GLUFOSINATE - AMMONIUM MONOGRAPH

SUMMARY

Background

Glufosinate-ammonium is a broad-spectrum herbicide, in use in relatively small quantities since the 1980s. However the recent development of more than 100 varieties of transgenic plants, genetically modified to be tolerant of the herbicide, is significantly increasing its use.

It carries unacceptable risks to humans, especially to the neurological development of the foetus, to agricultural biodiversity, and to the environment.

Operator exposure during crop spraying is unacceptably high even when protective clothing is worn.

Formulations are more toxic to humans and the aquatic environment than the active ingredient alone, but little information is publicly available on the inert, or adjuvant, ingredients in the formulated products.

Overall Risk

Risk statements from the European Food Safety Authority (EFSA 2005), based on European use conditions include:

- high risk to mammals
- low risk to aquatic organisms
- low risk to in-field non-target arthropods in potato and transgenic maize crops, but high risk to off-crop populations, requiring risk mitigation such as 5 meter buffer zones
- risk to non-target plants from use in orchards and maize is low if a 5 meter buffer zone is used
- low risk to birds, bees, earthworms, other soil non-target macro-organisms, and soil micro-organisms.

KEMI (2002a, 2002b), the Swedish National Chemicals Inspectorate, had proposed the following risk statements:

- harmful: danger of serious damage to health by prolonged exposure if swallowed
- toxic: danger of serious damage to health by prolonged exposure through inhalation
- may cause harm to the unborn child
- possible risk of impaired fertility
- danger to the environment
- toxic to aquatic organisms, may cause long-term adverse effects in the aquatic environment
- wear suitable protective clothing
- during spraying wear suitable respiratory equipment.

Poisonings

A number of poisonings have resulted from intentional ingestion of glufosinate-ammonium, mainly in Japan.

Acute Toxicity

Acute toxicity effects are firstly gastrointestinal such as nausea, vomiting, abdominal pain and diarrhoea, followed by the onset of neurological symptoms such as convulsions and coma, then respiratory failure; death results from circulatory failure. There is no antidote.

Long-term Toxicity

Chronic effects are primarily neurological and reproductive.

Glufosinate-ammonium is structurally similar to a neurotransmitter, glutamate, and interferes with its proper functioning. The developing

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brain is particularly susceptible: the herbicide has serious effects on early embryonic development, including damage to the brain and neural tube. It causes the loss of many foetuses and damage to those actually born, including cleft lips. Transgenerational effects on brain function are reported.

Environmental Effects

It is moderately persistent in some soils, and has the potential to leach to groundwater, especially in sandy soils.

Despite being a herbicide it also has insecticidal properties. It is highly toxic to beneficial organisms including spiders, predatory mites and butterflies.

It is toxic to a number of soil micro-organisms, and may increase susceptibility to plant diseases, with consequent increased use of and dependence on pesticides. Long-term use is likely to give rise to herbicide-resistant weeds. Glufosinate tolerance transgenes have already escaped from GM plants and been found in weedy relatives in Japan and the UK.

There are indications of synergistic interactions with other herbicides, such as with metolachlor to cause damage to testes, and with metsulfuron-methyl to increase phytotoxicity.

Chemical Profile

Common name
Glufosinate-ammonium

Common trade names
Basta, Liberty

Other related chemicals
Glufosinate, phosphinothricin

Chemical names
- butanoic acid, 2-amino-4-(hydroxy-methylphosphinyl)-monoammonium salt
- ammonium-DL-homoalanin-4-yl (methyl)phosphinate (KEMI 2002a)

Molecular formula
$C_5H_{15}N_2O_4P$

Chemical group
phosphinic acid, ammonium salt

CAS number
7718-82-2

Trade names include
Aeh, Basta, Buster, Challenge, Conquest, Dash, Derringer, Finale, Harvest, HOE 00661, HOE 039866, Ignite, Liberty, Rely, Remove, Tepat.

Inerts
All formulations contain so-called 'inert' ingredients (adjuvants), but they are largely unidentified. Two have been at least partially identified for glufosinate-ammonium formulations. KEMI (2002a) partially identified a surfactant in Basta as alkylethersulfate (AES) and Koyama et al (1997) identified it as sodium polyoxyethylene alkyl ether sulphate. "Genapol' is a solvent in Basta and Liberty and possibly other formulations (KEMI 2002a,c).

Material Safety Data Sheets (MSDS) for some formulations name one inert, as follows:
- *Finale, Basta*: propylene glycol monomethyl ether
- *Liberty*: 1-methoxy-2-propanol (which is the same chemical as in Finale and Basta)
- *Rely*: alkyl hydroxy-poly(oxyethylene) sulfate-salts

(Bayer CropScience undated a,b, 2003a,b, 2006).

Genapol is described as a polyglycol ether and there are a number of versions of it. The OXD-080 version is reported to cause damage to mosquito fish (*Gambusia*) at low concentrations; and can be expected to cause significant population decline of the fish when added to rice irrigation ditches even at very low levels (Cabral et al 2001). Another version, used in the administration of insulin is suspected of causing auto-immune thyroiditis in diabetic patients (Chantelau 2000). Which version(s) of Genapol is/are used in glufosinate formulations and its/their effects on human health and the environment is not publicly known.

Human poisonings and laboratory studies indicate that the 'inert' ingredients, particularly the surfactants, may be more toxic than the glufosinate-ammonium itself. For example the

formulation Basta reduced blood pressure and altered the heart rate in rats, but glufosinate ammonium alone did not (Koyama et al 1997).

Another herbicide, metsulfuron-methyl, has a synergistic effect on glufosinate-ammonium in terms of increasing its phytotoxicity, but the effect is greater with formulations than the active alone (Kudsk & Mathiassen 2004). Glufosinate formulations are also more toxic to aquatic life than the active ingredient (KEMI 2002a).

Metabolites

In soil glufosinate-ammonium is degraded by microbial action to 3-methylphosphinico-propionic acid (MPP) and 2-methylphosphinico-acetic acid (MPA), and eventually to carbon dioxide under dark aerobic conditions (HSD 2003; EFSA 2005).

Another metabolite, disodium L-2-acetamido-4-methylphosphinato-butyrate or N-acetyl-glufosinate (NAG), is found only in transgenic plants treated with glufosinate-ammonium and not in normal plants, as transgenic plants metabolise glufosinate-ammonium differently. MPP is found in both types of crop (KEMI 2002a). An additional unidentified metabolite has been found in a crop planted following the use of glufosinate (EFSA 2005).

In humans and animals the principle metabolites in urine and faeces are MPP and NAG. Minor metabolites are MPA, 2-hydroxy-4-methylphosphinico-butanoic acid (MHB), and 4-methylphosphinico-butanoic acid (MHB) (HSD 2003; EFSA 2005).

In water (and in anaerobic conditions) the metabolites are MPP, MPA, 3-methylphosphinico-acrylic acid (P-X), and methylphosphinico-formic acid (P-Y) (EFSA 2005).

The metabolites MPP, MPA and NAG are all less toxic than the parent compound according to the European Food Safety Authority (EFSA 2005). However MPP is also reported to be neurotoxic, causing severe convulsions and forelimb spasms (Cox 1996 citing US EPA 1988a). It is more persistent than the parent chemical, persisting up to 267 days, and is more mobile in the soil (Cox 1996 citing US EPA undated; EFSA 2005).

Mode of Action

Glufosinate-ammonium is a phosphorus-containing amino acid derived from a natural toxin isolated from two species of Streptomyces fungi (Jewell & Buffin 2001).

It acts by inhibiting the enzyme glutamine synthetase in plants, leading to a complete breakdown of ammonia metabolism and consequent inhibition of photosynthesis (Kidd & James 1991; HSD 2003). Leaf chlorosis and necrosis follow and the plant dies in 1-2 weeks (KEMI 2002a).

Glutamine synthetase is also metabolically important in animals and humans. The herbicide has inhibited this enzyme in the brain, kidney and liver of rats, and in the heart of dogs. It has also inhibited the related enzymes glutamate carboxylase in rat brains and glutamate dehydrogenase in cow liver (Cox 1996 citing FAO, WHO & IPCS 1992). Inhibition of glutamate synthetase has also been reported in humans (HSD 2003).

Uses

Glufosinate-ammonium is a non-selective contact herbicide with systemic action. It is translocated only within leaves, not within underground rhizomes or stolons (Kidd & James 1991).

It is used on a wide range of annual and perennial broad-leaved weeds and grasses in orchards, vineyards, rubber and oil palm plantations, cotton, soy, ornamental trees, non-crop land, home gardens, greenhouses, nurseries, and pre-emergence in vegetables (Kidd & James 1991; Orme & Kegley 2006).

It is used as a pre-harvest desiccant on a variety of crops including potatoes, peas, beans and cereals (PAN UK 1998).

It is also used in increasingly greater quantities as a selective herbicide in genetically modified, or transgenic, crops.

GM Uses

More than 100 plant hybrids have been genetically modified to be tolerant of, or resistant to, the herbicide glufosinate-ammonium (Bayer CropScience 2007). They include varieties of banana, bean, cabbage, canola (InVigor), cauliflower, coffee, cotton

(LibertyLink, WideStrike), maize (Herculex, Quad-Stack), mustard, oil palm, pineapple, plantain, potato, rapeseed (InVigor, Topas), rice (LibertyLink), soybean, strawberry, sugarbeet, sugarcane, and wheat (FAO 2007).

The status of GM glufosinate-tolerant crops in the Asia Pacific region (FAO 2007):

India: mustard – field trials
brassical mustard – experimental phase
cauliflower – experimental phase
rape seed – field trials
rice – experimental phase
Malaysia: oil palm – experimental phase
Viet Nam: cabbage – experimental phase
cotton – experimental phase
maize – experimental phase
rice – experimental phase
sugarcane – experimental phase

Sweet potato may also be under development in Korea (Yi et al 2007), and *Acacia sunuata* (Vengadesan et al 2006) and mungbean (Sonia et al 2007) in India.

The genetic modification generally involves incorporation of a bacterial gene that codes for the enzyme phosphinothricin acetyl transferase (PAT). This enzyme inactivates glufosinate-ammonium by adding an acetyl group to it, and so confers herbicide tolerance to the modified plant (OECD 2002).

Manufacturers

Bayer CropScience is the major producer of glufosinate-ammonium (KEMI 2002a). The name of the company manufacturing the herbicide has changed as companies have merged and split: it was manufactured formerly by Hoechst, then AgroEvo, then Aventis CropScience, now Bayer CropScience. Glufosinate is manufactured at Bayer plants in Frankfurt, Germany and Muskegon in Michigan, USA.

In China, Zhejiang Yongnong Chem. Ind. Co., LTD is also producing the herbicide. Three other Chinese companies have been approved to produce it so it is expected that the production and use in China will increase rapidly in the future (Sun Jing 2007).

Bayer sales of glufosinate for 2005 were EUR 219 million, its third highest grossing product (Bayer 2007).

Regulatory status

Glufosinate-ammonium was developed by Hoechst in the 1970s and first introduced to the market in 1984, in Japan. It was also considered for registration in the UK in 1984 but, because of concerns about toxicity via dermal absorption, it did not win full approval there until 1996 (Jewell & Buffin 2001). It was introduced in the USA in 1993. By 2004 it was registered in over 80 countries (Bayer CropScience 2007b).

Glufosinate-ammonium is not banned or restricted in any country, nor is it a PIC pesticide.

Toxicological Assessment

Absorption

Glufosinate-ammonium is rapidly but poorly absorbed from ingestion: only 5-17 percent was absorbed in laboratory animals. It has low distribution to tissues and does not appear to concentrate, although both glufosinate and MPP were found in brain tissue. Most of it is excreted via the faeces (KEMI 2002a).

Acute toxicity

WHO Recommended Classification by Acute Hazard: Class III, slightly hazardous.

Lethal doses

Lethal dose, LD_{50}, is the dose that kills 50 percent of test animals:

- Oral LD_{50}
  - = *medium to moderate: 1510mg/kg* (rat) (KEMI 2002a). Also reported as 1625mg/kg (WHO 2005); 2000mg/kg (male rat), 1620mg/kg (female rat), 431mg/kg (male mouse), 416mg/kg (female mouse), 200-400mg/kg (dog) (Kidd & James 1991); and as 4010mg/kg (male rat) and 3,030mg/kg (female rat) (US EPA 2003).
- Dermal LD_{50}
  - = *low:* >4000 mg/kg (male rat); >2000mg/kg (male rabbit), 1500-2000mg/kg (female rabbit) (KEMI 2002a).

- Inhalation LC_{50} = *medium to moderate*: 1.26mg/l air (aerosol)/4hr (rat) (KEMI 2002a).

It is reported that, when absorbed through the skin, glufosinate formulations can be 2.5 times more toxic than glufosinate alone (PAN UK 1998 citing an internal US EPA 1985 memo).

Acute effects

Following oral, dermal and inhalation exposure, laboratory animals suffered a number of acute symptoms indicative of effects on the nervous system—including lethargy, abnormal postures, piloerection ('goose bumps'), hypersensitivity to touch, increased motor activity, tremors, convulsions, seizures, irregular respiration, salivation, diarrhoea, decreased spontaneous motility, disequilibrium, hyperactivity, aggressiveness, ataxia (unsteady motion), hyporeflexia (below normal reflexes), and a marked decrease in body weight (Masumara et al 2001; Lapouble et al 2002; KEMI 2002a).

Skin and eye irritation

The European Food Safety Authority characterised glufosinate-ammonium as being mildly irritating to eyes, but not to skin and showing no sensitising properties (EFSA 2005). However, formulations containing glufosinate-ammonium can cause irritation to both skin and eyes, resulting in redness, conjunctival discharge, and opaqueness of the cornea; and redness, swelling and dry or chapped skin, in rabbits (Cox 1996 citing US EPA 1986a).

No Observed Adverse Effects Levels

- Short term NOAEL (oral) = 4.5mg/kg of body weight (bw)/day from a 1-year study on dogs, based on mortality and decreased glutamine synthetase activity (EFSA 2005).

- Dermal NOAEL = 100mg/kg bw/day (male rat), 300mg/kg bw/day (female rat) (EFSA 2005).

- NOAEC = 0.012mg/litre air (concentration in air) (EFSA 2005).

- NOAEL (reproduction) = 4.3mg/kg bw/day based on high losses of pre- and post- implantation rat pups (EFSA 2005).

- NOAEL (development) = 6.3mg/kg bw/day in rabbits, based on premature deliveries, abortions and dead foetuses (EFSA 2005).

Sub-chronic toxicity

Sub-chronic effects observed in dogs include a dose-related decrease in heart rate; and in rats decreased body weight gain, decreased white blood cell count, increased kidney weight, slow blood coagulation, increased aggressive behaviour, and increased weight of the adrenal glands (Cox 1996 citing FAO, WHO, & IPCS 1992, and US EPA 1984,1988b.c).

Chronic toxicity

Cardiovascular effects

Two dogs fed 8mg/kg bw/day for 10 and 14 days died of heart and circulatory system failure (Cox 1996 citing US EPA 1988c).

The formulation Basta, and its surfactant alone, both caused decreased blood pressure, increased heart rate at low doses, and decreased heart rate at very high doses, in rats; but the active ingredient glufosinate-ammonium by itself did not cause these effects (Koyama et al 1997).

Immune system

No effects appear to have been reported.

Endocrine disruption

No effects appear to have been reported.

Nervous system

Glufosinate-ammonium is structurally similar to glutamate (Hack et al 1994), an amino acid important to proper functioning of the nervous system, and the herbicide causes signs of neurotoxicity in most species of animals on which it has been tested (Cox 1996). Glutamate is an important neurotransmitter, especially in brain function, and the herbicide causes activation of the neurotransmitter receptors for glutamate (Matsumara et al 2001). It also stimulates production of nitric acid in the brain, through stimulation of N-methyl D-aspartate receptors (Nakaki et

al 2000). The increased production of nitric acid is held to be responsible for the epileptic (tonic-clonic) seizures resulting from exposure to glufosinate-ammonium (Lapouble et al 2002).

Developing brains appear to be particularly susceptible to glufosinate-ammonium. Exposure of the foetal brain of rats (through maternal exposure) resulted in functional abnormalities in the offspring (Fujii 1997).

Glufosinate specifically affects the neuroepithelium of the brain vesicle and neural tube, leading to neuroepithelial cell death and deformities. It has caused underdevelopment of the forebrain, blisters in the head, cleft lips, and inhibited the differentiation of midbrain cells in rats (Watanabe & Iwase 1996; Watanabe 1997).

There is no readily available information on delayed neurotoxicity; because glufosinate-ammonium is not an organophosphate, regulatory authorities do not generally require delayed neurotoxicity studies (KEMI 2002c).

Reproductive & developmental effects

Glufosinate has toxic effects on early embryonic development. Studies on rats, mice and rabbits have found the following:

- increased pre- and post-implantation losses
- vaginal haemorrhage
- intra-uterine death of foetus
- resorptions and abortions
- premature deliveries
- decreased litter size (KEMI 2002a).

In the pups that were born there was distension of the renal pelvis and ureter, and retardation of skeletal ossification (Watanabe & Iwase 1996; KEMI 2002a).

Glufosinate administered to the mother was measured in the foetus indicating that it crosses the placenta (KEMI 2002a).

KEMI (2002a) concluded that glufosinate is toxic to reproduction and the product should carry risk warnings that it “may cause harm to the unborn child”, and “possible risk of impaired fertility”.

Glufosinate-ammonium was found to be teratogenic (causes birth defects) in a study on rats (Watanabe & Iwase 1996; Watanabe 1997), and it caused transgenerational effects on brain function (Fujii 1997)—as reported above under neurological effects.

Genotoxicity / mutagenicity

No evidence of genotoxicity appears to have been reported (KEMI 2002a; US EPA 2003).

Cancer

KEMI (2002a) reported no evidence of carcinogenicity in the studies it reviewed. Glufosinate-ammonium has not been assessed for carcinogenicity by IARC or US EPA. One study, described by the US EPA as inadequate because it did not use sufficient doses, found an increased frequency of adrenal medullary tumours (Cox 1996 citing US EPA 1992a).

Toxic interactions

When glufosinate was combined with another herbicide, metolachlor, for a 4-hour inhalation study with rats, it produced atrophied and withdrawn testes, an effect that appears to be related to interaction between the two herbicides (Cox 1996 reporting on US EPA 1986b).

Gender differences

It is more acutely toxic to the females than the males of all species tested, although dermal NOAEL was higher for female than male rats.

Sensitive populations

The foetus.

Health Effects and Poisonings

Exposure

Exposure guidelines

- ADI (acceptable daily intake) set by the WHO/FAO = 0.02mg/kg bw/day (IPCS 2003).

- ADI set by the European Food Safety Authority = 0.021mg/kg bw/day, based on the NOEL (no observed effects levels) of 6.3mg/kg bw/day in rabbits (EFSA 2005).

- AOEL (Acceptable operator exposure level) = 0.0021mg/kg bw/day, based on

the NOEL for rabbits and a correction for 10per cent oral absorption (EFSA 2005).

- ARfD (Acute reference dose) = 0.021mg/kg bw/day for women of child-bearing potential, based on reproductive effects; and 0.045mg/kg bw/day for the general population (EFSA 2005).

The Australian label for the formulation Finale carries the following warning regarding stock withholding period: "do not graze or cut treated area for stock feed for 8 weeks after application" (Bayer CropScience undated a).

Occupational exposure

Exposure can occur through inhalation of spray or dust, and dermal contact, where glufosinate is used or manufactured.

The European Food Safety Authority (2005) has judged that operator exposure exceeds the AOEL when Basta SL14/SL18 is used for potato desiccation and when Liberty SL 18 is used on transgenic maize—even when personal protective equipment, including gloves, headwear, coverall and sturdy footwear (KEMI 2005), is worn. Exposure was regarded as unacceptable. However exposure in apple orchards at approved rates of use was regarded as acceptable.

Non-occupational – food residues

Residues in food are of concern, especially when glufosinate is used as a pre-harvest desiccant—such as on dried peas, field beans, barley, oilseed rape, linseed and potatoes—and in the liver and kidneys of animals fed cereal straw or pea and bean stalks that have been treated with glufosinate (PAN UK 1998 citing MAFF 1990).

When wheat grain containing residues was turned into flour, 10-100per cent of the residue was retained. Residue levels in bran were 10-600per cent of those in grain (PAN UK 1998 citing MAFF 1990).

Significant residues of glufosinate have been found in the UK in animal feed, potatoes and other crops. Additionally residues of an 'inert' ingredient and the metabolite, MPP, were found in milk and the tissues of animals fed 'treated' straw, resulting in a restriction on the feeding of 'treated' straw to reduce health risks to livestock and consumer intake of residues in animal products (PAN UK 1998 citing MAFF 1990).

The European Food Safety Authority (EFSA 2005) found that residues of glufosinate in potatoes can be up to 0.5mg/kg and are not altered by cooking in boiling water. They concluded that these residues pose an acute risk for small children, as they were 114per cent of the acute reference dose (EFSA 2005).

Residues are also stable in frozen food for 1 year in potatoes, 2 years in apples and transgenic maize. They are also stable in poultry and cattle for up to 15 months (KEMI 2002a).

Glufosinate has been found in spinach, radish, wheat and carrots planted 120 days after the soil was treated with a glufosinate herbicide (Cox 1996 citing US EPA 1988d), but KEMI (2002a) concluded that the potential for residues of glufosinate or its metabolites in soil and subsequent crops is low.

The main residue found when glufosinate is used as a non-selective herbicide is MPP; when it is used as a desiccant it is glufosinate itself, and on transgenic maize it is NAG. The other metabolites may also occur as residues in lesser amounts (KEMI 2002a).

According to Jewell & Buffin (2001), data from Aventis indicates that the metabolite NAG, formed in transgenic plants, can be reconverted into the active herbicidal form by micro-organisms in the digestive tract of warm-blooded animals, including humans.

Absorption

Absorption is described by the EFSA (2005) as poor. It reports dermal absorption of the formulations Basta SL14 and Basta SL18/Liberty SL18 as 16per cent and 7per cent for undiluted product and 14per cent and 9per cent for spraying dilution.

Treatment

People who have been poisoned should be immediately taken to hospital and placed under surveillance and treatment by properly trained medical staff. Do not induce vomiting. For eye exposure irrigate eye with copious amounts of room temperature water for at least 51 minutes (HSD 2003).

Treatment is symptomatic and supportive; there is no known antidote (KEMI 2002a).

Symptoms and consequences of poisonings

Most clinical experience of glufosinate poisoning comes from intentional ingestion in Japan (KEMI 2002a).

Neurotoxicity is characteristic of glufosinate-ammonium poisoning (Watanabe & Sano 1998). Acute effects such as convulsions appear to be mediated by activation of the glutamate neurotransmitter receptors by the herbicide (Matsumara et al 2001).

Acute effects

The first effects are nausea, vomiting, abdominal pain, and diarrhoea shortly after acute exposure (oral). This is followed by a short asymptomatic period, and then by neurological symptoms including loss of consciousness, coma, and seizures, and/or respiratory failure which may develop suddenly 8-24 hours after ingestion (HSD 2003).

Other reported acute effects include fever, disturbances of speech and eye movements, loss of short-term memory, general oedema (swelling resulting from accumulation of lymph in tissues and organs), systemic tremor, and erosion of gastric membranes (HSD 2003).

A case of transient diabetes following ingestion of glufosinate has been reported (Takahashi et al 2000).

Clinical signs include elevated white blood cell count and liver enzymes, lowered blood pressure, metabolic acidosis (low blood pH), depressed serum cholinesterase, and inhibition of the enzyme glutamine synthetase (HSD 2003).

Death results from circulatory failure usually, 1-3 days after ingestion. It has resulted from doses as low as 3.7 grams, but others have survived doses as high as 92.5 grams (HSD 2003).

The acute oral toxic dose for humans has been identified as 1.6-1.8ml/kg body weight of Basta formulation, or 298-333mg/kg of glufosinate ammonium (KEMI 2002c).

Long-term effects

An epidemiological study of parental occupational exposure to pesticides in Spain found possible risk of congenital malformations from parental exposure to glufosinate (Garcia et al 1998).

Cases of Poisonings

KEMI (2002c) stated that all known cases of poisoning (at that time) arose from intentional oral poisonings, and a 'few' incidences of accidental misuse. About 230 cases of poisoning are known to have occurred between 1985 and mid-1999, almost all in Japan, with 9 in France. Mortality from all reported cases was 19.4per cent (KEMI 2002c). Poisoning resulted from both the active ingredient and the surfactant.

Environmental Effects

Aquatic toxicity

For a number of aquatic organisms, such as fish, water fleas and oysters, formulated products containing glufosinate are more toxic than the active ingredient alone, indicating the presence of a toxic surfactant or other 'inert' ingredient. KEMI (2002a) described toxicity as 'moderate' for most aquatic species.

Freshwater fish

LC_{50} Rainbow trout = 580mg/l (96 hours) (Kidd & James 1991)

LC_{50} Bluegill, Carp, Golden orfe = >1000mg/l (98 hrs) (HSD 2003)

Freshwater invertebrates

Daphnia (water flea):

- EC_{50} (48hrs) = 668mg/l (15-18mg/l for Basta and Liberty)
- NOEC (no observed effects concentration) (21 days) = 18mg/l

Mysid shrimp:

- EC_{50} (48hrs) = 7.5mg/l
- NOEC (21 days) = 0.75mg/l

Estuarine/marine

Clams: the Ignite formulation is highly toxic to clam larvae (Cox 1996 citing US EPA 1993).

Oysters: glufosinate is acutely toxic to embryos and larvae, and at least one formulated product is more so (Cox 1996

citing US EPA 1990a,b).

Shrimp: 1ppm glufosinate caused loss of equilibrium, and at higher concentrations (7.5ppm) death of half the shrimps tested (Cox 1996 citing US EPA 1990c).

Aquatic plants

Green algae:

- EC_{50} (72 hrs) = 780mg/l [36mg/l for formulated product]
- NOEC (72hrs) = 2.5mg/l

Lemna giba (duckweed):

- EC_{50} (14 days) = 1.5mg/l [10mg/l for formulated product]
- NOEC (14 days) = 0.8mg/l [3.2 mg/l for formulated product]

Terrestrial ecotoxicity

Birds

Acute $LD_{50:}$ for 4 species = > 2000mg/kg bw
Dietary: bobwhite quail = 1100mg/kg bw/day
Reproductive toxicity: bobwhite quail = 40mg/kg bw/day (KEMI 2002a).

At lower levels of exposure effects on reproduction occur, principally fewer eggs are laid (Cox 1996 citing Roberts et al 1981).

Effects noted in 4 day-old partridges given a dose of 2,000 mg/kg of 96per cent glufosinate showed signs of central nervous system damage including ataxia, disequilibrium, convulsions, trembling, and wing flapping (PAN UK 1998 citing MAFF 1990).

KEMI (2002a) identified that there may be a long-term risk to bird populations where glufosinate is used in genetically modified crops.

Bees

LD_{50} (96 hrs) is > 345ug/bee by contact and >600ug/bee for inhalation (KEMI 2002a). However the formulation Basta SL14 is more than 10 times more toxic by the oral route than the active ingredient alone; for Basta SL18 and Liberty SL 18 significant effects were seen at 100ug/bee (KEMI 2002a).

Beneficial micro-organisms

Studies of 12 agricultural and 10 forest soils in Canada found that glufosinate selectively reduced the number of fungi and bacteria in soils. In agricultural soils it reduced fungi by 40 percent and bacteria by 20 percent, and in forest soils it reduced bacteria by 20 percent. However the least affected species were plant pathogens such as *Verticillium, Fusarium and Pythium.* The most affected species were beneficial micro-organisms such as *Bacillus subtillus*, *Pseudomonas fluroescens*, and *Trichoderma* species which parasitise disease-causing species. The glufosinate therefore impairs their ability to control disease-causing organisms, for example impairing *Trichoderma*'s control of *Fusarium oxysporum* and *Pythium aphanidermatum* (Ahmad & Malloch 1995; Ahmad et al 1995a, b).

It also significantly inhibits decomposition of cellulose in soils (Ismail & Wong 1994) and is toxic to nitrogen-fixing soil bacteria such as *Rhizobium meliloti* (Kriete & Broer 1996).

Earthworms

Low toxicity: LC_{50} (14 days) >1000 mg/kg dry wgt (KEMI 2002a).

Beneficial insects

Glufosinate-ammonium has insecticidal properties. It is highly toxic to beneficial insects and KEMI (2002a) determined that the risk to them was unacceptable. Recommended field rates caused nearly 100per cent mortality in 5 out of 8 arthropod species tested, with spiders being particularly sensitive. Spider populations in the field took 2-3 months to recover. Even at doses 10 times lower than recommended field rates serious effects were observed on the Linyphiid spider *Pardosa*. Predatory mites were the most sensitive species (KEMI 2002a).

Glufosinate-ammonium was found to be highly toxic to nymphs and adults of three predatory mite species *Amblyseius womersleyi* Schicha, *Phytoseiulus persimilis* Athias-Henriot, and *Tetranychus urticae* Koch. It also caused 71.2per cent mortality to eggs, 65.0per cent mortality to nymphs and 57.7per cent mortality to adults of the predatory insect species *Orius strigicollis* Poppius (Ahn et al 2001).

Glufosinate-ammonium is also toxic to butterfly caterpillars, with an LD_{50} of 400mg/kg for the 5th-instar caterpillars of the skipper butterfly *Calpodes ethlius*. Caterpillars stopped feeding, became dehydrated as a

result of the loss of rectal function, showed symptoms of neurotoxicity, such as tremors in larval 'prolegs' (abdominal structures), body convulsions and complete paralysis before death. The amount of glufosinate-ammonium that caused this effect was described by the authors as "comparable to the amount that might realistically by acquired from feeding on [glufosinate-ammonium]-treated crops" (Kutlesa & Caveney 2001).

Herbicide resistance

There are serious concerns regarding the potential of the gene conferring glufosinate-resistance in transgenic plants to escape from the plant and be transferred to weeds, thus transferring the resistance to weeds. Such gene flow can occur through pollen dispersal or vectors such as viruses and nematodes.

In Japan scientists have already confirmed the presence of glufosinate-resistant transgenes in weedy relatives of oilseed rape (*Brassica napa*). They found the transgene–infected *Brassica rapa* and *Brassica juncea* at five of six ports and along two of four roadsides in the Kanto District. As transgenic oilseed rape had not been grown in Japan, the genetic escape is assumed to have come from transgenic seeds spilled during transportation (Saji et al 2005).

Also in 2005, scientists in the UK found that glufosinate-resistance transgenes had been transferred from an experimental crop of genetically modified oilseed rape to a related weed, charlock (wild mustard). The GM weed was found growing amongst others in a field that had been used to grow the GM rape (Brown 2005).

Environmental fate

Soil persistence

Glufosinate has been found in Canadian soils after 113 days (Cox 1996 citing Smith & Belyk 1989); and in greenhouse soils after 172 days (Cox 1996 citing US EPA 1988d). The half-life in Californian vineyard soils has been reported as 12-70 days, with an average of 40 days (Cox 1996 citing US EPA 1992b). Other reported half-lives vary from 3-20 days, with degradation occurring more rapidly at higher temperatures (KEMI 2002a; HSD 2003). Persistence increases in sandy soils and with low carbon content (Allen-King et al 1995). Degradation is largely microbial (HSD 2003).

However EFSA (2005) described glufosinate as having low persistence, the metabolites MPP and MPA as having low to moderate persistence, and the metabolite NAG formed in transgenic plants as having very low persistence, in soil under aerobic conditions. Degradation is much slower under anaerobic conditions, with the half-life (DT_{50}) calculated to be 388 days (KEMI 2005).

Soil mobility

Laboratory studies indicate glufosinate and its metabolites, especially MPP, are moderate to highly mobile in soils, particularly sandy soils (EFSA 2005). KEMI (2002a) identified a high potential for both glufosinate and its metabolites to leach in sandy soils. However limited field studies have not found it to leach significantly (HSD 2003; KEMI 2002a). Most residues are found in the top 0-30cm of soil, but it has been found down to 150cm (KEMI 2005).

***Groundwater contamination**:* It can contaminate groundwater where there is low clay content and low labile organic carbon (HSD 2003). MPP may contaminate groundwater in vulnerable areas (EFSA 2005).

Water: It is highly soluble in water, and not degraded by photolysis or hydrolysis in water. The half life in water is >300 days; the range of Koc values (coefficient for sorption) indicates it can have low to high absorption to suspended solids and sediment; and so may be persistent in water. It is not likely to volatilise from the surface (HSD 2003). The half-life in water is >300 days; the range of Koc values indicates it can have low to high adsorption to suspended solids and sediment; and it is not likely to volatilise from the surface (HSD 2003). Glufosinate and the metabolite MPP are described by KEMI (2005) as being "ecotoxicologically relevant for surface water and sediments".

The dissipation half-life of glufosinate in water at 20°C is 1.4-13 days, and for MPP it is estimated to be 150 days. The degradation of metabolites MPA and P-Y is slower, and the rates for NAG and P-X are not known (EFSA 2005).

Biodegradation: Glufosinate-ammonium is not readily biodegraded (EFSA 2005).

Air: Glufosinate-ammonium has low volatility and short persistence in the atmosphere so concentrations in air are expected to be negligible (EFSA 2005).

Plants: De Snoo et al (2002) concluded that glufosinate-ammonium can cause adverse effects on off-crop vegetation as a result of drift. In their experiments they found significant phytotoxic effects on non-target vegetation, a decrease of the biomass of the vegetation, and a small decrease in species number and cover, especially for monocotyledons.

The US EPA has identified glufosinate as having "high risk potential to sensitive plants" based on scientific evidence of damage to plants at sublethal levels (Lee et al 2005).

Bioaccumulation: An estimated bioaccumulation factor (BCF) of <3.2 suggests low potential to bioaccumulate in aquatic organisms (HSD 2003). KEMI (2002a) gives the BCF as <1.

Alternatives to Glufosinate

Alternative herbicides

There are many other synthetic chemical herbicides on the market, but these also have a range of adverse health and environmental effects, such as endocrine disruption, cancer, neurological damage, reproductive toxicity, groundwater contamination, persistence, etc. Hence their use is NOT recommended as replacements for glufosinate-ammonium.

There are some herbicides derived from natural plant extracts that can kill or suppress weeds, such as extracts from pine oil and coconut oil. But care must be taken to ensure that formulations do not include toxic surfactants, solvents or other inert or adjuvant ingredients. Some formulations are permitted in certain circumstances in organic growing systems.

However, generally a herbicide, even a natural one, should be regarded as the choice of last resort, with the primary focus being placed on alternative weed management practices that prevent the need for a spray.

Alternative weed management

Alternative weed management focuses on sustainable ecological solutions that minimise the incursion and build up of weeds. It takes a holistic approach to crop management that recognises weeds as an integral part of the whole agroecosystem, forming a complex with beneficial insects, weeds, and crops. The self-regulatory mechanisms of a highly biodiverse farming system help keep both weed and pest species in balance. Although weeds are generally regarded by the modern agricultural institution as reducing crop productivity and encouraging pests and diseases, there are many instances where the reverse is true. Weeds can play a vital role in suppressing pest and disease populations, improving soils and increasing yields.

So-called weeds can provide valuable ground cover, protecting the soil from sun and rain damage and erosion. Balanced weed populations can provide favourable microclimates that assist crop growth. Weed roots can help improve soil biological activity and structure. They can be useful green manures. Weeds can also produce chemicals that are beneficial to crop plants—for example corncockle produces the chemical agrostemmin, which can increase the yield and gluten content of wheat (Lampkin 1990).

Weeds can attract insect pests away from crops and/or provide habitat for beneficial insects that control pest species, for example for ladybirds that control aphids (Lampkin 1990); or the use of Napier grass in East African maize and sorghum systems: the grass produces an odour which attracts stem borer and a sticky substance which kills the larvae (Ho & Ching 2003).

Indian farmer Poorak Kheti, in Mohanpur, Uttah Pradesh uses the weeds baru (*Sorghum halepense),* doob (*Cynodon dactylon*), tipatuiya and motha (*Cyperus rotundus*) to improve soil fertility and the yields of his sugar cane (Scialabba & Hattam 2002).

Weeds can also be very useful as prized herbal remedies or valuable additions to the diet because of their nutritive quality. Plants that are called weeds by some are in fact highly valued plants for others—for example the Napier grass mentioned above as a weed

is highly prized as a source of food and fodder by some African communities and can be a lifeline for them.

Weeds are excellent indicators of problems with soil structure and fertility. Weed species can be read to indicate problems with ph, poor drainage, compaction, low friability of soils and nutrient deficiency (Lampkin 1990). Spraying the weeds with glufosinate-ammonium or any other herbicide will not fix the problem, but solving the soil health problem will control the weed as well as increase productivity and resistance to pests and diseases.

Elements of alternative weed management can include:

- designing a farm ecosystem that encourages biodiversity, providing habitats for beneficial insects, and utilising weeds as an element of useful biodiversity whilst minimising the need for intervention to control them;
- reading weeds to identify soil problems and then making the necessary improvements to soil health (Lampkin 1990);
- timely and appropriate cultivation prior to sowing crop to either bury weed seeds or encourage their germination before crop sowing, although mechanical disturbance of the soil should be minimised in order to protect the soil structure (Lampkin 1990; BIO-GRO 2001);
- sowing of green manures between crops helps weed seed germination, then turning the green manure and weeds in before they flower;
- increasing competitiveness of the crop through appropriate nutrient use and improving soil health;
- controlling the spread of weed seeds through good sanitation practices, such as cleaning machinery, cleaning seeds for saving, careful use of animal manures, good composting practices, and avoiding letting weeds go to seed;
- selection of optimum planting dates with respect to crop choice, lunar cycles and weather patterns (BIO-GRO 2001);
- appropriate mechanical methods and cultural practices such as hand and mechanical weeders, mulches, smothering methods, thermal weed control, solarisation, livestock grazing, rotations, use of under-sowed species (Lampkin 1990; BIO-GRO 2001);
- flame weeders can be used pre-emergence, post-emergence and even as pre-harvest defoliants for crops such as potatoes and onions (Lampkin 1990);
- introducing ducks into rice growing systems to eat weed seeds and seedlings (Ho 1999);
- introducing insect species that provide biological control of weeds.

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Pesticide Action Network Asia and the Pacific (PAN AP) is one of five regional centres of PAN, a global network working to eliminate the human and environmental harm caused by pesticides, and to promote biodiversity-based ecological agriculture.

"Our vision is a society that is truly democratic, equal, just, culturally diverse, and based on food sovereignty, gender justice and environmental sustainability". **Thus PAN AP asserts people's food sovereignty based on the right to food for all, founded on the right to land and productive resources and the right of communities to decide on our own food and agriculture policies. We are committed to protect the safety and health of people and the environment from pesticide use, and genetic engineering in food and agriculture. We strive to protect and promote the rights, equality and dignity of women. We will promote and protect biodiversity based ecological agriculture. Our goal is to strengthen people's movements to eliminate hunger and achieve food sovereignty. We endeavour to achieve these goals by empowering people within effective networks at the Asia and the Pacific, and global levels.**

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