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September 21, 2009

Office of Pesticide Programs
(OPP) Regulatory Public Docket (7502P),
Environmental Protection Agency,
1200 Pennsylvania Ave., NW.,
Washington, DC 20460–0001.

Re: Registration Review; Glyphosate Docket Opened for Review and Comment.
Docket Number: EPA–HQ–OPP–2009–0361

Dear Sir/Madam,

Thank you for the opportunity to comment on the docket for glyphosate. Glyphosate (N-phosphono-methyl glycine), first registered in 1974, has become one of the most popular herbicides in the US market, with use dramatically increasing in recent years. Most commonly formulated as Monsanto's Roundup herbicide, approximately 135 million pounds per year[1] of glyphosate end-use products are used on a variety of agricultural crops (and non-agricultural sites such as lawns, gardens and parks), more than seven times its annual reported usage when its first Reregistration Eligibility Decision (RED) document was published in 1993.[2] During this time of increasing glyphosate popularity, and since the publication of the agency's 1993 RED document, data has been emerging that point to various health and environmental consequences resulting from glyphosate and Roundup use. These include an increased risk of non-Hodgkin's Lymphoma (NHL), genetic damage, neurological impacts, as well as water contamination, impacts on amphibians and increasing weed resistance. As a result of these human and environmental health impacts, Beyond Pesticides and the signatories to this letter strongly urge the agency, as it moves forward with the Registration Review process, to seriously consider the new and emerging science which illustrates that glyphosate and its formulated products pose unreasonable risk to human and environmental health, and as such should not be considered eligible for continued registration.

Human Exposures to Glyphosate Pose Unacceptable Risks
There are hundreds of glyphosate products currently registered with the agency[3] under numerous formulations. The agency considers the active ingredient glyphosate (most commonly used as its three salts—isoproplyamine, sodium and monoammonium salts—

[1] USEPA. 2009. Registration Review—- Preliminary Problem Formulation for the Ecological Risk and Drinking Water Exposure Assessments for Glyphosate and Its Salts. Office of Prevention, Pesticides and Toxic Substances. Washington DC
[2] USEPA. 1993. Reregistration Eligibility Decision (RED) Glyphosate. Office of Prevention, Pesticides and Toxic Substances. Washington DC
[3] USEPA. 2009. Glyphosate—Active Product Registrations. Office of Prevention, Pesticides and Toxic Substances. Washington DC

as well as the technical acid, in registered pesticide products) to be of low acute toxicity (Toxicity Category III). In June 1991, EPA classified glyphosate as a Group E carcinogen—evidence of non-carcinogenicity for humans—based on the lack of convincing evidence of carcinogenicity in adequate studies. Since this decision, a 1999 study found that people exposed to glyphosate are 2.7 times more likely to contract non-Hodgkin Lymphoma (NHL)[4]. In 2002, study of Swedish men showed that glyphosate exposure was *significantly* associated with an increased risk of NHL, and hairy cell leukemia—a rare subtype of NHL. [5] Further, a 2003 review of studies conducted on farmers by researchers at the National Cancer Institute showed that exposure to glyphosate was associated with an increased incidence of NHL.[6] According to the American Cancer Society, non-Hodgkin lymphoma is a cancer that starts in cells called lymphocytes, which are part of the body's immune system.[7]

Researchers evaluated associations between glyphosate exposure and cancer incidence in the Agricultural Health Study (AHS)[8], a cohort study of 57,311 licensed pesticide applicators and found that glyphosate had a suggested association with multiple myeloma, a cancer that starts in plasma cells, a type of white blood cell.[9] This association with multiple myeloma was observed with use of glyphosate and cumulative exposure days of use (a combination of duration and frequency).

There is also a tentative association between ADD/ADHD,[10] increased risks of late abortion,[11] and endocrine disruption[12] with glyphosate use.

Researchers agree that glyphosate deserves further epidemiologic study in light of these data and the widespread use of this chemical. Given that the reported incidence of NHL

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[4] Hardell, L., & Eriksson, M. 1999. A Case-Control Study of Non-Hodgkin Lymphoma and Exposure to Pesticides. *Cancer*, 85(6), 1353–1360.

[5] Hardell L, Eriksson M, & Nordstrom M. 2002. Exposure to pesticides as risk factor for non-Hodgkin's lymphoma and hairy cell leukemia: pooled analysis of two Swedish case-control studies. *Leuk Lymphoma*, 43(5), 1043-1049.

[6] De Roos, et al. 2003. Integrative assessment of multiple pesticides as risk factors for non-Hodgkin's lymphoma among men. *Occup Environ Med*, 60(9).

[7] American Cancer Society. Detailed Guide: Lymphoma, Non-Hodgkin Type:What Is Non-Hodgkin Lymphoma? Cancer Reference Information. Available at http://www.cancer.org/docroot/CRI/content/CRI_2_4_1X_What_Is_Non_Hodgkins_Lymphoma_32.asp

[8] De Roos, A. J. D., Blair, A., Rusiecki, J. A., Hoppin, J. A., Svec, M., Dosemeci, M., Sandler, D. P., & Alavanja, MC .2005. Cancer Incidence among Glyphosate-Exposed Pesticide Applicators in the Agricultural Health Study. *Environmental Health Perspectives*, 113(1), 49-54.

[9] National Cancer Institute. 2008. What You Need to Know About: Multiple Myeloma. Available at http://www.cancer.gov/cancertopics/wyntk/myeloma/page2

[10] Garry, V. F., et al. 2002. Birth defects, season of conception, and sex of children born to pesticide applicators living in the Red River Valley of Minnesota, USA. *Environ Health Perspect*, 110(Suppl 3), 441–449.

[11] Arbuckle, T.E., Z. Lin, and L.S. Mery. 2001. An Exploratory Analysis of the Effect of Pesticide Exposure on the Risk of Spontaneous Abortion in an Ontario Farm Population. *Environmental Health Perspectives* 109:851-857.

[12] Walsh, L. P., McCormick, C., Martin, C., & Stocco, D. M. 2000. Roundup Inhibits Steroidogenesis by Disrupting Steroidogenic Acute Regulatory (StAR) Protein Expression. *Environ Health Perspect*, 108, 769–776.

has increased during the period 1990-2005 (0.4% for men, 1.2% for women)[13] coincides with the increased use of Roundup during that period (glyphosate was the most used active ingredient in agriculture and ranked #2 for non-ag uses in 2001),[14] it is hard to ignore these statistics.

Roundup Formulations Are Toxic, Yet Go Unevaluated

For each of the many Roundup product formulations on the market, people are exposed to not only the active ingredient glyphosate, but the various "inert" ingredients included to make the product a more effective and/or long lasting herbicide. Beyond Pesticides and many of the signatory organizations have long advocated for the full disclosure and evaluation of all chemical ingredients in pesticide products. These ingredients are neither inert nor inactive, and are responsible for serious human health implications.

An increasing number of studies have found that formulated glyphosate products are more toxic than the active ingredient glyphosate alone. One study by Walsh, et al. found that Roundup decreased steroidogenesis, indicating that at least one other component of the formulation is required to disrupt steroidogenesis since it was observed that glyphosate alone did not alter steroid production.[15] In 1998, researchers found that Roundup was able to induce a dose-dependent formation of DNA adducts in the kidneys and liver of mice.[16] The researchers concluded that the Roundup-related DNA adducts were not related to the active ingredient (the isopropylammonium salt of glyphosate) but to another, "unknown" component of the herbicide formulation. Dallegrave et al. (2003 & 2007), in studies with Wistar rats, also found that Roundup induces developmental retardation of the fetal skeleton, a decrease in sperm number, an increase in the percentage of abnormal sperms and a dose-related decrease in the serum testosterone level at puberty.[17] A 2004 study examining glyphosate effects on cell cycle regulation concluded that glyphosate-based pesticides are clearly of human health concern based on results that demonstrated a molecular link between glyphosate-based products and cell cycle dysregulation—a hallmark of tumor cells and human cancers.[18]

A 2008 study confirmed that the adjuvants in Roundup formulations kill human cells, particularly embryonic, placental and umbilical cord cells, even at very low concentrations.[19] These researchers found that Roundup formulations cause total cell death within 24 hrs, through an inhibition of the mitochondrial succinate dehydrogenase activity, and necrosis, by release of cytosolic adenylate kinase measuring membrane

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[13] CDC. Non-Hodgkin Lymphoma Trends. Hematologic (Blood) Cancers. Available at http://www.cdc.gov/cancer/hematologic/lymphoma/statistics/trends.htm

[14] U.S.EPA. 2004. Most Commonly Used Conventional Pesticide Active Ingredients in the U.S. Agricultural Market Sector. Pesticide Industry Sales and Usage: 2000-2001 Pesticide Market Estimates

[15] Ref #12 *Walsh*

[16] Marco, P., Armelle, M., Claudia, B., & Silvio, P. 1998. ^{32}P-postlabeling detection of DNA adducts in mice treated with the herbicide roundup. *Environmental and Molecular Mutagenesis*, 31(1), 55-59.

[17] Dallegrave, E., et al. 2003. The teratogenic potential of the herbicide glyphosate-Roundup® in Wistar rats. *Toxicology Letters*, 142(1-2), 45-52.; Dallegrave, E., et al. 2007. Pre- and postnatal toxicity of the commercial glyphosate formulation in Wistar rats. *Arch Toxicol*, 81(9), 665-673.

[18] Marc, J., Mulner-Lorillon, O., & Bellé, R. (2004). Glyphosate-based pesticides affect cell cycle regulation. *Biology of the Cell*(96), 245-249.

[19] Benachour, N., & Seralini, G.-E. 2008. Glyphosate Formulations Induce Apoptosis and Necrosis in Human Umbilical, Embryonic, and Placental Cells. *Chemical Research in Toxicology*, 22(1), 97-105.

damage. Polyethoxylated tallowamine or POEA—a surfactant used in herbicidal products—was found to be the most potent "inert" and was responsible for the elevated toxic effects. Another study found that the cytotoxicty of Roundup formulations were amplified with time and that exposure affects human reproduction and fetal development.[20] Roundup reduces human placental JEG3 cell viability at least two times more efficiently than glyphosate, disrupts aromatase activity, and mRNA levels.[21]

EPA is aware that certain glyphosate formulations are toxic due especially to inert ingredients. In EPA's 1993 RED document for glyphosate the agency noted that "a toxic inert in glyphosate end use products" was toxic to fish and necessitates labeling requirements.[22] In EPA's recently published registration review summary document for glyphosate, the agency requests toxicity data for POEA "due to uncertainty about its risk to aquatic animals."[23] We are urging the agency to look into human toxicity regarding POEA and other potentially toxic "inert" ingredients in glyphosate formulations based on the above mentioned data and in light of increased residential, occupational and recreational use exposures. EPA tends to perform risk assessment on individual active ingredients and ignore formulations that include other ingredients that can either amplify the toxicity for the active ingredient or that are toxic themselves.

The continued exemption of inert ingredients in the registration process highlights the primary flaw with the agency's regulatory process for both active and inactive ingredients in pesticide products. Rather than adopt a precautionary approach when it comes to chemicals with unknown toxicity, EPA continues to allow chemicals to remain "innocent until proven guilty," and relies on a flawed risk assessment process that does not adequately address exposure and risk. Once proven guilty, these pesticides, both active ingredients and "inerts", have already left a toxic trail in the environment and people's well-being. EPA now has the opportunity and the obligation to address concerns about "inert" ingredients in glyphosate products.

FQPA 10x Factor Must be Reinstated.

EPA, in its 2006 aggregate human health risk assessment for glyphosate, decided that the Food Quality Protection Act (FQPA) safety factor of 10x be removed and reduced to 1x citing that "no evidence of quantitative or qualitative increased susceptibility of the young demonstrated in the prenatal developmental studies in rats and rabbits and pre/post natal reproduction study in rats."[24] This decision is flawed in light of the science. The purpose of the FQPA 10-fold margin of safety is to "to protect infants and children, taking into account the potential for pre- and post-natal toxicity." 21 USC §346a(b)(2)(C). As mentioned above, the data shows that glyphosate and its formulated products

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[20] Benachour N, Sipahutar H, Moslemi S, Gasnier C, Travert C, & Séralini GE. 2007. Time- and dose-dependent effects of roundup on human embryonic and placental cells. *Arch Environ Contam Toxicol.*, 53(1), 126-133.

[21] Richard S, Moslemi S, Sipahutar H, Benachour N, & Seralini GE. 2005. Differential effects of glyphosate and roundup on human placental cells and aromatase. *Environ Health Perspect*, 113(6), 716-720.

[22] Ref#2

[23] U.S.EPA. 2009. Glyphosate Summary Document Reregistration Review: Initial Docket (p10). Office of Prevention, Pesticides and Toxic Substances.

[24] U.S.EPA. 2006. Glyphosate Human Health Risk Assessment for Proposed Use on Indian Mulberry and Amended Use on Pea, Dry. Office of Prevention, Pesticides and Toxic Substances. Washington DC

adversely affect embryonic, placental and umbilical cord cells, and impacts fetal development. Richard, et al. reports that glyphosate is toxic on human placental JEG3 cells within 18 hrs with concentrations lower than those used in agriculture.[25] This effect was found to increase with concentration and time, or in the presence of Roundup adjuvants. Arbuckle, et al. found that preconception exposures to glyphosate moderately increased the risk for spontaneous abortions[26] in mothers exposed to glyphosate products.

Residential uses of glyphosate expose infants and children to variable and high concentrations of active and "inert" ingredients, via drift and direct or indirect contact with treated lawns. A Farm Family exposure study found that all but one of the 79 children evaluated had detectable concentrations of glyphoste in their urine.[27] While most of the active ingredient glyphosate is excreted quickly from the body, "a part may be retained or conjugated with other compounds that can stimulate biochemical and physiological responses."[28] Other studies, including those cited above, name glyphosate as an endocrine disruptor due to its activity on aromatase activity and mRNA levels. The agency still lags behind in finalizing a screening process for endocrine disruption and has still not been able to screen any chemicals for endocrine disruption to date.

As a result of the evidence against glyphosate for endocrine disruption, placental cell damage, and the potential for impaired fetal development, the agency must reinstate the 10X safety factor in accordance with FQPA.

Polyethoxylated Tallowamine (POEA) Surfactant

The agency is specifically requesting data for the surfactant POEA. The agency also proposed two approaches for assessing the hazard posed by POEA: (1) use of structural activity relationships for surfactants, and (2) toxicity testing for a subset of the surfactants. The use of structural activity relationships (SAR) is a useful tool to help determine the potential toxicity of a chemical in the absence of real data. They can be used to make predictions about the physical, chemical and/or biological activity of the chemical being assessed. However, it is by no means to be used as a substitute for available, reliable, measured data, especially in this case, where data is already available. Therefore the agency must utilize toxicity testing and toxicity data, which can be supplemented with SARs, in its assessment of POEA.

As noted above, POEA has been found to be highly toxic. A study by Brausch and Smith found that three formulations of POEA consisting of a 5:1, 10:1, and 15:1 average oxide:tallowamine ratio were all extremely toxic to fairy shrimp (*Thamnocephalus*

[25] Richard, S., Moslemi, S., Sipahutar, H., Benachour, N., & Gilles-Eric Seralini (2005). Differential Effects of Glyphosate and Roundup on Human Placental Cells and Aromatase. *Environmental Health Perspectives*, 113(6), 716-720.

[26] Arbuckle, T. E., Lin, Z., & Mery, L. S. (2001). An Exploratory Analysis of the Effect of Pesticide Exposure on the Risk of Spontaneous Abortion in an Ontario Farm Population. *Environ Health Perspect*, 109, 851–857.

[27] Acquavella, J. F., et al. (2004). Glyphosate Biomonitoring for Farmers and Their Families: Results from the Farm Family Exposure Study. *Environmental Health Perspectives*, 112(3), 321-326

[28] Ref #25 *Richard*

platyurus),[29] and toxic to *Daphnia magna.*[30] POEA toxicity was also found to increase as the tallowamine chain length was reduced. POEA accounted for more than 86% of Roundup toxicity on microalgae and crustaceans in a study conducted by Tsui and Chu.[31] This study also found that an increase in pH (6-9) and increase of suspended sediment concentration (0-200 mg/l) significantly increased the toxicity of Roundup to *Ceriodaphnia dubia*. Interestingly, this study determined that the order of toxicity to aquatic invertebrates were as follows; POEA>Roundup>glyphosate acid>IPA salt of glyphosate. In light of these and other available data on POEA, the agency must determine that POEA poses unreasonable risk to human health and the environment.

Glyphosate and Roundup Threaten Water Quality and Aquatic Life

More than 135 million pounds of glyphosate are used annually in the U.S., applied to agricultural fields, lawns, right-of-ways and other areas where weeds are not wanted. The prevalence of Roundup-ready crops—genetically modified to tolerate glyphosate, has contributed to the high rates of glyphosate use on agricultural sites. Many sites where Roundup is used are in proximity to water ways and wetlands.[32] In 2002, the U.S. Geological Survey collected 154 water samples from 51 streams in nine Midwestern States and glyphosate was detected in 55 (36%) of the samples, and aminomethylphosphonic acid or AMPA (a degradation product of glyphosate) was detected in 107 (69%) of the samples.[33] APMA is typically detected much more frequently, especially in urban environments.[34] This study found that glyphosate contamination endured from spring through to fall when many researchers presumed it would have already degraded so late in the growing season. Glyphosate and AMPA are more frequently detected in surface water rather than ground water.[35] EPA acknowledges that glyphosate has the potential to contaminate surface water because it does not readily break down in water or sunlight. Due to glyphosate's potential for water contamination, the agency has established a maximum contaminant level (MCL) for glyphosate (0.7ppm).[36] The agency lists the short- and long-term heath effects for

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[29] Brausch, J. M., & Smith, P. N. 2007. Toxicity of Three Polyethoxylated Tallowamine Surfactant Formulations to Laboratory and Field Collected Fairy Shrimp, Thamnocephalus platyurus. *Archives of Environmental Contamination and Toxicology*, 52(2), 217-221.

[30] Wang, N., et al. 2005. Influence of sediment on the fate and toxicity of a polyethoxylated tallowamine surfactant system (MON 0818) in aquatic microcosms. *Chemosphere.*, 59(4), 545-551; Brausch, J. M., Beall, B., & Smith, P. N. 2007. Acute and Sub-Lethal Toxicity of Three POEA Surfactant Formulations to Daphnia magna. *Bull Environ Contam Toxicol*, 78, 510–514.

[31] Tsui, M., & Chu, L. 2003. Aquatic toxicity of glyphosate-based formulations: comparison between different organisms and the effects of environmental factors. *Chemosphere.*, 52(7), 1189-1197.

[32] U.S. EPA. 2006. Consumer Factsheet on: GLYPHOSATE. Ground Water & Drinking Water. Available at http://www.epa.gov/OGWDW/contaminants/dw_contamfs/glyphosa.html

[33] Scribner, E. A., Battaglin, W. A., Dietze, J. E., & Thurman, E. M. 2003. Reconnaissance Data for Glyphosate, Other Selected Herbicides, Their Degradation Products, and Antibiotics in 51 Streams in Nine Midwestern States, 2002 *U.S. Geological Survey*, Open-File Report 03–217(101 p).

[34] Kolpin, D. W., et al. 2006. Urban contributions of glyphosate and its degradate AMPA to streams in the United States *Science of The Total Environment*, 354(2-3), 191-197

[35] Scribner, E. A., Battaglin, W. A., Gilliom, R. J., & Meyer, M. T. 2007. Concentrations of Glyphosate, Its Degradation Product, Aminomethylphosphonic Acid, and Glufosinate in Ground- and Surface-Water, Rainfall, and Soil Samples Collected in the United States, 2001-06. *US Geological Survey*, Scientific Investigations Report 2007-5122(111p).

[36] Ref #23

drinking water exposures: for relatively short periods of time, congestion of the lungs and increased breathing rate; for lifetime exposure at levels above the MCL kidney damage and reproductive effects.

Glyphosate has registered use for control of emergent aquatic weeds in ditches, wetlands, and margins of water bodies. However, glyphosate and its formulated end-use products have been proven to be toxic to aquatic organisms. It is therefore unclear why the agency would allow such uses for glyphosate. Glyphosate was measured at high concentrations (highest-328 µg/l) in vernal pools and adjacent flowing waters in Washington, D.C.—a concentration that exceeds the freshwater aquatic life standard for glyphosate—in a 2008 study conducted by researchers at the USGS.[37] A study by Relyea in 2005 found that Roundup alone is "extremely lethal" to amphibians in concentrations found in the environment.[38] Another study found that *Rana pipiens* tadpoles chronically exposed to environmentally-relevant concentrations of glyphosate formulations, containing POEA, exhibited decreased snout-vent length at metamorphosis, increased time to metamorphosis, tail damage, and gonadal abnormalities. These effects were due in part to disruption of hormone signaling, because thyroid hormone receptor beta mRNA transcript levels were elevated by exposure to formulations containing glyphosate and POEA.[39] The authors of this study concluded that surfactant compositions must be considered in the evaluation of toxicity of glyphosate-based herbicides. Native freshwater mussels, *Lampsilis siliquoidea,* were found to be the most sensitive aquatic organisms tested with glyphosate-based chemicals and its surfactants.[40]

EPA's and Monsanto's message is that the application of Roundup, at recommended rates, should not adversely affect resident populations of fish or invertebrates in these systems. Yet the agency requires the labeling of some glyphosate products "toxic to fish" as these products are applied directly to aquatic environments. The agency has not taken into consideration concentrations of glyphosate or Roundup that have already contaminated these water bodies via transport of residues adsorbed in soil particles suspended in runoff water, leaching and drift, phenomena it is well aware occurs.[41] Other environmental factors such as high sedimentation, increases in temperature and pH levels have been shown to increase the toxicity of Roundup, especially to young fish,[42] though they go unaddressed by the agency. The agency has already determined that glyphosate and its salts, as well as its metabolite AMPA, are likely to adversely impact the

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[37] Battaglin, W. A., Rice, K. C., Focazio, M. J., Salmon, S., & Barry, R. X. 2008. The occurrence of glyphosate, atrazine, and other pesticides in vernal pools and adjacent streams in Washington, DC, Maryland, Iowa, and Wyoming, 2005–2006 *Environmental Monitoring and Assessment*, 155(1-4), 281-307.

[38] Relyea, R. 2005. "The lethal impact of Roundup on aquatic and terrestrial amphibians." *Ecological Applications*, 15(4), 1118–1124

[39] Howe CM, B. M., Pauli BD, Helbing CC, Werry K, and Veldhoen N. 2004. Toxicity of glyphosate-based pesticides to four North American frog species. *Environ Toxicol Chem*, 23(8), 1928-1938.

[40] Bringolf RB, Cope. WG., Mosher S, Barnhart MC and Shea D. 2007. Acute and chronic toxicity of glyphosate compounds to glochidia and juveniles of Lampsilis siliquoidea (Unionidae). *Environ Toxicol Chem.*, 26(10), 2094-2100.

[41] Ref#23

[42] Folmar, L. C., Sanders, H. O., & Julin, A. M. 1979. Toxicity of the herbicide glyphosate and several of its formulations to fish and aquatic invertebrates *Archives of Environmental Contamination and Toxicology*, 8(3), 269-278.

endangered California red-legged frog based on prey and habitat reduction.[43] EPA must now determine that glyphosate, its salts, metabolite and its formulated end-use products, pose unreasonable harm not only to frogs, but to other aquatic organisms.

Glyphosate and Roundup-Ready Crops Lead to Increasing Resistance

Herbicide-resistant weeds have ballooned in recent years, due particularly to the expansion of Roundup-ready crops, including soybeans and alfalfa. According to the USDA's National Agricultural Statistics Service, biotechnology plantings as a percentage of total crop plantings in the U.S. are 46% for corn, 76% for cotton, and 85% for soybeans. With rising prevalence of herbicide-tolerant crops, challenges caused by increased herbicide resistance have arisen. When genetically engineered food products, such as Roundup-ready crops, were commercially developed in the 1990's, they were sold to the public as a technology that, among other things, would reduce pesticide use. In reality, it has done just the opposite. The use of Roundup-ready crops increases the use of glyphosate products and in turn increases the onset of resistant species. One survey of farmers' herbicide use patterns found that glyphosate use continued to increase, with concomitant decreases in utilization of other herbicides, with a high number of farmers making one to three post applications per year.[44] Glyphosate-resistant horseweed was first reported in 2000 in Delaware[45] and has since been found in several other states, including Mississippi, Arkansas and Tennessee.[46] In 2005, University of California researchers identified strains of mare's tail, also known as horseweed (*Conyza canadensis*), that are resistant to herbicide.[47] Data showed that clusters of horseweed can grow robustly even when sprayed with four times the recommended amount of the herbicide glyphosate. The researchers hypothesized that resistance evolved due to the repeated use of glyphosate over a number of years over large treated areas. A follow-up study in 2007 showed that a glyphosate-resistant biotype of horseweed also exists in non-crop areas.[48] In addition, resistance is found in rigid ryegrass (*Lolium rigidum*) populations, the inheritance of which appeared to be nuclear, incompletely dominant, multigenic, and pollen-transmitted.[49]

In general, in regions of the U.S. where Roundup-ready crops dominate, there are now

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[43] U.S.EPA. 2009. Glyphosate Summary Document Reregistration Review: Initial Docket (p10). Office of Prevention, Pesticides and Toxic Substances.

[44] Givens, W. A., Shaw, D. R., Johnson, W. G., Weller, S. C., Young, B. G., Wilson, R. G., Owen, M. D. K., & Jordan, D. 2009. A Grower Survey of Herbicide Use Patterns in Glyphosate-Resistant Cropping Systems. *Weed Technology*, 23(1), 156-161.

[45] VanGessel, M. J. (2001). Glyphosate-resistant horseweed from Delaware. *Weed Science*, 49(6), 703-705.

[46] Koger, C. H., Poston, D. H., Hayes, R. M., & Montgomery, R. F. 2004. Glyphosate-Resistant Horseweed (Conyza canadensis) in Mississippi1. *Weed Technology*, 18(3), 820-825; Koger, C. H., & Reddy, K. N. 2005. Role of absorption and translocation in the mechanism of glyphosate resistance in horseweed (Conyza canadensis). *Weed Science*, 53(1), 84-89.

[47] Hembree, K., & Shrestha, A. 2005. Glyphosate-Resistant Horseweed In California. *University of California, Davis*.

[48] Shrestha, A., Hembree, K. J., & Va, N. 2007. Growth stage influences level of resistance in glyphosate-resistant horseweed. *California Agriculture*, 61(2), 67.

[49] Simarmata, M., Bughrara, S., & Penner, D. 2005. Inheritance of glyphosate resistance in rigid ryegrass (*Lolium rigidum*) from California. *Weed Science*, 53(5), 615-619; Simarmata, M., Kaufmann, J. E., & Penner, D. (2003). Potential basis of glyphosate resistance in California rigid ryegrass (Lolium rigidum). *Weed Science*, 51(5), 678-682.

evolved glyphosate-resistant populations of economically-damaging weed species including *Ambrosia artemissifolia* L., *Ambrosia trifida* L., *Amaranthus palmeri* S, *Amaranthus rudis, Amaranthus tuberculatus* (Moq) *Conyza* and *Lolium* spp.[50] In other parts of the world where Roundup-ready crops are used, weed resistance has also appeared. In Argentina and Brazil, for example, there are now evolved glyphosate-resistant populations of *Sorghum halepense* L. and *Euphorbia heterophylla* L. The proliferation of glyphosate-resistant weeds presents an ever-growing economic concern to farmers, since a widespread distribution of hard-to-control weeds has the potential to cause significant economic losses. Scientists studying the phenomenon agree that it is of economic concern and advise against the dependence on glyphosate, and advise the use of crop rotations and the rotation to non Roundup-ready crops.[51]

Human Incidents Are Too High

EPA's Incident Data System recorded 289 incidents involving glyphosate between 2002 and 2008. Symptoms recorded included dermal, neurological, gastro-intestinal symptoms among others. The agency states that their findings were "moderately large" and warrants searching other databases for consistency and reproducibility of incident data. The American Association of Poison Control Centers 2007 data for the National Poison Data System reported 4,593 cases involving glyphosate with 25% involving children younger than six yrs and over 85% being unintentional (accidental) exposures. This figure is considerably larger than EPA 'moderate' 289 incidents. Glyphosate is one of the highest-ranking herbicides that causes pesticide-induced illness or injury. EPA must make a greater effort to protect consumers from pesticide injuries and reevaluate the "low toxicity" rating given to glyphosate products.

Conclusion

As the agency moves forward with the registration review process for glyphosate, we hope that the scientific evidence provided in these comments, and others, would be duly considered. Glyphosate and Roundup have been linked to so many serious adverse human and environmental health concerns that the agency must take care to thoroughly evaluate the data. Most important is the finding that glyphosate end-use products are more toxic then glyphosate itself. This is important since the agency has traditionally ignored the effects of pesticide mixtures and inerts and tends to focus solely on the active ingredient. The ingredient POEA found in many glyphosate products (and others) has been found to be incredibly toxic to human cells and aquatic organisms. The agency must reconsider the use of this ingredient in any pesticide formulation, based on its potential to cause unreasonable harm.

Arguments will be made that glyphosate and Roundup are valuable herbicidal tools in the agricultural and lawn care markets. Roundup is cheaper and more readily available than many other chemicals and provides an economic benefit to many farmers for the short-term control of weeds. However, based on current science, the risks posed by glyphosate to human health and the environment, and to agriculture in the form of increasing weed

[50] Powles, S. B. 2008. Evolved glyphosate-resistant weeds around the world: lessons to be learnt. *Pest Manag Sci*, 64(4), 360-365.

[51] Culpepper, A. S. 2006. Glyphosate-Induced Weed Shifts. *Weed Technology*, 20(2), 277–281.

resistance, far outweigh any short-term economic benefit that it may provide. Conversely, there are safer alternatives for lawn care and agriculture that have proven effective at controlling weeds, provide long term economic benefit and environmental sustainability. These include organic and sound IPM practices. The agency has been tasked to protect and uphold human and environmental health and now is the time to do so.

Thank you for your attention to our comments.

Sincerely,

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