



## MOTHER LODE CHAPTER

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Feb. 15<sup>th</sup>, 2010

I am writing on behalf of the Mother Lode Chapter of the Sierra Club and the Battle Creek Alliance to comment on the "Notice of Intent to Renew Resolution R5-2005-0052, Conditional Waiver of Waste Discharge Requirements for Discharges Related to Timber Harvest Activities (Waiver)". I am writing based on my on-the-ground experience of living near approximately 20,000 acres of completed or proposed clearcuts in the headwaters of the Battle Creek watershed that began in 1998. I have lived in Manton for 21 years and know the land and creeks quite well. As the anti-clearcutting organizer for the Sierra Club I have also done extensive research about forest practices, water, air, soil, chemical usage and wildlife issues.

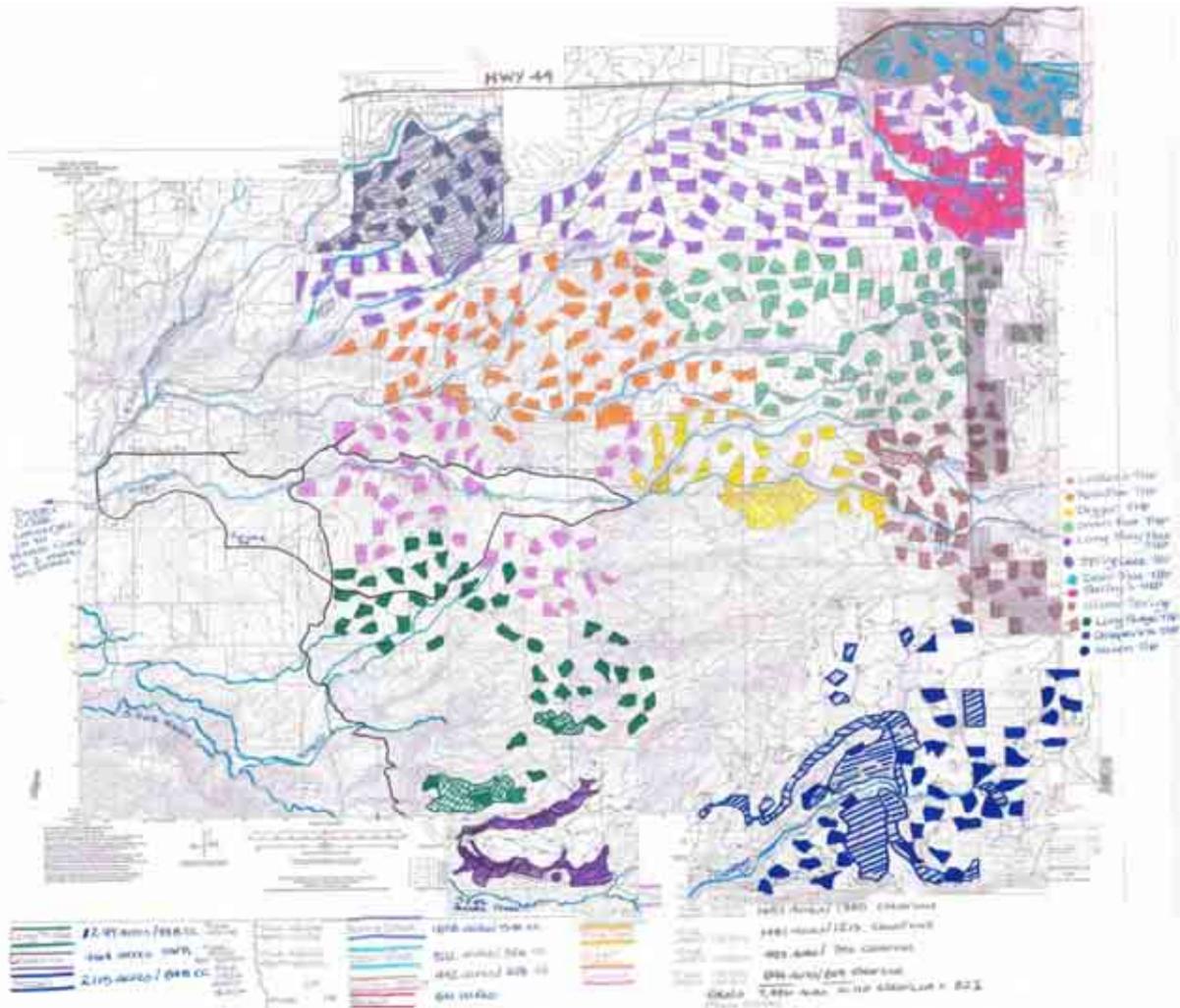
First of all, to understand what our concerns are about water issues and the impacts from timber harvest, here are some visual images of the area:



This photo was taken in Oct. of 2008 and is the most up to date that we have of the area directly west of Mt. Lassen, between Highways 44 and 36 in the Battle Creek watershed, Shasta and Tehama counties. The clearcuts range in size from 11 to 27 acres.

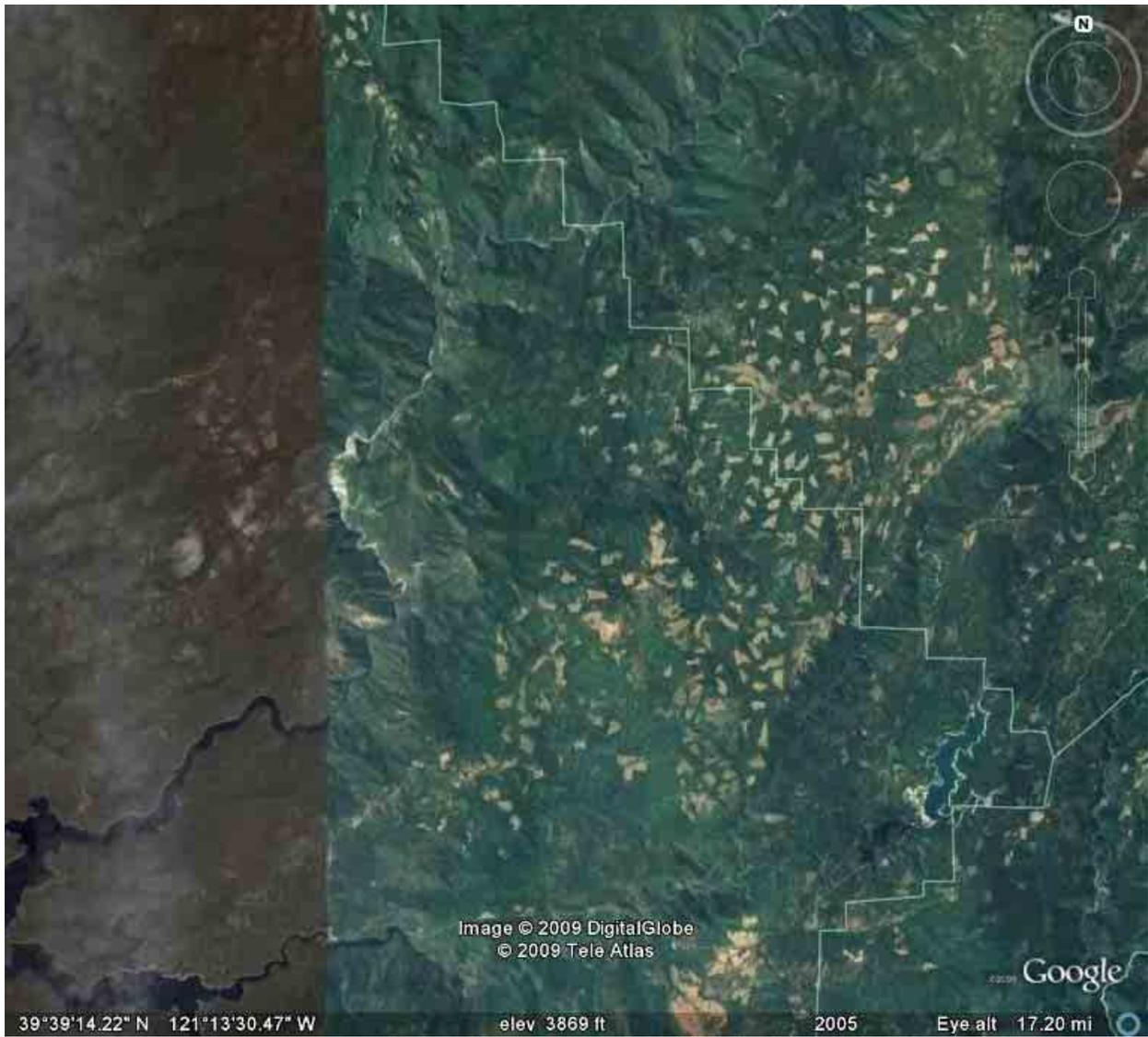


This is the Google Earth image of the same area. The last time we checked, it had not been updated since 2005, so there are many more clearcuts than are shown here. The darker green jagged lines are the canyons that the creeks flow through.



This is a map of the creeks and timber harvest plans in roughly the same area that the Google Earth image shows. The turquoise blue lines are the creeks. (These do not include the unnamed seasonal drainages that water flows through during storms.) The different colors on the map represent the different Timber Harvest Plans filed since 1998. Except for the pink and the dark green areas, these have all been completed. There are 2 new plans that have not yet been added to the map. SPI is the owner of this land, and have themselves expressed their intent to continue to return within 10 years to cut the adjacencies, i.e. the uncut areas between the clearcuts. In fact, one of the new (unmapped) plans is for that purpose, in the dark blue areas just south of Hwy 44 at the top of the map. It has been 6 years since the previous clearcutting was completed.

The previous images are site specific to the Battle Creek watershed, but the same excessive and damaging clearcutting is happening throughout the other watersheds of northern California. Following are Google Earth Images of some of those. (Google has not updated most of these images since 2004 or 2005, so it is safe to assume that there are more clearcuts in these areas by now.)



East of Lake Oroville, Butte and Plumas counties.



Red Bluff to Chico, a continuous trail of clearcuts on the right side of the picture.



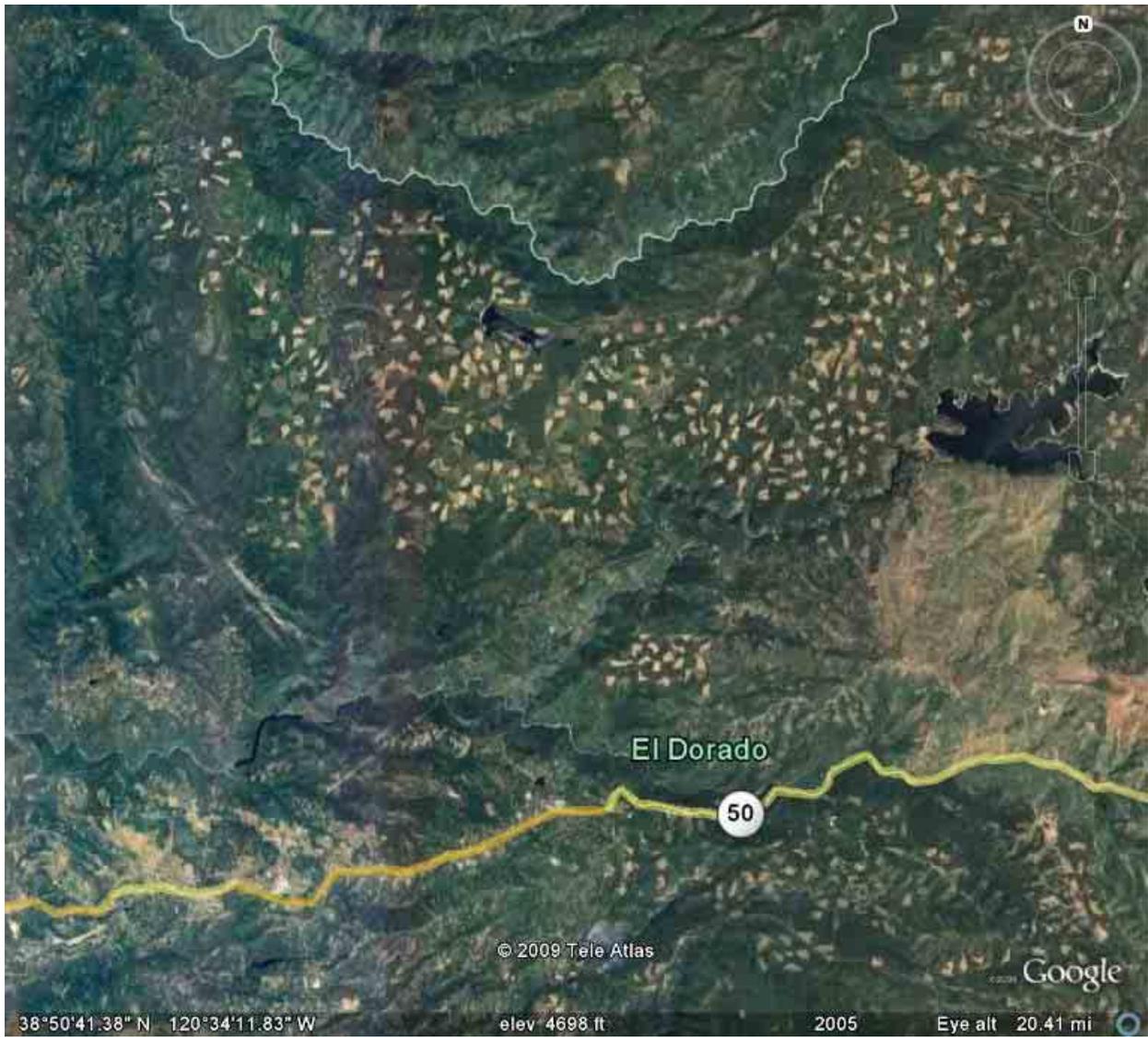
Burney area.



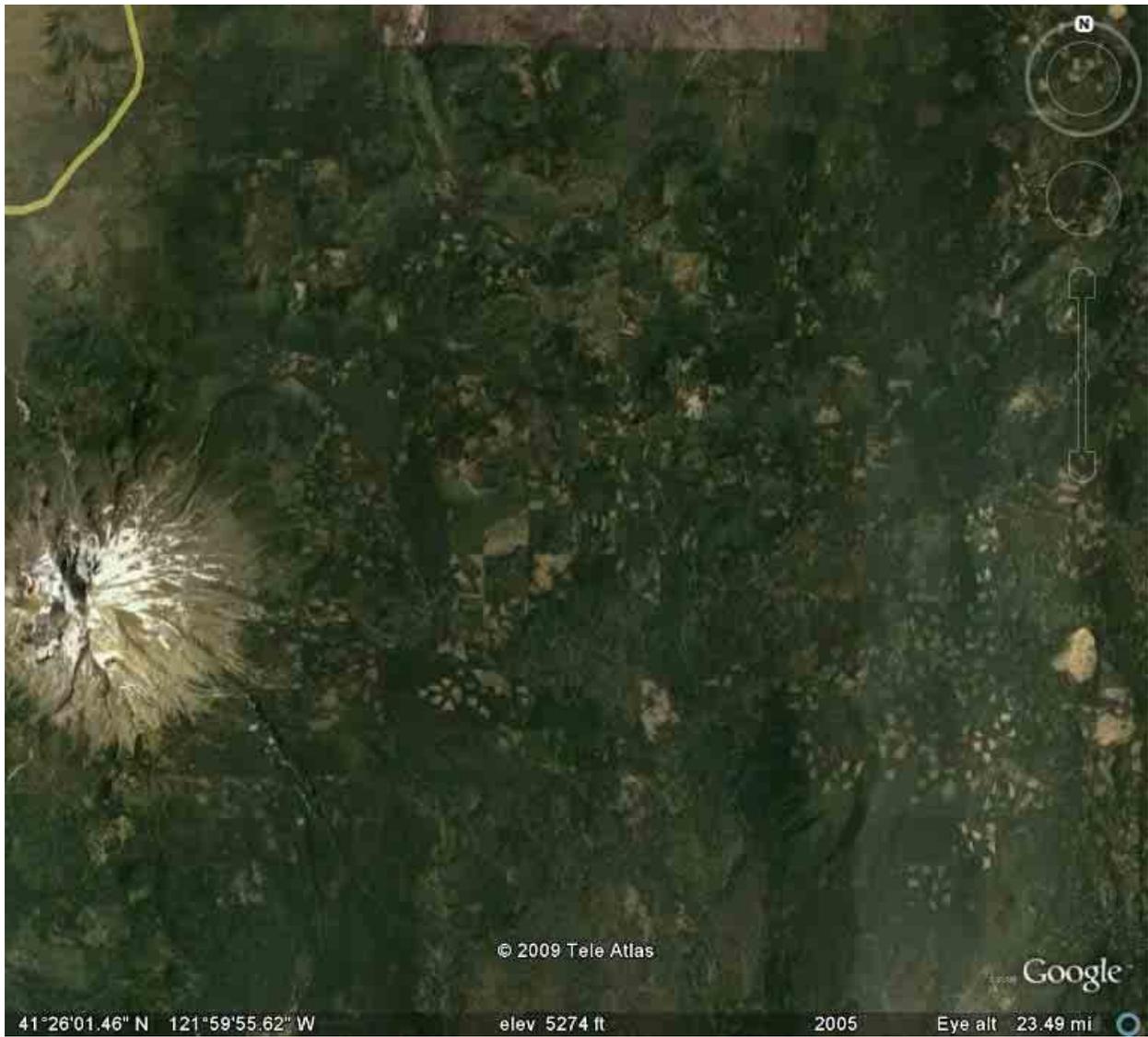
Highway 44 west of Susanville.



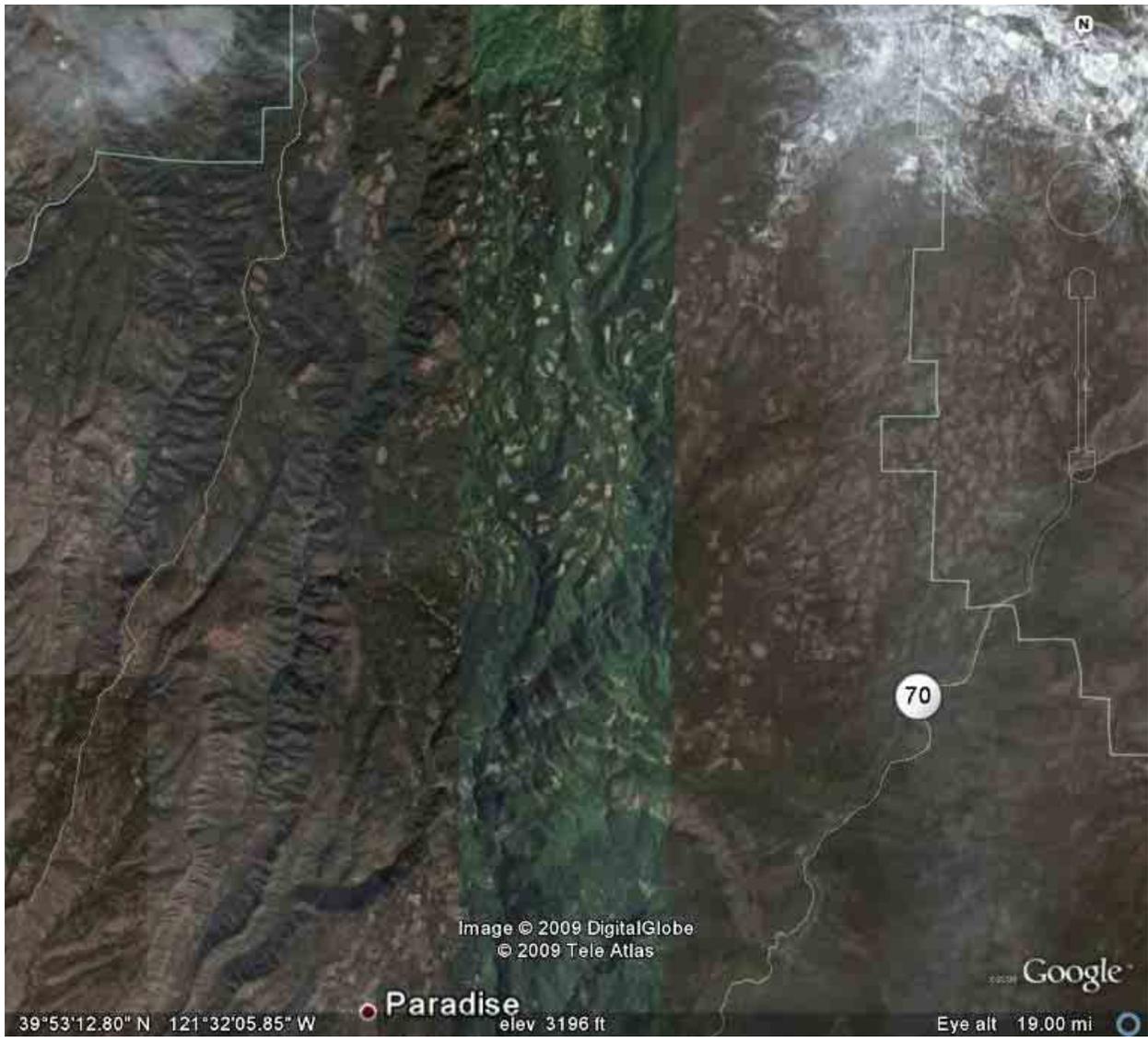
Tehama County, south of Highway 36.



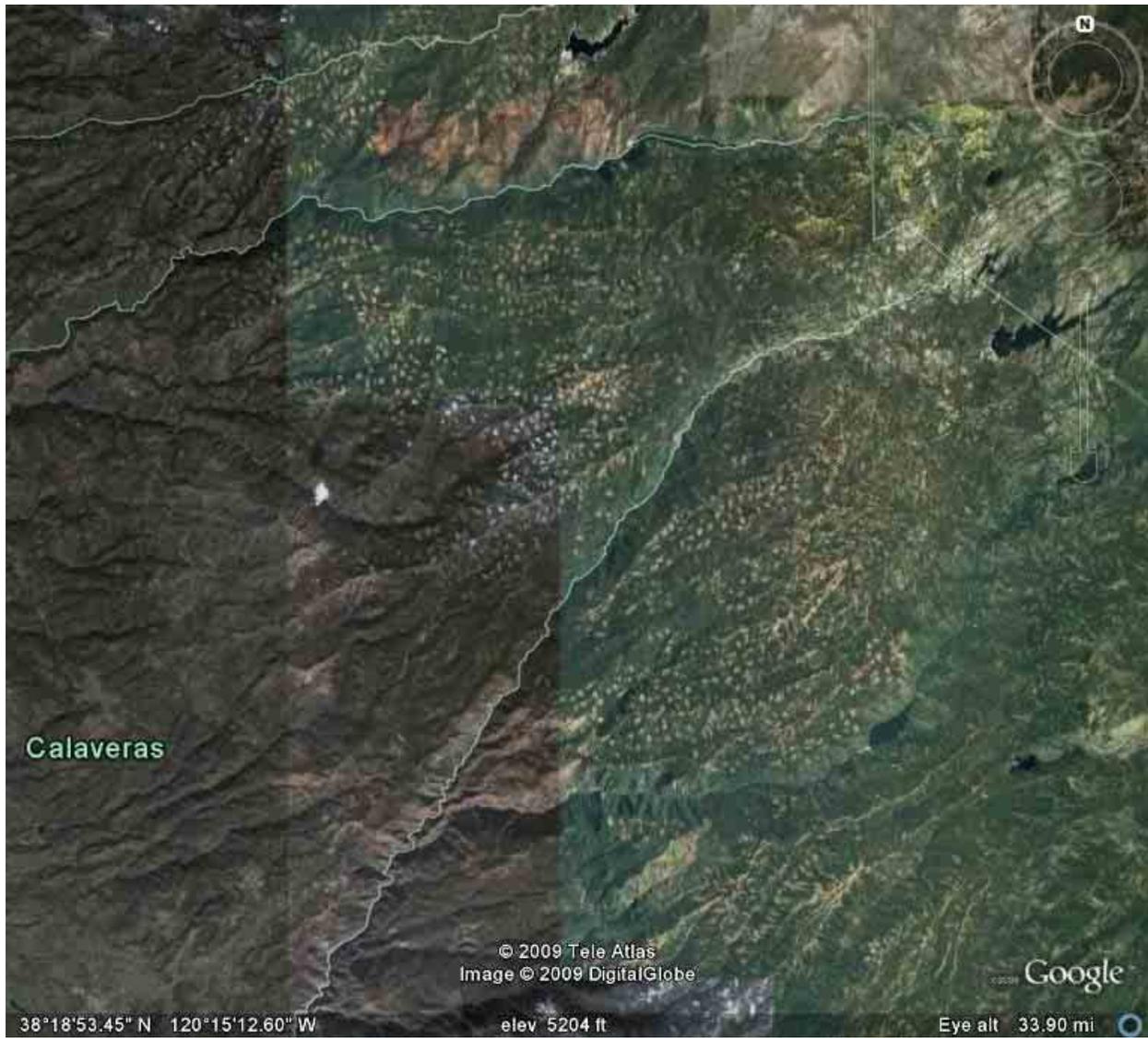
El Dorado county, north of Highway 50.



Mt. Shasta/Siskiyou County



Butte/Plumas County, Highway 70



Calaveras and adjoining counties.

Battle Creek Alliance and Sierra Club are very concerned that the Water Quality Control Board does not know: how extensive the clearcutting is that has taken place; what a short period of time it has occurred in; or the extent of the impacts it is having on water quality and quantity. These are serious issues that must be addressed before renewing, extending or granting waivers to the waste discharge requirements.



2009: A creek approximately 7 miles from the nearest logging that was cut in 2008.



2009: A creek approximately 2 miles from the nearest logging that was completed in 2008.

We have not been able to ascertain how many thousands of miles of logging roads there are throughout the state's watersheds. This is of concern because roads are intricately linked with the health and functional abilities of watersheds. A significant amount of erosion from roads is ultimately delivered to streams, because much of the road network is hydrologically connected to streams (Kattlemann, 1996; Wemple et al., 1996). Roads are also directly connected to streams by gullies and ditches. Logging inevitably increases soil erosion and stream sedimentation, regardless of how carefully it is designed and implemented, as the USFS has concluded (USFS and USBLM, 1997a; c). Stream sedimentation degrades water quality and has many adverse impacts on water quality, aquatic habitats, and aquatic biota (Beschta et al., 2004; Karr et al., 2004).

The construction, reconstruction, and elevated use of roads greatly increases erosion and consequent sediment delivery (Beschta et al., 2004; Karr et al., 2004). Research has consistently shown that elevated road use for timber haul significantly increases road erosion and the delivery of sediment to streams (Reid and Dunne, 1984; Beschta et al., 2004).

Battle Creek Alliance and the Sierra Club wonder why there has been a waiver given for timber harvest in the past and why it might be renewed now when the negative impacts of timber harvest have been researched and documented for so long? If waivers are given, do they not completely circumvent the point of such rules and regulations as the Clean Water Act? Was not the point of such rules and regulations to protect the waters that belong to everyone and to stop the same persistent and damaging impacts that we are writing about here?



A road across from a creek in 2009, the year after the clearcut was done.



A steep clearcut that extends to the edge of a gully that flows into the creek at the bottom of the hill. This is common practice.

In December of 2009, Battle Creek Alliance began a water monitoring project of the tributary streams of Battle Creek throughout the logging area delineated in the photographs and map at the beginning of this comment. We are studying the turbidity of these ancillary streams upstream of the majority of the logging, adjacent to recent logging and downstream of logging. Preliminary results show consistently higher readings in the streams near the most recent logging. In the past several weeks, during and after rainstorms, we have noticed that the water is an odd milky grey color and that there is a strange foam in those streams as well:



Foam appearing in creeks, January and February 2010.



We collected a sample of the foam and took it to be tested for surfactants at Basic Laboratory in Redding on Feb. 3<sup>rd</sup>, 2010 and received the results on Feb. 10<sup>th</sup>, 2010.

The foam tested positively for surfactants at 1.6ppm. Surfactants are added to the herbicides that are applied to land after it has been logged and replanted. These herbicides are used to kill any regenerating native growth. These herbicides and surfactants are known to be toxic to amphibians and have links to a wide range of human health problems also. We have been informed that 1.75ppm is a toxic/lethal level to some amphibians.

Battle Creek Alliance began our water monitoring program because, as far as we have been able to ascertain, public agencies are not monitoring the water quality here in regards to turbidity or chemicals. The timber companies are allowed to self monitor. There is nothing we have seen that gives us any confidence their monitoring is even vaguely adequate. The owner of the land in the Battle Creek watershed owns 1.7 million acres in California. They have said in their recent timber harvest plans that they have instituted a monitoring program that “started in 2000 and through January 22, 2008, has resulted in 3,730 samples being taken for testing”. This works out to a one time test per every 4,558 acres over 8 years of time. There is no data given as to the weather conditions or proximity to logging or the time elapsed between the logging and

herbicide application and the sample being collected. In their Xeroxed section about herbicides that is inserted into each timber harvest plan, they conclude that there will be no “significant adverse impact on the environment.” Battle Creek Alliance and Sierra Club find this conclusion to be wholly devoid of any basis in reality.

Battle Creek Alliance has found that herbicide testing is very expensive and beyond our financial means. We would like to know why this is falling on the public though? Why are the public agencies not performing these tests? Why is the timber industry not performing open and careful tests? Why is the timber industry being allowed to evade the regulations that are supposed to protect our water by being given waivers to the rules?

Battle Creek Alliance and the Sierra Club would like to know why the Water Quality Control Board is not studying the impacts of timber harvest and chemical use? We would also like to know why waivers have been granted to an industry that has so many impacts on the state’s watersheds?

The following is an overview of chemical usage that we wish the Water Quality Control Board to consider when making their decisions:

## We Interact with 100,000+ Chemicals, and the Dangers Are Barely Understood

By Monona Rossol, AlterNet

October 7, 2009

<http://www.alternet.org/story/143130/>

Last month, the Chemical Abstract Service, an agency that registers every new chemical as it is invented or discovered, assigned a registry number to the 50 millionth chemical. It's a landmark to be sure, but not one we're likely to look back on fondly.

The Chemical Abstract Service began to register chemicals in 1956, and it took 33 years to register the first 10 million new chemicals.

They identified these chemicals primarily from research papers accumulated from worldwide sources. But the last 10 million chemicals were registered in nine months at the rate of 25 per minute!

Even more important, their primary source for identifying these chemicals was not research papers. Instead, 60 percent were from major patent offices worldwide. And the next significant category was chemicals already available in chemical catalogs!

In other words, these chemicals are already out of the box and out there.

Of these 50 million chemicals, there are various estimates of the total number of chemicals in commercial use. The number used by most people in the U.S. is 100,000, based on EPA estimates.

This is probably a low estimate since the European Union has registered 140,000 and at this moment still registering more.

You could be forgiven for thinking these chemicals are tested before making their debuts, but that wouldn't make you any less wrong. Our regulatory system works according to a kind of "guilty until proven innocent" logic, where new chemicals are available and safe, until the day we realize they aren't. (Even if they're remarkably similar to chemicals we already know are dangerous.)

How many of these chemicals have been studied for safety? Not many.

If we restrict our concerns to the 100,000 chemicals in commercial use, it is absolutely frightening that only 642 of these have been studied sufficiently for American Conference of Governmental Industrial Hygienists to set workplace air-quality guidelines for them.

As for long-term testing, only about 900 chemicals have been studied for cancer effects with enough depth to be assessed by the major cancer-research agencies, and about 300 chemicals have been assessed for reproductive and developmental effects and birth defects.

Obviously, we can't assume that majority of the 140,000 or even the 50 million chemicals are nontoxic. There are probably 140,000 surprises out there for us. We are really clueless about this swamp of chemicals through which we slog.

The advertising from most manufacturers leaves consumers with the assumption that all of the ingredients they use in their products have been tested for all kinds of toxic effects, including cancer.

If you want to know how they actually test, look at their lab rats. There's one in your mirror.

### **New Formula! New and Improved!**

Sounds great. But since the "new and improved" products usually function almost the same as the originals, it's likely that one or more of the chemicals in the products have been switched with other chemicals that do the same job.

This is called "chemical substitution," and it's a big part of the reason we "need" to keep inventing new chemicals.

Chemical substitution is possible because chemicals that are closely related by formula and structure also have similar physical properties. So chemical substitution is a way for manufacturers to alter their products to avoid regulation -- often without making the product safer for us to use.

For example, if a chemical is banned, manufacturers can look for another chemical that is almost identical to the banned chemical.

Unfortunately, similar chemicals also often have toxic properties that are similar. And often the replacement chemicals are not as well studied and don't even have to be reported as toxic on labels or material safety-data sheets.

If we learn more about chemical substitution, we can use these same principles to select truly safer products. And to begin, we need to look at how chemicals are found to be so toxic that they are banned and must be replaced with substitutes.

### **European Union to the Rescue?**

The U.S. industry practice of creating chemicals and putting them into commerce without testing has been observed critically by the rest of the world and particularly by the newly forming European Union.

It did not want to operate on the faulty U.S. principle that chemicals are "innocent until proven guilty."

The E.U. chose to frame its approach to this problem in the reverse. In short, its position is that chemicals should be "guilty until proven innocent."

In essence, E.U. regulations to say to industries, "if you can't prove your chemicals are safe, you can't put them on our market."

This is called the "precautionary principle." It assumes that in the absence of test data, you cannot assume a chemical is safe and that precautions should be instituted as if the chemical was toxic, until or unless the manufacturer proves otherwise.

This simple principle forms the basis for the E.U. 's regulatory programs.

First on the E.U.'s agenda is getting the testing done. It understood the absurdity of trying to set safety and environmental policies in the absence of toxicity data. It passed regulations that require physiochemical, toxicological and ecotoxicological testing of "all substances manufactured or imported in quantities of 1,000 tons or more."

Estimates are there are over 30,000 of these large-volume commercial chemicals on which there are almost no data. And there are even more smaller-volume chemicals for which testing will have to wait.

The program under which the E.U. requires this chemical testing is called Registration, Evaluation, Authorization [and Restriction] of Chemical Substances. Now, REACH requires each industry to submit the basic test data or the manufacturer will not be allowed to import or sell either the chemical or products containing the chemical anywhere in the E.U.

The first REACH report of chemical test data is scheduled for 2012. By that time, industry will have invented millions of new chemicals, so it's still a race in which industry is winning.

But the E.U. regulations are at least making it a race rather than the compete rout we see here in the U.S.

*Monona Rossol is a chemist, artist and industrial hygienist. She is the president and founder of Arts, Crafts and Theater Safety Inc., a nonprofit corporation dedicated to providing health and safety services to the arts.*

Following is information about Glyphosate and 2,4-D, just two of the many herbicides that are used:



## Glyphosate Factsheet

### Part 1 of 2

[ Part 1 | Part 2 ]

Caroline Cox / Journal of Pesticide Reform v.108, n.3 Fall98

rev.Oct00

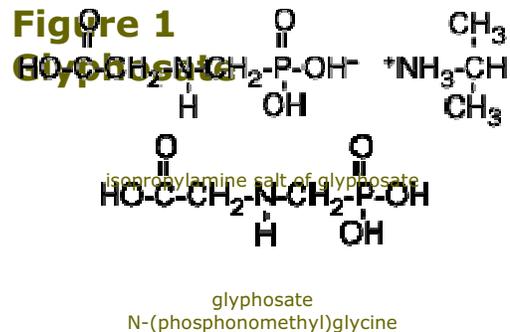
[More on **Monsanto** and its products]

*Caroline Cox is JPR's editor.*

Glyphosate is a broad-spectrum herbicide widely used to kill unwanted plants both in agriculture and in nonagricultural landscapes. Estimated use in the U.S. is between 38 and 48 million pounds per year. Most glyphosate-containing products are either made or used with a surfactant, chemicals that help glyphosate to penetrate plant cells.

Glyphosate-containing products are acutely toxic to animals, including humans. Symptoms include eye and skin irritation, headache, nausea, numbness, elevated blood pressure, and heart palpitations. The surfactant used in a common glyphosate product (Roundup) is more acutely toxic than glyphosate itself the combination of the two is yet more toxic.

Given the marketing of glyphosate herbicides as benign, it is striking that laboratory studies have found adverse effects in all standard categories of laboratory toxicology testing. These include medium-term toxicity (salivary gland lesions), long-term toxicity (inflamed stomach linings), genetic damage (in human blood cells), effects on reproduction (reduced sperm counts in rats; increased frequency of abnormal sperm in rabbits), and carcinogenicity (increased frequency of liver tumors in male rats and thyroid cancer in female rats).



**In studies of people (mostly farmers) exposed to glyphosate herbicides, exposure is associated with an increased risk of miscarriages, premature birth, and the cancer non-Hodgkin's lymphoma.**

**Glyphosate has been called "extremely persistent" by the U.S. Environmental Protection Agency, and half lives of over 100 days have been measured in field tests in Iowa and New York. Glyphosate has been found in streams following agricultural, urban, and forestry applications.**

**Glyphosate treatment has reduced populations of beneficial insects, birds, and small mammals by destroying vegetation on which they depend for food and shelter.**

**In laboratory tests, glyphosate increased plants' susceptibility to disease and reduced the growth of nitrogen-fixing bacteria.**

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Described by their manufacturer as pesticides of "low toxicity and environmental friendliness,"<sup>1</sup> glyphosate-based herbicides can seem like a silver bullet when dealing with unwanted vegetation. However, glyphosate poses a variety of health and environmental hazards. The following article is a summary of those hazards.

Glyphosate, N-(phosphonomethyl) glycine (Figure 1), is a systemic and nonselective herbicide used to kill broadleaved, grass, and sedge species.<sup>2</sup> It has been registered in the U.S. since 1974 and is used to control weeds in a wide variety of agricultural, urban, lawn and garden, aquatic, and forestry situations.<sup>3</sup> Most glyphosate herbicides contain the isopropylamine salt of glyphosate.<sup>4</sup>

Glyphosate products are manufactured by Monsanto Company worldwide. They are marketed under a variety of trade names: Roundup, Rodeo, and Accord are the most common names in the US.<sup>2</sup>

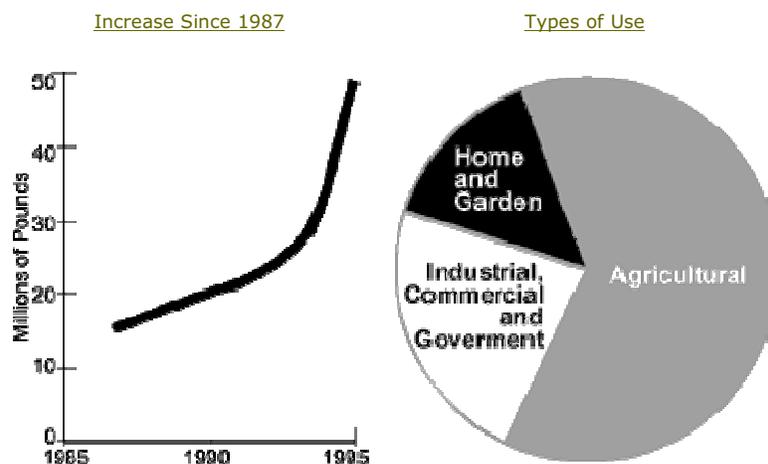
Unlike most other herbicides, chemicals which are closely related to glyphosate are not effective herbicides.<sup>1</sup>

## **Use**

Glyphosate is the seventh most commonly used pesticide in U.S. agriculture, the third most commonly used pesticide on industrial and commercial land, and the second most commonly used home and garden pesticide. Estimated annual use according to the U.S. Environmental Protection Agency (EPA) is between 38 and 48 million pounds.<sup>6</sup> The largest agricultural uses are in the production of soybeans, corn, hay and pasture, and on fallow land.<sup>7</sup> Glyphosate use is currently (1998) growing at a rate of about 20 percent annually, primarily because of the recent introduction of crops which are genetically engineered to be tolerant of the herbicide.<sup>8</sup> (See Figure 2.)

In the U.S., 25 million applications are made yearly on lawns and in yards.<sup>9</sup>

## **Figure 2** **Glyphosate Use in the U.S.**



Aspelin, A. 1. 1990; 1994; 1997. *Pesticide industry sales and usage: 1988 market estimates; 1992 and 1993 market estimates; 1994 and 1995 market estimates.* U.S. EPA. Office of Prevention, Pesticides and Toxic Substances. Office of Pesticide Programs. Biological and Economic Analysis Division. Washington, D.C.

## Mode of Action

Glyphosate's mode of action is "not known at this time,"<sup>4</sup> according to EPA. However, considerable research has established that glyphosate inhibits an enzyme pathway, the shikimic acid pathway, preventing plants from synthesizing three aromatic amino acids. These amino acids are essential for growth and survival of most plants. The key enzyme inhibited by glyphosate is called EPSP synthase.<sup>10</sup> Glyphosate also "may inhibitor repress"<sup>4</sup> two other enzymes, involved in the synthesis of the same amino acids.<sup>4</sup> These enzymes are present in higher plants and microorganisms but not in animals.<sup>10</sup>

Two of the three aromatic amino acids are essential amino acids in the human diet because humans, like all higher animals, lack the shikimic acid pathway, cannot synthesize these amino acids, and rely on their foods to provide these compounds. One is synthesized in animals through another pathway.<sup>11</sup>

Glyphosate can affect plant enzymes not connected with the shikimic acid pathway. In sugar cane, it reduces the activity of one of the enzymes involved in sugar metabolism.<sup>12</sup> It also inhibits a major detoxification enzyme in plants.<sup>13</sup>

Roundup affects enzymes found in mammals. In rats, Roundup decreased the activity of two detoxification enzymes in the liver and an intestinal enzyme.<sup>14</sup>

## "Inert" Ingredients in Glyphosate-containing Products

Virtually every pesticide product contains ingredients other than what is called the "active" ingredient(s), the one designed to provide killing action. These ingredients are misleadingly called "inert." The purpose of these "inerts" is to make the product easier to use or more efficient. In general, they are not identified on the labels of pesticide products.

In the case of glyphosate products, many "inerts" have been identified. See "Toxicology of 'Inert' Ingredients of Glyphosate-containing Products," (at right), for basic information about these "inerts."

Many of the toxicology studies that will be summarized in this factsheet have been conducted using glyphosate, the active ingredient, alone. Some have been conducted with commercial products containing glyphosate and "inert" ingredients. When no testing is done with the product as it is actually used, it is impossible to accurately assess its hazards.

We will discuss both types of studies, and will identify insofar as is possible what material was used in each study.

## Acute Toxicity to Laboratory Animals

Glyphosate's acute oral median lethal dose (the dose that causes death in 50 percent of a population of test animals; LD<sub>50</sub> in rats is greater than 4,320 milligrams per kilogram (mg/kg) of body weight. This places the herbicide in Toxicity Category III (Caution)<sup>4</sup> Its acute dermal toxicity (dermal LD<sub>50</sub>) in rabbits is greater than 2,000 mg/kg of body weight, also Toxicity Category

## **TOXICOLOGY OF "INERT" INGREDIENTS IN GLYPHOSATE CONTAINING PRODUCTS**

Three glyphosate products contain ammonium sulfate.<sup>29, 30, 32</sup> It causes eye irritation, nausea and diarrhea, and may cause allergic respiratory reactions. Prolonged exposure can cause permanent eye damage.<sup>46</sup> One glyphosate product contains benzisothiazolone.<sup>47</sup> It causes eczema, skin irritation,<sup>48</sup> and a light-induced allergic reaction in sensitive people.<sup>49,50</sup> Four glyphosate products contain 3-iodo-2-propynyl butylcarbamate (IPBC).<sup>39-41, 47</sup> It is severely irritating to eyes and increases the incidence of miscarriages in laboratory tests.<sup>51</sup> It also can cause allergic skin reactions.<sup>52</sup> One glyphosate product contains isobutane.<sup>30</sup> It causes nausea, nervous system depression, and difficulty breathing. It is a severe fire hazard.<sup>53</sup> One glyphosate product contains methyl pyrrolidinone.<sup>20</sup> It causes severe eye irritation.<sup>54</sup> It has caused fetal loss and reduced fetal weights in laboratory animals.<sup>55</sup> Three glyphosate products contain pelargonic acid.<sup>29, 30, 32</sup> It causes severe eye and skin irritation and may cause respiratory tract irritation.<sup>56</sup> Nine glyphosate products contain polyethoxylated tallowamine (POEA).<sup>21-24, 31, 35-38</sup> It causes eye burns; skin redness, swelling, and blistering; nausea; and diarrhea.<sup>23, 45</sup> Three glyphosate products contain potassium hydroxide.<sup>29, 30, 32</sup> It causes irreversible eye injury, deep skin ulcers, severe digestive tract burns, and severe irritation of the respiratory tract.<sup>57</sup> One glyphosate product contains sodium sulfite.<sup>34</sup> It may cause eye and skin irritation with vomiting and diarrhea's as well as skin allergies.<sup>59</sup> Exposure to small amounts can cause severe allergic reactions.<sup>60</sup> Three glyphosate products contain sorbic acid.<sup>35, 36, 37</sup> It may cause severe skin irritation, nausea, vomiting, chemical pneumonitis, and sore throat.<sup>61</sup> It also causes allergic reactions.<sup>62, 63</sup> Isopropylamine is used in some Roundup products.<sup>47,64</sup> It is "extremely destructive to tissue of the mucous membranes and upper respiratory tract."<sup>65</sup> Symptoms of exposure are wheezing, laryngitis, headache, and nausea.<sup>65</sup>

### III.<sup>4</sup>

Commercial glyphosate herbicides are more acutely toxic than glyphosate. The amount of Roundup (containing glyphosate and the surfactant POEA) required to kill rats is about 1/3 the amount of glyphosate alone.<sup>15</sup> Roundup is also more acutely toxic than POEA.<sup>15</sup>

Glyphosate-containing products are more toxic via inhalation than orally. Inhalation of Roundup by rats caused "signs of toxicity in all test groups,"<sup>16</sup> even at the lowest concentration tested. These signs included gasping, congested eyes, reduced activity,<sup>16</sup> and body weight loss.<sup>16</sup> Lungs were red or blood-congested.<sup>17</sup> The dose required to cause lung damage and mortality following pulmonary administration of two Roundup products and POEA (when forced into the trachea, the tube carrying air into the lungs) was only 1/10 the dose causing damage orally.<sup>15, 18</sup>

**Effects on the Circulatory System:** When dogs were given intravenous injections of glyphosate, POEA, or Roundup so that blood concentrations were approximately those found in humans who ingested glyphosate, glyphosate increased the ability of the heart muscle to contract. POEA reduced the output of the heart and the pressure in the arteries. Roundup caused cardiac depression.<sup>19</sup>

**Eye Irritation:** NCAP surveyed eye hazards listed on material safety data sheets for 25 glyphosate-containing products. One of the products is "severely irritating,"<sup>20</sup> four cause "substantial but temporary eye injury,"<sup>21-24</sup> eight "cause eye irritation,"<sup>25-32</sup> five "may cause eye irritation,"<sup>33-37</sup> one is "moderately irritating,"<sup>38</sup> and three are "slightly irritating."<sup>39-41</sup> The other three products require addition of a surfactant (wetting agent) before use,<sup>42-44</sup> and the surfactant sold by glyphosate's manufacturer for this purpose "causes eye burns."<sup>45</sup>

**Skin Irritation:** Glyphosate is classified as a slightly irritating to skin. Roundup is a "moderate skin irritant," and recovery can take over two weeks.<sup>16</sup>

## Table 1 Symptoms Following Unintentional Exposure to Glyphosate Herbicides

eye irritation	blisters	chest pains	facial numbness
painful eyes	skin rash	congestion	burning sensation on skin
burning eyes	rapid heartbeat	coughing	itchy skin
blurred vision	heart palpitations	headache	tingling skin
swollen eye, face, joints	elevated blood pressure	nausea	recurrent eczema

Temple, W.A. and N.A. Smith. 1992. Glyphosate herbicide poisoning experience in New Zealand. *N.Z. Med. J.* 105:173-174.  
Calif. EPA. Dept. of Pesticide Regulation. 1998. Case reports received by the California Pesticide Illness Surveillance Program in which health effects were attributed to glyphosate, 1993-1995. Unpublished report.

## Acute Toxicity to Humans

The acute toxicity of glyphosate products to humans was first publicized by physicians in Japan who studied<sup>56</sup> suicide attempts; nine cases were fatal. Symptoms included intestinal pain, vomiting, excess fluid in the lungs, pneumonia, clouding of consciousness, and destruction of red blood cells.<sup>66</sup> They calculated that the fatal cases ingested on average about 200 milliliters (3/4 of a cup). They believed that POEA was the cause of Roundup's toxicity.<sup>66</sup> More recent reviews of poisoning incidents have found similar symptoms, as well as lung dysfunction,<sup>67-69</sup> erosion of the gastrointestinal tract,<sup>67, 69</sup> abnormal electrocardiograms,<sup>69</sup> low blood pressure,<sup>67, 69</sup> kidney damage,<sup>67, 68, 70</sup> and damage to the larynx.<sup>71</sup>

Smaller amounts of Roundup cause adverse effects, usually skin or eye irritation as well as some of the symptoms listed above. (See Table 1.) For example, rubbing of Roundup in an eye caused eye and lid swelling, rapid heartbeat and elevated blood pressure. Wiping the face after touching leaky spray equipment caused swelling of the face. Accidental drenching with horticultural Roundup caused eczema of the hands and arms lasting two months.<sup>63</sup> A spill resulted in dizziness, fever, nausea, palpitations, and sore throat.<sup>72</sup>

## Toxicology Overview

Glyphosate is often portrayed as toxicologically benign: "extensive investigations strongly support the conclusion that glyphosate has a very low level of toxicity..."<sup>73</sup> NCAP's review of glyphosate's toxicology comes to a different conclusion. Adverse effects have been identified in each standard category of testing (subchronic, chronic, carcinogenicity, mutagenicity, and reproduction). NCAP's review has been challenged by the assertion that these effects were found because standard test protocols *require* finding adverse effects at the highest dose tested. However, the following five sections of this article summarize adverse effects did *not* result from this requirement: they were all found at less than the highest dose tested. (The few exceptions are clearly identified.)

## Subchronic Toxicity

In subchronic (medium term) studies of rats and mice done by the National Toxicology Program (NTP), microscopic salivary gland lesions were found in all doses tested in rats (200 - 3400 mg/kg per day) and in all but the lowest dose tested in mice (1,000-12,000 mg/kg per day). (See Figure 3.) A follow-up study by NTP found that the mechanism by which glyphosate caused these lesions involved the hormone adrenalin.<sup>74</sup>

The NTP study also found increases in two liver enzymes at all but the two lowest doses tested. Other effects found in at least two doses in this study were reduced weight gain in rats and mice; diarrhea in rats; and changes in kidney and liver weights in male rats and mice.<sup>74</sup>

Another subchronic laboratory test found that blood levels of potassium and phosphorus in rats increased at all doses tested (60-1600 mg/kg/day).<sup>4</sup>

Glyphosate-containing products are more toxic than glyphosate in subchronic tests. In a 7 day study with calves, 790 mg/kg per day of Roundup caused pneumonia, and death of 1/3 of the animals tested. At lower doses decreased food intake and diarrhea were observed.<sup>2</sup>

## Chronic Toxicity

Glyphosate is also toxic in long-term studies. At all but the lowest dose tested, excessive cell division in the urinary bladder occurred in male mice<sup>2</sup> and inflammation of the stomach lining occurred in both sexes of rats.<sup>2</sup>

## Carcinogenicity

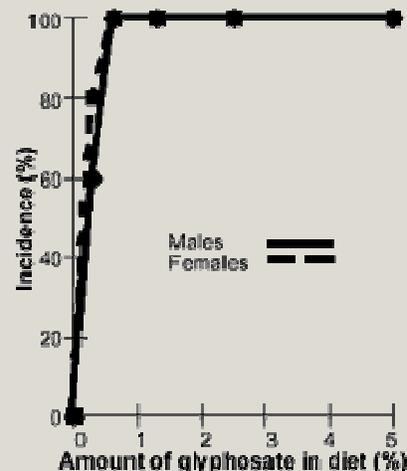
A recent Swedish study of hairy cell leukemia (HCE), a form of the cancer non-Hodgkin's lymphoma, found that people who were occupationally exposed to glyphosate herbicides had a threefold higher risk of HCE. A similar study of people with non-Hodgkin's lymphoma found exposure to glyphosate herbicides was associated with an increase in risk of about the same size.<sup>74ab</sup>

The publicly available laboratory studies of glyphosate's ability to cause cancer were all conducted by or for its manufacturer.<sup>2</sup> The first carcinogenicity study submitted to EPA (1981) found an increase in testicular tumors in male rats at the highest dose tested as well as an increase in the frequency of a thyroid cancer in females. Both results occurred at the highest dose tested (30 mg/kg of body weight per day).<sup>75,76</sup> The second study (1983) found an increasing trend in the frequency of a rare kidney tumor in male mice.<sup>77</sup> The most recent study (1990) found an increase in pancreas and liver tumors in male rats together with an increase of the same thyroid cancer found in the 1983 study in females.<sup>78</sup>

All of these increases in tumor or cancer incidence are "not considered compound-related"<sup>78</sup> according to EPA (This means that EPA did not consider glyphosate the cause of the tumors.) For the testicular tumors, EPA accepted the interpretation of an industry pathologist who said that the incidence in treated groups (12 percent) was similar to those observed (4.5 percent) in other rats not fed glyphosate.<sup>78</sup> For the thyroid cancer, EPA stated that it was not possible to distinguish between cancers and tumors of this type, so that the two should be considered together. The combined data are not statistically significant.<sup>76</sup> For the kidney tumors, the manufacturer reexamined the tissue and found an additional tumor in untreated mice so that statistical significance was lost. This was despite the opinion of EPA's pathologist that the lesion in question was not really a tumor.<sup>77</sup> For the pancreatic tumors, EPA stated that there was no dose-related trend. For the liver and thyroid tumors, EPA stated that pairwise comparisons between treated and untreated animals were not statistically significant.<sup>78</sup>

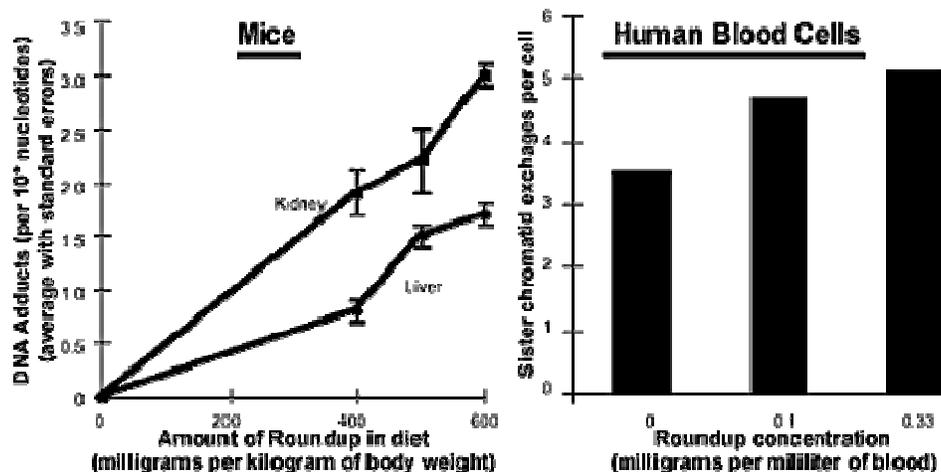
## Figure 4 Genetic Damage Caused by Roundup

**Figure 3**  
**Salivary Gland Lesions in Rats Fed Glyphosate**



U.S. Dept. of Health and Human Services. Public Health Service. National Institutes of Health. 1992. NTP technical report on toxicity studies of glyphosate (CAS No. 1071-83-6) administered in dosed feed to F344/N rats and B6C3F1 mice. Research Triangle Park, NC: National Toxicology Program.

**Glyphosate causes salivary gland lesions in rats, mediated by the hormone adrenalin.**



Peluso, M. et al. 1998. 32P-Postlabeling detection of DNA adducts in mice treated with the herbicide Roundup. *Environ. Molec. Mutag.*31:55-59.

Bolognesi, C. et al. 1997. Genotoxic activity of glyphosate and its technical formulation Roundup. *J. Agric. Food Chem.* 45:1957-1962.

**Roundup causes genetic damage in laboratory animals and in human blood cells.**

EPA concluded that glyphosate should be classified as Group E, "evidence of non-carcinogenicity for humans."<sup>78</sup> They added that this classification "should not be interpreted as a definitive conclusion."<sup>79</sup> The cancer tests leave many questions unanswered. Concerning one of the carcinogenicity studies, an EPA statistician wrote, "Viewpoint is a key issue. Our viewpoint is one of protecting the public health when we see suspicious data. Unfortunately, EPA has not taken that viewpoint in its assessment of glyphosate's cancer-causing potential."

There are no publicly available laboratory studies of the carcinogenicity of Roundup or other glyphosate-containing products.

## Mutagenicity

Although glyphosate's manufacturer describes "a large battery of assays"<sup>80</sup> showing that glyphosate does not cause genetic damage,<sup>80</sup> other studies have shown that both glyphosate and glyphosate products are mutagenic. Glyphosate-containing products are more potent mutagens than glyphosate.<sup>81</sup> The studies include the following:

**In fruit flies,** Roundup and Pondmaster (an aquatic herbicide consisting of glyphosate and a trade secret surfactant<sup>82</sup>) both increased the frequency of sexlinked, recessive lethal mutations. (These are mutations that are usually visible only in males. Only a single concentration was tested in this study.)<sup>83</sup>

**A study of human lymphocytes** (a type of white blood cell) showed an increase in the frequency of sister chromatid exchanges following exposure to the lowest dose tested of Roundup.<sup>84</sup> (Sister chromatid exchanges are exchanges of genetic material during cell division between members of a chromosome pair. They result from point mutations.) A 1997 study of human lymphocytes (see Figure 4) found similar results with Roundup (at both doses tested and with glyphosate (at all but the lowest dose tested)).<sup>81</sup>

**In *Salmonella* bacteria,** Roundup was weakly mutagenic at two concentrations. In onion root cells, Roundup caused an increase in chromosome aberrations, also at two concentrations.<sup>85</sup>

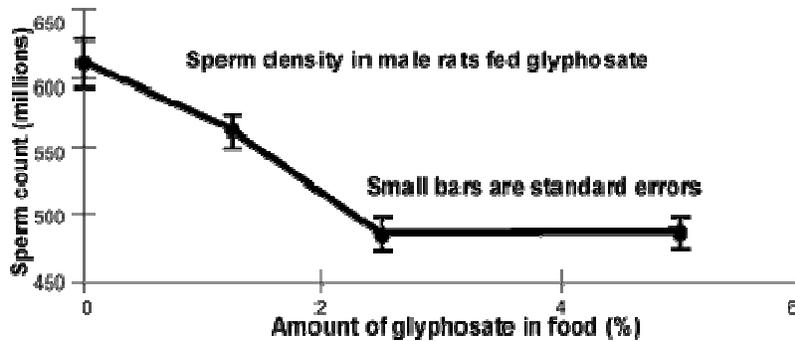
**In mice injected with Roundup,** the frequency of DNA adducts (the binding to genetic material of reactive molecules that lead to mutations) in the liver and kidney increased at all three doses tested.<sup>86</sup> (See Figure 4.)

**In another study of mice injected with glyphosate and Roundup,** the frequency of chromosome damage and DNA damage increased in bone marrow, liver, and kidney. (Only a single concentration was tested in this study.)<sup>81</sup>

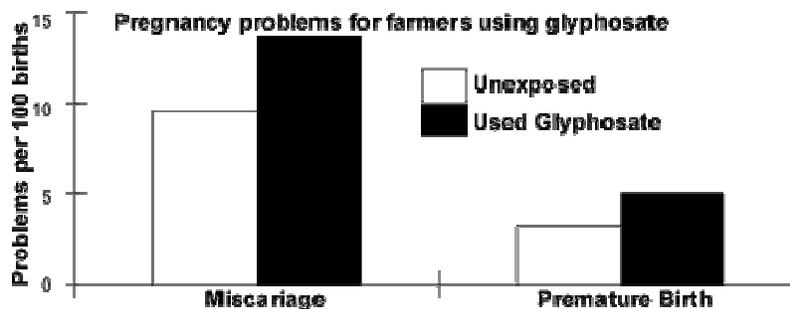
## Reproductive Effects

Glyphosate exposure has been linked to reproductive problems in humans. A study in Ontario, Canada, found that fathers' use of glyphosate was associated with an increase in miscarriages and premature births in farm families.<sup>87</sup> (See Figure 5.) In addition, a case report from the University of California discussed a student athlete who suffered abnormally frequent menstruation when she competed at tracks where glyphosate had been used.<sup>88</sup>

**Figure 5**  
**Effects of Glyphosate on Male Reproductive Success**



U.S. Dept. of Health and Human Services. Public Health Serv. National Inst. Health. 1992. NTP technical report on toxicity studies of glyphosate (CAS No. 1071-83-6) administered in dosed feed to F344/N rats and B6C3F1 mice. Research Triangle Park, NC: National Toxicology Program.



Savitz, D.A. et al. 1997. Male pesticide exposure and pregnancy outcome. *Am. J. Epidemiol.* 146:1025-1036.

**Glyphosate exposure is associated with reproductive problems in both laboratory animals and farmers.**

Laboratory studies have also demonstrated a number of effects of glyphosate on reproduction.

In rats, glyphosate reduced sperm counts at the two highest doses tested. (See Figure 5.) In male rabbits, glyphosate at doses of 1/10 and 1/100 of the LD<sub>50</sub> increased the frequency of abnormal and dead sperm.<sup>89</sup>

Using cells taken from Leydig cell testicular tumors in mice, researchers from Texas Tech University showed that exposure to Roundup (but not glyphosate alone) caused a decrease in the production of sex hormones. Specifically, Roundup inhibited the expression of a protein that carries cholesterol (the molecule from which sex hormones are made to the site where these hormones are synthesized). Lacking necessary amounts of cholesterol, the testicle cells' production of sex hormones decreased about 90 percent.<sup>89a</sup>

In a study of female rabbits, glyphosate caused a decrease in fetal weight in all treated groups.<sup>90</sup>

## **Toxicology of Glyphosate's Major Metabolite**

In general, studies of the breakdown of glyphosate find only one metabolite, aminomethylphosphonic acid (AMPA).<sup>2</sup> Although AMPA has low acute toxicity (its LD<sub>50</sub> is 8,300 mg/kg of body weight in rats),<sup>16</sup> it causes a variety of toxicological problems. In subchronic tests on rats, AMPA caused an increase in the activity of an enzyme, lactic dehydrogenase, in both sexes; a decrease in liver weights in males at all doses tested; and excessive cell division in the lining of the urinary bladder in both sexes.<sup>16</sup> AMPA is more persistent than glyphosate; studies in eight states found that the half-life in soil (the time required for half of the original concentration of a compound to break down or dissipate) was between 119 and 958 days.<sup>2</sup> AMPA has been found in lettuce and barley planted a year after glyphosate treatment.<sup>90a</sup>

## **Quality of Laboratory Testing**

Tests done on glyphosate to meet registration requirements have been associated with fraudulent practices.

Laboratory fraud first made headlines in 1983 when EPA publicly announced that a 1976 audit had discovered "serious deficiencies and improprieties" in studies conducted by Industrial Biotest Laboratories (IBT). Problems included "countless deaths of rats and mice" and "routine falsification of data."<sup>91</sup>

IBT was one of the largest laboratories performing tests in support of pesticide registrations.<sup>91</sup> About 30 tests on glyphosate and glyphosate-containing products were performed by IBT, including 11 of the 19 chronic toxicology studies.<sup>92</sup> A compelling example of the poor quality of IBT data comes from an EPA toxicologist who wrote, "It is also somewhat difficult not to doubt the scientific integrity of a study when the IBT stated that it took specimens from the uteri (of male rabbits for histopathological examination."<sup>93</sup> (Emphasis added.)

In 1991, EPA alleged that Craven Laboratories, a company that performed studies for 262 pesticide companies including Monsanto, had falsified tests.<sup>94</sup> "Tricks" employed by Craven Labs included "falsifying laboratory notebook entries" and "manually manipulating scientific equipment to produce false reports."<sup>95</sup> Roundup residue studies on plums, potatoes, grapes, and sugarbeets were among the tests in question.<sup>96</sup>

The following year, the owner of Craven Labs and three employees were indicted on 20 felony counts.<sup>97</sup> The owner was sentenced to five years in prison and fined \$50,000; Craven Labs was fined 15.5 million dollars, and ordered to pay 3.7 million dollars in restitution.<sup>95</sup>

Although the tests of glyphosate identified as fraudulent have been replaced, this fraud casts shadows on the entire pesticide registration process.

## **Illegal Advertising**

In 1996, Monsanto Co. negotiated an agreement with the New York attorney general that required Monsanto to stop making certain health and environmental claims in ads for glyphosate products and pay the attorney general \$50,000 in costs." Claims that glyphosate products are "safer than table salt,"<sup>98</sup> safe for people, pets, and the environment, and degrade "soon after application" <sup>98</sup> were challenged by the attorney general because they are in violation of the Federal Insecticide, Fungicide and Rodenticide Act (FIFRA), the national pesticide law.<sup>98</sup> According to the attorney-general, Monsanto had engaged in "false and misleading" advertising.<sup>98</sup>

In 1998, Monsanto Co. negotiated a similar agreement with the New York attorney-general about a different advertisement. The attorney general found that the advertisement featuring a horticulturist from the San Diego Zoo also was "false and misleading" because it implied to consumers that Roundup could be used (contrary to label directions) in and around water.<sup>98a</sup> Monsanto paid \$75,000 in costs.<sup>98a</sup>

EPA made a similar determination about Roundup ads in 1998, finding that they contained "false and misleading"<sup>98</sup> claims and were in violation of FIFRA. However, EPA took no action and did not even notify Monsanto Co. about the determination because two years had elapsed between the time that the ads were submitted to EPA and the time that EPA made the determination<sup>99</sup>

## **Human Exposure**

People are exposed to glyphosate through workplace exposure (for people who use glyphosate products on the job), eating of contaminated food, exposure caused by off target movement following application (drift), contact with contaminated soil, and drinking or bathing in contaminated water. The next five sections of this factsheet summarize information about these five routes of exposure. The third section, discussing drift, also covers impacts on plants.

## Contamination of Food

Analysis of glyphosate residues is "in general laborious, complex, and costly."<sup>2</sup>

**" Glyphosate's manufacturer reported that drift from a ground application in Minnesota damaged 25 acres of corn, and the Washington Department of Agriculture reported damage to 30 acres of onions from a ground application of a glyphosate herbicide."**

For this reason, it is not included in government monitoring of pesticide residues in food.<sup>2</sup> The only information available about contamination of food comes from research studies.

Monsanto's studies of residues in food crops found glyphosate in lettuce over five months after treatment (the lettuce was planted four months after treatment). Monsanto also found glyphosate in barley over four months after treatment (the barley was planted one month after treatment).<sup>90a</sup>

"Significant residues,"<sup>2</sup> according to the World Health Organization, have been identified from pre-harvest use of glyphosate on wheat (to dry out the grain). Bran contains between 2 and 4 times the amount on whole grains. Residues are not lost during baking.<sup>2</sup>

## Occupational Exposure

In California, the state with the most comprehensive program for reporting of pesticide-caused illness, glyphosate-containing herbicides were the third most commonly-reported cause of pesticide illness among agricultural workers.<sup>100</sup> Among landscape maintenance workers, glyphosate herbicides were the most commonly reported cause.<sup>101</sup> (Both these statistics come from illness reports collected between 1984 and 1990.) Even when glyphosate's extensive use in California is considered, and the illness statistics presented as "number of acute illnesses reported per million pounds used in California," glyphosate ranked twelfth.<sup>100</sup>

While many of the California reports involve "irritant effects,"<sup>102</sup> mostly to the eyes and skin, NCAP's survey of about 100 reports made in 1993, 1994, and 1995 found that over half of them involved more serious effects: burning of eyes or skin, blurred vision, peeling of skin, nausea, headache, vomiting, diarrhea, chest pain, dizziness, numbness, burning of the genitals, and wheezing.<sup>103</sup>

Other occupational symptoms were observed in a flax milling operation in Great Britain. A study compared the effects of breathing dust from flax treated with Roundup with the effects of dust from untreated flax. Treated dust caused a decrease in lung function and an increase in coughing, and breathlessness.<sup>104</sup>

## Drift

In general, movement of a pesticide through unwanted drift is "unavoidable."<sup>105</sup> Drift of glyphosate is no exception. Glyphosate drift, however, is particularly significant because drift "damage is likely to be much more extensive and more persistent than with many other herbicides."<sup>106</sup> This is because glyphosate moves readily within plants so that even unexposed parts of a plant can be damaged. Damage to perennial plants (when not exposed to enough glyphosate to kill them) is persistent, with some symptoms lasting several years.<sup>106</sup> In addition, plant susceptibility varies widely. Some wildflowers are almost a hundred times more sensitive than others; drift in amounts equal to 1/1000 of typical application rates will damage these species.<sup>107</sup>

A simple answer to the question, "How far can I expect glyphosate to travel off site?" is difficult, since drift is "notoriously variable."<sup>108</sup> However, extensive drift of glyphosate has been measured since the 1970s when a California study found glyphosate 800 m (2600 feet) from aerial and ground applications. Similar drift distances were found for the 8 different spray systems tested in this study.<sup>109</sup>

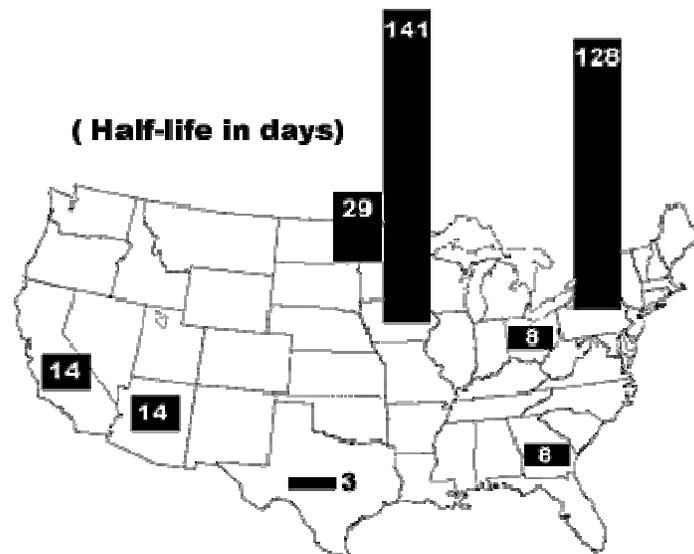
Drift distances that have been measured more recently for the major application techniques include the following:

**Ground Applications:** A study of 15 noncrop plants found seedling mortality (killing about 10 percent of seedlings) for most of the species tested at 20 meters (66 feet) downwind when using a tractor-mounted sprayer. Seedlings of some sensitive species were killed at 40 meters (131 feet).<sup>110</sup> A drift model predicted some native species would be damaged at distances of 80 meters (262 feet).<sup>107</sup> Glyphosate's manufacturer reported that drift from a ground application in Minnesota damaged 25 acres of corn,<sup>111</sup> and the Washington Department of Agriculture reported damage to 30 acres of onions from a ground application of a glyphosate herbicide.<sup>112</sup>

**Helicopter applications:** A study done in Canada<sup>113</sup> measured glyphosate residues 200 meters (656 feet) from target areas following helicopter applications to forest sites. In this study, 200 meters was the farthest distance at which samples were taken, so the longest distance glyphosate traveled is not known.

**Fixed-wing aircraft:** Long drift distances occur following applications of glyphosate made from airplanes. Two studies on forested sites conducted by Agriculture Canada (the Canadian agricultural ministry) showed that glyphosate was found at the farthest distance from the target areas that measurements were made (300 and 400 meters, or 984 and 1312 feet).<sup>114, 115</sup> One of these studies<sup>115</sup> calculated that buffer zones of between 75 and 1200 meters (246 feet - 0.75 miles) would be required to protect nontarget vegetation. According to Monsanto, drift from single aerial applications of glyphosate has been extensive enough to damage 1000 trees in one case,<sup>116</sup> 250 acres of corn in another,<sup>117</sup> and 155 acres of tomatoes in a third incident.<sup>118</sup>

## Figure 6 Persistence of Glyphosate in U.S. Agricultural Soils



Note: Numbers, as well as the length of the columns, give the half-life, in days, of glyphosate in soil. Half-life is the length of time required for half the applied glyphosate to break down or move out of the test site.

Source: U.S. EPA. Environmental Fate and Effects Division. 1993. Pesticide environmental fate one line summary; Glyphosate. Washington, D.C., May 6.

**Glyphosate's persistence in soil varies widely, but its half-life in agricultural soil can be over 4 months.**

## Persistence and Movement in Soil

Glyphosate's persistence in soil varies widely, so giving a simple answer to the question "How long does glyphosate persist in soil?" is not possible. Half-lives (the time required for half of the amount of glyphosate applied to break down or move away) as low as 3 days (in Texas) and as long as 141 days (in Iowa) have been measured by glyphosate's manufacturer.<sup>119</sup> (See Figure 6.) Initial degradation (breakdown) is faster than the subsequent degradation of what remains.<sup>120</sup> Long persistence has been measured in the following studies: 55 days on an Oregon Coast Range forestry site<sup>121</sup>; 249 days on Finnish agricultural soils<sup>122</sup>; between 259 and 296 days on eight Finnish forestry sites<sup>120</sup>; 335 days on an Ontario (Canada) forestry site<sup>123</sup>; 360 days on 3 British Columbia forestry sites<sup>124</sup>; and, from 1 to 3 years on eleven Swedish forestry sites.<sup>125</sup> EPA's Ecological Effects Branch wrote, "In summary, this herbicide is extremely persistent under typical application conditions."<sup>126</sup>

Glyphosate is thought to be "tightly complexed [bound] by most soils"<sup>127</sup> and therefore "in most soils, glyphosate is essentially immobile."<sup>127</sup> This means that the glyphosate will be unlikely to contaminate water or soil away from the application site. However, this binding to soil is "reversible." For example, one study found that glyphosate bound readily to four different soils. However, desorption, when glyphosate unbinds from soil particles, also occurred readily. In one soil, 80 percent of the added glyphosate desorbed in a two hour period. The study concluded that "this herbicide can be extensively mobile in the soil ...."<sup>123</sup>

## Water Contamination

When glyphosate binds readily to soil particles, it does not have the chemical characteristics of a pesticide that is likely to leach into water.<sup>2</sup> (When it readily desorbs, as described above, this changes. However, glyphosate can move into surface water when the soil particles to which it is bound are washed into streams or rivers.<sup>4</sup> How often this happens is not known, because routine monitoring for glyphosate in water is infrequent.<sup>2</sup>

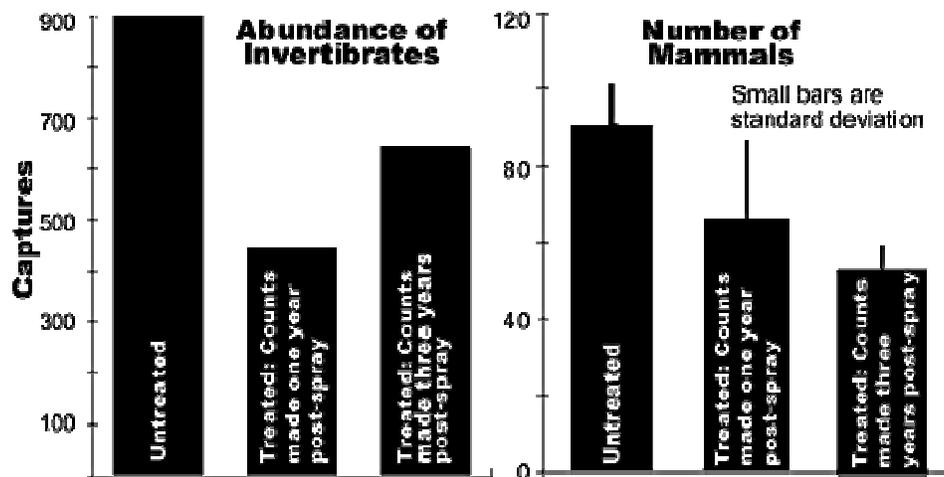
Glyphosate has been found in both ground and surface water. Examples include farm ponds in Ontario, Canada, contaminated by runoff from an agricultural treatment and a spill<sup>129</sup>; the runoff from a watershed treated with Roundup during production of no-till corn and fescue<sup>130</sup>; contaminated surface water in the Netherlands'; seven U.S. wells (one in Texas, six in Virginia contaminated with glyphosate<sup>131</sup>; contaminated forest streams in Oregon and Washington<sup>132, 133</sup>; contaminated streams near Puget Sound, Washington<sup>134</sup>; and contaminated wells under electrical substations treated with glyphosate.<sup>135</sup>

Glyphosate's persistence in water is shorter than its persistence in soils. Two Canadian studies found glyphosate persisted 12 to 60 days in pond water.<sup>136,137</sup> Glyphosate persists longer in pond sediments (mud at the bottom of a pond). For example, the half-life in pond sediments in a Missouri study was 120 days; persistence was over a year in pond sediments in Michigan and Oregon.<sup>4</sup>

## Ecological Effects

Glyphosate can impact many organisms not intended as targets of the herbicide. The next two sections describe both direct mortality and indirect effects, through destruction of food or shelter.

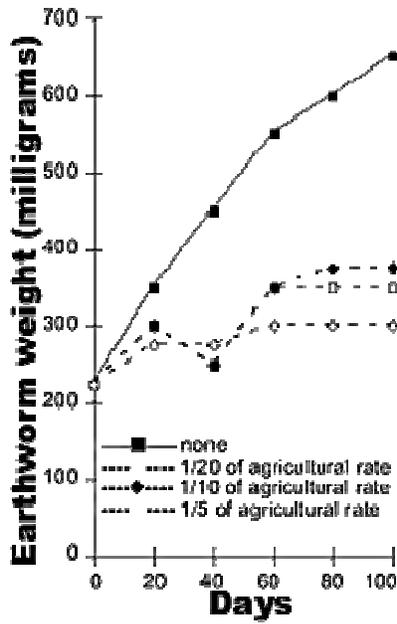
### Figure 7 Impacts of Glyphosate on Nontarget Animals on Maine Clear-cuts



Santillo, D.J., D.M. Leslie, and P.W. Brown. 1989. Responses of small mammals and habitat to glyphosate application on clearcuts. *J. Wildl. Manage.* 53(1):164-172.

**Glyphosate treatment reduced invertebrate and small mammal populations for up to 3 years.**

### Figure 8 Effect of Glyphosate on the Growth of Earthworms



Springer, J.A. and R.A.J. Gray. 1992. Effect of repeated low doses of biocides on the earthworm *Aporrectodea caliginosa* in laboratory culture. *Soil Biol. Biochem.* 24(12):1739-1744.

**Repeated applications of glyphosate reduce the growth of earthworms.**

[ [Part 1](#) | [Part 2](#) ]

## Glyphosate Factsheet

Part 2 of 2

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Caroline Cox / Journal of Pesticide Reform v.108, n.3 Fall98

rev.Oct00

[[More on Monsanto and its products](#)]

Effects on Nontarget Animals

**Beneficial insects:** Beneficial insects kill other species that are agricultural pests. The International Organization for Biological Control found that exposure to freshly dried Roundup killed over 50 percent of three species of beneficial insects: a parasitic wasp, a lacewing, and a ladybug. Over 80 percent of a fourth species, a predatory beetle, was killed.<sup>138</sup> Impacts on beneficial insects have also been shown in field studies, probably due to destruction of their habitat by the herbicide. In North Carolina wheat fields, populations of large carabid beetles declined after treatment with a glyphosate product and did not recover for 28 days.<sup>139</sup> A study of Roundup treatment of hedgerows in the United Kingdom also showed a decline in carabid beetles.<sup>140</sup>

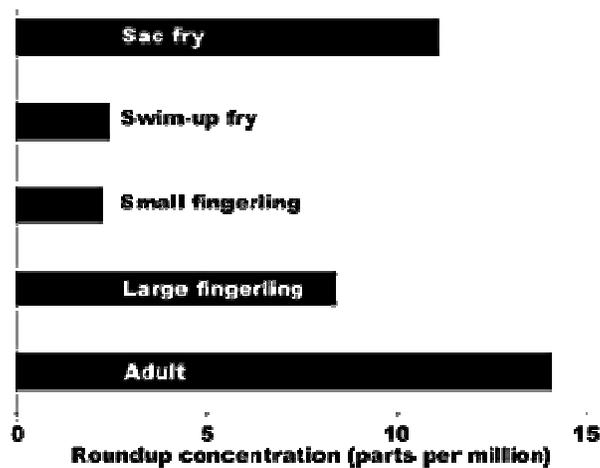
**Other insects:** Roundup treatment of a Maine clear-cut caused an 89 percent decline in the number of herbivorous (plant-eating insects because of the destruction of the vegetation on which they live and feed. (See Figure 7.) These insects serve as food resources for birds and insect-eating small mammals.<sup>141</sup>

The U.S. Fish and Wildlife Service has identified one endangered insect, a longhorn beetle, that would be jeopardized by use of glyphosate herbicides.<sup>142</sup>

**Other arthropods:** Glyphosate and glyphosate-containing products kill a variety of other arthropods. For example, over 50 percent of test populations of a beneficial predatory mite were killed by exposure to Roundup.<sup>138</sup> In another laboratory study, Roundup exposure caused a decrease in survival and a decrease in body weight of woodlice. These arthropods are important in humus production and soil aeration.<sup>143</sup> Roundup treatment of hedgerows reduced the number of spiders, probably by killing the plants they preferred for web-spinning.<sup>140</sup> The water flea *Daphnia pulex* is killed by concentrations of Roundup between 3 and 25 ppm.<sup>144-141</sup> Young *Daphnia* are more susceptible than mature individuals.<sup>145</sup> The red swamp crawfish, a commercial species, was killed by 47 ppm of Roundup.<sup>147</sup>

**Earthworms:** A study of the most common earthworm found in agricultural soils in New Zealand showed that repeated applications of glyphosate significantly affect growth and survival of earthworms. Biweekly applications of low rates of glyphosate (1/20 of typical rates caused a reduction in growth (see Figure 8), an increase in the time to maturity, and an increase in mortality.<sup>148</sup>

## Figure 9 Toxicity of Roundup to Rainbow Trout at Different Ages



Folmar, L.C., H.O. Sanders, and A.M. John. 1979. Toxicity of the herbicide glyphosate and several of its formulations to fish and aquatic invertebrates. Arch. Environ. Contam. Toxicol. 8:269-278.

**Young rainbow trout (swim-up fry and small fingerlings) are more susceptible to Roundup than adult rainbow trout.**

**Fish:** Both glyphosate and the commercial products that contain glyphosate are acutely toxic to fish. In general, glyphosate alone is less toxic than the common glyphosate product, Roundup, and other glyphosate products have intermediate toxicity. Part of these differences can be explained by the toxicity of the surfactant (detergent-like ingredient) in Roundup. It is 20 to 70 times more toxic to fish than glyphosate itself.<sup>144</sup>

Acute toxicities of glyphosate vary widely: median lethal concentrations (LC<sub>50</sub>s; the concentrations killing 50 percent of a population of test animals from 10 ppm to over 200 ppm have been reported depending on the species of fish and test conditions.<sup>2</sup>

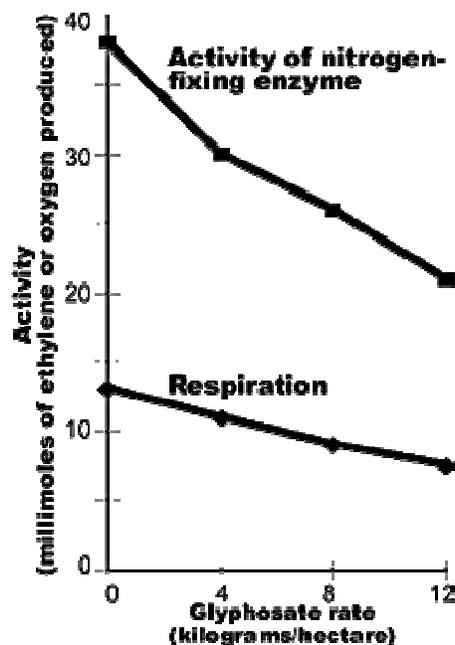
Acute toxicities (LC<sub>50</sub>) of Roundup to fish range from 2 ppm to 55 ppm.<sup>2</sup> Part of this variability is due to age: young fish are more sensitive to Roundup than are older fish.<sup>144</sup> (See Figure 9.) Acute toxicities of Rodeo (used with the surfactant X-77 per label recommendations) vary from 120 to 290 ppm.<sup>149</sup>

In soft water there is little difference between the toxicities of glyphosate and Roundup.<sup>150</sup> Also, if fish have not recently eaten, the toxicity of glyphosate (LC<sub>50</sub> = 2.9 ppm) is similar to that of Roundup.<sup>151</sup>

Roundup toxicity increases with increased water temperature. In both rainbow trout and bluegills, toxicity about doubled between 7 and 17°C (45 and 63°F).<sup>144</sup> Treatment of riparian areas with glyphosate causes water temperatures to increase for several years following treatment<sup>152</sup> because the herbicide kills shading vegetation. This means that use of glyphosate could cause increased toxicity to fish. In addition, the temperature increase could be critical for fish, like juvenile salmon, that thrive in cold water.

Sublethal effects of glyphosate occur at low concentrations. In rainbow trout and *Tilapia* concentrations of about 1/2 and 1/3 of the LC<sub>50</sub> (respectively) caused erratic swimming.<sup>153, 154</sup> The trout also exhibited labored breathing.<sup>153</sup> These effects can increase the risk that the fish will be eaten, as well as affecting feeding, migration, and reproduction.<sup>154</sup> Less than 1 percent of the LC<sub>50</sub> caused gill damage in carp and less than 2 percent caused changes in liver structure.<sup>155</sup>

## Figure 10 Effect of Glyphosate on a Nitrogen-Fixing Bacteria



Santos, A. and M. Flores. 1995. Effects of glyphosate on nitrogen fixation of free-living heterotrophic bacteria. *Let. Appl. Microbiol.* 20:349-352.

**Birds:** Glyphosate has indirect impacts on birds. Because glyphosate kills plants, its use can create a dramatic change in the structure of the plant community. This affects bird populations, since the birds depend on the plants for food, shelter, and nest support.

For example, a study of four glyphosate-treated clear-cuts (and an unsprayed control plot) in Nova Scotia found that the densities of the two most common species of birds (whitethroated sparrow and common yellowthroat) decreased for two years after treatment. By the fourth year post-spray, densities had returned to normal for these two species. By then the unsprayed plot had been colonized by new species of birds (warblers, vireos, and a hummingbird) which were not found on the sprayed plots.<sup>156</sup>

An earlier three year study of songbird abundance following glyphosate treatment of clear-cuts in Maine forests showed similar results. Abundances of the total number of birds and three common species decreased. The decrease in bird abundance was correlated with decrease in the diversity of the habitat.<sup>157</sup>

Black grouse avoided glyphosate-treated clearcuts in Norway for several years after treatment.<sup>158</sup> Researchers recommended that the herbicide not be used near grouse courtship areas.

**Small mammals:** In field studies, small mammals have been indirectly affected when glyphosate kills the vegetation they (or their prey) use for food or shelter. On clear-cuts in Maine,<sup>141</sup> insect-eating shrews declined for three years post-treatment; plant-eating voles declined for two. (See Figure 7.) A second study in Maine after a Roundup treatment<sup>159</sup> found similar results for voles. In British Columbia, deer mice populations were 83 percent lower following glyphosate treatment.<sup>160</sup> Another study from British Columbia found declines in chipmunk populations after Roundup treatment.<sup>161</sup> In Norway, there was a "strong reduction" in use of sprayed clear-cuts by mountain hare.<sup>162</sup> Other studies have not found impacts on small mammals,<sup>163</sup> suggesting that the particular characteristics of the site and the herbicide application are significant.

**Wildlife:** Canadian research has documented that plants serving as important food sources for wildlife are significantly damaged by glyphosate. "Severe" or "very severe damage" was recorded for 46 percent of the important food species eaten by moose, between 34 and 40 percent of the species eaten by elk, and 36 percent of the species eaten by mule deer.<sup>164</sup>

## Effects on Nontarget Plants

As a broad-spectrum herbicide, glyphosate has potent acutely toxic effects on most plant species. There are also other kinds of serious effects. These include effects on endangered species, reduced seed quality, reduction in the ability to fix nitrogen, increased susceptibility to plant diseases, and reduction in the activity of mycorrhizal fungi.

**Endangered species:** Because many plants are susceptible to glyphosate, it can seriously impact endangered plant species. The U.S. Fish and Wildlife Service has identified 74 endangered plant species that it believes could be jeopardized by glyphosate. This list is based on the use of glyphosate on 9 crops, and does not include over 50 other uses.**142**

**Seed Quality:** Sublethal treatment of cotton with Roundup "severely affects seed germination, vigor and stand establishment under field conditions." At the lowest glyphosate rate tested, seed germination was reduced between 24 and 85 percent and seedling weight was reduced between 19 and 83 percent.<sup>165</sup>

**Nitrogen fixation:** Most living things cannot use nitrogen in its common form and instead use ammonia and nitrates, much rarer compounds. Ammonia and nitrates are created by processes called nitrogen fixation and nitrification. They are carried out by bacteria which can be found in soil and in nodules on roots of legumes and certain other plants.<sup>166</sup>

Studies showing effects of glyphosate on nitrogen fixation include the following: At a concentration corresponding to typical application rates, glyphosate reduced by 70 percent the number of nitrogen-fixing nodules on clover planted 120 days after treatment<sup>167</sup>; a similar concentration of a glyphosate herbicide reduced by 27 percent the number of nodules on hydroponically grown clover<sup>168</sup>; a similar concentration of glyphosate reduced by 20 percent nitrogen-fixation by a soil bacteria<sup>169</sup> (see Figure 10); a concentration of glyphosate approximately that expected in soybean roots following treatment inhibited the growth of soybean's nitrogen-fixing bacteria between 10 and 40 percent<sup>170</sup>; and treatment with a glyphosate herbicide at the lowest concentration tested (10 times typical application rates) reduced the number of nodules on clover between 68 and 95 percent.<sup>171</sup>

All of the studies summarized above were done in the laboratory. In the field, such effects have been difficult to observe. However, use of genetically-engineered glyphosate-tolerant crop plants means that nitrogen-fixing bacteria in field situations "could be affected by repeated applications of glyphosate."<sup>170</sup>

Glyphosate also impacts other parts of the nitrogen cycle. A Canadian study found that treatment of a grass field with Roundup increased nitrate loss up to 7 weeks after treatment. The increase was probably caused by the nutrients released into the soil by dying vegetation.<sup>172</sup>

**Mycorrhizal fungi:** Mycorrhizal fungi are beneficial fungi that live in and around plant roots. They help plants absorb nutrients and water and can protect them from cold and drought.<sup>173</sup> Roundup is toxic to mycorrhizal fungi in laboratory studies. Effects on some species associated with conifers have been observed at concentrations of 1 part per million (ppm), lower than those found in soil following typical applications.<sup>174, 175</sup> In orchids, treatment with glyphosate changed the mutually beneficial interaction between the orchid and its mycorrhizae into a parasitic interaction (one that does not benefit the plant).<sup>176</sup>

**Plant diseases:** Glyphosate treatment increases the susceptibility of crop plants to a number of diseases. For example, glyphosate increased the susceptibility of tomatoes to crown and root disease<sup>177</sup>; reduced the ability of bean plants to defend themselves against the disease anthracnose<sup>178</sup>; increased the growth of take-all disease in soil from a wheat field and decreased the proportion of soil fungi which was antagonistic to the take-all fungus<sup>179</sup>; and increased soil populations of two important root pathogens of peas.<sup>180</sup> In addition, Roundup injection of lodgepole pine inhibited the defensive response of the tree to blue stain fungus.<sup>181</sup>

Both the inhibition of mycorrhizae and the increased susceptibility to disease have been observed in laboratory, not field, studies. Given the serious consequences these kinds of effects could have, more research is crucial.

## Plant Resistance

Plants that are resistant to glyphosate are able to tolerate treatment without showing signs of toxicity. Although many weed scientists argue that "it is nearly impossible for glyphosate resistance to evolve in weeds,"<sup>182</sup> others argue that "there are few constraints to weeds evolving resistance." The second group of scientists appears to be correct. In 1996 an Australian researcher reported that a population of annual ryegrass had developed resistance and tolerated five times the recommended field application rate.<sup>183</sup>

## References

*mindfully.org note: hyperlinks within references have not been checked for accuracy.*

1. Franz, J.E., M.K. Mao, and J.A. Sikorski. 1997. *Glyphosate: A unique global herbicide*. ACS Monograph 189. Washington D.C.: American Chemical Society.
2. World Health Organization, United Nations Environment Programme, the International Labour Organization. 1994. *Glyphosate. Environmental Health Criteria #159*. Geneva, Switzerland.
3. U.S. Environmental Protection Agency. 1986. Pesticide fact sheet: Glyphosate. No. 173. Washington, D.C.: Office of Pesticide Programs, June.
4. U.S. EPA. Office of Pesticide Programs. Special Review and Reregistration Division. 1993. *Reregistration eligibility decision (RED): Glyphosate*. Washington, D.C., Sept.
5. Ref.#1, p. 14.
6. Aspelin, A.L. 1997. *Pesticide industry sales and usage: 1994 and 1995 market estimates*. U.S. EPA. Office of Prevention, Pesticides and Toxic Substances. Office of Pesticide Programs. Biological and Economic Analysis Division. Washington, D.C., Aug.
7. Gianessi, L.P. and J.E. Anderson. 1995. Pesticide use in U.S. crop production. Washington, D.C. National Center for Food and Agricultural Policy, Feb.
8. Bureau of National Affairs. Pile & Fisher. 1998. Monsanto reports higher Q2 income for ag chems. *Green Markets Pesticide Report* (Aug. 3):2.
9. Whitmore, R.W., J.E. Kelly, and P.L. Reading. 1992. *National home and garden pesticide use survey*. Final report, Vol. 1: *Executive summary, results, and recommendations*. Research Triangle Park, NC: Research Triangle Institute.
10. Ref.#1, pp.9-10.
11. Metzler, D.E. 1977. *Biochemistry: The chemical reactions of living cells*. Pp. 849-850. New York, NY: Academic Press.
12. Su, L.Y. et al. 1992. The relationship of glyphosate treatment to sugar metabolism in sugarcane: New physiological insights. *J. Plant Physiol.* 140:168-173.
13. Lamb, D.C. et al. 1998. Glyphosate is an inhibitor of plant cytochrome P450: Functional expression of *Thlaspi arvensae* cytochrome P45071B1/ reductase fusion protein in Escherichia coli. *Biochem. Biophys. Res. Comm.* 244:110114.
14. Hietanen, E., K. Linnainmaa, and H. Vainio. 1983. Effects of phenoxy herbicides and glyphosate on the hepatic and intestinal biotransformation activities in the rat. *Acta Pharma. et Toxicol.* 53:103-112.
15. Martinez, T.T. and K. Brown. 1991. Oral and pulmonary toxicology of the surfactant used in Roundup herbicide. *Proc. West. Pharmacol Soc.* 34:43-46.
16. Agriculture Canada. Food Production and Inspection Branch. Pesticides Directorate. 1991. *Discussion document: Pre-harvest use of glyphosate*. Ottawa, Ontario, Canada., Nov. 27.
17. U.S. EPA. Office of Pesticides and Toxic Substances. 1982. Memo from William Dykstra, Toxicology Branch, to Robert Taylor, Registration Division, April 29.
18. Martinez, T.T., W.C. Long, and R. Hiller. 1990. Comparison of the toxicology of the herbicide Roundup by oral and pulmonary routes of exposure. *Proc. West. Pharmacol. Soc.* 34:43-46.

19. [Tai, T. 1990. Hemodynamic effects of Roundup, glyphosate and surfactant in dogs. Jpn. J. Toxicol. 3\(1\):63-68. Cited in World Health Organization, United Nations Environment Programme, the International Labour Organization. 1994. \*Glyphosate. Environmental Health Criteria #159\*. Geneva, Switzerland.](#)
20. [Monsanto Co. 1995. Material safety data sheet: Landmaster BW. \[www.monsanto.com/ag/\]\(http://www.monsanto.com/ag/\), Mar.](#)
21. [Monsanto Co. 1997. Material safety data sheet: Roundup RT. \[www.monsanto.com/ag/\]\(http://www.monsanto.com/ag/\), May.](#)
22. [Monsanto Co. 1997. Material safety data sheet: Roundup Original RT. \[www.monsanto.com/ag/\]\(http://www.monsanto.com/ag/\), Nov.](#)
23. [Monsanto Co. 1994. Material safety data sheet: Roundup. \[www.monsanto.com/ag/\]\(http://www.monsanto.com/ag/\), Jan.](#)
24. [Monsanto Co. 1995. Material safety data sheet: Roundup Super Concentrate Weed & Grass Killer.](#)
25. [Monsanto Co. 1995. Material safety data sheet: Roundup Ultra. \[www.monsanto.com/ag/\]\(http://www.monsanto.com/ag/\), Nov.](#)
26. [Monsanto Co. 1995. Material safety data sheet: Roundup Ultra RT. \[www.monsanto.com/ag/\]\(http://www.monsanto.com/ag/\), Dec.](#)
27. [Monsanto Co. 1998. Material safety data sheet: Roundup DPak. \[www.monsanto.com/ag/\]\(http://www.monsanto.com/ag/\), Feb.](#)
28. [Monsanto Co. 1995. Material safety data sheet: Roundup Pro. \[www.monsanto.com/ag/\]\(http://www.monsanto.com/ag/\), Nov.](#)
29. [Monsanto Co. 1997. Material safety data sheet: Roundup Fence and Yard Edger.](#)
30. [Monsanto Co. 1996. Material safety data sheet: Roundup Sure Shot Foam.](#)
31. [Monsanto Co. 1996. Material safety data sheet: GroundClear Super Edger Grass & Weed Control. \[www.ortho.corn/content/products/Solaris-msds/SOLMSDS.HTML\]\(http://www.ortho.corn/content/products/Solaris-msds/SOLMSDS.HTML\), Oct.](#)
32. [Monsanto Co. 1997. Material safety data sheet: Roundup Ready-To-Use Weed & Grass Killer.](#)
33. [Monsanto Co. 1998. Material safety data sheet: Roundup SoluGran. \[www.monsanto.com/ag/\]\(http://www.monsanto.com/ag/\), Apr.](#)
34. [Monsanto Co. 1994. Material safety data sheet: Roundup Dry Pak. \[www.monsanto.com/ag/\]\(http://www.monsanto.com/ag/\), Feb.](#)
35. [Monsanto Co. 1995. Material safety data sheet: Roundup Concentrate Brush Killer.](#)
36. [Monsanto Co. 1995. Material safety data sheet: Roundup Concentrate Weed & Grass Killer.](#)
37. [Monsanto Co. 1995. Material safety data sheet: Roundup Tough Weed Formula.](#)
38. [Monsanto Co. 1995. Material safety data sheet: Kleeraway Systemic Weed & Grass Killer. \[www.ortho.corn/content/products/Solaris-msds/SOLMSDS.HTML\]\(http://www.ortho.corn/content/products/Solaris-msds/SOLMSDS.HTML\), July.](#)
39. [Monsanto Co. 1995. Material safety data sheet: Yard Basics Weed & Grass Killer. \[www.ortho.corn/content/products/Solarismsds/SOLMSDS.HTML\]\(http://www.ortho.corn/content/products/Solarismsds/SOLMSDS.HTML\), Aug.](#)
40. [Monsanto Co. 1994. Material safety data sheet: KLEENUP Grass & Weed Killer. \[www.ortho.com/content/products/Solarismsds/SOLMSDS.HTML\]\(http://www.ortho.com/content/products/Solarismsds/SOLMSDS.HTML\), June.](#)
41. [Monsanto Co. 1995. Material safety data sheet: Kleeraway Grass & Weed Killer. \[www.ortho.com/content/products/Solarismsds/SOLMSDS.HTML\]\(http://www.ortho.com/content/products/Solarismsds/SOLMSDS.HTML\), July.](#)
42. [Monsanto Co. 1996. Roundup Custom specimen label. \[www.monsanto.com/ag/\]\(http://www.monsanto.com/ag/\), Oct.](#)
43. [Monsanto Co. 1997. Rodeo specimen label. \[www.monsanto.com/ag/\]\(http://www.monsanto.com/ag/\), July.](#)
44. [Monsanto Co. 1997. Accord specimen label. \[www.monsanto.com/ag/\]\(http://www.monsanto.com/ag/\), Aug.](#)

45. [Monsanto Co. 1997. Material safety data sheet: Entry II Surfactant. www.monsanto.com/ag/](http://www.monsanto.com/ag/) , Aug.
46. [Fisher Scientific. 1997. Material safety data sheet: ammonium sulfate. www.fisher1.com/fb/tv?16..f97.1.msa0002.68..1.9](http://www.fisher1.com/fb/tv?16..f97.1.msa0002.68..1.9) , Dec. 12.
47. U.S. EPA. Office of Prevention, Pesticides and Toxic Substances. Office of Pesticide Programs. 1998. Letter from Linda Travers, director, Information Resources and Services Division, to Caroline Cox, NCAP, July 28.
48. [Damstra, R.J., W.A. van Vloten, and C.J.W. van Ginkel. Allergic contact dermatitis from the preservative 1,2benzisothiazolin-3-one \(1,2-BIT; Proxel®\): a case report, its prevalence in those occupationally at risk and in the general dermatological population, and its relationship to allergy to its analogue Kathon® CG. \*Cent. Dermal.\* 27:105-109.](#)
49. [Hindson, C. and B. Diffey. 1993. Phototoxicity of glyphosate in a weedkiller. \*Cent. Derm.\* 10:51-52.](#)
50. [Hindson, C. and B. Diffey. 1993. Phototoxicity of a weedkiller: a correction. \*Cont. Doom.\* 11:260.](#)
51. U.S. EPA. Prevention, Pesticides and Toxic Substances. 1997. Reregistration eligibility decision (RED): 3-Iodo-2-propynyl butylcarbamate (IPBC). Washington, D.C., Mar.
52. [Bryld, L.E. et al. 1997. Iodopropynyl butylcarbamate: a new contact allergen. \*Cent. Dermal.\* 36:156-158.](#)
53. [MG Industries. 1997. Material safety data sheet: Isobutane. www.mgindustries.com/msds/SubLookup.asp?SubName=11600](http://www.mgindustries.com/msds/SubLookup.asp?SubName=11600) , Dec. 9.
54. [FisherScientific.1997.Materialsafetydatasheet:lmethyl-2pyrrolidinone,99%. www.fisher1.com/fb/itv?16..f97.1.msa0010h14k.1.9](http://www.fisher1.com/fb/itv?16..f97.1.msa0010h14k.1.9) , Dec. 12.
55. [Hass, U. B.M. Jakobsen, and S.P Lund. 1995. Developmental toxicity of inhaled n-methylpyrrolidinone in the rat. \*Pharm. Toxicol.\* 76:406-409.](#)
56. [Acros Organics. 1997. Material safety data sheet: Nonanoic acid, tech., 90%. www.fisher1.com/fb/itv?16..f97.1.msa0011.592..1.9](http://www.fisher1.com/fb/itv?16..f97.1.msa0011.592..1.9) , Sept. 2.
57. [Acros Organics. 1997. Material safety data sheet: potassium hydroxide, c.p., flakes. www.fisher1.com/fb/itv?16..f97.1.msa0012.838..1.9](http://www.fisher1.com/fb/itv?16..f97.1.msa0012.838..1.9) , Sept. 2.
58. [Acros Organics. 1997. Material safety data sheet: sodium sulfite. www.fisher1.com/fb/itv?16..f97.1.msa0013.666..1.9](http://www.fisher1.com/fb/itv?16..f97.1.msa0013.666..1.9) , Sept. 2.
59. [Lodi, A. et al. 1993. Contact allergy to sodium sulfite contained in an antifungal preparation. \*Cent. Dermatit.\* 29:97.](#)
60. [Anonymous. 1986. MSDS for sodium sulfite, anhydrous. www.chem.utah.edu/MSDS/S/SODIUM\\_SULFITE\\_ANHYDROUS](http://www.chem.utah.edu/MSDS/S/SODIUM_SULFITE_ANHYDROUS) , Aug. 18.
61. [Acros Organics. 1997. Material safety data sheet: 2,4hexadienoic acid, 99%. www.fisher1.com/fb/itv?16..f97.1.msa0008.574..1.9](http://www.fisher1.com/fb/itv?16..f97.1.msa0008.574..1.9) , Nov. 10.
62. [Lamey, P; J., A.B. Lamb, and A. Forsyth. 1987. Atypical burning mouth syndrome. \*Cont. Dermatit.\* 17:242-2443.](#)
63. [Giordano-Labadie, F., C. Pech-Ormieres, and J. Bazek. 1996. Systemic contact dermatitis from sordid acid. \*Cent. Dermatit.\* 34:61-62.](#)
64. [Monsanto Co. Undated. Monsanto backgrounder: Roundup herbicide ingredients. St. Louis, MO.](#)
65. [Sigma Chemical Co., Aldrich Chemical Co., and Fluke Chemical Corp. 1994. Material safety data sheet: Isopropylamine. St. Louis, MO, Milwaukee, WI, and Ronkonkoma, NY.](#)
66. [Sawada, Y., et al. 1988. Probable toxicity of surface-active agent in commercial herbicide containing glyphosate. \*Lancet\* 1\(8580\):299.](#)
67. [Tominack, R.L. et al. 1991. Taiwan National Poison Center: Survey of glyphosate-surfactant herbicide ingestions. \*Clin. Toxicol.\* 29\(1\):91-109.](#)
68. [Temple, W.A. and N.A. Smith. 1992. Glyphosate herbicide poisoning experience in New Zealand. \*N.Z. Med. J.\* 105:173-174.](#)

69. Talbot, A.R. et al. 1991. Acute poisoning with a glyphosate-surfactant herbicide ('Roundup'): A review of 93 cases. *Human Exp. Toxicol.* 10:1-8.
70. Menkes, D.B., W.A. Temple, and I.R. Edwards. 1991. Intentional self-poisoning with glyphosate-containing herbicides. *Human Exp. Toxicol.* 10:103-107.
71. Hung, D., J. Deng, and T. Wu. 1997. Laryngeal survey in glyphosate intoxication: a pathophysiological investigation. *Hum. Exp. Toxicol.* 16:596599.
72. U.S. EPA. Office of Pesticide Programs. Hazard Evaluation Division. Health Effects Branch. 1980. Summary of reported pesticide incidents involving glyphosate (isopropylamine salt). Report No. 373. Washington, D.C., Oct.
73. Ref.#1, p.128.
74. U.S. Dept. of Health and Human Services. Public Health Service. National Institutes of Health. 1992. NTP technical report on toxicity studies of glyphosate (CAS No. 1071-83-6) administered in dosed feed to F344/N rats and B6C3F1 mice. (NIH Publication 92-3135). Toxicity Reports Series No. 16. Research Triangle Park, NC: National Toxicology Program.
- 74a. Nordstrdm, M et al. 1998. Occupational exposures, animal exposure and smoking as risk factors for hairy cell leukemia evaluated in a case-control study. *Brit. J. Cancer* 77(11):20482052.
746. Hardell, L. and M. Eriksson. Undated. Case-control study of non-Hodgkin's lymphoma and exposure to pesticides. Unpublished poster.
75. U.S. EPA. Office of Pesticides and Toxic Substances. 1982. EPA Reg. #524-308; Lifetime feeding study in rats with glyphosate. Memo from William Dykstra, Health Effects Division to Robert Taylor, Registration Div. Washington, D.C., Feb. 18.
76. U.S. EPA. Office of Pesticides and Toxic Substances. 1983. Glyphosate; EPA Reg. #524-308; A lifetime feeding study of glyphosate in Sprague-Dawley rats; a preliminary addendum to review dated 2/18/83. Memo to Robert Taylor, Registration Div. Washington, D.C., Feb. 15.
77. U.S. EPA. Office of Pesticides and Toxic Substances. 1985. Glyphosate -Evaluation of kidney tumors in male mice. Chronic feeding study. Memo from L. Kassa, Toxicology Branch, to W. Dykstra, Toxicology Branch. Washington, D.C., Dec. 4.
78. U.S. EPA. Office of Pesticides and Toxic Substances. 1991. Second peer review of glyphosate. Memo from W. Dykstra and G.Z. Ghali, Health Effects Division to R. Taylor, Registration Division, and Lois Rossi, Special Review and Reregistration Division. Washington, D.C., Oct. 30.
79. U.S. EPA Office of Pesticides and Toxic Substances. 1985. Use of historical data in determining the weight of evidence from kidney tumor incidence in the glyphosate two-year feeding study; and some remarks on false positives. Memo from Herbert Lacayo to Reto Engler (both Office of Pesticide Programs, Health Effects Division). Washington, D.C., Feb. 26.
80. Ref.#1, p.108.
81. Bolognesi, C. et al. 1997. Genotoxic activity of glyphosate and its technical formulation Roundup. *J. Agric. Food Chem.* 45:1957-1962.
82. Monsanto Co. 1988. Material safety data sheet: Pondmaster aquatic herbicide. St. Louis, MO., Apr.
83. Kale, P.G. et al. 1995. Mutagenicity testing of nine herbicides and pesticides currently used in agriculture. *Environ. Mol. Mutagen.* 25:148-153.
84. Vigfusson, N.V. and E.R. Vyse. 1980. The effect of the pesticides, Dexon, Caftan and Roundup on sister-chromatid exchanges in human lymphocytes in vitro. *Mut. Res.* 79:53-57.
85. Rank, J. et al. 1993. Genotoxicity testing of the herbicide Roundup and its active ingredient glyphosate isopropylamine using the mouse bone marrow micronucleus test, Salmonella mutagenicity test, and Allium anaphase-telophase test. *Met. Res.* 300:29-36.
86. Peluso, M. et al. 1998. <sup>32</sup>P-Postlabeling detection of DNA adducts in mice treated with the herbicide Roundup. *Environ. Molec. Mutag.* 31:55-59.
87. Savitz, D.A. et al. 1997. Male pesticide exposure and pregnancy outcome. *Am. J. Epidemiol.* 146:1025-1036.

88. Barnard, R.J. and G. Hauser. 1995. Commonly used pesticides may help maintain facilities but can hinder athletes. *NCAA Sports Sciences Education Newsletter* (Fall):2.
89. Yousef, M.I. et al. 1995. Toxic effects of carbofuran and glyphosate on semen characteristics in rabbits. *J. Environ. Sci. Health B30*(4):513-534.
- 89a Welsh, L.P. et al. 2000. **Roundup inhibits steroidogenesis by disrupting steroidogenic acute regulatory (StAR) protein expression.** *Environ. Health Persp.* 108:769-776.
90. U.S. EPA. Office of Toxic Substances. 1980. EPA Reg. #524308; glyphosate; submission of rat teratology, rabbit teratology, dominant lethal mutagenicity assay in mice. Memo from W. Dykstra, Health Effects Division, to Robert Taylor, Registration Division. Washington, D.C., June 17.
- 90a Monsanto Agricultural Company. 1990. Confined rotational crop study of glyphosate. Part II: Quantitation, characterization, and identification of glyphosate and its metabolites in rotational crops. St. Louis MO, June 22.
91. U.S. Congress. House of Representatives. Committee on Government Operations. 1984. Problems plague the Environmental Protection Agency's pesticide registration activities. House Report 98-1147. Washington, D.C.: U.S. Government Printing Office.
92. U.S. EPA. Office of Pesticides and Toxic Substances. 1983. Summary of the IBT review program. Washington, D.C., July.
93. U.S. EPA. 1978. Data validation. Memo from K. Locke, Toxicology Branch, to R. Taylor, Registration Branch. Washington, D.C., Aug. 9.
94. U.S. EPA. Communications and Public Affairs. 1991. Note to correspondents. Washington, D.C., Mar. 1.
95. U.S. EPA. Communications, Education, And Public Affairs. 1994. Press advisory. Craven Laboratories, owner, and 14 employees sentenced for falsifying pesticide tests. Washington, D.C., Mar. 4.
96. U.S. EPA. Communications and Public Affairs. 1991. Press advisory. EPA lists crops associated with pesticides for which residue and environmental fate studies were allegedly manipulated. Washington, D.C., Mar. 29.
97. U.S. Dept. of Justice. United States Attorney. Western District of Texas. 1992. Texas laboratory, its president, 3 employees indicted on 20 felony counts in connection with pesticide testing. Austin, TX., Sept. 29.
98. Attorney General of the State of New York. Consumer Frauds and Protection Bureau. Environmental Protection Bureau. 1996. In the matter of Monsanto Company, respondent. Assurance of discontinuance pursuant to executive law § 63(15). New York, NY, Nov.
- 98a Attorney General of the State of New York. Consumer Frauds and Protection Bureau. Environmental Protection Bureau. 1998. In the matter of Monsanto Company, respondent. Assurance of discontinuance pursuant to executive law § 63(15). New York, NY, Apr.
99. U.S. EPA. Region VII. 1998. Letter from L.A. Flournoy, chief, Pesticides Branch, to Pete Haws, NCAP, Mar. 4.
100. Pease, W.S. et al. 1993. Preventing pesticide-related illness in California agriculture: Strategies and priorities. Environmental Health Policy Program Report. Berkeley, CA: University of Calif. School of Public Health. Calif. Policy Seminar.
101. Robinson, J.C. et al. 1994. Pesticides in the home and community: Health risks and policy alternatives. Environmental Health Policy Program Report. Berkeley, CA: University of Calif. School of Public Health. Calif. Policy Seminar.
102. Calif. EPA. Dept. of Pesticide Regulation. 1996. California pesticide illness surveillance program: Summary report. 1994. Health and Safety Report HS-1734.
103. Calif. EPA. Dept. of Pesticide Regulation. 1998. Case reports received by the California Pesticide Illness Surveillance Program in which health effects were attributed to glyphosate, 1993-1995. Unpublished report. Sacramento, CA, Aug.
104. Jamison, J.P., J.H.M. Langlands, R.C. Lowry. 1986. Ventilatory impairment from pre-harvest ratted flax. *Brit. J. Ind. Med.* 43:809-813.
105. Ware, G.W. et al. 1983. Reducing pesticide application *drift-losses*. Tucson, AZ: Univ. of Arizona. College of Agriculture. Coop. Extension Service.

106. Atkinson, D. 1985. Glyphosate damage symptoms and the effects of drift. Appendix I. In Grossbard, E. and D. Atkinson. The herbicide glyphosate. London Butterworths.
107. Breeze, V., G. Thomas, and R. Butler. 1992. Use of a model and toxicity data to predict the risks to some wild plants from drift of four herbicides. Ann. Appl. Biol. 121:669-677.
108. Freedman, B. 1990-1991. Controversy over the use of herbicides in forestry, with particular reference to glyphosate usage. J. Envir. Sci. Hlth. C8(2):277-286.
109. Yates, W.E., N.B. Akesson, and D.E. Bayer. 1978. Drift of glyphosate sprays applied with aerial and ground equipment. Weed Sci. 26(6):597-604.
110. Marrs, R.H. et al. 1993. Determination of buffer zones to protect seedlings of non-target plants from the effects of glyphosate spray drift. Ague. Ecosys. Environ. 45:283-293.
111. Monsanto Co. 1992. Letter from R.M. Weppelman, Product Registration and Regulatory Affairs Manager, to U.S. EPA Office of Pesticide Programs, June 16.
112. Washington State Dept. of Health. 1993. Pesticide incident reporting and tracking review panel. Annual report 1992. Olympia, WA, Feb.
113. Riley, C.M., C.J. Weisner, and W.A. Sexsmith. 1991. Estimating off-target spray deposition on the ground following the aerial application of glyphosate for conifer release in New Brunswick. J. Environ. Sci. Health B26(2):185-208.
114. Payne, N.J. 1993. Spray dispersal from aerial silvicultural applications. Crop Protec. 12(6):463-469.
115. Payne, N.J. 1992. Off-target glyphosate from aerial silvicultural applications, and buffer zones required around sensitive areas. Pestic. Sci. 34:1-8.
116. Monsanto Co. 1991. Letter from R.M. Weppelman, Product Registration and Regulatory Affairs Manager, to U.S. EPA Office of Pesticide Programs, Oct 28.
117. Monsanto Co. 1992. Letter from R.M. Weppelman, Product Registration and Regulatory Affairs Manager, to U.S. EPA Office of Pesticide Programs, Aug. 21.
118. Monsanto Co. 1992. Letter from R.M. Weppelman, Product Registration and Regulatory Affairs Manager, to U.S. EPA Office of Pesticide Programs, May 5.
119. U.S. EPA. Environmental Fate and Effects Division. 1993. Pesticide environmental fate one line summary; Glyphosate. Washington, D.C., May 6.
120. Torstensson, L. and Stark, J. 1979. Persistence of glyphosate in forest soils. In Weeds and weed control. 20th Swedish Weed Conference. Uppsala. 31 January-2 February 1979. Uppsala, Sweden: Swedish Univ. of Agricultural Sciences.
121. Newton, M. et al. 1984. Fate of glyphosate in an Oregon forest ecosystem. J. Ague. Food. Chem. 32:1144-1151.
122. Muller, M. et al. 1981. Fate of glyphosate and its influence on nitrogen-cycling in two Finnish agricultural soils. Bull. Environ. Contam. Toxicol 27:724-730. 123. Fang, J.C. and D.G. Thompson. 1990. Fate of glyphosate in a Canadian forest watershed. 2. Persistence in foliage and soils. J. Ague. Food. Chem. 38:1118-1125. 124. Roy, D.N. et al. 1989. Persistence, movement, and degradation of glyphosate in selected Canadian boreal forest soils. J. Agric. Food. Chem. 37:437-440.
125. Torstensson, N.T.L., L.N. Lundgren, and J. Stenström. 1989. Influence of climate and edaphic factors on persistence of glyphosate and 2,4-D in forest soils. Ecotoxicol. Environ. Safety 18:230-239.
126. U.S. EPA. Ecological Effects Branch. 1993. Science chapter for reregistration eligibility document for glyphosate. Washington, D.C., May 1.
127. Ref.#1. p.79.
128. Piccolo, A. et al. 1994. Adsorption and desorption of glyphosate in some European soils. J. Environ. Sci. Health B29(6):1105-1115.

129. Frank, R. et al. 1990. Contamination of rural ponds with pesticide, 1971-1985, Ontario, Canada. *Bull. Environ. Contam. Toxicol.* 44:401409.
130. Edwards, W.M., G.B. Triplett, Jr., and R.M. Kramer. 1980. A watershed study of glyphosate transport in runoff. *J. Environ. Qual.* 9(4):661665.
131. U.S. EPA. Prevention Pesticides and Toxic Substances. 1992. *Pesticides in groundwater database. A compilation of monitoring studies: 1971-1991. National summary.* Washington, D.C.
132. Rashin, E. and C. Grader. 1993. Effectiveness of best management practices for aerial application of forest pesticides. TFW-WQ1-93-001. Olympia, WA: Washington State Dept. of Ecology, Oct.
133. Oregon Dept. of Forestry. Forest Practices Program. 1992. Forest herbicide application water sampling study. Salem, OR, Jan.
134. Bortleson, G.C. and D.A. Davis. 1997. Pesticides in selected small streams in the Puget Sound Basin, 1987-1995. U.S. Geological Survey. Fact Sheet 067-97. Tacoma, WA, June.
135. Smith, N.J., R.C. Martin, and R.G. St. Croix. 1996. Levels of the herbicide glyphosate in well water. *Bull. Environ. Contam. Toxicol.* 57:759756.
136. Goldsborough, L.G. and A.E. Beck. 1989. Rapid dissipation of glyphosate in small forest ponds. *Arch. Environ. Contam. Toxicol.* 18:537-544.
137. Goldsborough, L.G. and D.J. Brown. 1993. Dissipation of glyphosate and aminomethylphosphonic acid in water and sediments of boreal forest ponds. *Environ. Toxicol. Chem.* 12:1139-1147.
138. Hassan, S.A. et al. 1988. Results of the fourth joint pesticide testing programme carried out by the IOBC/WPRS-Working Group "Pesticides and Beneficial Organisms." *J. Appl. Ent* 105:321329.
139. Brust, G.E. 1990. Direct and indirect effects of four herbicides on the activity of carabid beetles (Coleoptera: Carabidae). *Pestle. Sci.* 30:309-320.
140. Asteraki, E.J., C.B. Hanks, and R.O. Clements. 1992. The impact of the chemical removal of the hedge-base flora on the community structure of carabid beetles (Col., Carabidae) and spiders (Aransas) of the field and hedge bottom. *J. Appl. Ent.* 113:398-406.
141. Santillo, D.J., D.M. Leslie, and P.W. Brown. 1989. Responses of small mammals and habitat to glyphosate application on clearcuts. *J. Wildl. Manage.* 53(1):164-172. 142. U.S. EPA. Office of Pesticides and Toxic Substances. 1986. Guidance for the reregistration of pesticide products containing glyphosate as the active ingredient. Washington, D.C., June.
143. Mohamed, A.I. et al. 1992. Effects of pesticides on the survival, growth and oxygen consumption of *Hemilepistus reaumuri* (Audouin & Savigny 1826) (Isopoda Oniscidea). *Trop. Zool.* 5:145-153.
144. Folmar, L.C., H.O. Sanders, and A.M. Julin. 1979. Toxicity of the herbicide glyphosate and several of its formulations to fish and aquatic invertebrates. *Arch. Environ. Contam. Toxicol.* 8:269278.
145. Hartman, W.A. and D.B. Martin. 1984. Effect of suspended bentonite clay on the acute toxicity of glyphosate to *Daphnia pulex* and *Lemna minor*. *Bull. Environ. Contam. Toxicol.* 33:355361.
146. Servizi, J.A., R.W. Gordon, and D.W. Martens. 1987. Acute toxicity of Garlon 4 and Roundup herbicides to salmon, *Daphnia*, and trout. *Bull. Environ. Contam. Toxicol.* 39:15-22.
147. Holck, A.R. and C.L. Meek. 1987. Dose-mortality responses of crawfish and mosquitoes to selected pesticides. *J. Am. Mosqu. Contr. Assoc.* 3:407-411.
148. Springett, J.A. and R.A.J. Gray. 1992. Effect of repeated low doses of biocides on the earthworm *Aporrectodea caliginosa* in laboratory culture. *Soil Biol. Biochem.* 24(12):1739-1744.
149. Mitchell, D.G., P.M. Chapman, and T.J. Longs. 1987. Acute toxicity of Roundup and Rodeo herbicides to rainbow trout, chinook, and coho salmon. *Bull. Environ. Contam. Toxicol.* 39:10281035.
150. Wan, M.T., R.G. Watts, and D.J. Moul. 1989. Effects of different dilution water types on the acute toxicity to juvenile Pacific salmonids and rainbow trout of glyphosate and its formulated products. *Bull. Environ. Contam. Toxicol.* 43:378385.

151. Holdway, D.A. and D.G. Dixon. 1988. Acute toxicity of permethrin or glyphosate pulse exposure to larval white sucker (*Catostomus commersoni*) and juvenile flagfish (*Jordanella floridae*) as modified by age and ration level. *Environ. Toxicol. Chem.* 7:63-68.
152. Holtby, L.B. 1989. Changes in the temperature regime of a valley-bottom tributary of Carnation Creek, British Columbia, over-sprayed with the herbicide Roundup (glyphosate). In Reynolds, P.E. (ed.) *Proceedings of the Carnation Creek Herbicide Workshop*. Sault Ste. Marie, Ontario, Canada: Forest Pest Management Institute.
153. Morgan, J.D. et al. 1991. Acute avoidance reactions and behavioral responses of juvenile rainbow trout (*Oncorhynchus mykiss*) to Garlon 4, Garlon 3A and Vision" herbicides. *Environ. Toxicol. Chem.* 10:73-79.
154. Liong, P.C., W.P. Hamzah, and V. Murugan. 1988. Toxicity of some pesticides towards freshwater fishes. *Malaysian Aque. J.* 54(3):147-156.
155. Neskovic, N.K. et al. 1996. Biochemical and histopathological effects of glyphosate on carp, *Cyprinus carpio* L. *Bull. Environ. Toxicol. Chem.* 56:295-302.
156. MacKinnon, D.S. and B. Freedman. 1993. Effects of silvicultural use of the herbicide glyphosate on breeding birds of regenerating clearcuts in Nova Scotia, Canada. *J. Appl. Ecol.* 30(3):395-406.
157. Santillo, D., P. Brown, and D. Leslie. 1989. Responses of songbirds to glyphosate-induced habitat changes on clearcuts. *J. What. Manage.*
158. Eggestad, M. et al. 1988. Glyphosate application in forest-ecological aspects. VIII. The effect on black grouse (*Tetrao tetrix*) summer habitat. *Beard. J. For. Res.* 3:129-135.
159. D'Anieri, P., D.M. Leslie, and M.L. McCormack. 1987. Small mammals in glyphosate-treated clearcuts in northern Maine. *Can. Field-Nat.* 101(4):547-550.
160. Ritchie, C., A.S. Harestad, and R. Archibald. 1987. Glyphosate treatment and deer mice in clearcut and forest. *Northw. Sci.* 6(3):199-202.
161. Sullivan, T. 1990. Demographic responses of small mammal populations to a herbicide application in coastal coniferous forest: population density and resiliency. *Can. J. Zool.* 68:874883.
162. Hjeljord, O. et al. 1988. Glyphosate application in forest-ecological aspects. VII. The effect on mountain hare (*Lepus timidus*) use of a forest plantation. *Beard. J. For. Res.* 3:123-127.
163. Runciman, J.B., and T.P. Sullivan. 1996. Influence of alternative conifer release treatments on habitat structure and small mammal populations in south central British Columbia. *Can. J. For. Res.* 26:2023-2034.
164. Balfour, P.M. 1989. Effects of forest herbicides on some important wildlife forage species. Victoria, British Columbia, Canada: B.C. Ministry of Environment.
165. Locke, D., J.A. Landivar, and D. Moseley. 1995. The effects of rate and timing of glyphosate applications on defoliation efficiency, regrowth inhibition, lint yield, fiber quality and seed quality. *Proc. Beltwide Cotton Conf., National Cotton Council of America*: 1088-1090.
166. Hutchinson, G.L. 1995. Nitrogen cycle interactions with global change processes. In Nierenberg, W.L. (ed.) *Encyclopedia of Environmental Biology*. Volume 2. San Diego: Academic Press. Pp.563-557.
167. Eberbach, P.L. and L.A. Douglas. 1983. Persistence of glyphosate in a sandy loam. *Soil Biol. Biochem.* 15(4):485-487.
168. Eberbach, F.L. and L.A. Douglas. 1989. Herbicide effects on the growth and nodulation potential of *Rhizohium trilolii* with *Trilolium subterraneum* L. *Plant and Soil* 119:15-23.
169. Santos, A. and M. Flores. 1995. Effects of glyphosate on nitrogen fixation of free-living heterotrophic bacteria. *Lett Appl. Microbial.* 20:349352.
170. Moorman, T.B. et al. 1992. Production of hydrobenzoic acids by *Bradyrhizohium japonicum* strains after treatment with glyphosate. *J. Agric. Food Chem.* 40:289-293.
171. Mårtensson, A.M. 1992. Effects of agrochemicals and heavy metals on fast-growing *Rhizohia* and their symbiosis with small-seeded legumes. *Soil Biol. Biochem.* 24(5):435-445.

172. Tenuta, M. and E.G. Beauchamp. 1995. Denitrification following herbicide application to a grass sward. *Can. J. Soil. Sci.* 76:15-22.
173. Towle, A. 1989. *Modern biology*. Austin, TX: Holt, Rinehart and Winston. p.342.
174. Estok, D., B. Freedman, and D. Boyle. 1989. Effects of the herbicides 2,4-D, glyphosate, hexazinone, and triclopyr on the growth of three species of ectomycorrhizal fungi. *Bull. Environ. Contam. Toxicol.* 42:835-839.
175. Chakravarty, P. and S.S. Sidhu. 1987. Effects of glyphosate, hexazinone and triclopyr on in vitro growth of five species of ectomycorrhizal fungi. *Eur. J. For. Path.* 17:204-210.
176. Bayne, H.F. et al. 1995. Colonization of *Orchis morio* protocorms by a mycorrhizal fungus: effects of nitrogen nutrition and glyphosate in modifying the responses. *Can. J. Bot* 73:1128-1140.
177. Brammall, R.A. and V.J. Higgins. 1988. The effect of glyphosate on resistance of tomato to *Fusarium* crown and root rot disease and on the formation of host structural defensive barriers. *Can. J. Bot* 66:1547-1555.
178. Johal, G.S. and J.E. Rahe. 1988. Glyphosate, hypersensitivity and phytoalexin accumulation in the incompatible bean anthracnose host-parasite interaction. *Physiol. Molec. Plant Pathol.* 32:267-281.
179. Mekwatanakarn, P. and K. Sivassithamparam. 1987. Effect of certain herbicides on soil microbial populations and their influence on saprophytic growth in soil and pathogenicity of take-all fungus. *Biol. Fertil. Soils* 5:175-180.
180. Kawate, M.K. et al. 1997. Effect of glyphosate-treated henbit (*Lamium amplexicaule*) and downy brome (*Bromus tectorum*) on *Fusarium solani* f. sp. pisi and *Pythium ultimum*. *Weed Sci.* 45:739743.
181. Bergvinson, D.J. and J.H. Borden. 1992. Enhanced colonization by the blue stain fungus *Ophiostoma claverum* in glyphosate-treated sapwood of lodgepole pine. *Can J. For. Res.* 22:206-209.
182. Gressel, J. 1996. Fewer constraints than proclaimed to the evolution of glyphosate-resistant weeds. *Resist. Pest Manage.* 8:2-5.
183. Sindel, B. 1996. Glyphosate resistance discovered in annual ryegrass. *Resist. Pest Manage.* 8:5-6.

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## Overview of the toxic effects of 2,4-D

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### Introduction

2,4-D (2,4-dichlorophenoxyacetic acid) is a common herbicide used around the home and garden,

on golf courses, ball fields, parks, in agriculture and forestry. Agricultural uses include pasture land, wheat, corn, soybeans, barley, rice, oats, and sugar cane. In Canada, there are currently 205 registered products containing 2,4-D.<sup>1</sup>

Despite industry efforts claiming the safety of this chemical, there is a large body of evidence

indicating major health effects, from cancer to immunosuppression, reproductive damage to neurotoxicity. Environmental contamination, particularly in wetlands has also been demonstrated,

in direction infringement of the *Fisheries Act* R.S., c. F-14, s. 36.

This paper aims to provide an overview of the scientific body of evidence demonstrating the toxic effects of 2,4-D.

### **Health Effects**

In mammals, 2,4-D disrupts energy production (Zychlinski & Zolnierowicz, 1990), depleting the body of its primary energy molecule, ATP (adenosine triphosphate) (Palmiera et al., 1994). 2,4-D has been shown to cause cellular mutations which can lead to cancer. This mutagen contains dioxins, a group of chemicals known to be hazardous to human health and to the environment (Littorin, 1994).

Numerous epidemiological studies have linked 2,4-D to non-Hodgkin's lymphoma (NHL) among farmers (Zahm, 1997; Fontana et al, 1998, Zahm & Blair, 1992, Morrison et al. 1992). Multi-center studies in Canada and in Sweden of members of the general public found a 30-50% higher odds of 2,4-D exposure among people with NHL (McDuffie et al. 2001, Hardell & Eriksson, 1999, Sterling & Arundel, 1986).

The teratogenic, neurotoxic, immunosuppressive, cytotoxic and hepatotoxic effects of 2,4-D have been well documented (Blakely et al., 1989; Sulik et al, 1998; Barnekow et al., 2000; Rosso et al.,

2000; Venkov et al., 2000; Charles et al., 2001; Madrigal- Bujadar et al., 2001; Osaki et al., 2001;

Tuschl & Schwab, 2003).

Other researchers publishing in the open scientific literature have reported oxidant effects of 2,4-D, indicating the potential for cytotoxicity or genotoxicity. For example, Bukowska (2003) reported that treatment of human erythrocytes in vitro with 2,4-D at 250 and 500 ppm resulted in decreased levels of reduced glutathione, decreased activity of superoxide dismutase, and increased levels of glutathione peroxidase.<sup>56</sup> These significant changes in antioxidant enzyme activities and evidence of oxidative stress indicate that 2,4-D should be taken seriously as a cytotoxic and potentially genotoxic agent.

<sup>1</sup> PMRA, Electronic Labels: Search and Evaluation (ELSE). <http://eddenet.pmra-arla.gc.ca/4.0/4.01.asp>. Accessed January 13, 2005.

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2,4-D causes significant suppression of thyroid hormone levels in ewes dosed with this chemical (Rawlings et al., 1998). Similar findings have been reported in rodents, with suppression of thyroid hormone levels, increases in thyroid gland weight, and decreases in weight of the ovaries and testes (Charles et al., 1996). The increases in thyroid gland weight are consistent with the suppression of thyroid hormones, since the gland generally hypertrophies in an attempt to compensate for insufficient circulating levels of thyroid hormones. Thyroid hormone is known to play a critical role in the development of the brain. Slight thyroid suppression has been shown to adversely affect neurological development in the fetus, resulting in lasting effects on child learning and behavior (Haddow et al., 1999).

2,4-D causes slight decreases in testosterone release and significant increases in estrogen release from testicular cells (Liu et al., 1996). In rodents, this chemical also increases levels of the hormones progesterone and prolactin, and causes abnormalities in the estrus cycle (Duffard et al., 1995). Male farm sprayers exposed to 2,4-D had lower sperm counts and more spermatic

abnormalities compared to men who were not exposed to this chemical (Lerda & Rizzi, 1991). In Minnesota, higher rates of birth defects have been observed in areas of the state with the highest use of 2,4-D and other herbicides of the same class. This increase in birth defects was most pronounced among infants who were conceived in the spring, the time of greatest herbicide use (Garry et al, 1996).

2,4-D also interferes with the neurotransmitters serotonin and dopamine. In young organisms, exposure to 2,4-D results in delays in brain development and abnormal behavior patterns, including apathy, decreased social interactions, repetitive movements, tremor, and immobility (Evangelista de Duffard et al, 1995). Females are more severely affected than males. Rodent studies have revealed a region-specific neurotoxic effect on the basal ganglia of the brain, resulting in an array of effects on critical neurotransmitters and adverse effects on behavior (Bortolozzi et al., 2001).

A peer-reviewed, developmental neurotoxicity study demonstrated severe neurotoxicity in young rats exposed to 2,4-D from postnatal days 12 to 25 at doses of 70 mg/kg/day. These pups showed decreases in GM1 level, diminution in myelin deposition and alterations in all behavioral tests at all doses (Rosso et al, 2000). This herbicide specifically appears to impair normal deposition of myelin in the developing brain (Duffard et al., 1996). The neurotoxic and anti thyroid effects of 2,4-D make it highly likely that fetuses, infants, and children will be more susceptible to longterm

adverse health effects from exposure to this chemical although they may appear normal at birth.

Young animals can also be exposed to 2,4-D through maternal milk. Recent research has revealed that 2,4-D is excreted in breast milk, thereby resulting in potentially significant exposures to the nursling. The researchers detected 2,4-D residues in stomach content, blood, brain and kidney of 4-day-old neonates fed by 2,4-D exposed mothers (Sturtz et al., 2000). When maternal exposures stopped, the chemical continued to be excreted in maternal milk for a week. Thus, postnatal exposures to this chemical during the critical period for development of the infant brain are of serious scientific concern.

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#### *Agricultural Workers*

Workers applying chlorinated phenoxy herbicides frequently have nervous system disorders, are exposed to a higher risk of soft tissue sarcoma, and show symptoms of hormonal and internal organ irregularities. (Kogevinas, 1995; National Research Council of Canada, 1983). A study of farmers in Alberta, Saskatchewan and Manitoba linked use of 2,4-D to an increased incidence of prostate cancer (Morrison et al, 1993). An Italian study by Miligi et al (2003) showed that an associated between NHL and 2,4-D in women. Hardell & Eriksson (1999) also demonstrated the link between exposure of 2,4-D and NHL. Their research identified a latency period between exposure and diagnosis of NHL, which could be a reason why there is conflicting research on the issue.

The risk to farm workers is pronounced because of the use of sunscreen. Agricultural workers are encouraged to wear sunscreen to protect their skin from UV-related skin cancer. However, studies have shown that the use of sunscreen increases the rates of penetration of 2,4-D. This has also been shown for the insect repellent DEET (Windheuser et al, 1982). One study demonstrated 14% palmar absorption of 2,4-D after skin application of DEET (Moody et al., 1992). Studies have also shown that most commercial sunscreen formulations enhance the penetration of 2,4-D through hairless mouse skin. One such study found that sunscreens increase

penetration of 2,4-D by over 60 percent, from an average penetration of 54.9% to 86.9% (Pont et al., 2004). Another study found more than a doubling in absorption from an average penetration of 39.1% for the no sunscreen control to 81.0% for mice pre-treated with Neutrogena Oil Free Sunscreen (Brand et al., 2002). These results in the mouse appear also to be relevant to humans (Pont et al., 2004). In addition to penetration enhancement due to commonly-applied topical products, one study in rodents has demonstrated a 2.2- fold enhancement in dermal absorption after regular ethanol consumption over a 6 to 8 week period (Brand et al., 2004). This scenario was not examined by the USEPA in its evaluation of 2,4-D. However, it is a reality of agricultural workers and must be examined. It is important to note that the reevaluation document produced by the USEPA also did not use any form of occlusion over the applied 2,4-D.

Therefore the effect of 2,4-D that soaks clothing, or is subsequently covered by clothing or gloves would not be adequately assessed. Existing research on other chemicals indicates that occlusion is known to significantly enhance skin absorption of dermally-applied materials (Riviere et al., 2003). As much of the reevaluation process is harmonized between countries, this could be the case in the PMRA's evaluation as well, and it is crucial that these issues are not overlooked.

Children living in agricultural communities are heavily exposed to pesticides, whether or not they work in the fields (Lu et al., 2000; Fenske, 1997). Farm children come in contact with pesticides through residues from their parents' clothing, dust tracked into their homes, contaminated soil in areas where they play, food eaten directly from the fields, drift from aerial spraying, contaminated well water, and breast milk. Furthermore, farm children often accompany their parents to work in the fields, raising their pesticide exposures even higher.

#### *Household Use*

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Perhaps the most documented effect of household use of 2,4-D is its association of exposure with cancer in canines. Particularly the study by Hayes et al (1991) has implicated 2,4-D with an odds ratio of 1.32 linking malignant lymphoma and 2,4-D exposure. This study was reviewed by a number of industry sponsored initiatives, and the authors of the original study released their response in 1995 which demonstrated that their scientific methods were sound and that the study had indeed demonstrated this increased risk of malignant lymphoma.

These risks are elevated when one discovers that homeowners using 2,4-D are likely to track the pesticide into their home where it is expected to persist for up to one year (Nishioka et al. 1999). This persistence is seen after a single turf application at a concentration of approximately 0.5µg/g (Nishioka, 1996).

Surrounding and in the home is also where most exposure to children will occur. The levels of exposure to small children are pronounced for dermal exposure and have not been studied for dermal penetration of 2,4-D. We do know that the skin surface area of an infant per unit of body weight is double that of an adult and that all studies which have investigated dermal exposures to pesticides in children have found that this is a major route of exposure. Also, hands moist with saliva collect about 100 times more pesticide residue than dry hands, and children's hands are much more likely to be moist. A study of rats perinatal exposure of 2,4-D did not express effects of exposure until adulthood (Garcia et al., 2001). This demonstrates the insidious nature of the compound, and the enormous threat it poses to children.

Given that the "PMRA considers the unique biological characteristics and exposure patterns of children in its risk assessments," we trust that the studies such as those by Nishioka et al. (1996,

1999) and that of Lu et al., (2000) and Fenske (2003) will be included in the assessment process.

### **Environmental Effects**

2,4-D is a moderately persistent chemical with a half-life between 20 and 200 days.

Unfortunately, the herbicide does not affect target weeds alone. It can cause low growth rates, reproductive problems, changes in appearance or behaviour, or death in non-target species.

Due to the widespread use of 2,4-D on agricultural land, the environmental effects of this use are emerging in scientific studies. Donald et al. (1999) found agricultural pesticides in wetlands, and 2,4-D was the most commonly detected pesticide. Although its concentrations in wetlands exceeded the guidelines in less than 1% of the wetlands, these guidelines are created in isolation, not accounting for the synergistic effects of pesticides. For example, Forsyth et al. (1997) found synergistic effects of picloram and 2,4-D on macrophytes. The chemical will also be carried by run-off into the local river systems. This has been demonstrated here in Ottawa, where a city report on pesticide monitoring of local tributaries showed that 60% of all samples contaminated with phenoxy herbicides. Due to the numerous acceptable uses of 2,4-D, it is likely that the majority of watersheds in rural and urban Canada are contaminated.

### *Wildlife*

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2,4-D has been shown to have negative impacts on a number of groups of animals. In birds, 2,4-D exposure reduced hatching success and caused birth defects (Duffard et al., 1981). It is also indirectly affects birds by destroying their habitat and food source. The toxicity of 2,4-D to fish is variable, with the ester form of 2,4-D expressing greater toxicity than other forms. 2,4-D has also been demonstrated to bio-accumulate in fish (Wang et al., 1994). A product of the breakdown process of 2,4-D is 2,4-dichlorophenol. This chemical is extremely toxic to earthworms, 15 times more toxic than 2,4-D itself (Roberts & Dorough, 1984). Beneficial insects have reduced fecundity when exposed to 2,4-D.

The use of 2,4-D has had drastic affects for both agricultural and wildlife animals including, the deaths of cattle and horses grazing of treated plants, and the destruction of plant food sources of moose, gopher and voles.

### **Conclusion**

Given the effects outlined above, Sierra Club of Canada insists that use of this chemical discontinued. Perhaps the most promising outcoming of this proposed action would be a decline in cancer, which we have seen in Sweden after the banning of phenoxy herbicides (Hardell and Eriksson, 2003). Cancer prevention could start with this step.

### **Bibliography**

Barnekow DE, AW Hamburg, V Puvanesarajah, M Guo. Metabolism o 2,4-dichlorophenoxyacetic acid in laying hens and lactating goats. *Journal of Agricultural and Food Chemistry*. **2000**, 49(1):156-163.

Blakley PM, JS Kim, GD Firneisz. Effects of preconceptional and gestational exposure to Tordon 202c on fetal growth and development of CD-1 mice. *Teratology*, **1989**, 39:547-553.

Bortolozzi A, AM Evangelista de Duffard, F Dajas, R Duffard, R Silveira. Intracerebral administration of 2,4-dichlorophenoxyacetic acid induces behavioral and neurochemical alterations in the rat brain. *Neurotoxicology*, **2001**, 22(2):221-32.

Brand RM, AR Charron, L Dutton, TL Gavlik, et al. Effects of chronic alcohol consumption on dermal penetration of pesticides in rats. *J Toxicol Environ Health A*, **2004**, 67(2):153-61.

Brand RM, Spalding M, Mueller C. Sunscreens can increase dermal penetration of 2,4-dichlorophenoxyacetic acid. *J Toxicol Clin Toxicol*, **2002**, 40(7):827-32.

Bukowska B. Effects of 2,4-D and its metabolite 2,4-dichlorophenol on antioxidant enzymes and level of glutathione in human erythrocytes. *Comp Biochem Physiol C Toxicol Pharmacol*, **2003**, 135(4):435-41.

Charles JM, TR Hanley Jr., TR Wilson, B van Ravenzwaay, JS Bus. Developmental toxicity studies in rats and rabbits on 2,4-dichlorophenoxyacetic acid and its forms. *Toxicological Sciences*, **2001**, 60(1):121-131.

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Charles JM, HC Cunney, RD Wilson, JS Bus. Comparative subchronic studies on 2,4-dichlorophenoxyacetic acid, amine, and ester in rats. *Fundamental & Applied Toxicol*, **1996**, 33:161-165.

Donald DB, J Syrgiannis, F Hunter, G Weiss. Agricultural pesticides threaten the ecological integrity of northern prairie wetlands. *Sci Total Environ*. **1999**, Jul 1;231(2-3):173-81.

Duffard R, G Garcia, S Rosso, A Bortolozzi, M Madariaga, O di Paolo, AM Evangelista de Duffard. Central nervous system myelin deficit in rats exposed to 2,4-dichlorophenoxyacetic acid throughout lactation. *Neurotoxicol Teratol*, **1996**, 18(6):691-696.

Fenske RA. Pesticide exposure assessment of workers and their families. *Occup Med*, **1997**, 12:221-37.

de Duffard AME, A Bortolozzi, RO Duffard. Altered behavioral responses in 2,4-dichlorophenoxyacetic acid treated and amphetamine challenged rats. *Neurotoxicology*, **1995**, 16(3):479-488.

Fontana A, Picoco C, Masala G, Prastaro C, Vineis P. Incidence rates of lymphomas and environmental measurements of phenoxy herbicides: ecological analysis and case-control study. *Arch Environ Health*, **1998**, 53:384-7.

Forsyth DJ, PA Martin, GG Shaw. Effects of herbicides on two submersed aquatic macrophytes, *Potamogeton pectinatus* L. and *Myriophyllum sibiricum* Komarov, in a prairie wetland. *Environmental Pollution*, **1997**, 90:259-268.

Garcia G, P Tagliaferro, A Bortolozzi, MJ Madariaga, A Brusco, AME de Duffard, R Duffard, JP Saavedra. Morphological study of 5-HT neurons and astroglial cells on brain of adult rats perinatal or chronically exposed to 2,4-dichlorophenoxyacetic acid. *Neurotoxicology*, **2001**, 22:733-741.

Garry VF, D Schreinemachers, ME Harkins, et al. Pesticide applicators, bio cides, and birth defects in rural Minnesota. *Environ Hlth Perspect*, **1996**, 104:394-399.

Haddow JE, GE Palomaki, WC Allan, JR Williams, GJ Knight, J Gagnon, CE O'Heir, ML Mitchell, RJ Hermos, SE Waisbren, JD Faix, RZ Klein. Maternal thyroid deficiency during pregnancy and subsequent neuropsychological development of the child. *New Eng J Med*, **1999**, 341(8):549-555.

Hardell L, Eriksson M. A case-control study of non-Hodgkin lymphoma and exposure to pesticides. *Cancer*, **1999**, 85: 1353-60.

Hardell L, Eriksson M. Is the decline of the increasing incidence of non-hodgkins lymphoma in Sweden and other countries a result of cancer preventive measures? *Environ Health Perspect*, **2003**, 111(14):1704-6.

*Sierra Club of Canada Overview of the toxic effects of 2,4-D*  
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Hayes HM, RE Tarone and KP Cantor. On the Association between Canine Malignant Lymphoma and Opportunity for Exposure to 2,4-Dichlorophenoxyacetic Acid. *Environmental*

Research, **1995**, 70(2):119-125.

Hayes HM, RE Tarone, KP Cantor, CR Jessen, DM McCurrin, and RC Richardson. Casecontrol

study of canine malignant lymphoma: Positive association with dog owner's use of 2,4-dichlorophenoxyacetic acid herbicide. *J. Natl. Cancer. Inst.* **1991**, 83:1226-1231.

Kogevinas, M. Soft Tissue Sarcoma and non-Hodgkins Lymphoma in Workers exposed to phenoxy-herbicides, chlorophenols, and dioxins – 2 nested case studies. *Epidemiology*, **1995**, 6(4):396-402.

Lerda D, R Rizzi. Study of reproductive function in persons occupationally exposed to 2,4-D. *Mutation Research*, **1991**, 262:47-50.

Littorin, M “Dioxins in Blood from Swedish Phenoxy Herbicide Workers.” In *Lancet* Vol.344 (8922), August 27, **1994**, pp.611-612.

Liu RC, C Hahn, ME Hurtt. The direct effect of hepatic peroxisome proliferators on rat leydig cell function in vitro. *Fundamental & Applied Toxicol*, **1996**, 30:102-108.

Duffard R, Bortolozzi A, Ferri A, Garcia G, Evangelista de Duffard AM. Developmental neurotoxicity of the herbicide 2,4-dichlorophenoxyacetic acid. *Neurotoxicology*, **1995**, 16(4):764.

Lu C, RA Fenske, NJ Simcox, D Kalman. Pesticide exposure of children in an agricultural community: evidence of household proximity to farmland and take home exposure pathways. *Environ Res*, **2000**, 84:290-302.

Madrigal-Bujaidar E, Hernandez-Ceruelos A, Chamorro G. Induction of sister chromatid exchanges by 2,4-dichlorophenoxyacetic acid in somatic and germ cells of mice exposed in vivo. *Food Chem Toxicol*, **2001**, 39(9): 941-6.

McDuffie HH, Pahwa P, McLaughlin JR, et al. Non-Hodgkin's lymphoma and specific pesticide exposures in men: cross-Canada study of pesticides and health. *Cancer Epidemiol Biomarkers Prev.*, **2001**, 10(11): 1155-63.

Miligi L, Adele Seniori Costantini, Vanessa Bolejack, Angela Veraldi, Alessandra Benvenuti, Oriana Nanni, Valerio Ramazzotti, Rosario Tumino, Emanuele Stagnaro, Stefania Rodella, Arabella Fontana, Carla Vindigni, Paolo Vineis. Non-Hodgkin's lymphoma, leukemia, and exposures in agriculture: Results from the Italian multicenter case-control study. *Am J Ind Med.* **2003** 44(6):627-636.

Moody RP, RC Wester, JL Melendres, HI Maibach. Dermal absorption of the phenoxy herbicide 2,4-D dimethylamine in humans: effect of DEET and anatomic site. *J Toxicol Environ Health*, **1992**, 36(3):241-50.

*Sierra Club of Canada Overview of the toxic effects of 2,4-D*  
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Morrison HI, Wilkins K, Semenciw R, Mao Y, Wigle D. Herbicides and cancer. *J Natl Cancer Inst*, **1992**, 84:1866-74.

Morrison, H. et al. Farming and Prostate Cancer Mortality. *American Journal of Epidemiology*, **1993**, 137(30):270-280.

National Research Council of Canada. Associate committee on Scientific Criteria for Environmental Quality; Subcommittee on Pesticides and Industrial Organic Chemicals. “2,4-D Some Current Issues” NRCC No. 20647, **1983**, Pp. 29-55.

Nishioka MG, Burkholder HM, Brinkman MC, Gordon SM. Measuring lawn transport of lawn-applied

herbicide acids from turf to home: Correlation of dislodgeable 2,4-D turf residues with carpet dust and carpet surface residues. *Environmental Science and Technology*, **1996**, 30: 3313-

3320.

Nishioka, M.G. et al. Distribution of 2,4-dichlorophenoxy acetic acid in floor dust throughout homes following homeowner and commercial lawn applications: Quantitative effects of children, pets and shoes. *Environ. Sci. Technol.*, **1999**, 33:1359-1365.

Osaki K, JF Mahler, JK Hasemann, CR Moomaw, ML Nicolette, A Nyska. Unique renal tubule changes induced in rats and mice by the peroxisome proliferators 2,4-dichlorophenoxyacetic acid (2,4-D) and WY-1643. *Toxicologic Pathology*, **2001**, 29(4):440-450.

Palmeira, C.M, A.J Moreno and V.M.C. Madeira. Interactions of herbicides 2,4-D and dinoseb with liver mitochondrial bioenergetics. *Toxicol. Appl. Pharmacol.*, **1994**, 127:50-57.

Pont AR, Anna R. Charron and Rhonda M. Brand. Active ingredients in sunscreens act as topical penetration enhancers for the herbicide 2,4-dichlorophenoxyacetic acid. *Toxicology and Applied Pharmacology*, **2004**, 195(3):348-354.

Rawlings NC, SJ Cook, D Waldbillig. Effects of the pesticides carbofuran, chlorpyrifos, dimethoate, lindane, triallate, trifluralin, 2,4-D, and pentachlorophenol on the metabolic endocrine and reproductive endocrine system in ewes. *J Toxicol Environ Hlth*, **1998**, 54:21-36.

Riviere JE, Baynes RE, Brooks JD, Yeatts JL, Monteiro-Riviere NA. Percutaneous absorption of topical N,N-diethyl-m-toluamide (DEET): effects of exposure variables and coadministered toxicants. *J Toxicol Environ Health A*, **2003**, 66(2):133-51.

Roberts BL, HW Dorough. Relative toxicity of chemicals to the earthworm. *Environ Toxic Chem*, **1984**, 3:67-78.

Rosso SB, AO Caceres, AM de Duffard, RO de Duffard, S Quiroga. 2,4-dichlorophenoxyacetic acid disrupts the cytoskeleton and disorganizes the Golgi apparatus of cultured neurons. *Toxicological Sciences*, **2000**, 56(1):133-140.

*Sierra Club of Canada Overview of the toxic effects of 2,4-D*  
January 2005 9

Rosso SB, GB Garcia, MJ Madariaga, AM Evangelista de Duffard, RO Duffard. 2,4-Dichlorophenoxyacetic acid in developing rats alters behaviour, myelination and regions brain gangliosides pattern. *Neurotoxicology*, **2000**, 21(1-2):155-63.

Sterlineg TD, AV Arundel. Health effects of phenoxy herbicides – A review. *Scand J Work Environ Health*, **1986**, 12:161-173.

Sturtz N, AM Evangelista de Duffard, R Duffard. Detection of 2,4-dichlorophenoxyacetic acid (2,4-D) residues in neonates breast-fed by 2,4-D exposed dams. *Neurotoxicology*, **2000**, 21(1-2):147-54.

Sulik M, W Kisilewski, B Szyaka, A Kemonia, M Sulkowska, M Baltziak. Morphological change in mitochondria and lysosome of hepatocytes in acute intoxication with 2,4-dichlorophenoxyacetic acid (2,4-D). *Materia Medica Polona*, **1998**, 30(1-2):16-19.

Tuschl H, C Schwab. Cytotoxic effects of the herbicide 2,4-dichlorophenoxyacetic acid in HepG2 cells. *Food Chem Toxicol*, **2003**, 41:385-393.

Venkov P, M Topashka-Ancheva, M Georgieva, V Alexieva, E Karanov. Genotoxic effect of substituted phenoxyacetic acids. *Arch Toxicol*, **2000**, 74:560-6.

Wang Y, C Jaw, Y Chen. Accumulation of 2,4-D and glyphosate in fish and water. *Water Air Soil Poll*, **1994**, 74:397-403.

Weisenburger, DD. Epidemiology of non-Hodgkin's lymphoma: recent findings regarding an emerging epidemic. *Ann. Oncol.*, **1994**, 5:19-23.

Windheuser JJ, JL Haslam, L Caldwell, and RD Shaffer. The use of N,N-diethyl-m-toluamide to enhance dermal and transdermal delivery of drugs. *J. Pharm. Sci.*, **1982**, 71:1211-1213.

Zahm SH. Mortality study of pesticide applicators and other employees of a lawn care service

company. J Occup Environ Medicine, **1997**, 39:1055-67.

Zahm SH, Blair A. Pesticides and non-Hodgkin's lymphoma. Cancer Res, **1992**, 52: 5485s-5488s.

Zeljezic D, V Garaj-Vrhovac. Chromosomal aberrations, micronuclei and nuclear buds induced in human lymphocytes by 2,4-dichlorophenoxyacetic acid pesticide formulation. Toxicology, **2004**, 200:39-47.

Zychlinkski, L. and S. Zolnierowicz. Comparison of uncoupling activities of chlorophenoxy herbicides in rat liver mitochondria. Toxicol. Lett., **1990**, 52:25-34.

Battle Creek Alliance and Sierra Club hope you will carefully consider the information presented here and not continue to condone practices that degrade and destroy watersheds by granting waivers to industrial timber harvest. If you do continue to grant waivers, we would like an explanation of why these companies are above the rules, regulations and laws that pertain to others?

Sincerely,

Marily Woodhouse, for Battle Creek Alliance and Mother Lode Chapter, Sierra Club  
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