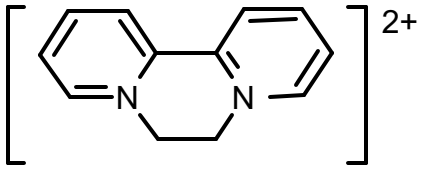
III.2 DIQUAT



DIQUAT

SUMMARY

Diquat (6,7-dihydrodipyridol[1,2-a:2',1'-c] pyrazinediium ion) is a water-soluble contact type, nonselective herbicide that is used to control many submerged and floating aquatic macrophytes and some types of filamentous algae in static and low-turbidity water (Klingman, Aston and Noordhoff, 1975 as cited in Aquatic Plant Identification and Herbicide Use Guide, 1988). Diquat binds very strongly and rapidly to sediments and once bound, it is very persistent (Reinert and Rodgers, 1987). When used as an aquatic herbicide at recommended application rates, diquat residues in water decrease rapidly to essentially undetectable levels within 7-14 days (State of Washington, 1984). The rate of diquat bioconcentration in fish is negligible (Reinert and Rodgers, 1987).

The common name, diquat, refers to the cation, which is responsible for the herbicidal action of the salt. The associated anion (i.e., bromide) has no effect on the herbicidal activity.

Many studies have been conducted using diquat addressing both toxicity and environmental fate and persistence. The EPA approved a Reregistration Eligibility Decision (RED) for diquat dibromide in July, 1995.

REGISTERED PRODUCTS IN MASSACHUSETTS

The current list of aquatic herbicides containing diquat that are registered in Massachusetts can be accessed at http://www.state.ma.us/dfa/pesticides/water/Aquatic/Herbicides.htm

on the Massachusetts Department of Agricultural Resources (DAR) Aquatic Pesticide Website. The DAR updates this list regularly with changes. In addition, the DAR can be contacted directly at (617) 626-1700 for more specific questions regarding these products.

DIQUAT USES AND APPLICATION

Diquat can be used to control both submerged and floating weeds. For submerged weeds, the diquat can be injected below the water surface or it can be applied directly into the water while moving slowly over the water surface in a boat. For floating plants, the foliage should be thoroughly wetted with diquat using either surface or aerial spraying (Herbicide Handbook, 1983; Aquatic Plant Identification and Herbicide Use Guide, 1988). Turbid or muddy water or mud-coated vegetation greatly reduces the effectiveness of diquat as the herbicide becomes adsorbed to particles (Aquatic Plant Identification and Herbicide Use Guide, 1988). Improved efficacy of diquat can often be achieved when applied in a mixture with complexed copper formulations (Aquatic Plant Identification and Herbicide Use Guide, 1988). In some cases it is recommended that diquat be applied with water carrier, thickener or invert emulsion carrier. The following adjuvants are recommended for use with diquat: for aerial applications, a nonionic surfactant to improve the ability of diquat to penetrate waxy plant cuticles (e.g., Ortho X-77 Spreader); for submersed growth, a polymeric thickener to improve sinking, herbicide confinement and contact properties (e.g., Nalquatic) (Aquatic Plant Identification and Herbicide Use Guide, 1988).

A formulation of diquat dibromide aquatic herbicide) targets the list of aquatic plants in Table III-2-1 (Zeneca, 1994). This herbicide controls the aquatic plants in Table III 2-1.

Diquat can be used at anytime during the growing season although control of early growth is recommended. Treatment of dense weed areas may result in oxygen loss from decomposition of dead weeds. The loss of oxygen may cause fish suffocation. Therefore, treat only 1/3 to 1/2 of the dense weed areas at a time and wait 14 days between treatments (Zeneca, 1994).

For specific information on recommended application rates for a particular product, the product label should be consulted. The USEPA Office of Pesticide Programs (OPP) has a link to a database of product pesticide labels at http://www.epa.gov/pesticides/pestlabels/. A list of the weeds that these products control, which has been compiled from the Environmental Protection Agency (EPA) registration labels for these products, is contained in Table III.2-1.

MECHANISM OF ACTION

Diquat's herbicidal activity and organic chemical reactions of diquat formulations are dependent only on the diquat cation and are not influenced by the nature of the associated anion, since the salts are mostly dissociated in aqueous solution (Herbicide Handbook, 1983). Diquat is absorbed readily by foliage through the cuticle of the leaf. Absorption is rapid, resulting in concentrations in plant tissues well above that in surrounding water so that very low concentrations (i.e., 0.1-1.5 ppm) in water will give effective control (HSDB, 1994). No absorption through buried plant roots occurs due to the rapid binding and inactivation of diquat by sediments (Aquatic Plant Identification and Herbicide Use Guide, 1988). Diquat is translocated only locally in plant tissues (Aquatic Plant Identification and Herbicide Use Guide, 1988). Diquat's mode of action is not clear but it is known that the mechanism is light-dependent (USEPA, 1992a; HSDB, 1994). Diquat interferes with the photosynthetic process, releasing strong oxidizers that rapidly disrupt and inactivate cells and cellular functions (Aquatic Plants Management Program for Washington State, 1992). This action results in the rapid death of the foliar parts of practically all plant species (HSDB, 1994).

ENVIRONMENTAL FATE/TRANSPORT

The available database for diquat indicates that dissipation following application is very rapid, initially by mixing and subsequently by adsorption by plants and sediments (USEPA, 1994). Once diquat reaches the sediments, it is tightly bound and is biologically unavailable.

Diquat is stable in neutral or acid conditions but hydrolyzes in the presence of alkaline materials including alkaline waters (Herbicide Handbook, 1979 as cited in HSDB, 1994). Volatilization and oxidation of diquat are insignificant fate processes.

Diquat is subject to photochemical degradation in surface layers of water in 1-3 or more weeks when not adsorbed to particulate matter (Sanborn, 1977). A 50% loss of diquat was noted within 48 hours when exposed to a UV source (Simsiman *et al.*, 1976 as cited by Reinert and Rodgers, 1987). A photodecomposition half-life of 1.6 weeks was calculated from the results of a study in which diquat in 20-cm glass petri plates was subjected to natural sunlight (Smith and Grove, 1969 as cited in Reinert and Rodgers, 1987). Diquat has a reported photolysis half-life of 2-11 days (Reinert and Rodgers, 1987). Despite the above information, photodegradation is not considered a major fate process for diquat in aquatic environments (Simsiman *et al.*, 1976 as cited by Reinert and Rodgers, 1987).

The photochemical breakdown of diquat on plant surfaces and in water exposed to sunlight releases 1,2,3,4-tetrahydro-1-oxopyrido-[1,2-a]-5-pyrazinium ion (TOPPS) as the major degradation product.

Further irradiation produces picolinamide and then degrades further via picolinic acid to volatile fragments (Smith and Grove, 1969 as cited in Aquatic Plants Management Program for Washington State, 1992). When a solution containing 5 ppm of diquat was exposed to sunlight during May and June, 70% of the diquat was degraded in 3 weeks. Picolinic acid and TOPPS were major photodegradation products (Smith and Grove, 1969 as cited in HSDB, 1994). A secondary degradation pathway results in diones and, to a limited extent, to monopyridone (Aquatic Plants Management Program for Washington State, 1992).

The major fate process for diquat in water is its propensity for rapidly binding to sediments. This property is due to its double positively charged diquat cation and clay minerals present in soil. The diquat cation may also insert itself between the layer planes of certain minerals such as montmorillonite (Reinert and Rodgers, 1987). Diquat may also incorporate into humus and/or become physically adsorbed to organic matter and particles (Aquatic Plants Management Program for Washington State, 1992). About 80-95% of diquat introduced into a flask containing sediment/water was sorbed to the sediment within 2 days (Simsiman and Chesters, 1976). Diquat is characterized by a fairly high octanol-water (K_{ow}) partition coefficient of 603 and adsorption coefficients (K_{oc}) ranging from 205-691ml/g based on various sediment types (Reinert and Rodgers, 1987). Once bound, diquat is no longer bioavailable. See Table III.2-4 for a list of environmental parameters of diquat.

Studies have shown that unbound, biologically available diquat can be biodegraded by bacteria in the laboratory. However, because of the rapid adsorption of diquat to sediments in the environment which renders it unavailable to biodegradation, the opportunity for microbial decomposition is not very great (Calderbank, 1968 as cited in Hamer, 1994). Thus, while diquat may disappear relatively quickly from water, it does tend to persist in sediments. In one study conducted with diquat in pond water, diquat disappeared from the water within days of treatment but persisted in the sediments for over 160 days (Frank and Comes, 1967 as cited in Reinert and Rodgers, 1987). Nevertheless, it has been shown that biodegradation does occur in various sediment/water systems although at a very slow rate. After 65 days, only 0.88% and 0.21% of diquat was converted to CO_2 and water under aerobic and anaerobic conditions using water and sediment from a eutrophic lake and negligible using water from an oligotrophic lake (Simsiman and Chesters, 1976 as cited by HSDB, 1994).

Table III.2-1. List of Weeds Controlled by Diquat

Common Name	Scientific Name
SUBMERSED AQUATICS:	
Bladderwort	*Utricularia*
Coontail	*Ceratophyllum demersum*
Elodea	*Elodea* spp.
Naiad	*Najas* spp.
Watermilfoil	*Myriophyllum* spp.
Hydrilla	*Hydrilla verticillata*
Pondweeds	*Potamogeton* spp.
FLOATING AQUATICS:	
Salvinia	*Salvinia* spp.
Water Hyacinth	*Eichhornia crassipes*
Water Lettuce	*Pistia stratiotes*
Duckweed	*Lemna* spp.
Pennywort	*Hydrocotyle* spp.
MARGINAL WEEDS:	
Cattails	*Typha* spp.
ALGAE:	
Filamentous green algae	*Pithophora* spp.
	Spirogyra spp.

(Zeneca, 1994)

Diquat does not tend to bioconcentrate to an appreciable degree in fish and other aquatic organisms. No diquat residues were detected in channel catfish collected from pools five months after a single application or two months after a second treatment of 1 ppm diquat (HSDB, 1994). Diquat did not significantly accumulate in fish with bioconcentration factors of $\leq$2.5X with rapid depuration once fish are in pesticide-free water. In laboratory flow-through systems, diquat did not accumulate to a significant degree in *Daphnia*, mayfly nymphs and oysters, with maximum bioconcentration factors of 32X. Depuration was rapid for all organisms (USEPA, 1994). Reported bioconcentration factors for aquatic (non-plant) organisms range from <1 - 62 (USEPA, 1994).

When sprayed on the surface of ponds in a dissipation study conducted in Florida, diquat mixed quickly both laterally and by depth in the water column. Diquat was removed from the water column with a half-life of $\leq$ 2 weeks. Most of the recovered diquat was bound to the first five centimeters of soil, with small amounts recovered from the 5-10 cm layer. Diquat is very persistent but due to its strong soil absorptive properties, it is unlikely to be a groundwater contaminant. When applied to surface water systems, diquat will most likely be associated with the sediment (USEPA, 1994).

PHARMACOKINETICS

In rats given oral doses of ^{14}C-labeled diquat dibromide or diquat dichloride, absorption of diquat through the gastrointestinal tract was very low. About 4-11% of the original dose was excreted within 48 hours in the urine and about 84-97% of the original dose was excreted in the feces. Biliary excretion in rats administered an oral dose of diquat was less than 5% of the administered dose within 24 hours. Most of the recovered radioactivity in rats was found to be unchanged diquat. Metabolic breakdown products of diquat include diquat monopyridone and diquat dipyridone in the urine and diquat monopyridone in the feces (USEPA, 1992a).

Absorption of diquat in dogs was somewhat higher than in rats. 29-32% of the orally administered dose was recovered in the urine within 3 days after dosing. 51-62% was recovered in feces (USEPA, 1992a).

Absorbed diquat tends to preferentially accumulate in the kidney, although it was also detected in other tissues. Single oral doses of 116-230 mg diquat ion/kg/day of diquat in dogs yielded diquat tissue concentrations of less than 3 µg/g and kidney concentrations of up to 10 µg/g after 4 hrs. Four to 48 hours after the dose was administered, the diquat residues decreased (USEPA, 1992a).

In an 8-week feeding study with rats administered 12.5 mg diquat ion/kg/day, tissue concentrations of diquat were less than 1 µg/g in the brain, liver, lung, stomach and small and large intestines. During the latter part of the experimental exposure period, diquat concentrations in the kidney and large intestine increased to greater than 1 µg/g. Within one week of return to a control diet, no diquat was detected in any tissue (USEPA, 1992a).

In rats given 116-125 mg diquat ion/kg/day, absorbed diquat was relatively uniformly distributed among tissues. At 2-30 hours postexposure, concentrations were slightly higher in the kidney than in other tissues. In rats given an oral dose of 231 mg diquat ion/kg/day, elevated levels of diquat were found in heart and lung tissue 2 hours after dosing but by 24 hours these levels had decreased and the levels in the kidney had increased between 24-48 hours. *In vitro* studies indicate that diquat accumulates in the kidney but not in the other tissues (USEPA, 1992b).

HEALTH EFFECTS

Avian:

A series of lethal doses and lethal concentrations of diquat were identified for birds in acute toxicity studies. A number of these have been summarized below:

Table III.2-2. Acute Toxicity Studies with Diquat in Birds

SPECIES	TYPE	RESULTS	REFERENCE
3-4 mo. old mallard chicks	oral LD50	564 mg/kg	USDWFS, 1984
mallard	oral LC50	>5,000 ppm	USDIFWS, 1975
bobwhite chicks	oral LC50	2932 ppm	USDIFWS, 1975
14-day old Japanese quail chicks	oral LC50	1346 ppm	USDIFWS, 1975
10-day old ring-necked pheasants	oral LC50	3742 ppm	USDIFWS, 1975

Mammalian:

Acute:

Symptoms of diquat poisoning include vomiting, diarrhea, general malaise, possible kidney and liver damage, dyspnea and pulmonary edema. Tremor and convulsions may occur with very large doses (Herbicide Handbook, 1979 as cited in HSDB, 1994). Workers who have skin contact with concentrated diquat solutions have shown a change in color and softening of one or more fingernails. Inhalation of dust or mist of the compound has led to nosebleeds and the mists may also cause skin irritation, irritation of the mouth and upper respiratory tract, cough and chest pain (Booth and McDonald, 1982 as cited in HSDB, 1994). Ingestion of concentrated solutions of diquat can cause severe irritation to the mucous membranes of the mouth, pharynx, esophagus and stomach. Ulceration and perforation may follow (Arena, 1979 as cited in HSDB, 1994).

Diquat is known to have a profound effect on the distribution of body water. Oral exposure with diquat increases gastrointestinal water content and results in hemoconcentration (USEPA, 1992a). Ingestion of diquat results in dehydration and gastrointestinal ulceration resulting in the vomiting of blood. Acute tubular necrosis of the kidney has also been reported resulting in anuria and increased blood levels of BUN and creatinine (USEPA, 1982 as cited in HSDB, 1994). Dehydration usually plays a key role in causing death from ingestion of diquat (USEPA, 1992a). Diquat administered subcutaneously is expected to be up to 20 times more toxic than via the oral route (USEPA, 1992a).

A number of cases of acute diquat poisoning in humans were reported in the literature. Of ten cases involving ingestion of diquat, six resulted in death. All six cases involved ingestion of at least 15 ml diquat and were characterized by clinical symptoms of toxicity involving the gastrointestinal tract, the brain and the kidney. The quantities ingested by these individuals were much higher than the amounts individuals swimming in waters treated at recommended application rates would ingest or absorb. In the remaining cases, which were characterized by ingestion of no greater than 5 ml diquat, no deaths occurred but gastrointestinal and renal tract damage was observed (USEPA, 1992a).

The acute oral toxicity of diquat in mammals is moderate. Reported acute toxicity values for diquat in mammals include an oral LD50 of 430 mg diquat ion/kg/day in the rat and >26 mg diquat ion/kg/day in the dog. These relatively high levels are attributed to the poor absorption of diquat through the gastrointestinal tract (USEPA, 1992a).

Rats exposed to 100-200 mg/kg diquat ion/kg had minor histopathological changes in the gastrointestinal tract, kidney and liver. An oral Lowest Observed Adverse Effect Level (LOAEL) was determined to be 18.4 mg diquat ion/kg for a single dose of diquat (based on an increase in water content in the gastrointestinal tract) (USEPA, 1992a).

Monkeys that died after being exposed to 100-400 mg of diquat ion/kg showed distinct exfoliation of the gastrointestinal tract epithelium and distinct pathological changes in kidneys (USEPA, 1992a).

Rats that were administered an LD50 dose of 166 mg diquat ion/kg were lethargic, showed signs of piloerection and weight loss, uncharacteristic, off-color feces, gross abdominal swelling, muscular twitching, erratic gait and, the most notable effect, an increase in gastrointestinal water content and hemoconcentration (USEPA, 1992a).

At LD50 doses of 100-200 mg diquat ion/kg in dogs and 100 mg diquat ion/kg in rabbits, perforation of the stomach wall was noted (USEPA, 1992a).

A single oral dose of 99 mg diquat ion/kg produced a marked decrease in renal excretory function. At 166 mg ion/kg, hemoconcentration and a significant reduction in renal plasma flow were observed. At LD50 levels, minimal pathological changes in the kidney were observed in rats at LD50 dose levels. Researchers have concluded that effects on kidney function observed after exposure to diquat are mainly due to body fluid redistribution. Pathological changes were observed in kidneys of monkeys receiving single oral doses of 100-400 mg diquat ion/kg (USEPA, 1992a).

In rats receiving acute lethal doses of diquat intraperitoneally and in monkeys receiving oral doses of diquat, minimal effects on the liver were noted. An increase in liver glycogen and blood glucose appeared to be mediated by altered adrenal secretion. Selenium-deficient rats, given 3.6 mg/kg diquat via intraperitoneal exposure were characterized by rapid and massive liver necrosis accompanied by a marked increase in hepatic liver peroxidation (USEPA, 1992a). A series of other acute toxicity studies conducted with various species yielded the toxicity values summarized in Table III 2-3.

Subchronic:

In a 4-week dietary study, a No Observed Adverse Effect Level (NOAEL) of 6.7 mg diquat ion/kg/day was identified in Charles River CD female rats (USEPA, 1992a).

Oral exposure of rats with either 2.1 or 4.3 mg/kg/day of diquat for four and one-half months produced lung damage characterized by apparently dose-related papillomatous proliferations of the bronchial and bronchiolar epithelia. In addition, moderate to severe alveolar damage was produced in mice exposed either intratracheally or intraperitoneally (USEPA, 1992a).

Table III.2-3. Acute Toxicity Studies With Diquat

SPECIES	TYPE	RESULTS	REFERENCE
Rabbit	dermal LD50	>750 mg/kg	Hartley and Kidd, 1983 as cited in HSDB, 1994
Cattle	oral LD50	30 mg/kg	Clark and Hurst, 1970 as cited in HSDB, 1994
guinea pig	oral LD50	100 mg/kg	Clark and Hurst, 1970 as cited in HSDB, 1994
Mouse	oral LD50	106-146 mg/kg	Clark and Hurst, 1970 as cited in HSDB, 1994
Rabbit	oral LD50	72-138 mg/kg	Clark and Hurst, 1970 as cited in HSDB, 1994
Rat	oral LD50	194-274 mg/kg	Clark and Hurst, 1970 as cited in HSDB, 1994

In rats exposed to 500 and 1,000 mg/l diquat in drinking water for 20 and 8 days respectively and in rabbits exposed to 100 and 500 mg/l for 6 and 10 days respectively, no irritation of the digestive mucosa was noted (USEPA, 1992a).

Chronic:

Chronic feeding studies conducted in dogs, guinea pigs and rats resulted in the formation of cataracts. Cataract formation is cited as the most sensitive toxic indicator of diquat exposure. Diquat-induced formation of cataracts was found to be both dose and time-dependent in laboratory animals. In rats exposed for two years or longer to doses as low as 1.8 mg diquat ion/kg/day, a high frequency of cataract formation was noted. The minimal effective dose in rats was 2 mg/kg/day in drinking water. In a two-year study conducted with rats, cataracts were noted in animals exposed to 3.28 and 17.16 mg diquat ion/kg/day. A NOAEL of 0.22 mg/kg/day was identified for rats in this study. In another study in rats exposed to concentrations up to 36 mg diquat ion/kg/day, an extensive examination of hematology, urinalysis and gross and microscopic pathological examination showed no effects (other than in the eye) at any treatment level. Animals exposed to higher doses experienced more serious effects sooner. No effects were noted in rats exposed to 0.36 mg diquat ion/kg/day. No cataracts were noted in dogs exposed to 1.2 mg/kg/day for 4 years or at a dose level of 0.58 mg diquat ion/kg/day for 3 years. A LOAEL of 3.6 mg diquat ion/kg/day for dogs was identified from this study . A NOAEL for guinea pigs was identified in another study as 0.1 mg diquat ion/kg/day (USEPA, 1992a).

Developmental/Reproductive Effects:

No reproductive or teratogenic effects were observed in mice, or rats after oral diquat administration. In a mouse study in which animals received five daily oral doses of 10 mg diquat ion/kg as well as in a three-generation rat study in which animals were given 25 mg diquat ion/kg, no reproductive effects were reported in the parental, F_1 or F_2 generations. No significant teratogenic effects were observed in mice, rats or rabbits. However, teratogenic effects were produced with diquat administered intraperitoneally or intravenously. In rats administered a single intravenous dose of 8 mg diquat ion/kg, an increase in the number of dead and resorbed fetuses was observed. In addition, skeletal abnormalities were found in the embryos of mice exposed to 1.4 and 5.9 mg diquat ion/kg and rats exposed to 7.5 mg diquat ion/kg after treatment of dams with single intraperitoneal doses (USEPA, 1992a).

Mutagenicity:

The potential mutagenicity of diquat was tested in a number of bacterial and eukaryotic systems with contradictory results. Both positive and negative results were found in *Salmonella* assay, unscheduled DNA synthesis and mitotic gene conversion assay. Diquat induced recessive lethal damage in *Aspergillus* but not in *Drosophila* (USEPA, 1992a).

Carcinogenicity:

In four feeding studies conducted in rats and one in mice (in which only doses of up to 75 mg diquat ion/kg were given for periods of up to two years) no tumors were detected. However, two of these studies had insufficient data upon which to base any final conclusions. Under the old EPA carcinogen classification system, the U.S. EPA OPP determined that diquat was an E carcinogen (i.e., having evidence of noncarcinogenicity in humans) based on a lack of tumor production in rats and mice. Under EPA's current classification system involving the designation of descriptors for summarizing weight-of-evidence, the old E designation corresponds to the descriptor, "not likely to be carcinogenic to humans".

Available Toxicity Criteria:

The Environmental Protection Agency (EPA) Carcinogen Risk Assessment Verification Endeavor (CRAVE) RfD/RfC Workgroup has developed an oral Reference Dose (RfD) of 0.0022 mg/kg/day for diquat based upon a 1985 2-year dietary rat study. The RfD is an estimate, (with uncertainty spanning perhaps an order of magnitude) of a daily exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime (USEPA, 1992b). The World Health Organization (WHO) has also developed an RfD of 0.002008 mg/kg/day. The EPA Office of Pesticide Programs (OPP) has developed an RfD of 0.005 mg/kg/day based upon a 1-year feeding study in dogs. (USEPA, 1995).

In addition, the EPA has also developed a Maximum Contaminant Level Goal (MCLG) of 0.02 mg/l for drinking water and has promulgated this value as a Maximum Contaminant Level (MCL) standard (USEPA, 1992b; USEPA, 1995b). Massachusetts has adopted this value as a drinking water standard, known as a Massachusetts Maximum Contaminant Level (MMCL).

ECOLOGICAL TOXICITY

Aquatic Organisms :

Acute flow-through type bioassays have been conducted with a variety of freshwater and marine fish and invertebrates. Because dissipation of diquat is very rapid, a comparison of toxicity data generated in the laboratory to expected diquat concentrations following application and dissipation in the field, indicates that acute effects on organisms in the field are unlikely at rates used for vegetation control (Hamer, 1994; MacKenzie, 1971 as cited in Aquatic Plants Management Program for Washington State, 1992).

Invertebrates:

LC50 toxicity values for invertebrates show a range of sensitivities to diquat. The most sensitive organisms tested were *Daphni*a and *Hyalella* with 24-hour LC50 values of 1-2 ppm and 0.6 ppm, respectively (Hamer, 1994). Studies of estuarine organisms in Florida, showed no adverse effects on oysters, shrimp or fish (Wilson and Bond, 1969 as cited in Aquatic Plants Management Program for Washington State, 1992). In a pond study, diquat had no direct effect on aquatic insects but a decrease in pond weeds after treatment did result in migration of some species to shoreline vegetation (Hilsenhoff, 1966 as cited in Aquatic Plants Management Program for Washington State, 1992). An application with 0.5 ppm diquat in another pond led to loss of aquatic vegetation. The decaying vegetation appeared to benefit certain organisms such as Oligochaetes, indicated by an increased number (Tatum and Blackburn, 1962 as cited in Aquatic Plants Management Program for Washington State, 1992).

Dragonflies, damselflies and tendipedids exposed to diquat concentrations 40 times the maximum field application rate, survived. *Hyalella* was very sensitive to diquat as was *Cladocera* although *Cladocera* populations returned to normal levels after diquat concentrations disappeared from the water (Gilderhus, 1967 as cited in Aquatic Plants Management Program for Washington State, 1992.)

The species discussed above are all water column or epibenthic organisms (with the exception of the Oligochaetes). Because diquat is very persistent in sediments, it would seem that infauna or deposit-feeding organisms would have the highest potential for exposure to this compound. No specific studies pertinent to this issue were available.

Vertebrates:

The toxicity of diquat varies with the size and type of fish as well as the softness or hardness of the water. Reported LC50 values from one source ranged from 12-90 mg/l for 24-hour exposures, 6-44 mg/l for 48-hour exposures and 4-36 mg/l for 96-hour exposures (Calderbank, 1972 as cited in State of Washington, 1984). Another source reports acute toxicity values for specific fish ranging from a 96-hour LC50 value of 5 mg/l for rainbow trout to a 96-hour LC50 value of 140 mg/l for bluegill sunfish (Aquatic Plant Identification and Herbicide Use Guide, 1988). The EPA AQUIRE database contains the results of acute toxicity tests ranging from a 24-hour acute LC50 value of 1.0 mg/l in striped bass (an anadromous fish) to a 24-hour acute LC50 value of 5967 mg/l in grass carp (AQUIRE, 1995). In a survey of the results of diquat toxicity tests, the manufacturer has identified a range in toxicity of diquat to fish, with 96 hour LC50 values of 0.5-245 mg/l (Hamer, 1994).

The results of 13 experiments conducted with diquat indicate that diquat did not cause direct mortality to any fish species at 1.0 ppm and below. (MacKenzie, 1971 as cited in Aquatic Plants Management Program for Washington State, 1992). The highest concentration of diquat allowed by the manufacturer's label would equal an initial in-water diquat concentration of 1.5 ppm (Aquatic Plants Management Program for Washington State, 1992).

Studies conducted with fish exposed to solutions of diquat indicate that diquat concentrations in fish do not accumulate above the concentration of diquat in the surrounding water. In addition, when water concentrations decrease, fish diquat residues also decrease. In salmon, trout and goldfish kept in water containing a 1 µg/ml diquat concentration, diquat residues in fish were less than external water concentrations and were mostly found in the nonedible portion of the fish including skin and viscera (Valent U.S.A. Corporation, 1989 as cited in Aquatic Plants Management Program for Washington State, 1992).

Following 24-hour exposure to diquat, changes in rheotaxis and swimming speeds were noted in rainbow trout (Dodson and Mayfield, 1979 as cited in HSDB, 1994).

Trout immersed in water containing 0.5 ppm and 1.0 ppm diquat for 16 days had diquat residue levels of 0.4 and 0.6 ppm, respectively. When fish were returned to non-contaminated water, these levels slowly returned to non-detectable levels. Similar results were obtained with goldfish. (Aquatic Plants Management Program for Washington State, 1992).

Diquat is used to treat disease in fish at hatcheries and for the species tested did not affect the breeding rate in bluegills or cause mortality in young fish. 1 ppm diquat applied up to 3 times and 3 ppm applied once or twice, with 8-week intervals between applications did not affect hatching and growth rates of bluegills in seven different pools. Channel catfish fry were not affected at 10 ppm and bluegill fry were not affected at 4 ppm diquat. Largemouth black bass fry were affected at 22.5°C at levels greater than 1.0 ppm and at 26.0°C at levels greater than 0.5 ppm (Jones, 1965 as cited in Aquatic Plants Management Program for Washington State, 1992).

Decaying vegetation caused by treatment with diquat may deplete oxygen content in the water. For this reason, it is recommended that only 1/3 to 1/2 of an area containing dense vegetation be treated with diquat at a time with a 14-day waiting period in between (Aquatic Plants Management Program for Washington State, 1992).

Plants:

Since diquat is effective in treating a large range of plants, it may have a widespread effect on non-target plants. In addition to direct toxic effects of the herbicide, treatment of a pond with diquat may also cause indirect impacts including dissolved oxygen depletion and habitat loss. These impacts may cause general weakening and/or death of plants on a large scale (Aquatic Plants Management Program for Washington State, 1992).

Microorganisms:

Incubation with diquat caused rapid loss of potassium and phosphate from *Aspergillus niger*, *Penicillium frequentans*, *Mucor hiemalis* and *Zygorrhynchus heterogamus*. At higher concentrations of diquat, the rate of loss is greater, especially with *Zynchorrhynchus* and *Mucor*. Short-term incubation with diquat is followed by sustained loss of potassium when colonies of the above four species are transferred to water (Sahid *et al.*, 1981).

A 50% decrease in O_2 evolution was noted in the following algae organisms: *Chlorococcum sp*, *Dunaliella tertiolecta*, *Isochrysis galbana* and *Phaedactylum tricornutum* in water containing >500 ppm, >500 ppm, 15 ppm and 15 ppm of diquat dibromide, respectively. A 50% decrease in growth was noted in *Chlorococcum sp*, *Dunaliella tertiolecta*, *Isochrysis galbana* and *Phaedactylum tricornutum* in water containing 200 ppm, 30 ppm, 15 ppm and 15 ppm of diquat dibromide, respectively (Verschueren, 1983 as cited in HSDB, 1994).

Table III.2-4. Properties of Diquat

CAS #:	85-00-7
Synonyms	Dipyrido(1,2-a:2',1-c)pyrazinediium, 6,7-dihydro-dibromide; Deiquat; Diquat; Ethylene dipyridylium dibromide; 1,1-Ethylene 2,2-dipyridylium dibromide; 5,6-Dihydro-dipyrido(1,2a:2,1c)pyraziniu m dibromide; 6,7-Dihydropyrido(1,2-a:2',1'-c)pyrazinedium dibromide 9,10-Dihydro-8A,10A-diazoniaphenanthrene dibromide
Molecular formula (salt)	$C_{12}H_{12}N_2Br_2$
Molecular weight (salt)	344.07
Physical properties	yellow solid (pure salt monohydrate); aqueous solution is dark reddish-brown.
Melting point	salts decompose at high temperatures, charring rather than melting; decomposition temperature is >300°C
Density	1.20-1.27 g/ml @20°C/20°C
Vapor Pressure	nonvolatile
Photolysis half-life	2-11 days
Hydrolysis half-life	insignificant
Biodegradation half-life	32 days
K_{ow}	603
K_{oc}	205-691 ml/g
BCF	<1-62
Water solubility	568 mg/l

(HSDB, 1994; Aquatic Plants Identification and Herbicide Use Guide, 1988; Herbicide Handbook, 1983)

Diquat References

Aquatic Plant Identification and Herbicide Use Guide. November, 1988. Volume I: Aquatic Herbicides and Application Equipment. Howard E. Westerdahl and Kurt D. Getsinger, eds. Environmental Laboratory. Department of the Army. Vicksburg, Mississippi.

Aquatic Plants Management Program for Washington State - Final Supplemental Environmental Impact Statement and Responsiveness Summary. January, 1992. Washington State Department of Ecology. vol. I.

AQUIRE (Aquatic Toxicity Information Retrieval Database). 1995. Environmental Research Laboratory. U.S. Environmental Protection Agency.

Arena, J.M. 1979. Poisoning: Toxicology, Symptoms, Treatments. Fourth Edition. Springfield, Illinois: Charles C. Thomas, Publisher.

Booth, N.H., and McDonald, L.E. 1982. Veterinary Pharmacology and Therapeutics. 5th ed. Ames, Iowa: Iowa State University Press.

Calderbank, A. 1968. The bipyridylium herbicides. In: Advances in pest control. 8:127-235.

Calderbank, A. 1972. Experimental considerations in the development of diquat and paraquat as aquatic herbicides. Outlook Agric. 7:51-54.

Clark, A. and Hurst, E. 1970. Brit J Ind Med. 27:51-55.

Corte-Real, Lee. 1995. Personal communication. Massachusetts Department of Food and Agriculture. Pesticide Bureau.

Davidson, Andrew A. 1995. Regulatory Product Manager, Zeneca Professional Products. (personal communication).

Dodson, J.J. and Mayfield, C.I. 1979. Envirn Pollut. 18(2):147-57.

Frank, P.A. and Comes, R.D. 1967. Herbicidal residues in pond water and hydrosoil. Weeds. 16:210-213.

Gilderhus, P.A. 1967. Effects of diquat on bluegills and their food organisms. Prog Fish-Cult. 29:67-74.

Hamer, M.J. May, 1994. Diquat: Fate and effects in aquatic environments. Zeneca, Jealott's Hill Research Station, Berkshire, UK. Report TMJ3204B.

Hartley, D. and Kidd, H. (eds.). 1983. The Agrochemicals Handbook. Old Woking Surrey. United Kingdom: Royal Society of Chemistry/Unwin Brothers Ltd. p. A121/Oct 83.
Herbicide Handbook. 1979. Weed Science Society of America. Champaign, Illinois. 4th edition.

Herbicide Handbook. 1983. Weed Science Society of America. Champaign, Illinois. 5th edition. pp. 184-188.

Hilsenhoff, W. 1966. J Econ Ent. 59:1520.

HSDB (Hazardous Substances Database). October, 1994. Environmental Protection Agency.

Jones, R.O. 1965. Tolerance of the fry of common warm-water fishes to some chemicals employed in fish culture. Proc SE Assoc Game and Fish Comm. 16:436-445.

Klingman, G.C., Aston, F.M. and Nordhoff, L.J. 1975. Weed Science: Principles and Practice, John Wiley and Sons, New York.

MacKenzie, J.W. 1971. Ph.D. Thesis. Oregon State University.

Reinert, K.H. and J.H. Rodgers. 1987. Fate and persistence of aquatic herbicides. Rev. Envtl Contamin Toxicol. 98:61-98.

Sahid, I.B. *et al.* 1981. New Phytol 89(3): 401-9.

Sanborn, J.R. 1977. The fate of selected herbicides in the aquatic environment. USEPA-660/3-74-025. pp. 76-89.

Simsiman, G.V., Daniel, T.C. and Chesters, G. 1976. Diquat and endothall: their fates in the environment. Residue Reviews. 62:131-174.

Simsiman, G.V. and Chesters, G. 1976. Persistence of diquat in the aquatic environment. Water Res. 10:105-112.

Smith, A.E. and Grove, J. 1969. Photochemical degradation of diquat in dilute aqueous solution and on silica gel. J. Agric Food Chem. 17:609-613.

State of Washington. 1984. Paraquat and Diquat. Environmental Health Criteria #39. Countway, Washington.

Tatum and Blackburn, 1962 in Pimentel, D. 1971 Ecological effects of pesticides on non-target species. EPA Report No. EPA-540/9-71-006. EPA, Washington, D.C. 225 pp.

U.S. Department of the Interior, Fish and Wildlife Service. Bureau of Sports Fisheries and Wildlife. 1975. Lethal Dietary Toxicities of Environmental Pollutants to Birds. Special Scientific Report - Wildlife No. 191. Washington, DC. U.S. Government Printing Office.

U.S. Department of the Interior, Fish and Wildlife Service. 1984. Handbook of Toxicity of Pesticides to Wildlife. Resource Publication 153. Washington, DC: U.S. Government Printing Office.

U.S. Department of Transportation. 1984. Emergency Response Guidebook, Guide No. 55 (1984) DOT No. p.5800.3.

USEPA (U.S. Environmental Protection Agency). 1982. Recognition and Management of Pesticide Poisoning. EPA 540/9-80-005. p. 33.

USEPA (U.S. Environmental Protection Agency). 1992a. Final Drinking Water Criteria Document for Diquat. Health and Ecological Criteria Division. Office of Science and Technology. Office of Water. Washington, D.C.

USEPA. (U.S. Environmental Protection Agency). 1992b. Integrated Risk Information System (IRIS).

USEPA. (U.S. Environmental Protection Agency). February 7, 1994. memo re: Diquat Dibromide - RED Candidate from Henry M. Jacoby, Chief, Environmental Fate and Groundwater Branch. Environmental Fate and Effects Division.

USEPA. (U.S. Environmental Protection Agency). 9/10/95. Office of Pesticide Programs Dose Tracking Report.

USEPA. (U.S. Environmental Protection Agency). May, 1995b. Drinking Water Regulations and Health Advisories. Office of Water. U.S. Environmental Protection Agency. Washington, D.C.

Valent U.S.A. Corporation. 1989. Correspondence to Washington Department of Ecology.

Verschueren, K. 1983. Handbook of Environmental Data of Organic Chemicals. 2nd ed. New York, NY: Van Nostrand Reinhold Co.

Wilson, D.C. and Bond, C.E. 1969. The effects of the herbicides diquat and dichlobenil (Casoron) on pond invertebrates. Part I. Acute Toxicity. Trans Am Fish Soc 98(3):438-443.

Zeneca Professional Products. 1994. Reward Aquatic and Noncrop Herbicide. (EPA registration label).