Health and environmental impacts of glufosinate ammonium

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Contents

Executive summary

The introduction of genetically modified crops resistant to glufosinate ammonium will lead to a significant increase in the use of this herbicide. Aventis, the company producing these crops, is claiming that its introduction will be of benefit because of the low toxicity and environmental safety of the product. However, there are many gaps in the evidence for this claim and research indicates that glufosinate ammonium may pose a threat to human health and the environment. It is essential that questions on the safety of this product are answered before its use is scaled up with the introduction of genetically modified crops. The following table provides a comparison of Aventis* claims and the findings of independent research.

Aventis*s claims	Independent research findings
Glufosinate is safe for users and consumers.	C Glufosinate and its metabolite, MMPA-3 are neurotoxins. C Glufosinate effects the central nervous system development in baby rats. C Teratogenic effects of glufosinate include growth retardation and deformities of the brain in rats and mice. • The surfactant used in glufosinate formulations is also toxic.
Glufosinate is not a threat to drinking water.	Glufosinate is persistent and mobile in soils. Under some soil conditions such as sandy soils which overlie many aquifers, glufosinate is persistent and mobile.
Glufosinate is environmentally safe.	C Glufosinate is toxic to beneficial soil micro-organisms. C Glufosinate is a threat to wild plant communities. C Glufosinate is toxic to some aquatic organisms. C Glufosinate may increase nitrogen leaching from arable fields.
Glufosinate-resistant crops will reduce the impact of agriculture to the environment.	C Herbicide resistant crops are likely to lead to glufosinate resistant volunteers, feral populations, and resistant weeds and will intensify and increase dependency on herbicide use rather than lead to any significant reductions. C Foreign genetic material may be introduced into wild populations and effect the structure of plant communities.

Preface

In the last 50 years, UK agriculture has become increasingly dependent on artificial chemicals to control weeds, pests and diseases. The chemical revolution in agriculture of the 1950s was heralded as the solution to all the problems of farming. The weeds and pests which had plagued farming for centuries would be eliminated and food would be cheap and plentiful for all. Fifty years later, we see the situation is not so simple. Many of the pesticides proved to be toxic to humans and the environment, and the pests they were designed to kill often developed resistance to them. This has led to a seemingly endless demand for more powerful pesticides to try and keep ahead of this resistance. In developing countries, the agro-chemicals and hybrid seeds on offer after the 1950s were often inappropriate to local conditions or were too expensive for poor farmers. Increasingly, it is being recognised that it is essential to reduce reliance on chemicals in agriculture around the world and move towards less damaging systems of farming.

We are now faced with another revolution in agriculture - genetic engineering or genetic modification. In this case, the same companies who were behind the chemical revolution are manipulating the genetics of crop plants for a variety of purposes. Not surprisingly, the most well-advanced development from the genetic engineering companies is herbicide resistance. The biotechnology company Aventis has spent the last ten years developing genetically modified crops tolerant to glufosinate ammonium which will be sold in a package with the herbicide which Aventis itself manufactures. Genes from a soil bacterium which are resistant to the herbicide are spliced into the plants. Once done this gene enables the crops to be sprayed with glufosinate ammonium without being affected. The weeds under and around the crop are killed, leaving the crop to grow on free from competition for light, water and plant nutrients.

This sounds like a boon to farmers. Aventis claims its glufosinate-resistant crops offer the prospect of higher yields, reduced use of herbicides, bigger profits and a route towards a sustainable system of farming. However, these crops will also benefit Aventis hugely. Not only will they provide Aventis with a foothold in the seeds market but the uptake of glufosinate-tolerant crops will increase sales of the company's core product.

Friends of the Earth asked the Pesticides Action Network UK to review the safety of glufosinate for human health and the environment, and to look at the implications of its widespread use in agriculture due to the introduction of GM crops. The report shows the widespread use of glufosinate on these crops could have significant implications. Glufosinate is a "broad spectrum" herbicide and, if applied widely to GM crops, could leave fields almost devoid of wild plant species. This would have a serious impact on the wild creatures which depend on them for food and cover. The UK is a heavily farmed island. We do not have the space to be able to create huge wilderness areas for wildlife conservation; even our National Parks are heavily managed. Our agricultural land is, therefore, our nature reserve. The massive change in farming practice which the introduction of herbicide-tolerant crops represents could wreak havoc with wildlife in arable areas, wildlife which is already suffering under the current regime of chemical intensive farming. Friends of the Earth believes there are many other options available to reduce dependence on herbicides and place farming on a more sustainable footing.

The UK countryside is being used as a giant outdoor laboratory to test herbicide-tolerant crops. Aventis has many test sites looking at glufosinate-resistant oilseed rape, sugar beet and maize, scattered around the country, and it has started more than 100 farm-scale trials of GM maize, beet and oilseed rape in the run up to commercial introduction. This review of glufosinate raises many questions about the long-term impacts of a huge increase in glufosinate use and they must be answered before the commercial approval for glufosinate-tolerant crops is given. However, as the recent GM contamination of Advanta UK's spring oilseed rape in 2000 has highlighted, the ability of GM pollen to escape from crop fields into neighbouring crops and related wild species means the future of herbicide tolerant crops is in jeopardy. The potential economic impact on farmers and beekeepers is considerable. Advanta UK has been forced to offer hundreds of farmers compensation after they were sold GM-contaminated seed in 2000. Honey has also been found to be contaminated by a survey carried out in one area of the UK by Friends of the Earth.

Given the lack of a complete picture of the impact of herbicide-resistant crops, Friends of the Earth believes the only option for the Government is to impose a five-year moratorium on all commercial releases of herbicide-resistant crops. This will give the necessary breathing space to allow scientists, the farming community and the public to debate whether or not herbicide-tolerant crops are worth the risk. An important factor in that debate will be the impact of increased use of broad-spectrum herbicides such as glufosinate.

We hope this report will contribute to that debate.

Introduction

Glufosinate is produced by Aventis, a joint venture established in 1999 by the merger of the German company AgrEvo (owned by chemical corporations Hoechst and Schering) and the French chemical company, Rhone Poulenc. Glufosinate was developed by Hoechst in the 1970s and is now produced at its Frankfurt plant in Germany, where work began in 1995 to double production capacity in anticipation of the launch of genetically modified glufosinate-tolerant crops.[1] In 1996, Aventis invested DM 150m, most of which was spent on the expansion of glufosinate production facilities in Germany and the US.[2]

The herbicide was introduced into Japan in 1984. In the UK, glufosinate was first considered in 1984 but was not approved for toxicological reasons (see below). Glufosinate was later given provisional approval in the UK in 1991 and full approval in 1996. It was registered in the US in 1993. The product is now registered for use in more than 40 countries and is marketed under a number of trade names including Basta, Rely, Finale and Challenge. Basta contains 18.5 per cent glufosinate ammonium and 30 per cent of an anionic surfactant, sodium polyoxyethylene alkylether sulphate (AES).[3]

In the UK, relatively small amounts of glufosinate are used at present: 21 tonnes of glufosinate on 50,000 ha. The main crops are oilseed rape and potatoes,[4] on which it is used as a crop dessicant prior to harvest. These figures may change dramatically if genetically modified, glufosinate-tolerant crops are introduced on a commercial scale.

Aventis aims to promote the fast spread of glufosinate resistance into popular crop varieties, including maize, sugar beet and oilseed rape and to profit from the consequent sales of glufosinate. Aventis market Liberty, a glufosinate product for use on crops tolerant to glufosinate. This allows Aventis to enter the herbicide market for crops that command a large share of herbicide sales. In Canada, Liberty Link canola has been on sale since 1995. In 1997, Liberty Link soybean and maize were approved for commercial use in the US where the maize and soybean herbicide markets account for 40 per cent of all US pesticide sales.[5]

Aventis planned for glufosinate to become its linchpin product by 2000, with an annual turnover of about $680m by 2002.[6] The introduction of Liberty Link varieties is expected to increase sales of glufosinate to more than $300m.[7]

What is glufosinate?

Glufosinate is a broad-spectrum, contact herbicide. It is used to control a wide range of weeds after the crop emerges or for total vegetation control on land not used for cultivation. Glufosinate herbicides are also used to desiccate (dry out) crops before harvest.

Glufosinate is a short name for the ammonium salt, glufosinate ammonium. It is derived from phosphinothricin, a natural microbial toxin isolated from two species of Streptomyces fungi.

Glufosinate is a phosphorus-containing amino acid. It inhibits the activity of an enzyme, glutamine synthetase, which is necessary for the production of the amino acid glutamine and for ammonia detoxification. The application of glufosinate leads to reduced glutamine and increased ammonia levels in the plant's tissues. This causes photosynthesis to stop and the plant dies within a few days.[8] Glufosinate also inhibits the same enzyme in animals.

Uptake of glufosinate is through the leaves and stem, and damage is restricted to those parts of the plants which are in direct contact with the spray. The active ingredient can move within leaves but cannot move to other parts of the plant such as underground rhizomes or stolons. Long-term control of perennial weeds is therefore limited. The anionic surfactant, sodium polyoxyethylene alkylether sulphate (AES), is added to enhance the plant’s absorption of glufosinate. This surfactant is widely used in shampoos and detergents.

Health impacts

Hoechst claimed that under conditions of recommended use of glufosinate ammonium, a "detrimental effect on the health of both users and consumers is extremely unlikely".[9]

Glufosinate ammonium structurally resembles glutamate, a typical excitatory amino acid in the central nervous system. It is recognised that excess release of glutamate results in the death of nerve cells in the brain.[10] In mammals, both glufosinate and the surfactant, AES, are rapidly absorbed through the gut.[11] Ingestion of glufosinate affects the nervous system and evidence of neurotoxicity has been found in most species of laboratory animals exposed to glufosinate.[12]

Glufosinate was first considered by the UK Ministry of Agriculture, Fisheries and Food (MAFF) Scientific Sub-Committee in 1984. At the time, the herbicide was not granted approval because of the greater toxicity of the formulation (containing 30 per cent surfactant) over the active ingredient when administered through the skin.[13]

In 1991, The Scientific Sub-Committee recommended "provisional approval for six products for five years with a data submission deadline of three years subject to a number of specific conditions and label amendments".[14] However, the Sub-Committee remained concerned about the increased dermal toxicity of one of the six formulations, requiring the applicants to submit further studies.

Acute toxic effects in humans

Ingestion of large quantities of the glufosinate formulation Basta causes death in humans: 10-20 suicides a year in Japan have resulted from Basta poisoning.[15] The amount used is well above what would be expected in normal usage, but it gives an indication of the toxic effects of high doses of this formulation on humans. Clinical findings in poisoning cases are believed to be due to damage to the central nervous system and include consciousness disturbance, convulsions, pyrexia, and respiratory failure.[16] Death is usually due to circulatory failure, which may mainly come from the surfactant.[17] [18]

The metabolite, MPPA-3, is, like glufosinate, a neurotoxin.[19]

Low-dose neurotoxic effects

Low doses of glufosinate have been shown to affect central nervous system development in baby rats. Seven days of dosing baby rats with 1 mg/kg per day led to changes in glutamate receptor response in their brains.[20]

Teratogenic effects (birth defects)

In recent studies, sub-lethal doses of glufosinate ammonium were found to cause abnormalities in the development of embryos in mammals both in vitro and in vivo. Deformities in the brain were the main finding of these studies:

- Mouse embryos exposed to glufosinate in vitro developed apoptosis (fragmentation of the cells leading to cell death) in the neuroepithelium of the brain.[21] An earlier study found that all the embryos in the treated groups had specific defects including overall growth retardation, increased death of embryos, hypoplasia (incomplete development) of the forebrain at 10 µg/ml, and cleft lips at 20 µg/ml. Histological examination showed cell death throughout the neuroepithelium in the brain vesicle and neural tube.[22]

! The effects on embryos after exposure of pregnant rats to glufosinate during the time of neurogenesis (central nervous system development) was determined. Pregnant rats were injected subcutaneously with 3 or 5 mg/kg of glufosinate once daily from days 13-20 of gestation. Six weeks after birth, the progeny of the rats treated with 5 mg/kg glufosinate showed a decrease in the glutamate receptor response to kainic acid. The results suggested that glufosinate exposure at a crucial stage in pregnancy causes a decrease in the number of glutamate receptors in offspring.[23]

Concerns about potential teratogenic effects of glufosinate on humans

A recent study in Italy has found a weak correlation between exposure of fathers to glufosinate and congenital malformations in babies.[24] The study found increased risk of congenital malformation with exposure to glufosinate, but the increase was not significant statistically. Although this study does not prove an association, it does raise concerns, and more research is clearly required.

Residues in food

Residues in food are an area of concern, especially when glufosinate is used as a pre-harvest desiccant. In the UK, MAFF found that adult consumers are most likely to be exposed to residues of glufosinate in potatoes and dried (or processed) peas and in liver and kidney from animals fed on contaminated cereal straw.[25] The WHO/FAO recommended ADI for glufosinate is 0.02 mg/kg body weight.

MAFF*s 1990 evaluation document on glufosinate states that when glufosinate is used as a desiccant, glufosinate residues will be found in dried peas, field beans, wheat, barley, oilseed rape, and linseed. The highest likely residue levels in commodities for human consumption were considered to be: 3 mg/kg in peas, 1 mg/kg in wheat grain, and 0.5 mg/kg in oilseed rape seeds. The reported residue levels in animal feed were high, including 50 mg/kg in barley straw and pea stalks and 20 mg/kg in wheat straw and field bean stalks.

MAFF reported that when wheat grain containing residues was turned into flour, 10-100 per cent of the residue was retained. Residue levels in bran were 10-600 per cent of those in grain. Data on the effects of processing on residues in oilseeds were not available. In addition, MAFF found that the use of glufosinate as a herbicide and/or a desiccant in potato crops can lead to residues in the tubers in the order of 0.1 mg/kg. Residues of the metabolite, MPPA-3, were found in potato tubers 77 days after treatment, 0.07 mg/kg were detected after a single treatment, and 0.24 mg/kg following double rate treatment.[26] A report produced by the UK Pesticides Safety Directorate in 1998 stated that it was probable that GM crops would receive at least two applications of glufosinate.[27]

In 1991, the MAFF Advisory Committee on Pesticides, the body responsible for advising Government ministers concerning pesticide registration in the UK, was concerned that significant residues of glufosinate were found in the crops at the time of harvest.[28] In particular, they were concerned that residues of "additive ingredient" and the metabolite, MPPA-3, were found in milk and the tissues of animals fed treated straw. The Sub-Committee proposed a restriction on straw feeding to reduce health risks to livestock and consumer intake of residues in animal products.

Additional fears with residues in food from GM crops

Glufosinate-tolerant oilseed rape may pose a particular risk of its own. In Germany, the Robert Koch Institute, the competent authority for authorising the marketing of glufosinate-tolerant oilseed rape, noted that a metabolite (N-acetyl-L-phosphinothricin) is formed in glufosinate-treated transgenic plants which does not appear to degrade, or to only degrade at a very slow rate.[29]

Data provided by Aventis (then AgrEvo) demonstrate that this metabolite can be reconverted into the active herbicidal form by micro-organisms in the digestive tract of warm blooded animals. The Robert Koch Institute concluded that the use of glufosinate herbicides in genetically modified oilseed crops may result in metabolites whose physiological activity corresponds to that of the herbicide glufosinate. This finding has implications for consumers, as oil extracted from the crop is used in cooking oil, margarine, mayonnaise, dressings and cocoa butter substitutes.

Residues in drinking water

Aventis claims that glufosinate is unlikely to leach into groundwater.[30] However, glufosinate is highly soluble in water and is also classified as persistent and mobile (see above). MAFF*s Scientific Sub-Committee considers that "under certain conditions significant run-off or leaching could occur, leading to contamination of ground or surface water".[31] The dangers of soluble pesticides contaminating water supplies as a result of recommended agricultural use is now generally recognised by both the industry and governments throughout the European Union.

The use of glufosinate could rise dramatically in the next few years with the commercialisation of glufosinate-tolerant crops. This could increase the likelihood of water contamination. Only monitoring over time will establish if glufosinate is a threat to drinking water. Leaflets published by Aventis in 2000 suggest the company may have carried out further research on the mobility of glufosinate in the soil but these data have not been published and requests made by Friends of the Earth in January 2000 to access the information through the Pesticide Safety Directorate have not yet been resolved in May 2001. Because of the current relative low use of glufosinate, the Environment Agency and water companies do not routinely monitor ground or surface waters for its presence.

Environmental fate

Glufosinate's principal breakdown product found in plants, animals and soil is 3-(methylphosphinyl)propionic acid (MPPA-3). 2-(methylphosphinyl)acetic acid (MPAA-2) and CO2 are also produced.

Persistence in soil and water

The US Environmental Protection Agency (US EPA) classifies glufosinate ammonium as “persistent” and “mobile”. Degradation of glufosinate is largely by microbial activity. The half life has been determined in numerous laboratory studies and varies from 3 to 42 days in some studies and up to 70 days in others.[32] The shortest half life tends to be in soils with a high clay and organic matter content.[33]

A number of factors can affect persistence including temperature,[34] soil moisture content, and whether the soil is uncovered or covered with grasses.[35] The presence of nitrogen fertilisers may also affect the persistence of glufosinate. Some bacteria appear to use glufosinate as a source of nitrogen and the availability of inorganic nitrogen would provide an alternative source for the bacteria.[36]

In one study, residues of glufosinate were found in the edible parts of spinach, radishes, wheat and carrots planted 120 days after glufosinate had been applied.[37] In sandy soils, which overlie many aquifers, glufosinate has been found to be highly persistent due to lack of biodegradation. Its transport through the soil was also determined to be “essentially unretarded”.[38]

Glufosinate*s metabolite, MPPA-3, has been found to be more persistent and more mobile than glufosinate.[39] In a soil column leaching experiment the amount of MPPA-3 leached was greater by a factor of 20 than the amount of glufosinate leached.[40]

Effects of glufosinate on wildlife and the environment

Very little information is available on the effects of glufosinate on aquatic and terrestrial wildlife. Most of the experimental work to date has been produced as a requirement of registration and has focused on the lethal dose rates for different organisms. Information on the sub-lethal effects of glufosinate on plants or animals is sparse. Researchers at the department of Animal Ecology, Justus-Liebig University, Giessen, Germany are concerned about the lack of data on the impacts of glufosinate in the environment. They are particularly concerned about the commercialisation of glufosinate tolerant crops and say “it has become a matter of urgency to make a study of the behaviour of this substance [glufosinate] in conjunction with natural systems”.[41]

Effects on soil micro-organisms

Aventis claims that glufosinate will not harm most common soil bacteria.[42] However, Phosphinothricin is a natural microbial toxin and there has been a relatively large interest in the effects of glufosinate soil microbial populations. Many studies have found that glufosinate significantly decreases bacterial and fungi populations with some species being more susceptible than others:

! In vitro studies of the sensitivity of 227 soil and water bacteria to glufosinate found that 38 strains (17 per cent) were wholly resistant at concentrations up to 3 mM (millimoles) of glufosinate, while the growth of 84 (37 per cent) was inhibited at concentrations of less than 1 mM. Inhibition could be reversed with glutamine supplements. Glufosinate-tolerant mutants appeared in all sensitive isolates. These mutants were found to have glutamine synthetase levels on average 10 times higher than glufosinate-sensitive wild types.[43]

! A study of agricultural and boreal forest soils found that the number of fungi and bacteria isolated from agricultural soils was reduced by 20 per cent and 40 per cent respectively in the presence of 1mM of glufosinate. In the boreal forest soils, bacteria isolates were suppressed by 20 per cent.[44]

Aquatic animals

Glufosinate is toxic to a number of aquatic animals including the larvae of clams and oysters,[45] daphnia and some freshwater fish species.[46] The commercial formulations are more toxic than the technical grade glufosinate. For example, for the aqueous formulation, the LC_{50}s for the fish tested were between 12.3 and 79 mg/l and for the active ingredient they were between 320 and 1000 mg/l.[47] The rainbow trout, *Oncorhynchus mykiss*, was the most sensitive species in these tests. It is not clear what a worst-case exposure of the aquatic environment to poorly-applied glufosinate could be.

Effects on beneficial insects

Very little data is available on the impacts of glufosinate on beneficial insects. The indirect effects through changes in habitat are likely to be similar for glyphosate. There is evidence that Basta is highly toxic to phytoselids and bagworm moths, both useful predators in the arable field.[48]

Terrestrial plants

Glufosinate is a broad-spectrum herbicide and is, therefore, damaging to most plants that it comes into contact with.

Agricultural impacts

The widespread use of glufosinate herbicides could have a number of impacts on the arable environment. It can inhibit some beneficial bacteria and fungi and may increase the susceptibility of some crop plants to disease. Over long periods of continuous use, weeds may become tolerant to glufosinate, and a possible consequence of this is an increase in the application of other agrochemicals including fertilisers, insecticides, fungicides and herbicides. The introduction of GM glufosinate tolerant crops will increase the use of glufosinate and may enhance these adverse impacts. There are also new risks specific to GM crops, which are discussed later.

Nutrient cycles

Glufosinate is toxic to a number of nitrogen-fixing soil bacteria. For example the nitrogen-fixing bacteria, *Rhizobium melilot,* was found to be sensitive to low doses of glufosinate. Growth of the bacteria was more strongly inhibited in sterile soils than in unsterile soils, where only a transient growth reduction was detectable. In sterile soil, rhizobial nodulation rates of glufosinate tolerant alfalfa plants was drastically reduced when exposed to glufosinate. Nitrogen fixation in the few nodules that arose was unaffected but because there were so few nodules the overall fixation rate was strongly diminished. In unsterile soil, nodulation and nitrogen fixation rates were not changed, probably due to rapid degradation of glufosinate in the unsterile soils under laboratory conditions.[49] In the real world soil in unsterile, but these results raise clear concern about potential impacts on nitrogen fixation.

Effects on cellulose decomposition

Glufosinate ammonium has been found to inhibit decomposition of cellulose in soil. The decomposition rate of cellulose substrates buried in soil immediately after the soil was treated with glufosinate (at 150 ppm) decreased by 78 per cent compared to the control.[50]

Effects on plant diseases

Some plant pathogens are highly resistant to glufosinate ammonium. Soil fungi grown in the presence of 1 mM glufosinate ammonium exhibited a wide range of tolerance to increasing herbicide concentration over the range of 0-50 mM. The plant pathogen *Verticilium alboatrum* was among the most resistant, while the parasitic fungi species *Trichoderma harzianum* and *T. longipilus* were among the most sensitive.[51]

Several studies have found that glufosinate ammonium inhibits a number of soil micro-organisms that are antagonistic to disease-causing micro-organisms in higher plants. Glufosinate ammonium was found to be inhibitory to antagonistic soil micro-organisms, including *Bacillus subtilis*, *Pseudomonas fluorescens***,** and many species of *Trichoderma*. *Trichoderma* parasitize the plant pathogens, *Fusarium oxysporum* and *Pythium aphanidermatum* which were found to be highly resistant to glufosinate. When glufosinate was added to soil containing both antagonist and the plant pathogen species, the ability of the antagonist to exclude the plant pathogen was greatly reduced.[52]

Effects on nitrate leaching

The use of glufosinate may increase nitrate content in soils and increase risks of nitrate leaching. In an open-field experiment with no plant growth, the application of Basta led to a rise in nitrate content. Heavy rainfall (about 100mm) between the first and second stages of the experiment led to the leaching of nitrates. The raised nitrate content continued throughout the duration of the experiment.[53]

Impacts of genetically modified glufosinate-tolerant crops

Some of the recognised implications of planting herbicide-tolerant crops include:

C the spread of genetically engineered herbicide tolerant genes to related weed species and to neighbouring crops
C increased risks of weeds naturally developing resistance to the herbicide
C change in the use of herbicides
C transgenic crops as weeds
C potential loss of farmland biodiversity.

Cross-pollination with related weeds

The transfer of glufosinate-resistance genes to the wild or weedy relatives of GM crop plants can take place by pollen dispersal (by insects or wind) or through vectors such as viruses or nematodes. The potential for transfer of the foreign gene depends on the local ecology in areas of transgenic crop production. It is most likely to occur in crops such as rice, barley, maize, oats, potatoes and brassicas, depending in which region of the world they are grown. For example in the US, where many of the GM crops are developed, there are no wild relatives of maize, soybeans, wheat or cotton. In Spain, there are many wild relatives of crops, which could potentially harbour modified crop genes.[54] In the UK, the Government's Advisory Committee on Releases to the Environment has acknowledged that oilseed rape is likely to be able to cross-breed with several related UK plant species.[55]

Until recently, it was widely assumed that isolation from other crops and the use of non-GM border rows around test sties would reduce any pollen transfer to a minimum. However, monitoring carried out by the National Institute of Agricultural Botany (NIAB) in the UK has found that pollen was dispersed up to 400m from a 9 hectare field of GM oilseed rape, despite the field being surrounded by a 20m deep boundary of unmodified oilseed rape.[56]

Studies of oilseed rape have shown that pollen densities around large agricultural fields are very much higher and have dispersal characteristics unlike those of experimental plots.[57] Moreover, recent studies have shown that wind dispersal of oilseed rape pollen from agricultural fields occurs over much greater distances and at higher concentrations than originally predicted. Feral and volunteer populations of GM oilseed rape further complicate the problem, as these can potentially act as additional sources of GM pollen. In a report examining existing patterns of gene flow in oilseed rape farming areas, it was concluded that "potential exists for a continuous network of cross-pollination of oilseed rape across any region growing the crop".[58]

Indications that cross-pollination and the transfer of herbicide-tolerant genes is a possibility include:

- Tests with experimental plots detected gene flow between neighbouring spring- and autumn-sown fields and between fields and feral populations. It was concluded that given the close proximity of fields and feral populations within the agricultural environment, significant levels of gene flow from transgenic oil seed rape fields is inevitable.[101]
- A Danish study found that spontaneous hybridisation between oilseed rape and wild turnip (*Brassica rapa*) could occur under field conditions. The hybrid plants were highly fertile and carried a transgene from the oilseed rape. The researchers concluded that the rapid spread of genes from oilseed rape to the weedy relative *B. rapa* is possible.[59]
- In a report for the UK Advisory Committee on Releases to the Environment it was concluded that, in the case of wild turnip, "the production of herbicide-tolerant hybrids must be considered a realistic prospect".[60]
- Research in France demonstrated that hybridisation can occur in the field between oilseed rape and wild radish (*Raphanus raphanistrum*). The progeny of the hybrid exhibited characteristics of both parents and the authors of the study recommended further careful research before planting large areas of herbicide-tolerant crops.[61]
- Other studies have shown that hybridisation occurs between oilseed rape and hoary mustard (*Hirschfeldia incana*). It was found that, under competitive conditions, the hybrid plants do better than the hoary mustard suggesting that hybridisation may be an

important avenue for gene escape for oilseed rape.[62]

- In an experiment in which wild radish was crossed with oilseed rape carrying a gene for resistance to glufosinate, the tolerant gene persisted after breeding the hybrids for four generations. The study also predicted that other transgenes in oilseed rape, such as antibiotic tolerant genes used as markers, will readily transfer to wild plants.
- A study in France found that gene flow occurred between GM herbicide tolerant sugar beet and weed beet in a neighboring field within one growing season.[63]
- In Africa, sorghum hybridises with weedy relatives producing a weed pest that looks very similar to the crop and is therefore very hard to control. ICI (now Zeneca) have ceased work on herbicide-resistance in sorghum for this reason.[64]
- The US Department of Agriculture has acknowledged that natural hybridisation is known to occur between cultivated soya bean, *Glycine max*, and a wild relative, *Glycine soja.*[65] *G. max* is thought to be derived from *G. soja* and where the two species overlap, a number of intermediate forms are found[66]. *G. Soja* is a principle weed in Japan. It is also found in Australia, southern China, Taiwan, the Philippines and Papua New Guinea. Transfer of glyphosate tolerant genes from Roundup Ready Soya could increase the problems with this weed in Japan and elsewhere.

The initial benefits manufacturers claim for herbicide-resistant crops may be eliminated quickly by the impacts of cross-breeding with weedy relatives. In particular, crops such as sugar beet for which problem weeds are closely related to the crop, will rapidly lose the benefits of herbicide tolerance for weed control.

Weed resistance

Weed resistance is a major area of concern for farmers all over the world. It can have serious agronomic and economic impacts especially when weeds gain multiple resistance to a range of herbicides. In a 1995/6 International Survey of Herbicide Resistant Weeds, 183 herbicide-resistant weed biotypes were recorded in 42 countries.[67] A resistant weed biotype is a naturally occurring group of individual weeds with the same genetic predisposition to resist a herbicide. The study recorded 124 weed species with herbicide resistance. Most of the resistant biotypes were found in countries where herbicides are the primary weed control method. For example, there were 49 resistant weed biotypes in the US, 24 in France and Spain, 22 in Canada and 16 in the UK.

Although glufosinate is classed as a non-selective herbicide, variation in the response of plant species to exposure has been widely observed. In one study a 70-fold difference was observed in the sensitivity of seven plant species to glufosinate,[68] and in another it was found that in the case of fat hen (*Chenopodium album*) acceptable levels of control were not achieved even at high rates of application.[69] In literature produced by Hoechst it is stated that some of the weeds most tolerant to glufosinate include docks (*Rumex spp*), perennial sowthistle (*Sonchus arvensis*) and couch grass (*Agropyron repens*).[70] It seems highly possible that repeated and widespread use of glufosinate may lead to the rapid natural development of glufosinate-resistant weed populations.

While industry is proclaiming that herbicide-resistant crops will make weed control even simpler, weed scientists are calling for the adoption of longer-term, more complex, non-chemical, weed control strategies that reduce risks of resistance. For instance, field experiments in the southern Canadian prairies looked at ways to reduce both costs and the risks of herbicide resistance.[71] Conventional use of repeated applications of the herbicides glyphosate and 2,4-D controls weeds in fallow rotations with winter and spring wheat. However, methods involving a combination of herbicides and tillage gave the best results for all weed species and also minimised the risks of soil erosion. Soil water retention and succeeding wheat yields were similar to, or greater than, those attained with repeated herbicide use and no tillage.

Herbicide use

Aventis claims that the use of glufosinate-tolerant crops will reduce the quantity of older, more toxic herbicides used by farmers. In the short term, the use of herbicides may appear to decline, but in the long term there are other considerations which will affect the levels of herbicides and possibly other pesticides used. These include:

- The development of glufosinate resistance in weeds or the use of practices to minimise the risks of weed resistance will perpetuate the practice of applying mixtures of herbicides that have a range of adverse environmental impacts.
- The presence of glyphosate-resistant volunteers will require the use of additional applications of herbicides other than glufosinate. The Dutch Government has expressed concern that if herbicide-resistant crops are commercialised, then the greater volumes and varieties of herbicides required in removing volunteer and feral plants will have impacts on soil and groundwater.[72]
- The impact on plant diseases after long-term use of glufosinate may mean that farmers are increasing the use of fungicides.

Rather than making significant reductions in pesticide use, in the long term, herbicide-resistant crops will intensify chemical dependence in agriculture. Other weed control strategies are being developed that aim to reduce dependency on herbicides and minimise environmental impact. For example, scientists working for DLO-NL, the Dutch Government-funded organisation for agricultural research, are assessing new systems of weed control that are reducing the level of herbicide used in sugar beet and maize drastically. The researchers are worried that widespread use of herbicide-resistant plants will remove the incentives for farmers to adopt these non-chemical, more environmentally benign control systems.[73]

Loss of farmland biodiversity

There is a great deal of controversy in the UK about whether herbicide-tolerant crops will pose a threat to the health of plants and animals. The Government has commissioned various research projects to examine this issue, in particular farm scale trials of herbicide-tolerant crops. This will not be complete until 2003. In a paper produced by the UK Pesticide Safety Directorate it was stated, in reference to the impact of herbicide tolerant crops on biodiversity, that “there is currently a lack of independent research to allow an accurate prediction of the potential impacts”.[74] However, it has been widely suggested that the switch to broad-spectrum herbicides which are partnered to these crops could have significant impacts upon the biodiversity of UK arable farmland.

In July 1998, English Nature produced a position statement on genetically modified organisms. The accompanying press statement said: “More research is needed and existing research needs to be completed before we can have a better idea of the possible effects of GMOs on our already hard-pressed farmland wildlife ... [the] environmentally un-tested introduction of GMOs could be the final blow for species such as the skylark, corn bunting and the linnet."[75]

This position is backed up by other conservation organisations. In 1997, the Countryside Council for Wales supported an English Nature proposal that no commercial releases of herbicide-tolerant crops be approved for a five-year period.[76] Scottish Natural Heritage's response to a MAFF discussion paper on herbicide-tolerant crops, was: "Genetically modified herbicide-tolerant varieties appear to offer the possibility of virtually weed-free crops, so removing the remaining food source for these [farmland] birds (many of which have already seriously declined in numbers during the past 20 years). Although the popular focus has been on skylarks and song thrushes, this is potentially serious for a far wider range of farmland birds."[77]

And according to the Royal Society for the Protection of Birds (RSPB): "The development of genetically modified, herbicide-tolerant crops could lead to a massive increase in the use of broad-spectrum herbicides which would have a huge impact on biodiversity by increasing indirect effects of pesticides and further diminishing the food chain." Referring specifically to oilseed rape, the RSPB said: "Broadleaved crops, such as oilseed rape and sugar beet, support large populations of broadleaved weeds, making them important in the arable rotation to wildlife. Genetically modified herbicide-tolerant crops could effectively be 'sterilised' of weeds by broad spectrum sprays."[78]

Impact on farmers and beekeepers

The most likely plants to be cross-pollinated by GM herbicide-tolerant crops are the non GM crops of the same species growing near by. There has been much debate in the UK about the separation distance required to prevent or minimise cross pollination between neighbouring crops and what would be an acceptable contamination threshold. It seems highly probable that such cross pollination will occur in wind pollinated and insect pollinated crops such as oilseed rape and maize. Farmers suffering such contamination would be faced with the task of controlling glufosinate-tolerant oilseed volunteers, referred to above, leading to increased expenditure on herbicides. There may also be an economic impact if farmers were unable to sell the contaminated crop in a food market that is increasingly demanding GM-free raw materials. In 2000, several hundred farmers who had been sold GM-contaminated spring oilseed rape faced this possibility. Their seed supplier, Advanta UK Ltd, was eventually forced to compensate farmers after it became clear that their was no market for the crop.

Honey can also be contaminated by GM pollen collected by bees - which creates marking problems for beekeepers. In 2000, the British Bee Farmers association advised its members to move their hives six miles from the nearest GM trail site to minimise risk of contamination. In these circumstances, farmers growing fruit (both top fruit and soft fruit) could lose the vital pollination service provided by local hives, leading to lower fruit yields.

Conclusions

The development of herbicide-resistant crops is a strategy developed by a number of chemical companies to increase profits and ensure key product lines can compete in the market place. Aventis has targeted the broad-spectrum herbicide, glufosinate, as its linchpin product for the future and initiated a fast-track programme to produce a range of crops resistant to glufosinate.

Aventis claims glufosinate is an environmentally safe herbicide. However, the studies outlined in this report demonstrate that it is a neurotoxin and can cause serious damage to growing foetuses in experimental animals; it may leach to drinking water sources; it could increase nitrate leaching; and is toxic to beneficial soil micro-organisms. The introduction of glufosinate-resistant crops increases the likelihood of these harmful effects in humans and the environment. MAFF*s Advisory Committee on Pesticides has highlighted concerns for levels of glufosinate residues in crops destined for both human and livestock consumption and the potential for glufosinate to contaminate surface and groundwater.

References

1.S. Watkins, 1995. *Agrow*s top twenty five*. Report ref: DS 106. PJB Publications, London
2.Agrow No. 268, November 15th 1996, p. 7.
3.Koyama, K., K. Koyama and K. Goto, 1997. *Cardiovascular effects of a herbicide containing glufosinate and a surfactant: in vitro and in vivo analyses in rat*. Tox. And App. Pharm. 145, pp. 409-414.
4.MAFF, *Pesticides usage survey report*. MAFF, York
5.Agrow No. 273 January 31st 1997, p. 21
6.Watkins S, 1995. *Agrow's top twenty five*. Report ref: DS 106, PJB Publications, London.
7.Agrow, 1997. *Agrow*s top twenty five* 1997 edition. p. 24
8.Rasche E, J. Cremer, G. Donn, J, Zink. 1995. *The Development of Glufosinate Ammonium Tolerant Crops into the Market*. In Brighton Crop Protection Conference, Weeds, 1995. British Crop Protection Council, Farnham, Surrey
9.Hack, R. , E Ebert, G. Ehling, and K.H. Leist, 1994. *Glufosinate-ammonium - some aspects of its mode of action in mammals*. Food and Chemical Toxicology, Vol. 32, No. 5, pp. 461-470.
10. Fujii, T. 1997. *Transgenerational effects of maternal exposure to chemicals on the functional development of the brain in the offspring*. Cancer Causes and Control, Vol. 8, No. 3, pp. 524-528.
11. Koyama, K., K. Koyama and K. Goto, 1997. Op. Cit 3
12. Cox, C. , 1996. *Herbicide Fact Sheet : Glufosinate. J. of Pesticide Reform, North West Coalition for Alternatives to Pesticides (NCAP)*, Oregan, USA
13. MAFF, Health and Safety Executive, 1991. Advisory Committee on Pesticides Annual Report 1991. HMSO, London.
14. MAFF, Health and Safety Executive, 1991. Ibid.
15. Koyama, K., K. Koyama and K. Goto, 1997. Op. Cit. 3.
16. Koyama K, Y. Andou, K. Saruki, and H. Matsuo, 1994. *Delayed and severe toxicities of a herbicide containing glufosinate and a surfactant*. Vet. Human Toxicol. Vol. 36, No. 1, pp. 17-18.
17. Koyama K, Y. Andou, K. Saruki, and H. Matsuo, 1994. Ibid
18. Koyama, K., K. Koyama and K. Goto, 1997. Op. Cit. 3.
19. U.S. EPA, 1988. *Catecholamines and glutamine synthetase determinations on Hoe 039866 and Hoe061517 treated rats*. Data Evaluation report. Cited in C.Cox, 1996. Op. Cit. 12.
20. Fujii, T., T. Ohata, M. Horinaka, 1996. *Alternations in the response to kainic acid in rats exposed to glufosinate-ammonium, a herbicide, during infantile period*. Proc. Of the Japan Acad. Series B-Physical and Biological Sciences, Vol. 72, No. 1, pp. 7-10.
21.Watanabe, T. , 1997. *Apoptosis induced by glufoosinate ammonium in the neuroepithelium of developing mouse embryos in culture*. Neuroscientific Letters, Vol. 222, No. 1, pp.17-20.
22. Watanabe, T. and T. Iwase, 1996. *Development and dymorphogenic effects of glufosinate ammonium on mouse embryos in culture*. Teratogenesis carcinogenesis and mutagenesis, Vol. 16, No. 6, pp. 287-299.
23. Fujii, T. 1997. Op. Cit. 10.
24.
Garcia, A.M., Benavides, F.G., Fletcher, T., Orts, E, 1998. *Paternal exposure to pesticides and congenital malformations*. Scandinavian Journal of Work and Environmental Health, Vol. 24, Ni. 6, pp 473-480.

25 MAFF, 1990. Op. Cit. 13.
26 MAFF, 1990. Ibid.
27.Pesticides Safety Directorate, 1998. Scientific Review of the impact of herbicide use on genetically modified crops 15 December 1998
28 MAFF, Health and Safety Executive, 1991. Advisory Committee on Pesticides Annual Report 1991. HMSO, London.
29.Robert Koch Institute, Notification for the placing on the market under Part C, Article 13 of Directive 90/220/EEC; Notification No. C/DE/96/5: Application for the placing on the market of glufosinate-tolerant, genetically modified rape (Brassica napus): Statement of the competent authority of the Federal Republic of Germany, 25 October 1996.
30. Pesticides Trust, 1997. *Crops Resistant to Glutamine Synthetase Inhibitors*. Pesticides Trust, London.
31. MAFF, Health and Safety Executive, 1991. Op. Cit. 13.
32. Cox, C. , 1996. Op. Cit. 12.
33. Ismail BS, and A.R. Ahmed, 1994. *Attenuation of the herbicidal activities of glufosinate-ammonium and imazapyr in 2 soils*. Agric. Ecosystems and Environ., Vol. 47, No. 4, pp. 279-285.
34. Behrendt, H. and M. Matthies, 1990. *Leaching and transformation of glufosinate ammonium and its main metabolite in a layered soil column*. Environ. Toxicol. and Chemistry, Vol. 9, pp. 541-549.
35. Ismail BS, and A.R. Ahmed, 1994. Op. Cit. 23.
36. Allenking, R.M., B.J. Butler. And B. Reichert, 1995. *Fate of the herbicide glufosinate-ammonium in the sandy, low organic-carbon aquifer at CFB Borden, Ontario, Canada*. J. of Contaminant Hydrology, Vol. 18, No. 2, pp. 161-179.
37. US EPA, 1988. *Glufosinate Ammonium: Review and assessment of individual studies and environmental fate assessment*. Submitted by Dynamac Corp. (Sept 8). Cited in Cox, C. 1996. Op. Cit. 12.
38. Allenking, R.M., B.J. Butler. And B. Reichert, 1995. Op. Cit. 26.
39. Gallina, M.A. and G.R. Stevenson, 1992. *Dissipation of [c-14] glufosinate ammonium in 2 Ontario soils.* J. of Agric. And Food Chem., Vol. 40, No. 1., pp. 165-168.
40. Behrendt, H. and M. Matthies, 1990. Op. Cit. 34.
41. Meyer, H., and V. Wolkers, (unpublished) *Herbicides Containing glufosinate-ammonium and their effect on micro-organisms and animals in both terrestrial and aquatic eco-systems*. (Language: German).
42. Bartsch, K. and C.C. Tebbe, 1989. *Initial steps in the degradation of phosphinothricin (glufosinate) by soil bacteria*. Appl. And Environ. Microb., Vol. 55, No. 3, pp. 711-716.
43. Quinn, J.P., J.K. Heron, and G. McMullan, 1993. *Glufosinate tolerance and utilisation by soil and aquatic Bacteria. Biol. And Environ*.-Proceedings of the Royal Irish Academy, Vol. 93B, No. 3, pp. 181-186.
44. Ahmed, I., and D. Malloch, 1995. *Interaction of soil microflora with the bioherbicide phosphinothricin*. Agric. Ecosystems and Environ. Vol. 54, No. 3, pp. 165-174.
45. Cox, C., 1996. Op. Cit. 12.
46. MAFF, 1990. Op. Cit. 14????.
47. MAFF, 1990. Ibid.
48. Meyer, H., and V. Wolkers, (unpublished) Op cit 41
49. Kriete, G., and I. Broer, 1996. *….nodulation capacity of Rhizobium meliloti.* Appl. Microbiology and Biotechnology, Vol. 46, No. 5-6, pp. 580-586.

50. Ismail, B.S. and L.K. Wong, 1994. *Effects of herbicides on cellulolytic activity in peat soil. Microbios.* , Vol. 28, No. 315, pp. 117-123.
51. Ahmed, I., and D. Malloch, 1995. Op. Cit. 44.
52. Ahmed, I., J. Bisset, and D. Malloch, 1995. *Influence of the bioherbicide phosphinothricin on interactions between phytopathogens and their antagonists.* Canadian J. of Botany-Revue Canadienne de Botanique, Vol. 73., No. 11, pp. 1750-1760.
53. Malkomes, H.P., 1988. *Einfluss von Glufosinat-ammonium (Basta) und Glyphosat (Roundup) auf Bodenmikroorganismen und deren Aktivitaten. Zeitschrift Pflanzenkrankheiten Pflanzenschutz, Sonderheft* 11, pp. 277-286. Cited in Meyer, H., and V. Wolkers, (unpublished) *Herbicides containing glufosinate-ammonium and their effect on micro-organisms and animals in both terrestrial and aquatic eco-systems*. (Language: German).
54. OECD, 1994. *Field Releases of Transgenic Plants, 1986-1992.* OECD, Paris. Cited in *Agrow* 202, February 18th 1994, p18.
55. Gray, AJ and AF Raybould, 1998. *Environmental Risks of Herbicide Tolerant oilseed rape: a review of the PGS hybrid oilseed rape* Report for the Advisory Committee on releases to the Environment December 1998
56.Simpson, EC, Norris CE, Law JR, Thomas JE and Sweet JB. 1999. *Gene flow in genetically modified herbicide tolerant oilseed rape (Brassica napus) in the UK* in *Gene Flow and Agriculture: Relevance for Transgenic Crops* PJW Lutman (ed). British Crop Protection Council Symposium Proceedings No 72 pp 75-81
57. M.J. Wilkinson, Timmons, A.M., Charters, Y., Dubbels, S., Robertson, A., Wilson, N., Scott, S., O'Brien, E. and Lawson, H.M., 1995. *Problems of Risk Assessment with Genetically Modified Oilseed Rape.* In *Brighton Crop Protection Conference, Weeds, 1995.* British Crop Protection Council, Farnham, Surrey.
58. Thompson, CE, Squire G, Mackay GR, Bradshaw JE, Crawford J and Ramsay G. R. 1999. *Regional patterns of gene flow and its consequence for GM oilseed rape* in *Gene Flow and Agriculture: Relevance for Transgenic Crops* PJW Lutman (ed). British Crop Protection Council Symposium Proceedings No 72 pp 95-100
59. Mikkelsen, T.R., Andersen, B. and Jorgensen, R.B., 1996. The risk of crop transgene spread. *Nature* 380, 31.
60.Gray, AJ and AF Raybould, 1998. Op cit 55
61. Darmency, H., Fleury A. and Lefol, E. , 1995. *Effect of transgenic release on weed biodiversity: oilseed rape and wild radish.* In *Brighton Crop Protection Conference, Weeds, 1995.* British Crop Protection Council, Farnham, Surrey.
62. Lefol E., Danielou, V., Darmency, H., Boucher, F. Maillet, and Renard, M., 1995. Gene Dispersal from Transgenic crops. I. Growth of interspecific hybrids between oilseed rape and the wild hoary mustard. *Journal of Applied Ecology* 32, 803-808.
63.Vigouroux, Y, Darmency H, Gestat de Garambe T and Richard-Molard M. 1999. *Gene flow between sugar beet and weed beet* in *Gene Flow and Agriculture: Relevance for Transgenic Crops* PJW Lutman (ed). British Crop Protection Council Symposium Proceedings No 72 pp 83-88
64. The Pesticides Trust, 1991. *The Use of Herbicide on Crops Resistant to Glutamine Synthetase Inhibitors*. Pesticides Trust, London.
65. USDA/APHIS, 1994. *Response to Monsanto petition P93-258-01 for the determination of non-regulated status for glyphosate-tolerant soybean line 40-3-2.* United States Department of Agriculture, Animals and Plant Health Inspection Service, Biotechnology, Biologics, and Environmental Protection. Cited in Pesticides Trust, 1997. *The Price of Resistance. The consequences of Roundup Ready Soya Beans.* Pesticides Trust, London.

66. Norman, M.J.T., Pearson, C.J. and Searle, P.G.E., 1995. *The ecology of tropical food crops.* Cambridge University Press, Cambridge, UK. Cited in Pesticides Trust, 1997. *The Price of Resistance. The consequences of Roundup Ready Soya Beans.* Pesticides Trust, London.
67. Heap, I.M., 1997. The occurrence of herbicide-resistant weeds worldwide. *Pestic. Sci.* 51, 235-243.
68. Ridley, SM and McNally SF. 1985. Effects of phosphinothricin on the isoenzymes of glutamine synthetase isolated from plant species which exhibit varying degrees of susceptibility to the herbicide *Plant Science* 39 31-36
69.Steckel GJ, Wax LM, William Simmons F, Phillips WH. 1997. Glufosinate efficacy on annual weeds is influenced by rate and growth stage *Weed Technology* 11 484-488
70. Hoechst, undated. *Basta. The new non-selective herbicide. Tested worldwide - effective worldwide*
71. Blackshaw , R.E. and Lindwall, C.W., 1995. Management-systems for conservation fallow on the southern Canadian prairies. *Canadian J. of Soil Sci.*75(1), 93-99.
72. RIVM/RIZA, 1991. *Sustainable Use of Groundwater: problems and threats in the European Communities.* Report No. 600025001. RIVM/RIZA, Netherlands.
73. ENDS Daily, 20 February 1997.
74. Pesticides Safety Directorate, 15/12/1998. *Scientific Review of the Impact of Herbicide Use on Genetically Modified Crops*
75. English Nature Press Release - 8 July 1998. *Government Wildlife Advisor urges caution on genetically modified organisms - the new agricultural revolution*
76. Dr M Smith, Director, Policy and Science, Countryside Council for Wales. 15/12/1997. Pers comm.
77. Scottish Natural Heritage, July 1997. *Management of genetically modified herbicide tolerant crops*
78. RSPB, 10/9/1997. Comments on *Weed control on the farm: management of genetically modified herbicide tolerant crops*