

Glufosinate ammonium

Chemical name: 2-amino-4-(hydroxymethylphosphonyl)butanoic acid monoammonium salt

Synonym: butanoic acid

CAS: 77182-82-2

MF: C₅H₁₅N₂O₄P

MW: 198.6

Liquid, with pH 6.5±1.

Solubility: soluble in water.

Major use

Glufosinate ammonium (GA) is the active ingredient of non-selective, broad-spectrum herbicides. It is available in liquid formulation often under name "BASTA", consisting of 20% w/v GA and 33% w/v polyoxyethylene alkylether, an anionic surfactant. Some other names of the pesticides containing GA are: Rely, Finale, Challenge and Pestanal [1].

Human toxicity

Poisoning from accidental and suicidal ingestion is gradually increasing in Japan, where this herbicide is widely used (15-20 deaths occurred annually after 1991). Cases of poisoning may occur worldwide, because this herbicide is now available in major European, American, and Asian countries.

Ingestion of BASTA with suicidal intention, ranging from 20 to 500 ml (corresponding 3.7 to 92.5 g GA) was fatal for 19.4% patients (6 of 31) by the fourth hospital day. Two patients died from respiratory failure when GA doses of BASTA were greater than 5.5 ml/kg (corresponding 1036 mg/kg GA) [2].

In cases of acute intentional GA poisoning, when specimens of serum from 15 victims were analysed, the GA concentrations in serum varied over a wide range between 0.1 and 1000 mg/l [3].

Among clinical symptoms occurring shortly after ingestion are nausea, vomiting and diarrhea. Other symptoms at the acute exposure are lowered blood pressure, fever and metabolic acidosis. Later, 8 to 24 h after ingestion, the neurological symptoms including seizures, disturbances in eye movement and consciousness disturbance may occur. In fatal cases, respiratory failure, systemic circulatory failure, general edema, gastric erosion, and coma were reported [1].

The anionic surfactant presenting in GA containing herbicides, polyoxyethylene alkylether alone may cause circulatory failure, respiratory failure, seizures, lung edema, and gastric erosion [1].

The World Health Organization (WHO) classifies GA in a toxicity class III, "slightly hazardous". The WHO classifying system is based on the LD50 values for rats.

Carcinogenicity: not listed (EPA, 2004).

Kinetic data

Absorption: GA is rapidly absorbed from the gastrointestinal tract.

Volume of distribution (V_d) is > 1 l/kg, e.g. 1.44 l/kg, respective 1.92 l/kg, in two human cases [4].

Distribution half-life following ingestion of 60 g GA was reported to be 1.8 h in 65-year-old man [5].

Peak blood concentration was reached within 90 min after ingestion [5].

Serum level at 3 h after ingestion of 60 g GA was reported to be 0.44 mg/ml, which decreased by a bi-exponential curve up to 42 h after ingestion [5].

Elimination half-life following an ingestion of 60 g GA was 9.6 h [5].

Metabolism and excretion

GA is a structural analogue of glutamate, or more precisely, it is an analogue of glutamate-containing phosphine amino acid, the active ingredient of non-selective herbicides.

Data about metabolism in humans are limited; however, it is known that in rat GA undergoes oxidative deamination.

Main GA metabolites in rat are:

3-(hydroxy(methyl)phosphinoyl)propionic acid and 3-methyl-phosphinico propionic acid [1].

Excretion: 78 to 95 % of GA is excreted in the urine within 24 to 36 h [1].

Toxicological mechanism

Toxic effects of GA on the CNS may be possibly explained by interference with the neurotransmitter function of endogenous glutamate. GA inhibits glutamine synthetase resulting in decreased glutamine level [6].

In a post-mortem study, the most significant lesions found in the brain were a primary injury of astrocytes, which likely contributed to GA effects on the CNS [7].

Target organ: CNS.

References

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