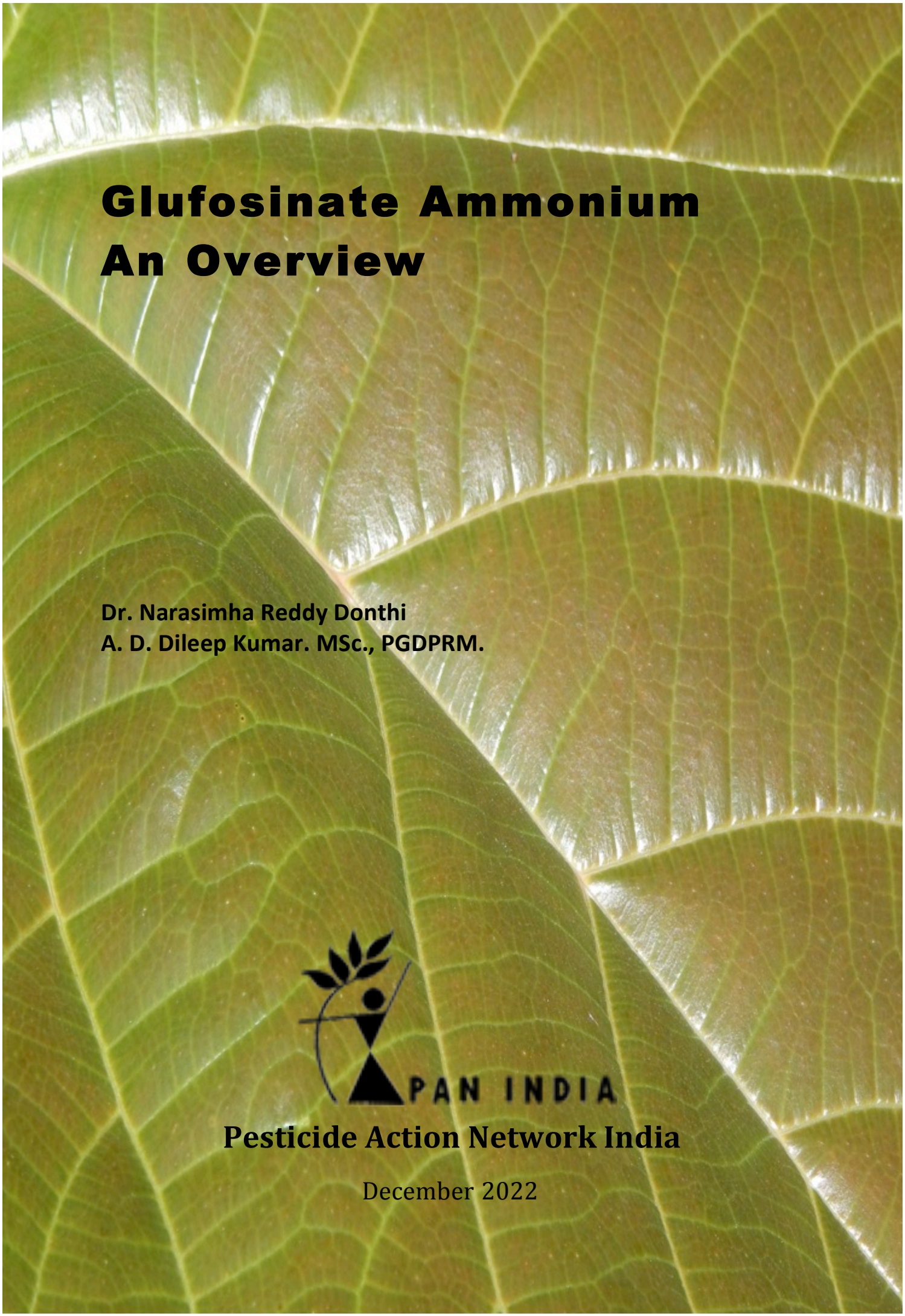

Glufosinate Ammonium
An Overview

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PAN INDIA

Pesticide Action Network India

December 2022

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About PAN India

Pesticide Action Network India (PAN India) is a public interest research and advocacy non-profit organisation formed in 2013. PAN India is a national independent organization in India, working closely with the PAN Asia Pacific and PAN international community. PAN India's objective is to help communities and governments to reduce dependence on toxic chemicals for pest control in agriculture, household as well as public health and to increase the use of sustainable alternatives. PAN India is committed to safe farming, safe living, and safe working place. PAN India is working to make India a world leader in Agroecology by empowering farming communities to keep away from toxic pesticides and agrochemicals, and to take up non-chemical methods of farming practices that champion traditional knowledge, biodiversity, and farmer participated research in attaining food sovereignty.

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SUMMARY

Glufosinate ammonium is being used as broad-spectrum post emergence herbicide globally, and the use is expected to increase due to the development of herbicide tolerant crops. It is a structural analog of glutamate and inhibits the glutamine synthetase enzyme, kill plants by ammonia accumulation and accumulation on reactive oxygen. Since glufosinate irreversibly inhibits glutamine synthetase, leading to intracellular accumulation of ammonia, hyperammonemia is considered one of the main mechanisms of glufosinate ammonium toxicity in humans. It is highly hazardous herbicide, owing to the fact that it can cause reproductive toxicity, neurotoxicity, and cardiovascular effects. It is also capable of causing damages to developing foetuses. Studies reports premature birth and intra uterine death and abortions in experimental animals, and human brain toxicity; pointing to high risk to mammals. Due to its acute and chronic adverse health effects, it has been banned in 29 countries.

Glufosinate, being a soluble volatile compound, spraying can result in inhalational and contact exposures and poisoning among the farming community, and people residing near agricultural fields where it is applied, vulnerability exists for all sections in the society. Registration authority in India specifies that full protective suit needs to be used while working with this herbicide. Given the reality of pesticide use conditions in India, there are high risks of exposure to users, due to the fact the recommended personal protective equipments of required quality is not available, or accessible; and even if available, the prevailing climatic conditions does not allow users to wear them. Because no antidote is available to glufosinate, there is less chance for saving poisoned individuals.

While, it is registered and approved for use in India, for weed control in tea and cotton crops, wide spread non approved use was reported in an ICAR study. Residue tests and monitoring is not been done for herbicides in India. As the reference values are not set for crops other than approved tea and cotton crops, non-approved spraying remains unmonitored for food safety standards and thereby putting consumers at risk of exposure to such pesticides unknowingly. Residues of herbicides/ planticides are generally not monitored in India. Consequently, the level and extent of contamination of food and fibre products remains unknown. Presence of residues in farm produce can have profound impact on health of food consumers and can be a factor in export markets.

Available information on environmental impacts shows harmful effects of glufosinate usage on living organisms, high risk to mammals, high toxicity on beneficial insects low to moderate risk to aquatic animals, birds, bees, earthworms, other soil non-target macro-organisms and soil micro-organisms reported. Development of resistance in plants is another adverse effects and impact on biodiversity is also reported.

Glufosinate is widely used to control weeds on crops that are tolerant to it in several countries. Studies demonstrated that glufosinate could cause adverse health and environmental problems. Introduction of glufosinate tolerant crops will pave way for its wider use, as it tends to increase dependency on this herbicide. It would result in higher exposure to humans and animals, thus increasing likelihood of adverse effects. It will be dangerous to farmers, farm workers as well as consumers (literally all the sections in the society).

Since glufosinate being a light dependent herbicide and is fully effective in full sunlight, spraying has to be during mid-day. This would mean pesticide workers have to necessarily spray in day time, usually under hot and humid conditions, leading to higher exposure. Even if proper protective measures were used harmful effects would be there, especially on respiratory functions.

Major features on glufosinate ammonium

- Synthetic Organophosphate agrochemical
- Used as broad spectrum herbicide
- It is a Neurotoxin, reproductive toxin, and foetotoxin/ teratogen.
- Not approved in EU, considered as a candidate for substitution
- A PAN Highly Hazardous Pesticide
- Banned in 29 countries, including in EU member countries, Morocco and UK.
- Approved in India for cotton and tea, but used for several crops.
- No antidote is known

INTRODUCTION

Glufosinate-Ammonium is a synthetic organophosphate glutamine synthetase inhibitor and neurotoxin that is used as an agrochemical. It is characterized as a highly soluble and volatile white to light yellow crystalline solid with a slightly pungent odor, and exposure occurs by inhalation, ingestion, or contact. Glufosinate is used for controlling weeds in orchards, rubber plantations, non-arable land, and no-till land all over the world. Glufosinate-ammonium is a non-selective, post emergence herbicide introduced in 1981 for vegetation control. Glufosinate-ammonium has come to be used mostly in transgenic glufosinate-ammonium tolerant crops and in vegetation management applications.

Glufosinate-ammonium was first derived from cultures of soil bacteria *Streptomyces viridochromogenesa* and was used as an herbicide in 1976. Glufosinate is a naturally occurring broad-spectrum herbicide produced by several species of Streptomyces soil bacteria. However, Glufosinate (dl-2-amino-4-(hydroxy(methyl) phosphonoyl) butanoic acid) is the chemically synthesized racemic mixture of the d and l stereoisomers of the natural compound l-phosphinothricin, which is produced by several taxa of *Streptomyces* (Copping and Duke, 2007; Lydon and Duke, 1999). Only the natural form, the l-isomer, is herbicidally active.

As transgenic glufosinate-tolerant crops became available, and glyphosate-resistance increased among weeds, glufosinate came to be projected as a key, broad-spectrum herbicide. Although glufosinate use has increased exponentially over the past decade, the treated area with this herbicide is still far less than that of glyphosate. Experts attribute inconsistent performance of glufosinate as the main reason, even though other factors including environmental conditions, application technology, and weed species are mentioned.

PROFILE OF GLUFOSINATE AMMONIUM

- **IUPAC Name**: Ammonium (2RS)-2-amino-4-(methylphosphinato) butyric acid
- **Substance group**: Organophosphate / Phosphonoglycine herbicide
- **Uses** : Broad spectrum, non-selective, systemic/contact post emergence herbicide
- **Mode of action :** Inhibition of glutamine synthetase enzyme in plant cells.
- **Trade names** : Basta (BASF) and Liberty (Bayer), Sweep Power (UPL), Himax (Insecticides India), Ferio (SWAL)
- **Acute Toxicity Classification** (WHO): Class II- Moderately hazardous
- **Herbicide resistance Classification (WSSA)**: 10
- **Manufacturing companies:** Bayer, UPL, Etc.
- **Signs and Symptoms of Poisoning:** Dizziness, tremors, convulsions and loss of consciousness
- **Antidote**: No specific antidote is known.

MODE OF ACTION

According to BASF, Glufosinate-ammonium is a plant protection product that works by inhibiting an enzyme central to plant metabolism. Plants absorb this substance primarily through their leaves and other green parts. As a contact herbicide, Glufosinat-ammonium is effective only where it comes into contact with the plant.

This allows it to control weeds without affecting the roots or requiring tillage, which is important especially for erosion-prone areas such as slopes[1].

Glufosinate ammonium (phosphinothricin ammonium) (GLA) is the active ingredient of Basta and several other herbicides used worldwide (Hoerlein G, 1994). Glufosinate is a fast-acting herbicide that was first discovered as a natural product, and is the only herbicide presently targeting glutamine synthetase (GS) (Takano & Dayan, 2020). It is also used as a desiccant. Glufosinate is a structural analog of glutamate and inhibits the glutamine synthetase. The result is a rapid build-up of a high ammonia level and a concomitant depletion of glutamine and several other amino acids in the plant. These effects are accompanied by a rapid decline of photosynthetic CO2-fixation and are followed by chlorosis and desiccation.

Glutamine Synthetase is the enzyme that catalyses the reaction of ammonia (NH3) and Glutamic acid to form Glutamine in plants. When Glufosinate-ammonium is sprayed on the plants, leaves absorbs it and collects ammonia. This gradually turns toxic, killing the cells and destroying the plants. It can exert similar effects in animals including human beings.

Recent studies indicate that the contact activity of glufosinate results from the accumulation of reactive oxygen species (ROS) and subsequent lipid peroxidation. Glufosinate disrupts both photorespiration and the light reactions of photosynthesis, leading to photoreduction of molecular oxygen, which generates reactive oxygen species (Takano & Dayan, 2020). Glufosinate is also highly hydrophilic and does not translocate well in plants, generally providing poor control of grasses and perennial species. The report claims that glufosinate is rapidly degraded by microorganisms, leaves no residual activity in the soil. The mode of action of glufosinate has been controversial, and the causes for the rapid phytotoxicity have often been attributed to ammonia accumulation.

Glufosinate efficacy depends on several factors related to the spraying conditions, temperature and humidity, and the target weeds. A good application technology and weather conditions can increase uptake levels, but the final concentration of glufosinate in leaves depends on metabolism rates. Glufosinate is a light-dependent herbicide and more effective when sprayed at full sunlight compared to night application. Some other findings are the following (Takano & Dayan, 2020):

- In addition to the accumulation of ammonia, glufosinate can also inhibit carbon assimilation
- Glufosinate performance substantially differs across weed species.
- Grass species tend to be less susceptible to glufosinate than broadleaves, with a few exceptions.
- Some weed species tend to develop symptoms faster than others, which may suggest the existence of different phytotoxic mechanisms or variable capacity to cope with reactive oxygen species (ROS) accumulation.
- Glufosinate efficacy was proportional to the concentration of the herbicide in the leaf tissue of 5 different weeds.

[1] https://agriculture.basf.com/global/en/innovations-for-agriculture/innovation-for-herbicides/glufosinate-ammonium/basics/how-does-glufosinate-ammonium-work.html

- Only the *pat* and *bar* genes have been used to develop glufosinate-tolerant crops. Expression of *pat* or *bar* at high levels allows the application of glufosinate postemergence in glufosinate-tolerant crops such as soybean, cotton, corn and canola.
- However, reliance on lower level of *pat* expression to provide protection from glufosinate in transgenic crops has been cautioned. The low mRNA expression level of the *pat* gene in those cultivars does not provide the same level of protection against glufosinate as of that achieved with the high expression of the *bar* gene in glufosinate-tolerant cotton (Carbonari, et al, 2016).

The Federal register of USA notes that, "the toxicology database for glufosinate is complete. A primary effect associated with glufosinate is inhibition of glutamine synthetase in the brain, which may be of significant concern for possible neurotoxicity and/or expression of clinical signs. Clinical signs of neurotoxicity were seen in several studies, including the subchronic, developmental, and chronic studies in rats and dogs. In addition to a variety of clinical signs, retinal atrophy was also observed in the subchronic and chronic rat studies. The rat developmental neurotoxicity (DNT) study demonstrated altered brain morphometrics" (USEPA, 2022).

While there have been concerns regarding glufosinate toxicology, study claims its proper use is recommended as safe. However, ingestion of undiluted glufosinate ammonium herbicide results in grave clinical outcomes (Mao, et al. 2012). In severe cases, patients either die or suffer severe toxic reactions, such as shock, respiratory arrest (apnea), unconsciousness, convulsions, and amnesia. A study has shown sixth cranial nerve palsy as a neurologic manifestation in acute glufosinate ammonium intoxication (Park, et al, 2013). Some toxicological studies assert that glufosinate ammonium and its commercial formulations are safe for users and consumers under the conditions of recommended use. As per these studies, it will degrade fast and completely in soil and surface water giving no scope for residues joining groundwater. The toxicological threshold levels for all the non-target organisms tested are well above the potential exposure levels and therefore do not reflect any hazard for non-target organisms in the ecosystem.

REGULATORY STATUS

International regulation: In US, Glufosinate ammonium is registered for use on a variety of food crops, and non-crop areas (golf course turf and residential lawns). At registration stage, EPA has conducted a human health risk assessment for citrus, pome, and stone fruits completed on July 25, 2012. An addendum was completed on January 24, 2013, addressing the scenarios that were not covered in the former assessment. An ecological risk assessment was also conducted including a screening-level listed species assessment[2].

Approval of glufosinate for agriculture usage in the EU has expired in 2018 and is currently not approved, and marked it as a candidate chemical for substitution[3].

Glufosinate ammonium is currently banned in 29 countries. They include 27 EU member countries, Morocco and UK. It is also included in the PAN International list of Highly Hazardous Pesticides. The new EU regulation in 2009 declares a ban on all CRM (carcinogenic, reprotoxic and mutagenic) pesticides from categories I and II. Glufosinate is classified as falling in reprotoxic category II. Already in 2006 Swedish authorities demanded an EU-wide ban. European Parliament members voted in January, 2009, not to renew permits for 22 substances, with glufosinate listed among them. Currently, agriculture use of Glufosinate Ammonium is not allowed in the EU as its approval expired 31st July, 2018 (Bohn T, et al, 2020).

French health and environment regulator ANSES in October, 2017, had withdrawn the license for Bayer's Basta F1 weedkiller made with glufosinate-ammonium, citing uncertainty over its effect on health following a review[4].

The Italian Ministry of Health banned it under the Directive 2009/2/EC. This directive was in relation to the classification, packaging and labelling of dangerous substances, which classified glufosinate as toxic for reproduction and capable of producing damage to foetuses. For the same reasons, this herbicide was subjected to a thorough human health impact assessment. However, subsequently, with two decrees signed on the 26th and 27th of April 2012, this ban on glufosinate ammonium was removed. This powerful herbicide produced by Bayer, was allowed to be used in agriculture for another five years, until its authorization expired in 2017. It was allowed up to 2017 for the weeding of important crops, especially grapevine, apple, pear, olive, and citrus, with the exception of some other uses, such as for wilting potato leaves and the weeding of seedbeds[5]. Even though this company concedes that this herbicide has its risks of producing adverse effects on the reproductive system of mice, it believes that this data cannot be transferred to humans.

In US, evidence of both qualitative and quantitative susceptibility following glufosinate exposure was found. A 28-day inhalation toxicity study demonstrated toxicity at the lowest dose as lung and bronchial congestion. The estimated drinking

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[2] USEPA, https://www.regulations.gov/document/EPA-HQ-OPP-2008-0190-0021

[3] https://ec.europa.eu/food/plant/pesticides/eu-pesticidesdatabase/start/screen/active-substances/details/79

[4] Article, https://www.reuters.com/article/us-france-health-glufosinate/french-health-regulator-withdraws-license-for-bayer-weedkiller-idUSKBN1CV2OT

[5] https://news.agropages.com/News/NewsDetail---7197.htm

water concentrations (EDWCs) of glufosinate are estimated to be 201 per billion (ppb) for acute dietary exposures and 24.4 ppb for chronic dietary exposures. However, Glufosinate was classified as "not likely to be a human carcinogen." USEPA has concluded that glufosinate does not pose a cancer risk to humans. Yet, the acute dietary exposure has been flagged as a cause of concern for all crop harvests and livestock commodities.

There is scepticism over EPA's findings. According to skeptics, birth defects have been caused by exposure of the human father to the herbicide (based on peer-reviewed studies in the published literature). They wanted EPA to thoroughly investigate these findings and reconsider the tolerance limits for glufosinate. It was noted there are no peer reviewed studies on the metabolism of the high levels of acetyl glufosinate in harvested GM crops to highly neurotoxic and teratogenic glufosinate. Certainly, gut bacteria are well known to contain enzymes that remove acetyl groups from glufosinate and mammalian enzymes may also be capable of removing the acetyl group from glufosinate. EPA was requested to obtain more technical data and information to better define the neurotoxicity and teratogenicity of glufosinate and its metabolites, especially in humans[6]. It was pointed out that Glufosinate, for example, was found to trigger apoptosis (programmed cell death) in the developing brain of the embryonic mouse. Numerous, well established studies showing brain damage and birth defects seem to have been ignored by those regulating use of the herbicide. It is apparent that adverse health effects of glufosinate and its metabolites is under studied, under researched and under reported. Glufosinate safety is far from proven. Its impact on humans and farm animals is difficult to trace because the GM products are not labeled for consumption.

USEPA stresses that no evidence of estrogenic or other endocrine effects of glufosinate and its metabolites have been noted in any of the toxicology studies that have been conducted with this product. But this was objected. USEPA was asked to determine if this herbicide has endocrine-disrupting potential.

A European Food Safety Authority (EFSA) evaluation states that glufosinate poses a high risk to mammals. The substance is classified as reprotoxic, with laboratory experiments causing premature birth, intra-uterine death and abortions in rats. Japanese studies show that the substance can also hamper the development and activity of the human brain. In an assessment of the occurrence of glufosinate residues in plants, processed commodities, rotational crops and livestock, possible acute risk to consumers was identified by EFSA in 2015[7].

Bayer acknowledges the risk of adverse effects with very high doses of the active Glufosinate on the reproductive system of mice, but objects to the transfer of such data to humans. It is currently banned in 27 EU states as per the PAN International consolidated list of banned pesticides updated on May 2022.

[6] USEPA, https://www.federalregister.gov/documents/2003/09/29/03-24565/glufosinate-ammonium-pesticide-tolerance
[7] EFSA, https://www.efsa.europa.eu/en/efsajournal/pub/3950

Regulation in India

It is registered for use (introduced in India during 1981)**.**

- **Approved use In India**: For tea and cotton.
- **Waiting period:** 96 days in cotton and 15 days in tea.
- **FSSA MRL:** 0.01 (tea)

Reported Use in India: Glufosinate-ammonium herbicide is used in the India to control weeds in many crops. Several non-approved uses including in food crops have been reported in a bulletin published by the Indian Council of Agriculture Research (ICAR).

#	**State**	**Crops used in**
1	Chatthisgarh	Rice and other crops
2	Haryana	Cotton and vegetables
3	Punjab	Cotton, rice and other crops
4	West Bengal	Tea
5	Assam	Tea
6	Andhra Pradesh	Cotton, sugarcane, rice, bengal gram, chillies, banana, grapes and other crops
7	Karnataka	Sugarcane, pomegranate and other crops
8	Goa	Sugarcane, pomegranate and other crops
9	Tamilnadu	Rice and other crops
10	Gujarat	Cotton and banana
11	Maharashtra	Grape and vegetables

(Source: ICAR, Herbicide Use in Indian Agriculture. 2016)

Non-approved use happening in India

Glufosinate ammonium 13.5% w/w SL is the formulation approved in India for cotton and tea. As pointed above, glufosinate use has been reported in India over several food and non-food crops in the country. These literally are non-approved use in India as glufosinate has been approved for weed control in tea and cotton.

In India, pesticides are approved for specific crop-pest combinations with a pre-harvest waiting period (although many approved uses in India do not have waiting periods set). A waiting period denotes the interval to be followed between the last application and harvest. Use of pesticides for the crops not approved for poses significant threat to food safety. There exists significant risk when food crops and vegetables fall under non-approved uses (Dileep Kumar, 2022).

The dangers of a pesticide being applied for non-approved crops are many, including that waiting periods are not known. Generally, for many of the vegetable crops,

farmers are not able to follow the recommended waiting period between last application and harvest (even in the case of approved uses) because the yields are harvested either once or twice in a week. As per the approved uses, the highest waiting of glufosinate is 96 days. In reality, farmers may not be able to follow this requirement, posing food safety risks. In addition to waiting periods, the maximum residue limits (MRLs) are not set for such non-approved crops. A 2013 report showed that there are a number of pesticides in India for which MRLs are not fixed (Bhushan, C., et al., 2013). Therefore, technically, such non approved crops and pesticides may not come under the purview of residue tests and monitoring as the reference values are not set and thus it may leave many of the non-approved uses not monitored for food safety standards and thereby putting consumers at risk of exposure to such pesticides unknowingly. As the residues of herbicides are generally not monitored in India, the level and extent of contamination of food articles is unknown.

The estimated operator exposure levels using glufosinate ammonium, when personal protective equipment (PPE) was used, and when PPE is not available, have not been assessed.

Recommended Precautions (Source: Pesticide Registration Authority, India)

1. Keep away from foodstuffs, empty foodstuff containers and animals feed.
2. Avoid contact with mouth, eyes and skin.
3. Avoid inhalation the spray mist. Spray in the direction of wind.
4. Wash thoroughly the contaminated clothes and parts of the body after spraying.
5. Do not smoke, drink, eat and chew anything.
6. **Wear full protective clothing while mixing and spraying.**

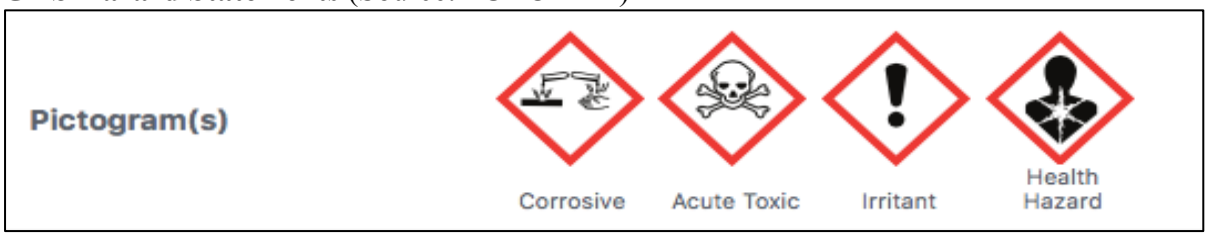
Application Methods and Time

"Tea -Basta15 SL is to be used as post emergence herbicide. It is to be applied when weeds are at active vegetative growth stage/ flowering stage with a spray shield to prevent drift on tea plants and Cottonseed in active vegetative growth**.** It is a non-selective, post emergence herbicide recommended for control of annual and perennial weeds infesting tea gardens, cotton crops and non-crop area. it contains 15% weight /volume glufosinate ammonium (a.i.) which is equal to 13.5% on weight / weight basis[8]." Glufosinate-ammonium is used in the solution form, sprayed on the weeds when the weeds reach the actual growth or vegetation stage. It usually is intended to affect the leaves and the stem of the weeds.

[8] Directorate of PPQS, Government of India
http://www.ppqs.gov.in/sites/default/files/glufosinate_ammonium_13.5_sl93fbasf_india_limited_1.pdf

HEALTH HAZARDS

GHS Hazard Statements (Source: PUBCHEM)



Source: (PUBCHEM https://pubchem.ncbi.nlm.nih.gov/compound/Glufosinate-ammonium#section=GHS-Classification&fullscreen=true)

- **H302** : Harmful if swallowed [Warning Acute toxicity, oral]
- **H312** : Harmful in contact with skin [Warning Acute toxicity, dermal]
- **H314 :** Causes severe skin burns and eye damage [Danger Skin corrosion/irritation]
- **H317 :** May cause an allergic skin reaction [Warning Sensitization, Skin]
- H318 : Causes serious eye damage [Danger Serious eye damage/eye irritation]
- **H332** : Harmful if inhaled [Warning Acute toxicity, inhalation].
- H336 : May cause drowsiness or dizziness [Warning Specific target organ toxicity, single exposure; Narcotic effects]
- **H360** : May damage fertility; Suspected of damaging the unborn child [Danger Reproductive toxicity]
- H370: Causes damage to organs [Danger Specific target organ toxicity, single exposure]
- H372: Causes damage to organs through prolonged or repeated exposure [Danger Specific target organ toxicity, repeated exposure]

RISK ASSESSMENTS

EU: high long-term risk for mammals and High risk to non-target arthropods (EFSA 2012).

Limits on Exposure – residues and tolerances

Exposure route*:* Occupational exposure may occur via inhalation and dermal contact. Glufosinate ammonium is classified as Toxicity Category III or IV for acute oral, dermal, and inhalation toxicity. However, it is not a dermal irritant or a sensitizer. It is not an eye irritant. According to EFSA, use of glufosinate ammonium will lead to farmers/worker exposures that exceed acceptable exposure levels during application, even with PPE (EFSA, 2005). Since glufosinate being a light dependent herbicide and is fully effective in full sunlight, spraying has to be during mid-day. This would mean pesticide workers have to spray in hot and humid conditions, leading to higher exposure.

There are no exposure limits established by the Occupational Safety & Health Administration or the American Conference of Governmental Industrial Hygienists. The WHO/FAO recommended acceptable daily intake (ADI) for glufosinate is

0.02 mg/kg. The European Food Safety Authority has set an ADI of 0.021 mg/kg. The Acute reference dose (ARfD) for child-bearing women is 0.021 mg/kg. Glufosinate ammonium was classified as "not likely to be a human carcinogen." There was no evidence of a treatment-related increase in tumors in either rats or mice[9].

***Residues**:* Residues of weedicides/herbicides in general have not been detected in India. No official data is available in the public domain, on the level and extent of contamination by weedicide/ herbicides in water, soil, and food. However, a study conducted in China (Yan et al, 2022) reported presence of glufosinate in surface water, sediments, cray fish.

Toxicity

- **Acute toxicity:** Acute toxicity is associated with symptoms like nausea, vomiting, convulsions, respiratory distress. Similar symptoms were observed in poisoned victims. A 60-year-old man who ingested this herbicide developed symptoms like respiratory distress, convulsions and increased output of urine (Takahashi et al, 2000). In another reported poisoning cases on the same herbicide the victims developed speech ataxia, systemic tremor, mental disturbances, loss of short-term memory and haematological changes (Hirose et al, 1999; Watanabe and Sano, 1998). Acute toxic effects like hypersensitivity, irregular respiration, hypersalivation, disequilibrium, ataxia, hyporeflexia, diarrhoea and decreased body weight have been observed in exposed laboratory animals (Matsumara et al, 2001; Lapouble et al, 2002; KEMI, 2002). This herbicide can also cause conjunctival discharge, redness and opaqueness in cornea of rabbits (Cox,1996).

  As per a European Food Safety Authority (EFSA 2005) report, the actual health hazards can be harmful effects on living organisms, high risk to mammals, low risk to aquatic animals, birds, bees, earthworms, other soil non-target macro-organisms, and soil micro-organisms. Also, it would be highly harmful if the intake happened, like swallowing or consuming directly into the body. It is also a high risk for the unborn child, and also harmful to the aquatic environment. Even if proper protective measures were used effects would be there, especially on respiratory functions.

- **Chronic toxicity:** It is a reproductive and neurotoxin. It is a possible kidney, blood, bladder and lungs toxicant as well. Exposure towards this herbicide has reported to induce lung inflammations, increased myeloperoxidase, disruption of alveolar septae and seizures in mice (Maillet et al, 2016).

  - **Neurotoxicity:** Glufosinate ammonium poisoning can lead to hippocampal lesions (causes memory impairment) (Mao et al, 2012). In a reported poisoning case, a 57-year-old male developed symptoms like memory loss and cognitive dysfunction following the poisoning (Lee and Kang, 2021). The cause of neurotoxicity can be the binding of glufosinate and its metabolites to NMDA receptors leading to its overstimulation. They are expressed highly in hippocampus. This overstimulation can also lead to neuronal damage and is

[9] USEPA, https://www.federalregister.gov/documents/2012/09/26/2012-23738/glufosinate-ammonium-pesticide-tolerances

associated with memory impairments. They also inhibitis the glutamine synthetase. This causes the irreversible inhibition of glutamine from ammonia leading to accumulation of ammonia in cells. Hyperammonemia leads to neuronic cell death and tissue necrosis (Kim and Min, 2018; Lantz et al, 2014; Mao et al, 2011). It also stimulates the production of nitric acid in the brain leading to symptoms like epileptic seizures after exposure from this herbicide (Nakaki et al, 2000; Lapouble et al, 2002). Excitotoxic mechanism of glufosinate ammonium and the development of associated reversible splenial lesion was studied in a 39-year-old poisoned victim. The victim also developed confusion and amnesia (Jeong et al, 2015). In rats, this herbicide has been reported to induce neuroepithelial cell death, blisters in head, cleft lips and inhibited differentiation of midbrain cells (Watanabe & Iwase, 1996; Watanabe, 1997). Transgenerational effects on brain function was also reported (Fujii, 1997)

- **Reproductive toxicity:** The reproductive toxicity effects of glufosinate ammonium were studied in male lizards exposed to herbicide contaminated soil, against unexposed control lizards. It was clear that testes were affected by oxidative damage and lesions. Alterations in plasma sex hormone levels were also observed as an effect of exposure (Zhang et al, 2019). When the effects of glufosinate ammonium were tested in human sperm mitochondria in a dose-dependent manner, researchers could observe that this herbicide negatively affected mitochondrial respiration efficiency (Ferramosca and Lorenzetti, 2021). Animal studies reported reproductive toxicity and neurological imbalances. In rats' symptoms observed included vaginal haemorrhage, premature deliveries, increased pre & post implantation losses and intra-uterine death of foetuses (KEMI, 2002).
- **Foetotoxicity/teratogenicity:** Adverse effects on embryos were reported, with effects on early embryonic development, damage to neural rube and brain. (Watanabe and Iwase, 1996)
- **Cardiovascular effects:** The formulation Basta at low doses, is known to cause decreased blood pressure and increased heart rate in rats. At higher doses it induced decreased heart rate (Koyama et al, 1997).

Other Human health risk

Studies report that glufosinate is harmful when inhaled, swallowed or through skin contact. Serious health risk may manifest from exposure over longer periods. Assessment of exposed patients shows that acute exposure causes convulsions, respiratory circulatory and central nervous system damages. Chronic exposure studies noted memory loss, changes in brain regions, and autism traits in mice offspring. Potential damage to brain, reproduction, and adverse effects on embryos can happen upon exposure.

The visible effects of the herbicide are neurological and reproductive problems; these are the long-term effects of the herbicide. Human health impacts due to Glufosinate-ammonium can start with the contamination of the drinking water. It is one of the primary routes that cause adverse effects on human health, and also its effects vary according to the sensitivity. The human eye is more sensitive than the skin; on the skin, it would cause itching and, on the eyes, it causes severe eye irritation.

Vulnerable groups: Farmers and farm workers, including women and children, and literally the entire population.

Intentional poisoning is another harmful effect of Glufosinate-ammonium. As it is easily accessible, it becomes handy for intentional poisoning. An ingestion of glufosinate even in small quantities can cause severe poisoning, affecting the central nervous system and heart rhythm. Signs and symptoms may not appear for several hours and may persist for several days and cause severe damages.

There is no antidote known for glufosinate, and hence results in high risk for farming community in India. There are high risks of exposure to users in India, given the fact the recommended PPE of required quality is not available, or accessible, or even if available, the prevailing humid climatic conditions do not allow users to wear them. Because no antidote is available, there is less chance for proper treatment for poisoning (both occupational and intentional). India's Pesticide registration authority information shows that no specific antidote is known. Usually poisoning treatment is done on the basis of the symptoms, and diazepam may be given to control convulsions.

Report of the special rapporteur on right to food (2017) notes that achieving right to adequate food and health requires proactive measures to eliminate harmful pesticides.

"Despite the harms associated with excessive and unsafe pesticide practices, it is commonly argued that intensive industrial agriculture, which is heavily reliant on pesticide inputs, is necessary to increase yields to feed a growing world population, particularly in the light of negative climate change impacts and global scarcity of farmlands. Indeed, over the past 50 years, the global population has more than doubled, while available arable land has only increased by about 10 per cent. Evolving technology in pesticide manufacture, among other agricultural innovations, has certainly helped to keep agricultural production apace of unprecedented jumps in food demand. However, this has come at the expense of human health and the environment. Equally, increased food production has not succeeded in eliminating hunger worldwide. Reliance on hazardous pesticides is a short-term solution that undermines the rights to adequate food and health for present and future generations."

"Implementing the right to adequate food and health requires proactive measures to eliminate harmful pesticides. Corporations have the responsibility to ensure that the chemicals they produce and sell do not pose threats to these rights. There continues to be a general lack of awareness of the dangers posed by certain pesticides, a condition exacerbated by industry efforts to downplay the harm being done as well as complacent Governments that often make misleading assertions that existing legislation and regulatory frameworks provide sufficient protection."

Source: Report of the special rapporteur on right to food. United Nations Human Rights Council. A/HRC/34/48. Dated 24th January 2017. https://documents-ddsny.un.org/doc/UNDOC/GEN/G17/017/85/PDF/G1701785.pdf?OpenElement

Glufosinate Ammonium Poisoning – Global Data

Even though, Glufosinate-containing herbicides are used worldwide, data on acute human glufosinate poisoning however remain scarce (Mao, et al. 2012). A retrospective review of the medical records of all glufosinate poisoned cases reported to the Taiwan National Poison Control Center and two medical centers in Taiwan from August 1993 through February 2010, severe effects can occur and may be associated with older age, larger amount of ingestion and absence of concomitant ethanol consumption. Among patients with oral exposure, 25 were asymptomatic, while the others developed gastrointestinal, neurological, cardiovascular, and/or respiratory manifestations. Glufosinate inhibits glutamine synthetase, an enzyme catalyzing the synthesis of glutamine from glutamate and ammonia. Glutamine synthetase inhibition leads to the accumulation of intracellular ammonia (Takano, et al. 2020).

Since glufosinate irreversibly inhibits glutamine synthetase, leading to intracellular accumulation of ammonia, hyperammonemia is considered one of the main mechanisms of glufosinate ammonium toxicity in humans. Whether hyperammonemia causes neurotoxicity has not yet been studied fully. One study however concludes, hyperammonemia cannot be assumed as the cause of neurotoxicity in glufosinate ammonium poisoning (Kim, J. et al, 2022).

Acute glufosinate poisoning could result in various moderate-to-severe central nervous system (CNS) toxicities such as stupor, coma, seizures, and amnesia (Watanabe & Sano, 1998). Glufosinate, structurally similar to glutamate, could lead to both CNS excitation and depression in animals and humans. Neurologic effects observed after glufosinate exposure are proposed to be caused by glufosinate, its metabolites, or glufosinate induced imbalance between glutamate, ammonia and glutamine; and the toxic mechanism mediated through N-methyl-aspartate (NMDA) receptor. Gastrointestinal effects were the most common manifestations following glufosinate-containing herbicide exposure, which might result from the irritative effect of the anionic surfactant present in the formulation. Other reported acute symptoms of poisoning include disturbances in speech and vision, coma, seizures, elevated WBC count, liver enzymes, metabolic acidosis and depressed serum cholinesterase (HSD, 2003).

Management of acute glufosinate poisoning remains supportive, since there is no antidote. Early GI decontamination and administration of activated charcoal may be recommended for patients with significant oral ingestion, similar to paraquat. Appropriate fluid therapy and close monitoring of major central nervous system, cardiovascular, and respiratory effects should also be performed on a routine basis, especially for those patients who are prone to develop severe toxicity. Intensive care would then be needed for patients developing life-threatening manifestations. Hemodialysis and/or hemoperfusion had been conducted in some glufosinate poisoned patients without definite efficacy. Glufosinate is cleared by the kidney 1.6 – 1.8 times more than that by hemodialysis. Hence, the usefulness of hemodialysis/hemoperfusion in severe glufosinate poisonings awaits better evaluation.

ENVIRONMENTAL FATE AND EFFECTS

Impact of glufosinate on natural resources and environment remains under-studied. However, impacts cannot be ruled out, given the widespread and consistent usage over years.

In aquatic systems, this herbicide is expected to adsorb onto suspended solids and sediments. Volatilization is not a major process. It dissolves in the soil simply as compared to other herbicides, and therefore it pollutes the water sources quickly. It dissolves in the liquid media easily, therefore, it contaminates the water sources. Thus, it can reach the groundwater, especially in sandy soils. The half-life in water of glufosinate is expected to be less than 300 days.

Impact on Soil

In soil, glufosinate ammonium primarily undergoes microbial degradation with a half-life of 3-11 (aerobic)/ 5-10 (anaerobic) days. It is transformed to 3-methylphosphinyl-propionic acid and 2-methylphosphinyl-acetic acid and then to carbon dioxide. But there are reports which reveals the persistence of this herbicide. It persisted for 113 days in Canadian soils and 12-70 days in Californian vineyard soils (Smith and Belyk, 1989; US EPA 1992b).

The effect of Glufosinate Ammonium 13.5% SL on soil physico-chemical properties and microflora population on tea soil of Darjeeling, West Bengal, India was investigated over two years (2014 and 2015). Effect of herbicides on bulk density, water holding capacity, moisture content, soil pH, organic matter content, electrical conductivity, as well as total nitrogen, available phosphorus and available potassium contents were analyzed along with microflora population of rhizosphere soil (total bacteria, actinomycetes and fungi) (Ghosh, et al. 2017). This study, declared as supported by M/S Crystal Crop Protection Pvt. Ltd., Corp., concludes that "Ammonium 13.5% SL applied at different doses did not show any long run adverse effect on rhizosphere region of Tea crop."

As per a 2008 USEPA, it has received 48 ecological incident reports that have been linked to the use of glufosinate-ammonium. The majority of the incidents (45) involved damage to corn. Another of these incidents involved a 'massive' fish kill in a farm pond in Maryland after application of glufosinate-ammonium to plants surrounding the farm pond[10].

Impact on Food Chain

As glufosinate is often used as a pre-harvest desiccant, residues can also be found in foods that humans ingest. Such foods include potatoes, peas, beans, corn, wheat, and barley. In addition, the chemical can be passed to humans through animals who are fed contaminated straw. Flour processed from wheat grain that contained traces of glufosinate was found to retain 10-100% of the chemicals' residues (*Watts, M. 2008*).

[10] USEPA, https://www.regulations.gov/document/EPA-HQ-OPP-2008-0190-0003

The herbicide is also persistent; it has been found to be prevalent in spinach, radishes, wheat and carrots that were planted 120 days after the treatment of the herbicide. Its persistent nature can also be observed by its half-life which varies from 3 to 70 days depending on the soil type and organic matter content. Residues can remain in frozen food for up to two years and the chemical is not easily destroyed by cooking the food item in boiling water. The EPA classifies the chemical as 'persistent' and 'mobile' based on its lack of degradation and ease of transport through soil.

All India Network Project on Pesticide Residues in India has not studied on glufosinate ammonium residues. This Network was established by the government to organise, promote, co-ordinate and conduct location-specific research at national level on analysis of pesticide residues in food commodities, soil and in water for ensuring food safety and security[11].

Impact on Biodiversity

Many other organisms that are helpful like spiders, predatory mites, and butterflies are endangered due to the toxicity of this herbicide. Microorganisms help in the breakdown of minerals, and decomposition, and also increase productivity, this herbicide is toxic to them and increases susceptibility to plant diseases. Long-term use of herbicide gives rise to herbicide-resistant weeds and therefore the production units are insisted to produce more strong herbicides, this results in more toxicity and depletes the sustainability of soil, fields, and seeds.

Eco toxicology – Acute toxicity (Source: PPDB)

Mammals	- Moderate acute toxicity
Birds	- Low acute toxicity
Earthworms	- Low acute toxicity
Honeybees	- Low acute toxicity
Fish	- Low acute toxicity
Aquatic invertebrates	- Low acute toxicity
Aquatic crustaceans	- Moderate acute toxicity
Aquatic plants	- Moderate acute toxicity
Algae	- Low acute toxicity
Beneficial insects-	- High toxicity (KEMI 2002).

[11] ICAR, https://aicrp.icar.gov.in/pesticide/

GLUFOSINATE AMMONIUM STATISTICS

In India, according to the data available in the web site of the Directorate of Plant Protection, Quarantine and Storage, Ministry of Agriculture, Government of India, the volume consumption of glufosinate is 1 MT for the year 2021-2022. Production/import/export data is not available. Approved sources of import for India include BAYER (Germany and USA) and M/S Shijiazhuang Richem Co. Ltd., China. Indigenous manufacture in India is UPL Limited, Mumbai[12].

United States of America, Ecuador, Vietnam were the leading Importers of Product Glufosinate Ammonium from India (HS CODE: 38089390) with a market share of 95.89% with an exports value of US$ 13.7 Million[13]. United States of America has a market share with 92.18%, followed by Ecuador with 2.27% and Vietnam with 1.44%. JNPT is the leading export port for Product Glufosinate Ammonium goods. It handled 79.1% of the country's Product Glufosinate Ammonium cargo. This port handled roughly US$ 11.3 Million worth of exports in 2,017. Hazira comes second and it handled 18.45% of the country's Product Glufosinate Ammonium exports. This port handled roughly US$ 2.6 Million worth of exports in 2,017.

Ankleshwar comes third in the rank and it handled 2.46% of the country's Product Glufosinate Ammonium exports items. This port handled roughly US$ 351.7 Thousand worth of exports in 2,017. As per Global buyer's Directory compiled from global Glufosinate Ammonium exports data, there are total 3 Exporters and 7 Importers of Glufosinate Ammonium.

Markets and Growth of Glufosinate

Glufosinate which started off as a natural herbicide, quickly became synthetic. Glufosinate was first commercialized in the USA and Canada in 1993-1994. Its growth matches the emergence of genetically modified crops, especially herbicide-tolerant crops. It is also linked with glyphosate growth and the challenges therein. Historical data of consumption of glufosinate has to be collected to understand this growth phenomena, essentially in United States. The estimated treated area with glufosinate in the world was approximately 12 million ha per year in 2014.

Indicating powerplay and market competition, growth of glufosinate in China is attributed to the ban of paraquat. Ban on paraquat in China, Brazil and Thailand has led to growth of glufosinate. As per a industry report, "Glufosinate is worth 2.8 times the cost paraquat in terms of cost per hectare, and will replace glyphosate at a ratio of 6:4. Together, with the amount of glyphosate to be limited and banned, it is estimated that the additional demand for glufosinate arising from the substitution will exceed 4,000 metric tons in 2020. In the long-term, the ban on paraquat in many major agricultural countries will drive a considerable demand for glufosinate."

[12] Approved source of import and list of indigenous manufacturers of insecticides in India, as on 01.07.2021.
http://ppqs.gov.in/sites/default/files/source_of_import_and_list_of_indigenous_manufacturers_of_insecticide_as_on_01.07.2021.pdf

[13] https://www.voleba.com/india/product-glufosinate-ammonium-exports-data.html

Glufosinate is widely used in the Midwest and Southern United States where the majority of glufosinate-tolerant soybean and cotton are planted in North America. In rice, orchards, vineyards, minor crops, and non-agricultural areas of the western United States glufosinate use has been found. Continous application of herbicides including glufosinate contributed to the evolution of herbicide-tolerant weeds, known as super weeds.

Approximately 800,000 pounds of glufosinate were sold in Minnesota in 2017.[1] A 2013 USDA: NASS Survey, indicated that glufosinate was applied to 1% of corn acres, and 2% of soybean acres in the state. It was applied on up to 11% of the acres in specific counties (Wilkins). As glyphosate resistant weeds become more prevalent, glufosinate use was expected as alternative herbicide option. As per a survey conducted in 2021 to estimate the amounts and types of pesticides applied in 2020, in Maryland, USA, glufosinate-ammonium usage was 5,496 pounds and was ranked 38 among the pesticides used.

Since 1995, glufosinate tolerant and multi-tolerant GM crops have been successively promoted and applied on a commercial scale, especially in field crops, such as soybean, rape and cotton. The market demand for glufosinate was 8,000 metric tons in 2018, and is expected to maintain a 5% growth rate in 2020, an increase of some 1,000 metric tons. It is estimated that the global glufosinate market will grow at a rate of 4.5% to 5.0% from 2020 to 2025, exceeding 40,000 metric tons, based on market for herbicide-tolerant crops, ban on paraquat, the planting of glufosinate-tolerant crops, combined formulations and other factors.

Table: Effective Global Production Capacity and Output of Glufosinate in 2019 (metric tons)[14]

S. No.	Company	Location	Capacity/t	Production
1.	BASF	Frankfurt, Michigan	12,000	12,000
2.	LIER Chemical	Mianyang and Guang'an in Sichuan Province	12,000	11,500
3.	UPL	India	6,000	5,500
4.	Shandong Yisheng	Jining in Shandong Province	5,000	5,000
5.	Yongnong Bioscience	Shangyang in Zheijiang, Ningxia	5,000	2,500
6.	Veyong biochemical	Shijiazhuang in Heibei Province	1,600	1,000
7.	Shijiazhuang Ruikai Chemical	Shijiazhuang in Heibei Province	1,500	1,000
			43,100	38,500

[14] https://www.kingquenson.com/Industry_News/glufosinate-ammonium.html

Global Market

The global glufosinate ammonium market was highly fragmented in 2019. Key manufacturers operating in the global glufosinate ammonium market include:

- Bayer
- Syngenta
- Aventis
- Certis
- Fargro
- Hoechst
- Lier Chemical
- Jiangsu Huangma
- Dow AgroSciences
- Hebei Veyong Biochemical Co., Ltd.

Glufosinate use will likely continue to increase for 3 reasons: 1. With glyphosate-resistance growing among weeds, glufosinate is seen as an alternative, and also to other herbicides likes paraquat and dicamba 2. glufosinate is not considered volatile and the newer transgenic technologies for herbicide-tolerant crops tend to stack multiple events, including glufosinate resistance as one of their traits in most varieties. 3. no new effective and non-selective herbicide is expected to launch in the near future.

Glufosinate-ammonium's trade names and identifiers include:

- Glufosinate-ammonium
- GLUFOSINATE AMMONIUM
- Basta
- Ammonium glufosinate
- Basta F1
- Gluphosinate ammonium
- Liberty (pesticide)
- Finale 14SL
- Ignite ISC Herbicide
- Caswell No. 580I
- BIP0679
- Glufosinate-ammonium [ISO]
- HSDB 6666
- DL-Phosphinothricin
- EINECS 278-636-5
- EPA Pesticide Chemical Code 128850
- Ammonium (DL-homoalanine-4-yl)methylphosphinate
- Ammonium (3-amino-3-carboxypropyl)methylphosphinate

Source: https://pubchem.ncbi.nlm.nih.gov/compound/11564649#section=Names-and-Identifiers

HERBICIDE RESISTANCE

Resistance to glufosinate

Current herbicide and herbicide trait practices are in a status of limbo, owing to the rapid spread of glyphosate-resistant weeds. Researchers thought glyphosate is needed when glyphosate-tolerant crops became available. An argument was developed that because weeds were becoming widely resistant to most commonly used herbicides, turning weed management too complex and time consuming for large farm operations, Glyphosate usage can be easy and efficient by controlling all emerged weeds at a wide range of application timings. But then weeds developed resistance to glyphosate too. The intensive use of glyphosate led to the widespread resistance among weeds. Research has not come up with any answer yet. Today, weeds that are resistant to glyphosate and other herbicides are threatening current crop production practices.

All commercial herbicide usage methods are over 20 years old and have resistant weed problems. Yet, the research to discover new weed management technologies is continuing. Companies are making efforts to bring new generation of crops with resistance to glyphosate, glufosinate and other herbicides, aimed to bring monopoly using dangerous technologies. Other practices include new chemical, biological, cultural and mechanical methods for weed management. India has been known for its cultural and physical methods of weed management.

Herbicide Tolerant Crops

Glufosinate-tolerant crops were first introduced for commercial production in 1995. Herbicide-tolerant crops have had a profound impact on weed management. Most of the impact has been by glyphosate-tolerant maize, cotton, soybean and canola. Continuous use of glyphosate with glyphosate-tolerant crops over broad areas facilitated the evolution of glyphosate-resistant weeds, which have resulted in increases in the use of tillage and other herbicides with glyphosate. Transgenic crops with resistance to auxinic herbicides, as well as to herbicides that inhibit acetolactate synthase, acetyl-CoA carboxylase and hydroxyphenylpyruvate dioxygenase, stacked with glyphosate and/or glufosinate resistance, were expected in the market (Duke, 2015). As the pesticide and seed industry are working together, herbicides with new modes of action and technologies such as bioherbicides, sprayable herbicidal RNAi and/or robotic weeding may affect the role of transgenic, herbicide-tolerant crops in weed management.

Conventional cotton varieties could not withstand glufosinate (Carbonari, 2016). Insertion of the gene encoding phosphinothricin acetyltransferase (PAT) has resulted in cotton plants becoming tolerant to the herbicide glufosinate. The presence of this gene provided a level of resistance to glufosinate in terms of visual injury and effect on photosynthetic electron transport. The injury is proportional to the amount of ammonia accumulation. The strong promoter associated with bar expression in the glufosinate-tolerant cultivar led to high RNA expression levels and PAT activity which protected this cultivar from glufosinate injury. In comparison, even though the insect-tolerant cultivar demonstrated a good level of resistance to glufosinate, its safety margin is lower than that of the glufosinate-tolerant cultivar. Therefore, it was suggested that the farmers should be extremely careful in using glufosinate on cultivars not expressly designed and commercialized as tolerant to it.

Later, a synthetic phosphinothricin acetyltransferase (PAT) gene has been introduced via Agrobacterium tumefaciens into dicot crops, such as like tobacco, tomato, spring and winter rapeseed, alfalfa, and several horticultural crops. This gene was also introduced into maize. All transgenic crop plants tolerated a two- to threefold field dosage of Basta.

Phosphinothricin acetyltransferase (pat) gene confers resistance to glufosinate by transforming this herbicide into N-acetyl-l-glufosinate (NAG). The pat gene was inserted in six maize hybrids (Herculex, Agrisure TL, Herculex Yieldgard, Leptra, Viptera 3, Power Core) as a selectable marker (Krenchinski, 2018). Glufosinate-tolerant transgenes has been identified in weedy relatives of *Brassica napa (*Oilseed rape) in Japan (Saji, et al, 2005).

Adverse effects related to herbicide tolerant crops

Several studies reported adverse effects as a result of cultivating genetically engineered crops, including increased incidences of chronic health effects among people, and increase in use of pesticides. An Argentina study on the health effects of transgenic herbicide tolerant crops reported fourfold increase in rate of birth defects and three time increase in childhood cancers in a decade where rice and soy crops are heavily sprayed with herbicides (Houllier F. 2012). A report (Bardocz, et al. 2012) based on clinical data from Argentina noted increased incidences of acute and chronic health effects in people associated with increased cultivation of genetically modified crops. It noted increased incidences of birth defects, miscarriages, and cancers, increased risk for conceiving and genetic damage leading to cancers and birth effects, increased liver toxicity, impaired neurodevelopment functions in children, kidney failure, respiratory problems and allergy.

An analysis by Benbrook (2012) concluded that, in contrary to the claim that genetically engineered crops would reduce pesticide usages; herbicide tolerant crops in effects had led to increased usage of herbicides, indicating overall increase of pesticide use in number and volume between 1996 and 2011 in the United States.

Joint FAO/WHO Meeting On Pesticide Residues (JMPR) and The Joint FAO/WHO Meeting On Pesticide Specifications (JMPS)

Glufosinate-ammonium was previously evaluated by JMPR in 1991 and 1999[15]. In 1999, the Meeting considered reports on the relevance of glutamine synthetase activity in the liver, kidney and brain of experimental animals and humans. The 2012 JMPR Meeting confirmed the conclusion of the 1999 JMPR, concluded that glufosinate-ammonium is not carcinogenic in mice or rats. Glufosinate-ammonium was tested for genotoxicity in an adequate range of studies of genotoxicity in vitro and in vivo. No evidence for genotoxicity was observed in any test. In view of the lack of genotoxicity and the absence of carcinogenicity in mice and rats, the JMPR 2012 Meeting concluded that glufosinate-ammonium is unlikely to pose a carcinogenic risk to humans.

The WHO Panel of the 2012 JMPR established an Acute Reference Dose (ARfD) of 0.01 mg/kg bw for glufosinate-ammonium. On the basis of the data obtained from supervised residue trials the Meeting concluded that the residue levels listed below are suitable for establishing maximum residue limits and for IEDI and IESTI assessment.

Definition of the residue for (compliance with the MRL and for estimation of dietary intake) for animal and plant commodities: *sum of glufosinate, 3 [hydroxy(methyl)phosphinoyl]propionic acid and N-acetyl-glufosinate, calculated as glufosinate (free acid). The residue is not fat soluble.*

The JMPR 2014 meeting concluded that the long-term intake of glufosinate–ammonium resulting from the uses considered by the current JMPR is unlikely to present a public health concern.

[15] https://www.fao.org/agriculture/crops/thematic-sitemap/theme/pests/jmpr/jmpr-rep/en/

CONCLUSION

In conclusion, glufosinate is a neurotoxin, reproductive toxin and can have adverse effects on biodiversity, it is advisable that use of glufosinate use is not allowed. Given the socio-economic conditions and use patterns of pesticides in India, all sections in the society, including farmers and farm workers, women and children, and literally the entire population are vulnerable to exposure either directly or indirectly.

Allowing herbicide tolerant crops will increase the use of this highly hazardous herbicide in the near future, possibly causing adverse effects in the long run for people, and environment. Sustainable agriculture production can be adversely affected, and can lead to failure of farm produce from meeting domestic and international standards.

Use of technology or development in agriculture should be something that does not harm the environment, ensuring farmer and worker safety as well as safe food production. However, development of transgenic, herbicide tolerant crops will pave way for monopoly in seed and agrochemical sector at the cost of farmers, farm labour and people in India as well as the environment. In the light of precautionary principle and assuring the rights guaranteed by the Article 21 of the Constitution of India, it is wiser not to allow glufosinate use, as well as any agriculture seed technology that demands use of this hazardous herbicide.

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PAN INDIA

GLUFOSINATE AMMONIUM
An Overview

It presents information related to the regulation, use, toxicity, health effects and adverse effects on environment. Glufosinate is a broad-spectrum herbicide acknowledged to have adverse effects on nervous system, reproductive system, cardiovascular system, and on developing fetus in animals. Glufosinate ammonium does not have known antidotes, making it difficult for saving lives of individuals who are poisoned. It is banned in 29 countries. It is approved for use in India for tea and cotton, however, wide spread non-approved use has been reported. Studies show harmful effects on organisms including mammals, beneficial insects, aquatic animals, birds, bees, earthworms, as well as micro organisms. Introduction of glufosinate tolerant crops will pave way for its wider use, as it tends to increase dependency on this herbicide. It would result in higher exposure to humans and animals, thus increasing likelihood of adverse effects.

About the Authors

Dr. Narasimha Reddy Donthi is a public policy expert, and has been a passionate campaigner on environmental and development issues. He was a Board member of IFOAM-Asia, Member of a Cotton Advisory Board Committee and Advisory Council, of Textile Exchange (a global non-profit). He has contributed to public discourse and policy changes in electricity, seed, rice, cotton, sugarcane, sericulture, handloom and textiles, land, water and other related areas. An intense team player, he has built campaigns, advocacy programmes and policy change projects. He is an author, writing on different subjects in regional, national and international publications. He also guides students on their Ph.D and other research activities. He has extensively worked on pesticide related issues such as poisoning, community health, and policy over the past 30 years.

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