Glyphosate fact sheet

From Pesticides Action Network UK, 1996

www.pan-uk.org/pesticide-news

Glyphosate is claimed to be the world's biggest selling herbicide by its manufacturer Monsanto(1). It is also said to be highly effective at killing weeds, safe to users and members of the public and harmless to the environment. Is it the perfect product that herbicide users want and that anti-pesticide campaigners can find no fault with?

What is glyphosate?
Glyphosate was first reported as a herbicide in 1971. Three related products are now manufactured under the name glyphosate: glyphosate-isopropylammonium and glyphosate-sesquisodium patented by Monsanto, and glyphosate-trimesium patented by ICI (now Zeneca). In pure chemical terms glyphosate is an organophosphate in that it contains carbon and phosphorous. However, it does not affect the nervous system in the same way as organophosphate insecticides, and is not a cholinesterase inhibitor.

Glyphosate is a broad spectrum, non-selective systemic herbicide. It is effective in killing all plant types including grasses, perennials and woody plants. As a herbicide glyphosate works by being absorbed into the plant mainly through its leaves but also through soft stalk tissue. It is then transported throughout the plant where it acts on various enzyme systems inhibiting amino acid metabolism in what is known as the shikimic acid pathway. This pathway exists in higher plants and microorganisms but not in animals. Plants treated with glyphosate slowly die over a period of days or weeks, and because the chemical is transported throughout the plant, no part survives.

Usage
Glyphosate is sold around the world and is formulated into dozens of products by many pesticide companies. Glyphosate product sales are currently worth approximately US$1,200 million annually and represent about 60% of global non-selective herbicides sales(2). The total world herbicide market was worth about US$14,285 million in 1995(3).

In UK arable agriculture, glyphosate was the 12th most extensively used pesticide active ingredient; the 5th most extensively used herbicide by weight with 251 tonnes being used; and 38th most widely applied herbicide, being applied over 334,529 ha annually in 1994(4). In the US nearly 8,500 tonnes was being used on 5-8 million hectares annually in the years leading up to 1991(5).

Acute toxicity
The acute toxicity of glyphosate itself is very low. According to the World Health Organisation, the oral LD50 in the rat of pure glyphosate is 4,230 mg/kg, or 5,600 mg/kg according to Monsanto(6). The low acute toxicity of glyphosate can be attributed to its biochemical mode of action on a metabolic pathway in plants (called the shikimic acid pathway) which does not exist in animals(7). However, glyphosate can also disrupt functions of enzymes in animals. In rats it was found to decrease the activity of some detoxification enzymes when injected into the abdomen(8). In general, controlled toxicity tests report adverse symptoms from exposure to glyphosate only at extremely high doses, ie several grammes per kg body weight.

While glyphosate itself may be relatively harmless, some of the products with which it is formulated have a rather less benign reputation. Marketed formulations of glyphosate generally contain a surfactant. The purpose of this is to prevent the chemical from forming into droplets and rolling off leaves which are sprayed. Some of these surfactants are serious irritants, toxic to fish, and can themselves contain contaminants which are carcinogenic to humans.

The most widely used type of surfactants in glyphosate formulations are known as ethylated amines. POEA (polyoxy-ethyleneamine) has been frequently mentioned as a surfactant, but in fact it refers to a group of ethylated amine products used in glyphosate formulations. Members of this group of surfactants are significantly more toxic than glyphosate. They are serious irritants of eyes, the respiratory tract and skin, and have been
found to contain dioxane (not dioxin) contaminants which are suspected of being carcinogenic. Accordingly, the UN FAO has set standards of 1ppm for levels of the contaminant 1,4 dioxane which may be present in POEA surfactants.

Monsanto states that all surfactants used in its glyphosate formulations fall well within the FAO standard. However, being aware of the irritant and toxic potential of the surfactants in general, the company has now developed new surfactants which have none of these toxic effects. Products containing the new formulates are sold in the UK and elsewhere and are recognised by approval authorities as being non-irritant(9). Currently in the UK, all garden products contain the new surfactant, and most local authorities are using it. However, the new formulations are more expensive and as long as there is demand for the cheaper, old formulations they will continue to be sold. Currently these are available in UK agriculture and horticulture and for professional amenity use(10).

In the UK, a local authority was prosecuted after a child was accidentally sprayed with a glyphosate formulation and suffered allergic reactions. Recently there have also been claims from residents of St. Just in Cornwall that they have suffered severe reactions following application of glyphosate for weed control(11).

In the UK, glyphosate is the most frequent cause of complaints and poisoning incidents recorded by the Health and Safety Executive's Pesticides Incidents Appraisal Panel (PIAP). Between 1990 and 1995, 33 complaints were received and 34 poisonings recorded including a single death by suicide in 1990(12,13). In California, glyphosate is one of the most commonly reported causes of illness or injury to workers from pesticides. The most common complaints are eye and skin irritation(14). The US authorities have recommended a no re-entry period of 12 hours where glyphosate is used in agricultural or industrial situations. No such recommendation exists in the UK.

**Chronic toxicity**

Some literature suggests that glyphosate can cause some chronic health effects and birth defects in certain test animals when administered at high doses over prolonged periods(15). Chronic feeding studies have shown reduced weight gain, blood and pancreatic effects, but no evidence of carcinogenicity to humans. A US EPA report says: "Effects on pregnant mothers and foetuses included diarrhoea, decreased weight gain, nasal discharge and death of mothers and kidney and digestive disorders in rat pups”(16).

It is extremely unlikely that human users or members of the public would be exposed to doses as high as those used in the trials, but extrapolating toxicity data from rats, mice and rabbits on which trials are run, to humans can be inaccurate and misleading.

**Glyphosate in the environment**

Glyphosate is inactivated when it comes into contact with soil since it is adsorbed onto soil particles. This mechanism is not fully understood, but in part glyphosate binds to soil in the same way as inorganic phosphates(17). Un-bound glyphosate is rapidly degraded by microbial activity to carbon dioxide, and bound glyphosate is degraded more slowly, sometimes remaining un-degraded but inactive in soil for years(18). Glyphosate has been found to inhibit anaerobic nitrogen fixation in soil(19,20).

Because of its adsorption to soil, glyphosate is not easily leached and is unlikely to contaminate ground water. However, glyphosate is used in water for the control of aquatic weeds, and it can be carried with eroded soil into surface waters where natural breakdown processes are much slower. On rare occasions glyphosate has been detected in water, but generally it is not looked for because it is extremely difficult to isolate and is not considered to be of major concern as a water contaminant(21).

The Forestry Commission believes that glyphosate and other herbicides commonly affect hedgerow trees causing die-back. In the US it has been suggested that herbicides, including glyphosate reduce winter hardiness in trees and their resistance to fungal disease(22). It has been suggested that damage to maple trees increases during the second year following treatment with glyphosate, and that clover planted 120 days following treatment showed reduced nitrogen fixation and growth. This implies that glyphosate which is bound to soil particles can remain active and may be released from soil and taken up by plants(23). The US-EPA has also stated that many endangered plants may be at risk from glyphosate use(24).

There may also be cause for concern where glyphosate is used extensively in programmes to eradicate drug producing plants such as coca, opium poppies and marijuana. Glyphosate is sprayed indiscriminately over vast areas and will inevitably kill non-target vegetation some of which may be endangered.

The toxicity of glyphosate to mammals and birds is generally relatively low. However, its broad spectrum of herbicidal activity has led to the destruction of habitats and food sources for some birds and amphibians leading to population reductions(25). The Houston toad is an extreme case in that it is now an endangered species due to
destruction of its habitat by glyphosate(26).

Fish and aquatic invertebrates are more sensitive to glyphosate and its formulations. Its toxicity is increased with higher water temperatures and pH. Some soil invertebrates including springtails, mites and isopods are also adversely affected by glyphosate. Of nine herbicides tested for their toxicity to soil microorganisms, glyphosate was found to be the second most toxic to a range of bacteria, fungi, actinomycetes and yeasts(27).

However, while glyphosate alone has low toxicity, the formulation of glyphosate with the surfactant polyoxyethylene amine (POEA), which is widely used, is significantly more toxic.

In Australia most formulations of glyphosate have been banned from use in or near water because of their toxic effects on tadpoles and to a lesser extent on adult frogs. There is also concern about non-lethal effects of the herbicide on frogs. New non-irritant formulations such as Roundup Biactive are excluded from the ban(28,29).

Resistance

Crops with genetically engineered resistance to glyphosate are being developed so that weeds can be controlled in fields where the crops are growing without harming the crop plants themselves. This strategy will make farmers more dependent on particular pesticidal products and will probably lead to increased use. There is also concern that the genes which display glyphosate resistance may be transferred to non-crop species including weeds.

Recent reports in professional journals indicate that resistance to glyphosate has developed in annual ryegrass in Australia(30,31). Anecdotal evidence from users in the UK suggests that similar signs of resistance in annual ryegrass and knotgrass have existed for some time.

Conclusion

Glyphosate can be an effective tool in weed control programmes and is relatively less harmful than many of the products which compete with it in the market place. There is nevertheless evidence of toxic effects on humans as well as environmental toxicity, indirect environmental damage and resistance in some target weed species.

Since glyphosate is being marketed as a safe and environmentally friendly product and its use is so extensive, there is a danger that damage to non-target plants including endangered species will increase. Habitat damage and destruction will occur more frequently and more instances of weed resistance will appear. Cultivation of glyphosate resistant crops will potentially exacerbate these problems.

So while glyphosate provides a welcome move away from chemicals which are highly toxic to humans and other non target organisms, and from chemicals which cause direct and lasting damage to the environment, it may be introducing more subtle indirect forms of damage of which users need to be aware.

References
6. Monsanto Company, Toxicology of glyphosate and Roundup herbicide, 5 June 1985, Department of Medicine and Env. Health, Monsanto Co. Missouri, US.
28. Our frogs: are they heading for the last round-up?, Sydney Morning Herald, 13 September 1995.
31. Australian ryegrass resists glyphosate, PANUPS internet bulletin board, 8 July 1996.

[This article first appeared in Pesticides News No.33, September 1996, p28-29]