

Comments from the public: EFSA opinion on NK603 maize

Organisation: Greenpeace-Neumarkt Group, Bavaria, Germany

Country: Germany

Type: Individual

a. Assessment:

b. Food Safety Assessment:

Toxicology

Glufosinate ammonium (Glufosinate Herbicides used with all GMOs)fact sheet Glufosinate is produced by AgrEvo, a joint venture established by the German chemical corporations Hoechst and Schering. Current usage levels raise concern because of the marketing of genetically engineered crops resistant to this herbicide. Glufosinate is produced at Hoechst's Frankfurt plant in Germany where work began in 1995 to double production capacity in anticipation of the launch of genetically engineered glufosinate resistant crops. The herbicide was first introduced into Japan in 1984. In the UK, glufosinate was first considered in 1984. It was not approved then for toxicological reasons, but was given provisional approval in 1991 (see below). US registration was achieved in 1993. The product is now registered for use in more than forty countries and is marketed under a number of trade names including Basta, Rely, Finale and Challenge. In the UK agriculture, relatively small amounts of glufosinate are used: 25 tonnes on 60,000 ha. The main crops are oilseed rape and potatoes(1). These figures may change dramatically if genetically engineered, glufosinate resistant crops are commercialised. In North America, commercialisation has already begun. AgrEvo recently launched a formulation called Liberty, a glufosinate product for use on crops resistant to glufosinate. In Canada, Liberty Link canola has been on sale since 1995. In 1997, Liberty Link soybean and maize were approved in the US. The US maize (corn) and soybean herbicide markets account for 40% of all US pesticide sales(2). AgrEvo expects glufosinate to become its linchpin product by 2000, with an annual turnover of about US\$680 million by 2001-2(3). AgrEvo aims to promote the fast spread of glufosinate resistance into popular crop varieties, including sugar beet. The profits could be considerable: introduction of Liberty Link varieties is expected to increase sales of glufosinate to over US\$300 million(4). What is glufosinate Glufosinate is a short name for the ammonium salt, glufosinate-ammonium. It is a broad-spectrum contact herbicide and 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 off) crops before harvest. Glufosinate is a natural compound isolated from two species of *Streptomyces* fungi. It inhibits the activity of an enzyme, glutamine synthetase, which is necessary for the production of glutamine and for ammonia detoxification. The application of glufosinate leads to reduced glutamine and increased ammonia levels in the plant tissues. This causes photosynthesis to stop and the plant dies within a few days(5). Glufosinate also inhibits the same enzyme in animals. Health impacts Hoechst claims that under conditions of recommended use of glufosinate ammonium, a "detrimental effect on the health of both users and consumers is extremely unlikely" (6). Glufosinate ammonium structurally resembles glutamic acid, a natural amino acid that can stimulate the central nervous system. It is recognised that excess release of glutamic acid results in the death of nerve cells in the brain(7). The surfactant, AES, which is used in

formulations, has also been found to cause toxic effects and may be a cause of some of the clinical symptoms observed in suicide cases involving glufosinate. The metabolite, MPPA-3, is, like glufosinate, a neurotoxin. The US EPA reported that MPPA-3 injected into the brain of rats caused severe convulsions(8). Acute Toxicity The toxicity of glufosinate varies in different laboratory animals. The oral LD50 is 436-464 mg/kg in mice and 1,510-1,660 mg/kg in rats. Dogs are the most sensitive. They can be twice as susceptible as mice (the LD50 for beagles is 200-400 mg/kg(9)). The LD50 of the surfactant, AES, is 1,995-2,138 mg/kg in rats. The World Health Organisation (WHO) classifies glufosinate in toxicity Class III, "slightly hazardous". The WHO classification system is based on the LD50 for rats and aims to take account of the sensitivities of more vulnerable test animals. The dermal LD50 for glufosinate is about the same as for oral exposures. However, through the skin, glufosinate formulations can be 2.5 times more toxic than glufosinate alone(10). Glufosinate was first considered by the UK Ministry of Agriculture, Fisheries and Food (MAFF) Scientific Sub-Committee in 1984. The herbicide was refused approval because of the toxicity of the formulation (containing 30% surfactant) when absorbed through the skin(11). 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" (12). However, the Sub-Committee remained concerned about the dermal toxicity of one of the six formulations, requiring the applicants to submit further studies.

Neurotoxic Effects Glufosinate has been found to cause a number of neurological symptoms in laboratory animals following both oral and dermal exposure. At lethal doses, overt signs of toxicity include: convulsions, salivation, hypersensitivity, irregular breathing, and trembling(13). Some of the behavioural changes lasted several days(14). At sub-lethal doses, glufosinate can have significant, but not so easily observable impacts. For example, a recent study found that low doses of glufosinate affected central nervous system development in young rats. One-day old rats were exposed to a dose of 1, 2 or 5 mg/kg of glufosinate daily for seven days. At six weeks they were tested for the 'wet-dog shakes' induced by administering kainic acid. Kainic acid stimulates glutamate receptors in the brain. The frequency of wet-dog shakes decreased significantly in all the glufosinate exposed rats. The results suggested that exposure to even low doses of glufosinate in the infantile period in rats causes changes in the kainic acid receptor in the brain(15).

Teratogenic effects (birth defects) In recent studies, sub-lethal doses of glufosinate ammonium was 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(16). 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 g/ml, and cleft lips at 20 μ development) of the forebrain at 10 g/ml(17).
- The effects on embryos after exposure of pregnant rats to μ 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. The results suggested that glufosinate exposure at a crucial stage in pregnancy causes a decrease in the number of glutamate receptors in offspring(18).

Residues in food and water Residues in food are an area of concern, especially when glufosinate is used as a pre-harvest desiccant. MAFF in the UK states 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(19). The WHO/FAO recommended acceptable daily intake (ADI) for glufosinate is 0.02 mg/kg. MAFF's 1990 evaluation document on glufosinate states that when it 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. 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% of the residue was retained. Residue levels in bran were 10-600% of those in grain. 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. In 1991, the MAFF Advisory Committee on Pesticides, the body responsible for registering pesticides in the UK, was concerned that significant residues of glufosinate were found in the crops at the time of harvest(20). 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 intakes of residues in animal products. AgroEvo claims that glufosinate is unlikely to leach into groundwater(21), but independent evidence suggests otherwise. Glufosinate is highly soluble in water and is also classified as persistent and mobile (see below). The dangers of soluble pesticides contaminating water supplies as a result of recommended agricultural use is recognised by both the industry and governments throughout the European Union. Environmental Fate 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(22) and up to 70 days in others(23). The shortest half life tends to be in soils with a high clay and organic matter content(24). In one study, residues of glufosinate were found in spinach, radishes, wheat and carrots planted 120 days after glufosinate had been applied(25). 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' (26). Glufosinate's metabolite, MPPA-3, has been found to be more persistent and more mobile than glufosinate(27). Effects on wildlife 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, 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 resistant 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" (28). Glufosinate is toxic to a number of aquatic animals including the larvae of clams and oysters(29), daphnia and some freshwater fish species(30). The commercial formulations are more toxic than the technical grade glufosinate. For example, for the aqueous formulation, the LC50s for the fish tested were between 12.3 and 79 mg/l and for the active ingredient they were between 320 and 1,000 mg/l(31). The rainbow trout, *Oncorhynchus mykiss*, was the most sensitive species in these tests. The acute oral LD50 for birds is 2,000 mg/kg. 4 day old partridges given a dose of 2,000 mg/kg of 96% glufosinate showed signs of central nervous system damage including ataxia, disequilibrium, convulsions, trembling, and wing flapping(32). Effects on non-target plants Glufosinate is a broad spectrum herbicide and is damaging to most plants that it comes into contact with. The US EPA has stated that glufosinate is "expected to adversely affect non-target terrestrial plant species" (33). Conclusion The development of herbicide resistant crops is a strategy developed by a number of chemical companies to increase profits and ensure that key product lines can compete in the market place. AgrEvo has targeted the broad spectrum herbicide, glufosinate, as their linchpin product for the future and initiated a fast track programme to produce a range of

crops resistant to glufosinate. However, studies demonstrate that it causes adverse health effects in animal studies, is likely to leach to drinking water sources, could increase nitrate leaching, and is toxic to beneficial soil micro-organisms. The introduction of glufosinate resistant crops and a greater exposure to glufosinate increases the likelihood of these harmful effects in humans and the environment. Glufosinate resistance will tend to intensify and increase dependency on herbicide use rather than lead to significant reductions. This fact sheet is taken from Health and Environmental Effects of Glufosinate (in press) written by Topsy Jewell for Friends of the Earth. References 1. Pers. Comm., MAFF, Pesticides Usage Survey Group. MAFF, York. 2. Agrow No. 273 January 31st 1997, p. 21. 3. S. Watkins, 1995. Agrow's Top Twenty Five. Report ref: DS 106, PJB Publications, London. 4. Agrow, 1997, Agrow's Top 25 1997 edition. p. 24. 5. E. Rasche, J. Cremer, G. Donn, J. Zink. 1995, The Development of Glufosinate Ammonium Tolerant Crops into the Market. In Brighton Crop Protection Conference, Weeds, 1995, BCPC, Farnham, Surrey. 6. Hack, R. , E Ebert, G. Ehling, and K.H. Leist, Glufosinate-ammonium - some aspects of its mode of action in mammals, Food and Chemical Toxicology, 1994, Vol. 32, No. 5, pp. 461-470. 7. Fujii, T., Transgenerational effects of maternal exposure to chemicals on the functional development of the brain in the offspring. Cancer Causes and Control, 1997, Vol. 8, No. 3, pp. 524-528. 8. Cox, C., Herbicide Fact Sheet: Glufosinate, Journal of Pesticide Reform, North West Coalition for Alternatives to Pesticides, Oregon, US, 1996. 9. MAFF, Evaluation No. 33 : HOE 399866 (Glufosinate-ammonium), Ministry of Agriculture Fisheries and Food, London, 1990. 10. US EPA, Office of Pesticides and Toxic Substances, Experimental Use permit (6340-EUP-RN) and Temporary Tolerance Petition (4G3156) for HOE 39866. Memo from D.S. Saunders to R. Mountfort, Registration Division, 18th April 1985. 11. MAFF, Health and Safety Executive, 1991. Advisory Committee on Pesticides Annual Report 1991, HMSO, London. 12. Ibid. 13. Op. cit. 9. 14. Op. cit. 8. 15. Fujii, T., T. Ohata, M. Horinaka, 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, 1996, Vol. 72, No. 1, pp. 7-10. 16. Watanabe, T. , Apoptosis induced by glufosinate ammonium in the neuroepithelium of developing mouse embryos in culture. Neuroscientific Letters, 1997, Vol. 222, No. 1, pp.17-20. 17. Watanabe, T. and T. Iwase, Development and dymorphogenic effects of glufosinate ammonium on mouse embryos in culture. Teratogenesis carcinogenesis and mutagenesis, 1996, Vol. 16, No. 6, pp. 287-299. 18. Op. cit. 7. 19. Op. cit. 9. 20. MAFF, Health and Safety Executive, 1991. Advisory Committee on Pesticides Annual Report 1991, HMSO, London. 21. Pesticides Trust [now PAN UK], Crops Resistant to Glutamine Synthetase Inhibitors. Pesticides Trust, London, 1997. 22. Op. cit. 9. 23. Op. cit. 8. 24. B.S. Ismail, and A.R. Ahmed, Attenuation of the herbicidal activities of glufosinate-ammonium and imazapyr in 2 soils. Agric. Ecosystems and Environ., 1994, Vol. 47, No. 4, pp. 279-285. 25. US EPA, Glufosinate Ammonium: Review and assessment of individual studies and environmental fate assessment . Submitted by Dynamac Corp, Sept 8 1988 Cited in Cox, C. 1996. Op. cit. 8. 26. Allenking, R.M., B.J. Butler. and B. Reichert, Fate of the herbicide glufosinate-ammonium in the sandy, low organic-carbon aquifer at CFB Borden, Ontario, Canada, Journal of Contaminant Hydrology, 1995, Vol 18, No 2, pp161-179. 27. Gallina, M.A. and G.R. Stevenson, Dissipation of [c-14] glufosinate ammonium in 2 Ontario soils, J. of Agric. And Food Chem., 1992, Vol. 40, No. 1., pp.165-168. 28. 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). 29. Op. cit. 8. 30. Op. cit. 9. 31. Op. cit. 9. 32. Op. cit. 9. 33. US EPA, EEB review of glufosinate/ignite herbicide . Memo from D. Urban, Ecological Effects Branch, to J. Miller Registration Div. , June 16th 1993, Cited in Cox, C. 1996. Op. cit. 8.

3. Environmental risk assessment

Glufosinate ammonium (the active ingredient in all of the chemical herbicides used with all GMOs) - fact sheet Glufosinate is produced by AgrEvo, a joint venture established by the German chemical corporations Hoechst and Schering. Current usage levels raise concern because of the marketing of genetically engineered crops resistant to this herbicide. Glufosinate is produced at Hoechst's Frankfurt plant in Germany where work began in 1995 to double production capacity in anticipation of the launch of genetically engineered glufosinate resistant crops. The herbicide was first introduced into Japan in 1984. In the UK, glufosinate was first considered in 1984. It was not approved then for toxicological reasons, but was given provisional approval in 1991 (see below). US registration was achieved in 1993. The product is now registered for use in more than forty countries and is marketed under a number of trade names including Basta, Rely, Finale and Challenge. In the UK agriculture, relatively small amounts of glufosinate are used: 25 tonnes on 60,000 ha. The main crops are oilseed rape and potatoes(1). These figures may change dramatically if genetically engineered, glufosinate resistant crops are commercialised. In North America, commercialisation has already begun. AgrEvo recently launched a formulation called Liberty, a glufosinate product for use on crops resistant to glufosinate. In Canada, Liberty Link canola has been on sale since 1995. In 1997, Liberty Link soybean and maize were approved in the US. The US maize (corn) and soybean herbicide markets account for 40% of all US pesticide sales(2). AgrEvo expects glufosinate to become its linchpin product by 2000, with an annual turnover of about US\$680 million by 2001-2(3). AgrEvo aims to promote the fast spread of glufosinate resistance into popular crop varieties, including sugar beet. The profits could be considerable: introduction of Liberty Link varieties is expected to increase sales of glufosinate to over US\$300 million(4). What is glufosinate Glufosinate is a short name for the ammonium salt, glufosinate-ammonium. It is a broad-spectrum contact herbicide and 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 off) crops before harvest. Glufosinate is a natural compound isolated from two species of *Streptomyces* fungi. It inhibits the activity of an enzyme, glutamine synthetase, which is necessary for the production of glutamine and for ammonia detoxification. The application of glufosinate leads to reduced glutamine and increased ammonia levels in the plant tissues. This causes photosynthesis to stop and the plant dies within a few days(5). Glufosinate also inhibits the same enzyme in animals. Health impacts Hoechst claims that under conditions of recommended use of glufosinate ammonium, a "detrimental effect on the health of both users and consumers is extremely unlikely" (6). Glufosinate ammonium structurally resembles glutamic acid, a natural amino acid that can stimulate the central nervous system. It is recognised that excess release of glutamic acid results in the death of nerve cells in the brain(7). The surfactant, AES, which is used in formulations, has also been found to cause toxic effects and may be a cause of some of the clinical symptoms observed in suicide cases involving glufosinate. The metabolite, MPPA-3, is, like glufosinate, a neurotoxin. The US EPA reported that MPPA-3 injected into the brain of rats caused severe convulsions(8). Acute Toxicity The toxicity of glufosinate varies in different laboratory animals. The oral LD50 is 436-464 mg/kg in mice and 1,510-1,660 mg/kg in rats. Dogs are the most sensitive. They can be twice as susceptible as mice (the LD50 for beagles is 200-400 mg/kg(9)). The LD50 of the surfactant, AES, is 1,995-2,138 mg/kg in rats. The World Health Organisation (WHO) classifies glufosinate in toxicity Class III, "slightly hazardous". The WHO classification system is based on the LD50 for rats and aims to take account of the sensitivities of more vulnerable test animals. The dermal LD50 for glufosinate is about the same as for oral exposures. However, through the

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Zink. 1995, The Development of Glufosinate Ammonium Tolerant Crops into the Market. In Brighton Crop Protection Conference, Weeds, 1995, BCPC, Farnham, Surrey. 6. Hack, R. , E Ebert, G. Ehling, and K.H. Leist, Glufosinate-ammonium - some aspects of its mode of action in mammals, Food and Chemical Toxicology, 1994, Vol. 32, No. 5, pp. 461-470. 7. Fujii, T., Transgenerational effects of maternal exposure to chemicals on the functional development of the brain in the offspring. Cancer Causes and Control, 1997, Vol. 8, No. 3, pp. 524-528. 8. Cox, C., Herbicide Fact Sheet: Glufosinate, Journal of Pesticide Reform, North West Coalition for Alternatives to Pesticides, Oregon, US, 1996. 9. MAFF, Evaluation No. 33 : HOE 399866 (Glufosinate-ammonium), Ministry of Agriculture Fisheries and Food, London, 1990. 10. US EPA, Office of Pesticides and Toxic Substances, Experimental Use permit (6340-EUP-RN) and Temporary Tolerance Petition (4G3156) for HOE 39866. Memo from D.S. Saunders to R. Mountfort, Registration Division, 18th April 1985. 11. MAFF, Health and Safety Executive, 1991. Advisory Committee on Pesticides Annual Report 1991, HMSO, London. 12. Ibid. 13. Op. cit. 9. 14. Op. cit. 8. 15. Fujii, T., T. Ohata, M. Horinaka, 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, 1996, Vol. 72, No. 1, pp. 7-10. 16. Watanabe, T. , Apoptosis induced by glufosinate ammonium in the neuroepithelium of developing mouse embryos in culture. Neuroscientific Letters, 1997, Vol. 222, No. 1, pp.17-20. 17. Watanabe, T. and T. Iwase, Development and dymorphogenic effects of glufosinate ammonium on mouse embryos in culture. Teratogenesis carcinogenesis and mutagenesis, 1996, Vol. 16, No. 6, pp. 287-299. 18. Op. cit. 7. 19. Op. cit. 9. 20. MAFF, Health and Safety Executive, 1991. Advisory Committee on Pesticides Annual Report 1991, HMSO, London. 21. Pesticides Trust [now PAN UK], Crops Resistant to Glutamine Synthetase Inhibitors. Pesticides Trust, London, 1997. 22. Op. cit. 9. 23. Op. cit. 8. 24. B.S. Ismail, and A.R. Ahmed, Attenuation of the herbicidal activities of glufosinate-ammonium and imazapyr in 2 soils. Agric. Ecosystems and Environ., 1994, Vol. 47, No. 4, pp. 279-285. 25. US EPA, Glufosinate Ammonium: Review and assessment of individual studies and environmental fate assessment . Submitted by Dynamac Corp, Sept 8 1988 Cited in Cox, C. 1996. Op. cit. 8. 26. Allenking, R.M., B.J. Butler. and B. Reichert, Fate of the herbicide glufosinate-ammonium in the sandy, low organic-carbon aquifer at CFB Borden, Ontario, Canada, Journal of Contaminant Hydrology, 1995, Vol 18, No 2, pp161-179. 27. Gallina, M.A. and G.R. Stevenson, Dissipation of [c-14] glufosinate ammonium in 2 Ontario soils, J. of Agric. And Food Chem., 1992, Vol. 40, No. 1., pp.165-168. 28. 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). 29. Op. cit. 8. 30. Op. cit. 9. 31. Op. cit. 9. 32. Op. cit. 9. 33. US EPA, EEB review of glufosinate/ignite herbicide . Memo from D. Urban, Ecological Effects Branch, to J. Miller Registration Div. , June 16th 1993, Cited in Cox, C. 1996. Op. cit. 8.

4. Conclusions and recommendations

Glufosinate herbicides (used heavily with all Genetically Modified crops) are dangerous to the health of consumers.

Organisation: GMO-fritt Europa

Country: Sweden

Type: Individual

a. Assessment:

b. Food Safety Assessment:

Toxicology

Majs NK603 skall inte godkännas för det är inte säkerställt till 100% att det inte är giftigt och att det inte innebär allvarlig fara för den biologiska mångfalden.

Translation

Maize NK603 will not be approved until it is established with absolute certainty that it is not toxic and does not constitute a serious danger to biodiversity.

Nutritional assessment

Majs NK603 skall inte godkännas för det är inte säkerställt till 100% att det inte innebär allvarlig risk för människor och djur att äta eller komma i kontakt med den.

Translation

Maize NK603 will not be approved until it is established with absolute certainty that it does not constitute a serious danger to humans or animals eating or coming into contact with it.

3. Environmental risk assessment

Majs NK603 skall inte godkännas för det är inte säkerställt till 100% att det inte innebär allvarlig fara för miljön.

Translation

Maize NK603 will not be approved until it is established with absolute certainty that it does not constitute a serious danger to the environment.

4. Conclusions and recommendations

Stoppa alla GMO-tillstånd. Gör Europa till en GMO-fri zon!

Translation

Stop all authorisations for GMOs. Make Europe a GMO-free area!

Organisation: AEAC/SV

Country: Spain

Type: Non Profit Organisation

a. Assessment:

4. Conclusions and recommendations

The cultivation of NK603 maize genetically tolerant to glyphosate in the EU is expected to offer new tools for soil conservation and reduction of CO₂ emissions, thanks to the use of low risk herbicides instead of repeated tillage operations. As a non profit association interested in the promotion of conservation agriculture, the Spanish Association for Conservation Agriculture/Live Soils (AEAC/SV) believes that the approval for this technology should be advanced as soon as possible. With this technology, farmers in the EU can contribute to soil conservation, better wildlife habitats and reduction of CO₂ emissions in the same way as American farmers have been doing since 2001.

Organisation: APOSOLO - ASSOCIAÇÃO PORTUGUESA DE MOBILIZAÇÃO DE CONSERVAÇÃO DO SOLO

Country: Portugal

Type: Association

a. Assessment:

4. Conclusions and recommendations

Herbicide tolerant crops have enabled farmers in many parts of the World to convert soybean, corn, cotton into Conservation Tillage practices like No-Till, Zone Till and Minimum Till. As a result heavy fuel consuming equipment used to plow and other tillage techniques are not needed anymore. Crop residue kept on the fields provide a shield against erosion and runoff streams. It also provides winter shelter and food for wildlife. Fewer herbicides applications and no or little tillage operations allow to conserve fuel (by less spending) and considerably reduces gas emissions. Allowing herbicide tolerant crops in Europe will have the same positive impact on environment. The same to workers and farmers by less contact with pesticides. Using glyphosate to control weeds other than residual herbicides will provide many benefits to environment. Without herbicide tolerant crops there is no future for Conservation Agriculture (CA) in Europe and the environment of this continent will not

benefit from all the advantages of those practices www.ecaf.org. CA is probably one of the most sustainable agriculture technology. It allows farmers to produce a great part of the food needed to feed the growing global population. Experts estimate that growers will need to double their current production by the year 2050 in order to meet global food demand. New skills and technologies are the only way to make that happen. European farmers need GMO's and other biotechnology events to produce on a sustainable way. Producing these crops will enable researchers to find some more biotech crops which will allow farming to be an activity more environmental friendly without which farmers may not be able to produce all the food we need. Additionally, the control that is already being done to such crops provides consumers a more safe food.