

GM Herbicide Tolerant Crops – Less Equals More

Herbicides needed to support glyphosate and combat resistant weeds: Glufosinate ammonium

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The rapid increase in the number of, and area affected by, weeds with resistance to glyphosate (sold as Monsanto's Roundup) in the US and South America has led to recommendations that farmers should use other herbicides to control weeds in GM crops tolerant to the herbicide (known as or Roundup Ready, or RR, crops).

One such chemical is glufosinate ammonium.

GM crops tolerant to the weedkiller glufosinate ammonium (GA) have been around for many years in North and South America but have failed to gain a significant market share of the seed and herbicide market. The rapid development of weed resistance to glyphosate due to its overuse in GM Roundup Ready (RR) crops has allowed Bayer CropScience (who manufacture GA) to promote GM GA herbicide tolerant (GAHT) crops as a "solution". The company is urging farmers to rotate different GM herbicide tolerant crops to reduce the risk of resistance developing in their "Respect the Rotation" marketing campaign.ⁱ

This increased use of all chemical weedkillers should put the final nail in the coffin of Monsanto's claim that RR crops would lead to cheaper, easier and safer weed control and demonstrate that on the contrary RR crops are now another proven staging post in the pesticides "arms race" that began in the 1940s.

What is glufosinate ammonium?

GA works by inhibiting the production of the amino acid glutamine, which leads to a build up of lethal levels of ammonia in the plant. GA has the same effect in mammals. GA breaks down into five main metabolites products: MPP¹, MPA², P-X³, P-Y⁴ and NAG.⁵ The European Food Safety Authority (EFSA) has also reported an unknown metabolite at very low levelsⁱⁱ.

GA was first approved for commercial use in 1992 in the US and in 1996 in the UK. GA herbicide is sold under the Bayer CropScience brand names Basta, Liberty and Ignite.

Uses for glufosinate ammonium

GA is used to control weeds in a range of circumstances, including around fruit trees, on GM herbicide tolerant crops and to desiccate crops such as peas, beans, potatoes and cereals prior to harvest. Direct application to growing crops, such as on GM crops, can produce higher residues of the herbicide and its breakdown products in food and animal feed.

GA is not widely used as a herbicide compared to other, older products. To date the adoption of GA tolerant GM crop varieties has been limited, due to:

- Aggressive marketing by Monsanto of their competitor RR crops (tolerant to glyphosate).
- Bayer's failure to gain EU commercial approval for GA tolerant oilseed rape and beet.
- GA being a contact herbicide, killing only the green parts of plants and not the whole plant (making it less attractive as a weed killer than glyphosate).

¹ MPP = 3, methylphosphonico-propionic acid

² MPA = 2, methylphosphonico-acetic acid

³ P-X = 3, methylphosphonico-acrylic acid

⁴ P-Y = 3, methylphosphonico-formic acid

⁵ NAG = disodium L-2-acetamido-4-methylphosphinato-butyrate

Many crops have been genetically modified to tolerate GA around the world. An increase in the cultivation of GAHT varieties could trigger a major increase in the volume of GA applied in agriculture, leading to increased exposure of the food chain and the environment to the chemical and its breakdown products.

GAHT crops are often prefixed with LL (Liberty Link), although they are now being re-branded as “Ignite” in the US. All formulated products contain other chemicals to ensure that GA is effective as a weedkiller, such as surfactants, which ensure the product sticks to leaves to allow time for GA to penetrate the tissue.

Weed resistance

Bayer CropScience has embarked on a major marketing campaign in North America to promote GA tolerant crops, under the name Ignite, as a solution to the growing problem of glyphosate resistant weeds in US RR crops.ⁱⁱⁱ Bayer is now promoting the rotation of different herbicide tolerance traits as a means to delay weed resistance becoming established, along with a field marker system based on colour flags to designate different GM herbicide tolerant crops to avoid accidentally spraying with the wrong herbicide (and killing the crop).

The fragility of this strategy is illustrated by the 2010 confirmation that Italian rye grass (*Lolium multiflorum*) is resistant to both glufosinate and glyphosate^{iv} in a small number of orchard sites in Oregon covering up to 10,000 acres. Italian ryegrass with glyphosate resistance has already been confirmed in cotton and soya crops in Mississippi. A glufosinate resistant grass (Goosegrass, *Eleusine indica*) has also been confirmed in Malaysian orchards in 2009.

GA tolerance genes could be transferred to crop relatives by cross-pollination,^v and wider resistance could evolve through repeated use over a number of years. GA is just as vulnerable to resistance developing as other herbicides when overused by farmers and other land managers in the same area over several years.

Health concerns about Glufosinate ammonium

GA is linked to several health problems. Table 1 summarises human health concerns related to glufosinate ammonium hazards.

Table 1 Human health concerns for glufosinate ammonium

General human health issues	Public exposure	Occupational exposure issues
Reproductive/Development effects. Neurotoxicant. Possible skin and eye irritant.	Slight risk to bystanders in orchards.	Exposure risk acceptable under label recommendations for use for personal protective clothing and equipment.

Source: Compiled from the [Pesticide Properties Database](#) (known as FOOTPRINT) maintained by University of Hertfordshire as a readily accessible summary source of data for users in the EU.

PAN International pesticide of concern

Glufosinate ammonium features in Pesticide Action Network (PAN) International’s *Highly Hazardous Pesticide List (HHP)*, the modern update to PAN’s famous Dirty Dozen of harmful pesticides first published in the 1980s) due to its hazards for chronic harm to human health (EU reproductive toxin). The PAN HHP List⁶ is based on official health and environmental hazard classifications from global, US and EU authorities and serves as PAN’s recommendations to the joint UN agencies’ initiative for a progressive ban on highly hazardous pesticides.

Acute toxicity

GA has been classified as “slightly hazardous” in respect of its acute toxicity by the WHO. It produces a range of symptoms from unsteadiness to lethargy to slower reactions. The European

⁶ The PAN HHP List and its rationale is available at http://www.pangermany.org/download/PAN_HHP-List_1101.pdf

Food Safety Authority (EFSA) says the metabolites MPP, MPA and NAG are all less toxic than GA. Nevertheless formulated products containing GA can be more toxic than GA alone.^{vi} According to EFSA exposure of operators applying GA to potatoes and to GM maize, “Exceeded the AOEL [acceptable operator exposure level] even when PPE [personal protective equipment] was used.”^{vii}

Embryonic development

GA interferes with the functioning of the neurotransmitter glutamate. During embryo development it causes damage to the brain and neural tube development leading to losses and deformities, including cleft palate. Recent research using the product BASTA-15 found that it, “[M]ight affect the development of pre-implantation embryos and suggest that the responsibility for this effect lies probably not solely with glufosinate ammonium, but in combination with the herbicide’s secondary compounds.”^{viii}

Chronic effect

Chronic effects from GA include decreased blood pressure and increased heart rate when given to dogs in the commercial formulation of Basta.

Exposure in food

The direct application of GA to a growing GM crop, or as a desiccant of crops such as potatoes prior to harvest, brings the greatest risk of exposure through food. In 2005 EFSA^{ix} concluded that toddlers were at risk from eating potatoes when their foliage had been killed prior to harvest using GA because the Acute Reference Dose for GA is exceeded at 114% of the permitted limit.

Exposure to GA breakdown products via animal feed can also occur. EFSA say that cattle liver and kidney can contain 1 and 2mg/kg MPP respectively when the animals are fed GM glufosinate tolerant maize or potatoes,^x although no specific risk was identified.

Environmental effects

Table 2 summarises ecotoxicological rankings of moderate or high concern related to glufosinate ammonium hazards to wildlife.

Table 2 Ecotoxicological concerns for glufosinate ammonium

Mammalian hazard rankings	Other wildlife hazard rankings
Acute oral toxicity: Moderate Short term dietary: High	Birds, aquatic crustaceans, earthworms - Moderate Harmful to arthropod natural enemies tested.

Source: Compiled from [Pesticide Properties Database](#).

GA in water and soil

There is good evidence that GA and its breakdown products are mobile in water and can leach from soils once applied. GA is moderate to highly mobile, MPP is highly mobile and MPA is high to moderately mobile in soil. GA is hydrolytically stable and it is not degraded by light in water. It is not readily biodegradable. MPP has the highest leaching potential, according to EFSA, with levels 260 times the EU maximum permitted concentration for pesticides in drinking water of 0.1µ/l found at 120cm depth in tests – suggesting groundwater could be contaminated in some soils.

Effects on soil

GA residues suppress the beneficial nitrogen fixing bacteria *Rhizobium melilot* in the soil. Researchers have also found that GA affects other soil living fungal and bacteria species. In particular some pathogenic species of fungi are increased while other microbes which normally suppress harmful species are impaired.^{xi, xii and xiii}

Toxicity to mammals

EFSA identified that GA posed “a high risk to mammals”^{xiv} and said, “Based on the available data, a high acute risk to small herbivorous mammals cannot be excluded for the representative use in orchards.”^{xv}

Aquatic toxicity

Formulated products containing GA can be more toxic to aquatic life than GA alone^{xvi}, suggesting that formulations of GA may contain toxic surfactants or adjuvants added to ensure that the active ingredient is effective or that these additives enhance the toxicity of GA. EFSA^{xvii} reported that MPP affected the reproduction of the water flea (*Daphnia* species).

Non-target invertebrates

GA is toxic to insects and other invertebrates at rates applied in the field.^{xviii} The most recent review of environmental impacts of GA by the EFSA concluded:

“A field study was submitted to address the risk to non-target arthropods^{xix}. It was noted that recovery within the season/year was not observed in all of the taxa, and effects on mites from the use of glufosinate could not be excluded. Based on the available data, the risk to non-target arthropods was assessed as high for the representative use in orchards.”

Non-target plants

GA is very toxic to plants and non-target wild plants are vulnerable to spray drift at levels below 8% of the recommended application rate.^{xx}

Conclusion

The shift from glyphosate tolerant crops to glufosinate tolerance is a classic example of “out of the frying pan, into the fire”. GA is not a sustainable option for controlling weeds. In fact there are sufficient doubts about its safety for health and the environment to suggest that its approval should be withdrawn at once.

The escalation of use that would follow the introduction of GA tolerant varieties in response to weed resistance to glyphosate would merely prop up a failing technology for a short while. GA resistant weeds will surely evolve quite soon, add to the complexity and costs of weed control for farmers and will eventually lead to more resistance in weed populations. This model of arable crops production is flawed and is now failing.

Integrated Weed Management can greatly reduce dependency on chemical weed control without using GM crops and can provide an important transitional step to agroecological methods of weed management based on crop rotations, break crops, grazing, mechanical weed control and mulches. Such approaches provide safer alternatives and deal with all types of weed – herbicide resistant or not.

Notes

ⁱ Bayer CropScience, press release 14 September 2011. “Respect the Rotation: Glyphosate-resistant weeds one year later”. See www.bayercropscience.us/news/press-releases?storyId=6fb6bb8e-69ed-4a47-8795-6e6896ef0204

ⁱⁱ European Food Safety Agency, 2005. Conclusion regarding the peer review of the pesticide risk assessment of the active substance glufosinate finalised: *EFSA Scientific Report* 27, 1-81, See page 3.

ⁱⁱⁱ GM Freeze 2011, Weed resistance in RR crops- an update.

http://www.gmfreeze.org/site_media/uploads/publications/glyphosate_brief_final.pdf

^{iv} Heap, I. The International Survey of Herbicide Resistant Weeds. Online. Internet. **April 30 2012**. Available www.weedscience.com

^v Daneils R. et al., 2005. The potential for dispersal of herbicide tolerance genes from genetically-modified, herbicide-tolerant oilseed rape crops to wild relatives. Contract reference EPG 1/5/151 http://webarchive.nationalarchives.gov.uk/20081023141438/http://www.defra.gov.uk/environment/gm/research/pdf/epg_1-5-151.pdf

^{vi} Koyama K. and Goto K., 1997. Cardiovascular effects of a herbicide containing glufosinate and a surfactant: in vitro and in vivo analyses in rats. *Toxicology and Applied Pharmacology* 145:409-14.

^{vii} European Food Safety Agency, 2005. Conclusion regarding the peer review of the pesticide risk assessment of the active substance glufosinate finalised: *EFSA Scientific Report* 27, 1-81, See page 3.

^{viii} Fabian B., Bystriansky J., Burkus J., Rehak P., Legath J. and Koppel J., 2011. The effect of herbicide BASTA 15 on the development of mouse preimplantation embryos in vivo and in vitro. *Toxicology in Vitro* 25:

73-79.

^{ix} European Food Safety Agency , 2005. Conclusion regarding the peer review of the pesticide risk assessment of the active substance glufosinate finalised: *EFSA Scientific Report 27*, 1-81,

^x European Food Safety Agency , 2005. Op cit

^{xi} Ahmad I, and Malloch D. 1995. Interaction of soil microflora with the bioherbicide phosphinothricin. *Agric Ecosys Environ* 54(3):165-174.

^{xii} Ahmad I., Bissett J. and Malloch D., 1995. Effect of phosphinothricin on nitrogen metabolism of *Trichoderma* species and its implications for their control of phytopathogenic fungi. *Pestic biochem physiol* 53(1):49-59

^{xiii} Ahmad I, Bissett J, Malloch D. 1995b. Influence of the bioherbicide phosphinothricin on interactions between phytopathogens and their antagonists. *Can J Bot /Rev Can Bot* 73(11):1750-1760.

^{xiv} European Food Safety Agency, 2005. Op cit.

^{xv} European Food Safety Agency, 2012. Conclusion On Pesticide Peer Review. Conclusion on the peer review of the pesticide risk assessment of confirmatory data submitted for the active substance glufosinate. *EFSA Journal* 10: 2609 [14 pp.]. doi:10.2903/j.efsa.2012.2609.

^{xvi} European Food Safety Agency , 2005. Op cit

^{xvii} European Food Safety Agency , 2005. Op cit

^{xviii} KEMI. 2002a. Draft Assessment Report (DAR) – public version. Initial risk assessment provided by the rapporteur Member state Sweden for the existing active substance Glufosinate (based on the variant glufosinate-ammonium) of the second stage of the review programme referred to in Article 8(2) of Council Directive 91/414/EEC.

^{xix} European Food Safety Agency, 2012. Conclusion On Pesticide Peer Review. Conclusion on the peer review of the pesticide risk assessment of confirmatory data submitted for the active substance glufosinate. *EFSA Journal* 10: 2609 [14 pp.]. doi:10.2903/j.efsa.2012.2609

^{xx} Carpenter D. and Boutin C., 2010. Sublethal effects of the herbicide glufosinate ammonium on crops and wild plants: short-term effects compared to vegetative recovery and plant reproduction. *Ecotoxicology* 19: 1322-1326.