● HERBICIDE FACTSHEET

GLUFOSINATE

Glufosinate is a new broad-spectrum herbicide whose use is expected to increase rapidly in the near future. It kills plants by inhibiting the enzyme glutamine synthetase, an enzyme also found in animals including humans.

Glufosinate chemically resembles glutamine, a molecule used to transmit nerve impulses in the brain. Neurotoxic symptoms observed in laboratory animals following ingestion, dermal exposure, or inhalation of glufosinate include convulsions, diarrhea, aggressiveness, and disequilibrium.

Dogs appear to be the laboratory animal most sensitive to glufosinate. Ingestion of glufosinate for two weeks caused heart and circulatory failure resulting in death.

Exposure of pregnant laboratory animals to glufosinate caused an increase in premature delivery, miscarriages, the number of dead fetuses, and arrested development of fetal kidneys.

Concentrations of a glufosinate-containing herbicide of less than one part per million cause mortality of oyster and clam larvae.

Several species of disease-causing fungi are resistant to glufosinate, while a beneficial fungi that parasitizes disease-causing fungi is very susceptible to glufosinate. This means that use of glufosinate can have "important microbiological consequences."

BY CAROLINE COX

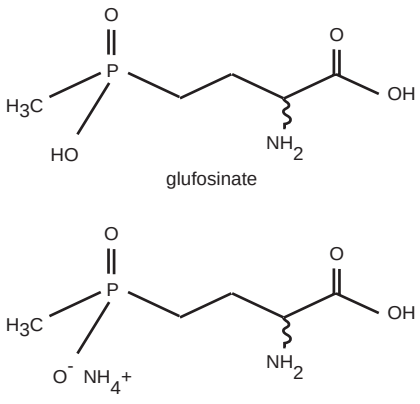
Glufosinate (see Figure 1) is a broad-spectrum herbicide derived from a molecule isolated from two species of *Streptomyces* fungi.[1] The ammonium salt of glufosinate (see Figure 1) was first registered in the U.S. for use as an herbicide in 1993 by Hoechst Celanese.[2] It is currently distributed in the U.S. by AgrEvo USA Company under the brand names Rely and Finale.[3,4]

Use

Glufosinate is currently used to kill unwanted plants in landscape areas where a complete vegetation kill is desired (for example, around the base of shrubs, in sand traps on golf courses, or around fence and sign posts) in industrial, recreational, and public areas such as airports, schools, parking lots, roadsides, and railroad rights-of-way. It is also used as a directed spray (away from crop plants) around ornamental plants and in Christmas tree plantations, fruit and nut orchards, and vineyards.[3,4]

Use of glufosinate is expected to increase dramatically in the next few years because of the development of crop plants that have been genetically engineered to tolerate glufosinate. These crops include corn,[5] soybeans,[6] canola,[7] sugar beets,[8] sugar cane,[9] and sweet potato.[10] Genetically-engineered corn and soybeans appear close to commercial use.

Figure 1
Glufosinate and
Glufosinate-ammonium



glufosinate

glufosinate-ammonium
(monoammonium
2-amino-4-(hydroxymethylphosphinyl)
butanoate)

Note: Glufosinate is sometimes called phosphinothricin.

Mode of Action

Glufosinate inhibits the activity of an enzyme involved in the synthesis of the amino acid glutamine. This enzyme is called glutamine synthetase. Essentially, glufosinate acts enough like glutamate, the molecule used by this enzyme to make glutamine, that it blocks the enzyme's usual function. Glutamine synthetase is also involved in ammonia detoxification. Treatment with glufosinate leads to reduced glutamine and increased ammonia levels in plant tissues. This causes photosynthesis to stop, the plant to wither, and then die within a few days.[1]

Glufosinate inhibits the same enzyme in animals. For example, a single oral dose in mice or rats caused "marked" inhibition of this enzyme in the kidney and liver.[11] In female rats, glutamine synthetase activity decreased in the brain, kidney, and liver at all doses tested.[12] In dogs, a small dose (1 milligram per kilogram (mg/kg) of body weight per day) caused a decrease in the levels of glutamine in the heart. Other related ani-

■

Caroline Cox is JPR's editor.

mal enzymes that are inhibited by glufosinate are glutamate carboxylase in rat brains and glutamate dehydrogenase in cow liver.[11]

Acute Toxicity

The amount of glufosinate required to kill an animal varies widely among species. The oral LD_{50} (the amount that kills 50 percent of a population of test animals) is 1500-2000 mg/kg in rats, but less than 1/3 that amount in mice.[11] Dogs are the most sensitive animal tested: they can be up to twice as susceptible to glufosinate as mice.[13]

The LD_{50} for exposure through the skin is about the same as for oral exposure. Through the skin, glufosinate-containing products can be about 2 1/2 times more toxic than glufosinate itself.[14] Via injection, glufosinate is more toxic (LD_{50} 82-204 mg/kg) than it is through skin exposure.[11]

If humans are as susceptible to glufosinate as dogs, it would require between 0.4 and 0.8 ounces to kill a typical adult (weighing about 60 kilograms).

Neurotoxicity

Although most herbicides are not nerve poisons, glufosinate can affect the nervous system. Glutamate is an "excitatory" neurotransmitter in the brain, and it appears to affect some of the processes in the nervous system that normally involve glutamate.[14]

Signs of neurotoxicity have been seen in most species of laboratory animals that have been exposed to glufosinate or glufosinate-containing herbicides. Symptoms following oral exposure include protruding eyes, convulsions, trembling and irregular respiration in rats,[15,16] convulsions and irregular respiration in mice,[17,18] and trembling, diarrhea, and disequilibrium in dogs.[13]

Following dermal exposure, symptoms include hyperactivity, convulsions, and aggressiveness in rats.[19,20] One study found abnormal behaviors at all doses tested.[21] Following inhalation exposure, symptoms in rats include hyperactivity, disequilibrium, and jerky respiration.[22]

Effects on the nervous system can be relatively persistent. A single oral dose given to female rats caused behavioral impairments for five days, while a larger dose caused more severe symptoms (convulsions and spasms) lasting for three days.[12] In rabbits, a single dose of either 10 or 40 mg/kg of body weight (given intravenously) resulted in abnormal electroencephalograms (EEGs) that persisted for four days.[11] In dogs, a single oral dose entered the brain and could still be detected after four days.[23]

A recent study indicates that developing brains may be particularly susceptible to the neurotoxic effects of glufosinate. Daily exposure of baby rats (one week old) resulted in alterations in one of the nervous system receptors in the brain, called the kainic acid receptor. Effects were measured one month following the glufosinate treatment.[24]

> "Signs of neurotoxicity have been seen in most species of laboratory animals that have been exposed to glufosinate."

Eye and Skin Irritation

Glufosinate-containing products can irritate both eyes and skin. One product, Ignite, is called a "moderate" eye irritant. In all rabbits tested, it caused redness, conjunctival discharge, and opaqueness of the cornea. Eyes returned to normal after seven days.[25] Another product, Ignite 1SC, is classified in the most severe eye irritation category, with discharge and irritation requiring 21 days to return to normal, and opaqueness of the cornea requiring 14 days to return to normal.[26]

On rabbit skin, Ignite (called a "slight" skin irritant) caused redness and swelling with dry or chapped skin lasting 14 days.[25]

Subchronic Toxicity

The toxicity that can occur following medium-length exposure to glufosinate is most dramatically illustrated in a study in which beagle dogs were fed glufosinate. After 10 and 14 days, a male and a female dog receiving the high dose (about 8 mg/kg of body weight per day) died of heart and circulatory system failure. Prior to their death, both dogs showed signs of abnormal behavior, including hyperactivity followed by somnolence, spasms, and stiff gait. In addition, all treated animals showed a dose-related decrease in heart rate after six months of glufosinate ingestion.[27]

Subchronic effects noted in rats include the following: decreased body weight gain and white blood cell count in a 14 day feeding study,[28] an increase in kidney weights in all treated females in a 28 day feeding study,[29] slow blood coagulation in females in a 28 day inhalation study,[29] an increase in aggressive behavior following 28 days of dermal exposure[28] and an increase in the weight of the adrenal glands in all treated males in a 13 week feeding study.[11]

In mice, feeding glufosinate for 13 weeks caused an increase in liver weights in males at the high and the middle dose.[30]

Effects on Reproduction

Exposure to glufosinate during pregnancy negatively impacts the developing fetus in rabbits. The highest dose tested (20 mg/kg of body weight per day) caused a decrease in the number of mother rabbits with live fetuses. The frequency of premature delivery and miscarriages increased. An increase in the number of dead fetuses per litter was found in all treated rabbits.[31]

The effects of glufosinate on rat reproduction have been extensively studied. One of the first studies found that glufosinate caused a sharp decrease in the number of pups lost as fetuses. At the two highest doses tested, all fetuses died, and at the middle dose almost half of the fetuses died. Researchers used lower doses in a subsequent two-generation study: litter size was reduced in both generations[32] at doses over 12 mg/kg of body weight.[1] The researchers concluded that glufosinate had "toxic effects on early embryonic development."[32]

In another series of tests, glufosinate was shown to arrest the development of the kidney and ureter at doses of 10 mg/kg of body weight and greater.[2,33] In addition, glufosinate caused "fetal wastage" (fetal loss) at the mid and high dose tested (50 and 250 mg/kg of body weight).[34]

Two other studies support the conclu-

sion that glufosinate poses reproductive hazards. In rats, the testes are one of three organs where glufosinate accumulates; only the kidneys accumulated more.[35] Exposure of embryos to glufosinate during laboratory culture caused "significant" growth retardation in mice and rats along with birth defects. When mice embryos were exposed on day ten of development, over 80 percent developed defects.[36]

There are no publicly available studies about the ability of glufosinate-containing products to cause reproductive effects.

Carcinogenicity

Whether or not glufosinate causes cancer is ambiguous. One of the studies submitted in support of its registration, a long-term feeding study in rats, showed an increase in the frequency of adrenal medullary tumors. Both this study and a long-term feeding study in mice, were "inadequate for a reliable assessment of carcinogenic potential" because they did not use sufficient doses. EPA recommended they be repeated.[37]

There are no publicly available studies about the ability of glufosinate-containing products to cause cancer.

Effects on Animals

It is often assumed that herbicides are primarily toxic to plants. However, glufosinate has demonstrated adverse effects in the following animals:

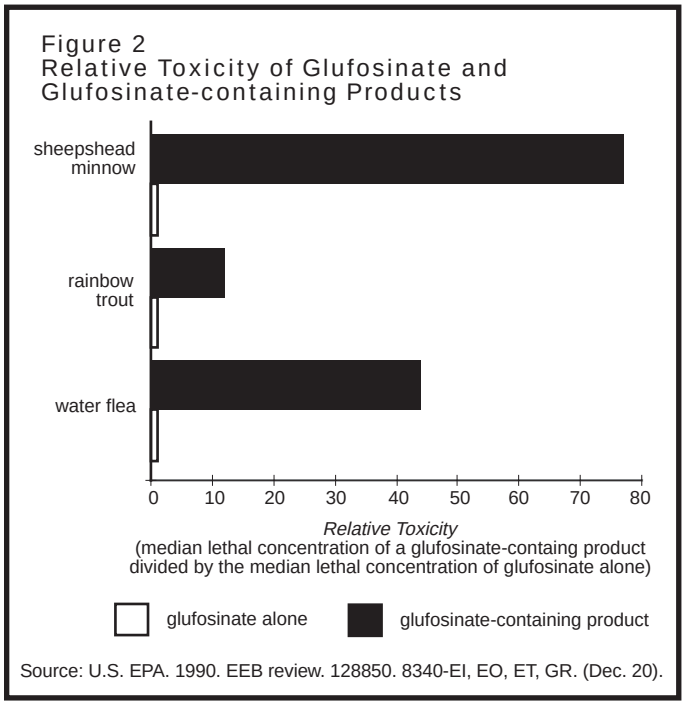
Clams: The glufosinate-containing product Ignite is "highly toxic" to clam larvae. Concentrations over about 1/2 part per million (ppm) cause adverse effects.[38]

Oysters: Glufosinate is "acutely toxic to embryos and larvae" at concentrations above 8 ppm, reducing the production of normal larvae by 66 percent.[39] A glufosinate-containing product is more toxic, reducing the number of normal larvae by half at a concentration of about 1/2 ppm.[40]

Shrimp: Glufosinate at concentrations above 1 ppm causes loss of equilibrium in shrimp.[41] Similar concentrations of a glufosinate-containing herbicide caused similar effects.[42] A concentration of 7.5 ppm caused death of half of the shrimp tested.[41]

Daphnia (water flea): The LC_{50} (the concentration which kills half of a test population) of a glufosinate-containing product for water fleas is 15 ppm.[43] Concentrations of glufosinate alone above 32 ppm inhibited reproduction.[44]

Fish: Concentrations of a glufosinate-containing herbicide above 7 ppm killed sheepshead minnows. The LC_{50} was 13 ppm.[45] For rainbow trout, the LC_{50} was 27 ppm with a four day exposure[46] and 7 ppm with a 21-day exposure.[1] For both fish, as well as for *Daphnia*, glufosinate-containing products show higher toxicity than glufosinate alone. (See Figure 2.)



Tests of a glufosinate-containing herbicide showed that it is much more toxic to fish and aquatic invertebrates than glufosinate alone.

Birds: The amount of glufosinate needed to kill birds via ingestion is relatively high, more than 2000 mg/kg of bodybweight.[2] However, there appear to be effects on reproduction at much lower doses. A study of mallard ducks found that females who ingested up to 60 mg/kg of body weight per day laid 20 percent fewer eggs than untreated birds. This resulted in production of about 30 percent fewer ducklings.[47] These results were not statistically significant according to the laboratory that performed the study,[47] but given glufosinate's striking reproductive effects in laboratory animals, further studies are needed.

Effects on Nontarget Plants

As might be expected from a broad-spectrum herbicide, glufosinate is damaging to plants with which it comes in contact. As EPA stated, it is "expected to adversely affect non-target terrestrial plant species."[38] In addition, there are complex effects. These include the following:

Plant diseases: A study of 12 agricultural and 3 forest soils from Canada showed that treatment with glufosinate reduced the number of fungi in the agricultural soils about 20 percent and the number of bacteria about 40 percent. Bacteria from forest soils were reduced about 20 percent.[48] One result of the study was startling. Plant disease-causing fungi were among the most resistant species tested.[50] *Trichoderma* species, regarded as beneficial because they parasitize disease-causing species, were among the most sensitive.[48] (See Fig. 3) Further study showed that glufosinate impaired *Trichoderma's* control of two plant pathogens.[49] The researchers caution that use of glufosinate has "important microbiological consequences."[50]

Endangered species: EPA's Ecological Effects Branch review of glufosinate stated, "Any off-target movement … is expected to adversely affect nontarget endangered terrestrial plant species"[51] and recommends that "Ignite [a glufosinate-containing herbicide] must be disallowed in those counties (or areas within counties) where endangered/threatened plants exist in or adjacent to non-crop areas."[51] This recommendation was later changed to a recommendation for a100 yard buffer zone around areas that have endangered species.[38]

Soil Mobility and Persistence

EPA classifies glufosinate as "mobile" in

soil. It's solubility in water is high: 1370 grams of glufosinate can be dissolved in a liter of water.[2] In laboratory tests of mobility in loam or sandy loam soils, up to 80 percent of the glufosinate leached enough to be classified as "mobile" and up to 7 percent leached enough to be classified as "very mobile." Only in a volcanic ash was glufosinate mostly immobile.[52] This means that "the potential for groundwater contamination does exist."[2]

EPA classifies glufosinate as "persistent."[2] However, as with all herbicides, persistence of glufosinate varies depending on soil, climate, weather, and other characteristics. A study in California vineyards found that the half-life of glufosinate (the time required for half of the applied amount to break down or move away from the study area varied from 12 to 70 days, with an average of about 40 days.[53] Other half-lives reported by EPA include 20 days in Maryland, 15 days in Washington, and 6 days in Illinois.[54]

As with most pesticides, persistence of glufosinate is longer than the half-life. In Canadian soils, glufosinate persisted until the end of the interval over which measurements were taken (60, 67, and 113 days).[55] In a greenhouse, glufosinate persisted in soil for 172 days.[54]

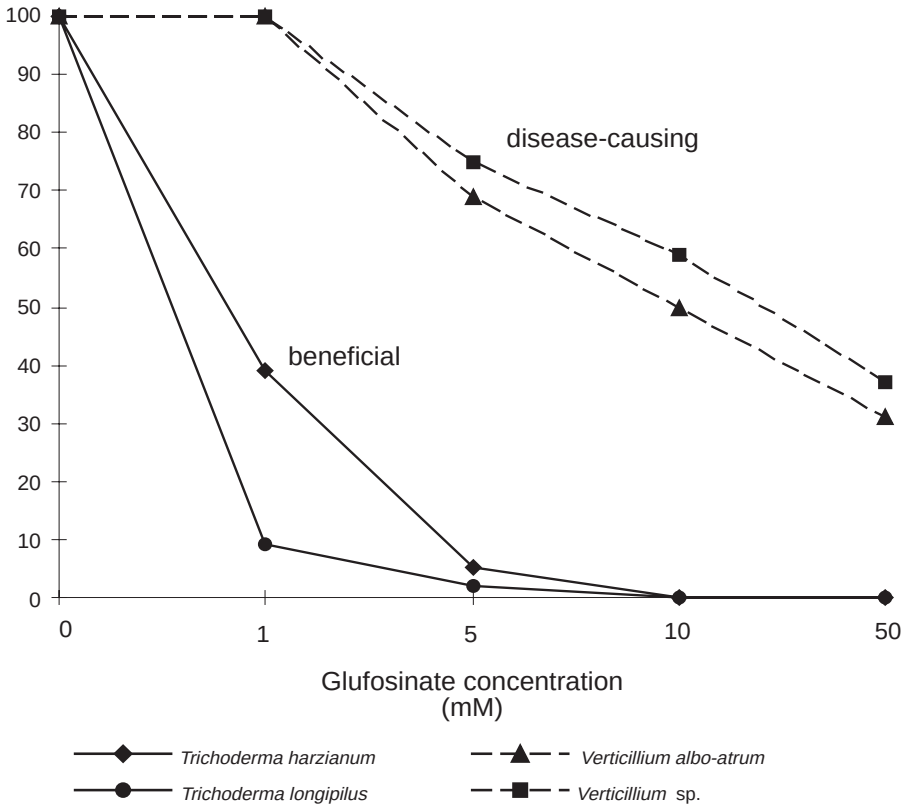
Glufosinate in Food

Glufosinate was found in the edible parts of spinach, radishes, wheat, and carrots that had been planted 120 days after treatment with a glufosinate-containing herbicide.[54]

Metabolites

A major breakdown product of glufosinate found in both plants and animals that have been exposed to glufosinate is 3-methylphosphinicopropioninc acid. (See Fig. 4)[56,57] This metabolite is, like glufosinate, a neurotoxin. In rats, injection into the brain caused severe convulsions and forelimb spasms.[58] It is more persistent than glufosinate: one study found it persisted for 267 days after treatment[59] and another found it in wheat that had been planted 120 days after a glufosinate treatment.[60] It is more mobile than glufosinate in soil.[61]

Figure 3
Glufosinate's Effect on the Growth of Beneficial and Disease-causing Fungi



Source: Ahmad, I. and D. Malloch. 1995. Interaction of soil microflora with the bioherbicide phosphinothricin. *Agric. Ecosys. Environ.* 54:165-174.

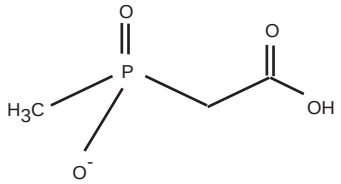
Source: U.S. EPA. 1990. EEB review. 128850. 8340-EI, EO, ET, GR. (Dec. 20).

Glufosinate strongly inhibits the growth of several species of beneficial fungi, while disease-causing species can be quite tolerant of this herbicide.

Inert Ingredients

All glufosinate-containing herbicides contain ingredients that are identified only as "inert" ingredients by EPA.[3,4] AgrEvo has identified one of these ingredients, alkyl hydroxypoly (oxyethylene) sulfate.[62,63] It is a surfactant, a detergent-like chemical used in glufosinate products to help the glufosinate penetrate leaf surfaces. There is little publicly available information about this surfactant. However, as described above, the glufosinate-containing products studied are significantly more toxic to aquatic organisms than glufosinate alone. (See Figure 2.) The surfactant is likely to be a major cause of this increased toxicity.[64]

Figure 4
Glufosinate's major metabolite



3-methylphosphinicopropionic acid

Interactions

When glufosinate was combined with the herbicide metolachlor for a 4-hour in-

halation study with rats, the following symptoms occurred: loss of coordination, convulsions, diarrhea, emaciation, atrophied testes, and withdrawn testes.[65] While some of these symptoms were found in inhalation studies of glufosinate alone, the adverse effects on the testes seem to be related to an interaction between the two herbicides.

References

1. Hoerlein, G. 1994. Glufosinate (phosphinothricin), a natural amino acid with unexpected herbicidal properties. *Rev. Environ. Contam. Toxicol.* 138:73-145.
2. U.S. EPA. Office of Pesticides and Toxic Substances. 1993. Pesticide fact sheet: Glufosinate ammonium. (June 29.)
3. AgrEvo USA Co. 1996. Finale specimen label. Wilmington, DE. (May.)
4. AgrEvo USA Co. 1996. Rely specimen label. Wilmington, DE. (May.)
5. U.S. Dept. of Agriculture. 1995. Availability of determination of nonregulated status for genetically engineered corn. *Fed. Reg.* 60(134):36095-36096. (July 13.)
6. U.S. Dept. of Agriculture. 1996. Availability of determination of nonregulated status for soybeans genetically engineered for glufosinate herbicide tolerance. *Fed. Reg.* 61(160):42581-42582. (Aug 16.)
7. U.S. Dept. of Agriculture. 1995. Availability of environmental assessments and findings of no significant impact. *Fed. Reg.* 60(14):4398. (Jan. 23.)
8. U.S. Dept. of Agriculture. 1995. Availability of environmental assessments and findings of no significant impact. *Fed. Reg.* 60(108):29819. (June 6.)
9. U.S. Dept. of Agriculture. 1995. Availability of environmental assessments and findings of no significant impact. *Fed. Reg.* 60(187):49824. (Sept. 27.)
10. U.S. Dept. of Agriculture. 1996. Availability of environmental assessments and findings of no significant impact. *Fed. Reg.* 61(170):45933. (Aug. 30.)
11. Food and Agriculture Organization, World Health Organization, and the International Programme on Chemical Safety. 1992. *Pesticide residues in food - 1991: Toxicology evaluations.* Pp. 209-239. Geneva, Switzerland.
12. U.S. EPA. 1988. Observational assessment of neurotoxicity and determination of glutamine synthetase (GS) activity, NH4+ and glutamate levels in brain, liver, and kidney of female Wistar rats. Data evaluation report. Reviewed by W. Phang, M. van Gemert, and W. Sette. Sept. 19.
13. U.S. EPA. 1984. Acute oral toxicity of HOE 39866 to the male and female dog. Unpublished evaluation. (Sept. 24.)
14. U.S. EPA. Office of Pesticides and Toxic Substances. 1985. Experimental use permit (6340-EUP-RN) and temporary tolerance petition (4G3156) for HOE 39866. Memo from D.S. Saunders to R. Mountfort, Registration Div. (April 18.)
15. U.S. EPA. 1984. Acute oral toxicity of HOE 39866 to the male rat. Unpublished evaluation. (Sept. 25.)
16. U.S. EPA. 1984. Acute oral toxicity of HOE 39866 to the female rat. Unpublished evaluation. (Sept. 25.)
17. U.S. EPA. 1984. Acute oral toxicity of HOE 39866 to the male mouse. Unpublished evaluation. (Sept. 25.)
18. U.S. EPA. 1984. Acute oral toxicity of HOE 39866 to the female mouse. Unpublished evaluation. (Sept. 25.)
19. U.S. EPA. 1984. Acute percutaneous toxicity of HOE 39866 to the male rat. Unpublished evaluation. (Sept. 25.)
20. U.S. EPA. 1984. Acute percutaneous toxicity of HOE 39866 to the female rat. Unpublished evaluation. (Sept. 25.)
21. U.S. EPA. 1984. Acute dermal toxicity study in rats. Unpublished evaluation. (Aug. 9.)
22. U.S. EPA. 1984. Aerosol inhalation of HOE 39866 to the male and female SPF-Wistar rat. Unpublished evaluation. (Sept. 25.)
23. U.S. EPA. 1988. Metabolism (dogs). Data evaluation report. Reviewed by W. Phang and M. van Gemert. (August 31.)
24. Fujii, T., T. Ohata, and M Horinaka. 1996. Alterations in the response to kainic acid in rats exposed to glufosinate ammonium, a herbicide, during infantile period. *Proc. Japan Acad.* 72 (Ser. B): 7-10.
25. U.S. EPA. Office of Pesticides and Toxic Substances. 1986. Experimental use permit and temporary tolerance for Ignite herbicide (HOE-39866). Memo from D.S. Saunders, Toxicology Branch, to R. Mountfort, Registration Div.. Sept. 26.
26. U.S. EPA. Office of Pesticides and Toxic Substances. 1991. Ignite 1SC herbicide - evaluation of acute data. Memo from S.L. Makris, Health Effects Div., to J. Miller and J. Mayes, Registration Div.. Oct. 7.
27. U.S. EPA. 1988. Ignite (HOE 039866): Chronic toxicity feeding study in dogs. Reviewed by M.E. Brower and W.L. McLellan. Dynamac Corp. (May 17.)
28. U.S. EPA. 1984. Two-week subacute toxicity in rats. Unpublished evaluation. September 25.
29. U.S. EPA. Office of Pesticides and Toxic Substances. 1988. Ignite: Evaluation of the toxicology studies on Ignite or HOE 039866. Memo from W. Phang, Toxicology Branch, to R. Mountfort, Registration Div.. (Dec. 1).
30. U.S. EPA. 1988. 13-week feeding study in mice. Data evaluation report. Reviewed by W. Phang and M. van Gemert. May 17.
31. U.S. EPA. 1988. Teratology study in rabbits. Data evaluation report. Reviewed by W. Phang and M. van Gemert, Toxicology Branch. May 11.
32. U.S. EPA. 1988. Ignite: Two generation reproduction study in rats. Data evaluation record. Reviewed by Dynamac Corp. May 18.
33. U.S. EPA. 1993. Response to Rfd/peer review report of glufosinate-ammonium (Ignite) re: Historical control data (rat). Memo from J.N. Rowe, Health Effects Div., to J. Miller, Registration Div. (Jan. 28.)
34. U.S. EPA. 1984. Embryotoxic effect in Wistar rats after oral administration. (Sept. 25.)
35. EPA. 1984. Study on kinetics and residue analysis in rats. Unpublished evaluation. (Sept. 25.)
36. Watanabe, T. 1995. Developmental effects of glufosinate ammonium on mammalian embryos in vitro. Japan. Teratol. Soc. Abs. *Teratology* 52(4): 25-26B. (Abstract.)
37. U.S. EPA. Office of Pesticides and Toxic Substances. 1992. RfD/Peer review report of glufosinate-ammonium (Ignite). Memo from G.Z. Ghali, Health Effects Div., to J. Miller, Registration Div.. (May 13.)
38. U.S. EPA. 1993. EEB review of glufosinate/Ignite herbicide. Memo from D. Urban, Ecological Effects Branch, to J. Miller, Registration Div.. (June 16.)
39. U.S. EPA. 1990. Mollusc 48-hour embryo-larvae study. HOE 39866 technical. Data evaluation record. Reviewed by K.D. Rhodes, KBN Eng. Appl. Sci., Inc. (June 1.)
40. U.S. EPA. 1990. Mollusc 48-hour embryo-larvae study. HOE 039866 200 g/l soluble concentrate. Data evaluation record. Reviewed by K.D. Rhodes, KBN, Eng. Appl. Sci., Inc. (June 1.)
41. U.S. EPA. 1990. Estuarine invertebrate toxicity test. HOE 039866 technical. Data evaluation record. Reviewed by K.D. Rhodes, KBN Eng. Appl. Sci., Inc. (June 1.)
42. U.S. EPA. 1990. Estuarine invertebrate toxicity test. HOE 039866 200 g/l soluble concentrate. Data evaluation record. Reviewed by K.D. Rhodes, KBN Eng. Appl. Sci., Inc. (June 1.)
43. U.S. EPA. 1986. Aquatic invertebrate acute toxicity. Soluble concentrate 200 g/l. Data evaluation record. Reviewed by Carol Natella, Health Effects Div. (Nov. 20).
44. U.S. EPA. 1988. Life-cycle (21-day renewal) chronic test. Technical HOE 039866. Data evaluation record. Reviewed by P. Kosalwat, KBN Eng. Appl. Sci., Inc. (Oct. 4)
45. U.S. EPA. 1990. Estuarine fish toxicity test. HOE 039866 200 g/l soluble concentrate. Data evaluation record. Reviewed by K.D. Rhodes, KBN Eng. Appl. Sci., Inc. (June 1.)
46. U.S. EPA. 1986. Freshwater fish acute toxicity. Soluble concentrate 200 g/l. Data evaluation record. Reviewed by Carol Natella, Health Effects Div. (Nov. 20).
47. Roberts, N.R. et al. 1981. The effects of dietary inclusion of HOE 039866 - active ingredient technical - on reproduction in the mallard duck. Huntingdon, U.K.: Huntingdon Research Centre Ltd. Unpublished. Submitted to U.S. EPA. (March 18.)
48. Ahmad, I. and D. Malloch. 1995. Interaction of soil microflora with the bioherbicide phosphinothricin. *Agric. Ecosys. Environ.* 54:165-174.
49. Ahmad, I., J. Bissett, and D. Malloch. 1995. Influence of the bioherbicide phosphinothricin on interactions between phytopathogens and their antagonists. *Can. J. Bot.* 73: 1750-1760.
50. Ahmad, I., J. Bissett, and D. Malloch. 1995. Effect of phosphinothricin on nitrogen metabolism of *Trichoderma* species and its implications for their control of phytopathogenic fungi. *Pest. Biochem. Physiol.* 53:49-59.
51. U.S. EPA. 1990. EEB review. 128850. 8340-EI, EO, ET, GR. (Dec. 20).
52. Gildemeister, H. and U. Scheinkönig. 1985. Hoe 039866-14C leaching study. Hoechst Aktiengesellschaft. Submitted to U.S. EPA. (Sept. 4.)
53. U.S. EPA. 1992. EFGWB review of ammonoium-DL-homoalanin-4-yl methylphosphinate (glufosinate ammonium). Memo from A.A. Abramovitch to J. Miller and J. Mayes. (July 23.)
54. U.S. EPA. 1988. Glufosinate ammonium: Review and assessment of individual studies and environmental fate assessment. Submitted by Dynamac Corp. (Sept. 8.)
55. Smith, A.E. and M.B. Belyk. 1989. Field persistence studies with the herbicide glufosinate-ammonium in Saskatchewan soils. *J. Environ. Qual.* 18:475-479.
56. U.S. EPA. 1984. Metabolism study on female rats after a single oral dose of the active ingredient. Submitted by Hoechst Analytical Lab. (Sept. 25.)
57. Komoβa, D. and H. Sandermann. 1992. Plant metabolism of herbicides with C-P bonds: Phosphinothricin. *Pestic. Biochem. Physiol.* 43:95-102.
58. U.S. EPA. 1988. Catecholamines and glutamine synthetase determinations on Hoe 039866 and Hoe 061517 treated rats. Data evaluation report. Review by W. Phang and M. van Gemert. (Aug. 15.)
59. U.S. EPA. Undated. Rotational crop accumulation, confined. Data evaluation review 8. Review by E.B. Conerly, Environmental Fate and Groundwater Branch (EFGWB).
60. U.S. EPA. Office of Pesticides and Toxic Substances. Undated. Glufosinate ammonium review. Memo from H. Jacoby, EFGWB, to J. Miller and J. Mayes, Registration Div.
61. U.S. EPA. 1986. EAB review of 8340-EUP-10 HOE 39866 herbicide. Memo from E. Regelman, Hazard Evaluation Div. to R. Mountfort, Registration Div. (July 30)
62. AgrEvo USA Co. 1995. Material safety data sheet. Finale herbicide. Wilmington, DE. (Sept. 22.)
63. AgrEvo USA Co. 1995. Material safety data sheet. Rely herbicide. Wilmington, DE. (Sept. 22.)
64. U.S. EPA.. Office of Pesticides and Toxic Substances. 1988.EEB request for EFGWB determination on Ignite formulation. Memo from H. Jacoby, EFGWB, to D.J. Urban, Ecol. Effects Br. (Dec. 13.)
65. U.S. EPA. Office of Pesticides and Toxic Substances. 1986. EPA experimental use permit file symbol 100EUP-II. Dual/Ignite 4EW. Memo from M.L. Waller, Registration Div. to R. Mountfort, Registration Div. (Nov. 25.)