



M O N \$ A N T O

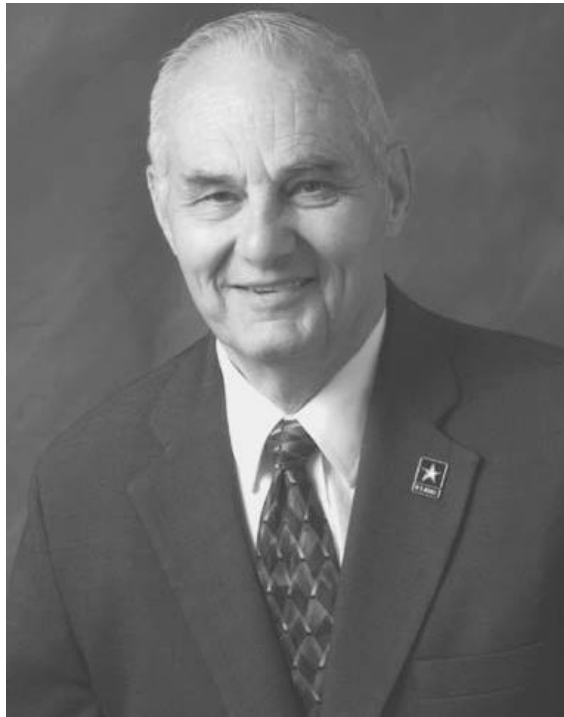
Memo n°7 : Don HUBER

Biologist

La Haye, October 15th-16th, 2016

CONTACTS :

emilie@monsanto-tribunal.org
witnesses@monsanto-tribunal.org



Brief Biographical Sketch for Don M. Huber

Dr. Don M. Huber, Professor Emeritus of Plant Pathology at Purdue University, holds B.S. and M.S. degrees from the University of Idaho (1957, 1959), a Ph-D from Michigan State University (1963), and is a graduate of the US Army Command & General Staff College and Industrial College of the Armed Forces. He was Cereal Pathologist at the University of Idaho for 8 years before joining the Department of Botany & Plant Pathology at Purdue University in 1971. His agricultural research the past 55 years has focused on the epidemiology and control of soilborne plant pathogens with emphasis on microbial ecology, cultural and biological controls, nutrient-disease interactions, pesticide-disease interactions, physiology of host-parasite relationships and techniques for rapid microbial identification. He is author or co-author of over 300 journal articles, Experiment Station Bulletins, book chapters and review articles; three books, and 84 special invited publications and an active scientific reviewer; consultant to academia, industry, and government; and international research cooperator.

Dr. Huber has had several concurrent careers including 14 years as a professional labor-relations conciliator with 7 years service on the Indiana Education Employment Relations Board as a Mediator/Fact-Finder/Conciliator. He retired in 1995 as Associate Director of the Armed Forces Medical Intelligence Center (Colonel) after 41+ years of active and reserve military service. Dr. Huber is past Chairman of the USDA-APS National Plant Disease Recovery System; a member of the US Threat Pathogens Committee; former member of the Advisory Board for the Office of Technology Assessment, U.S. Congress; and OTSG Global Epidemiology Working Group.

Synopsis of testimony to be presented at the Hague Tribunal. Professor Emeritus Don M. Huber Ph-D

Forty+ years ago, U.S. agriculture started a conversion to a primarily monochemical herbicide program focused around glyphosate (Roundup®). The near simultaneous shift from conventional tillage to no-till or minimum tillage stimulated this chemical conversion that was reinforced by the subsequent introduction of genetically modified crops tolerant to glyphosate. The introduction of genetically modified (Roundup Ready®, RR) crops has greatly increased the volume and scope of glyphosate usage, and the conversion of major segments of crop production to a monochemical herbicide strategy. The promotion of glyphosate based herbicides (GBH) as readily biodegradable, non-toxic and environmentally friendly by the Monsanto Company (masked the fact that glyphosate is a powerful mineral chelator, artificial amino acid, and broad spectrum antibiotic that interferes with mineral nutrition to increase plant, environmental, animal and human disease. Fraudulent safety reports of non-toxicity provided a false-sense of safety as adoption expanded throughout the environment (Wikipedia, 2015). Although previously over-looked, long-term damage to the soil, environment, crop, animal and human diseases have become more prevalent each year as glyphosate accumulation and residual effects escalate and become more apparent.

The extensive use of glyphosate, and the rapid adoption of genetically modified glyphosate-tolerant crops such as soybean, corn, cotton, canola, sugar beets, potato and alfalfa; each with its own unknown toxicity, have greatly increased the indiscriminate application of glyphosate and intensified deficiencies of numerous essential micronutrients and some macronutrients. Additive nutrient inefficiency of the Roundup Ready® (RR) genetics and glyphosate herbicide necessitate extensive nutrient remediation, and increase the need well above previously established soil and tissue levels for nutrients considered sufficient for specific crop production. Previous sufficient nutrient levels also may be inadequate indicators in the less nutrient efficient glyphosate weed management program because of its extensive antibiotic effects on soil biology essential for nutrient cycling, soil structure, and symbiotic nutrition (Huber, 2010).

Understanding glyphosate's mode of action and impact of the RR genetics, are necessary to understand the numerous long-term negative impacts of this monochemical system on plant nutrition and its predisposition to disease. An absolute consideration in this regard should be a much more regulated use of glyphosate, if not a total ban. Because of its persistence and broad impact on the physical-chemical and biological environment, glyphosate damage is often subtle and attributed to other causes such as drought, cool soils, deep seeding, high temperatures, crop residues, water fluctuations, etc. Some of the common symptoms of drift and residual glyphosate damage to crops presented in Table 1 reflect nutrient and disease interactions affected by glyphosate and the RR genetics as presented in scientific publications (Johal and Huber, 2009).

Understanding Glyphosate

Glyphosate (N-(phosphonomethyl)glycine) as a strong mineral chelator was first patented by Stauffer Chemical Co. in 1964 (U.S. Patent No. 3,160,632) and used to descale boilers and steam pipes. Glyphosate chelates and immobilizes essential mineral nutrients that are essential for plant and animal physiological processes. It was subsequently patented as an herbicide and later as a general biocide whereby it immobilizes mineral co-factors (Co, Cu, Fe, Mn, Ni, Zn, etc.) essential for enzyme activity

and cell function. In contrast to some compounds that chelate with a single or only a few mineral species, glyphosate is a broad-spectrum chelator with both macro and micronutrients (Ca, Co, Cu, Fe, Mg, Mn, Ni, Zn). It is this strong, broad-spectrum chelating ability that also makes glyphosate a broad-spectrum herbicide, a potent antimicrobial agent and potent environmental toxicant since the function of numerous essential enzymes is affected (Ganson and Jensen, 1988).

Primary emphasis in understanding glyphosate's herbicidal activity has focused on inhibition of the enzyme 5-enolpyruvylshikimate-3-phosphate synthase (EPSPS) at the start of the Shikimate physiological pathway for secondary metabolism. This enzyme requires reduced FMN as a co-factor (catalyst) whose reduction requires manganese (Mn). Thus, by immobilizing Mn by chelation, glyphosate denies the availability of reduced FMN for the EPSPS enzyme. It also can affect up to 25 other plant enzymes that require Mn as a co-factor and 290 other enzymes in both primary and secondary metabolism that require other mineral co-factors (Ca, Co, Cu, Fe, Mg, Ni, Zn). Several of these enzymes also function with Mn in the Shikimate pathway that is responsible for plant responses to stress and defense against pathogens (amino acids, hormones, lignin, phytoalexins, flavenoids, phenols, etc.). By inhibiting these enzymes, a plant becomes highly susceptible to various ubiquitous soilborne pathogens (*Fusarium*, *Pythium*, *Phytophthora*, *Rhizoctonia*, etc.). It is this pathogenic activity that actually kills the plant as "the herbicidal mode of action" (Johal and Rahe, 1984; Levesque and Rahe, 1992, Johal and Huber, 2009). If glyphosate is not translocated to the roots because of stem boring insects or disruption of the vascular system, aerial parts of the plant may be stunted, but the plant is not killed.

Recognizing that glyphosate is a strong chelator to immobilize essential plant micronutrients provides an understanding for the various non-herbicidal and herbicidal effects of glyphosate. Glyphosate is a phloem-mobile, systemic chemical in plants that accumulates in meristematic tissues (root, shoot tip, reproductive organs, legume nodules) and is released into the rhizosphere through root exudation (from RR as well as non-RR plants) or mineralization of treated plant residues. Degradation of glyphosate in most soils is slow or non-existent since it is not readily 'biodegradable' and is primarily by microbial co-metabolism when it does occur. Although glyphosate can be immobilized in soil (also spray tank mixtures, and plants) through chelation with various cat-ions (Ca, Mg, Cu, Fe, Mn, Ni, Zn), it is not readily degraded and can accumulate for years (in both soils and perennial plants). Very limited degradation may be a "safety" feature with glyphosate since most degradation products are toxic to normal as well as RR plants. Phosphorus fertilizers can desorb accumulated glyphosate that is immobilized in soil to damage and reduce the physiological efficiency of subsequent crops . Some of the observed affects of glyphosate are presented in table 1.

TABLE 1. Some things we know about glyphosate that influence plant nutrition and disease.

1. Glyphosate is a strong mineral chelator (for Ca, Co, Cu, Fe, Mn, Mg, Ni, Zn) – in the spray tank, in soil and in plants.
2. It is rapidly absorbed by roots, stems, and leaves, and moves systemically throughout the plant (normal and RR).
3. Accumulates in meristematic tissues (root, shoot, legume nodules, and reproductive sites) of normal and RR plants.
4. Inhibits EPSPS in the Shikimate metabolic pathway and many other plant essential enzymes.
5. Increases susceptibility to drought and disease.
6. Non-specific herbicidal activity (broad-spectrum weed control).

7. Some of the applied glyphosate is exuded from roots into soil.
8. Immobilized in soil by chelating with soil cations (Ca, Co, Cu, Fe, Mg, Mn, Ni, Zn).
9. Persists and accumulates in soil and plants for extended periods (years) – it is not readily ‘biodegradable,’ but is immobilized by chelation generally.
10. Desorbed from soil particles by phosphorus and is available for root uptake by all plants.
11. Toxic to soil organisms that facilitate nutrient access, availability, or absorption of nutrients.
12. Inhibits the uptake and translocation of Fe, Mn, and Zn at very low, non-herbicidal rates.
13. Stimulates soilborne pathogenic and other soil microbes to reduce nutrient availability.
14. Reduces secondary cell wall formation and lignin in RR and non-RR plants.
15. Inhibits nitrogen fixation by chelating Ni for ureide synthesis and is toxic to *Rhizobiaceae*.
16. Reduces physiological availability and concentration of Ca, Cu, Fe, K, Mg, Mn, and Zn in plant tissues and seed.
17. Residual soil activity can damage plants through root uptake at 1/40th of herbicidal concentrations.
18. Increases mycotoxins in stems, straw, grain, and fruit.
19. Reduces photosynthesis (CO₂ fixation).
20. Causes fruit (bud) drop and other hormonal effects.
21. Accumulates in food and feed products to enter the food chain as a concern for food safety.

Understanding the Roundup Ready® Genetics

Plants genetically engineered for glyphosate-tolerance contain an alternate EPSPS pathway (EPSPS-II) that is not blocked by glyphosate. The purpose of these gene inserts is to provide herbicidal selectivity so glyphosate can be applied directly to these plants rather than only for preplant applications. This duplicate pathway (Roundup Ready® genetics, RR) tolerance of glyphosate requires energy from the plant that otherwise could be used for yield. The RR genes are ‘silent’ in meristematic tissues where glyphosate accumulates so that these rapidly metabolizing tissues are not provided an active alternative EPSPS pathway to counter the physiological effects of glyphosate’s inhibition of EPSPS. Meristematic tissues have high physiologic activity requiring a higher availability of the essential micronutrients needed for cell division and growth that glyphosate immobilizes by chelation.

Residual glyphosate in RR plant tissues can immobilize Co, Fe, Mn, Ni, Zn or other nutrients applied as foliar amendments for 8-35 days after it has been applied. This reduces the availability of micronutrients required for photosynthesis, disease resistance, and other critical physiological functions. The presence of the RR genetics reduces nutrient uptake and physiological efficiency and may account for some of the ‘yield drag’ reported for RR crops when compared with the ‘normal’ isolines from which they were derived (Table 2). Reduced physiological efficiency from the RR gene is also reflected in reduced water use efficiency (WUE) and increased drought stress (Zobiolo, 2009) to predispose plants to more extensive damage from abiotic factors in the ecology. Application of glyphosate to a plant insures the systemic distribution of this powerful antibiotic into the soil throughout the root zone, often below areas of microbial activity for potential degradation.

The wide-spread presence of the glyphosate tolerance genetics into a high percentage (70-100 %) of our essential and economically important plants (alfalfa, canola, maize, soybean, cotton, etc.) increases societies’ vulnerability to natural stress and overt disease damage. The subsequent application of glyphosate to plants engineered to tolerate it further erodes food and feed production sustainability and increases vulnerability to disease and stress. This vulnerability is further enhanced by disruption of the biotic ecological factors essential for efficient feed and food production as well as

maintaining a healthy supportive ecology of the organisms within. Soil degradation, void of important microbial activity to provide an abundance of nutritional and disease suppression benefits has generational impacts on the ecology.

TABLE 2. Some things we know about the glyphosate-tolerance (RR) genetics.

1. Provides selective herbicidal activity for glyphosate.
2. Inserts an alternative EPSPS pathway that is not sensitive to glyphosate action in mature tissue.
3. Reduces the plant's physiological efficiency of Fe, Mn, Ni, Zn, etc.
4. Inactive (silent) in meristematic tissues (root and shoot tips, legume root nodules, and reproductive tissues).
5. Reduces nutrient uptake and efficiency to predispose to disease.
6. Increases drought stress.
7. Reduces N-fixation.
8. Lowers seed nutrient content.
9. Transferred in pollen to plants, and from degrading plant tissues to microbes.
10. Generally causes a yield 'drag' compared with near-isogenic normal plants from which it was derived.
11. Has greatly increased the application of glyphosate.
12. Permanent in plants once it is introduced.

Glyphosate as a potent antibiotic

The falsified promotion of glyphosate as a benign chemical that is non-persistent and safe for the environment has led to its indiscriminant, extensive application at increasingly high rates to result in its accumulation in the soil, air, water and food chain. In addition to glyphosate's direct impact on the ecology through reduced mineral availability, it also damages the ecology through its potent antibiotic activity. whereby it drastically disrupts the natural biological balance of soil, animal and human microbiomes. Soil structure, water holding capacity, fertility, and ecology are dependent on biological activity that is damaged by the antibiotic glyphosate to reduce crop production, increase disease and threaten agricultural sustainability.

Few areas have escaped contamination with this potent antibiotic that affects all living matter. As a consequence, the population of bees, frogs, lizards and other animals have been decimated in many areas because of the extensive contamination of their habitat with glyphosate that is toxic to the *Lactobacillus*, *Bifidobacteria* and other species essential for their digestion and disease resistance. The populations of pollinators essential for seed production of many plants especially have been decimated. Gut dysbiosis of animals and humans predisposes them to many diseases because of compromised immunity and the absence of functional vitamins and compounds otherwise produced by their gastrointestinal microbiome.

In contrast to glyphosate's toxicity to many beneficial and essential microbes, many pathogens are stimulated directly by it or indirectly through its toxicity to the natural biological controls that would function to suppress pathogen activity (Fernandez et al, 2009; Johal and Huber, 2009). This has resulted in the emergence of new economic diseases and the reemergence of endemic diseases in more virulent forms that are not readily managed by available control measures. Loss of productivity,

income, and nutrition are observed on the one hand; while mycotoxins and other toxic entities in the food chain have greatly increased to threaten the health and well-being of those consuming the products.

The irresponsible application of this massive experiment with glyphosate and GMO crops appears to be more of a generalized ecocide than a benefit to society as commercially promoted. Future historians may well look back upon our time and write, not about how many pounds of pesticides we did or did not apply, but about how willing we are to sacrifice our children and jeopardize future generations for this massive experiment we call genetic engineering that is based on failed promises and flawed science, just to benefit the bottom line of a commercial enterprise.

Some selected references:

- Bott, S., Tesfamariam, T., Kania, A., Eman, B., Aslan, N., Roemheld, V., and Neumann, G. 2011. Phytotoxicity of glyphosate soil residues re-mobilised by phosphate fertilization. *Plant Soil* 315:2-11. DOI 10, 1007/s11104-010-0689-3.
- Fernandez, M.R., Zentner, R.P., Basnyat, P., Gehl, D., Selles, F. and Huber, D. 2009. Glyphosate associations with cereal diseases caused by *Fusarium* spp. In the Canadian Prairies. *European Journal of Agronomy*. Vol. 31. pp 133-143.
- Ganson, R.J. and Jensen, R.A. 1988. The essential role of cobalt in the inhibition of the cytosolic isozyme of 3-deoxy-D-arabino-heptulosonate-7-phosphate synthase from *Nicotiana glauca* by glyphosate. *Arch Biochem. Biophys.* 260(1):85-93.
- Huber, D.M. 2010. Ag chemical and crop nutrient interactions – current update. *Proc. Fluid Fert. Forum*, Scottsdale, AZ February 14-16, 2010. Vol. 27. Fluid Fertilizer Foundation, Manhattan, KS.
- Johal, G.R. and Huber, D.M. 2009. Glyphosate effects on diseases of plants. *Eur. J. Agron.* 31:144-152.
- Johal, G.R. and Rahe, J.E. 1984. Effect of soilborne plant-pathogenic fungi on the herbicidal action of glyphosate on bean seedlings. *Phytopathology* 74:950-955.
- Levesque, C.A. and Rahe, J.E. 1992. Herbicide interactions with fungal root pathogens, with special reference to glyphosate. *Ann. Rev. Phytopathol.* 30:579-602.
- Wikipedia. 2011. Glyphosate: Legal Cases: Monsanto Fined in France for 'False' Herbicide Ads.
- Zobiolo, L.H.S. 2009. Effect of increasing doses of glyphosate on water use efficiency and photosynthesis in glyphosate-resistant soybeans. *World Soybean Conference*, Beijing, China P.R.

Dr. Don M. Huber is Emeritus Professor, Purdue University; Former Chairman, USDA National Plant Disease Recovery Program; member, US Threat Pathogens Committee; former member of the Advisory Board, Office of Technology Assessment, US Congress; and OTSG Global Epidemiology Working Group.