E X T O X N E T

Extension Toxicology Network

A Pesticide Information Project of Cooperative Extension Offices of Cornell University, Michigan State University, Oregon State University, and University of California at Davis. Major support and funding was provided by the USDA/Extension Service/National Agricultural Pesticide Impact Assessment Program.

Pesticide
Information
Profile

Diquat dibromide

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TRADE OR OTHER NAMES

Aquacide, Dextrone, Reglone, Reglox, Weedtrine-D, Aquakill, Vegetrole, Deiquat, Reglon, Tag.

REGULATORY STATUS

Diquat dibromide is classified as a general use herbicide by the U.S. Environmental Protection Agency (EPA) (7). A registration standard was issued by EPA in June, 1986. Check with specific state regulations for local restrictions that may apply to this chemical.

Based on its assessment by EPA as a moderately toxic material, containers of diquat bear labels with the signal word, "WARNING" (34).

INTRODUCTION

Diquat dibromide is an herbicide and plant growth regulator. It is a quick-acting contact herbicide, causing injury only to the parts of the plant to which it is applied (18, 38). It is nonselective, meaning that it does not spare 'nontarget' plants from its herbicidal effects (21). Diquat is referred to as a desiccant because it causes a leaf or an entire plant to dry out quickly. It is not residual, that is, it does not leave any trace of herbicide on or in plants, soil, or water. It is used to desiccate potato vines and seed crops, to control flowering of sugarcane, and for industrial and aquatic weed control (35, 39).

TOXICOLOGICAL EFFECTS

ACUTE TOXICITY

Diquat dibromide is a moderately toxic chemical (34). It may be fatal to humans if swallowed, inhaled, or absorbed through the skin (4). Concentrated solutions may cause severe irritation of the mouth, throat, esophagus and stomach followed by nausea, vomiting, diarrhea, severe drying out of bodily tissues, gastrointestinal discomfort, chest pain, diarrhea, kidney failure, and toxic liver damage (24, 36, 42). Very large doses of the herbicide can result in convulsions and tremors (36). Rats given lethal doses of diquat showed few signs of illness during the first 24 hours. They then exhibited lethargy, pupil dilation, respiratory distress, weight loss weakness and finally death over

the course of 2 to 14 days after dosing. Similar patterns of symptoms occurred in mice, guinea pigs, rabbits, dogs, cows and hens (38).

Diquat dibromide is acutely toxic when it is absorbed through the skin and the possibility for poisoning increases with repeated exposure (35). Dermal adsorption is higher where the skin is cut or abraded (36). Although absorption is reportedly low following dermal exposure, the demonstrated toxicity of this compound is sufficient to raise serious human health concerns. Small amounts of diquat can cause skin irritation and sores, as well as delayed healing of cuts and wounds (37). When absorbed through the skin, some commercial concentrate formulations of diquat can cause symptoms similar to those that occur when it is eaten. There have been reports of workers who have had softening and color changes in one or more fingernails after contact with concentrated diquat dibromide solutions. In some instances, the nail was shed, and did not grow in again (18). A single dose of diquat was not irritating to the skin of rabbits. Repeated dermal doses cause mild redness, thickening, and scabbing (38).

Diquat dibromide also causes eye irritation (5, 27). Several cases of severe injury to human eyes have been reported after accidental splashings. In each case, initial irritation was mild, but after several days, serious burns and sometimes scarring of the cornea developed (12). Moderate to severe membrane irritation occurred when diquat was put in the eyes of rabbits (5).

Direct or excessive inhalation of diquat dibromide spray mist or dust may result in oral or nasal irritation, nosebleeds, headache, sore throat, coughing, and symptoms similar to those from ingestion of diquat (5, 18).

The amount of a chemical that is lethal to one-half (50%) of experimental animals fed the material is referred to as its acute oral lethal dose fifty, or LD50. The lower the LD50 is for a chemical, the more toxic it is; the higher the LD50, the less toxic it is. The oral LD50 for diquat in rats is 120 mg/kg, 233 mg/kg in mice, 188 mg/kg in rabbits, 187 mg/kg in guinea pigs and dogs. Cows appear to be particularly sensitive to this herbicide, with an oral LD50 of 30 to 56 mg/kg (1, 42). The acute dermal LD50 for diquat dibromide is 250-400 mg/kg in rabbits (37).

CHRONIC TOXICITY

Cataract formation is the most significant effect of chronic exposure to diquat that is currently recognized (35). Cataracts, a clouding of the eyes which interferes with light entering the eye, occurred in rats and dogs given 2.5 mg/kg and 5 mg/kg of diquat, respectively (12). The number of cases of cataracts increased in test animals (cats and dogs) as the amount of diquat was increased in their diets. This is referred to as a dose-dependent association between cataracts and diquat ingestion (5, 1). A single, near-fatal dose will not produce cataracts. Chronic exposure is necessary (38).

The effects of repeated, or prolonged, dermal contact with diquat dibromide range from inflammation of the skin, to general bodily ('systemic') poisoning, as evidenced by injury to internal organs, primarily the kidneys. Chronic exposure may damage skin, which allows more absorption of the herbicide (5). Repeated applications of 42 mg/kg of diquat killed four out of six rabbits tested (24). While rats fed 50 mg/kg of diquat for two years did not die from testing, their food intake and growth was decreased (1).

Repeated inhalation exposure of rats to 1.9 mg/m3 caused inflammatory changes in connective tissues, damage to the kidneys and heart, abnormal levels of several liver enzymes, low white blood cell counts, high red blood cell counts, and depressed cholinesterase activity (42).

Reproductive Effects

Diquat dibromide does not cause reproductive effects (35). It did not reduce fertility when tested in experimental animals (4). Rats receiving 25 mg/kg decreased their food intake and showed slowed growth, but had unchanged reproduction. Fertility was reduced in male mice given diquat dibromide during different stages of sperm formation (18). Neither fertility nor reproduction was affected in a three-generation study in rats given dietary doses of 0, 12.5 or 25 mg/kg/day of diquat dibromide, although some growth retardation was seen at the 25 mg/kg/day dose (38).

Teratogenic Effects

EPA does not consider diquat capable of causing teratogenic effects (35). However, diquat dibromide is thought by other researchers to have the potential to cause birth defects. It is referred to as an experimental teratogen based on a study which showed teratogenic effects in six-day pregnant rats given intravenous injections of diquat. A lowest published toxic dose, or TDLO, of 7 mg/kg resulted from this study (27). Growth retardation was seen in test animals given extremely high doses of diquat. No deformities were found in the unborn offspring of pregnant rats that were injected intraperitoneally with 0.5 mg/kg of diquat daily during organogenesis, the stage of fetal development in which organs are formed (12). Pregnant rats died when they were injected with 14 mg/kg of diquat dibromide. Upon examination of the unborn rats, there was evidence of skeletal defects of the collar bone, as well as little or no ear bone formation (28). While no actual teratogenesis occurred in rats given single abdominal injections during the 7th to 14th days of pregnancy, many rats did not have normal weight gain and bone formation in the unborn was decreased (27).

Mutagenic Effects

EPA has required more testing on the capability of this herbicide to cause mutations, since available information is contradictory (35). Diquat dibromide is not known to cause permanent changes in genetic material and is therefore not considered a mutagen (18). No mutagenic effects were seen in mice given ten mg/kg of diquat orally for five days (6).

Carcinogenic Effects

Diquat dibromide is not classified as a tumor-causing chemical (6, 15, 35). An 80-week feeding study showed that dietary doses of 15 mg/kg/day of diquat did not cause tumors in rats (10). Likewise, dietary levels of 36 mg/kg/day for two years did not induce tumors in rats (38).

Organ Toxicity

Based on records of suicidal ingestion of diquat by humans as well as diquat-feeding studies of monkeys, it has been concluded that diquat is most harmful to the gastrointestinal tract (GIT), kidneys, and liver. Severe congestion and ulceration of the stomach and bowel are produced by the herbicide. After large doses of diquat are given, there is evidence of stretching and irritation of the GIT and thickening of the walls of the alveoli, or air cells of the lungs (15). When enough diquat is given, the fat in the liver goes through extreme changes. Acute death occurs in the cells of the small glandular tubes that process urine in the kidney. Cataracts are caused when smaller amounts of diquat are given. While diquat dibromide appears to primarily affect the tissue lining of the eye lens and the kidneys, water is apparently removed from other tissues as well. Dehydration can result. The amount of water which is removed depends on how much diquat dibromide is given (18). Poisoning by diquat may affect the liver and kidneys (42).

Fate in Humans and Animals

Diquat dibromide may be absorbed by humans following oral ingestion, dermal exposure or inhalation of spray mists. Studies indicate that absorption from the gut into the bloodstream is low. Oral doses of diquat are metabolized mainly within the intestines rather than in the body proper, with metabolites being excreted in the feces. Only a small percentage of oral doses are absorbed into the bloodstream and then excreted in the urine. When rats were fed radio labeled diquat, only 6% of the dose was recovered in the urine. When exposure occurs through routes other than oral, diquat enters the bloodstream and is rapidly eliminated in the urine. Following subcutaneous injection in rats, excretion of about 90% of the dose occurred in the urine on the first day and almost all of the remainder on the next day (15, 21, 31, 35, 38). Complete elimination of the herbicide was seen in urine and feces of rats within four days of administration of oral doses of five to ten mg/kg of diquat dibromide (17). When diquat was fed to hens, 70- 80% was unchanged when it was excreted in the feces (6). In cattle 0.004 to 0.015% of an oral dose was recovered in the milk and 0.4 to 2.6% of the dose was found in the urine (38).

ECOLOGICAL EFFECTS

Effects on Birds

Diquat dibromide ranges from moderately toxic to practically nontoxic to birds, depending on the species (35). Its acute oral LD50 in twelve young male mallards was 564 mg/kg. Signs of poisoning in these birds included instability, wing-drop and lack of movement (19). The oral LD50 for diquat was 200-400 mg/kg in hens (6).

Effects on Aquatic Organisms

Diquat dibromide is slightly toxic to fish. Its toxicity to fish, and food organisms on which fish survive, has been reported in many studies. It appears to be less toxic in hard water (30). The lethal concentration fifty, or LC50 is that concentration of a chemical in air or water that kills half of the experimental subjects exposed to it for a specific time period. The 8-hour LC50 for diquat in rainbow trout is 12.3 ppm, and 28.5 ppm in Chinook salmon (25). The 96-hour LC50 in northern pike is 16 ppm and 20.4 ppm in fingerling trout (13, 29). The shell growth of eastern oysters was not noticeably affected with exposure to 1 ppm of diquat for 96 hours (25).

Some species of fish may be harmed, but not actually killed, by sublethal levels of diquat dibromide. Oxygen can become depleted in diquat-treated water by decaying aquatic plants. This decreases the amount of oxygen available for fish survival. Research indicates that yellow perch suffer significant respiratory stress when herbicide concentrations in the water are similar to those normally present during aquatic vegetation control programs (3). Strip application of the herbicide over water is recommended to prevent large scale fish kills (4).

There is little or no bioconcentration of diquat dibromide in fish. Bioconcentration is the buildup or accumulation of a chemical in plants and/or animals. One investigation into the persistence of diquat in fish showed that one half of the herbicide was lost in less than three weeks (20, 25).

Effects on Other Animals (Nontarget species)

Diquat dibromide is not toxic to honey bees. Cows are particularly sensitive to the toxic effects of this material (1, 17).

ENVIRONMENTAL FATE

Breakdown of Chemical in Soil and Groundwater

When diquat dibromide comes in contact with soil, it becomes strongly adsorbed to clay particles or organic matter in the soil for long periods of time (Koc - 100,000 g/ml). The strong chemical bonds formed by diquat adsorption to soil particles make the herbicide biologically and chemically inactive. That is, in certain soils it is unlikely to be:

a. carried away, or leached, by water seeping through the soil;
b. taken up by plants;
c. broken down by microbes in the soil in a process called microbial degradation; or
d. broken down by sunlight (photochemical degradation).

Traces, or residues, of diquat have been found to persist in soil for many years with very little degradation (8, 21, 32, 36, 40).

Soil capacity for adsorption of diquat is so high in comparison to the rates at which it is applied that there is little possibility that leaching or groundwater contamination will occur. Field and laboratory tests show that diquat usually remains in the top inch of soil for long periods of time after it is applied (31, 32). However, there is also evidence that diquat has the ability to eventually use up, or saturate, all the available adsorption sites on soil clay particles. Groundwater quality can be affected if soil adsorption sites become totally saturated because water moving down through the soil can carry any non adsorbed herbicide into the groundwater. More research is needed for a better understanding of the potential effects on groundwater of long- term, repeated use of diquat.

Studies on the erosion of diquat-treated soils near bodies of water indicate that diquat stays bonded to soil particles, remaining biologically inactive in surface waters, such as lakes, rivers and ponds (32).

Breakdown of Chemical in Water

Since diquat is purposely applied to water to control the growth of aquatic weeds, its ability to last as an effective residue has been studied carefully. These studies suggest that diquat is not persistent in water (14). When diquat is applied to open water, it disappears rapidly because it binds to suspended particles in the water. These particles are then taken up by plants. Diquat dibromide's half-life, or the period of time that it usually takes for half of the amount of the material to be broken down by natural processes, is less than 48 hours in water (31). As affected plants decompose, the adsorbed diquat rapidly disappears from open waters. Disappearance may be due to degradation by microbes or sunlight, or due to adsorption to bottom sediments. Twenty-two days after a weed infested artificial lake was treated, only 1% of the applied diquat remained in the water and 19% was adsorbed to sediments. Adsorbed diquat was subject to microbial degradation (41). Diquat has been found in the bottom soil of pools and ponds four years after application (12). Diquat will photodegrade in surface layers of water in 1 to 3 or more weeks when it is not adsorbed to suspended particles (32, 41).

The EPA requires a 14-day interval between treatment of water with diquat dibromide and use of treated waters for domestic, livestock, or irrigation purposes. Swimming, fishing and watering of domestic animals should not be allowed for at least 14 days after application of the herbicide to water. The herbicide cannot be used for any purpose in commercial fish processing areas (35).

Breakdown of Chemical in Vegetation

Diquat is rapidly absorbed by the leaves of plants. Usually, plant tissues are killed too quickly to allow translocation to other parts of the plant. The herbicide interferes with cell respiration, the process by which plants take in oxygen. The chemical structure of diquat is not changed or degraded within plants. Rather, after it is sprayed on plants or dead/decaying vegetation, diquat is broken down on the surface by photochemical degradation (17). The resulting intermediate residues of diquat are incorporated with the plant materials into the soil, where, through a process called 'microbial degradation, they are changed into carbon dioxide by soil microorganisms (21, 32, 36). Diquat dibromide is also rapidly absorbed by weeds in water, which causes the concentrations of the material in plant tissue to be higher than in surrounding water. Low concentrations of the herbicide in water are adequate for controlling aquatic weeds (31).

Since diquat dibromide is a nonselective herbicide with non-crop use patterns that overlap endangered plant habitats, it may present a danger to nontarget plants, including endangered species (35).

PHYSICAL PROPERTIES AND GUIDELINES

Technical diquat, which is greater than 95% pure, forms white to yellow crystals (38). It is usually marketed in an aqueous solution which is dark reddish-brown (22, 36).

Diquat is highly soluble in water. It usually decomposes in alkaline solutions but is stable in neutral or acidic solutions (2). Diquat degrades when it is exposed to the ultraviolet rays of sunlight (17).

Diquat contains small amounts of a highly toxic chemical impurity, ethylene dibromide (EDB). The National Institute for Occupational Safety and Health (NIOSH) has recommended an occupational exposure limit for EDB of 0.13 ppm during any 15-minute sampling period (5). When heated to decomposition, very toxic fumes are emitted from diquat (27). Diquat is a nonvolatile chemical. This means that it will not vaporize and become a gas (24).

Diquat is stable under normal temperatures and pressures, but it may pose a slight fire hazard if exposed to heat or flame. It poses a fire and explosion hazard in the presence of strong oxidizers. Thermal decomposition of diquat will release toxic oxides of nitrogen and carbon and toxic and corrosive fumes of bromides (42).

Diquat is corrosive. Commercial use formulations of diquat contain corrosion inhibitors, ingredients that slow down or prevent corrosion. In spite of corrosion inhibitors, concentrated formulations will corrode metals, especially aluminum, and should be stored only in the original container. It should not be put into food or drink containers (31). While it can be mixed with most herbicides, diquat dibromide is incompatible with anionic wetting agents such as alkyl sulfamates (17).

To reduce skin exposure to diquat dibromide, protective clothing should be worn when handling the concentrated product, and splashes should be immediately washed away from eyes and skin. Skin and eye contact with diquat, especially from drift, should be avoided (17). Breathing diquat spray (mist) should also be avoided (2). Respiratory equipment is recommended, especially in situations in which exposure to drifting spray is possible.

A 24-hour reentry interval is set by EPA for crop sites, golf courses, and rights-of-way that are treated with diquat dibromide. A 14-day period is also required by EPA between diquat treatment of water bodies and use of treated water for domestic, swimming, irrigation, and/or livestock

feeding purposes (35). Diquat dibromide may not be used where commercial fish processing is practiced (9, 32).

Occupational Exposure Limits:

0.5 mg/m3 OSHA TWA
0.5 mg/m3 ACGIH TWA
0.5 mg/m3 NIOSH recommended TWA (42)

Physical Properties:

CAS #:	85-00-7
Specific gravity:	1.22 to 1.27 (36)
H2O solubility:	very soluble, 70 gm/100 ml in 20 degrees C water (38)
Solubility in other solvents:	insoluble in non-polar organic solvents such as chloroform, diethyl ether, and petroleum ether (1); slightly soluble in alcohol and hydroxylic solvents (35)
Melting point:	335 - 340 degrees C (22)
Decomposition point:	decomposes above 300 degrees C (38)
Vapor pressure:	negligible, less than 10-5 mbar at 20 degrees C; does not volatilize very much from water or soil (17)
Koc:	100,000 g/ml (42)
Kow:	Log octanol/water coefficient = -3.05 (31)
Chemical Class/Use:	heterocyclic cationic herbicides -- bipyridilium quaternary ammonium salts
NOEL:	for cataract formation in rats: 0.22 mg/kg/day; in dogs: 1.7 mg/kg/day (35)
ADI:	0.005 mg/kg, based on a NOEL of 0.5 mg/kg and a safety factor of 100 (11)
PEL:	Health Advisory for drinking water (EPA): tolerances have been established in potable water (0.01 ppm) (2)
STEL:	1.0 mg/m3 (31)

BASIC MANUFACTURER

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Review by Basic Manufacturer:

Comments solicited: November, 1992.
Comments received:

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