Diquat dibromide

Chemical name: 1,1'-ethylene-2,2'-bipyridylium dibromide ion
CAS: 85-00-7
MF: $C_{12}H_{12}Br_2N_2$
MW: 344.06
logKow= –4.6 at 20°C.
Solubility: very well soluble in water (700g/l at 20°C); also soluble in methanol (25 g/l); slightly soluble in alcohols and hydroxylic solvents; practically insoluble in nonpolar organic solvents [1, 2].

Major uses
Diquat dibromide is a non-selective herbicide used for pre-harvest desiccation of seed crops, e.g. rice, clover, rape, peas, beans, maize etc.; for desiccation of potato haulm; and as a herbicide in carrots, onions, vines etc. It is also used for aquatic weed control [3].

Trade names include Weedol, Weedtrine-D, Reglone, Vegetrole, Aquacide and many others. The water solution of diquat dibromide is often formulated as 200 g/l [1, 3]. Technical concentrate contains 502 g/l diquate dibromide [2].

Human toxicity
Diquat is a moderately toxic compound, with a little evidence of a neurotoxic effect. An accidental or intentional human poisonings by diquat occur seldom.

At the lower acute oral doses (less than 1 g diquat), nausea, vomiting, abdominal pain and diarrhea (often bloody) may occur within a few hours after oral ingestion. Renal impairment may develop, but recovery generally occurs after several days [4].

Ingestion of concentrated diquat solution may result in severe systemic toxicity. Moderate to severe poisoning develops after ingestion of 1 to 12 g. Ingestion of 6 g (100 mg/kg for a 60 kg person) or more may be fatal. Toxicity can affect all organs and result in death within 24 to 48 h. Larger doses can produce ulcerations of the digestive tract, pulmonary edema, acute liver and kidney failure, cardiac arrest, and rapid loss of consciousness [5]. Deaths have occurred after ingestion of 20 to 50 ml of Reglone (diquat dibromide 200 g/l) [5].

Diquat serum concentration levels greater than 0.5 mg/l during the first 24 h after ingestion have been associated with systemic poisoning (data from Chevron Emergency Information Center) [4]. In one case of fatal intent poisoning (ingestion of 50 ml of Reglone), the diquat serum concentration level of 4.5 mg/l (13 μM) was measured [6].

The intact skin is a very effective barrier against the absorption of diquat. Human studies have demonstrated that only about 0.3% can penetrate through the skin [4].

TLV/TWA* is 0.5 mg/m^3 for 8 h [3].

Carcinogenicity: diquat dibromide is not considered to be a carcinogen in humans [4].

Kinetic data
Absorption: Only a small portion of an oral dose is absorbed from the gut in experimental animals.

Elimination half-life of diquat is expected to be less than 24 h (animal data) [4].

Metabolism and excretion
Only animal data are available. In rats, only a small percentage undergoes metabolism. Two metabolites were detected: diquat monopyridone and diquat dipyridone. Both these metabolites are less toxic than diquat itself.

Excretion: diquat and its metabolites are excreted in the urine and feces. Based on ^{14}C-diquat rat oral dosing studies, about 85% to 90% of the dose remained in the feces, the rest recovered in the urine [4].

Toxicological mechanisms
Diquat belongs to the same class of bipyridyl compounds as paraquat, however, its acute and chronic effects differ from those of paraquat: effect on the lung is not observed in the case of diquat [7]. Diquat causes, first of all, the kidney damage, namely renal tubule damage, which can progress to acute oliguric renal failure.

The mechanism of diquat action in plants and in mammals is similar: diquat, like paraquat, undergoes a single electron addition to form a free radical, in the presence of NADPH and cytochrome P450 reductase. The diquat radical reacts with oxygen to form a superoxide anion radical which is highly reactive. The superoxide radicals can react with each other forming hydrogen peroxide and molecular oxygen, a reaction that may occur spontaneously or via the enzyme superoxide dismutase [9]. The hydroxyl radical attacks unsaturated lipids of cell membranes and produce lipid hydroperoxide. The latter forms lipids free radicals with consequent membrane damage and cell death [7, 8].

Target organs: kidney, gastro-intestinal tract.

References
1. Extention Toxicology Network (EXTOXNET). http://extoxnet.orst.edu/pips/diquatdi.htm
2. Handbook of Pesticide Toxicology (1991) W.J.Hayes, Jr., E.R. Laws, Jr., eds., V. 3, pp. 1376-1380, Academic Press, Inc.
3. European Commission, Peer Review Programme, Diquat, (1996) V. 3, Annex B.
4. Poisindex, Thomson Micromedex (2005).
5. Ellenhorn's Medical Toxicology, 2nd ed. (1997) pp. 1637-1638, William & Wilkins.
6. Vanholder R, Colardyn F, De Reuck J, Praet M, Lameire N, Ringoir S (1981) Diquat intoxication: Report of two cases and review of the literature. Am J Med 70, 1267-1271.
7. Casarett and Doull's Toxicology (1986). C.D. Claassen, M.O. Amdur, J. Doull, eds. 3rd ed., Macmillan Publishing Company, New York.
8. Jones GM, Vale JA (2000) Mechanisms of toxicity, clinical features, and management of diquat poisoning: A review. Clin Toxicol 38 (2) 123-128.

*Threshold Limit Value / Time Weighted Average. The average concentration under which most people can work consistently for eight hours.

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