Glyphosate-Based Aquatic Herbicides
An Overview of Risk

by Susan Monheit, CDFA-IPC

[Editor's note: The following article is the result of a thorough literature review and multiple consultations with pesticide chemists and eco-toxicologists. It does not constitute an endorsement for any commercial product. The Noxious Times staff is aware that many of the issues around the safety of Glyphosate-based herbicides concern surfactants rather than the active ingredient. A similar review is being conducted on surfactants and will be presented in a future Noxious Times. Persons wishing to obtain a electronic copy of this article may email loosestrife@cdfa.ca.gov.]

Introduction

Public awareness of the potential adverse effects of pesticide use has been growing over the past several decades. Environmentalists now raise concerns when pesticides are used, and demand proactive assessments of the potential impacts of pest management activities. What has not been widely recognized by many environmentalists is the magnitude of difference between highly toxic, persistent, and lipophillic (fat soluble) organochlorine insecticides such as DDT, and the relatively low toxicity, quick degradability, and lipophobic (fat insoluble) nature of glyphosate-based herbicides such as Aquamasterâ (Monsanto Company, St. Louis MO), Rodeoâ (Dow Agrosciences, Indianapolis, IN), and Roundup â (Monsanto Company, St. Louis MO).

Integrated pest management (IPM) is a multi-disciplinary approach to tackling the problem of invasive species. In integrated weed management programs, effective tools to control the spread of noxious weeds may include mechanical, chemical and biological control techniques. Each method has its strength, and when used together or in succession, can increase the effectiveness of a program substantially. The use of herbicides to control noxious weeds is an integral part of this process. Efficacy and potential toxicity to non-target organisms are the two factors that most often the guiding herbicide selection. When used with discretion, and a carefully selected surfactant, glyphosate-based herbicides can offer a highly effective option, with relatively little adverse effect.

In this paper, current risk assessments, bioassay toxicity tests, and other studies performed on glyphosate or glyphosate-based herbicides, are reviewed. In a follow-up article, surfactant toxicity will be addressed. Together, these articles are intended to establish a basis for confidence in the use of glyphosate-based herbicides as an environmentally sound technique for the management of noxious invasive aquatic plant species.

Risk values and toxicity data for the herbicide Roundupâ (labeled only for terrestrial uses) are included because most available literature on glyphosate-based herbicides examined Roundupâ formulations rather than the aquatic labeled products Rodeoâ or Aquamasterâ. The formulations of Aquamaster® and Rodeo® differ from Roundup® herbicide in that they have a higher concentration of the active ingredient, glyphosate, but contain no surfactant.

How Glyphosate-Based Herbicides Work

There are many formulations of glyphosate-based herbicides, many of which have the same basic ingredients: isopropyl amine (IPA) salt of glyphosate, water and some type of surfactant (specific surfactants are chosen by the herbicide products' manufacturer, or are added after market by the consumer). The exact formulation of Aquamaster®/Rodeoâ is isopropylamine (IPA) salt of glyphosate (53.5%), water (46.5%). A surfactant must be added to these products before application to enable glyphosate to penetrate cuticular waxes and allow uptake by plants. The formulation of Roundupâ is isopropylamine (IPA) salt of glyphosate (480 g/L), water and ethoxylated tallow amine surfactant, POEA.

Aquamasterâ/Rodeoâ herbicide, once mixed with a surfactant, is generally applied by direct spray to foliage. Glyphosate is assimilated by leaves and other green plant tissue, and is then rapidly translocated within the phloem throughout the entire plant including its roots. Glyphosate's mode of action is to prevent a plant from producing the essential amino acids tryptophan, tyrosine, and phenylalanine, which reduces the production of protein within the plant, thereby inhibiting plant growth (Herbicide Handbook, 1994; Glyphosate Pesticide Fact Sheet, USDA; Williams et al 2000). The biochemical (protein production) pathway by which glyphosate acts on plants is not found in animals. This helps explains the low risk to animal species from labeled uses of glyphosate.

Glyphosate

Glyphosate-based herbicides are among the most widely used broad-spectrum herbicides in the world because they are highly efficacious, cost effective, practically non-toxic, and degrade readily in the environment. Glyphosate is soluble in water, and tends to bind tightly to sediment, suspended particulates, organic matter and soil, becoming essentially unavailable to plants or other aquatic organisms. Glyphosate does not bioaccumulate, in terrestrial or aquatic animals (Giesy et al. 2000; Williams et al 2000). Herbicidal effects are therefore limited to foliar contact, cut stump or stem injected applications on plants. Glyphosate rapidly dissipates from surface waters, and soil microflora quickly biodegrade glyphosate into AMPA and CO_2 (Gardner & Grue 1996). AMPA also undergoes rapid degradation to CO_2 in soil (Rueppel et al 1977).

Formulations of glyphosate including Rodeoâ, Roundupâ, and Aquamasterâ have been extensively investigated for their potential to produce adverse effects in non-target organisms. Governmental regulatory agencies, international organizations, and others have reviewed and assessed the available scientific data for glyphosate formulations and independently judged them to be of minimal risk to the environment (Agriculture Canada 1991; USEPA 1993; WHO 1994).

Since glyphosate's development in the 1970's, there have been no documented cases of adverse effects on fish or aquatic invertebrates associated its use for the control of aquatic weeds (Giesy et al. 2000). Several field studies have investigated effects of aquatic weed control applications on aquatic animals (Solberg and Higgins 1993; Findlay and Jones 1996; Simenstad et al. 1996; Linz et al 1997). No measurable increases in effects on density, abundance, or survival of aquatic invertebrates have been reported from the direct effects of glyphosate in field studies (Haag 1986; Henry et al. 1991; Gardner & Grue 1996; Simenstad et al. 1996; Linz et al. 1999).

Pesticide Registration

"A pesticide product may be sold or distributed in the United States only if it is registered or exempt from registration under the Federal Insecticide, Fungicide and Rodneticide Act (FIFRA). Before a product can be registered unconditionally, it must be shown that it can be used without causing any unreasonable risk to man or the environment." (USEPA 1993c). Periodically, pesticides and their active ingredients undergo re-registration. As part of the re-registration process of pesticide active ingredients, new toxicity studies and risk assessments are reviewed. Glyphosate was re-registered by the EPA in 1993 (USEPA 1993d).

Ecological Risk

Risk is an assessment of the potential for adverse effects that result from some activity. Practically anything however, can be toxic if the dose or level of exposure is high enough. Toxicity alone does not indicate risk. This concept was first elaborated by Paracelsus (1492-1541), who said "What is there that is not poison?" In other words, the dose makes the poison.

To assess the potential effects of a known chemical to wildlife, exposure (average daily dose - ADD) is compared to some conservative reference dose of known toxicity (toxicity reference value -TRV). Together ADD/TRV give a hazard quotient (HQ), which is a measure of risk. If calculated HQ's are less than 1, no adverse effects are expected from the defined exposure. If HQ's are greater than 1, either more site-specific information is needed, or adverse effects are indicated.

Several toxicity values derived from laboratory tests can be used as TRV's. An LC_{50} is a toxicity value that indicates a concentration at which 50% of the test organisms will die (lethal concentration). A no-observable-effects-level (NOEL), and lowest effects level (LEL) are other commonly used TRV's.

Toxicological Effects

Numerous tests to study the toxicity of glyphosate herbicides have been conducted on rodents, dogs, rabbits, birds, fish, aquatic invertebrates and aquatic vegetation. These tests show that glyphosate, while highly toxic to plants, is largely non-toxic to other animals (Williams et al 2000).

In wildlife assessments, aquatic toxicity values are derived from bioassay tests. Avian and mammalian toxicity values are derived from field sampling studies. Toxicity to humans however, is extrapolated from carefully controlled studies using laboratory animals. Little direct toxicity data exist for human exposure to glyphosate. The California Department of Pesticide Regulation (DPR) maintains the few anecdotal or physician reported records that exist for human adverse health effects as a result of exposure to glyphosate-based herbicides.

Aquatic Toxicity: In aquatic bioassays tests, test organisms are exposed to a range of contaminant concentrations over time. When 50% of the test organisms die, the test is stopped, and an LC_{50} (lethal concentration) or LD_{50} (lethal dose) is calculated. The smaller the amount of chemical required to kill 50% of the test organisms, the more toxic the chemical is. A highly toxic compound might have an LC_{50} between 0.1 and 1 mg/kg for aquatic animals, and 50 to 500 mg/kg for birds (See the Table 1). Glyphosate is only slightly toxic to wild birds, and practically non-toxic to fish. LC_{50} values for both mallards and bobwhite quail are greater than 4,500 ppm (Forest Service 1984, Giesy et al. 2000).

Table 1. Toxicity Classification For Aquatic and Avian Species (Giesy et al. 2000)

U.S. EPA Toxicity Classifications[a]	European Toxicity Classification[b] (Aquatic)	Acute aquatic LC_{50} or EC_{50} (mg/L)	Avian dietary LC_{50} (mg/kg)
Practically nontoxic	--	>100	>5000
Slightly toxic	Harmful	>10, ≤ 100	>1000, ≤ 5000
Moderately toxic	Toxic	>1, ≤ 10	>500, ≤ 1000
Highly toxic	Very toxic	≥ 0.1, ≤ 1	>50, ≤ 500
Very highly toxic	Very toxic	≤0.1	< 50

To put these toxicity values in context, it is valuable to compare receptor toxicity response with concentrations of glyphosate found in water and sediment after direct application of formulated herbicide. A review of current literature showed environmental glyphosate concentrations after herbicide application to range between 0.090-1.700 mg/L in ponds, 0.020-1.237 mg/L in streams, 0.162-1.0 mg/L in surface waters, and 0.11-2.82 mg/kg in a variety of sediments (Giesy et al. 2000). Aquatic organisms would need to be exposed to concentrations of glyphosate 100 times greater than that which is present after ordinary (following label instructions) use around streams, and 60 times greater than is present after ordinary use around ponds to show toxic effects. The same holds true for glyphosate-bound sediments – birds would have to consume incredibly large amounts of sediment to reach the level of glyphosate consumption that caused adverse health effects in test animals.

The acute LC_{50} values for fish exposed to Roundup[â] (a glyphosate-based formulation with POEA surfactant), glyphosate IPA salt, the metabolite AMPA, and the surfactant POEA, are listed in Table 2. Comparison of these values clearly shows that the surfactant POEA, and the Roundup formulation (with surfactant included), are more toxic than glyphosate itself. In acute toxicity studies with amphibians, glyphosate was found to be practically nontoxic to slightly toxic, while Roundup[â] was slightly to moderately toxic (Giesy et al. 2000).

Table 2. LC_{50} Values for fish exposed to components of the herbicide Roundup.

Test Compound	**Test Species**	**LC_{50} Values (**mg/L)
Roundup[â]	Rainbow trout	8.2-27 (NOEL is 6.4)
Glyphosate	Rainbow trout	140-240
AMPA	Rainbow trout	520
POEA	Rainbow Trout	0.65-7.4

(Giesy et al. 2000)

Mammalian Toxicological Data: In toxicological studies performed with mammals, the test animal is dosed with a specific amount of material, and observed in a laboratory for a variety of acute and chronic effects. LC_{50} values measure acute effects (death). Chronic evaluations include assessments of overall health, behavior, biochemical/physiological processes, and focused evaluations for reproductive toxicity, teratogenicity, and carcinogenicity. Toxicity evaluation endpoints include survival, growth, reproduction, cancer and teratogenic abnormalities (birth defects).

Glyphosate is poorly absorbed by the digestive tract in mammals, and is excreted essentially unmetabolized (Williams et al 2000; EXTOXNET database, Cornell Univ). This is reflected by the large amount of glyphosate needed to cause acute toxicity (death) in test animals. The acute oral LD_{50} value for glyphosate in rats is greater than 5000 mg/kg (WHO, 1994), and 8300 mg/kg for AMPA (Williams et al 2002: Birch 1973). Other oral LD_{50} values for glyphosate are 1,538 mg/kg to greater than 10,000 mg/kg for mice, rabbits, and goats (Extoxnet database, University of Cornell; National Library of Medicine 1992; Williams et al 2000). Toxicological data for Rodeo[â] specifically include: Oral LD_{50} rat >5000 mg/kg; Dermal LD_{50} rabbit >5000 mg/kg; 4-h Inhalation LC_{50} rat >1.3 mg/L; Skin irritation rabbit, none; Skin sensitivity in guinea pig, none; Eye irritation in rabbit, none (Herbicide Handbook 1994).

The USEPA made the determination in its re-registration eligibility decision (RED), that glyphosate is not carcinogenic to humans based on a large body of data from tests performed with laboratory animals (USEPA 1993a). Subchronic and chronic toxicity studies performed with rats, dogs, mice and rabbits ranging from 21 days to two years, and diet concentrations of 3125 ppm to 50,000 ppm (Williams et al 2000: NPT 1992) revealed few treatment-related changes, and the effects observed were confined to the highest doses tested (US EPA RED, 1993).

New chronic rat and two generation rat reproduction studies were submitted as part of the re-registration process for glyphosate. These studies showed no adverse histological consequences on any reproductive or endocrine tissue from either male or female rats even at exaggerated dosage levels (Williams et al 2000).

The U.S. EPA bases its risk assessment for humans on the lowest NOAEL recorded in the various studies. The NOEL for glyphosate, 175 mg/kg/day, comes from a rabbit teratology study. In human health evaluations for glyphosate, the reference does (RfD) has been set at 1.75 mg/kg/day (175 mg/kg/day NOAEL divided by the uncertainty factor of 100X = 1.75 mg/kg/day).

Neurotoxicity, Immunotoxicity and Endocrine Disruption.

In a recent risk report commissioned by the USFS (SERA 1996) on three commonly used herbicides, the potential for glyphosate to cause neurotoxicity, immunotoxicity and endocrine disruption was evaluated. No evidence was found to support glyphosate as a neurotoxicant, immunotoxicant or endocrine disruptor (SERA 2002). SERA found no evidence that glyphosate is a direct neurotoxicant in humans or other species. Several long-term experimental studies of dogs, mice and rats did not find evidence of neurotoxicity to the brain. Nor was there any evidence of neurological effects found among forest workers who mixed and sprayed Roundupâ in a small clinical investigation of worker exposure.

Glyphosate does not appear to be an immunotoxicant in humans or other animals, based on results from the available studies in humans and experimental studies in rodents. “This conclusion is supported not only by an extensive set of standard mammalian bioassays on toxicity, but also by an *in vivo* assay specifically designed to detect humoral immune response, and an *in vitro* assay specifically designed to detect cell mediated immune response” (SERA 2002).

Three specific tests on the potential effects of glyphosate on the endocrine system were conducted. No effects were reported in any of the tests. “The conclusion that glyphosate is not an endocrine disruptor is reinforced by epidemiological studies that have examined relationships between occupational farm exposures to glyphosate formulations and risk of spontaneous miscarriage, fecundity, sperm quality and serum reproductive hormone concentrations” (SERA 2002). None of these studies have found positive associations between exposure to glyphosate formulations and any reproductive or endocrine outcomes.

Application Studies Using Rodeo®/Aquamaster® and Roundup®

Giesy et al 2000: In the “Ecotoxicological Risk Assessment for Roundupâ Herbicide” (Giesy et al. 2000) Roundupâ, glyphosate, and the surfactant POEA where subjected to current ecological risk assessment methodology to provide an index of environmental safety. “Worst-case” assumptions, and NOELs from the most sensitive test species were used to calculate very conservative HQ’s.

The results of the acute risk assessment for Roundupâ showed minimal risk (HQ’s < 1.0) for all aquatic taxa (microorganisms, aquatic macrophytes, fresh-water invertebrates, fish, and amphibians) in environments 2-meters deep. In shallow water (0.15 meters), acute hazard values approached, or in some instances exceeded, minimal risk levels (HQ’s > 1.0) warranting further investigation. An examination of risk assessment assumptions revealed that herbicide degradation, sorption, and interception by target vegetation of greater than 50% would mitigate the potential for effects in shallow waters (i.e.: bringing the HQ values back below 1.0).

Evaluations of chronic risk looked at the components and metabolites of Roundupâ independently. Chronic risk evaluations indicated minimal risk for all components and metabolites, even in shallow waters. While Roundup is not registered for aquatic use, Giesy et al. (2000) concluded that the use of Roundupâ for aquatic habitat restoration can be safely carried out, but requires consideration of items such as application rate, depth of water and percent vegetation coverage.

Trumbo 2000: In an assessment of the non-target aquatic impacts of the herbicide Rodeoâ and the nonylphenol ethoxylate surfactant R-11â, chemical analysis from two of three sites yielded no herbicidal or surfactant constituents, and no toxic effects. Water chemistry from a third site (a still water pond) showed a statistically significant mortality of 30%, and the presence of surfactant constituents in water analysis. This study will be discussed in greater detail in a subsequent article addressing surfactant toxicity.

Gardner & Grue 1996: Results of a series of laboratory and in-situ bioassay test performed on water from wetlands in Washington State treated with Rodeoâ show that the herbicide did not pose a hazard to aquatic invertebrates or fish. The same study however, showed reduced growth of duckweed 48 hours after exposure to Rodeoâ (Gardner & Grue 1996). Based on their findings, the authors of this study concluded that Rodeoâ might pose a greater hazard to non-target aquatic vegetation than to other aquatic organisms. (The surfactant used was not identified).

Simenstad et al 1996: In another study, benthic invertebrate response to the use of Rodeoâ and X-77 Spreaderâ (NPEO surfactant) to control smooth cordgrass *(Spartina alterniflora)* was undetectable. Neither short-term (28-d) or long-term (119-d), population effects were observed following the use of the herbicide/surfactant mix (Simenstad et al 1996).

Surfactants

To effectively control weeds, a surfactant must be added to Rodeoâ/Aquamaster® before application. This allows the user to select a surfactant that meets the specific needs of the weed control program. Efficacy and potential toxicity to non-target aquatic organisms are the two factors that most often guide surfactant selection.

While there is a plethora of data available on the relatively non-toxic effects of glyphosate, the active herbicidal ingredient in Rodeoâ/Aquamaster®, and Roundup®, there is a dearth of information regarding the toxicity of adjuvants (surfactants) which must be added to activate herbicides designated for aquatic use.

On the whole, available toxicity data for surfactants indicate that they are more toxic than glyphosate. Surfactant toxicity data will be presented in a subsequent Noxious Times article, and the choices available to resource conservation/weed control program managers will be discussed. One thing is clear, there is little information published on this subject, and more studies of surfactant toxicity are needed to fill the data gap.

Secondary Effects

The creation of open water habitat in wetlands through the use of herbicides such as Rodeo®/Aquamaster®, create trade-offs for wildlife populations. Studies have noted an increase in populations of some aquatic invertebrates, and species of birds following treatment of cattail-choked-wetlands with Rodeoâ (Linz et al. 1999; Baltezore, Leitch & Linz 1994). Rails, shorebirds and waterfowl will increase when vegetation is thinned, while numbers of red-winged blackbirds, wrens, upland game, furbearers and deer may decline (Baltezore, Leitch & Linz 1994). In some cases, short-term declines in populations may be anticipated because of changes in habitat (i.e.: temporary diminishment of food sources, and nesting or shelter sites). Therefore, ecological assessment endpoints of any habitat rehabilitation program, needs to reflect the long-term goals of the program.

Summary

A review of key documents and studies assessing the acute and chronic toxicity, neurotoxicity, immunotoxicity, and endocrine disruption risks of glyphosate-based herbicides, indicates that non-target organisms are exposed to minimal risk through the use of these herbicides. The surfactants used in the formulation of glyphosate-based herbicides, or mixed with the aquatic herbicides Aquamasterâ and Rodeoâ before application, are far more acutely toxic than the active ingredient itself. A well-administered management program for the control of noxious weeds can minimize potential exposure and risk to non-target organisms through use of BMPs. Application rate, depth of water, water movement and mixing, and percent vegetation converge are key factors in minimizing unwanted aquatic exposures. Surfactants of low toxicity can be selected to minimize the risk to aquatic organisms when using aquatic herbicides such as Rodeo®/Aquamaster®. Further investigation into toxicity values for a variety of surfactants would enable weed control program managers to make surfactant decisions that would be most protective of the environment.

The ecological risks of glyphosate use for focused, short-term eradication efforts has been shown to be small; especially in comparison to the potential ecological damage caused by noxious and invasive weeds that permanently establish themselves across the landscape. Stopping aggressive noxious weed species from invading new wetland and aquatic areas in California is a high priority. Integrated approaches work best.

Bibliography

Agricultural Canada (1991) Pre-harvests use of glyphosate herbicide. Discussion document. Pesticides Directorate, Ottawa, Ontario.

Brooke L (1993). Acute and chronic toxicity of nonylphenol to ten species of aquatic organisms. EPA 68 C-0034. U.S. Environmental Protection Agency, Duluth MN.

Felsot A. S. (2001). Herbicide Tolerant Genes, Part 4, Withering Wildlife? Agrichemical and Environmental News, Washington State University, Cooperative Extension, Pesticide Information Center Newsletter. Feb. 2001, No. 178 pp 1-8.

Folmar LC, Sanders HO, Julin AM (1979). Toxicity of the herbicide glyphosate and several of its formulations to fish and aquatic invertebrates. Arch Environ Contam Toxicol 8:269-278. (They tested the toxicity of surfactants separately).

Forest Service, 1984. Pesticide Background Statements, Vol. I Herbicides. United States Dept. of Agriculture, Agriculture Handbook No. 633.

Gardner S, Grue C (1996). Effects of Rodeo and Garlon 3A on Nontarget Wetland Species In Central Washington. Environ Toxicol Chem, Bol 15, No. 4 pp 441-451.

Giesy JP, Dobson S, Solomon KR, (2000). Ecotoxicological Risk Assessment for Roundup Herbicide. Rev Env. Contam Toxicol 167:35-120.

Herbicide Handbook, Weed Science Society of America, Seventh Edition (1994).

Linz GM, Bleier WJ, Overland JD, and Homan JH (1999). Response of Invertebrates to Glyphosate-Induced Habitat Alterations In Wetlands. Wetlands, Vol 19, No. 1, March 1999, pp. 220-227.

Mitchell, DG, Chapman PM, Long TJ, (1987). Acute toxicity of Roundup and Rodeo herbicides to rainbow trout, Chinook, and Coho salmon. Bull. Evniron Contam Toxicol. 39:1028-1035. (They tested the toxicity of surfactants separately).

Monsanto and Company, 1981 (also called Schroeder CA, 1981). A Three-generation reproduction study with glyphosate in rats. MRID No. 00093879. Available from EPA.

Monsanto Company, 1985. Toxicology of Glyphosate and Roundup Herbicide, Department of Medicine and Environmental Health, St. Louis, MO.

National Library of Medicine, 1992. Hazardous Substances Databank. TOXNET, Medlars Management Section, Bethesda, MD.

Pesticide Fact Sheet: Glyphosate. Office of Pesticide Programs, U.S. Environmental Protection Agency, Washington, DC. EPA Publication No. 540/FS-88-124, 1986.

Rueppel ML, Brightwell BB, Schaefer J, and Marvel JT, (1977). Metabolism and degradation of glyphosate in soil and water. J. Agric. Food Chem. 25:517-528.

Simenstad C A, Cordell J R, Tear C & L, Weitkamp L A, Paveglio F L, Kilbride K M, Fresh K L, and Grue C E, (1996). Use of Rodeo and X-77 Spreader to Control Smooth Cordgrass (*Spartina alterniflora*) in a Southwestern Washington Estuary: 2. Effects on Benthic Microflora and Invertebrates. Environ Tox & Chem, Vol. 156 No. 6, pp 969-978.

Staples C, Weeks J, Hall J, and Naylor C (1998). Evaluation of aquatic toxicity and bioaccumulation of C8- and C9-alkylphenol ethoxylates. Environ Toxicol Chem 17:2470-2480.

Syracuse Environmental Research Associates, (SERA) TR 01-43-08-04a, Neurotoxicity, Immunotoxicity, and Endocrine Disruption with Specific Commentary on Glyphosate, Triclopyr and Hexazinone: Final Report, (2002). Submitted to the Forest Service.

Trumbo J., (2002). California Dept Fish & Game, Pesticide Investigations Unit, Office of Spill Prevention and Response, Rancho Cordova, CA. An Assessment of the Non-target Aquatic Impacts of the Herbicide Rodeo and the Surfactant R-11 When Used to Control Purple Loosestrife, *Lythurm salicaria.*

USEPA, (1993a). Re-registration Eligibility Decision (RED), Glyphosate. Office of Prevention, Pesticides and Toxic Substances, U.S. Environmental Protection Agency, Washington, DC. 738-F-93-011. (**738-R-93-014)**

USEPA (1993b). A Review of Ecological Assessment Case Studies from a Risk Assessment Perspective. EPA/630/R-92/005.

WHO (1994). Glyphosate. Environmental Health Criteria No. 159. World Health Organization, Geneva.

Williams G.M, Kroes R., and Munro I.C. (2000). Safety Evaluation and Risk Assessment of the Herbicide Roundup and Its Active Ingredient, Glyphosate, for Humans. Regulatory Toxicology and Pharmacology 31, 117-165.