CATs Californians For Alternatives To Toxics

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Re: Headwaters Forest Acquision and the Draft Palco Habitat Conservation Plan and Sustained Yeild Plan and Supporting Draft Environmental Impact Report/Environmental Impact Statement

These comments are submitted on behalf of Californians for Alternatives to Toxics, Environmental Protection Information Center and the Center for Ethics and Toxics and are tiered, horizontally and vertically, to the comments of those organizations and others participating in this National Environmental Policy Act(NEPA)/California Environmental Quality Act (CEQ) procedure.

General Comments

CAT-1

The concept that a Sustained Yield Plan (SYP) and a Habitat Conservation Plan (HCP) could be incorporated in one document then submitted as The Plan along with an aquision agreement, then analyzed in a joint federal/state NEPA/CEQA procedure and still be in plain English that can be read and grasped by the interested and at least moderately well-educated member of the public is ludicrous on the face of it and impossible in practice. But, still, your agencies could have made a much better attempt and at least succeeded to some degree. Federal documents do exist which are written in clear and plain English and structured in a form that can be followed by a significant portion of the public. Yours does not fit in the same catagory.

Besides WHAT these documents contain is the problem of how they are structured. The lack of an understandable, straight-forward construction and of a keyed and corresponding link between the documents, so that the reader can relate back and forth between Plan and Analysis, creates a process of analysis that is so cumbersome and convoluted that all but the most dedicated, persistant and resource-rich members of the public are locked out.

Incorporated with these comments are sections of the recently published federal Drug

Enforcement Agency's Supplement EIS for Cannabis Eradication in the Contiguous United States and Hawaii and the Final EIS for Vegetation Management for Reforestation for the US Department of Agriculture, Forest Service Region V, published in 1988. Both EISs are programatic and describe complex programs, particularly the latter. Each is head and shoulders above yours in providing readers with plain English and a broad scope of issues as well as a fairly in-depth analysis. These are informative documents, though each has significant shortcomings. For the DEA's EIS, a more throrough analysis will occur at the state level before implimentation of the program; the FS EIS is a decade old and deficient of much relevant science that has been developed since its publication. Yet both documents are far and away superior to what you and Palco have prepared. CAT-1

The EIR/EIS should be site and species specific regarding the impacts of herbicide and other potential pesticide use because it is not programatic on the continental level, as is the DEA EIS, or regionally, as is the FS EIS. It supports information about a much more specific environment with site specific variations that must be considered

Within the documents there exist continual contradictions, unfinished thoughts, assumptions based on thin air rather than science, statements made to stand alone without documented evidence and large gaps of information that render the HCP/SYP and EIS/EIR incomprehensible, unfounded and inadequate to the law. It strikes the reader throughout purusal of the EIS/EIR that every effort is made to support the alternative that Palco created prior to the NEPA documents.

Nowhere is this more evident than in the sections dealing with the use of pesticides, including herbicide application, the one potential pesticide application you acknowledge but fall short of analyzing. Other reasonably forseeable uses of pesticide in the course of the permit and the environmental consequences of those uses are completely ignored.

Pesticide Definitions

At least some of the problems you have regarding descriptions of past and future pesticide use are demonstrated in the one definition of pesticides we were able to find. You apparently don't know what pesticides are.

3.4-19 EIR/EIS Pesticides are NOT merely "...chemicals used to control undesirable animal pests, while herbicides are chemicals used to control unwanted plants." CAT-2

As described by CA Department of Pesticide Regulation (DPR 1998), pesticides are "In California, any of the following: any spray adjuvant, any substance, or mixture of substances which is intended to be used for defoliating plants, regulating plant growth, or for preventing, destroying, repelling, or mitigating any pests which may infest or be detrimental to vegetation, man, animals, or households, or be present in any agricultural or nonagricultural environment. Includes fungicides, herbicides, insecticides, nematicides, rodenticides, desiccants, defoliants, plant growth regulators."

Providing such a wildly inaccurate and misleading definition of the word "pesticide" isn't too surprising to us; throughout the HCP/SYP EIR/EIS (the Documents), your position is that herbicides, though integral to the SYP, are not part of the HCP and not really subject to analysis. Because the treatment of pesticides in the EIR/EIS, as it arises from your definition, minimizes the potential consequences of past and future pesticide applications, it bias the EIS/EIR in favor of the preferred alternative. CAT-2 CON.

Neither pest control nor pesticide experts were consulted in the course of developing the Documents.

p 91– 92 Vol 1 HCP/SYP The Plan Preparation Team list does not include one pesticide/pest control specialist. CAT-3

p 5-1 – 5-7 EIR/EIS The list of Persons and Organizations Consulted does not include any governmental agency, state or federal, that regulates the use of pesticides. Nor is any environmental/ public interest organization which focuses on pest control options and pesticide use, including our organization, listed as having been consulted. You did did not consult with any Native American non-governmental cultural organizations, including the California Indian Basketweavers Association, to which our organization belongs, and which has serious concerns about the use of pesticides where traditional hunting, fishing and gathering may take place (CIBA 1997). CAT-4

p6-1– 6.4 EIR/EIS In the List of Preparers,
Foster Wheeler Environmental Corporation does not list any pest control or pesticide expert. Under Other Contributors is one Thomas Reid, an "EIS technical reviewer and contributor to Herbicides..." who apparently has a B.S. in Chemistry and unspecified graduate studies to his credit, hardly qualification as an expert. No pest control expert participated. CAT-5

Since you have not involved the guidence of federal and state pesticide regulators, preparers who have broad toxicological and pest control experiential and educational qualifications, or expert environmental organizations you have minimized the significance of pesticide use as part of the Plan, permitting and the National Environmental Policy Act (NEPA) process and skewed the analysis in favor of the preferred alternative.

Critique of 3.14 Herbicides of the EIR/EIS of the HCP/SYP

It is difficult to criticize that which is not there, but we are forced to do so because of the absolute lack of any meaningful description of the risks entailed with the use of herbicides on PL's land. What you do say defies science and common sense. We will restrict most of our comments to most of the most glaring omissions and inaccuracies. You must know you're in trouble on this one.

3.14-1 In the introduction, you say "Palco is seeking incidental take authorization for effects to covered species that may result from the use of herbicides...herbicide use is a component of the SYP and...long-term sustained yield depends on it ...Herbicide use on the PALCO onership is subject to a separate regulatory process and could continue CAT-6

even with the No Action alternative."

And again at 3.14-9 you say "Because herbicide use is not subject to the current permitting process, herbicide use could continue under the No Project, No Action alternative unless it was determined to result in a [sic] unauthorized take of a listed species."

CAT-6 CON.

This is a contradiction of the first degree and, as a result, highly confusing to even the most interested and educated participant. **Either Palco is seeking an incidental take permit for effects from herbicides OR herbicides are not a part of the permitting process.** Which is it? To put it mildly, just this one contradiction is a fatal flaw for the herbicide section of the EIS because the most fundamental question is not transparent, and it must be to satisfy NEPA.

It appears that perhaps at the last moment, when all was wrapped and ready to go, someone realized that herbicide effects would have to be included in the take permit, and so it was spliced in. But at that point there wasn't time enough to go through the HCP/SYP and specify herbicide use under all the various elements of the various alternatives, so the Plans remain almost completely devoid of even the mention of herbicides and the EIR/EIS is full of fatal flaws which bias the EIR/EIS in favor of the preferred alternative. Such as this one.

At p 4 Part G Justification of Calibration in the HCP/SYP, herbicide use is described as integral to Intensive Management. Thus you are bound to describe herbicide use in the Plan <u>even if there wasn't a significant likelyhood of a take as a result of its effects</u>, but you don't. As quoted earlier at p 3.14-1, you say "...long term sustained yield depends on it..."

You cannot say that it is not part of the permitting process because you have conceded that the use of herbicides could be construed as contributing to a take. You <u>must</u> deal with herbicide use in the EIS/EIR and cannot rely on the notion that you "could have done it anyway."

<u>Jurisdiction regarding herbicide use for reforestation</u>

California Department of Forestry denies that it has jurisdiction over the use of herbicides by timber companies, but your description of herbicide use for reforestation (p 3.14.1.2 "For routine vegetation vegetation control after clear cut...) makes it clear that this use of herbicides is part of the Timber Harvest Plan, which is a functional equivalent of CEQA and thus should be regulated and subject to mitigation under the jurisdiction of CDF and the mandates of the Forest Practices Act. Isn't it true that CDF should be regulating the use of herbicides for reforestation that is undertaken in the course of fulfilling the requirements of a THP?

CAT-7

<u>Need for vegetation control</u>

3.14-1 You immediately launch into Affected Environment. You do not even make a stab at describing the conditions that would create a need for herbicide spraying,

CAT-8

although it is clear that when considering the amount and extent of herbicide use that appears to be proposed to support the SYP, herbicide use alone appears to amount to a program of its own. How can informed decisions be made about herbicide use if you don't describe the pest targets and the management goal? You need to describe the conditions that make herbicide spraying necessary in the first place. See the Forest Service EIS for Vegetation Control (1988) which contains a description of the effects of competition on various commercial tree species. We cannot find an adequate description in the SYP and for you to forgo it entirely in this section slants the EIS/EIR in favor of the preferred alternative. CAT-8

Affected environment

There is no description of the affected environment. Where are you spraying? When you spray for reclaiming hardwood areas from older unmanaged clear cut harvests, for example, it is unclear if you are converting from an indiginous oak forest to a conifer plantation, or if you need to spray thousands of acres of whitethorn that have filled an unmanaged, older clearcut. Where are the 20,000 acres (see below) of hardwoods located that are to be sprayed in the next decade? You need to provide a map of locations and describe which species are the target, what is the target's density, the techniques used to manage it (backpack spray, hack and squirt, et cetera), the proposed chemicals (include herbicide formulations and adjuvants, the amount proposed to be used per acre), environmental conditions at the sites (wetlands? steep slopes? nearness to streams? presence of species of concern?). CAT-9

Alternative means of vegetation control

Nor do you describe various alternatives for vegetation control, including but not limited to herbicides. If herbicides could cause a take of a species of concern or pollute water, injure workers or cause other advere effects, don't you need to describe other ways of controlling vegetation? Wouldn't a description of various alternatives include one about forgoing clearcut prescriptions to avoid undertaking vegetation control in the first place? See the Forest Service EIS for Vegetation Control (1988); many of the described alternatives seem silly ten years later, but that is only because today much more information is available about alternatives. The FS EIS document can serve as an example of the least that can be done to fulfill this requirement. CAT-10

Scope of the proposed activity

It would be useful to early in your description of the proposed activity to provide some context in which the reader can understand if the activity is large in scale or small. Knowing the number of acres and amount of chemicals that would be used in the course of the SYP would certainly be critical to understanding whether the use of herbicides is significant or trivial. Surely the amount of acreage and chemicals is critical to providing a basis to understanding if to what extent individuals of species of concern could be seriosly impacted. CAT-11

Yet, rather than dealing with this important topic early, you bury it later in the text. When we tried to figure out the scale of the proposed action and went looking for

information on the number of acres herbicides that would be used in, for example, the first decade, we found the DEIS to be misleading and confusing. Harvest levels, and as noted above, harvest intensity, is directly related to herbicide use. CAT-11 CON.

p 3.9-36 Clear cuts in the first decade are described as at 34,903 acres. [emph added]

p 3.4-19 Herbicides will be used "wherever intensive management takes place on PALCO lands."

3.14.4 Yet here you say that "Under the proposed SYP, harvest in the first decade would result in 34,720 acres of clearcut. Of this total, 80 percent, or 27,776 acres would be subject to a one-or two-year herbicide program..." [emph added]

You do not describe how you arrived at the 80% figure. Pulled it out of a hat? We can't find any specified description of what's sprayed and what won't be in the SYP; although vegetation control is noted just what that entails, herbicides or hand grubbing or other, is never specified. You need to describe why you think 20% of the clearcuts will not need herbicides.

At 3.14.4 it appears that you will be using herbicides on 2,000 acres per year for rehabilitation and 2, 800 acres per year for reforestation, or site preparation, which comes to a total of 4,800 acres per year. Thus, under this line of reasoning, the first decade alone of the SYP would entail herbicide use on 48,000 acres of land.

But a serious flaw in your calculations is that you count the 27,776 acres of intensively managed, herbicide treated clearcuts as being 'treated' only once, yet you state at 3.14.4 that those acres would be subject to a "one- or two-year program," which sounds as if it is an annual rate higher than it is now.

To confuse the issue further, earlier, at 3.14.1.2 you say "For clear cuts, this treatment will be done once in the first year and once again one to three years later." That sounds unequivical, and it now looks as if PL will be spraying 5,000 acres of new clear cut every year if the currently proposed SYP gets rolling. So really, it's an arbitrarily set 20,000 acres of hardwood recovery, an arbitrarily set 27,776 acres of first year clear cut reforestation and an arbitrarily set 27,776 acres of second or third year herbicide application. Now we're looking at a grand total of 75,552 acres of spraying over the course of the first decade alone of this plan. Do YOU have any idea of how much it will be?

p 3.4-51 And you agee, saying here that "Increased herbicide use would occur under this [the proposed] alternative because of the intensive harvest of PALCO lands."

Back at 3.14.4, how can you keep a straight face and say that "The area treated and the types of treatment could thus be similar to and slightly less than recent historical treatment" – and refer us to a table (3.24-2) (which doesn't exist as that number) – when you obviously haven't thought this one out.

We shouldn't have to accept your uninformative and unsupported "80%" and "one- or

two-year program" without knowing what you mean; this knowledge is crucial to making an informed decision under NEPA and an at least somewhat wise decision under CEQA.

CAT 11 CON.

You need to spell out in the EIR/EIS just how many acres will be harvested and how many will be sprayed in each decade. If 47,776 to 75,552 acres will be sprayed the first decade of the SYP, and less in later decades because of a move away from clear-cuts (as it appears at p 3.9-36), how much less acreage will be sprayed later? Does PL only spray clear-cuts or do they spray other forms of harvest like overstory removal? and how do they do it (backpack, hack and squirt?) – and if not, why not?

Let's not forget the amount of chemical that will be applied. You don't tell us how much you want to use per acre of the herbicides and adjuvants. For forest rehab you currently use (Table 3.14-2) about 0.55 gallon (ga) of Garlon 3 or 4, mixed with 0.14 ga of R-11 adjuvant and an undescribed amount of diesel – we have reason to believe diesel is used from 2 up to 20 ga per acre (see below), so let's err on the side of caution and estimate a use of 10 ga of diesel per acre. At 2,000 acres per year that comes to 1,100 gallons of Garlon 3 & 4, 280 ga R-11 and 20,000 ga of diesel. That's some serious chemical.

But what about reforestation? Erring on the side of caution again, we'll estimate that an average 4,500 acres will receive this 'treatment' most years, or a conservative estimate of (at slightly over 1 ga of tank mix per acre) of 4,725 ga of herbicide, including 25 ga of adjuvant per year.

Another way of looking at it reveals a conservative estimate of a total of 58, 250 gallons of herbicide in the first decade of the SYP, and 200,000 ga of diesel!

You describe a "substitution of Oust for most of the Aatrex application" at 3.14.2.4. As we describe later, we are not comforted by the low amount of sulfometuron methyl/Oust that is required to kill vegetation. Common sense tells us to be cautious of anything that is so very toxic, and science backs that up.

If PL wants a fifty year permit they need to detail their fifty year SYP. Each type of harvest and each target species that they want to spray needs to be described. They need to show a map of areas sprayed in each WAA (including that done by other companies) in the last decade and describe what sprays were used and how much used, so these figures can be compared to present and future use maps in each WAA for the purpose of defining possible cumulative impacts.

History of herbicide use on PL land

CAT-12

Under both guiding statutes for assessing this Aquision and the Plans, NEPA and CEQA, you must describe the history of herbicide use at this site and assess whether it has had a significant adverse effect, if species may have already been taken and whether persistant adverse effects could exacerbate adverse effects of future herbicide use when the effects are cumulative. You have not done so.

The only historical information you provide is Table 3.14-2, and we are not sure it is accurate. You say that PL used a total of 107 ga of glyphosate-containing products (Roundup and Accord). Our figures, drawn from the records stored at the County Agricultural Commissioner's office, indicate that PL actually used 288 ga of these herbicides.

CAT-12 CON.

Following is a chart of PL herbicide use from 1996 through 1998, taken from Pesticide Use Reports at the Humboldt County Agriculture Commissioner's Office. Attached with documents accompanying this letter are maps printed on clear material which may be overlain on map #1 "Headwaters Agreement" of Vol.V Maps and Illustrations of PL's HCP/SYP to gain an idea of where herbicides were applied in each of the years, and if all maps are overlain at once, an idea of which watersheds have been the most severely impacted.

In 1995, Pacific Lumber used (no map supplied by CATs)

Oust- Sulfometuron methyl	139.00 pounds	840 acres
used with Glyphosate (Roundup)	152.25 pounds	
and R-11 Spreader/Activator	39.28 gallons	
Garlon 4- Triclopyr, Butoxyethyl ester	130.00 gallons	314 acres
R-11 Spreader/Activator	32.50 gallons	
Garlon 4- Triclopyr, Butoxyethyl ester	288.00 gallons	452 acres
Aatrex 4L- Atrazine	234.00 gallons	275 acres
and Glyphosate (Roundup)	55.50 gallons	
and R-11 Spreader/Activator	13.80 gallons	
Total: Liquid herbicide	945.33 gallons	1,881 acres
dry herbicide	139.00 pounds	

In 1996 (see map)

5,849 acres were sprayed with:
116.4 pounds of Oust (sulfometuron methyl),
525.4 gallon of Round-up and Accord (glyphosate),
2,216.0 gallons of Aatrex 4L (atrazine),
1,260.9 gallons of Garlon 4 (triclopyr-ester form),
234.0 gallons of Garlon 3A (triclopyr-amine salt form),
147.1 gallons of R-11 (adjuvant),
and an unknown amount of diesel – at a minimum of 2 ga to a maximum 20 ga per acre, we estimate per acre use of 3,000 to 30,000 ga of diesel mixed with Garlon..

In 1997 (see map),

5,243 acres were sprayed with:
2,788.5 gallons of Aatrex 4L (atrazine),
1,512.4 gallons of Garlon 4 (triclopyr-ester form),
288.5 gallons of Round-up and Accord (glyphosate),

90.4 gallons of R-11 (adjuvant)
and an unknown amount of diesel – 3,000 to 30,000 ga.

CAT 12 CON.

In 1998(see map),
5,175 acres were sprayed with:
732.4 pounds of Oust (sulfometuron methyl),
574.4 gallons of Round-up (glyphosate),
730.3 gallons of Garlon 4 (triclopyr-ester form),
60.0 gallons of Garlon 3A (triclopyr-amine salt form)
and an unknown amount of diesel – 1,600 to 16,000 ga.

Unfortunately state regulatory agencies require only the legal description of the square mile section in which forestry herbicide applications take place, unlike all other agricultural applications for which locations must be site specific. You need to describe historical herbicide appliction with site specificity so that the impact on watersheds of this large use of herbicides can be assessed in the proper context.

Diesel

Tens of thousands of gallons of diesel have been sprayed on PL's holdings in the last four years. Because this application does not by law need to be reported to the Agricultural Commissioner any more than if it was water, PL has withheld the information. But this information cannot be withheld in an EIR/EIS because it is essential to an assessment of contamination as a result of historical use and of potential use. You need to describe all diesel use, including amounts used per acre, and where it has been stored and mixed on the land.

Atrazine

CAT-13

We describe atrazine here as an example of what you need to describe when assessing the potential for significant risk and takes of species of concern in an EIR/EIS. This description is in no way designed to limit the scope of the assessment you need to do either in terms of information or specific chemicals. Rather than analyzing all the chemicals PL intends to use, we examine atrazine and a few others as examples.

PL has relied heavily on the use of atrazine in the form of Aatrex 5% in previous years. We are very concerned that atrazine and the Aatrex formulation are not anywhere near adequately described in the EIR/EIS. As a result of selecting only some science to support your 'assessment' of atrazine, you minimize the potential for serious harm due to the use of this chemical, including cumulative effects that may be felt for years into the future. You inadequate portrayal of the toxic effects of atrazine and its formulations biases the EIS in favor of the preferred alternative.

In fact, peer reviewed scientific research, published in scientific journals and in governmental documents, reveals a different image of atrazine than the one you display.

Herbicides with atrazine as the active ingredient are sold under trade names of Atrazine, AAtrex, Atratol, Gesaprim, and Zeaphos. Atrazine is also a component of other herbicides such as Alazine, Bicep, Bullet, Exrtazine, Prozine, Rastra,Stuazine, and Tomahawk. (EPA OPA 1990) Thus it is important that you define exactly which formulation will be used in the course of the SYP, if any. If you do not tell us which formula is planned for use, there is a great deal of information that will not be available for assessing the impact of atrazine because not all the chemicals in the formula will be known and metabolites will be uncertain.

CAT-13 CON.

Mode of Action: Atrazine kills plants by binding to the cell membrane on which photosynthesis occurs and inhibiting photosynthesis.

Acute Toxicity: According to the National Institute for Occupational Safety and Health, atrazine is a mild skin irritant and a severe eye irritant. The oral LD50, the dose that will kill 50 percent of a population of test animals, is 672 milligrams per kilogram of body weight (mg/kg) in rats (US DHHS 1991). When you say at p3.14-13 that "Atrazine is considered non-toxic to slighly toxic to humans and other animals," you totally misstate the meaning of this ranking. LD50, the measurment at the basis of your statement, describes how much chemical is necessary for one-half of test subjects to die after one dose. This in no way describes chronic and other effects of atrazine which put it in the class of quite toxic to humans.

Chronic Toxicity: Chronic feeding studies in dogs demonstrated increased mortality, decreased food consumption and weight gain, increased liver, ovary and heart weights (in females) with related electrocardiographic changes in the heart accompanied by detectable pathology in both sexes (the NOEL for these effects was 15 ppm). (CDFA 1990) Chronic effects such as these need to be assessed in light of herbivorous mammals which are the food source of the carnivorous mammals and birds of the species of concern, such as the Northern spotted owl or the Humboldt marten. Martens and other mammals may eat grasses to relieve digestive problems and be exposed to atrazine or its metabolites by this route.

Atrazine also has been classified as an endrocrine disrupting chemical, meaning that it mimics hormones in the body and can upset normal functioning of the reproductive and other systems (Colburn 1995). Endocrine disruption has been described in many classes of organisms, including amphibians, fish, birds and mammals (Colburn)

Carcinogenicity: Tests of atrazine's ability to cause cancer in rats using technical atrazine (the active ingredient) found dose-related breast tumors in females and tumors in the testicles of males. The NOEL was 10 ppm in females and 500 ppm in males. (CDFA 1990) Another rat study found a dose related increase in combined leukemia/lymphoma (cancer of the lymph system) incidence, an increase in benign mammary tumors in males, and an increase in cancer of the uterus in females. (Pinter et al 1990,EPA OPA 1998)

In a study of hamster ovary cells, the results indicated that atrazine has the potential to cause whole cell clastogenicity, even at a very low concentration. (Biradar, Rayburn 1995).

In humans the use of triazine herbicides has been associated with an increase in tumors of the ovary. (Donna et al 1989.) Women previously exposed to triazines developed the tumors 2.7 times as frequently as unexposed women. A study of eastern Nebraska residents found that exposure to atrazine was associated with an elevated risk of another cancer, non-Hodgkin's lymphoma. (Weisenburger. 1990) Atrazine is classified by EPA as a possible human carcinogen (Class C) Based on the increased incidence of mammary tumors in female rats. (U.S. EPA Office of Public Affairs1990.)

CAT-13 CON.

The results of studies about atrazine's effects on mammals are of special concern regarding workers. You need to assess the impact to workers' health from exposure to atrazine, not simply ignore it as you have in the EIR/EIS.

Mutagenicity: Field tests with commercial atrazine products have demonstrated genotoxic effects on maize pollen, indicating that the chemical may induce gene mutations in other plants as well as other classes of organisims (Plewa et al.1984) What effect might this have on species of concern such as Sanicula tracyi or Bensoniella oregona if simazine is used nearby and less-than-lethal doses contaminate the non-target plant?

Reproductive Effects: Treatment of rat mothers with atrazine and one of its metabolites during pregnancy and nursing resulted in slow maturation of their offsprings sexual organs. As a consequence, pituitary activity was modified in both male and female offspring, and certain hormone receptors were strongly inhibited. (Kniewald et al 1987) If White footed voles are exposed to atrazine by contamination of their burrows when atrazine leaches through soil, what effect might this have on the reproductive capacity of voles?

Treatment of rat mothers with atrazine caused a reduction in the weight of their offspring (Infurna et al 1988).The NOEL for this effect was 0.5 mg/kg/day. Atrazine also produced dose-related pattern of toxicity in the mothers, including mortality at high doses and decreases in food consumption, body weight, and body weight gain. (ibid) The maternal NOEL was estimated to be 10 mg/kg per day. At high doses (700 mg/kg day) maternal mortality was 78 percent and clinical signs included salivation, bloody vulvae, and swollen abdomens. Rabbits also experience similar effects: bloody vulvae, reduction in feed consumption, body weight, and body weight gain in mothers. reduced fetal weights, increase skeletal abnormalities, and increases in embryo loss were associated with this maternal toxicity. The maternal NOEL was 1 mg/kg/day and the fetal NOEL was 5 mg/kg/day. (ibid)

Environmental Fate: At least eleven degradation products (metabolites) have been determined for atrazine.(DeLuca 1988; UM 1998)

Two of atrazine's metabolites, deethylatrazine and deisopropylatrazine have been characterized by atrazine manufacturer, Ciba-Geigy as having a toxicology that "can be assumed to be similar to that of atrazine." In some samples of groundwater, deethylatrazine has been measured at concentrations four times as high as the

concentration of atrazine. (Belluck1991)

Mobility in Soil: Problems with atrazine's persistence are compounded by its mobility in soil. A Connecticut study detected atrazine continuously form the surface to the water table at 7.5 feet. Finding residues at this level is significant because numerous studies have shown that microbial degradation is slower in the subsoils. Thus, herbicide residues detected in deep soils could be sources of leaching to groundwater for many years. (Huang 1989) Under no-tillage conditions, the leaching potential of atrazine was evidenced by concentrations greatly exceeding those in control plots in the lowest sampled depths (up to 1.5 m, almost 5 feet) at 14, 42, and 124 days. While the movement of agrochemicals through soil is influenced by soil properties, soil management, weather conditions, and degradation processes, in general, atrazine is expected to maintain a very high to medium mobility in soils and should not strongly adsorb to sediments.

CAT-13 CON.

One study found that the fate of atrazine in soil depends on complex interactions betwen mass flow, diffusion, hydrodynamic dispersion, routes of water and solutes in soil, pesticide stability, sorption on soil and metabolization by the soil microflora (Tasli et al). You need to look at the whole body of what is known about atrazine and compare it to the specific site conditions where atrazine has been used on PL land. Only then can assessments of its impacts be made.

Water Contamination: Atrazine is a common contaminant found in groudwater, rivers, high mountain lakes, drinking water supplies, rain, and even fog. (Glotfelty et al 1987) Under the safe Drinking Water Act. EPA establishes maximum contaminant levels (MCLs) for public water supplies. The MCL for atrazine is 3 mg/1 and is based on the NOEL of 0.5 mg/kg/day for reproductive effects in the rat study described earlier. Possible carcinogenic effects are not part of this study; EPA applies an uncertainty factor of 10 in its calculations of the MCL to account for these. (US EPA 1991) Estimates of the cancer risk posed by lifetime consumption of drinking water contaminated with atrazine at the MCL are approximately one excess cancer death per hundred thousand people exposed. (Belleck et al 1991) The MCL is set for a single chemical, and does not apply to metabolites. However, Midwestern groundwater contamination specialists have proposed that the levels should either apply to the total concentration of atrazine plus metabolites, or be made more stringent for atrazine alone. (ibid)

The National Pesticide Survey EPA (1991), atrazine was the second most detected pesticide, frequently exceeding the Survey's minimum reportng limits.

A USGS study found atrazine in the rainwater in all 23 of Eastern and Midwestern states sampled. While highest concentrations for atrazine were found where atrazine usage is highest, atrazine was also found in nonfarm areas. Highest concentrations were four to five mg/l, exceeding the MCL. (Goering 1991)

A study of rural ponds in Ontario, Canada found that atrazine was the most common contaminant. Eighty-nine percent of the contaminations were due to drift or runoff; the rest were due to spills. (Frank et al 1990.)

A Swiss study found triazine herbicides in all of the lakes tested, with atrazine being the major herbicide present. (Plewa et al 1984) Atrazine was also detected in rainwater during the warmer season. Volatilization and wind erosion of treated soil particles are theorized to be the cause of its presence in rain and in high mountain lakes. (Buser 1990)

CAT-13 CON.

CA DPR found atrazine in 7 wells of those it sampled in 1997. (DPR 1998) It is especially interesting that atrazine's metabolite 2-amino-4chloro-6-isopropylamino-s-triazine (DEA) was found in 40 wells, its metabolite 2-amino-4-choloro-6thylamino-s-triazine (ACET) was found in 37 wells, and its metabolite 2,4-diamino-6-chloro-s-truazine (DACT) was found in in 27 wells.

This is especially of interest because you describe at 3.14.2.2 that the Northcoast Regional Water Quality Control Board (NCRWCB) tested water for atrazine and didn't find any. But bcause you do not divulge the protocol or results of NCRWCB's tests you are not adequately informative. Were tests done of atrazine's metabolites? Did you return and test months after the application or did you test only at the first rain event?

This information is critical to an understanding of whether your contention (supported NCRWCQB based apparently on very little information) that "...ground application as practiced on PALCO land poses little threat to water quality..." (3.14.2.2) is accurate. This claim cannot be made without supporting evidence, and it needs to be comprehensive given the danger posed by the use of this chemical and other for vegetation control by PL.

"Inerts" and Contaminants: Hexachlorobenzene is a reaction side product in the production of atrazine. (Verschueren 1983) and Tetrachlorodibenzofuran may also be found in atrazine.

A special concern related to atrazine's presence in ground and surface water is the possible production of N-nitrosoatrazine (NNAT). N-Nitroso derivatives as a class are potent carcinogens and are formed by the reaction of an amine (atrazine, for example) with nitrates (from, for example, nitrogen fertilizers) (Wolfe et al 1976). NNAT has been formed form atrazine under laboratory conditions, (Wolfe et al 1976), is persistent in soil when no sunlight is present, and is a more soluble than atrazine in water and thus is a potential groundwater contaminant. (Wolfe et al 1976) Extensive groundwater contamination by nitrate and atrazine has been reported. (Weisenburger 1990) In hamsters, NNAT is a strong mutagen.

With all that is known of the adverse effects of atrazine to mammals due to both laboratory and human epidemiological studies, isn't it true that a risk assessment of the effects of mammalian species of concern would be warranted?

Ecosystem level studies have shown that a whole ecosystem can produce or experience the effects of the chemical that are difficult to identify when only a portion of the ecosystem is used for the assessment. (DeNoyelles et al 1989)

Fish species: Studies using experimental ponds show that as little as 20 ppb of atrazine significantly affects the diet and reproductive success of bluegill although the bluegill feeds on insects that would not be directly harmed by atrazine. (Kettle et al 1987) Atrazine also produced physiologic changes in carp.(Gluth & Hanke 1984) CAT-14

Furthermore, studies have shown that different segments of the renal tubule in rainbow trout react not only with different levels of sensitivity to atrazine exposure, but also in a segment-specific way. Moreover, comparison of effects induced by atrazine with those resulting from chronic exposure to to the herbicide linuron revealed a distinct substance specificity in the reaction of different tubular segments. (Oulmi et al 1994)

Short-term exposure to a relatively low concentration (5 & mgr;g/l) of atrazine can affect various behaviors of fish not only directly but also indirectly by altering the chemical perception of natural substances of eco-ethological importance (Saglio, Trijasse 1998). The authors of this study found that "In consideration of the basic role of olfaction in fish behavior, these results also emphasize the need for further developments on the possible effects of aquatic toxicants on olfactory-mediated behaviors."

This should raise a red flag for you. You have threatened fish species to consider such as the Coho, which may not . Nowhere in the HCP is any form of a discussion of how a low exposure to any of PL's herbicides is likely to cause problems to fish, yet for atrazine and the other chemicals we discuss in this letter, very significant effects have been observed by credible scientists in replicable studies. You need to analyze the potential for adverse effects of atrazine to fish or other species of concen such as the endangered Tidewater goby.

Other aquatic organisms: Cricket frogs from the wild were tested and an "assiciation approaching significance" was found between the detection of atrazine and intersex individuals." (Bulog 1996) Resistant species and biotypes have also been found in non-agricultural systems. In some of the experimental pond studies mentioned above, researchers found that after initial decreases in populations of phytoplankton, algae an vascular aquatic plants, communities often recovered in mass. However, there was a change in the community profile, and resistant species became dominant. (deNoyelles et al 1989, Kettle et al 1987, Dewey 1986) Because frog species are rapidly crashing around the world, wouldn't it be critical to understand the effects atrazine may have had and is having on amphibians in the watersheds of PL's holdings? CAT-15

Wetlands can be affected by discharge from atrazine use. In a study of wetland macrophtes it was demonstrated that the organisms may not tolerate herbicide contamination equally. (Langan & Hoagland 1996)

Since atrazine is highly mobile as well as persistant, atrazine or its metabolites are likely to have reached water in many instances on PL land, although it was not found in tests done there by Regional Water Quality Control. You did not inform us of the protocol of the atrazine tests nor did you share any other details of the tests you claim occured on streams running through PL's land holdings. We cannot assess the CAT-16

science used in these tests unless you provide a chart and description of the herbicide sampling. Given the huge amount that is known of atrazine's propensity to contaminate water, and the fact that it can move to water slowly and may be leaching into streams on PL's holdings now from sprays done a year or more ago, and coupled with the information we have of atrazine's adverse effects on fish and other organisms, it is critical that you undertake an assessment of the historical use of atrazine at this site in order that a well-informed decision can be made. CAT-16 CON.

Earthworms exposed to atrazine experienced weight loss, reproductive failure, and death. Atrazine has been shown to have strong negative effects on the total abundance of several species of insects at concentrations as low as 20 ppb. Species richness, total abundance of emerging insects, and the total number of herbivorous insects were all reduced in controlled tests. (Dewey1986) CAT-17

Plant Toxicity Resistance: As of 1983, more than two dozen weed species in Europe, Canada, and the U.S. were resistant to atrazine. Researchers found that a change in a single gene can account for the development of resistance. (Marx 1988) Not only is atrazine resistance spreading, but this resistance is associated with resistance to other herbicides. Atrazine-resistant species have also been found to be resistant to phenmedipham, lenacil, fenuron, and diuron. (Solymosi & Lehoczki 1988) CAT-18

Regulatory Issues: In 1990, EPA classified atrazine as a restricted use pesticide and made label revisions in an effort to reduce worker exposure and groundwater contamination. EPA took these steps in response to a voluntary request by the manufacturers, but also noted that additional action may be necessary to reduce groundwater contamination. (EPA/OPA 1990) CAT-19

EPA began a special review of atrazine in 1994 which "could result in use restrictions or even cancellation...all three of the triazines included in the Special Review... produce mammary tumors in some strains of rats. Recently published human studies offer...suggestive evidence that exposure...may contribute to the still-increasing rates of breast cancer in American women."

In 1996 EPA proposed state management plans as a condition for use of pesticides that pose a threat to ground water contamination, including atrazine, which have had "numerous detections indicating contamination above levels of concern established under EPA's drinking water standards."

Non-agricultural use of atrazine was banned from Sept 1993 in the United Kingdom and has ben banned in other European countries.

Workers

Although you say at p3.14-12 that herbicide use itself is not subject to review in this EIS/EIR, you are wrong. NEPA requires that you do an environmental analysis of the whole project, including, among other topics, the potential for worker exposures. You are denying that herbicides are part of the project because herbicides would be used anyway (see above). But the SYP involves clear cutting and intensive management to CAT-20

which PL admits herbicide use is integral. Thus herbicides are part of the project and must be addressed for more than "potential effects to covered species, particularly aquatic organisms." (p 3.14-12) CAT-20 CON.

Regarding atrazine and workers, EPA (1994) said that "also of concern is the potential cancer risk to persons mixing, loading and applying one or more of these compounds in agricultural areas." CA DPR (1994) said that "Current regulations require handlers of atrazine and chlorothalonil to follow basic pesticide handling precautions and to follow the saety precautions printed on the pesticide lael. These safety precautions, however, do not address other chronic or reproductive hazards of the pesticides. Chronic and reproductive damage often occur at very low exposure levels that do not cause acute illness, thus the person exposed may be unaware of excessive exposure. [DPR] proposes to add atrazine...to the [Minimal Exposure List]. (emph added) CAT-21

As the above information illustrates, your one little paragraph on workers' exposure to herbicides is pathetic and sad, to say the least. You need to describe how chemicals are transported and stored, mixed and loaded into application equipment and applied. Merely saying 'hack and squirt (p 3.14-2) doesn't satisfy the requirement to be transparent about the project. The decisionmaker and the public need to understand what this application method entails and how great the risk of accidental exposure to the worker for this method and others will be. It is important that you describe the environment that workers enter because the steepness of slopes, density of brush and amount and nature of other environmental features will impact 1) the likihood of worker exposure and worker accidents and 2) subsequent spills of herbicide into the environment.

Several studies illustrate our concern about the above mentioned problems. A 1980 EPA study reported scores of worker exposures. The Emergency Medical Treatment for Acute Pesticide Exposures chart indicates some of the responses that can be anticipated from exposure to various pesticides.

One evaluation of worker exposures to pesticides stated that reviews of pesticides by the federal pesticide regulatory program have had little effect on occupational risks. Further, it found that the potential for chronic illnesses from pesticide exposure is much higher than for acute toxicity for the pesticides examined. (Woodruff et al 1994) The EIR/EIS needs to take into consideration the range of effects to workers since they are handling pesticides under very dangerous circumstances.

In risk assessments you need to undertake for worker exposure you need to consider the potential effects to applicators as well as other workers who may be occupied in or passing through herbicide treated areas. One review of biomakersof human exposure to pesticides found that there are several studies on the cytogentic effcts of work with pesticide formulations. The majority of these studies report increases in the frequency of chromosomal aberrations...among exposed workers. The review found that sores and abrasions may fadcilitate uptake thrugh the skin, that vapors or aerosol droplets are absorbed effectively through the lungs, absorption resulting from dermal exposure is the most important route of uptake for workers, and that acute toxic effects are not easily recognized, whereas the effects resulting from long-term exposure to low doses

are often difficult to distinguish. (Anwar 1997)

Occupational exposures of parents to pesticides have been linked to childhood cancers which are on the rise. (Daniels et al 1997) CAT-21 CON.

Your assurances at 3.14.2.2, 3.14.3 and3.14.8 that a hodgepodge of laws, regulations, voluntary programs, company oversight and regulatory inspections will prevent worker exposure (barely mentioned) and the environment do not make the grade as transparent and adequate treatment of how worker safety and worker compliance will be assured.

We are happy to see at p 3.14-20 that you will keep "a copy of the company's spill contingency plan...on site in case of an accidental spill of hazardous materials." Perhaps you can use it to sop up the mess. You need to describe the critical potential for adverse impact to workers and species of concern caused by release of pesticide into PL lands, accidental or otherwise.

Twice in 1996 reports were made by residents of Humboldt County of pesticide spills by workers employed by PL and another Humboldt County business, Barnum Timber Company (Pesticide Episode Investigation Report, Stafford/Winter Express Incident, Sample analysis Report for atrazine, Cleanup and Abatement Order). That the violations were seen and reported by residents was highly unusual; the workers are usually far from observation by anyone but their employers. These serious violations indicate that there are problems with compliance by workers; this problem and its mitigation should be discussed in the EIR/EIS.

Sulfometuron methyl

At 3.14.2.3 you say that usage of atrazine is declining and that its replacement is Oust, which is one of the formulations for which the active ingredient is sulfometuron methyl. You seem to think this is a good idea because you postulate that it will "...reduce the potential for groundwater contamination from atrazine." (p3.14-6) Of course, you deny repeatedly that atrazine is a problem, including in your assessment of its toxicity at p 3.14-13 where you say that it "is not expected to migrate to groundwater under normal application conditions." Now that we've throughly debunked that absurd contention, we are supposed to be relieved that PL will replace atrazine with sulfometuron methyl because you say at p 3.14-14 that it "is relatively immobile and is not expected to migrate to groundwater under mormal application conditions" and it "is considered a slightly toxic compound"? We took a look at the available literature and concluded that your search was shallow and selective. CAT-22

You need to conduct a full blown risk analysis for sulfometuron methyl and all the other chemicals to be used for the SYP.

Sulfometuron methyl is one compound in a large class of chemicals, the sulfonylureas. Over 375 patents have been granted for sulfonylurea herbicides. Du Pont markets Sulfometuron methyl under the trade name Oust. CAT-23

Mode of Action: Sulfometuron methyl, like all sulfonylurea herbicides, is a "potent inhibitor of plant root and shoot growth." (Brown 1990) This cessation of growth occurs very quickly, within hours after exposure, and precedes other visual signs of sulfonylurea exposure, including bud death, loss of leaf color, and decay of plant tissues. growth is inhibited because sulfonylureas stop the plant's cells from successfully dividing." (Brown 1990)

CAT-23 con.

At a molecular level, sulfonylureas kill plants by inhibiting the activity of an enzyme, acetolactate synthase (ALS), that is involved in the production of three chemically-related amino acids – valine, leucine, and isoleucine. (LaRossa & Schloss 1984) Why the inhibition of this enzyme should cause cessation of cell division is not completely understood, but appears to be related to the accumulation of a chemical used by ALS to make the amino acids. (Brown 1990;LaRossa,Van Dyk. 1988) Mammals synthesize these three amino acids and must obtain them through their diet.

Animal cells do not contain ALS. (Brown 1990) It is possible that sulfonylureas do impact systems in mammals. For example, several sulfonylureas are used to treat diabetes because they stimulate insulin secretion by pancreas cells and reduce the rate at which it is removed form the blood by the kidneys. (Melander 1989)) These drugs also relax smooth muscles, such as those found around blood vessels. (Nielsen-kudsk & Thirstrup1991) This possibility should be considered in the analysis conducted through the CEQA/NEPA proceding for the Plans.

Acute Toxicity: Oust is acutely lethal to animals only in large amounts. An oral dose of over 5,000 milligrams per kilogram (mg/kg) is required to kill rats. Other acute effects occur at lower doses: baldness and mottled, heavy lungs were observed in the animals used to determine the acute lethal dose. Rabbit skin exposed to Oust became red, slightly to severely swollen, and the rabbits sporadically lost weight. When applied to rabbits' eyes, Oust caused mild redness, selling, and corneas to become opaque. (U.S.EPA1981)

Chronic Toxicity: Dogs fed sulfometuron methyl for three months developed elevated numbers of white blood cells and lymphocytes (cells found in both the blood and lymph systems). Neutrophils, scavenger cells in the blood, were decreased in number. A year- long study of dogs fed sulfometuron methyl found effects on other blood components. Mild anemia, with a decrease in the numbers of red blood cells and the amount of hemoglobin, was a principal finding. The same study found that sulfometuron methyl exposure caused a significant increase in the weight of the liver, as well as some increase in kidney weights. (U.S.EPA1983)

Effects on Reproduction:
Males: An early study of sulfometuron methyl toxicity to rats found\d that several rats (out of the six tested in this small study) developed problems with their testes when fed sulfometuron methyl for ten days. One rat had an unusually small testis, while a second had testicular lesions. (U.S.EPA 1981) A larger, longer-term study of dogs found both testicular degeneration and testicular atrophy. (U.S.EPA1983) The same kinds of effects have been found in a study of the effect on male fertility of an anticancer drug chemically related to sulfometuron methyl (in the sulfonylurea family).

(Seyler, Cochran & Todd 1992)

Females: A study of pregnant rabbits fed sulfometuron methyl found that they became anorexic, depressed, thin, and they had lower " reproductive indices" than unexposed rabbits, and their pregnancies ended with miscarriages and fetal resorption. (U.S.EPA 1981) In the rabbits given the highest sulfometuron methyl dose, all fetuses were resorbed. Another study of rats found that mothers fed sulfometuron methyl ate less, gained less weight, and had babies that weighed less than did unexposed mothers. (U.S.EPA 1981) CAT-23 CON.

We are concerned that reproductive and other effects found in tests to laboratory mammals may impact species of concern. You need to discuss this throughly in a thorough analysis of the risks involved with the use of herbicides on PL land.

Carcinogenicity and Mutagenicity: Two of the chemicals produced by the breakdown of sulfometuron methyl in goats, sulfonilamide and saccharin, (Koeppe, Mucha 1991) have been associated with cancer and genetic damage in laboratory test. Sulfonilamide causes mutations in the bacteria *Escherichia coli* (Iyer, Szybalske 1958) and invasive lung tumors in rats. (Haerem 1948) Saccharin induces bladder cancer in rats (Arnold et al 1980) and sister chromatid exchanges in Chinese hamster and human cells. (Wolff, Rodin 1978) (Sister chromatid exchanges are a type of genetic damage in which genetic material is exchanged, within a pair of chromosomes.)

Secret "Inert" Ingredients:The identity of 25 percent of the ingredients in Oust is called a trade secret by Oust's manufactured, Du Pont. There is no publicly available information about the identity of these secrets. Much of EPA's hazard assessment of Oust is based on tests of sulfometuron methyl and not the complete Oust formulation.

Interestingly, EPA is concerned about "the potential that the inerts [phrase deleted by EPA] of the *combination* of inerts and active ingredient together, when applied directly to water, might be considerably more toxic than the active ingredient alone" (U.S. EPA 1982)

Effects on Nontarget Organisms:
Microorganisms: The growth of many soil microorganisms is inhibited by sulfonylurea herbicides. In a study of eleven typical soil microorganisms, five were inhibited by Oust. Oust was more inhibitory that the other sulfonylurea used in this study, chlorsulfuron. The researchers concluded that sulfonylureas "would have significant effects on the microbial ecological balance of the soil." (Burnet, Hodgson.1991) Furthermore, "the consequences would be most evident in agricultural situations where the microbial population played an important role in maximizing the productivity of crops. (ibid) CAT-24

Plants: The potential for sulfonylurea herbicides to affect plants at startlingly low doses is probably the most important reason to develop alternatives to their use. This is an area in which research is only just beginning, but one that deserves serious attention. In a recent study (1993), EPA researchers showed that spring applications of the

sulfonylurea herbicide chlorsulfuron, in amounts equivalent to 0.001 times the concentration used in agriculture could reduce the amount of fruit produced by cherry trees if the applications were made at the time that the fruit had reached half of its fulls size. The reduction in cherry production was caused by concentrations of chlorsulfuron that caused almost no damage to the vegetative parts of the trees. At slightly higher concentrations. applications at a wider range of times caused fruit yields to drop. Fall applications with similarly low concentrations caused decreases in fruit production the following year. (Fletcher et al 1993) CAT-24 CON.

The researchers conclude, "if the effect of chlorsulfuron on cherry trees is characteristic of other plant species, drifting sulfonylureas may severely reduce both drop yields and fruit development on native plants, an important component of the habitat and food web for wildlife." They discuss the "devastating impact" expanded use for sulfonylureas could have on crop productivity, natural plant communities, and wildlife food chains.

In other words, misuse of Oust and other sulfonylureas could affect entire ecosystems, while leaving few signs to identify what is causing the problem. As a broad spectrum herbicide, Oust also has potential to have a substantial direct impact on the diversity of plant species present in a treated area. For example, a study in Florida slash pine forests showed that annual applications of Oust, together with other broad spectrum herbicides, caused a dramatic shift" (250 in the distribution and diversity of plants. Almost 20 percent of the treated plots contained only slash pines of bare ground, while none of the untreated plots was bare of only pine. A less intensively managed site (two herbicide applications) also showed a decrease in diversity. (Neary et al 1990)

The importance of this kind of research is perhaps most clearly illustrated by the endangered plant species which the U.S. Fish and Wildlife Service has identified could be jeopardized by use of Oust along rights-of-ways. The list includes 25 species in 13 states, as well as the critical habitat for three of the species. (U.S. Dept. of the Interior FWS1983) As the Fish and Wildlife Service consultation points out, "Because of the limited population size of many of these plant species a local spraying program could virtually destroy the entire species. (U.S. Dept. of the Interior FWS1983)

Contamination of Soil and Water:
Soil: Sulfometuron methyl is moderately persistent in soils according to tests reviewed by EPA. In four soils from Delaware, Illinois, and Florida, the half-life (the time required for half the pesticide applied to disappear was one month. (U.S.EPA1981) Under some conditions, however, sulfometuron methyl residues can persist for much longer. For example, a study of soils in Delaware, North Carolina, Oregon, Colorado and Saskatchewan found that some sulfometuron methyl persisted in each soil for a year. In the Colorado soil, 18 percent of the sulfometuron applied was still present after one year. (Anderson & Dulka 1985) CAT-25

On sandy soils in Franklin County, Washington, it persisted at levels high enough to damage plants for over a year following a roadside application of Oust. (Turner 1987)

In a study of forestry applications in Mississippi and Florida, sulfometuron methyl was

detectable in soil 60 days after application, and in vegetation for 90 days. (Michael, Neary 1988) In New Brunswick forests, enough Oust persisted to kill spruce seedlings ten months after sulfonylurea treatment. (Harvey et al 1985)

CAT-25 CON.

Du Pont scientists have discovered that the rate at which sulfonylureas break down in the soil is "bi-exponential." This Term is used to describe the degradation of a pesticide that is relatively quick at first, then slows so that there is long persistence of some of the chemical. The Du Pont researchers believe that the residual herbicide diffuses into spaces that are too small for microbes and thus is protected, resulting in extended persistence. (Brown 1990)

Water: Laboratory tests of sulfometuron methyl's leaching potential found that a high proportion (75-87 %) of the amount applied leached from the soil column. (U.S.EPA, Garner) Chemicals that leach readily move through the soil easily and can potentially contaminate ground water.

Measurements for the mobility of sulfometuron methyl in soil showed that it was more mobile than atrazine(Wehtje et al. 1987), the herbicide that is the second most common contaminant of ground water in the U.S. (U.S.EPA 1990) The authors believe that "considerable movement may expected" under certain conditions. (Wehtje et al 1987)

Drift:
Blowing soil: Because tiny amounts of Oust can kill plants, any drift (movement of a pesticide away form the area in which it is intended for use) has the potential to cause significant damage. A 1985 incident in Franklin county, Washington, is an illustration of this type of problem. Almost all of the rainfall in Franklin county occurs during the fall, so that most of the applied Oust remained "on top of the very light, sandy" soil. (Turner. 1987) The soil blew from the roadsides into agricultural land and caused extensive damage. Corn, barley, wheat, asparagus, carrots, onions, beans, peas, potatoes, alfalfa, pasture, and fruit trees were all affected. Many crops were killed; some carrots,onions, and potatoes that received doses too small to kill them developed "growth disorders." (Turner 1987)

Flowing Water: Damage from Oust was also documented in Pierce County, in western Washington where rain is abundant. During the spring of 1991, transplanting and direct seeding failed at one agricultural property.

Oust applied during the spring of 1990 by Pierce County maintenance crews along the roads which bordered the property had washed into fields during winter rains and flooding. Oust residues were found in soil samples taken form the field in 1991. (Creger 1992)

Resistance of higher plants to sulfonylurea herbicides has been demonstrated. (Chaleff,Mauvais 1984) Sulfonylurea-resistant weeds have become an increasing problem. Resistance has been reported at over 200 sites in the western U.S. (Saari 1992)

CAT-26

Development of resistance occurs quickly. For example, perennial rye grass in California developed resistance after six annual roadside applications of Oust and Russian thistle in Washington developed resistance after only four annual applications. (ibid) CAT-26 CON.

The development of herbicide resistance is a direct warning that the use of herbicides is not a sustainable technique for managing vegetation. Du Pont in 1991 had to end the use of a sulfonylurea in seven Great Plains states. (Agrichemical Age-February1991) Many weeds that develop resistance to one sulfonylurea are resistant to other sulfonylureas and to herbicides whose mode of action is similar (This is called cross resistance). (Primiani 1990)

Perhaps more importantly, cross-resistance has also developed between sulfonylureas and herbicides with a completely different mode of action. (Kempet et al 1990)

Clearly you did not examine sulfometuron methy adequately in your mini-assessment. You were wrong to assume that you need only examine aquatic effects of the chemicals; NEPA and CEQA demand much more.

2,4-D

PL would like to use 2,4-D. We have attached a number of studies that indicate that 2,4-D is far more dangerously toxic than you will admit. (Knopp 1994; Clement & Colborn 1995; Weisenburger 1990; Reynolds et al 1994; Gerken 1995) Some of these studies focus on endocrine disruption, CAT-27

One of the issues that needs to be addressed is the fact that these pesticides may be endocrine disrupters, chemicals that mimic natural hormones, upsetting normal reproductive and developmental processes. 2,4-D is considered an endrocrine disrupting chemical, as is atrazine. (Colburn 1995) Though the damage these chemicals do has been recognized in both humans and wildlife for some time, it is only recently that an understanding of the processes involved, and that which is causing it, has come to light. Numerous disorders including defective sexual organs, hormone-related cancers, impaired fertility, learning disabilities and attention deficits in children and the loss of young are now blamed on these chemicals.

A recent study from England found that in two of eight rivers tested, 100% of the male fish tested had feminized reproductive tracts while the other six rivers showed anywhere from 20% to 80%. The massive drop in human sperm counts, which has been shown to be as much as 50%, is also linked to these chemicals.

Unfortunately no one knows for sure which chemicals are causing the problem. Though some have been identified, many others are suspected. The problem is now being taken very seriously by the EPA who have just ordered 62,000 chemicals that are already in use to be tested for their potential to be endocrine disrupters.

Of the three chemicals used extensively by PALCO, all three are known or suspected endocrine disrupters. Atrazine is a known endocrine disrupter which causes breast

tumors in laboratory animals. Triclopyr is very suspect. Some of the chemicals that seem to be causing the most trouble are chlorinated compounds, which triclopyr is. It is also known that any auxin-inhibiting compound has the potential to cause disruption, which includes triclopyr. Auxin is a plant growth hormone and triclopyr works by mimicking auxin, causing uncontrollable and inappropriate cell proliferation and subsequent exhaustion of the plants energy and nutrient reserves. Since it's mode of action is hormone disruption in plants, it is highly suspect as an endocrine disrupter in humans, fish and wildlife.

CAT-27 CON.

Glyphosate is also very suspect. A recent study by Yousef et al. found glyphosate to cause reduced sperm counts in rats and increased the frequency of abnormal and dead sperm in rabbits. The authors were unsure if the damage was being done through fetotoxicity or endocrine disruption. Since considerable research has established that glyphosate's mode of action is inhibiting enzymes, this too could cause endocrine disruption. Foreign chemicals may boost the body's hormone levels by interfering with the physiological processes that break down hormones so they can be excreted. Some chemicals deactivate the enzymes involved in this process. For example, a chemical that interferes with the enzymes that break down estrogen would cause more estrogen to be available to the receptors and indirectly create an estrogenic effect.

The damage from endocrine disrupters is as old as synthetic chemicals. Two of the worst and earliest disrupters were DDT and DES, the damage from which we are still experiencing today. The most important thing that needs to be understood is that it only takes small amounts of exposure to occur at a certain time to do massive and irreversible damage. I would like to quote from the most definitive piece of work on the subject, the book Our Stolen Future by Theo Colburn, Dianne Dumanoski and John Myers.

At levels typically found in the environment, hormone-disrupting chemicals do not kill cells, nor do they attack DNA. Their target is hormones, the chemical messengers that move about constantly within the body's communication network. Hormonally active synthetic chemicals are thugs on the biological information highway that sabotage vital communication....Because hormone messages orchestrate many critical aspects of development, from sexual differentiation to brain-organization, hormone-disrupting chemicals pose a particular hazard before birth and early in life. Relatively low levels of contaminants that have no observable impact on adults can have devastating impacts on the unborn. The process that unfolds in the womb and creates a normal, healthy baby depends on getting the right hormone message to the fetus at the right time (pg. 203-4).

It is also important to understand that our old way of looking for damage from synthetic chemicals does not fit endocrine disruption. The damage can be both obvious or subtle and short or long term. From the book Our Stolen Future:

"Hormone-disrupting chemicals can diminish individuals without making them sick. For this reason, there is an urgent need to look for impaired function as well as for disorders that fit the classic notions of disease. For example, having a poor short-term

memory or difficulty in paying attention because of exposure to PCB is is very different from having a brain tumor. The former are deficits, not diseases, but they can nevertheless have serious consequences over a lifetime and for a society. They erode human potential and undermine the quality of human life. They undermine the ways in which humans interact with one another and thereby threaten the social order of modern civilization, not to mention the basic survival." Colburn 1995; similar statements can be found at Wingspread Statement 1991)

CAT-27 CON.

Exposure to a hormone-disrupting chemical before birth does not produce just a single clear-cut effect, and in this way, too, the results defy our prevailing notion of chemically-induced disease. Depending on the dose and the timing, a foreign chemical can derail development in a variety of ways that will become evident at different times. For example, a boy exposed before birth to chemicals that mimic estrogen may have undescended testicles at birth, a low sperm count at puberty, or testicular cancer in middle age because of this prenatal hormone disruption (ibid).

Endocrine system damage is by no way limited to humans and has been shown to have serious impacts on species in the wild. In July, 1991, a group of scientific experts from numerous fields, including ecology, endocrinology, reproductive physiology and wildlife management, came together to address the issue of endocrine-disruption.

From the Wingspread Consensus Statement " We are certain of the following. Many wildlife populations are already affected by these compounds. The impacts include thyroid dysfunction in birds and fish; decreased fertility in birds, fish, shellfish and mammals; decreased hatching success in birds, fish, and turtles; gross birth deformities in birds, fish and turtles; metabolic abnormalities in birds, fish and mammals; demasculinization and feminization of male fish, birds and mammals; defeminization and masculinization of female fish and birds; and compromised immune systems in birds and mammals (pg. 253). Of special concern is the fact that birds are targeted by all conditions mentioned above and fish by all except compromised immune systems, which bodes ill for the recovery of marbled murrelets, northern spotted owls and anadromous fish."

As the above statement clearly points out, the damage that had already been identified in 1991 was wide-ranging and extremely serious. Since then the damage has been shown to be even more serious than previously thought and each day brings new information to our attention, as the article on fish populations in England shows. As we learn more about endocrine-disruption it becomes apparent that the potential for even greater damage exists. Once again from Our Stolen Future.

Serious detailed assessments should be undertaken to consider the role of hormone disrupters in several disturbing ecological trends, especially the dramatic decline and loss of frog populations around the world, the series of epidemics that have hit marine mammals, and other notable biological disruptions. Some classic wildlife crashes should be revisited to ask whether hormone disrupters contributed to the declines or perhaps are impairing recovery (p 224).

In recent weeks there have been stories released concerning a population crash of Alaskan seals, mysterious deaths to birds in Mexico and a mysterious disease that is affecting Maine lobsters because they are being weakened by some other, as yet unknown cause. The reason I mention these is to highlight the fact that so much damage is occurring worldwide that we have no understanding of, much of it via endocrine disruption. Has there been a thorough investigation by PALCO of the potential for any of the herbicides in question to be endocrine disrupters? If so, why has this not been mentioned? If not, has there been any investigation at all? Has there even been a discussion of this issue? CAT-27 CON.

All of this raises serious questions about potential impacts that may be generated by the application of the herbicides glyphosate and triclopyr, which are suspected of being endocrine disrupters, atrazine which is known to be an endocrine disrupter, and any other herbicides which may be used and that may be disrupters. Considering the amount of wildlife both in the proposed spray area and downstream, much of it which is either endangered, threatened or of special concern, we need to address these concerns and define the potential for impact. The issues here are of such great importance to the recovery of the marbled murrelet, northern spotted owl and anadromous fish that it shouldn't be left up to PALCO, especially in light of the fact that PALCO has shown a complete disregard for following environmental law and protecting public trust resources that exist on their lands.

Wan et al in 1990 points out that 2,4-D and diesel are extremely toxic to Pacific salmonids. Colburn in 1995 discusses endrocrinological effects on wildlife. And Jobling in 1998 discusses widespread sexual disruption in wild fish. Why have you ignored this important aspect of pesticide toxicity?

Are pesticides a problem? The experts think they are. (Envir. Health Perspectives 1993; Hodgson, Levi 1996) Check it out, then give us a risk assessment. The law demands it.

Triclopyr:

Another of the chemicals PL will rely on is triclopyr. We enclose it largely as an example of why PL cannot change formulations without re-opening the HCP. Formulations can vary dramatically in their toxicity, thus ALL the chemicals in a formulation need to be assessed for risk, and if a new formulation is introduced, a new assessment will be required. CAT-28

Triclopyr is the pyridine analog of 2,4,5-T, differing from 2,4,5-T by one atom. It has been commercially available since the 1970s and is the active ingredient found in Pathfinder, Garlon 4 (present as the ester of both), Garlon 3A (present as the amine salt), [USDA 1992] and Turflon (available both as an ester and an amine).[Willoughby 1992] Combinations with other herbicides such as 2,4-D and picloram are also available. [Willoughby 1992]

Like the phenoxy herbicides, triclopyr imitates a plant growth hormone, auxin, and kills plants by inducing disorderly growth. [Corbett 1984]

None of the toxicology studies and few of the studies of triclopyr's ecological impacts have been carried out by independent scientists: most have been carried out by independent scientists; most have been carried out or funded by triclopyr's manufactured. This makes it difficult to assess the effects of widespread triclopyr use.

CAT-28 CON.

Acute Toxicity:Triclopyr's acute oral median lethal dose (LD50, or the dose of a compound that kills fifty percent of the test animal population) in rats is 630 to 729 milligrams per kilogram (mg/kg) of body weight. This places triclopyr in Toxicity Category 111 (Caution) according to the U.S. Environmental Protection Agency's (EPA's) classification system. Its acute dermal toxicity in rabbits is greater than 2,000 mg/kg, again placing triclopyr in Toxicity Category III. [USDA 1992]

Triclopyr is a slight eye irritant (Toxicity Category III). [USDA 1992]
Garlon 3A is a severe eye irritant ((Toxicity Category I). [USDA 1992]
The material data safety sheet (MSDS) reports that Garlon 3A, "may cause severe irritation with corneal injury which may result in permanent impairment of vision, even blindness." [Dow Chemical 1990] In California, two eye injuries resulting from occupational triclopyr exposure occurred in 1988. [CDFA 1990]

Triclopyr is considered a slight to moderate skin irritant (Toxicity Category III to IV) and "prolonged or repeated exposure may cause skin irritation, even a burn." [Dow Chemical 1990] Garlon 4's MSDS mentions that, "prolonged or frequently-repeated skin contact may cause allergic reactions in some individuals." [Dow Chemical 1990] Exposure to technical triclopyr amine has resulted in flaking skin up to a week later. [Newton 1984] Skin injury as a result of occupational triclopyr exposure has been reported in California.

In rats, the lowest dose for which no adverse health effects were noted as a result of one hour of triclopyr inhalation was about 5 parts per million (ppm) triclopyr, again putting triclopyr in Toxicity Category III. [USDA-FS1984] However, rats exposed to less than 1ppm Garlon 4 MSDS warns, "Excessive exposure may cause irritation to upper respiratory tract" [Dow Chemical 1990]

In human volunteers, blood triclopyr levels peaked two to three hours after oral administration of triclopyr acid. More than 80 percent of the triclopyr was excreted in urine unchanged. Five of the six volunteers excreted detectable triclopyr residues over 60 hours after ingestion. Triclopyr residues were found in the blood three hours after dermal applications of triclopyr ester although less that two percent of the triclopyr dosage applied to the skin was absorbed. The half-life (the time it takes for one half of a chemical concentration to degrade or move to a different location of dermally absorbed triclopyr was estimated at 16 hours. [Carmichael 1989]

In a monkey fed triclopyr, a majority of the triclopyr was cleared form the system with a half-life of the remainder was 150 hours. Almost a quarter of the triclopyr dosage was not recovered. This suggests that concentrations for triclopyr may be temporarily or permanently stored somewhere in the monkey. [Newton1984]

In rats, the half-life was about four hours. Triclopyr residues were detected in the kidneys, liver, lugs, skin, and ovaries. [Timchalk et al 1990]

CAT-28 CON.

3,5,6-TCP (3,5,6-trichloro-2-pyridinol: is the primary metabolite of triclopyr, as well as the primary metabolite of the organophosphate insecticide chlorpyrifos. 3,5,6-TCP has an acute oral LD50 of 400 mg/kg for mice. [Newton 1984] A group of rats maintained on a diet containing 3000 ppm (0.3%) 3,5,6-TCP exhibited increased urination. At a reduced triclopyr dose of 1000 ppm, growth and body weights were reduced, and urination increased. [Newton 1984]

Chronic Toxicity: There are no publicly available chronic toxicity tests of triclopyr products as they are used, only tests on triclopyr itself. These tests have revealed indications of organ damage, possible reproductive and cancer-causing effects, and genetic damage.

Teratogenicity/Fetotoxictne study of birth defects conducted on rats, doses of 200 mg/kg triclopyr caused an increased incidence of delayed skull bone hardening, resulting in fetal death. There was evidence of maternal toxicity at all dosage levels use in the test, with such symptoms as increased hair loss, excessive salivation and reduced weight gain, but no apparent effects were observed in the live offspring. [Newton1984] [Hanley 1984] [CDFA 1990]

Other laboratory tests of chronic toxicity reported increased kidney effects in rats and dogs, and enlarged thymus glands and increased water consumption in mice. [CDFA1990]

Secret"Inert" Ingredients:
Many of the "inert" ingredients in triclopyr products are called trade secrets by Dow Elanco and are not publicly known. This section summarizes health concerns about ingredients which have been identified.

Kerosene is present in the formulation Garlon 4. [USDA-FS 1992] Kerosene is a skin irritant. It may adversely affect the central nervous system [Dow Chem. 1990] and cause chemical pneumonia or death if the liquid form is inhaled. [USDA-FS 1992] [Dow Chem. 1990] It has not been found to cause mutation of cancer, but kerosene does contain small concentrations of other petroleum compounds that are known to be carcinogenic. [USDA-FS 1992]

Ethanol (ethyl alcohol) is present in the formulation Garlon 3A as an "inert" ingredient. [USDA-FS 1992] Ethanol has been known to cause such health effects as birth defects, decreased male fertility, and adverse neurological and liver effects. .[USDA-FS 1992]

Ethylenediamine tetraacetic acid (EDTA) is used in Garlon as a chelating agent.(Letter from Stewart...1993) EDTA has been registered as a pesticide active ingredient in the U.S. It causes genetic damage mammalian cells and developmental abnormalities

and fetal death in rats. (U.S. Dept of Health and Human Services1991]

CAT-28 CON.

Diesel oil is often added to Garlon ester formulation when they are used. Symptoms of diesel oil exposure may include nausea, vomiting, coughing, lung irritation, and chemical pneumonia. Several studies have found that low concentrations of petroleum products applied to bird eggs could cause embryonic death. [Hoffman 1990] In one particular study, the application of diesel fuel to pheasant eggs reduced the matchability to zero. [Koplschke 1972]

Diesel oil is highly toxic to freshwater fish; 0.19 ppm in water is enough to cause death. [Monroe 1988] Diesel oil, like kerosene, also contains carcinogenic compounds such as Bunsen and other aromatic hydrocarbons. [Monroe 1988]

Environmental Fate:

CAT-29 CON.

Soil persistence: The persistence of triclopyr residues in soil varies widely. Triclopyr residues have been detected in soil from one week to over two years [Nilsson 1983] [Stark 1983] following application. Triclopyr's half-life was between 75 and 81 days at tow study sites in western Oregon. [Nilsson 1983]

In soil, triclopyr is primarily degraded by microbes. [Norris 1987]
Triclopyr has a solubility of 430-440 ppm in water. This means it can be leached form the soil along with water. It a strong rain occurs shortly after a triclopyr application, the triclopyr residues will either run off with the rain water, or be leached to lower soil layers. Microbial activity decreases with soil depth, so once the herbicide gets in the lower soil, it will be more persistent than it would be at the surface. [Nilsson 1983] For example, triclopyr residues were detectable 30 to 45 centimeters below the soil surface in a study conducted in western Oregon for 477 days following application. . [Norris 1987]

Triclopyr also has a tendency to adsorb, or adhere to soil particles. Chemical soil adsorption is increased with higher organic content or clay content, as well as with higher acidity. [Smith 1988][Somasundaram 1991] Adsorption of triclopyr reduces leaching, and tends to keep the chemical in the upper soil levels where it is metabolized by microbes. Soil flooding may slow triclopyr degradation. [Bush, Neary, & Taylor 1988]

3,4,5-TCP, Triclopyr's most common metabolite, has shown microbial toxicity. The addition of 3,5,6-TCP to soils with no previous chlorpyrifos history inhibited the microbial metabolism of chlorpyrifos. [Somasundaram 1991]

One study found that 90 per cent of the triclopyr applied to sandy soil dissipated within 4 weeks after the application. Dissipation of the remaining 10 per cent, however, was not apparent, and recoverable residues were found 489 weeks after treatment. [Stephenson 1990] The higher the dose of 3,5,6-TCP in a soil sample, the less degradation of this compound occurs, pointing to the microbial toxicity of this metabolite. 3,5, 6-TCP has reported half-lives from 10 to 325 days. [Somasundaram

1991] Half-lives for 2-methoxy-3,5,6-trichloropyridine (see Figure 4), the second most common metabolite of triclopyr, range from 50-300 days in three different soil types. [USDA-FS 1992]

CAT-29 CON.

Vetch, a plant highly sensitive to triclopyr residues, suffered severe inhibition of growth when planted in soil taken from a depth of 25 to 38 centimeters 1,2 and 12 weeks after triclopyr application, a good indication of triclopyr's leaching potential.[Devlin 1984]

Water persistence: The primary route of triclopyr degradation in water is photolysis, chemical reactions resulting from exposure to light. The half-life of triclopyr in water averages about 24 hours for surface waters exposed to sunlight, [USDA-FS 1992] but has been reported to be as little as 2 hours and as much as 3 days. [McCall & Gavit 1986] The presence of clouds may slow triclopyr degradation, as may turbidity of surface water. [McCall & Gavit 1986] Factors such as the acidity of the water and sedimentation may also affect the rate of triclopyr ester and amine formulation breakdown, as well as triclopyr acid degradation.

An experiment studying the persistence of triclopyr residues in a lake in northern Ontario following a direct triclopyr application found that 95 percent of the triclopyr acid had been degraded within 15 days after application. However, the remaining residues were found in the water for up to 42 days after the application. [Solomon, et al 1988] Triclopyr residues in rainwater running off from agricultural areas have been found in South Africa. [Letter form Roger Evans... 1989] In a watershed in northern Maine, the peak concentration in a stream was 56 parts per billion (ppb) as a result of direct application and drift form an aerial triclopyr application. Residue levels were 48 ppb after the first rain 6 days later. [Smith & McCormack 1988.]

EPA has reported the presence of triclopyr residues in five granddaughter samples taken in Texas and Virginia. [US EPA. Office of Prevention....1992]

Plant Tissue Residues: Foliar surfaces are not thought to be conducive to microbial degradation. [Newton et al. 1990 & 1992] The major route of triclopyr degradation on foliage is photolysis, particularly when other factors are not in operation, such as wash-off, metabolism and evaporation. [Benston & Norris1991] Triclopyr disappears form foliage faster as temperature increased, regardless of the presence of light, indicating the importance of evaporation. [Benston & Norris1991]

Specific leaf structure and physiology help determine how much triclopyr is taken up by the leaf. For instance, chinkapin leaves take up more triclopyr than madrone leaves. The triclopyr formulation used is also a factor: ester formulations penetrate foliage more rapidly than amine formulations. [Newton 1990]

Initial half-lives for triclopyr residues on tan oak foliage were found to be more than one month. [Newton 1982]

In a study conducted in southwestern Oregon triclopyr residues 'in evergreen foliage and twigs showed remarkable persistence." This study also found that triclopyr absorbed into the vegetation "is not readily removed until the vegetation becomes

litter" and is available for microbial decomposition. [Newton 1990] Triclopyr residues were measured in conifer vegetation for at least one year following the application. [Newton 1990] Triclopyr residues persisted in dead honey mesquite stems, both standing and fallen, for at least two years following treatment. [Bovey, Hein & Meyer1984] The subsequent decomposition of vegetation containing triclopyr residues can release triclopyr and its metabolites into the soil months after the initial application. CAT-29 CON.

Effects on Nontarget Organisms:
Desired Plant Species: Triclopyr can damage the crop plants it is meant to benefit. For example, damage incurred by ponderosa pine trees was more severe than that suffered by manzanita (the weed) in a forestry experiment. [Paley& Radpsevich 1984] In another example Douglas fir seedlings were damaged by triclopyr ester. Damage was seen greater to terminal and lateral buds when a diesel oil carrier was used. [Fischer & Carrithers 1992] CAT-30

In a study simulating triclopyr drift to a cotton field, cotton yields were reduced, particularly during early bloom stages. Irrigation water contaminated with less than 1 ppb triclopyr can cause injury when administered to sensitive crops. [Newton & Dost 1984]

Ecological Effects:
Of four forestry herbicides tested, triclopyr showed the most severe inhibition of germination of seeds on the forest floor. [Morash & Freedman 1989] Germination inhibition in the long term may alter stand vigor and affect secondary succession. Forestry herbicide treatments, including triclopyr, reduce the overall number of forest plants, and strongly reduce plant diversity. [Neary1990] CAT-31

Maintenance of forest plant diversity is essential to protect rare and endangered species of both plants and animals; as well as to provide for long term forest health.

Nitrate and calcium concentrations increased in forest soil solutions and stream water for two growing seasons following an aerial application of triclopyr. [Smith, Horn beck & McCormack 1988] The nitrate levels measured in this experiment were of the same magnitude as levels measured following clear-cuts. This indicates that triclopyr reduces vegetation enough to aid the leaching. [Smith, Horn beck & McCormack. 1988] Triclopyr applications reduce nutrient levels available to forest systems, as well as increase the potential for adverse health effects from higher nitrate levels in streams and water sources.

Microorganisms:
In one experiment triclopyr was found to be the most toxic of four herbicides tested to five species of mycorrhizal fungi. Fungal growth was reduced at concentrations of 0.1 ppm and above. [Chakravarty & Sidhu1987]

In another experiment studying the effects of herbicides on there development of mycorrhizae and subsequent conifer seedling growth, triclopyr was again found to be the most toxic of the herbicides tested. [Sidhu & Chakravarty 1990]

Triclopyr reduced mycorrhizal growth and colonization of the roots of lodgepole pine and white spruce. Concentrations as low as 1.0 micrograms per liter killed seedlings. Seedlings with mycorrhizae on their roots were more sensitive to the herbicide than those without mycorrhizae, possibly as a result of increased herbicide absorption facilitated by the fungi. [Sidhu & Chakravarty 1990]

CAT-32 CON.

In a study of the toxicity of triclopyr to bacteria, fungi and algae, algae was the most sensitive organism. Triclopyr's metabolite 3,4,6-TCP was much more toxic than triclopyr to all three kinds of organisms and the triclopyr acid was generally more toxic than to all three kinds of organisms and the triclopyr acid was generally more toxic than the triclopyr amine salt. [Baarschers, Donnelly, & Heitland 1988]

Aquatic organisms: The formulation is a critical factor in the toxicity of triclopyr to aquatic organisms. The ester formulation Garlon 4 is 167 times more toxic to fish than amine formulations (Garlon 3A). [Norris & Dost 1991] Garlon 4 is also significantly more toxic than triclopyr acid. [USDA-FS 1992] In addition, the ester formulations are more persistent, with the half-lives in water are that six times longer than those of triclopyr acid. [McCall & Gavit 1986.] Ester formulations are also less soluble than triclopyr acids and have a tendency to accumulate in the sediment, causing problems for ground-dwelling aquatic organisms. [McCall & Gavit 1986.]

Lethal concentration values vary for different species of fish, and for different triclopyr formulations. The LC50s (the concentration that kills 50 percent of a group of test animals for salmon are 1.4 ppm for Garlon 4; 7.8 ppm for triclopyr acid; and 275 ppm for Garlon 3A. [Johansen & Geen 1990]

For the fathead minnow, LC50s are between 101 to 120 mg/l for the triclopyr amine formulation. [Mayes, et al. 1984]

The greater toxicity of triclopyr ester formulations may not be due to greater toxicity for the compound itself, but may rather be due to the ester's ability to facilitate absorption of the triclopyr acid, especially through gill membranes. This causes faster triclopyr uptake by the organism. [Barron 1990] One study found evidence that triclopyr acid was the compound associated with lethality rather than the ester compound, due top the rapid breakdown of the ester formulation into triclopyr acid by coho salmon, and the subsequent accumulation of the acid in the fish tissues. [Johansen & Geen 1990]

Perhaps of more importance than the acute effects of triclopyr exposure are those effects occurring as a result of sublethal concentrations. These do not cause immediate death, but may alter behavior enough to result in lowered survival of exposed organisms. In an experiment conducted on rainbow trout, some fish exhibited disequilibrium and erratic swimming at concentrations as low as 0.12 ppm of Garlon 4. At 0.60 ppm of Garlon 4 (25 per cent of the LC50 for trout) a majority of the fish showed increased surfacing and respiration. For Garlon 3A, concentrations of 200 ppm (50 per cent of the LC50) resulted in similar significant behavioral changes., Trout did not show avoidance of poisoned water until concentrations reached two to eight times the LC50 for triclopyr formulations. [Morgan 1991]

A study examining sublethal responses of juvenile coho salmon to triclopyr ester formulations found hypersensitive reactions to stimuli and increased respiration at concentrations less than 0.10 mg/l (20 percent of the LC50). Greater concentrations resulted in fish lethargy followed by distress and ultimately death. [Janz, et al. 1991]

CAT-32 CON.

In a study conducted on twelve species of aquatic insect, Garlon 4 was found to be more toxic than another herbicide, hexazinone, causing significant mortality in two insect species at concentrations lower than 80 ppm. One insect species, *Dolophilodes distinctus*, showed significant downstream drifting and mortality at a concentration of 3.2 ppm of Garlon 4. [Kreutzwelser, Holmes, & Behmer 1992]

The LC50 for *Daphnia pules*, a water flea is 1.2 ppm of Garlon 4. [Swrvizi, Godon, & Martens. 1992] Reproduction (brood size) in *Daphnia magna* was reduced by triclopyr exposure. [Gerisch 1985]

The oyster is the most sensitive salt water invertebrate for which lethal concentrations have been calculated, with an LC50 of 56 ppm triclopyr amine. [Norris & Dost. 1991] In an experiment observing metabolism of triclopyr in crayfish, a crustacean invertebrate, triclopyr was excreted with a half-life of 7-17 days. Most of the residues were found in the shell and the hemolymph, the circulatory fluid. [Barron, Hansen, and Ball. 1991]

Birds and Other Terrestrial Wildlife:

Triclopyr carriers and "inerts" such as diesel oil and kerosene have been found to reduce bird egg matchability. [Koplschke. 1972] The tendency of triclopyr to persist in vegetation suggest s the potential for the poisoning of herbivorous wildlife consuming the contaminated foliage. Triclopyr residues in cowberries persisted up to 98 days following treatment. [USDA-FS 1984] Residues on foliage potentially eaten by deer and elk in northern Idaho persisted for a year (the duration of the study) on all of the species of plants tested that survived the herbicide treatment. [Whisenant & McArther 1989.]

Glyphosate

Another favored psticide is glyphosate, probably in the Roundup and Accord formulations. Glyphosate formulations are another example of the need to assess full formulations, not just active ingredients.

CAT-33

U.S. EPA recently reclassified glyphosate as a Group E chemical, which means that evidence exists that it is not likely to cause cancer in humans. Yet glyphosate's carcinogenic potential is subject to debate. Studies conducted by the manufacturer showed increased testicular tumors, thyroid cancer in females, and a rare kidney tumor in laboratory animals (U.S.EPA 1985, 1991). Studies submitted to California's Department of Pesticide Regulation indicated possible adverse cancer effects, with rare tumor formation in the kidneys and adrenal cortex (DPR 1992).

Formaldehyde, classified by U.S. EPA as a probable human carcinogen and as a

carcinogen under California's Safe Drinking Water and Toxic Enforcement Act of 1986 (OEHHA 1994), is a product of the breakdown of glyphosate in the environment (U of M 1998). N- nitrosoglyphosate, a contaminant of glyphosate, is a member of a chemical family of which approximately 75% are known carcinogens (U.S. HHS 1998).

CAT-33 CON.

Test animals exposed to glyphosate experienced inhibition of cytochrome P450 and two other enzymes which detoxify chemicals in mammals (Heitanen 1983). At least two enzymatic steps are involved when toxic chemicals are processed in the liver of humans; the first involves cytochrome P450 enzymes and the second involves glutathione S tranferases (GSTs). People who do not possess certain GSTs due to genetic variation (estimated to be 50% of 'Caucasians;' others unknown), may have a greater risk of some types of cancer (Perera 1996). No studies were found exploring of the effect of P450 inhibition by glyphosate may have on people who do not possess certain GSTs.

Laboratory studies undertaken with human blood cells showed mutations at low dose tests (Bolognesi et al 1997)

In developmental toxicity studies using pregnant rats and rabbits, glyphosate caused treatment-related effects in the high dose groups including diarrhea, decreased body weight gain, nasal discharge and death (EPA, 1994).

One reproductive toxicity study using rats showed kidney effects in the high dose male pups; another study showed digestive effects and decreased body weight gain (EPA, 1994).

In a low dose exposure study of experimental animals, salivary gland abnormalities related to changes in adrenalin levels were observed as were changes in the kidney, liver, and thymus (U.S. DHHS 1992).

An unknown percentage of Roundup's formulation is composed of polyethoxylated tallowamine (POEA), a surfactant added to enhance the performance of glyphosate. POEA is at least three times as acutely toxic as glyphosate (Sawada 1988), is irritating to eyes and skin, and causes gastrointestinal problems (Monsanto 1992). 1,4 dioxane is likely to contaminate POEA as a result of the manufacturing process. When, to test the theory, a sample of a glyphosate and POEA formulation was analyzed, it was found to be contaminated by 1,4 dioxane (Jyrkkanen 1989; Monroe 1990). 1,4 dioxane is regarded by U.S. EPA as a probable human carcinogen and is listed as a carcinogen under California's Safe Drinking Water and Toxic Enforcement Act.

In animal tests, a mixture of glyphosate and POEA caused cardiac arrest (UNEP/WHO/ILO 1994). The amount of Roundup — which combines glyphosate and POEA — required to kill rats is about 1/3 of a lethal dose of either compound applied separately (Martinez 1990,1991), suggesting that synergism of the two chemicals may enhance toxicity.

Another portion of Roundup's formula is composed of isopropylamine, a neutralizing

agent. It is extremely destructive to tissue of the mucous membranes and upper respiratory tract (Sigma Chemical 1994).

CAT-33 CON.

Some glyphosate end-use products are in Toxicity Categories I or II for primary eye irritation or skin irritation. In California, glyphosate ranks high among pesticides causing illness or injury to workers, who report numerous incidents of eye and skin irritation from splashes during mixing and loading (EPA 1994).

Formaldehyde, one of glyphosate's breakdown products, is classified as a toxic air contaminant (DPR 1994).

Between 14% and 78% of glyphosate applied as a ground spray drifts off site (Freedman 1991). It has been documented to affect plants as far as 131 feet away, and residues have been detected 1,312 feet downwind (Marrs 1993).

Glyphosate is highly persistent in soil; residues have been found up to three years after application (Torstensson, Lundgren, Stenstrom 1989). A federal review noted that glyphosate in soil "is extremely persistent under typical application conditions" (U.S. EPA 1993). Glyphosate does have the potential to contaminate surface waters due to its aquatic use patterns and through erosion, as it adsorbs to soil particles suspended in runoff. If glyphosate reached surface water, it would not be broken down readily by water or sunlight. (EPA 1994).

Glyphosate has been found in surface and ground water (Rashin, Graber 1993, Wan 1986). It has been found two months after application in water (Goldsborough, Brown 1993) and much longer, more than a year, in sediments found at the bottom of ponds (U.S. EPA 1993). At an application rate typical of conifer release operations (3 kg/hectare), the maximum glyphosate concentration in runoff was 55 ug/liter one month after application (Edwards et al., 1980). This indicates that application of Roundup during the rainy season will result in surface water contamination by glyphosate, though only as high as 2 ppm, accidents excluded. The mobility of the Roundup surfactant (POEA) and the formaldehyde breakdown product of glyphosate in surface water runoff have not been studies. This is unfortunate, especially considering that these compounds have a much greater potential for contaminating water and pose a significantly greater hazard to wildlife and human health than glyphosate (Monroe 1988).

Wan found in an aerial application in British Columbia in 1986 that "...in a sediment sample from the unprotected [by a buffer zone] stream...Peak levels of 0.400 mg/kg glyphosate were detected at three weeks and three months post-spray, and...persisted for 574 days post-spray. [Glyphosate metabolite amino-methyl-phosphonic acid] residues were also found in the sediments, peaking at 0.400 three months after treatment [and]...showed a more gradual decline for the duration of the study (574 days).

In soil samples collected in California, glyphosate residues were found in all the 0 DAT samples analyzed to a depth of 24 inches (Oppenhuizen 1992).

"Glyphosate residue in a sediment sample from a protected stream was 0.200 mg/kg one week after an aerial spray application at 3 kg a.i./ha. Herbicide residues from vegetation along the stream bank or in the treated area may have washed into the creek following major rainstorm events."

CAT-33 CON.

Glyphosate and the 'inert' ingredients of Roundup are acutely toxic to fish and aquatic organisms; the surfactant 'inert' ingredient is much more toxic than glyphosate and the combined effect of glyphosate and the surfactant were more than additive. (Servizi, et al, 1987). One ingredient is 20 to 70 times more toxic to fish than glyphosate (Folmar, Sanders and Julin 1979.) Juvenile fish are particularly sensitive to the toxic effects of Roundup. Physical and chemical factors such as temperature, pH and solute concentration in aquatic ecosystems influence the acute toxicity of glyphosate to aquatic organisms (Caltrans 1991).

Wan et al found (1989) in a study of the effects of reconstituted water and four natural sources of dilution water on the acute toxicity to juvenile Pacific salmonoids and rainbow trout of glyphosate and formulated products that " a notable variation of 96-h LC50 values is attained for the same fish species when different water types are used in the bioassay of glyphosate."

Glyphosate has been shown to inhibit the growth of mycorrhizal fungi (organisms which are essential to ecosystems and enhance plant survival (FEMAT 1993)); in one laboratory study, mycorrhizal inhibition was shown at residue rates well below those found in soils following typical applications (Chakravarty,Sidhu 1990).

Non-target plants have been found to be more susceptible to disease after exposure to glyphosate-containing herbicides (Brammall, Higgins 1988, Mekwatanakam, Sivassithamparam 1987, Kawate 1997).

Roundup herbicide has a high potential for damaging desirable conifer species. Fisher et al. (1986) noted that glyphosate caused unacceptable seedling injury with the rate required to control weeds (1.68 kg a.i./ha) (Monroe 1988).

Acute (immediate) toxicity to mammals, birds, and bees is low, but no information is available regarding long term effects of glyphosate to these organisms. No data is available regarding the toxicity of glyphosate to soil invertebrates, reptiles or amphibians (Caltrans 1991).

Wildlife, such as birds and mammals could be variously affected through changes in vegetation, and through secondary changes in the abundance of arthropods. These indirect effects of glyphosate spraying, which are considered within the purview of "ecotoxicology", could affect the abundance and reproductive success of wildlife on the spray site, irrespective of any direct effects due to glyphosate toxicity (Stott et al 1991).

According to Raymond et al (1996) "There is evidence that moose browse more in sections of clearcuts...missed by aerial spraying than in treated sections. More information is needed on conifer regeneration and moose foraging behavior in

untreated strips...As with moose, the consequences of forest management with herbicides on other taxa will depend largely on habitat requirements of individual species...and will change during stand regeneration." CAT-33 CON.

Please refer to the *Cannabis Eradication in the Contiguous United States and Hawaii*, US Department of Justice, Drug Enforcement Administration, April 1998 as an example for a risk assessment. It is clear from what we know on the toxicity of the pesticides currently used, and to be used, that there is a need for a complete risk assessment on glyphosate and other listed pesticides.

WILDLIFE

Birds

The toxicity of pesticides to wildlife depends on several factors, including the persistence and degree of toxicity of the chemical. Factors such as dose, time and duration of application play important roles in a pesticide's toxicity. Wildlife, for example, are more susceptible to pesticide effects during nesting, nursing of young or during times of low food availability (USFWS, vol 1, no. 3). CAT-34

Primary exposure includes eating, drinking, preening feathers, skin contact or breathing of vapors. Secondary exposure occurs from scavenging on contaminated food, such as exposed carcasses, or feeding upon insects. If pesticide levels are high enough, wildlife often die suddenly (USFWS, vol 1, no. 3). It is often difficult to find poisoned birds because after most pesticide sprayings, no one searches for dead birds; and in the rare event that an application is reported as "monitored," there is apt to be just a single searcher (Williams 1997).

Roughly 672 million birds annually exposed to pesticides in the United States, 10 percent--67 million--are killed. This is a conservative estimate because it is extrapolated from the low range of mortality seen in study areas and it does not include such factors as the bird losses caused by poisoning of invertebrate prey, eggs left to chill or young birds left to starve when adults die, and birds with blown-out nervous systems that may migrate to places where they can't reproduce or survive (Williams 1997; PANUPS 1997).

Part of the phesant declines at Tule Lake National Wildlife refuge is contributed to poor habitat, which resulted in increased adult mortality, reduced nesting success, and reduced brood survival (Groves 1994).

Most chance discoveries have shown that about 40 pesticides, the bulk of them still registered in the United States, can kill birds even whn used according to the direction on the label (Williams 1997).

Sublethal, or non-fatal consequences of ingesting pesticies are not readily observed in wildlife. Behavior changes, weight loss, impaired or unsuccessful reproduction, high offspring mortality or deformed embryos are results of sublethal exposure to pesticides. Affected wildlife become easy prey for predators, and many lose their

ability to adapt to enviromental changes (USFWS, vol 1, no. 3).

Pesticides can reduce food sources for young birds and fish, and habitat is similarly reduced when vegetation is destroyed (USFWS, vol 1, no. 3). CAT-34

Other chemicals

You do not discuss the chemical toxicity of dyes, adjuvants, metabolites, contaminants and Inerts all of which can be highly toxic. Dyes are used to mark spray sites and can be very toxic (USDA FS FEIS 1988). Adjuvants enhance the efficacy of the pesticide and contain toxic chemicals. (A guide to Ag. Spray Adj. 1998) Inert ingredients can be more toxic than the active ingredient. (Monroe 1998).
(Anwar 1997- Environmental Health Perspectives) CAT-35

Other media

Soil types needs to be analyzed in order to understand the various mechanisms of environmental fate of the herbicides and their effects especially to wildlife (J. Environ. Qual.,Vol.15, no. 4, 1986;Wolkomir 1997;Wehtje et al 1987;Stark 1982; Somasundaram et al 1991) CAT-36

Other potential uses of pesticides

There are whole areas of potential pesticide use that neither PL in the Plans or you in the EIR/EIS ever mention. These involve the increase of pest pressure caused by changes in tree composition, in creating an almost entirely new forest, by fire and other forces. CAT-37

As Dr. Fields Cobb in his comments and Dr. Donald Dahlsten in the attached letter point out, the change in species composition proposed by PL in the SYP is likely to cause greatly increased pest pressure. (OR Dept of Forestry March 1988; Kanaskie & Bordelon 1996)Greatly increased pest pressure will result in increased reliance on pesticides. This is a reasonably forseeable result of the proposed SYP. (Lawsen 1986)

Nitrogen fixation is accomplished by some of the very plants that PL seeks to kill as competitors, such as Ceanothus. (Binkley et al 1982; Binkley & Husted 1982; NTC 1997-98)

What will be the result of removal of species with herbicides which, while not of immediate concern, nevertheless may impact forest health in general?

Have you considered the potential effects of exposing and possibility of harming species not of concern which are integral to the success of species of concern? Recent studies show the interdependency of species (Jones et al 1998); you should too.

Poor forest management in other areas of the country have caused reduced forest health and similar results could be anticipated here. (Rice 1998) Reduced forest health

leads to the use of pesticides to defend trees from introduced non-indiginous pests and pest levels that get out of whack when the forest is young and a plantation, such as when gophers become a problem.

37 CON.

Fire has been the cause of increased pest pressure on Forest Service land in California and subsequent requirement for vegetation control which FS has applied herbicides to tens of thousands of acres in recent years. (USFS 1994: Hamm-Hasloe and Cottonood). How will fire affect PL land as a result of the change of species composition and age of the trees and other vegetation on its land? What would resultant herbicide use entail?

And, finally, raw wood from outside North America is being imported to Humboldt Bay and other destinations in California. Isn't it a reasonable assumption that non-indiginous species could infest PL lands, particularly after species composition has weakened the 'forest' and made it vulnerable to pest infestation?

SYP/HCP

Our previous comments have been restricted almost entirely to the EIR/EIS. That's because herbicide use is barely mntion in either document despite evidence, as we have presented, that herbicides can provide a significant adverse impact. Both th SYP and th HCP should be rejected until rewritten to include herbicide use (SYP) and herbicide impacts (HCP) to an adequate degree.

Sincerely,

Patricia Clary

Nina Hapner

Laurynda Allison

Dan Zimmerman

Dan Zimmerman for PC