at maximum doses.[5] However, effects other than death have been observed in tests of imazapyr's acute toxicity. Bleeding and congested lungs were observed in rabbits dermally exposed to imazapyr and in rats inhaling Arsenal Railroad Applicators Concentrate or Arsenal Herbicide Applicators Concentrate.[6,7,8] Congestion of the kidney, liver, and intestine was also observed in laboratory tests.[8]

Eye irritation

Imazapyr is "corrosive" to the eyes and "causes irreversible eye damage."[8] Imazapyr-containing products are also irritating to the eyes: Arsenal Herbicide Railroad Applicators Concentrate caused eye irritation which subsided by 24 hours post-treatment,[9] and Arsenal caused eye irritation which subsided by 72 hours post-treatment.[8]

Skin irritation

Arsenal caused reddening, scaling, and crusting of treated skin at all doses tested in rabbits dermally exposed over a 21 day period.[10] With a single exposure, Arsenal and Arsenal Herbicide Railroad Applicators Concentrate caused swelling and redness, or just redness depending on whether or not the skin was abraded.[6,8]

Subchronic Toxicity

Oral administration of imazapyr to female rabbits over a 12 day period caused stomach ulcers and intestinal lesions at most doses tested.[10]

Chronic Toxicity

Laboratory studies in which mice were fed imazapyr for two years found the following chronic effects: fluid accumulation in the air sacs of the lungs in females; an increased incidence of congestion of the brain in females; and an increased incidence of kidney cysts in males.[11] In a two year feeding study with female rats, different symptoms were observed: an increase in abnormal blood formation in the spleen; an increase of blood pooling in the liver; an increase in thyroid cysts;[12] and a decrease in food efficiency (the ability to transform ingested food into body weight gain).[13] Most of these effects were not considered significant by EPA.[5]

There are no publicly available data concerning chronic effects of imazapyr-containing products.

Reproductive Effects

In a review of imazapyr toxicity concluded in 1992, the U.S. Forest Service and two other federal agencies concluded that "the potential for causing adverse effects on fertility or reproduction has not been determined at this time."[14] This is the most recent publicly available information. There are no publicly available data regarding the reproductive hazards posed by imazapyr-containing products.

Carcinogenicity

EPA has evaluated the potential of imazapyr to cause cancer and placed it in Class E, "evidence of noncarcinogenicity."[15] However, it is important to look at the data produced by the two-year feeding studies of both rats and mice on which this evaluation was based. The study using rats indicated the following carcinogenicity concerns: an increase in the number of brain tumors in male rats, an increase in the number of thyroid tumors and cancers in male rats, and an increase in the number of tumors and cancers of the adrenal glands in female rats.[12] EPA found that the frequency of thyroid and adrenal gland tumors and cancers did not increase above the levels found in other studies done by the same laboratory.[12] With respect to the brain tumors, American Cyanamid reanalyzed tissues from the original study. They discovered an additional tumor in the high-dose group, as well as an additional tumor in the untreated (control) group. EPA found that with the addition of the new data the increased incidence of brain tumors was no longer statistically significant.[5]

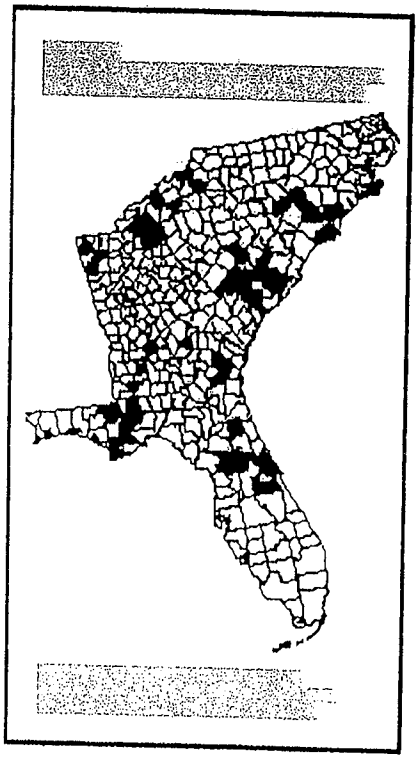
There is no publicly available data considering the carcinogenicity of imazapyr-containing products.

Effects on Nontarget Plants

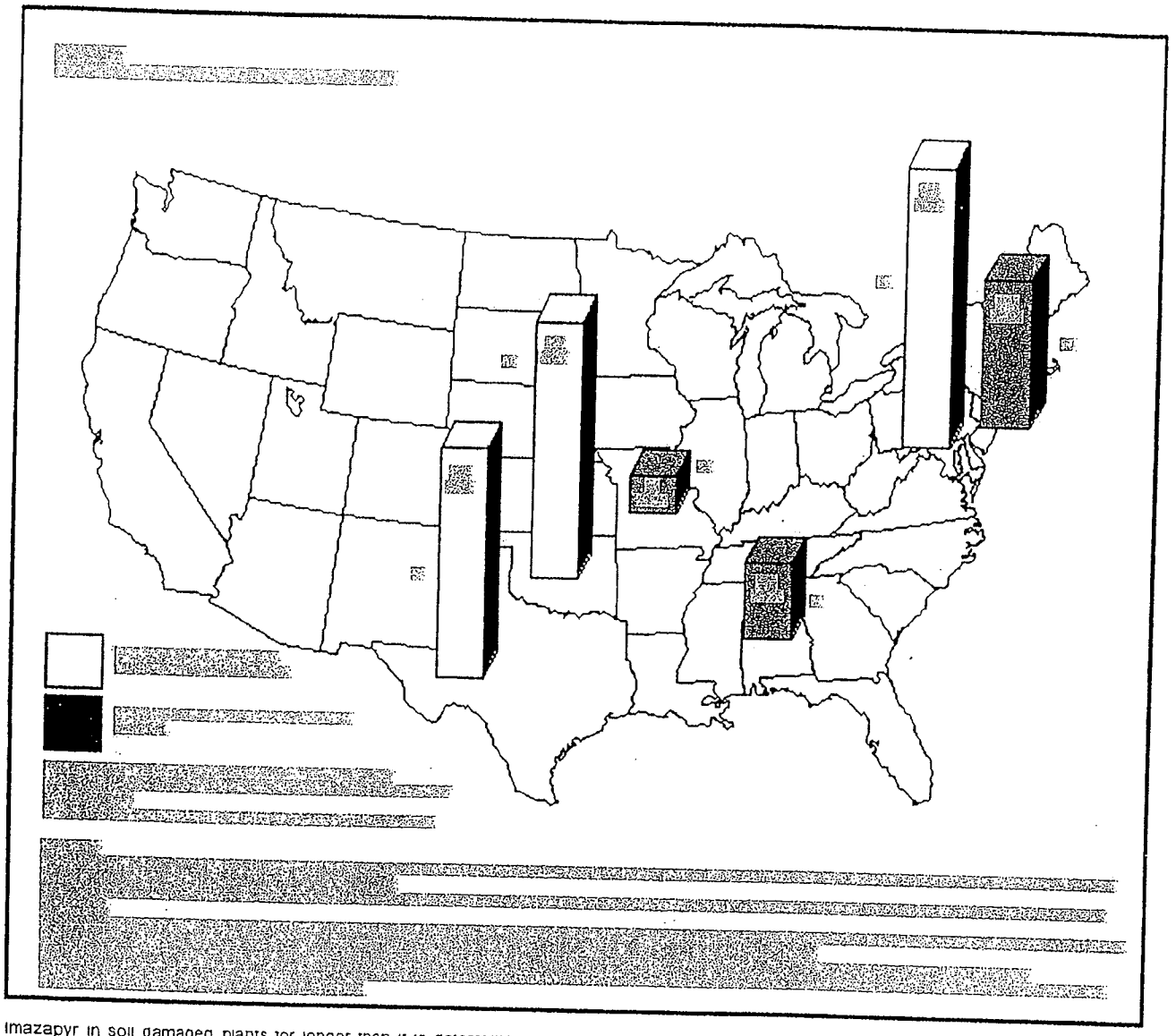
Like all broad spectrum herbicides, imazapyr efficiently kills most plants with which it comes in contact, even those not intended as targets of the herbicide. In addition to this acute toxicity to plants, a variety of other impacts have been reported in nontarget plants exposed to imazapyr. These include hazards to endangered species, increased susceptibility to disease, and disruption of nutrient cycling in soil.

Endangered species: Rare plants are particularly at risk from herbicide exposure because the loss of a few individuals can have significant consequences for a small population. EPA states that "a number of terrestrial and aquatic plant species are listed as being at jeopardy from the use of herbicides and that "jeopardy will also occur from the used of Arsenal."[16] The Fish and Wildlife Service has identified 100 counties in 24 states where endangered species could be at risk from forestry use of Arsenal. (See Figure 2 for a map of these counties in the southeastern U.S.) No such analysis for western states is publicly available.

Plant disease: When used in combination with the herbicide diuron, imazapyr



Counties containing endangered species that could be jeopardized by use of imazapyr. Similar information is available for most states east of the Mississippi River.



Imazapyr in soil damaged plants for longer than it is detectable through laboratory analysis. This difference is much greater than geographical differences in persistence.

increased the severity of the fungal leaf disease *Tubakia dryina* on water oak (*Quercus nigra*). The disease resulted in a significant decrease in stem growth when trees were exposed to the herbicides.[17]

Nutrient cycling: Decomposition of plant material (cellulose) is an important component of cycling nutrients through an ecosystem. Imazapyr can disrupt this cycling. In laboratory tests, imazapyr treatment of soil slowed decomposition of cellulose, and decreased the activity of an enzyme used by soil microbes to break down cellulose.[18]

Effects on Animals

According to three federal agencies, imazapyr's acute oral toxicity to birds, fish, and water fleas is low.[14] No studies have been conducted on imazapyr's chronic toxicity to any of these animals,[14] although a related herbicide (imazamethabenz-methyl) has high chronic toxicity to fish, with effects occurring at concentrations of less than 1 part per million.[19] In addition, there are no studies about the chronic toxicity of imazapyr-containing products.[14]

Persistence in Soil

Overall, imazapyr is a persistent herbicide. Persistence in field studies varies from 60[20] to 436 days,[21] with many studies reporting persistence of over a year.[21-23] (See Figure 3.) These are minimum estimates of persistence because imazapyr persisted, in most cases, until the last date tested.

Soil persistence of imazapyr, as with any

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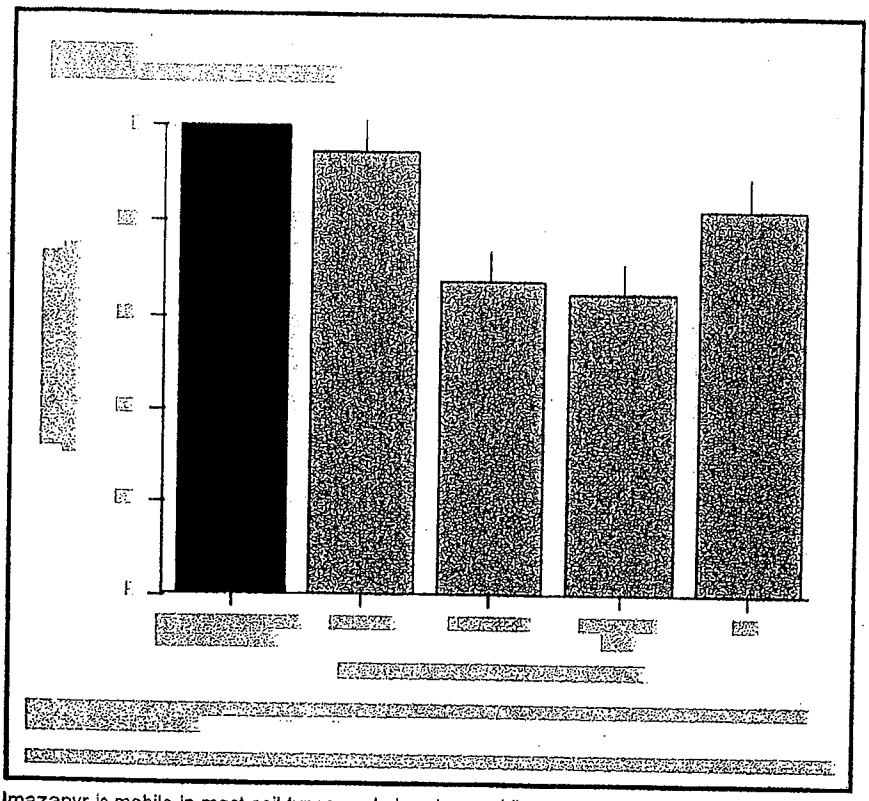

Imazapyr is mobile in most soil types, and almost as mobile as water in clay and clay loam.

pesticide, varies depending on climate, weather, soil type, and other factors. However, in the case of imazapyr, the most important factor appears to be the method used to detect imazapyr. The persistence studies measure the length of time between imazapyr application and the last detection of imazapyr residues by laboratory analysis or the last observation of imazapyr-caused plant injury. All of the studies using plant injury show longer persistence[21-23] than those that depend on laboratory analysis.[21,24,25] This suggests that imazapyr can cause plant damage at levels too low to detect by standard laboratory procedures. This problem has also been observed in another class of herbicides, the sulfonylureas, with the same mode of action as imazapyr.[26]

A common measure of persistence is half-life, the length of time required for half of the amount of a pesticide originally applied to break down or move away. EPA reports that imazapyr's half-life is 17 months in laboratory tests.[27] Half-lives ranging from 21 days to 49 months have been reported in field studies.[20,28] Consistent with the studies measuring persistence, the longest half-lives are reported in the studies that use plant injury to detect imazapyr.[28]

Water Contamination

Several of imazapyr's chemical characteristics mean that it is mobile in soil and thus likely to contaminate water. Researchers in Alabama found that it was more mobile in soil than the widespread water contaminant atrazine.[29] In this study it was nearly as mobile as water in some soil types. (See Fig. 4.) EPA found that it has a "moderate potential for sorption"[30] (ability to attach to the surface of soil particles) but a "high potential for desorption,"[30] when it would then be able to contaminate nearby water. One field study found that between 40 and 70 percent of applied imazapyr leached down to the lowest depth tested (45 cm).[28] Another study found that "significant" residues of imazapyr leached to a depth of between 1.5 and 3 meters (4.9 - 9.9 feet) depending on application rate.[31]

Little monitoring of imazapyr contamination of water has been done. However, the studies that have been conducted show that imazapyr does contaminate water.

In the southeastern U.S., imazapyr was found in surface water following aerial application at both of the two forestry sites for which data are publicly available. Management practices to reduce water contamination were employed at one of the sites.[32] Imazapyr was also found in groundwater following a forestry application using ground equipment in the only published study that tested for groundwater contamination.[33] In the Pacific Northwest, imazapyr was found in surface water in one out of the two sites monitored by the Washington Dept. of Ecology following aerial forestry applications. Again, management practices were used to reduce water contamination.[34]

Ozone degradation, a treatment used to remove pesticides from drinking water is not successful with imazapyr, removing only about half the imazapyr present.[34]

Drift

Imazapyr is a potent herbicide, so it is not surprising that drift of small amounts can severely damage valuable plants. For example, a study of the effect of simulated drift on yield and quality of potatoes found that amounts of imazapyr as small as 1/50 of the normal agricultural rate reduced potato yields to as little as one-third of unexposed plants. Yield of high quality (U.S. #1) potatoes decreased by 99 percent because folded, multiknobbed, and cracked potatoes were common.[36]

There are no publicly available data about the distance that imazapyr can drift.

Resistance

Resistance to imazapyr, the ability to tolerate amounts that typically would be lethal, has developed in a number of weed species from around the world. In general this resistance has not been observed following use of imazapyr. Instead, use of other herbicides with the same mode of action (primarily the sulfonylurea herbicides) has resulted in the development of cross-resistance, when resistance to one herbicide confers resistance to others. Species in which

resistance to imazapyr has been confirmed include rigid ryegrass (*Lolium rigidum*),[37] kochia (*Kochia scoparia*),[38] common chickweed (*Stellaria media*), Russian thistle (*Salsola iberica*), perennial ryegrass (*Lolium perenne*),[39] *Sonchus oleraceus*,[40] and *Arabidopsis thaliana*.[41] In addition, resistance has developed in an algae species, *Chlorella emersonii*.[42]

Plants that are resistant to imazapyr generally have a different form of the enzyme acetolactase synthase than susceptible plants. The resistant form of the enzyme is not as susceptible to inhibition by imazapyr as the susceptible form.[40,43] In at least one case, the resistant form of the enzyme is caused by a single point mutation.[41]

"Inert" Ingredients

Ingredients comprising about 47 percent of Arsenal Applicators Concentrate, the most common imazapyr-containing herbicide, are identified only as "inerts" by Arsenal's manufacturer.[44] There is no publicly available information about the identity of these ingredients. Most of EPA's hazard assessment of Arsenal is based on tests of imazapyr only and not on tests of all the ingredients.

Breakdown Products

There are two primary breakdown products of imazapyr when it is exposed to light.[45] One of them, quinolinic acid, is also a primary breakdown product in soil.[46] It is irritating to eyes, the respiratory system, and skin.[47] It is also a neurotoxin, causing nerve lesions and symptoms similar to Huntington's disease.[48]

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● HERBICIDE FACTSHEET

SULFOMETURON METHYL (OUST)

Sulfometuron methyl is an herbicide in the sulfonylurea chemical family. It is used mostly in nonagricultural situations, including roadsides and other rights-of-way, industrial facilities, and public lands. Oust is a common brand name for sulfometuron methyl products.

Sulfometuron methyl-containing herbicides cause eye discomfort, tearing, and blurred vision. In laboratory tests, sulfometuron methyl caused anemia, atrophied testicles and testicular lesions, and increased the incidence of fetal loss. A sulfometuron methyl breakdown product causes DNA damage in the colon of laboratory animals.

Because of limited monitoring, little is known about how often sulfometuron methyl contaminates rivers and streams. However, the U.S. Geological Survey found this herbicide in rivers in the Midwest, and the U.S. Forest Service found it in streams following forestry applications.

Enough sulfometuron methyl to kill desirable vegetation can persist in soil for a year after application.

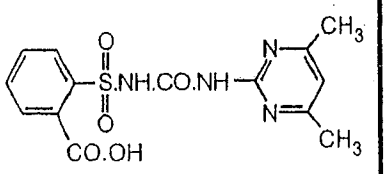
Minute amounts of sulfonylurea herbicides disrupt plant reproduction. For example, sulfometuron methyl's chemical relative chlorsulfuron reduces fruit production in cherry trees. This reduction is caused by amounts equivalent to 1/1000 of the typical agricultural rate. Experiments with peas, canola, soybeans, and smartweed had similar results.

Drift from roadside and noxious weed applications of Oust have resulted in widespread crop damage totaling millions of dollars.

BY CAROLINE COX

Sulfometuron methyl (see Figure 1) is an herbicide in the sulfonylurea chemical family. This is a relatively new family of herbicides, first marketed in the early 1980s. All sulfonylurea herbicides are extraordinarily potent, about 100 times more toxic to plants than older herbicides, and sulfometuron methyl is "one of the most potent"[1] of this family.[1] E.I. du Pont de Nemours and Company (DuPont) is the major manufacturer of sulfometuron methyl and markets the herbicide under the brand name Oust.[2-4]

Figure 1
Sulfometuron methyl



2-[[[[(4,6-dimethyl-2-pyrimidinyl)amino]carbonyl]amino]sulfonyl] benzoic acid

Use

Sulfometuron methyl is used as a broad spectrum herbicide (for total vegetation kill) in nonagricultural sites such as fence rows, roadsides and other rights-of-way, storage areas, industrial facilities, and public lands. It is also used as a selective herbicide on conifer plantations, hardwood plantations, and turf. To use it as a selective herbicide in these situations, applications must occur during times when the crop tree or turf is less susceptible to Oust (during winter dormancy for trees, and when turf is well established).[2-4]

Mode of Action

Sulfometuron methyl kills plants by stopping the division of growing cells, particularly cells in the tips of plant roots. On a molecular level, sulfometuron methyl inhibits the activity of an enzyme called acetolactate synthase (ALS). ALS is one of the enzymes used by plants to synthesize three specific amino acids, molecules that are used as components of proteins.[5] Animals do not have the ALS enzyme.[1]

"Inert" Ingredients

Like most pesticides, sulfometuron methyl herbicide products contain ingredients in addition to sulfometuron methyl. Many of these ingredients, according to U.S. pesticide law, are called "inert."[6] Some inert ingredients in sulfometuron methyl products have been identified by the U.S. Environmental Protection Agency (EPA). These include sugar, the sodium salt of a naphthalene-sulfonic acid formaldehyde condensate, the sodium salt of sulfated alkyl carboxylated and sulfated alkyl naphthalene, hydroxypropyl methylcellulose, and polyvinyl pyrrolidone. All are ingredients in Oust Herbicide (EPA Registration Number 352-401).[7]

The following six sections of this article discuss the toxicology of Oust herbicides, including information about sulfometuron methyl, Oust's inert

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ingredients, and sulfometuron methyl breakdown products. With the exception of the studies of eye irritation in the next section, all of the studies summarized were conducted on a single ingredient, not on the combination of ingredients found in sulfometuron methyl herbicide products.

Eye Irritation and Injury

Commercial sulfometuron methyl-containing herbicides can injure eyes. Oust and Oust XP both cause eye discomfort, tearing, and blurred vision. A third sulfometuron methyl herbicide product, Oustar, is corrosive to eyes and causes irreversible eye injury.[8-10]

Effects on the Circulatory System

Sulfometuron methyl causes anemia. In a laboratory study that was conducted by sulfometuron methyl's manufacturer (DuPont), dogs fed sulfometuron methyl for a year had fewer red blood cells and less hemoglobin in their blood than unexposed dogs. These effects occurred at lower doses in females than they did in males.[11] (See Figure 2.)

Sulfometuron methyl also affects white blood cells. In a laboratory study conducted by DuPont, rats fed sulfometuron methyl for three months had more white blood cells,[11] including a special kind of white blood cell called a lymphocyte,[12] than unexposed rats.[11] These effects occurred at a dose level of approximately 400 milligrams per kilogram (mg/kg) per day.[11]

Effects on the Lungs

According to the International Agency for Research on Cancer, exposure of people to polyvinyl pyrrolidone, an Oust ingredient, "may be accompanied by pulmonary fibrosis and pneumonia."[13] Pulmonary fibrosis is the development of fibrous tissue in the lungs.[12]

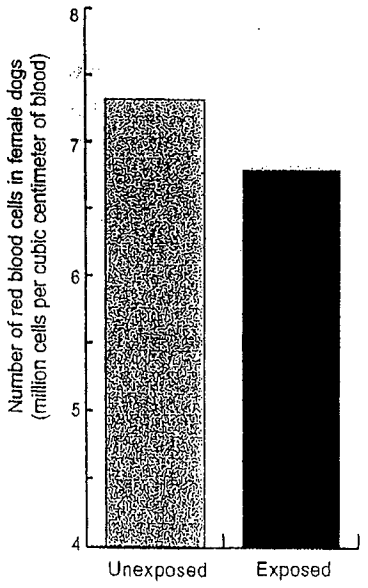
Effects on Reproduction

Exposure to sulfometuron methyl can disrupt successful reproduction in a variety of ways according to laboratory studies conducted by DuPont.

Effects on the testes have been documented in studies with both rats and dogs. The study with rats was a small study in which rats were fed high doses of sulfometuron methyl for 10 days. Two of the six rats developed testicular problems: one had abnormally small testes and another developed lesions.[14] In a study in which dogs were fed sulfometuron methyl for a year, the testes of three of the six dogs exposed to sulfometuron methyl degenerated and two dogs developed atrophied testicles.[11] (See Figure 3.) No abnormal testes occurred in unexposed animals.[11,14]

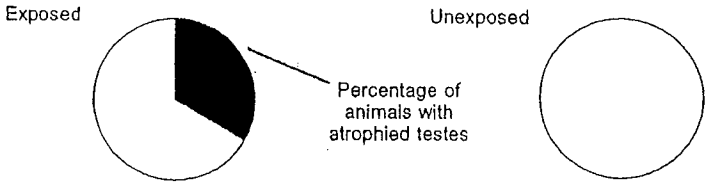
Effects on pregnant animals have been documented in both rats and rabbits. The offspring of rats exposed to sulfometuron methyl during the middle

Figure 2
Sulfometuron Methyl Causes Anemia



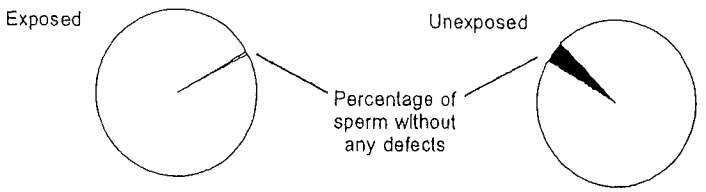
Source:
U.S. EPA. 1983. E.I. duPont. Oust Weed Killer, ID#352-401, Caswell #561D, Accession #250590. Memo from A. Arce, Toxicology Branch, to R. Taylor, Registration Div. Washington, D.C., Dec. 1.

Figure 3
Sulfometuron Methyl Causes Atrophied Testes



Source: U.S. EPA. 1983. E.I. duPont. Oust Weed Killer. Memo from A. Arce, Toxicology Branch, to R. Taylor, Registration Div. Washington, D.C., Dec. 1.

Figure 4
An Oust "Inert" Ingredient Damages Sperm



Source: Strehler, E. et al. 1998. Detrimental effects of polyvinylpyrrolidone on the ultrastructure of spermatozoa. *Hum. Reprod.* 13: 120-123.

Sulfometuron methyl caused anemia and atrophied testes in a laboratory test. In addition, an "inert" ingredient in Oust damages sperm.

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part of their pregnancies were smaller than offspring of unexposed animals. This effect occurred at a dose level of about 400 mg/kg per day.[11] In a two-generation study, feeding of sulfometuron methyl decreased the number of offspring in a litter. This effect also occurred at a dose level of about 400 mg/kg per day.[14] In rabbits exposed during the middle part of their pregnancies sulfometuron methyl increased the incidence of fetal loss.[15]

In addition, the Oust ingredient polyvinyl pyrrolidone damages sperm. In a study conducted by a researcher at the Institute for Reproductive Biology (Germany) and his colleagues, the proportion of undamaged (human) sperm was ten times less for sperm exposed to polyvinyl pyrrolidone than for unexposed sperm.[16] (See Figure 4.)

Mutagenicity (Ability to Cause Genetic Damage)

According to DuPont, "sulfometuron methyl did not produce genetic damage in bacterial or mammalian cell cultures."[10] However, sulfometuron methyl can break down into saccharin[17] (a compound that is used as an artificial sweetener) and that compound causes genetic damage. According to a study conducted by researchers at the Hachinohe National College of Technology (Japan), a single (large) dose of saccharin causes DNA damage in the colon of laboratory animals.[18] DNA is the molecule inside cells that carries genetic information.[12]

Carcinogenicity (Ability to Cause Cancer)

According to DuPont, "animal testing indicates that the active ingredient, Sulfometuron Methyl, does not have carcinogenic effects."[8] However, another Oust ingredient, polyvinyl pyrrolidone, "was tested for carcinogenicity in mice, rats, and rabbits by several routes of administration, providing local tumours" according to an evaluation by the International Agency for Research on Cancer.[13] These tumors were mostly a kind of cancer called sarcomas.[19]

Contamination of Water

Use of sulfometuron methyl can result in contamination of rivers and streams, although monitoring for sulfonylurea herbicides in water is limited and "little is known about their occurrence, fate, or transport in surface water or ground water in the United States."[20]

The U.S. Geological Survey found sulfometuron methyl in river and stream samples in agricultural areas in the Midwest, and the U.S. Forest Service found sulfometuron methyl in streams following forestry applications in Mississippi and Florida.[20,21]

" The U.S. Geological Survey found sulfometuron methyl in river and stream samples in agricultural areas in the Midwest, and the U.S. Forest Service found sulfometuron methyl in streams following forestry applications in Mississippi and Florida."

Incidents in which water contaminated with Oust damaged desirable vegetation have been reported from Tennessee, Pennsylvania, Arkansas,[22] and Washington.[23]

Sulfometuron methyl also has the potential to contaminate groundwater. Like all sulfonylurea herbicides, it is "relatively mobile in soil,"[24] according to EPA, and has a "high intrinsic leaching potential."[24] Its breakdown products have similar characteristics.[24]

Effects on Frogs

Sulfometuron methyl causes a variety of developmental effects on frogs. A study conducted by The Stover Group (an environmental consulting firm) showed that sulfometuron methyl inhibited tail resorption in tadpoles. (Tail resorption is part of the process by which a tadpole matures into a frog.) This inhibition occurred at a concentration of 10 parts per million (ppm) with chemically purified sulfometuron methyl and 1 ppm if not purified.[25]

Other effects on frog development of sulfometuron methyl (not chemically purified) include malformed limbs and increased mortality. These effects occurred at concentrations of 5 and 10 ppm respectively.[25]

The Stover Group researchers concluded that these developmental effects are caused by sulfonylurea herbicides' ability to disrupt thyroid function, and stated that such disruption "is capable of producing a myriad of deleterious effects."[25]

Effects on Fish

Concentrations of sulfometuron methyl required to kill fish are relatively high (over 12.5 ppm).[26] Because of this low aquatic toxicity, a fish kill related to Oust use is of particular interest. In spring 1983, the Tennessee Department of Transportation sprayed about 6,000 acres of rights-of-way. Heavy rains followed the spraying, resulting in fish kills.[22]

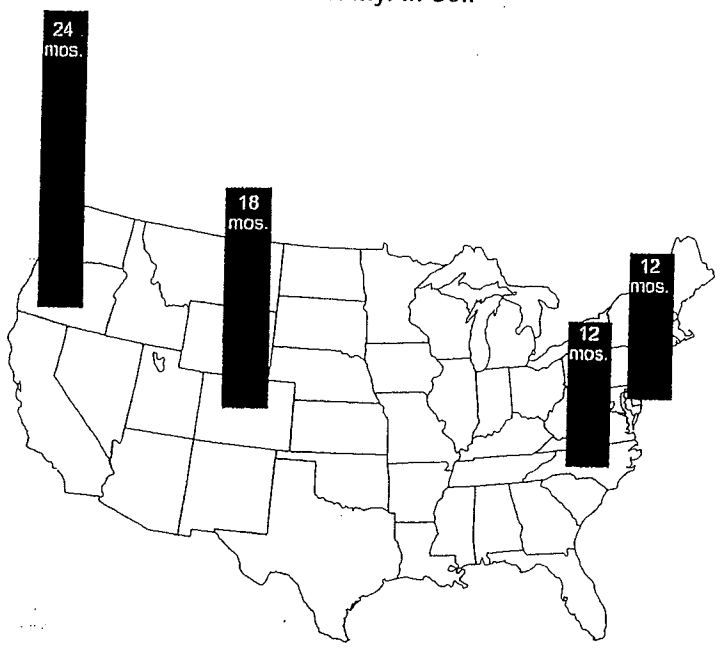
Effects on Algae

Low concentrations of sulfometuron methyl kill algae. In a laboratory test conducted by DuPont, concentrations above 0.63 parts per billion (ppb) killed the green algae *Selenastrum capricornutum*.[27]

Persistence in Soil

There is no simple answer to the question, "How long does sulfometuron methyl remain in soil?" EPA classifies sulfometuron methyl as "moderately persistent."[28] Oust's half-life (time required for half of the applied sulfometuron methyl to break down or move away from the application site) has been measured by DuPont researchers to vary between 12 and 25 days[29] and by Forest Service researchers to vary between 5 and 33

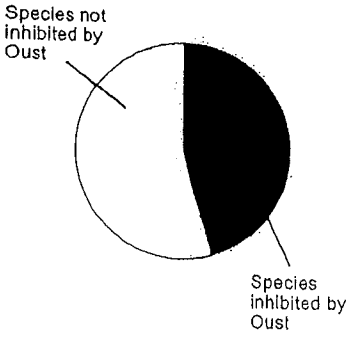
Figure 5
Persistence of Sulfometuron Methyl in Soil



Source: Anderson, J.J. and J.J. Dulka. 1985. Environmental fate of sulfometuron methyl in aerobic soils. *J. Agric. Food. Chem.* 33:596-602.

According to field studies conducted by Oust's manufacturer, sulfometuron methyl persists in soil between one and two years.

Figure 6
Effect of Oust on Soil Microorganisms



Source: Burnet, M. and B. Hodgson. 1991. Differential effects of the sulfonylurea herbicides chlorsulfuron and sulfometuron methyl on microorganisms. *Arch. Microbiol.* 155:521-525.

Oust inhibited almost half of the species of soil microorganisms studied in a laboratory test.

days.[21]

It takes at least a year for enough Oust to break down so that treated land can be used for crops. The label for Oust® Herbicide states, "If noncrop or forested sites treated with OUST® XP are to be converted to a food, feed, or fiber agricultural crop, or to a horticultural crop, do not plant the treated sites for at least one year after the OUST® XP application."[3] Even after one year, the label recommends field testing prior to planting a crop.[3]

This label requirement is based on the time required for nearly all of the applied sulfometuron methyl to break down. This can be long. For example, a second DuPont study measured sulfometuron methyl residues two years after applications made in Oregon and 18 months in Colorado.[30] (See Figure 5.)

One of Oust's common uses is to control vegetation along roadsides, which often have alkaline soils because of lime and limestone used during construction. Alkaline soils increase the persistence of sulfometuron methyl.[1,31]

Effects on Soil Microorganisms

Because some soil microorganisms rely on acetolactate synthase, the enzyme inhibited by sulfonylurea herbicides, they are quite susceptible to sulfometuron methyl. Microbiologists at the University of Melbourne (Australia) found that, of eleven soil bacteria studied, the growth of five species was inhibited by treatment with Oust. (See Figure 6.) The biologists concluded that application of Oust "would have significant effects on the microbial ecological balance of the soil."[32]

A study of Christmas tree weed management in Kentucky demonstrated this kind of effect on soil ecology. The study compared the use of Oust and sawdust mulches in a Christmas tree plantation. The foresters, from the University of Kentucky, showed that Oust treatment (compared to untreated areas) had a negative impact on the abundance of microorganisms and decreased the soil nitrogen content, while sawdust treatment increased both microorganisms and nitrogen content.[33]

Effects on Plants

Sulfometuron methyl is extraordinarily potent; for example, a concentration of 0.1 ppb in soil kills sugarbeets,[28] and a concentration of 0.06 ppb in water reduces growth of the native aquatic plant common water milfoil.[34]

Another illustration of its extraordinary potency is that the Oust label prohibits using equipment that has been used to apply Oust to apply any other pesticide. "This is extremely important," states DuPont, "as low rates of OUST® XP can kill or severely injure most crops."[3]

However, even more extraordinary is the ability of this herbicide family to disrupt plant reproduction at exposure levels that are far less than the tiny amounts needed to kill plants.

EPA researchers have conducted a series of studies with sulfometuron methyl's chemical relative chlorsulfuron that document how minute exposures

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reduce fruit or seed production. In the first study, looking at cherry trees, spring applications (when immature cherries were about half of their full size) of chlorsulfuron equivalent to one thousandth of typical agricultural application rates reduced the amount of fruit produced. Fall applications at similar low levels caused fruit production to drop the following year. Neither fall nor spring applications caused visual damage to leaves, branches, or other vegetative parts of the tree.[35]

The subsequent studies looked at impacts on other plants: garden peas, canola, soybeans, sunflower, and smartweed. Results were similar. Exposures equivalent to two thousandths of typical application rates reduced canola, soybean, and smartweed seed production; exposures of four thousandths of typical rates reduced pea production; and slightly higher exposures impacted sunflowers. (See Figure 7.) Again, reductions in fruit and seed production often occurred without visible signs of injury to the vegetative parts of the plant.[36,37]

The first of these studies begins by pointing out that sulfonylurea herbicides could have a "devastating impact."[35] The researchers' conclusions are sobering: "drifting sulfonylureas may severely reduce both crop yields and fruit development on native plants, an important component of the habitat and foodweb for wildlife."[35]

Effects on Endangered Species

Because sulfometuron methyl is a potent broad spectrum herbicide, it can kill endangered plants if they are exposed. In 1983, the U.S. Fish and Wildlife Service completed a formal consultation with EPA as required by the Endangered Species Act regarding potential impacts on endangered species of right-of-way and ditch bank use of Oust. The Fish and Wildlife Service identified 25 endangered plant species in 13 states that occur on or near rights-of-way and pointed out that "because of the limited population size of many of these plant species, a local spraying program could virtually destroy the entire species."[38]

In the 1983 consultation, the Fish and Wildlife Service suggested prohibiting rights-of-way uses of Oust in counties that were home to the 25 identified endangered species.[38] However, Oust labels merely prohibit use in five counties in Colorado;[3] there are only voluntary guidelines to protect endangered species elsewhere.[39]

Wind Transport

Off-target movement of sulfometuron methyl has caused dramatic crop damage. For example, an investigation by the Idaho Department of Agriculture recently (2002) concluded that several million dollars worth of crops were damaged by wind transport from an aerial Oust application made by the Bureau of Land Management to kill cheatgrass following a wildfire.[40] Over a hundred farmers and ranchers reported damage on over 100,000 acres.[41]

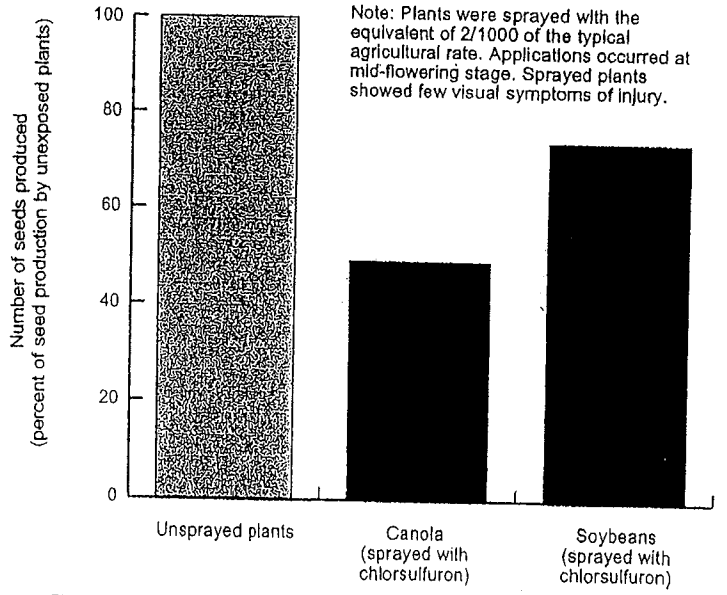
The first well-documented, large-scale Oust wind transport incident occurred in 1985. County and state road crews applied Oust to over 700 miles of roadsides in Franklin County, Washington, and subsequent wind transport caused over a million dollars of damage. In one nursery over 300,000 young trees were damaged.[42]

Investigation and documentation of these incidents has been hampered because, until recently, analytical methods for detecting sulfonylurea herbicides were not sensitive enough to detect the low concentrations of these herbicides that caused plant damage.[1] This meant that there was no way to analytically determine the cause of damage caused by low sulfometuron methyl concentrations. In 2001, however, the U.S. Geological Survey developed a sophisticated analytical method for detecting sulfonylurea herbicides at concentrations of 10 parts per trillion.[20]

Resistance

Weeds that are resistant (able to tolerate exposure) to sulfonylurea herbicides, including sulfometuron methyl,

Figure 7
Effects on Plants of Exposure to Minute Amounts of a Sulfonylurea Herbicide



Source: Fletcher, J.S. et al. 1996. Potential impact of low levels of chlorsulfuron and other herbicides on growth and yield of nontarget plants. *Environ. Toxicol. Chem.* 15: 1189-1196.

Exposure of plants to extremely small amounts of sulfonylurea herbicides while they are flowering causes significant reductions in seed production.

are widespread. At least 73 weed species have developed resistance.[43]

Characteristics of resistance to sulfometuron methyl that raise special concerns include the following:

- **Cross resistance.** This type of resistance occurs when plants that have developed resistance to one herbicide are also resistant to other herbicides. Weed populations that have developed resistance to sulfometuron methyl can be resistant to other unrelated herbicides. For example, scientists from Alberta, Canada's agriculture department studied a false cleavers population that was resistant to a broad range of sulfonylurea herbicides, including sulfometuron methyl. The population was also resistant to quinclorac,[44] an auxin-type herbicide that acts by mimicking natural plant growth hormones.[45] Cross resistance also occurs with herbicides that share sulfometuron methyl's mode of action but are from other chemical families.[46;47]
- **Frequent occurrence.** Genes that confer sulfometuron methyl resistance are found in weed populations that have never been sprayed with this herbicide. For example, University of Western Australia researchers studying rigid ryegrass populations that had never been treated with sulfonylurea herbicides found sulfometuron methyl-resistant individuals in all three populations studied.[48] This means that weed populations can develop resistance to sulfonylurea herbicides quickly. Resistant populations of weeds have appeared after only four applications of a sulfonylurea herbicide.[1] ✦

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● HERBICIDE FACTSHEET

CLOPYRALID

The herbicide clopyralid is commonly sold under the brand names Transline, Stinger, and Confront. It is used to kill unwanted plants in lawn and turf, range, pasture, rights-of-way, sugarbeets, mint, and wheat.

Clopyralid and the products containing it are irritating to eyes, some severely. The eye hazards of four clopyralid products include permanent impairment of vision or irreversible damage.

In laboratory tests, clopyralid caused what a U.S. Environmental Protection Agency (EPA) reviewer called "substantial" reproductive problems. These include a reduction in the weight of fetuses carried by rabbits who ingested clopyralid, an increase in skeletal abnormalities in these fetuses at all doses tested, and an increase in the number of fetuses with hydrocephaly, accumulation of excess fluid around the brain.

"Inert" ingredients in clopyralid products include cyclohexanone (produces tearing and burning of the eyes, vomiting, diarrhea, and dizziness), triethylamine (a severe eye irritant and cause of chemical pneumonia), and polyethoxylated tallow amines (cause eye burns, nausea, and are acutely toxic to fish).

Clopyralid is "persistent" in soil, according to an EPA review, and field studies have measured persistence as long as 14 months. It has the chemical characteristics that make it a likely water contaminant; despite its relatively low level of use it has been found in 2 of the 20 river basins studied by the U.S. Geological Survey.

Potatoes are extremely sensitive to clopyralid with damage occurring when plants are exposed to 0.07 percent of typical agricultural rates. When tubers from these damaged plants were grown in unsprayed fields, the new generation of plants also showed damage symptoms.

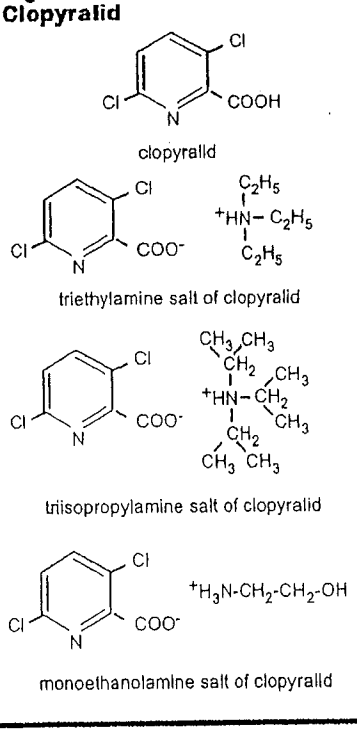
BY CAROLINE COX

Clopyralid (3,6-dichloropicolinic acid; see Figure 1) is an herbicide used to kill unwanted annual and perennial broadleaf plants in turf and lawn, range, pasture, rights-of-ways, and some agricultural crops.[1] Its primary manufacturer is Dow AgroSciences and it has been registered for use in the U.S. since 1987.[2] The acid form of clopyralid and three clopyralid salts (triethylamine, triisopropylamine, and monoethanolamine) are commonly used in commercial herbicide products.[3-11] Common brand names under which clopyralid is sold include Transline, Stinger, and Reclaim.[3-5] It is also sold in combination with other herbicides including triclopyr, MCPA, and 2,4-D; brand names of these mixes include Confront, Curtail, Scorpion, Hornet, and Accent Gold.[6-11]

■

Caroline Cox is JPR's editor.

Figure 1
Clopyralid



Use

The National Center for Food and Agricultural Policy estimates that agricultural uses of clopyralid in the U.S. total about 89,000 pounds per year. Most of this is used on sugarbeets, mint, and wheat.[12] (See Figure 2.)

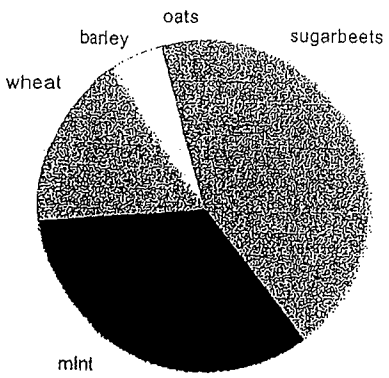
Mode of Action

Clopyralid is a synthetic plant growth hormone and has some structural similarities to naturally occurring hormones called auxins. It disrupts plant growth by binding to molecules that are normally used as receptors for the natural growth hormones.[1] Because clopyralid is more persistent in plant tissue than auxins,[13] the binding causes abnormal growth leading to plant death in a few days or weeks, depending on the species.[1] Clopyralid is similar in structure and mode of action to the herbicide picloram.[13]

Acute Toxicity

In laboratory rats, symptoms of

Figure 2
Agricultural Uses of Clopyralid in the U.S.



Total: 89,110 pounds per year

Gianessi, L.P. and J.E. Anderson. 1995. Pesticide use in U.S. crop protection. Washington, D.C: National Center for Food and Agricultural Policy, Feb.

clopyralid poisoning include watery eyes, diarrhea, and lethargy. These symptoms appear between 2 and 48 hours after clopyralid ingestion.[14] Acute exposure to clopyralid is also "severely irritating" to eyes, with symptoms (opaque cornea, inflamed iris, redness, and discharge) lasting up to 21 days after exposure.[15]

Commercial clopyralid products also damage eyes. Confront "may cause severe irritation"[6] with effects that can be "slow to heal."[6] These effects include "blurred, smoky, or halo vision."[6] Curtail, Scorpion, and Hornet "may cause permanent impairment of vision, even blindness."[7,9,10] Accent Gold "causes irreversible eye damage."[11]

In addition, clopyralid herbicides can irritate skin. Material safety data sheets for the products Hornet, Reclaim, Transline, Stinger, Curtail M, Curtail, and Scorpion all warn, "Prolonged exposure may cause skin irritation."[3-5,7,8,10] Repeated exposure to Confront "may cause allergic skin reactions in some individuals,"[6] and Accent Gold "may cause skin sensitization."[11]

Subchronic Toxicity

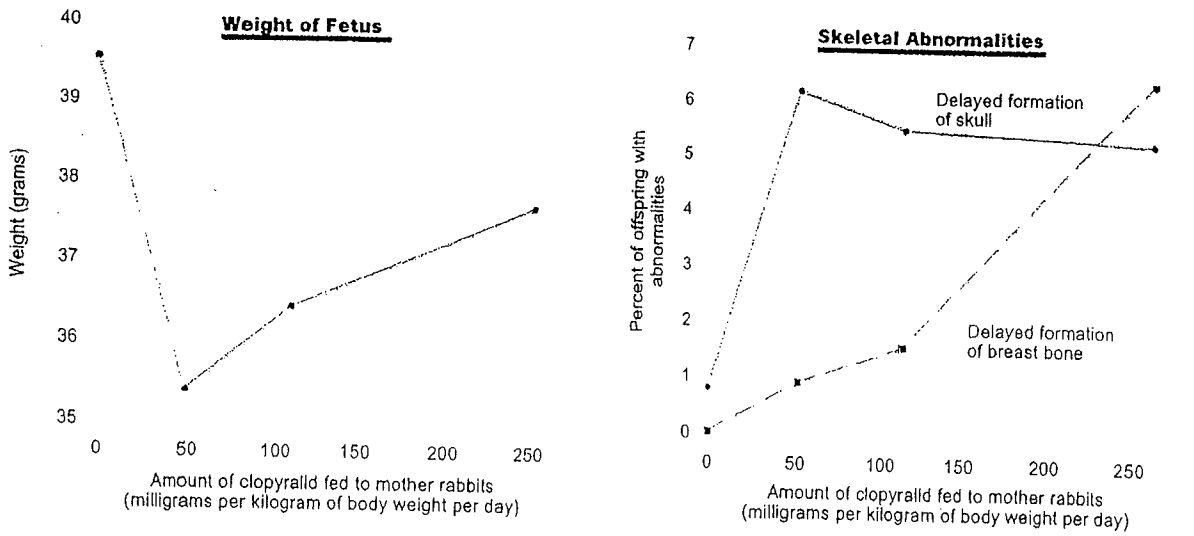
Subchronic (medium term exposure) studies of laboratory animals exposed to clopyralid have identified a variety of adverse effects. A three-month study with mice found an increase in the size of cells in the liver in females at two of the four doses tested. At the high dose, males showed similar effects and liver weights were increased in both sexes. A six-month study with dogs found increased liver weights in females, and urinary tract problems in males. Both effects were found at the highest dose tested.[16]

There are no publicly available studies of the subchronic effects of clopyralid-containing products.

Chronic Toxicity

Chronic (long-term exposure) studies of laboratory animals have identified effects on the stomach, liver, blood, and body weight. A two-year study with rats found hyperplasia of the stomach lining,

Figure 3
Reproductive Effects of Clopyralid



Source: U.S. EPA. Office of Pesticides and Toxic Substances. 1991. 3,6-dichloro-2-pyridinecarboxylic acid (clopyralid): Review of a rabbit teratology study submitted by the registrant. Memo from T.F. McMahon, Health Effects Division, to E. Wilson, Registration Division. Washington, D.C., Mar. 20.

Clopyralid's reproductive hazards include reduced weight of offspring and an increased incidence of skeletal abnormalities

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an increase in the number of cells leading to an enlargement of the lining, at all but the lowest dose tested. A one-year study with dogs found an increase in liver weights and a decrease in the number of red blood cells, also at all but the lowest dose tested. A two-year study with mice found decreased weight of males at the highest dose tested.[16]

There are no publicly available studies of the chronic effects of clopyralid-containing products.

Effects on Reproduction

According to a U.S. Environmental Protection Agency (EPA) reviewer, laboratory tests have demonstrated that exposure to clopyralid results in "substantial"[17] toxicity to fetuses and birth defects. (See Figure 3.) In a test with rabbits, clopyralid caused a decrease in the weight of the fetuses at both the low and the high dose tested. (At the middle dose the decrease in fetal weights occurred but was not large enough to be statistically significant.) In the same study, EPA also found that "developmental toxicity in the form of skeletal abnormalities was evident at all dose levels tested."[17] These skeletal abnormalities included delayed bone formation in the skull, pubic bone, and breast bone. At the highest dose tested, a "substantial increase was found in the number of fetuses with hydrocephaly,"[17] accumulation of excess fluid around the brain resulting in a small brain and an enlarged skull.

There are no publicly available studies of the reproductive effects of clopyralid-containing products.

Carcinogenicity

EPA has not evaluated the ability of clopyralid to cause cancer. In addition, there are no publicly available studies of the cancer-causing ability of clopyralid-containing products.[18,19]

"Inert" Ingredients

Like most pesticide products, clopyralid products contain so-called inert ingredients. These are ingredients added to the pesticide product to make it more potent or easier to use. Only limited information about their identities is publicly available. The following inert ingredients have been identified in clopyralid-containing products:

Curtail M contains cyclohexanone.[8] It produces eye irritation, tearing, and burning pain. It also causes skin irritation, nausea, vomiting, and diarrhea. It may cause liver and kidney damage, headache, dizziness, drowsiness, and nausea. Inhalation of cyclohexanone may be fatal as a result of spasms, inflammation, and fluid accumulation in the lungs.[20]

Curtail contains triisopropanolamine.[7] It causes eye irritation, and may cause skin irritation. Inhalation of triisopropanolamine may irritate the respiratory tract, and may be fatal as a result of spasms, inflammation, and fluid accumulation in the lungs.[21]

Confront, Confront Weedstick, Confront F, and two Confront-fertilizer combinations contain triethylamine (N,N-diethylanamine), ethylenediaminetetraacetic acid (EDTA), and polyethoxylated tallowamine (POEA).[22] Triethylamine causes severe eye irritation, and may cause "blue haze" vision. It also causes skin irritation, and respiratory tract irritation which may lead to chemical pneumonia. It may irritate the digestive tract.[23] EDTA causes eye irritation, skin irritation, and respiratory tract irritation. In laboratory tests it has caused reproductive problems, including stunting and death of fetuses and abnormal fetal development.[24,25] POEA causes eye burns; skin redness, swelling, and blistering; nausea; and diarrhea.[26,27] Concentrations of POEA between 1 and 5 parts per million kill fish.[26]

Persistence in Soil

Clopyralid is "persistent"[28] in soil, according to EPA, with a half-life (the time required for one-half of the amount of clopyralid to break down or move away from a test site) of "up to 11 months."[28] Several studies have measured persistence under field conditions; clopyralid persisted in soil between 2 and 14 months depending on soil type, climate, and other factors.[29-31] (See Figure 4.) Enough clopyralid persists in soil that lentils, safflower, and peas are damaged 220 days after treatment,[31] and a fall application caused "severe potato growth reductions" in potatoes planted the next spring.[32]

Persistence in Compost and Mulches

Compost and mulches made from clopyralid-treated plants contain residues. A study from Michigan State University measured clopyralid in grass clippings composted for up to one year.[33] Another study found that potatoes mulched with mint hay were damaged when the hay was made from plants treated with clopyralid the preceding spring.[34] In a greenhouse experiment in which clopyralid-damaged bean plants were used as a soil amendment for subsequent plantings of beans, damage was visible for three generations.[35]

Contamination of Water

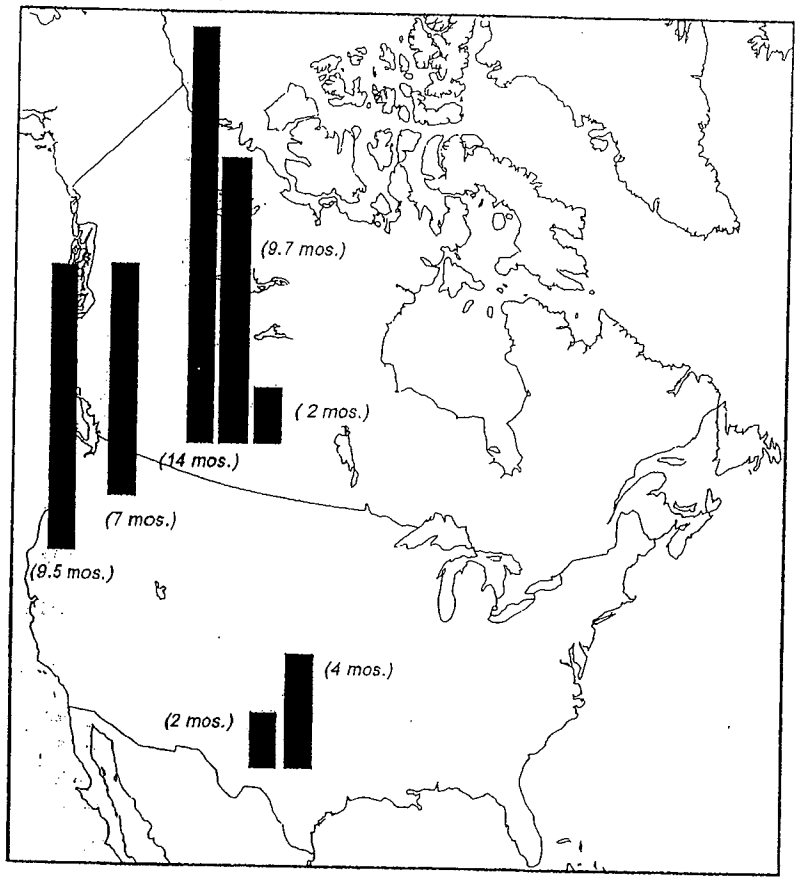
EPA described clopyralid as "very soluble"[36] in water and "very mobile"[36] in soil and concluded that it "has the potential to leach to ground water and/or contaminate surface water."[36] Relative to other herbicides, the amount of clopyralid used in the U.S. is small: for example, its use is about 0.1 percent of that of atrazine, the most widely used herbicide in the U.S.[12] Atrazine is widely found as a water contaminant.[37] Despite this low level of use, the U.S. Geological Survey has found clopyralid in two of the twenty river basins it has sampled for pesticides, the Central Columbia Plateau (Washington and Idaho) and the Trinity River Basin (Texas).[38,39]

Under "worst case" experimental conditions (application to a harvested, cultivated field followed by irrigation), clopyralid was found in soil water samples at all depths and dates tested, up to 30 days after treatment and down to a depth of 1.8 meters (almost 6 feet).[40]

Effects on Crop Plants

Clopyralid is "considered volatile,"[41] according to EPA, meaning that it can evaporate from foliage and soil after application, move away from the applica-

Figure 4
Persistence of Clopyralid in Soil



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Depending on soil type and climate, clopyralid can persist up to 14 months in soil.

tion site, and "adversely affect nontarget broadleaf plants."[41] EPA calculated that volatilization of only one percent of applied clopyralid would be enough to damage nontarget plants.[41] For a sensitive crop plant, the amount causing damage is even smaller. Potato plants showed damages after exposure to 0.07 percent of typical agricultural rates, and 0.7 percent reduced potato yields.[42] Potatoes are so sensitive to clopyralid that effects can be seen in plants the year following exposure. When tubers from clopyralid-damaged plants were grown in an unsprayed field, clopyralid injuries were visible in the new generation of potato plants.[43]

Hazards to Endangered Species

EPA has identified 11 species of endangered plants which could be jeopardized by the use of clopyralid.[44] Five of these species are rare cactus species. The impact of clopyralid on endangered cactus has been studied in Arizona, by doing experiments with close relatives of the endangered species. The experiments showed that some of the cactus species were impacted for months following clopyralid treatment. Of the five species studied, one showed reduced vigor 16 months after treatment, one showed reduced survival and vigor for six months (this species is not easy to keep alive, and could not be evaluated for 16 months), and one showed reduced survival for 16 months.[45]

Effects on Plant Communities

Clopyralid is used in plant community restoration efforts in an attempt to kill alien (weed) species and promote the growth of native species. A study conducted in Glacier National Park found that clopyralid-based restoration has mixed results. After a highway in the park was widened, researchers attempted to re-establish native vegetation along the roadside. Study plots were seeded with native seed mixes, and treated three times with clopyralid. The abundance of nonnative broad-leaved species decreased and the abundance of native grasses increased slightly. However, the herbicide treatment also reduced the abundance of native broad-leaved plants and increased the abundance of nonnative grasses.[46]

An experiment in the United Kingdom showed similar results. After sowing seed from native species, treatment with clopyralid reduced the abundance of one of the seeded broad-leaved species about 75 percent and completely eliminated flowering by that species. Clopyralid treatment also eliminated flowering by two other species and reduced flowering of a third species by 90 percent.[47]

Effects on Beneficial Insects

Since clopyralid is an herbicide, it is not surprising that plants are the primary target of its toxicity. However, clopyralid is also toxic to some beneficial insects, those insects that are economically important because they reduce populations

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of agricultural pests. The International Organization for Biological Control found that clopyralid is toxic to three species of beneficial insects: between 30 and 80 percent of test populations of a ladybug and a pirate bug were killed by clopyralid, as were between 25 and 50 percent of populations of a lacewing.[48]

Resistance

Resistance to clopyralid was documented in a yellow starthistle population in Washington. The starthistle grew in a pasture that was treated frequently with picloram over a ten year period. Greenhouse studies showed that the starthistle had 3-fold resistance to picloram. (The amount of picloram required to kill the plants was three times the amount required to kill plants that had not developed resistance.) For clopyralid, the starthistle showed 14-fold resistance.[49]

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● HERBICIDE FACTSHEET

2,4-D

2,4-D is one of the most widely used herbicides in the world. It is commonly used on rangeland and pasture, in the production of wheat, and on home lawns.

Symptoms of 2,4-D poisoning in exposed people include irritation and inflammation of eyes and skin, hives, nausea, vomiting, throat irritation, headache, dizziness, coughing, and difficulty breathing.

In laboratory animals, human cells, and exposed people 2,4-D caused genetic damage. Scientists have also demonstrated that 2,4-D affects hormones in exposed people and laboratory animals. Three recent laboratory studies indicate that 2,4-D has the ability to reduce the effectiveness of the immune system.

2,4-D (and the entire family of phenoxy herbicides) is classified as possibly carcinogenic by the International Agency for Research on Cancer. Studies of exposed farmers support this classification.

New studies indicate that 2,4-D reduces fertility in several ways. 2,4-D exposure is associated with low sperm counts. 2,4-D also damaged sperm and male sex organs in laboratory studies. When low doses of a commercial 2,4-D herbicide were fed to pregnant laboratory animals, average litter size was reduced by about 20 percent.

According to the most recent data collected by the U.S. Environmental Protection Agency, some 2,4-D is contaminated with 2,3,7,8-TCDD, a potent dioxin.

Monitoring by the U.S. Geological Survey showed that 2,4-D is frequently found in rivers and streams. It is also often measured in air samples.

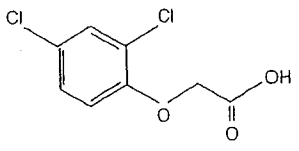
2,4-D use on lawns is linked with an increased risk of cancer in dogs.

2,4-D causes genetic damage in plants in amounts too small to cause visible damage to the plants.

BY CAROLINE COX

2,4-D (see Figure 1) is a chlorophenoxy herbicide. This herbicide family is said to have "initiated an agricultural revolution"[1] when it was first marketed in the 1940s. 2,4-D is also commonly used in weed and feed products[2] and is "one of the most widely used herbicides in the world."[1] There are over 600 2,4-D products currently on the market.[3]

Figure 1
2,4-D



2,4-dichlorophenoxyacetic acid

Scope of this Article

This article focuses on research published since 2000 that identifies hazards of 2,4-D use.

Use

2,4-D is used to kill broadleaf weeds.[2] Typically, grasses and their relatives are not killed by 2,4-D.[1]

The U.S. Environmental Protection Agency (EPA) recently estimated 2,4-D use based on data collected during the 1990s. A total of 46 million pounds of 2,4-D are used every year in the U.S.; about two-thirds of this is used in agriculture. Major uses include about 11 million pounds used on range and pasture, about 8 million pounds used by homeowners on their lawns, about 7 million pounds used in wheat production, and about 3 million pounds used by lawn care companies.[2]

How Does 2,4-D Kill Plants?

According to EPA, 2,4-D kills plants by increasing three characteristics of the plant: the plasticity of the cell walls, the amount of proteins being made in the plant, and the amount of ethylene being produced by the plant. The effect of these changes is to cause cells to divide and the plant to grow uncontrollably. The end result is that the tissues of the plant are damaged and death occurs.[2]

Forms of 2,4-D

There are eight salts and esters of 2,4-D, in addition to 2,4-D itself (an acid), that are used as herbicides.[3] Most toxicology tests use the acid form of

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"INERT" HAZARDS IN 2,4-D HERBICIDES

Chemical Name	Health Hazards Identified in Laboratory Tests Compiled by the National Institute for Occupation Safety and Health
Amorphous silica	Diarrhea, tears, and obstruction of lung blood vessels
Aromatic solvent naphtha	Reduced fertility, reduced litter size, and reduced growth of newborns
Attapulgite-type clay	Cancer and tumors
1,2-Benzisothiazolin-3-one	Genetic damage in human cells, skin sensitization
n-Butyl alcohol	Severe eye irritation, genetic damage in hamsters, reduced fertility, developmental abnormalities; depressed activity
Butyl cellosolve	Severe eye irritation, genetic damage in bacterial tests, sperm damage, reduced fertility, and developmental abnormalities
Diesel Fuel No. 2	Tumors
Dimethylpolysiloxane	Diarrhea
Ethylene diaminetetraacetic acid	Genetic damage in laboratory animals, developmental abnormalities, and reduced fertility
Ethylene glycol	Genetic damage in laboratory animals and human cells, developmental abnormalities, reduced litter size, effects on testes, reduced fertility, diarrhea, nausea, headache, reduced liver function, and damage to corneas
Hexylene glycol	Severe eye irritation, reduced kidney function
Hydrogenated aliphatic solvent	Some evidence of cancer in laboratory animals
8-Hydroxyquinoline sulfite	Genetic damage in bacterial tests and human cells
Kerosene	Severe skin irritation, genetic damage in bacterial tests, coughing, nausea, depressed activity, muscle weakness, and anemia
Kerosene/Fuel Oil No. 1	Skin inflammation
Methyl oleate	Leukemia, tumors
Methyl salicilate	Severe skin irritant, reduced newborn survival, reduced fertility, developmental abnormalities,and liver degeneration
Mineral spirits	Kidney damage, skin inflammation, and anemia
Octylphenol polyethoxylate	Genetic damage in human cells and laboratory animals, developmental abnormalities, and skin inflammation
Polyethoxylated isodecyl alcohol	Severe skin and eye irritation
Propylene glycol	Genetic damage in laboratory animals, reduced fertility, high blood sugar levels, anemia, and tumors
Quartz silica	Genetic damage in laboratory animals and human cells, cancer, lung fibrosis, diarrhea, and coughing
Sodium benzoate	Genetic damage in laboratory animals and human cells, developmental abnormalities, and reduced newborn survival
Sodium lignosulfonate	Genetic damage in laboratory animals, and reduced liver function
Titanium dioxide	Genetic damage in laboratory animals, cancer, tumors, and diarrhea

Inert ingredients in 2,4-D products identified by EPA Office of Prevention, Pesticides, and Toxic Substances's Public Information and Records Integrity Branch in response to NCAP's Freedom of Information Act request RIN-1178-99. Response dated January 28, 2004.

Hazards of inert ingredients taken from National Institute for Occupational Safety and Health's Registry of Toxic Effects of Chemical Substances. Accessed through NISC International, Inc's BiblioLine Basic Chemical Information System, www.nisc.com. Query done on November, 2005 by Chemical Abstract Services (CAS) numbers 7631-86-9 , 64742-95-6,12174-11-7, 2634-33-5, 71-36-3, 111-76-2, 68476-34-6, 63148-62-9, 60-00-4, 107-21-1, 107-41-5, 64742-47-8, 134-31-6, 8008-20-6, 64742-81-0, 112-62-9, 119-36-8, 8052-41-3, 9002-93-1, 61827-42-7, 57-55-6, 14808-60-7, 532-32-1, 8061-51-6, and 13463-67-7.

2,4-D. This article will identify other forms when they are used.

Inert Ingredients

Most commercial 2,4-D herbicides contain ingredients other than 2,4-D. According to U.S. pesticide law, many of these ingredients are called "inert."[4] Typically these ingredients are neither identified on pesticide labels nor included in most of the health and safety testing required to register a pesticide.[5,6]

NCAP has identified some of the inert ingredients used in 2,4-D products through the Freedom of Information Act. For hazards of some of these chemicals, see "'Inert' Hazards in 2,4-D Herbicides," left.

Symptoms of 2,4-D Poisoning

A review of chlorophenoxy herbicide incidents reported to poison control centers in the U.S. found that about 2,000 poisoning incidents are reported every year. (2,4-D is the most common herbicide in this family.)[7]

EPA's summary of 2,4-D poisoning incidents describes the most common symptoms as irritation, inflammation, and itching of eyes and skin. Other symptoms include hives, nausea, vomiting, throat irritation, headache, dizziness, coughing, and difficulty breathing. Eye exposures are more problematic than skin exposures.[8]

Mutagenicity (Ability to Cause Genetic Damage)

In its recent review of 2,4-D, EPA concluded that "2,4-dichlorophenoxyacetic acid was not mutagenic."[9] However, other recent evidence points to a different conclusion:

- The National Institute for Occupational Safety and Health labels three forms of 2,4-D (the acid, the sodium salt, and the dimethylamine salt) as mutagens.[10]
- Research from the University of Minnesota found that the frequency of a chromosome rearrangement in pesticide applicators was correlated with the level of 2,4-D in their urine.[11]
- Scientists at the Institute for Medical Research and Occupational Health (Croatia) found that a commercial 2,4-D herbicide caused chromosome

breaks in human blood cells.[12]

- Two studies (from the National Research Centre (Egypt) and the Bulgarian Academy of Sciences showed that 2,4-D caused chromosome breaks in mouse bone marrow.[13, 14]

Effects on Hormones

EPA's discussion of 2,4-D's ability to disrupt the normal functioning of hormones concludes: "Based on currently available toxicity data, which demonstrate effects on the thyroid and gonads [sex organs], there is concern regarding its endocrine disruption potential."[15] This conclusion is based on tests of laboratory animals sponsored by 2,4-D manufacturers showing that 2,4-D decreased levels of thyroid hormones and decreased the size of sex organs.[16]

Other recent research showing that 2,4-D has effects on hormones includes a study of 2,4-D applicators. The study, led by a University of Minnesota researcher, showed that 2,4-D exposure increased levels of a sex hormone in these applicators.[11]

Another University of Minnesota study shows that two commercial 2,4-D herbicides act like estrogens (sex hormones) in breast cancer cells.[17]

In addition, a recent study from the Netherlands shows that 2,4-D has the ability to displace sex hormones from the protein that normally transports these hormones in the blood.[18]

Effects on the Immune System

In EPA's recent review of 2,4-D, the agency asked 2,4-D manufacturers to conduct an additional laboratory test to address concerns about 2,4-D's toxicity to the immune system.[15]

However, research has already demonstrated that 2,4-D has significant effects on the immune system:

- Led by a toxicologist from the University of Saskatchewan, a team of Canadian researchers showed that exposure to "environmentally realistic" amounts of 2,4-D reduced the activity of at least three human genes that produce proteins with important immune system functions.[19]
- Scientists from the National Institute for Occupational Safety and Health and West Virginia University showed that 2,4-D decreased the production of cells that make antibodies in the bone marrow of mice.[20] (See Figure 2.) 2,4-D exposure also decreased the numbers of certain immune system cells made in the thymus.[21]

Carcinogenicity (Ability to Cause Cancer)

Whether exposure to 2,4-D causes cancer has been a controversial question for decades. In 1987, the International Agency for Research on Cancer classified all phenoxy herbicides, including 2,4-D, as "possibly carcinogenic to humans."[22] This classification was based primarily on studies of people who were exposed at work to phenoxy herbicides.[22] EPA evaluated 2,4-D's carcinogenicity in 1997 and concluded that 2,4-D is "not classifiable as to human carcinogenicity."[23]

Meanwhile, research continues to suggest that exposure to 2,4-D poses cancer concerns.

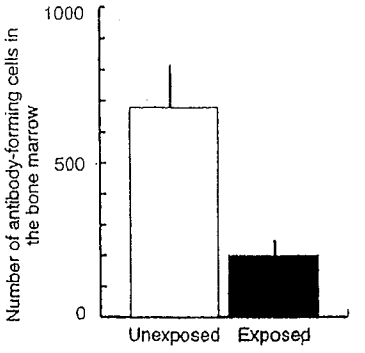
One type of research focuses on people who work with 2,4-D. A study led by a scientist from the University of Saskatchewan found that risk of the cancer non-Hodgkin's lymphoma was increased by exposure to 2,4-D. (See Figure 3.) This study confirms the results of four earlier studies that found a similar link.[24] A second study, conducted by an EPA researcher, found that increased cancer rates were associated with phenoxy herbicide use on farms. (This study used wheat acreage to estimate phenoxy herbicide use because 2,4-D and related herbicides are commonly used on wheat.)[25] A third study found that exposure to 2,4-D was associated with an increased risk of non-Hodgkin's lymphoma in California farmworkers. This study was conducted by scientists at the Cancer Registry of Central California.[25a]

Other recent research has focused on how 2,4-D exposure affects cells in ways that promote cancer. A study led by a researcher at St. Louis University showed that rapid and repeated division of blood cells occurs in pesticide applicators who use 2,4-D.[26] These results were confirmed by laboratory tests in a study led by a researcher at the University of California, Berkeley.[27] A study led by a researcher at the Medical College of Ohio found that 2,4-D increased the activity of a tumor gene in the liver.[28]

Effects on Sperm

Effects of 2,4-D on sperm have been

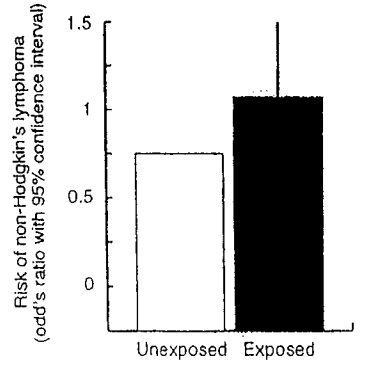
Figure 2
2,4-D and the Immune System



Salazar, K.D. et al. 2005. The polysaccharide antibody response after Streptococcus pneumoniae vaccination is differentially enhanced or suppressed by 3,4-dichloropropionanilide and 2,4-dichlorophenoxyacetic acid. Toxicol. Sci. 87:123-133.

In laboratory tests, 2,4-D reduced the production of antibodies, chemicals used to fight off infection.

Figure 3
2,4-D and Cancer



McDuffie, H.H. et al. 2001. Non-Hodgkin's lymphoma and specific pesticide exposures in men: Cross-Canada study of pesticides and health. Cancer Epidemiol. Biom. Prev. 10:1155-1163.

In a study of Canadian men, exposure to 2,4-D was associated with an increased risk of non-Hodgkin's lymphoma.

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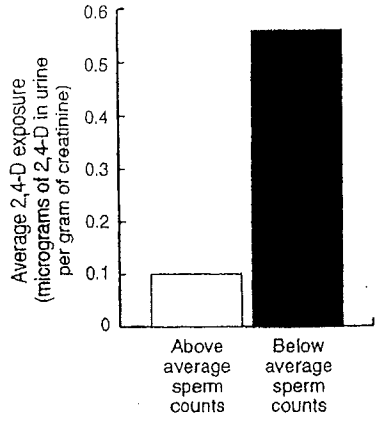
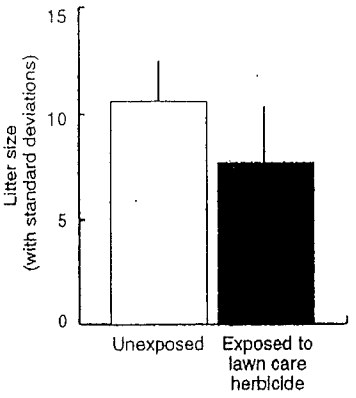
Figure 4
2,4-D and Low Sperm Counts



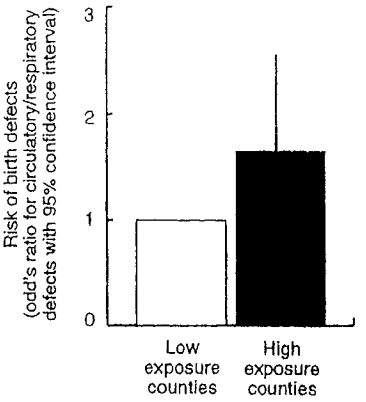
Swan, S.H. 2003. Semen quality in relation to biomarkers of pesticide exposure. Environ. Health Persp. 111:1478-1484.

Figure 5
2,4-D and Pregnancy Success



Cavieres, M.F., J. Jaeger, and W. Porter. 2002. Developmental toxicity of a commercial herbicide mixture in mice: 1. Effects on embryo implantation and litter size. Environ. Health Persp. 110:1081-1085.

Figure 6
2,4-D and Birth Defects



Schreinemachers, D.M. 2003. Birth malformations and other adverse perinatal outcomes in four U.S. wheat-producing states. Environ. Health Persp. 111:1259-1264.

2,4-D exposure has been linked with low sperm counts and birth defects. It also reduces fertility in laboratory tests.

identified in studies of both exposed people and laboratory animals.

A study led by a physician at the University of Missouri compared 2,4-D exposure (as measured by urine 2,4-D levels) with sperm counts. The study found that men with low sperm counts had 2,4-D levels five times as high as those found in men with above average sperm counts.[29] (See Figure 4.)

In addition, EPA lists a variety of effects on male sex organs that were identified in laboratory tests sponsored by 2,4-D manufacturers. These include atrophy of the testes, degeneration of sperm-producing tissues, and decreased numbers of sperm in the testes.[30] 2,4-D also caused an increase in the numbers of abnormal sperm in a study conducted at the National Research Centre (Egypt).[13]

Effects on Children

Some of the most troubling concerns about 2,4-D are its potential to harm children:

- **Pregnancy problems**. EPA's recent review of 2,4-D did not identify significant pregnancy problems caused by 2,4-D exposure except to note that spontaneous abortions increased in rabbits following high-dose exposure.[30]

However, research from the University of Wisconsin-Madison shows that environmentally relevant exposures to a commercial 2,4-D herbicide reduced litter size in laboratory animals. The study used a lawn care product containing three herbicide chemicals (2,4-D, mecoprop, and dicamba) and an unknown number of inert ingredients. When pregnant animals drank water during their pregnancies containing small amounts of this herbicide, their litters were about 20 percent smaller than litters from animals drinking uncontaminated water.[31] (See Figure 5.)

- **Birth defects**: An EPA researcher studying birth defects in rural parts of Minnesota, Montana, North Dakota, and South Dakota showed that defects related to the respiratory and circulatory system were more common in counties with high 2,4-D use than in low-use counties. Wheat acreage was used as an estimate of 2,4-D use.[32] (See Figure 6.)
- **Contaminated breast milk**: Two recent studies (one of rats, done at the University of Rosario [Argentina][33] and the other of goats, sponsored by a 2,4-D manufacturer[34]) show that mothers exposed to 2,4-D produce 2,4-D-contaminated milk. The Argentine study also showed that 2,4-D moved from the milk to the blood and brain of the offspring.[33]
- **Brain development**: EPA states that "there is a concern for developmental neurotoxicity resulting from exposure to 2,4-D."[35] (Developmental neurotoxicity is the ability of chemical exposures in the womb or during childhood to affect the developing brain and nervous system.) EPA's only response to this concern was to require another study from 2,4-D manufacturers.[35]

However, a series of studies by researchers at the University of Rosario have already demonstrated that 2,4-D exposure impacts brain development. Recent studies show that exposure of laboratory animals during pregnancy and nursing affected neurotransmitters[36,37] (the chemicals that allow nerve impulses to move between cells in the brain), reduced brain size,[38] and disrupted developing connections between nerve cells in the brain.[39]

Dioxin Contamination

According to EPA, 2,4-D is contaminated with dioxin (2,3,7,8-TCDD),[40] a stunningly toxic molecule. 2,3,7,8-TCDD, according to the National

Institute of Occupational Safety and Health, is carcinogenic, mutagenic, and causes reproductive problems at minute doses.[41]

EPA's data dates from the 1990s and shows that 2 out of 8 samples of 2,4-D analyzed were contaminated with 2,3,7,8-TCDD. Other related dioxins were also found. 2,4-D is the seventh largest source of dioxin in the U.S.[40]

Dioxins have also been found in a Japanese 2,4-D herbicide.[42]

Contamination of People

According to a survey conducted by the Centers for Disease Control and Prevention, about 25 percent of Americans carry 2,4-D in their bodies. (See Figure 7.) Levels of 2,4-D are higher in children than they are in adults.[43]

Contamination of Rivers, Streams, and Wells

2,4-D is found in rivers and streams in both agricultural and urban areas. The U.S. Geological Survey's (USGS's) national water quality monitoring program found 2,4-D in about 15 percent of the samples the agency collected in agricultural areas. Urban streams were contaminated equally often.[44]

Wells are also contaminated by 2,4-D, according to USGS, but not as often as rivers and streams.[45]

Contamination of Air

USGS compiled air monitoring data from across the country in 1995. The agency found that 2,4-D contamination of air is widespread; almost 60 percent of the samples in the USGS compilation were contaminated with 2,4-D.[46] (See Figure 8.)

Indoor Contamination

Although 2,4-D is used outdoors, it can be tracked inside after lawn care applications and contaminate homes. Researchers from EPA and Battelle Memorial Institute found 2,4-D on dust particles in the air inside homes after lawn treatments, as well as on tables, window sills, and floors.[47]

Drift Problems

Drift of 2,4-D is common. When the Association of American Pesticide Control Officials surveyed state pesticide agencies in 1999, 2,4-D was one of the top five pesticides involved in drift incidents in over 26 states.[48]

Effects on Pets

2,4-D is linked with both cancer and testicular problems in dogs.

Veterinarians from Purdue University studying bladder cancer in Scottish terriers showed that exposure of terriers to lawns treated with phenoxy herbicides is associated with an increased risk of bladder cancer. The risk of this cancer was four times greater in exposed dogs than in unexposed dogs. The results of this study are consistent with an earlier study showing that use of 2,4-D herbicides on lawns was associated with another cancer, lymphoma, in dogs.[49]

According to laboratory studies sponsored by 2,4-D manufacturers, exposure to 2,4-D also decreases the size of testicles in dogs.[50]

Effects on Birds

Although EPA recently concluded that "risks to birds from 2,4-D exposure are not of concern,"[51] 2,4-D impacts birds when its use alters the plant community that provides birds with food and shelter.

For example, reviews done by USGS regarding the sage-grouse state that "spraying of herbicides [often 2,4-D] not only eliminates large blocks of sagebrush, leading to increased habitat fragmentation, but also may poison insects and other invertebrates eaten by sage-grouse."[52]

In another review, USGS noted that 2,4-D spraying caused changes in what Brewer's sparrows eat, reducing their consumption of insects.[53]

Effects on Fish

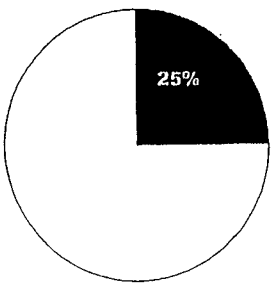
EPA requires 2,4-D products to be labeled with a warning about toxicity to fish.[54] Recent research shows that some of these toxic effects occur at minute concentrations.[55]

Researchers at the University of Maryland looked at an effect called "peroxisomal proliferation" in fish. This term refers to an increase in certain specialized cell structures and has been associated with disruptions of sex hormones and development. In this study effects occurred at a concentration of only 10 parts per billion.[55]

Effects on Frogs

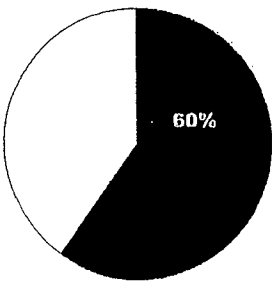
EPA's review of 2,4-D states the 2,4-D is "practically non-toxic"[56] to frogs. However, recent research shows that 2,4-D has troubling effects on frogs.

Figure 7
2,4-D in Our Bodies



Department of Health and Human Services. Centers for Disease Control and Prevention. 2005. Third national report on human exposure to environmental chemicals. http://www.cdc.gov/exposurereport/.

Figure 8
2,4-D in Air



Majewski, M.S. and P.D. Capel. 1995. Pesticides in the atmosphere: Distribution, trends, and governing factors. Chelsea, MI: Ann Arbor Press. Pp. 78-79.

A surprising number of Americans carry 2,4-D in their bodies. It is also frequently found in studies of air contamination.

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Researchers at Willamette University showed that 2,4-D interferes with a sex hormone and stops frog eggs from maturing.[57]

Effects on Plants

As an herbicide, it is not surprising that 2,4-D damages plants. What is surprising is that 2,4-D can cause genetic damage to plants at concentrations "that did not have any visible physiological effects."[58] Biologists at the University of Lethbridge (Canada) showed that 2,4-D caused mutations in a mustard at concentrations as low as 3 parts per billion, below drinking water guidelines in Canada.[58]

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ID 94

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Please consider utilization of small diameter material: markets, processing plants, compensation, haul distances, etc.

ID 95

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Urban forestry needs. Simple yet not so. Almost everyone agrees that trees are good and even necessary. The environmental changes ahead will show that those that took early steps to help offset it were much better off. Trees planted in the urban areas will have a greater positive impact. Now is the time to increase and promote the urban forest.

To do this it takes money.Unfortunately the Federal Government in its infinite wisdom has decided to cut its funds for urban forestry. This leaves the state and local governments a bigger share to pay or cut services. The later choice is not good. Let us not be as short sighted. Cutting funding now would be the worst. In fact funding should be increased in accordance to the importance and impact that urban forests will have in moderating temperature extremes.

Recent studies have shown that governments get a return of $2.70 for every $1 spent. Now if we could just figure a way for the private sector to get a return and we wouldn't have to worry about an urban forestry budget.

Please see to it that the state helps pick up what the federal government has dropped.The small urban forestry staff the state has now does an amazing job. Don't let it get cut, the state cann't afford it. Urban forests are only going to increase in value and importance.

Urban Forests- The Forests Where We Live

ID 96

Board of Forestry - 2007 Issue Scan

Printed Name: Richard Gibson
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Please describe the proposed issue [in 500 words or less]:

Review and update the Protection From Fire Program's administrative rules.

Many of the Protection From Fire Program's administrative rules (OAR 629-041-0005 to 629-047-0290) have not been comprehensively reviewed and updated in many years. As a result, a number of the rules contain requirements which are no longer appropriate, contain technical errors, require grammatical correction, or are in need of general review and validation.

Under this proposal, a small advisory committee of Department representatives, operator representatives and landowner representatives would be established. The committee would review the current rules, make recommendations for any needed amendments, deletions or additions and submit their findings and recommendations to the Board of Forestry.

Dear Sir,

I treasure our vast state forests, and have personal concerns about the use of herbicides in our forest industries. My beloved daughter and grandson live directly downstream and downwind from a unit that was logged last year and will be sprayed this summer. I don't want them to be afflicted with the sort of chemical sensitivities that I, myself suffer from as a result of being downwind of aerially sprayed pesticides. Unfortunately there are growing numbers of us whose bodies are reacting badly to the toxic stew we've created in the world - PCB's and dioxin and mercury and the myriad of other chemicals that our industries use by the ton.

We are seeing an undeniable link between human activities and global climate change, deminishing animal populations, etc. etc. Our state and region are taking these matters seriously and enacting more sustainable practices on all levels, from top to bottom. It is also time for the ODF to reexamine its practices and hopefully make the necessary changes. New voices are emerging from our universities, questioning the status quo in forest management. Some in industry and government would like to silence new and differing perspectives. But who are you going to listen to, lobbyists and talking heads who are beholden to industry profits, or local and regional researchers and citizens who intimately know our forests and our needs? It's time for some serious research concerning herbicide drift and its effects on our water quality, our salmon stocks and our rural communities. It's time to open up policy

discussions to include more environmentalists and public health advocates, and those directly effected by spray issues. It's time give respect to groups such as NCAP (Northwest Coalition Against Pesticides) who've worked on these issues for decades.

I understand that herbicides are used to suppress new understory growth that competes with newly planted Doug Fir seedlings. There is the alternative of manual release — forest crews weeding out plants that overshadow new trees. We could employ lots of young people to do this work. There can be such wonderful comraderie among folks doing this type of bioremidiation together. Decades ago, my ex was a Tree Dad, and I well remember the passion of those treeplanting crews and their drive to rehabilitate our forests. The use of less toxins, along with more manual labor is integral to natural and sustainable lifestyles and businesses of all types. Also, it seems like it would make sense to retain as much of the native understory as possible. Just yesterday, I was walking in a clearcut, and looking at the salal and ferns and such, and thinking how important they are — helping to hold the soil together, now that the forest canopy no longer shelters the slopes. Besides, biodiversity is nature's way to maintain the health of the ecosystem. Monocropping our forests invites pests and other problems.

Times are changing. Solutions that worked in the past may no longer be appropriate.

Thanks for listening. Sincerely, Janine Offutt

Janine Offutt
924 4th St
Oregon City OR 97045
503-722-1627
janineoffutt@hotmail.com

ID 98

Dan Postrel
Oregon Department of Forestry
Agency Affairs
2600 State Street
Building B
Salem, Oregon 97310

Dear Mr. Postrel,

It has come to my attention that the ODF is asking for comments from the public about what issues it should focus its research and budget on for the next few years.

The Oregon Toxics Alliance is promoting a campaign that is attempting to flood your office with letters stating something to the effect that the ODF should give a high priority to monitoring chemical use in the forestry environment. Ironically, in their communication to it's members, the OTA makes the statement that "good policy must be based on good science". They then supply the non-scientists on their mailing list with a plethora of non-scientifically substantiated bullet points upon which to base a letter to ODF. This is nothing but another attempt by the OTA to effectively ban the forestry use of pesticides.

I am a forester and have been a licensed pesticide applicator in the State of Oregon for over 25 years. I have been associated with the aerial application of pesticides every year over this time. Every applicator knows that when pesticides are applied according to the label, they do not pose any danger to the to the non-involved public or wildlife.

Drift is a non-issue. We do not spray in the fog or in high winds. We use nozzles that are designed and oriented not to produce the fine particles that may move off of a target area. When doing herbicide applications, we have the ability to control our application in such a manner that it appears that we have "painted" green stripes parallel to protected streams.

In over a quarter century of experience applying pesticides, I have never experienced any type of pesticide related injury or illness, nor have I ever known any other applicator who has had any type of injury or illness or has known anyone who has. On the other hand, when manual release methods have been used to control brush in the forest, chainsaw injuries are commonplace and sometimes permanently disabling.

The OTA notes that the ODF lags behind California and Washington in forest chemical impact awareness and in developing precautionary models. I say Oregon is far ahead of California and Washington in recognizing that there is no "problem" that needs to be addressed. Oregon understands that pesticides are a necessary tool in the effort to insure that Oregon's forest resource remains renewable.

Sincerely,

Marc V. Halley
Oregon Private Applicator #11249
P.O. Box 670
Brookings, Oregon 97415

ID 99

Board of Forestry – 2007 Issue Scan

Printed Name: Gary Groth
Affiliation: Director, Douglas County Land Department
Mailing Address: Douglas County Land Department
Courthouse Room 320
Roseburg, OR 97470

I have three issues that I would like to submit to the Board of Forestry issue scan.

ID 99

1. The most important issue in my view is the restoration of active management and timber harvest on public forest lands. The reduction of harvest on federal and state lands has had numerous direct and indirect negative effects on Oregonians. In recent years federal and state forests have been managed with preservation in mind rather than multiple use and this has resulted in both economic and environmental disasters. Large, long term investments in forest research and technology have shown we can harvest timber as well as enhance other values at the same time.

ID 100

2. Another topic important to many Oregonians is the intergenerational transfer of family owned forests. We should work towards making the federal inheritance tax reductions permanent as well as ensuring that the Oregon legislature does not pass new laws that discourage families from investing in their forests. In the past too many forest management decisions have been based on IRS (and Department of Revenue) tax bills.

ID 101

3. A third topic is funding for the Oregon Department of Forestry's Private Forest Program. This program has struggled for adequate funding in recent years. More regulations to enforce, with fewer field staff, make implementation of the Forest Practices Act difficult. I believe the Forest Practices Act needs to be adequately enforced in order for the Oregon Department of Forestry to maintain the public's confidence.

Thank you for the opportunity to participate in this process.

Associated Oregon Loggers, Inc.

P.O. Box 12339 • Salem, Oregon 97309-0339 • (503) 364-1330 • Fax: (503) 364-0836

July 27, 2007

Oregon Dept. of Forestry
Attn: Dan Postrel
2600 State St., Bldg. B
Salem, OR 97310

Subject: Board of Forestry -- 2007 Issue Scan

Dear Dan,

I am writing on behalf of Associated Oregon Loggers, Inc. (AOL), to express our interest in Board of Forestry activities and to comment on the Board's 2007 Issue Scan process, which addresses the Board's agenda for 2008 & 2009.

AOL represents more than 1,050 member logging and allied forest management businesses working statewide—Oregon's forest professionals. These forest professionals employ approximately 10,000 workers in the continuous improvement of operation technology for the sound management of the state's abundant & renewable forest resources. AOL member companies are stakeholders in actively conducting forest management of a majority of Oregon's public & private forests.

EXISTING ISSUES

AOL has remained engaged with the Board and Department of Forestry (ODF) during the previous issue scan and work planning. We believe that the Board has identified through their work plans a full plate of issues to address. AOL has in the past, and will continue to make suggestions and voice concerns on the Board's plans and agenda matters.

Additionally, the Forest Vitality Work Plan is a work in progress that promises to redress Board participation in shaping future policies surrounding federal forest land management, biomass utilization, and forest sector economies.

NEW ISSUES

FD 102 **Proposed issue #1: Aggressive Forest Management to Reduce Wildfire & Unhealthy Hazards:**
There is a clear and present danger in Oregon forests on a landscape scale, posed by unprecedented wildfire, insect, disease and storm damage hazards—primarily located on federal forest and rangelands. The vast scope of this problem has been recognized since the early 1990's by forest managers and scientists alike (refer to attached report summary from National Commission on Wildfire Disasters, 1994).

Active forest management is urgently needed to more aggressively reduce these hazards in a meaningful way. Federal efforts over the past 15 years have failed to significantly deal with the escalating dilemma. This should be among the highest priorities for both public and private lands in

Oregon. These problems transcend property boundaries, so landscape-level strategies and actions are required to be effective.

The Board and ODF should exert the bold leadership needed to create new policies—and if necessary encourage statutory changes—to promote extensive and timely actions that will effectively address these unhealthy forest problems on federal, state, and private forest lands. The future sustainability of Oregon forests at-large hinge upon curbing the ongoing devastating forest resource losses incurred over the past two decades to pests, wildfires and storms that have ravaged increasingly vulnerable, unhealthy forests. The on-going losses are wasteful, and such losses in timber and resource production are today dramatically reducing the future yields from Oregon forests.

The current effort of the *Federal Forestlands Advisory Committee* (FFAC) is one element of identifying needed remedies. However, the February 2009 completion date of a FFAC report , which re-states the obvious problems, is not anticipated to be the only constructive solution. More needs to happen to shape Oregon's broken federal forest health policy statewide. In the two years necessary for the FFAC to re-state the federal forest problem, another half million acres of Oregon forest & rangeland will likely be consumed by wildfire, pests, disease and storms. At best, only 2-5% of the killed timber will be salvaged for timber sale due to broken federal forest management. Ironically, in the same two years, a significant segment of the economic infrastructure critical to treating unhealthy forests will be eliminated due in-part to a lack of a cost-effective timber supply. As the Board pondered whether the federal forest debacle was a problem, Oregon continued to lose forest product mills and forestry infrastructure—another half dozen mills were shuttered in 2006 & early '07.

EO 103 **Proposed issue #2: Aggressive Forest Management to Salvage and Reforest Damaged Forests:** The chronic catastrophic forest losses due to wildfire (&other) hazards described in Issue #1 continue to decimate future Oregon forest sustainability, because vast acreages of federal forestlands remain deforested—and poorly stocked with trees. Hundreds on thousands of acres of Oregon federal forests, ravaged by wildfire, pests, disease and storms over the past twenty years, today largely remain grossly underproductive, because salvage site preparation and reforestation was not successfully completed by federal forest agencies. Since 1988, legal gridlock and broken federal policies have prevented meaningful dead timber salvage or reforestation on approximately 1 million acres of Oregon federal forests (an estimate, because not assessed or reported by agencies). And many of the federal attempts to reforest have concluded in poorly-stocked young forests, due to insurmountable policy hurdles and harmful prescriptive policies that hobble the very treatments necessary to accomplish reforestation success.

The vast scope of this problem has been recognized since the early 1990's by forest managers and scientists alike; but, this reforestation backlog has largely not been assessed or reported. Active forest management is urgently needed to more aggressively salvage dead trees, reduce fuel hazard of dead trees, and reforest killed federal forests. Federal efforts over the past 20 years have failed to

significantly deal with the escalating dilemma. This should be among the highest priorities for both public and private lands in Oregon.

The Board and ODF should exert the bold leadership needed to create new policies—and if necessary encourage statutory changes—to promote extensive and timely actions that will effectively address these reforestation problems on federal, state, and private forest lands. The future sustainability of Oregon forests at-large hinge upon the prompt reforestation and growth of forest resources after devastating losses to pests, disease, wildfires and storms that have ravaged increasingly vulnerable, unhealthy forests. The on-going failure to reforest is an irresponsible & tragic sacrifice of future forest resource growth; and such losses in timber and resource production are today dramatically reducing the future yields from Oregon forests.

The current effort of the *Federal Forestlands Advisory Committee* (FFAC) is one element of identifying needed remedies. However, the February 2009 completion date of a FFAC report , which re-states the obvious problems, is not anticipated to be the only constructive solution. More needs to happen soon to shape Oregon's federal forest reforestation policy statewide.

IO 104 **Proposed issue #3: Working Forest Policy to be Strengthened:**
The Board and ODF should do more to promote policies that encourage working forests on both private and public ownerships in Oregon. Working forests provide important economic benefits that help maintain private forest ownerships against competing non-forest land uses. In recent years, conversion of forestlands to other land uses has increased, in-part because forest regulations incrementally have depreciated the value of growing and harvesting trees as a land use. Maintaining working forests over competing land uses also helps sustain a multitude of ecological benefits. Management to promote growing and utilizing renewable forest & energy products is important to fulfill increasing human consumption needs.

IO 105 **Proposed issue #4: Increase Board Accomplishments:**
The current work plan is overly- ambitious. Although ODF provides substantial support, the Board needs to better recognize its limitations and more carefully prioritize its efforts. Similarly, Board discussion and decision-making protocols should be streamlined to make timely progress on priority issues. Lacking this focus and streamlining, Board function at times has slowed to a pace that is no longer responsive to many of the issues at hand.

IO 106 **Proposed issue #5: Forestry Professionals are Un-utilized as Stakeholders:**
The Board typically fails to seek input and advice from forestry professionals, who could provide their professional perspective *independent of their employer or client interests*. The consequence is missed opportunities to tap highly relevant and diverse expertise and experience about , the very policies being considered. The Society of American Foresters is willing & capable to provide stakeholder advice to the Board.

ID107 **Proposed issue #6: Lead and Codify Definition of Biomass:**
The Board should take the initiative to be a strong leader in the emerging issue of forest biomass utilization. Recent Oregon Legislatures tinkered with policymaking surrounding biomass—passing two bills that offered mixed value to biomass forestry—yet, there has been lacking coordination or leadership on this matter. The '07 Legislature defined "biomass" by statute in a very loose, haphazard manner, which needs further clarification. The Board and ODF should become the state's leader by codifying the state's definition of forest biomass, as well as developing—and implementing—proactive forest biomass policy. The Board needs to create effective forest biomass policy and funding mechanisms in future legislative and public venues.

ID 108 **Proposed issue #7: Private Forest Program Mandates Unfunded:**
The Board should establish a strategy to achieve sufficient promote funding for the multitude of regulatory and non-regulatory mandates of the Private Forest Program. Each session of the Oregon Legislature culminates in further erosion of funding necessary to accomplish statutory and administrative programs. At the same time the legislature is *reducing* the general fund contribution to the Private Forest Program, the actual administrative cost of delivering only the non-discretionary responsibilities has *risen* (to consume nearly all the available funding). Key areas are currently under-funded in a serious manner: a) field forester operations in forest practices; b) forest practices monitoring; c) FP education; d) FP reforestation compliance; e) interface forestry stewardship; and f) federal forestry grants/cooperation. The Board needs to advocate for no additional unfunded rules or laws under the Private Forest Program, until full funding of mandated policies occurs.

ID 109 **Proposed issue #8: Public Not Paying Their Share of Forest Protection**
The Board should establish an initiative or strategy to promote fair and equitable forest protection funding at both the statutory and administrative levels. Each session of the Oregon Legislature involves attacks on the balance of fire protection share-costing between forest landowners and the public general fund. As the legislature attempts to *reduce* the general fund contribution, the actual forest firefighting costs attributable to public ignition sources are *increasing*. The Board needs to defend the fairness of effective forest protection funding mechanisms in legislative and public venues.

ID 110 **Proposed issue #9: Curb the Loss of Family Forestlands**
The Board could do better at developing policies that would further encourage the viability of keeping non-industrial family forestland ownerships in Oregon. In recent years, conversion of family forestlands to other land uses and industrial ownership has increased, in-part because forest regulations incrementally have depreciated the value of growing and harvesting trees as a land use for non-industrial owners. The Board should act to address the key solutions recommended by the 2007 Family Forestland Symposium. Family forests provide important economic benefits that help maintain a mosaic of forest ecosystem, economic and social values—which keep Oregon forests sustainable. Promoting family forests is important to keeping Oregon's forests sustainable.

ID 111

Proposed issue #10: Federal Boundaries De-Value Neighboring Property

The danger in Oregon's federal forests posed by unprecedented wildfire, insect, disease and storm damage hazards—increasingly threatens non-federal neighboring properties with these costly unwanted problems. Along thousands of miles of federal forest property boundaries lie neighboring forest and rangelands that must bear the devastation consequences of unhealthy and dangerous federal lands. These federal threats should be directly addressed by the Board, as their impacts on their neighbors are escalating in the following areas: a) fire protection cost increase; b) wildfire hazard; c) rights-of-way uncooperative & untimely; d) spread of pest-disease; and e) spread of invasives.

ID 112

Proposed issue #11: Improved Industrial Fire Data Collection, for Forest Operations

AOL also proposes that the issue of data collection for industrial fires is included in the Wildfire Risk Management Work Plan. An improved record of statewide industrial fire data is needed to assure that forest operator and landowner fire prevention resources are effectively allocated to those specific machinery or methods having the highest probability of fire ignition and escape. At this time, we believe that there is insufficient history of reliable, credible fire source records, which clearly demonstrate which specific industrial practices result in fires [ignition, escape, suppression cost, resource damage cost]. Without such data, it is impossible to conduct a benefit/cost analysis to determine if precaution measures are effective and efficient.

Thank you for the opportunity to comment. If there is a need to further explain these suggestions, please don't hesitate to contact me.

Sincerely,

Rex Storm, CF
Forest Policy Manager
Associated Oregon Loggers, Inc.

Enclosures: 2-pages

SPOTLIGHTING IMPORTANT FOREST TRENDS AND EVENTS

REPORT OF THE NATIONAL COMMISSION ON WILDFIRE DISASTERS

Presented recently to Secretaries Babbitt and Espy, its message is clear: Without aggressive, proactive wildland management programs begun now, the worst is yet to come.

NATIONAL COMMISSION ON WILDFIRE DISASTERS
P.O. BOX 2000 WASHINGTON, DC 20013 • TEL: (202) 667-3300 FAX: (202) 667-7751

The Honorable Mike Espy
Secretary of Agriculture
The Honorable Bruce Babbitt
Secretary of the Interior

Gentlemen:

Here is the report we, the National Commission on Wildfire Disasters, have developed on an issue we believe is of extreme importance to our Nation's interest, both economically and environmentally. You will not find its contents comforting. Your responsibilities include management of a federal estate that is, in many places, an explosive time bomb waiting to ignite. What we have done is look to the future. The situation as we describe it exists. The facts are clear. The question is, What must be done now? We have tried to point the way to some of the answers.

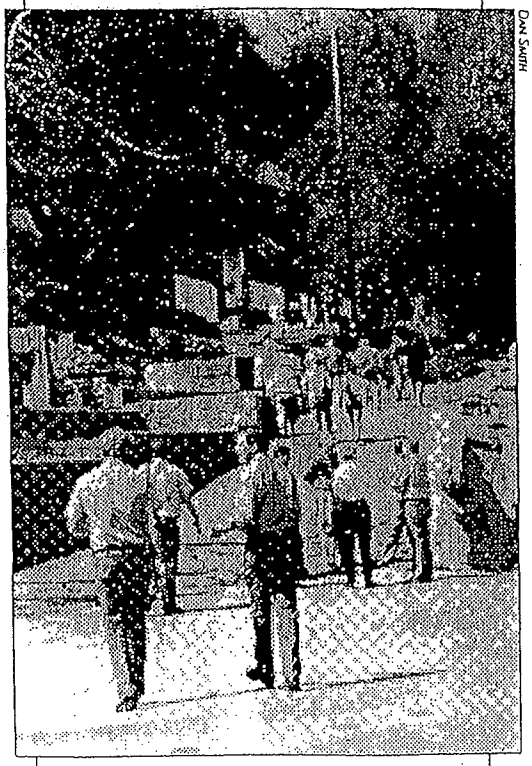
What emerges from this report is the recommendation that disastrous wildfires are a clear and present danger to major regions of the nation, and further, that every year's success in avoiding them—while also delaying the management work needed to prevent them in the future—builds fuels to make the inevitable wildfire disasters more costly and destructive.

In other words, the federal government must continue to improve suppression capability, because the threat grows more serious as each year passes. At the same time, it must begin to reduce that threat by improved, proactive wildland management, or in the end, no amount of suppression investment will be sufficient.

We are convinced that the costs of inaction are significantly greater than the costs of action, and we urge you to take these recommendations seriously. This is the opinion of a group of informed citizens, backed by the science developed within the agencies of your Departments.

R. Neil Sampson
Chairman

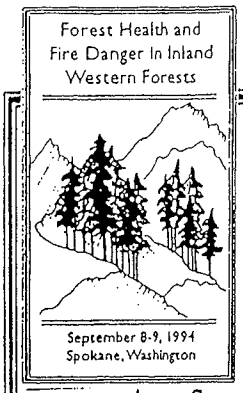
APPOINTED BY THE SECRETARIES OF AGRICULTURE AND THE INTERIOR TO STUDY AND REPORT UPON NATIONAL POLICY CHANGES NEEDED TO MEET THE FIRE MANAGEMENT CHALLENGE IN THE 21ST CENTURY.



DAN SMITH

Wildfire Commission members view the aftermath of California's fall 1993 firestorms—this site is in Pasadena Glen.

Full report is available for $5. Also available is a new book, *Assessing Forest Ecosystem Health in the Inland West* ($49.95, plus $4.20 shipping). Order from AMERICAN FORESTS, P.O. Box 2000, Washington, DC 20013, or call 800/368-5748.



Introduction

Last September, as wildfires raged in the Inland West during one of the worst fire seasons in memory, over 400 natural resource managers, forestry professors, elected officials, forest products industry representatives and members of the general public gathered in Spokane, Washington to attend a conference on forest health. During a two day period, they heard presentations from a variety of perspectives on the current state of forest health in the Inland West, on the implications of forest health conditions for our natural resource base and our communities, and on solutions to forest health problems.

This book collects the presentations made at the conference in order to make the information shared there available to a wider audience.

While many perspectives were brought to bear on the forest health issue at the conference, a number of areas of widespread agreement emerged. There was consensus that many forest ecosystems in the Inland West are in an unhealthy condition; that unhealthy forests pose risks to wildlife, habitat, human communities and industry; that if the conditions are not treated, they will worsen; that we have the science to treat unhealthy forests and restore them to more desired conditions; that gridlock over forest policy is an obstacle to restoring forest health; that an informed and educated public is the key to ending the policy gridlock and to allowing natural resource managers to use the tools at their disposal to treat unhealthy forest conditions.

It is with the hope that the information pooled at this conference can, if shared, help the public understand both the magnitude and the urgency of the current forest health problem in the Inland West that we offer this publication.

Judi Danielson
Conference Co-Chair

Neil Sampson
Conference Co-Chair

ID 115 –
ID 121

LONE ROCK TIMBER MANAGEMENT CO.
LONE ROCK LOGGING CO.

P.O. BOX 1127 * ROSEBURG, OR 97470
TELEPHONE: 541-673-0141
FAX: 541-440-2516 or 541-440-1573

July 26, 2007

Stephen D. Hobbs, Chair
Oregon Board of Forestry
State Forester's Office
2600 State Street
Salem, OR 97310

Chair Hobbs:

Thank you for the opportunity to participate in the Board of Forestry's 2007 Issue Scan. The work of the Board is vitally important to Oregonians and to be granted the opportunity to share items of interest with your Board is valuable.

After reviewing the Board's existing Work Plans, I find it difficult to add new items for consideration. However, I do have points to emphasize under several of the eight existing work plans.

ID 113 I notice that the existing work plans do not include a prioritization system, timeline, or measure of resources needed to accomplish the Plan. Having watched the Board work over the last several years struggling to come to resolution on many difficult issues, I would encourage prioritizing the existing work plans, establish a timeline for tasks to be completed and decisions made and most importantly an evaluation of the resources required to meet the goals of each work plan. I do not have a sure sense of how the Board plans on accomplishing existing plans and I am concerned over the ability of the Board and the Department to achieve success with the limited resources available.

ID 114 <u>State Forests Management</u>
I have reservations about the value of a Habitat Conservation Plan on state forests in Western Oregon. I would encourage the Board to investigate other methods for achieving sustainable forest management on state lands.

ID 115 <u>Forest Vitality</u>
The Governor's recently adopted "25% by 2025" renewable energy goal will require innovation and investment from all sectors. Forest vitality objectives can be coupled with innovation in new energy sources. The Board should be looking for ways to assist in the development of new energy technologies utilizing forest materials. Operations involving biomass are underway and support from the Board and the Department will encourage additional activities.

ID 121 The growing pressure to convert productive forestlands to other uses will continue into the future. It appears the 2007 Farm Bill allocates additional funds for existing conservation and technical assistance programs in forestry. Traditionally, Oregon struggles to efficiently and effectively administer these federal program dollars. The US Forest Service is charged with implementing these programs. As Forest Service budgets continue to struggle and staff reductions continue, it is apparent it is not the appropriate agency to successfully implement these programs. The culture and geographic isolation of Forest Service staff limits the ability to provide technical assistance to private landowners. Recently, the Natural Resources Conservation Service has succeeded in implementing some of these federal programs primarily on agriculture lands. While Forestry Programs are expanding under the Farm Bill, the NRCS lacks the technical expertise to capitalize on the opportunities provided. The Department of Forestry is poised to assist in implementing these programs and should be partnering with the Natural Resources Conservation Services and other federal agencies to provide the technical expertise needed by these programs.

ID 116 Outreach to Urban Populations

The increased attention on ecosystem services provided by forests allows for meeting this work plan objective. The Board and the Department should evaluate opportunities to link these items of interest together.

ID 117 Dynamic Ecosystems

The Board should continue supporting Department efforts to develop this concept by working with other agencies and interested parties.

ID 118 Forest Regulation

There has been much talk and discussion about increasing the use of incentives and other non-regulatory means to achieve desired outcomes. While the dialogue on this topic is always good, few results appear. Rather, rule concept 3 and 4 are approved. If the Board really believes that incentives can provide the means to achieving desired outcomes, then incentives should be available and the priority mechanism for achieving desired results on the ground.

ID 119 Wildfire Risk Management

The 2007 Legislature attempted to shift costs in the Department's Fire budget from the general fund to landowners to pay for fire program administration. While not successful in this legislative cycle, the Department's fire budget will likely be challenged again in the future. Efforts by the Board and the Department are necessary to quantify the contributions of private landowners to firefighting across the state.

ID 120 The Board should continue to support efforts to improve the ability of Smoke Management to accomplish its mission. The growing conflict between urban (and even not-so-urban) residents over smoke from burning will continue to escalate. Efforts need to be made to ensure that this tool is not removed from forest management.

I realize the Board has identified a tremendous amount of work for the near future. I hope these suggestions are helpful in demonstrating the issues important to Lone Rock Timber Management Company. If I can be of any further assistance, feel free to contact me.

Sincerely,

/s/ Jake Gibbs

Jake Gibbs

ED 122

First Name: Robin
Last Name: Winfree
Email: masstudio@earthlink.net
Address1: 29775 Fox Hollow Rd.
City: Eugene
State: OR
Zip: 97405
Company:
Title:
Phone: 541-343-1557
Contact by: E-Mail
Issue Description: I am very concerned about the use of chemical pesticides in our public forests. As far as I know, ODF has done no independent research on the substances that are used - industry research has been enough to allow countless applications of pesticide poisons. Current independent and medical research shows alarming statistics of the effects of these poisons on both human and animal populations.

ODF needs to know if current policy is keeping significant amounts of chemicals out of streams. IT IS IMPERATIVE THAT ODF MONITOR these applications to determine exactly what and how much is entering our watersheds!!

Also, a law suit against the EPA in 2003 required 60' buffers for ground applications and 300' for aerial applications of specific pesticides in certain stream systems with threatened runs of salmon and steelhead. ODF should justify the current buffer zones using the best available science.

I beg you to please amend ODF policies to prevent these chemicals from poisoning our environment.

FO 123

First Name: Mr. Pat
Last Name: Russell
Email: flanagan112@hotmail.com
Address1: 16358 SE Hearthwood Drive
City: Clackamas
State: OR
Zip: 97015
Company:
Title:
Phone: (503) 656-9681 lv mess
Contact by: E-Mail
Issue Description: Comment #2

The litmus test of whether forestry management in the Portland Metro region (within the UBG) is successful with regard to 4[d]-listed (ESA) salmon recovery riparian corridors, and especially in my watershed--the Kellogg-Mt. Scott Creek watershed (from Milwaukie to Happy Valley and Johnson City) is whether there are fish in the creek and spawning.

I can tell you that today there are none to be seen. I have been told by "old timers" that there use to be a lot of salmon in the stream.

This Kellogg Watershed has been ignored for years and it shows. However, surprisingly there are a signficant amount of mature Douglas Fir, Cedar, Oak, Big Leaf Maple, etc. our our watershed (in back yards and front yards). However, they are unprotected and ODF policies provide little or no protection.

So the true measure of ODF success in our urban areas is based upon whats in the stream.

ID 124

First Name: Dianne
Last Name: Ensign
Email: ensign@lclark.edu
Address1: 11010 SW Boones Ferry Rd
City: Portland
State: OR
Zip: 97219
Company:
Title:
Phone: 503-768-6692
Contact by: E-Mail
Issue Description: Dear Madam or Sir,

I am writing to try to convince you that ODF should give high priority to chemical monitoring. I am concerned about the health effects of pesticides both because I personally suffer from chemical sensitivities; and because I care about the effect of forestry herbicides on fish and wildlife. The vast number of pesticide "hits" that we're subjected to every day and absorb has caught up with me a little sooner than others, but we are all at risk for becoming chemically sensitive.

I would like for ODF to track the scientific research on pesticides, including chemical drift from aerially mobile herbicides. I also think that ODF should increase interest group representation from environmental and public health advocates. I also would like to see 60' buffer zones for ground applications and 300' zones for aerial applications of specific pesticides for people and domesticated animals, as well as threatened runs of salmon and steelhead.

Thank you for considering my comments.

First Name: Mr. Pat
Last Name: Russell
Email: flanagan112@hotmail.com
Address1: 16358 SE Hearthwood Drive
City: Clackamas
State: OR
Zip: 97015
Company:
Title:
Phone: (503) 656-9681 lv mess
Contact by: E-Mail
Issue Description: If the ODF is going to continue to administer "harvest" permits in the Portland region urban area (within the UGB), then the ODF needs to identify how "harvesting" approaches are protecting our 4[d]-listed ESA streams and declining fish runs. For example, here in the Kellogg Creek basin, salmon can't spawn because they can't get up the creek at its mouth (Hwy 99E dam and non-functioning fish ladder). Also the riparian fabric ("ribbons of green") is fragmented. Metro's "Nature in Neighborhood" will not help because too much focus was placed on possibly protecting the most regionally-significant riparian habitats while ignoring the need to repair (CPR) the most damaged habitat and provide a strategy of "sustainability" of our "upland forests", at least in my mind urban areas within 1/4th to 1/2 mile of Kellogg - Mt. Scott Creek. Consequently, ODF permits clearcutting (harvest) of most timber to within 100 to 50 feet of the creek banks, rather than focusing on building up the riparian corridor as part of the salmon recovery needs. ODF exercises no discretionary control over private interests. Local agencies who want a more thorough approach take over jurisdiction of tree cutting. ODF sort of ends up with the "black eye."

ID 126

First Name: Paul
Last Name: Engelmeyer
Email: tmnas@harborside.com
Address1: PO Box 694
City: Yachats
State: OR
Zip: 97498
Company:
Title:
Phone: 541-547-4097
Contact by: E-Mail
Issue Description: To: Oregon Department of Forestry/Issue Scan

I would like to take this opportunity to submit comments to your issue/scan process. I have for over 15 years worked throughout the Northwest forests doing a range of contract work, and for the last 10 years worked with the Midcoast Watersheds Council on restoration activities. So, as a result of my experiences I have grave concern over ODF's existing herbicide management rules and regulations. I urge you to address the following points in your issue scan process.

ODF has not been tracking the scientific research on pesticides. ODF needs to utilize the best available science to guide policies which direct the aerial applications of herbicides

ODF's research on long-term fates of herbicide applications was never completed. ODF needs to know if current policy is keeping significant amounts of chemicals out of streams. In particular, any monitoring effort needs to incorporate 1st and 2nd storm run-off events. Monitoring herbicide applications on dry days is just insufficient and I believe inaccurate.

ODF has never researched chemical drift from aerial applications on forested land to neighboring properties. There are numerous well-documented studies that demonstrate the movement of pesticide vapors over long distances and there is also reliable medical research on the impacts to the public. ODF needs to refer to existing research or conduct research on the fate of aerially mobile herbicides.

ODF involves "interest groups" and "stakeholders" in the review of policies but neighboring landowners and the more distant public in general can be impacted by forestry

chemical applications and they are not adequately represented as an "interest group" or a "stakeholder". These people have very good reason to be a voice in the ODF process.

ODF is behind California and Washington in Forest Chemical impact awareness and in developing precautionary models. ODF should make Chemical Monitoring a high priority.

I would also like to acknowledge that herbicide use on forestry lands in but one assault on terrestrial and aquatic biodiversity. I would urge ODF to actively work toward a joint research, monitoring and adaptive management program that would include all parties that use herbicide and pesticides in our landscape. I urge you to engage the local communities, watershed councils, EPA, ODA, DEQ as well as NOAA as partners in a comprehensive monitoring effort.

I look forward to your response. If there is a need to clarify or discuss the issues above do not hesitate to contact me.

Sincerely,

Paul Engelmeyer

EB 127

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Contact by: E-Mail
Issue Description:

Please take a look at the possibly appalling recombinations of chemicals that is occurring in our chemical society (ref. The Hundred Year Lie by Randall Fitzpatrick. The statistics on anomalies in higher life forms--like, mammals; like, human beings) are beyond alarming.

The amount of pesticides dumped on our forests is contributing to this disaster, and there are sustainable solutions to the problems they allege to solve by poisoning the environment-- consider selective cuts, human labor, etc. We just need to adjust our societal/corporate viewpoints (less greed, less consumption, less focus on profit, more focus on the long term). This can be done, to the better health of all.

ID 128

July 31, 2007

Stephen D. Hobbs, Chair
Oregon Board of Forestry
State Forester's Office
2600 State Street
Salem, Oregon 97310

Dear Chair Hobbs and Members of the Oregon Board of Forestry:

As part of the Board's 2007 Issue Scan, the Forest Resource Trust Advisory Committee is proposing the attached "Ecosystem Services" issue for consideration as a priority topic for future work by the Board. In addition to submitting the attached, this letter provides some background on how the advisory committee became engaged on the topic of ecosystem services.

As you are aware, the Forest Resource Trust Advisory Committee – a standing committee to the Board – reconvened in 2006 to review the Forest Resource Trust's existing Stand Establishment Program; and based on this review, to develop recommendations for improving the efficiency and effectiveness of the program. In addition, the advisory committee took the opportunity to look beyond the Stand Establishment Program to see if there were additional program opportunities that could be developed under the Forest Resource Trust statute. These efforts by the advisory committee led to the Board's adoption of administrative rule changes to the Stand Establishment Program in January, 2007, and passage of House Bill 2293 relating to the Forest Resource Trust by the 2007 Oregon Legislative Assembly. House Bill 2293, signed into the law by the Governor, establishes a cost-share program under the Forest Resource Trust as an alternative mechanism to the Trust's loan program for the delivery of financial assistance to participating forest landowners. House Bill 2293 also gives the Board the flexibility to expand the scope of Forest Resource Trust programs to include voluntary forest practices beyond stand establishment.

Originally, the advisory committee envisioned that the State Legislature would create an environmental (also known as ecosystem) services program that would build upon the Forest Resource Trust's role in securing carbon dioxide emission offset funds as a source of funding for financial assistance to family forest landowners. In developing this legislative vision, the advisory committee saw the development of markets for ecosystem services, and the Forest Resource Trust's participation in them, as a tool that landowners could use to help them meet their objectives. The advisory committee's experience was like turning over a big rock – there was a lot to discover, observe and learn. Developing markets for

ecosystem services is a dynamic and complex enterprise in part because the services themselves are dynamic and complex. The topic proved too big to be addressed by the advisory committee's proposed legislative action. Based on amendments developed by a work group established by the House Agriculture and Natural Resources Committee (Representative Arnie Roblan, Chair), the final bill removed the advisory committee's proposed ecosystem services program structure. Instead, the final bill adopted the provision that the Forest Resource Trust, as implemented by the Oregon Department of Forestry, take on the role of assisting participating landowners in securing payments for ecosystem services consistent with their land management strategies and objectives.

Even though further action on ecosystem services is now beyond the scope of the Forest Resource Trust programs, the advisory committee felt it worthwhile to communicate to the Board what it has learned about this important, emerging issue. At a minimum, further consideration and discussion by the Board will inform the Oregon Department of Forestry's implementation of House Bill 2293's direction that the Forest Resource Trust assist landowners in securing payments for ecosystem services. More broadly though, it is also the advisory committee's desire that the Board review emerging markets for ecosystem services to determine how activities in those markets may contribute to accomplishing the strategies detailed in the 2003 Forestry Program for Oregon.

As it turns out, the Board's 2007 Issue Scan provides a timely and appropriate forum for the advisory committee to pass along this topic. Please contact me at (503) 222-3148; patrik.norris@scotiacapital.com, or Peter Daugherty, Private Forests Program Director, Oregon Department of Forestry at (503) 945-7482; pdaugherty@odf.state.or.us with any questions or comments.

Sincerely,

Patrik Norris, Chair
Forest Resource Trust Advisory Committee

cc: Dan Postrel, Agency Affairs, Oregon Department of Forestry
Forest Resource Trust Advisory Committee
House Bill 2293 Work Group Members
Oregon Department of Forestry Executive Staff

ID 128

Board of Forestry – 2007 Issue Scan

Printed Name: Patrik Norris, Chair
Affiliation: Forest Resource Trust Advisory Committee
Mailing Address: c/o Oregon Department of Forestry
Private Forests Program
2600 State Street
Salem, Oregon 97310

Please describe the proposed issue [in 500 words or less]:

According to recent statute (House Bill 2293) adopted by the Oregon State Legislature:

> **"Ecosystem Services"** means environmental benefits arising from the conservation and management of forestland, including, but not limited to, fish and wildlife habitat, clean water and air, pollination, mitigation of environmental hazards, control of pests and diseases, carbon sequestration, avoidance of carbon dioxide emissions and maintenance of soil productivity.

Markets, or other mechanisms providing payments to private forest landowners for ecosystem services, are emerging – with non-governmental organizations and private businesses being in the lead in many cases. Examples include carbon dioxide emission offset payments, wetland mitigation banks and water quality trading programs.

Categories of sub-issues underlying ecosystem services identified by the advisory committee that need broader consideration by the Board are: 1) additionality/baseline, 2) the role of government in market development and participation, 3) fairness between regulatory triggers for markets and landowner property rights, 4) measurement and accounting, 5) complexity of transactions and other barriers for landowner access and participation, 6) the bundling of ecosystem services, and 7) state and federal agency coordination. The advisory committee found particularly challenging the sub-issue of additionality (i.e., changed behavior or action that might not otherwise occur but for the ecosystem service payment) and baseline (i.e., the threshold or floor that changed behavior or action must exceed to be eligible for an ecosystem service payment).

The advisory committee sees the following benefits arising from the Board's broad consideration of ecosystem services and emerging markets and other payment mechanisms to private forest landowners for them.

- Recognition as to how markets for ecosystem services can be used to accomplish existing strategies and actions in the 2003 Forestry Program for Oregon.
- Communication of the opportunities (and possible pitfalls including the relative costs and benefits) markets for ecosystem services provide for private forest landowners in simple, understandable terms.
- Clear direction by the Board to the Department on what needs to be looked at with respect to ecosystem services in the next (2010) long-term forest assessment.

FD 1729

Board of Forestry - 2007 Issue Scan

Printed Name: Wayne R. Giesy
Affiliation: Family
Mailing Address: P. O. Box 772
Philomath, OR 97370

Please describe the proposed issue [in 500 words or less]:

There is not one adequate excuse for State Forestland not to produce and harvest the maximum of timber on a sustained basis. A great amount of this land was transferred to the State with that understanding. The State of Oregon should lead the way to show the Federal Government how to manage the federal land. The patchwork of clearcuts from the 50's on provided many benefits, Timber for family wage jobs, home grown lumber for our citizens and not the least a variety of the best habitat for wildlife.

We better start now, this year, to manufacture and produce more home grown products or the United States imbalance of payments will destroy our economy.

THINK ABOUT IT.

Wayne R. Giesy

Return to: Oregon Department of Forestry
Attn: Dan Postrel, Agency Affairs
2600 State Street, Building B
Salem, OR 97310

before **July 31, 2007**

- Broader discussion, understanding and resolution of the issues of fairness arising from the early development of ecosystem services markets,
- Removal of barriers inherent in additionality and baseline in recognition that landowners with the historical foresight and objective to develop and maintain ecosystem services should be the first in line for ecosystem services payments, and
- Improved and effective coordination amongst the various agencies and natural resource sectors on resolving identified sub-issues.

In order for the above listed benefits to be realized, the Board of Forestry needs to be actively engaged in the discussion and development of proposed policy resolutions to these sub-issues underlying ecosystem services and payments to landowners for them. The discussion cannot just be within the Oregon Department of Forestry, or within the Board and the forestry community. There is opportunity for the Board to emerge as a leader and catalyst on this topic with other commissions such as the Fish and Wildlife Commission, the Environmental Quality Commission and the Board of Agriculture.

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before **July 31, 2007**