylphenols and Alkylphenol Polyethoxylates. *Water Quality Research Journal of Canada*, 34(1): 123-177

Sheehan et al 1999. No threshold dose for estradiol-induced sex reversal of turtle embryos: how little is too much? *Environ Health Perspect*; VOL 107, ISS 2, 1999, P155-9

Smith, G. R. 2001. Effects of acute exposure to a commercial formulation of glyphosate on the tadpoles of two species of anurans. *Bulletin of Environmental Contamination and Toxicology* 67:483–488.

Sohoni P, Sumpter JP. 1998. Several environmental oestrogens are also anti-androgens. *J Endocrinol* 158:327–339.

Sone et al 2004. Effects of 17beta-estradiol, nonylphenol, and bisphenol-A on developing *Xenopus laevis* embryos. *Gen Comp Endocrinol*. 2004 Sep 15; 138(3):228-236.

Tanaka, Y; Nakanishi, J; 2001; Life history elasticity and the population-level effect of p-nonylphenol on *Daphnia galeata*. *Ecological Research [Ecol. Res.]*, vol. 16, no. 1, pp. 41-48, Mar 2001

Tanaka Y, and Nakanishi J 2002. Chronic effects of p-nonylphenol on survival and reproduction of *Daphnia galeata*: multigenerational life table experiment. *Environ Toxicol*. 2002 Oct;17(5):487-92.

Teles et al, 2004 Juvenile sea bass biotransformation, genotoxic and endocrine responses to beta-

naphthoflavone, 4-nonylphenol and 17beta-estradiol individual and combined exposures. *Chemosphere*. 2004 Oct;57(2):147-58.

Thibaut R, Monod G, Cravedi JP. 2002; Residues of 14C-4n-nonylphenol in mosquitofish (*Gambusia holbrooki*) oocytes and embryos during dietary exposure of mature females to this xenohormone. *Mar Environ Res*. 2002 Sep-Dec;54(3-5):685-9.

Tsui, M. T., and L. M. Chu. 2003. Aquatic toxicity of glyphosate-based formulations: comparison between different organisms and the effects of environmental factors. *Chemosphere* 52:1189–1197.

Uguz C, Iscan M, Erguven A, Isgor B, Togan I. 2003; The bioaccumulation of nonylphenol and its adverse effect on the liver of rainbow trout (*Onchorynchus mykiss*). *Environ Res*. 2003 Jul;92(3):262-70.

Weber LP, Hill RL Jr, Janz DM. 2003; Developmental estrogenic exposure in zebrafish (*Danio rerio*): II. Histological evaluation of gametogenesis and organ toxicity. *Aquat Toxicol*. 2003 May 29;63(4):431-46.

Welshons, WV, KA Thayer, BM Judy, JA Taylor, EM Curran and FS vom Saal. 2003. Large effects from small exposures. I. Mechanisms for endocrine disrupting chemicals with estrogenic activity. *Environmental Health Perspectives* doi:10.1289

WHO, 2002; Global Assessment of the State-of-the-Science of Endocrine Disruptors. International Programme On Chemical Safety. (Damstra, T, S Barlow, A Bergman, R Kavlock and G Van Der Kraak (editors)). [http://www.who.int/pcs/emerg\\_site/edc/global\\_edc\\_TOC.htm](http://www.who.int/pcs/emerg_site/edc/global_edc_TOC.htm)

USEPA TRI. USEPA Toxics Release inventory.

Verslycke et al, 2004. Testosterone and energy metabolism in the estuarine mysid *Neomysis integer* (Crustacea: Mysidacea) following exposure to endocrine disruptors. *Environ Toxicol Chem*. 2004 May;23(5):1289-96.

Villeneuve DL, et al. 2002; Effects of waterborne exposure to 4-nonylphenol on plasma sex steroid and vitellogenin concentrations in sexually mature male carp (*Cyprinus carpio*). *Chemosphere*. 2002 Apr;47(1):15-28.

Vivacqua et al, 2003. The food contaminants bisphenol A and 4-nonylphenol act as agonists for estrogen receptor alpha in MCF7 breast cancer cells. *Endocrine*. 2003 Dec;22(3):275-84.

Walsh LP, McCormick C, Martin C, Stocco DM. 2000. Roundup inhibits steroidogenesis by disrupting steroidogenic acute regulatory (StAR) protein expression. *Environ Health Perspect* 108:769–776.

Weber LP, Hill RL Jr, Janz DM. 2003; Developmental estrogenic exposure in zebrafish (*Danio rerio*): II. Histological evaluation of gametogenesis and organ toxicity. *Aquat Toxicol*. 2003 May 29;63(4):431-46.

Weiss B. 1998. A Risk Assessment Perspective on the Neurobehavioral Toxicity of Endocrine Disruptors. *Toxicol Ind Health*; VOL 14, ISS 1-2, P341-59

Welshons, WV, KA Thayer, BM Judy, JA Taylor, EM Curran and FS vom Saal. 2003. Large effects from small exposures. I. Mechanisms for endocrine disrupting chemicals with estrogenic activity. *Environmental Health Perspectives* doi:10.1289

WHO, 2002; Global Assessment of the State-of-the-Science of Endocrine Disruptors. International Programme On Chemical Safety. (Damstra, T, S Barlow, A Bergman, R Kavlock and G Van Der Kraak (editors)).

Williams GM, Kroes R, Munro IC. 2000. Safety evaluation and risk assessment of the herbicide Roundup and its active ingredient, glyphosate, for human. *Regul Toxicol Pharmacol* 31:117–165.

Wu F, Safe S, 2004. Comparative Activation Of Estrogen Receptor alpha (ERalpha) And ERalpha/Sp1 In Breast Cancer Cells By Xenoestrogens. *Toxicologist* 2004 Mar;78(1-S):120

Yokota et al 2001 Life-cycle toxicity of 4-nonylphenol to medaka (*Oryzias latipes*). *Environ Toxicol Chem.* 2001 Nov;20(11):2552-60.

Yousef MI, Salem MH, Ibrahim HZ, Helmi S, Seehy MA, Bertheussen K. 1995. Toxic effects of carbofuran and glyphosate on semen characteristics in rabbits. *J Environ Sci Health B* 30:513–534

Yu Z, et al. 2003; Estrogenic activity of some environmental chemicals; Sheng Yan Jiu. 2003 Jan;32(1):10-2.

Zaga et al. 1998; Photoenhanced toxicity of a carbamate insecticide to early life stage anuran amphibians. *Environ Toxicol Chem* 17:2543–255

Zhang L & Baer KN, 2001; The effects of 4-nonylphenol on reproduction and embryo development in *Daphnia magna*. *Toxicologist* 2001 Mar;60(1):163

Zhang 2003 The effects of 4-nonylphenol and ethanol on acute toxicity, embryo development, and reproduction in *Daphnia magna*. *Ecotoxicol Environ Saf.* 2003 Jul;55(3):330-7.

Zhang et al 2003a. Adverse effects of nonylphenol on the reproductive function of adult male SD rats. *Sichuan Da Xue Xue Bao Yi Xue Ban.* 2003 Apr;34(2):295-7.

Zumbado M, et al. 2002; Evaluation of acute hepatotoxic effects exerted by environmental estrogens nonylphenol and 4-octylphenol in immature male rats. *Toxicology.* 2002 Jun 14;175(1-3):49-62.

#### Impacts to pregnant women, fetuses, and infants

Sandra Steingraber. *Having Faith: An Ecologist's Journey To Motherhood.* Perseus Publishing .2001.

Schreinemachers, D. National Health and Environmental Effects Research Laboratory. 2003. *Environmental Health Perspectives*, Vol. 111, No. 9.

B.M. Blatter et al., “Paternal Occupational Exposure Around Conception and Spina Bifida in Offspring.” *Am. J. of Industrial Medicine* 32(1997): 283-91

A.M. Garcia et al., “Paternal Exposure to Pesticides and Congenital Malformations,” *Scandinavian J. of Work and Environmental Health* 24(1998): 473-80

A.F. Olshan et al, “Paternal Occupational Exposures and the Risk of Down Syndrome,” *American J. of Human Genetics* 44(1989):646-51

Pew Environmental Health Commission, *Healthy from the Start*, p.22-23

D.H. Poyner et al., “Paternal exposures and the Question of Birth Defects,” *J. of the Florida Medical Association* 84(1997):323-26

- P.G. Schnitzer et al., "Paternal Occupation and Risk of Birth Defects in Offspring," *Epidemiology* 6(1995):577-83
- Poyner et al., "Paternal Exposures"
- T. Nurminen, "Maternal Pesticide Exposure and Pregnancy Outcome," *J. of Occupational and Environmental Medicine* 37(1995):935-40
- A.S. Rowland, "Pesticides and Birth Defects," *Epidemiology* 6(1995):6-7
- T. Nurminen et al., "Agricultural Work During Pregnancy and Selective Structural Malformations in Finland," *Epidemiology* 6(1995):23-30
- Schettler et al. *In Harm's Way: Toxic Threats to Child Development*. 2000. Greater Boston Physicians for Social Responsibility.
- U.S. Department of Health and Human Services. Agency for Toxic Substances and Disease Registry. 2002. Case Studies in Environmental Medicine (CSEM) □ Pediatric Environmental Health □ The Child as Susceptible Host: A Developmental Approach to Pediatric Environmental Medicine.
- A.M. Garcia et al., "Parental Agricultural Work and Selected Congenital malformations," *Am J. of Epidemiology* 149(1996):64-74
- J. Garcia-Rodriguez et al., "Exposure to Pesticides and Cryptorchidism: Geographical Evidence of a Possible Association," *EHP* 104(1996): 1090-95
- I.S. Weidner et al., "Cryptorchidism and Hypospadias in Sons of Gardeners and Farmers," *EHP* 106(1998):793-96
- P. Kristensen et al., "Birth Defects Among Offspring of Norwegian Farmers, 1967-1991," *Epidemiology* 8(1997):537-44
- E.M. Bell et al., "A Case-Control Study of Pesticides and Fetal Death Due to Congenital Anomalies," *Epidemiology* 12(2001):148-56
- V.F. Garry et al., "Pesticide Applicators, Biocides, and Birth Defects in Rural Minnesota," *EHP* 104(1996):394-99
- P.W. Nathanielsz, *Life Before Birth: The Challenges of Fetal Development* (New York: W.H. Freeman, 1996)
- B.M. Carlson, *Human Embryology and Developmental Biology*, 2d ed. (St. Louis: Mosby, 1999), p. 208-48.
- England, *Life Before Birth*, p. 51-70
- H.L. Needleman and D. Bellinger, eds., *Prenatal Exposure to Toxicants: Developmental Consequences* (Baltimore: Johns Hopkins University Press, 1994), p.89-111
- Carlson, *Human Embryology*, p. 208-48
- G.J. Harry, ed., *Developmental neurotoxicology* (Boca Raton: CRC Press, 1994), p. 1-7.

E. M. Faustman et al., "Mechanisms Underlying Chilfern's Susceptibility to Environmental Toxicants," *EHP* 108(2000, sup. 1):13-21

P.M. Rodier, "Comparative Postnatal Neurologic Development," in Needleman and Bellinger, *Prenatal Exposure to Toxicants*, p. 3-23

*In Harm's Way: Toxic Threats to Child Development* (Cambridge: Greater Boston Physicians for Social Responsibility, 2000), p. 23-28

## **Part two: Invasive species issues**

Harper, G. and R. Whitehead. 1994. Evaluation of forest vegetation community dynamics, biodiversity and wildlife forage values 8 years after herbicide and manual brushing treatments on the Bush River Trial site. Working Plan. E.P. 1179. B.C. Min. For., Research Branch, Victoria.  
[Available 1/2002 <http://www.for.gov.bc.ca/research/forprod/fordyn/projects/ep1179/ep1179htm.>]

Simard, Suzanne, Heineman, Jean, and Youwe, Phil. 1998. Brushing and Grazing Effects on Lodgepole Pine, Vascular Plants, and Range Forage in Three Plant Communities in the Southern Interior of British Columbia: Nine-Year Results. LMH 45. B.C. Ministry of Forests, Forestry Division Services Branch. Upper McKay Creek Study pgs. 35-45.  
[Available 1/2002 <http://www.for.gov.bc.ca/hfd/pubs/Docs/Lmh/Lmh45.htm>]

Belsky, A. Joy and Dana M. Blumenthal. 1997. Effects of Livestock Grazing on Stand Dynamics and Soils in Upland Forests of the Interior West. *Conservation Biology* Volume 11, Number 3, April.

Brooks, M.L., C.M. D'Antonio, D.M. Richardson, J.M. DiTomaso, J.B. Grace, R.J. Hobbs, J.E. Keeley, M. Pellant, D. Pyke. 2004. Effects of invasive alien plants on fire regimes. *Bioscience* 54:677-688.

D'Antonio, Carla M., Eric L. Berlow, and Karen A. Haubensak. 2002. Invasive exotic plant species in Sierra Nevada ecosystems. *Proceedings from the Sierra Science Conference 2002: Science for management and conservation.*

Zouhar, Kris. 2003. *Bromus tectorum*. In: Fire Effects Information System, [Online]. U.S. Department of Agriculture, Forest Service, Rocky Mountain Research Station, Fire Sciences Laboratory (Producer). Available: <http://www.fs.fed.us/database/feis/> [2006, January 6].

McDonald, Philip M., Everest, Glen A. 1996. Response of young ponderosa pines, shrubs, and grasses to two release treatments. Research Note PSW-RN-419-Web. Albany, CA: Pacific Southwest Research Station, Forest Service, U.S. Department of Agriculture; 8 p.  
[Available 11/2001 [http://www.psw.fs.fed.us/Tech\\_Pub/Documents/rn-419/McDonald419.html](http://www.psw.fs.fed.us/Tech_Pub/Documents/rn-419/McDonald419.html)]

McGinnis, Thomas W. April, 2005. Personal Communication. U.S. Geological Survey, Biological Resources Discipline, Western Ecological Research Center, Sequoia-Kings Canyon National Park.

McGinnis, Thomas W., Jon E. Keeley, Matt Brooks, Robert Sanford, Jayne Belnap. 2005. The Response of Cheatgrass (*Bromus tectorum* L.) and Native Flora to Ecological Manipulations in the Yellow Pine-Mixed Conifer Forest. USGS Research Project. Unpublished.

- Schwartz, Mark W., Daniel J. Porter, John M. Randall, and Kelly E. Lyons. 1996. Impact of Nonindigenous Plants. Sierra Nevada Ecosystem Project: Final report to Congress, Volume II, Assessments and Scientific Basis for Management Options. Davis: University of California, Centers for Water and Wildland Resources.
- Skinner, Carl N. and C. Chang. 1996. Fire Regimes, Past and Present. Sierra Nevada Ecosystem Project: Final report to Congress, Volume II, Assessments and Scientific Basis for Management Options. Davis: University of California, Centers for Water and Wildland Resources.
- Weed Research Information Center (WRIC). University of California Extension Service. Yellow Star-thistle Information: <http://wric.ucdavis.edu/yst/index.html>.
- Wooten, G. and M. Renwyck. 2001. Risky Business: Invasive Species Management on National Forests. Kettle Range Conservation Group. Spokane, Washington. [Available 12/2005 <http://www.kettlerange.org/weeds/>]
- Young, J.A., Clements, D.D. 2005. Species Diversity In Cheatgrass Communities. Society For Range Management Meeting Proceedings.
- Agee, J.K., 1996. The influence of forest structure on fire behavior. In: Proceedings of the 17<sup>th</sup> Forest Vegetation Management Conference. Redding, CA, pp 52-68.
- Agee, J.K., 2000. The use of shaded fuelbreaks in landscape fire management. Forest Ecology and Management 127, 55-66.
- Beaty, R.M., Taylor, 2001. A.H. Spatial and temporal variation of fire regimes in a mixed conifer forest landscapes, Southern Cascades, California, USA. J. Biogeography 28, 955-966.
- Bekker, M.F., Taylor A.H. 2001. Gradient analysis of fire regimes in montane forests of the Southern Cascade Range, Thousand Lakes Wilderness, California, USA. Plant Ecology 155, 15-28.
- Conrad, S.G., Radosevich, S.R. 1982. Post-fire succession in white fir (*Abies concolor*) vegetation of the northern Sierra Nevada. Madroño 29. 42-56.
- Countryman, C. C. 1955. Old-growth conversion also converts fireclimate. In: Proceedings of Society of American Foresters Annual Meeting, 158-160. Portland, OR: Society of American Foresters.
- Graham, Russell T., Alan E. Harvey, Threasa B. Jain, and Jonalea R. Tonn. 1999. The effects of thinning and similar stand treatments on fire behavior in western forests. General Technical Report PNW-GTR-463. September.
- Stephens, Scott Lewis. 1998. Evaluation of the effects of silvicultural and fuels treatments on potential fire behaviour in Sierra Nevada mixed-conifer forests. Forest Ecology and Management 105, 21-35.
- Taylor, A.H. 2000. Fire regimes and forest changes in mid and upper montane forests of the Southern Cascades, Lassen Volcanic National Park, California, U.S.A. J. of Biogeography 27.87-104.
- van Wagtenonk, Jan W. 1996. Use of a Deterministic Fire Growth Model to Test Fuel Treatments. In: Sierra Nevada Ecosystem Project: Final report to Congress, Volume II, Assessments and scientific basis for management options. Davis: University of California, Centers for Water and Wildland Resources. Chapter 43.

Weatherspoon, C. Phillip. 1996. Fire-silviculture relationships in Sierra forests. In: Sierra Nevada Ecosystem Project: Final report to Congress, Volume II, Assessments and scientific basis for management options. Davis: University of California, Centers for Water and Wildland Resources. Chapter 44.

DiTomaso, J. 2001. Yellow Starthistle Information. Weed Research and Information Center, The University of California. Available from: <http://wric.ucdavis.edu/yst/yst.html>

Drlik, T., I. Woo, and S. Swiadon, Editors. 1998. Integrated vegetation management technical bulletin: spotted, diffuse, and Russian knapweed. Bio-Integral Resource Center, Berkeley, CA. 19pp.  
Carpenter, T. Alan. 1998. Element Stewardship Abstract for Tamarix spp. The Nature Conservancy, Arlington, VA. Available <http://tncweeds.ucdavis.edu/esadocs/tamaramo.html>

Zouhar, Kris. 2003. Tamarix spp. In: Fire Effects Information System, [Online]. U.S. Department of Agriculture, Forest Service, Rocky Mountain Research Station, Fire Sciences Laboratory (Producer). Available <http://www.fs.fed.us/database/feis/plants/tree/tamspp/introductory.html>

Beck, K.G. 2005. Diffuse and Spotted Knapweed. Colorado State University Cooperative Extension – Natural Resources. <http://www.ext.colostate.edu/pubs/natres/03110.html>

California Department of Food and Agriculture, Integrated Pest Control Branch. Weed information sheet. Spotted knapweed, Diffuse knapweed, Squarrose knapweed. <http://www.cdfa.ca.gov/phpps/ipc/weedinfo/centaurea.htm>

Carpenter, Alan and Thomas Murray. *Centaurea diffusa* Lamarck, diffuse knapweed. Element Stewardship Abstract. The Nature Conservancy. Arlington, Virginia. <http://tncweeds.ucdavis.edu/esadocs/documents/centdif.html>

Carpinelli, Michael. 2003. Spotted Knapweed. Plant Conservation Alliance, Alien Plant Working Group. <http://www.nps.gov/plants/alien/fact/cebil.htm>

Drlik, T., I. Woo, and S. Swiadon, Editors. 1998. Integrated vegetation management technical bulletin: spotted, diffuse, and Russian knapweed. Bio-Integral Resource Center, Berkeley, CA. 19pp.

Engeland, Ron. 1988. Hand-Pulling of Diffuse and Spotted Knapweed. Columbia River Bioregional Education Project. Okanogan County Noxious Weed Control Board. Columbia Publishing, Oroville, WA.

Mauer, Teresa. 1987. *Centaurea maculosa* Spotted Knapweed. Element Stewardship Abstract. The Nature Conservancy. Arlington, Virginia. <http://tncweeds.ucdavis.edu/esadocs/documts/centmac.html>

U.S. Department of Agriculture. Biological Control of Spotted and Diffuse Knapweeds. Animal and Plant Health Inspection Service. Program Aid Number 1529. <http://aphis.usda.gov/oa/pubs/knapwpub.pdf>

Waldo, Amy Jo. 2001. Knapweed...Identification and Control. Four common knapweeds found in Central Oregon. Oregon State University Extension Service. <http://extension.oregonstate.edu/deschutes/knapweed.pdf>

California Department of Food and Agriculture, Integrated Pest Control Branch. Encycloweed website. Scotch broom, French broom, Spanish broom. <http://www.cdfa.ca.gov/phpps/ipc/weedinfo/brooms.htm>.

- Fuller, T.C., and G.D. Barbe. 1985. The Bradley method of eliminating exotic plants from natural reserves. *Fremontia* 13(2):24-25.
- Huckins, Eddie and Jonathan Soll. 2004. Controlling Scotch (Scots) Broom (*Cytisus scoparius*) in the Pacific Northwest. The Nature Conservancy. The Global Invasive Species Initiative. <http://tncweeds.ucdavis.edu/moredocs/cytsco01.pdf>.
- Hoshovsky, Marc. 1986. Element Stewardship Abstract for *Cytisus scoparius* and *Genista monspessulanus*, Scotch Broom, French Broom. The Nature Conservancy, Arlington, Virginia. <http://tncweeds.ucdavis.edu/esadocs/documnts/cytisco.pdf>.
- Parker, B., G. Miller, and L.C. Burrill. 1998. Scotch Broom, *Cytisus scoparius* (L.) Link. A Pacific Northwest Extension Publication. PNW 103. <http://eesc.orst.edu/agcomwebfile/edmat/pnw103.pdf>.
- Raj, Prasad. 2002. Scotch Broom, *Cytisus scoparius* L. in British Columbia. Natural Resources Canada. [http://www.pfc.cfs.nrcan.gc.ca/biodiversity/broom\\_e.html](http://www.pfc.cfs.nrcan.gc.ca/biodiversity/broom_e.html).
- California Department of Fish and Game (CDFG). 2000. Weed Control by Species: Elkhorn Slough National Estuarine Research Reserve. *Phalaris aquatica* or Harding grass. pg. 44-45. Available: [www.elkhornslough.org/plants/weeds.PDF](http://www.elkhornslough.org/plants/weeds.PDF).
- Holloran, P., Mackenzie, A., Farrell, S., Johnson, D. 2004. The Weed Workers' Handbook. A Guide to Techniques for Removing Bay Area Invasive Plants. Harding Grass, *Phalaris aquatica*. The Watershed Project and California Invasive Plant Council. pg. 42, 94-95. Available: [http://groups.ucanr.org/ceppc/WW\\_Handbook/](http://groups.ucanr.org/ceppc/WW_Handbook/)
- Huselid-Glass, Jesse and Miguel Hernandex. 2004. Invasive Weeds of the Salinas Creeks. Harding grass, *Phalaris aquatica*. Return of the Natives, Restoration Education Project. Available: [http://essp.csumb.edu/internships/internships/HandOuts/Jesse\\_H\\_Glass\\_weed%20manual.pdf](http://essp.csumb.edu/internships/internships/HandOuts/Jesse_H_Glass_weed%20manual.pdf).
- Peterson, David L. 1988. Element Stewardship Abstract for *Phalaris aquatica*, Harding Grass. The Nature Conservancy. Arlington, Virginia. Available: <http://tncweeds.ucdavis.edu/esadocs/phalaqua.html>.
- California Department of Food and Agriculture, Integrated Pest Control Branch. Weed information sheet. Dalmatian toadflax. <http://www.cdffa.ca.gov/phpps/ipc/weedinfo/linaria.htm>
- Carpenter, Alan and Thomas Murray. 1988. *Linaria genistifolia* (L.), Dalmatian toadflax. Element Stewardship Abstract. The Nature Conservancy. Arlington, Virginia. <http://tncweeds.ucdavis.edu/esadocs/documents/linadal.html>
- Hansen, Rich. Dalmatian Toadflax, Yellow Toadflax. Biological Control: A Guide to Natural Enemies in North America. Cornell University. <http://www.nysaes.cornell.edu/ent/biocontrol/weedfeeders/toadflax.html>
- Lajeunesse, Sherry. 2004. Dalmatian and Yellow Toadflax. Butte-Silver Bow Local Government. Montana Weed Control Association. [http://www.co.silverbow.mt.us/weeds/dalmatian\\_and\\_yellow\\_toadflax.htm](http://www.co.silverbow.mt.us/weeds/dalmatian_and_yellow_toadflax.htm)
- Moser, L., and D. Crisp. 2001. Dalmatian Toadflax, *Linaria genistifolia* spp. *dalmatica* (L.).

San Francisco Peaks Weed Management Area fact sheet on *Linaria dalmatica*. Coconino National Forest. [http://www.usgs.nau.edu/swepic/factsheets/lidasf\\_info.pdf](http://www.usgs.nau.edu/swepic/factsheets/lidasf_info.pdf)

Washington State Noxious Weed Control Board. 2003. Dalmatian toadflax (*Linaria dalmatica* (L.) P. Mill. ssp. *dalmatica*). [http://www.nwcb.wa.gov/weed\\_info/dyerswoad.html](http://www.nwcb.wa.gov/weed_info/dyerswoad.html)

Zouhar, Kris. 2003. *Bromus tectorum*. In: Fire Effects Information System, [Online]. U.S. Department of Agriculture, Forest Service, Rocky Mountain Research Station, Fire Sciences Laboratory (Producer). Available: <http://www.fs.fed.us/database/feis/> [2006, February 10].

California Department of Food and Agriculture, Integrated Pest Control Branch. Weed information sheet. Dyer's woad. <http://www.cdfa.ca.gov/phpps/ipc/weedinfo/isatis.htm>

Kedzie-Web, S., R. Sheley, and S. Dewey. 1996. Dyer's Woad: A Threat to Rangeland in Montana. Montguide. MT 9614. Montana State University, Bozeman, Montana.

Andrascik, Roger, Terry Cacek, Robert Doren, Lissa Fox, Ron Hiebert, Gary Johnston. 1996. A Strategic Plan for Managing Invasive Nonnative Plants on National Park System Lands. U.S. Department of the Interior, National Park Service. Available [Feb 2006]: [http://www.nature.nps.gov/biology/invasivespecies/strat\\_pl.cfm](http://www.nature.nps.gov/biology/invasivespecies/strat_pl.cfm)

Archer, Amy J. 2001. *Taeniatherum caput-medusae*. In: Fire Effects Information System, [Online]. U.S. Department of Agriculture, Forest Service, Rocky Mountain Research Station, Fire Sciences Laboratory (Producer). Available: <http://www.fs.fed.us/database/feis/> [2005, October 5].

(CDFA) California Department of Food and Agriculture. Integrated Pest Management Branch. Encycloweedia. Italian thistle. <http://www.cdfa.ca.gov/phpps/ipc/weedinfo/carduus.htm>

Pitcher, Don. 1986. Italian Thistle, Element Stewardship abstract for *Carduus pycnocephalus*. The Nature Conservancy, Arlington Virginia. <http://tncweeds.ucdavis.edu/esadocs>

Washington State Noxious Weed Control Board. Italian thistle. [http://www.nwcb.wa.gov/weed\\_info/written\\_findings/carduus\\_pyconocephalus.html](http://www.nwcb.wa.gov/weed_info/written_findings/carduus_pyconocephalus.html)

USDA Forest Service. 2000. Noxious Weed Management Strategy. Pacific Southwest Region. Prepared by Cheri Rohrer and James R. Shackelford. Recommended by Stephen Paulson. Approved by John Neisess.

Lassen National Forest. 2005. Creeks Forest Health Recovery Project. Final Environmental Impact Statement. USDA Forest Service. Pacific Southwest Region. Almanor Ranger District. Pg. B-5.

Clark, Janet. 2003. Invasive Plant Prevention Guidelines. Center For Invasive Plant Management. Bozeman, Montana. Available [February 2006]: [www.weedcenter.org](http://www.weedcenter.org).

California Department of Food and Agriculture, Integrated Pest Control Branch. Weed information sheet. Musk thistle. <http://www.cdfa.ca.gov/phpps/ipc/weedinfo/carduus.htm>

California Department of Food and Agriculture, Integrated Pest Control Branch. Encycloweedia website. Scotch broom, French broom, Spanish broom. <http://www.cdfa.ca.gov/phpps/ipc/weedinfo/brooms.htm>.

Huckins, Eddie and Jonathan Soll. 2004. Controlling Scotch (Scots) Broom (*Cytisus scoparius*) in the Pacific Northwest. The Nature Conservancy. The Global Invasive Species Initiative.

<http://tncweeds.ucdavis.edu/moredocs/cytsco01.pdf>.

Hoshovsky, Marc. 1986. Element Stewardship Abstract for *Cytisus scoparius* and *Genista monspessulanus*, Scotch Broom, French Broom. The Nature Conservancy, Arlington, Virginia.

<http://tncweeds.ucdavis.edu/esadocs/documnts/cytisco.pdf>.

Raj, Prasad. 2002. Scotch Broom, *Cytisus scoparius* L. in British Columbia. Natural Resources Canada.

[http://www.pfc.cfs.nrcan.gc.ca/biodiversity/broom\\_e.html](http://www.pfc.cfs.nrcan.gc.ca/biodiversity/broom_e.html).

Graham, Russell T., Harvey, Alan E., Jain, Theresa B., Tonn, Jonalea R. 1999. The effects of thinning and similar stand treatments on fire behavior in Western forests. Gen. Tech Rep. PNW-GTR-463. Portland, OR. U.S. Dept. of Ag. Forest Service, Pacific Northwest Research Station. 27p. Available [February 2006]:

<http://www.treearch.fs.fed.us/pubs/2979>.

Merriam, K.E., J.E. Keeley, and J.L. Beyers. 2005. The Role of Fuel Breaks in the Invasion of Nonnative Plants. Technical Report. U.S. Geological Survey, Western Ecological Research Center, Sequoia and Kings Canyon Field Station, California. (in preparation). Available [February 2006]:

<http://www.werc.usgs.gov/fire/seki/ffm/products.htm>.

Nature Trails and Water Coalition. 2002. Invasive Weeds Threaten Wildlands, Economic Health. Issues/Fact Sheets. Available [February 2006]: <http://www.naturaltrails.org/issues/>.

Dahlsten, Donald L., Richard Garcia, and Hilary Lorraine. 1989. Eradication as a Pest Management Tool: Concepts and Contexts. In: Eradication of Exotic Pests. Donald L. Dahlsten and Richard Garcia (eds.). Yale University Press. Pg. 3-15.