

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

Paraquat

Publication Date: 9/93

TRADE OR OTHER NAMES

Product names include Crisquat, Dextrone, Dexuron, Gramoxone Extra, Herba-xone, Ortho Weed and Spot Killer, and Sweep.

INTRODUCTION

Paraquat is a quaternary nitrogen herbicide widely used for broadleaf weed control. It is a quick acting, non-selective compound, that destroys green plant tissue on contact and by translocation within the plant. It has been employed for killing marijuana in the U.S. and in Mexico. It is also used as a crop desiccant and defoliant, and as an aquatic herbicide ([11](#)).

Paraquat is a Restricted Use Pesticide (RUP) and is banned for use in several Scandinavian countries. Restricted Use Pesticides may be purchased and used only by certified applicators.

TOXICOLOGICAL EFFECTS

ACUTE TOXICITY

Paraquat is highly toxic to animals by all routes of exposure, and is labeled with a DANGER-POISON signal word. A single large dose, administered orally or by injection to animals, can cause excitability and lung congestion, which in some cases leads to convulsions, incoordination, and death by respiratory failure.

Paraquat is exceedingly toxic to humans. Many cases of illness and/or death have been reported ([12](#)). The lethal ingestion dose of paraquat in humans is 35 mg/kg ([6](#)). A maximum of 3.5 mg/hour could be absorbed through the dermal or respiratory route without damage ([10](#)).

If swallowed, burning of the mouth and throat often occurs, followed by gastrointestinal tract irritation, resulting in abdominal pain, loss of appetite, nausea, vomiting, and diarrhea. Other symptoms of toxicity include thirst, shortness of breath, and rapid heart rate. Other toxic effects include kidney failure, lung sores, and liver injury. Some symptoms may not occur until days after exposure.

Persons with lung problems may be at increased risk from exposure. Evidence also suggests that long-term, heavy smoking of marijuana contaminated with paraquat may cause lung scarring ([10](#)).

Although nose bleeds and lung irritation may occur if paraquat is inhaled, the risk of serious damage due to occupational exposure to paraquat is low because of its low vapor pressure.

Direct contact with paraquat solutions or aerosol mists may cause skin burns, dermatitis, and damage to fingernails. Paraquat splashed in the eye can irritate, burn, and cause corneal damage and scarring of the eyes.

The oral LD50 of paraquat, in its various forms, for rats ranges from about 20 mg/kg ([10](#)) to 150 mg/kg ([6](#)). The oral LD50 of the dichloride form of paraquat is 196 mg/kg in mice, 50 mg/kg in monkeys, and 48 mg/kg in cats ([6](#)). The oral LD50 of paraquat in the cow is 50-75 mg/kg ([6](#)). The dermal LD50 in rabbits is 236-325 mg/kg.

CHRONIC TOXICITY

No long-term studies on paraquat's effects on humans were found. Several chronic effects for humans have been inferred from animal studies including Parkinson's disease and irreversible lung damage ([13](#)).

In animal studies, rats showed no effects after being exposed for two years to paraquat ([6](#)). Dogs, however, developed lung problems after being exposed to high doses for two years ([6](#), [9](#)).

Reproductive Effects

Overall, there is little evidence that paraquat causes adverse effects on reproduction.

In a long-term rat study, no adverse reproductive effects were reported ([8](#)). Paraquat, administered both orally and by injection to pregnant mice, did not transfer to the mouse embryos in appreciable quantities. Hens given high levels of paraquat in their drinking water for 14 days produced an increased percentage of abnormal eggs ([6](#)).

In one suicide, a seven-month pregnant woman swallowed about 2 ounces of paraquat. The fetal heartbeat disappeared on the 13th day. The mother died on the 17th day after poisoning. No symptoms of paraquat poisoning were noted in the body of the fetus ([6](#)).

Teratogenic Effects

The weight of evidence suggests that paraquat does not cause birth defects at doses which might reasonably be encountered. Offspring of mice dosed with high doses of paraquat during the sensitive period of pregnancy had less complete bone development than the mice given lower doses. Offspring of rats given similar treatment showed no developmental defects at any dose, but fetal and maternal body weights were lower than normal ([8](#)). Other studies of paraquat using rabbits and mice have shown no teratogenic effects ([6](#), [9](#)).

Mutagenic Effects

Paraquat has been shown to be mutagenic in human, microorganism, and mouse cell assays ([8](#)). Paraquat dichloride did not cause mutations in the sperm of fertile male mice, but pregnancy rates were reduced ([6](#)).

Carcinogenic Effects

Mice fed paraquat dichloride for 99 weeks at high levels did not show cancerous growths. Rats fed high doses for 113 (male) or 124 weeks (female) developed lung, thyroid, skin, and adrenal tumors (8). Paraquat's carcinogenic potential has not yet been thoroughly evaluated; however, the EPA has classified the pesticide as a possible human carcinogen (14).

Organ Toxicity

Paraquat is extremely toxic to mammalian lungs, where it can cause sores, bleeding, and disease. The lung tissue accumulates paraquat at much higher rates than do the other organs and tissues in the body (11). However, it can also damage the heart, liver, kidneys, cornea of the eyes, adrenal glands, skin, fingernails, and digestive system. Inhalation of paraquat can cause nasal mucous irritation and bleeding of the gastrointestinal tract (9, 10).

In a study of 30 workers spraying paraquat over a 12-week period, approximately one-half had minor irritation of the eyes and nose (6). Of 296 spray operators with gross and prolonged skin exposure, 55 had damaged fingernails as indicated by discoloration, nail deformities, or loss of nails (6).

Fate in Humans and Animals

Paraquat is not readily absorbed from the stomach, and is even more slowly absorbed across the skin. This chemical rapidly disappears from the blood.

In the stomach, the metabolites formed from paraquat breakdown are more readily absorbed than is paraquat (9) but their toxicity is unknown.

As paraquat concentrates in lung tissue, it is changed to highly reactive and potentially toxic forms.

Oral doses of paraquat in rats are excreted mainly in the feces, while paraquat injected into the abdomen leaves through urine (9).

In one study, farm animals excreted over 90% of the administered paraquat within a few days. It was slightly absorbed and metabolized in the gastrointestinal tract. Milk and eggs contained small amounts of two paraquat metabolites (9).

ECOLOGICAL EFFECTS

Paraquat is moderately to highly toxic to many species of aquatic life including rainbow trout, bluegill, and channel catfish (6).

At high levels, paraquat inhibits the photosynthesis of some algae in stream waters (5).

Paraquat is rapidly excreted by animals, so tissue accumulation is unlikely. In rainbow trout exposed for seven days to paraquat, the chemical was detected in the gut and liver, but not in the meat of the fish.

Aquatic weeds may be a source of bioaccumulation. In one study, four days after paraquat was applied as an aquatic herbicide, weeds sampled showed significant residue levels.

Paraquat is non-toxic to honey bees (14).

ENVIRONMENTAL FATE

Ultraviolet light, sunlight, and soil microorganisms can degrade paraquat to products which are less toxic than the parent compound.

High concentrations of paraquat in marijuana fields have been reported. Paraquat dichloride droplets decompose when exposed to light after being applied to maize, tomato, and broad-bean plants. Small amounts of residues were found in potatoes treated with paraquat as a desiccant, and boiling the potatoes did not reduce the residue (6).

Paraquat is quickly and strongly adsorbed by soil particles, especially in clay soil (3). Such bound residues are not available to plants, earthworms, and microorganisms. Because of its unavailability, and its resistance to microbial degradation and breakdown by sunlight, paraquat is long-lived. The bound residues persist indefinitely and are transported in runoff with the sediment. The reported half-life for paraquat in soil ranges from 16 months (aerobic laboratory conditions) to 13 years (field study) (7).

Paraquat is not mobile in silt loam and silty clay loams, and is slightly mobile in sandy loams (8). Paraquat not associated with soil particles can be decomposed to a non-toxic end product by soil bacteria (10).

Paraquat residues disappear rapidly from water by binding onto aquatic weeds and by strong adherence to the bottom mud. It has a half-life in laboratory stream water of 13.1 hours (5). In another study, paraquat dichloride was stable for up to 30 days. In a third study using low levels in water, paraquat had a half-life of 23 weeks (8).

Because paraquat quickly adsorbs to soil particles, it resists decomposition by sunlight. Paraquat sprayed on the surface of a sandy soil did not degrade when the soil was irradiated with natural sunlight for two years (8).

Of 721 groundwater samples, only one sample contained paraquat, at a concentration of 20 ppm (8).

COMMENTS AND PROBLEMS

Paraquat is similar to 1-methyl-4-phenyl-1,2,3-tetrahydropyridine, a compound which induces a Parkinsonian-like state in humans. In a Canadian study, the correlation between Parkinson's disease incidence and level of pesticide use was very strong.

PHYSICAL PROPERTIES AND GUIDELINES

Paraquat salts are colorless, white, or pale yellow crystalline solids, which are hygroscopic and odorless. The dichloride salt is stable except under alkaline conditions. The formulated technical grade product is a dark red solution with an ammonia-like odor. It is corrosive to metals, will react with strong oxidizers, and tends to be light-sensitive. Paraquat is stable to heat when in acid or neutral solutions, but is hydrolyzed by alkali solutions. Paraquat decomposes at high temperatures to form toxic gases and vapors such as hydrogen chloride, nitrogen oxides, sulfur oxides, and carbon monoxide. Its molecular weight is 257.18 (8).

The chemical name for paraquat is 1'-Dimethyl-4,4'-bipyridinium. Paraquat is often formulated as a dichloride salt or a dimethyl sulfate salt. This chemical is also known as N,N'-dimethyl-4,4'-

bipyridinium; N,N'-dimethyl-4, 4'-bipyridinium dication, and o-paraquat dichloride. Under normal storage conditions, paraquat's shelf-life is indefinite ([10](#)).

Exposure Guidelines:

ADI: .002 mg/kg (paraquat dichloride); or 0.0045 mg/kg/day ([8](#)) (paraquat ion)
TLV: air.
TWA: 0.1 mg/m³ [dichloride and bis(methosulfate)salts]
Drinking water health advisory: Drinking Water Equivalent Level (DWEL): 160 ug/L ([8](#))

Physical Properties:

CAS #: 1910-42-5
Solubility in water: very soluble (20 degrees C) ([2](#))
Solubility in solvents: Paraquat has little to no solubility in organic solvents. The dichloride salt is sparingly soluble in lower alcohols and is insoluble in hydrocarbons.
Boiling point: 175-180 degrees C (decomposes) ([2](#))
Vapor pressure: <10⁻⁷ torr([10](#)) Not volatile ([2](#))
Log P: Less than 7.0
Kow: 2.44 (calculated) ([8](#))
Koc: 15,473-51,856 ([1](#), [4](#))
BCF: 0.3 (calculated) ([6](#))

BASIC MANUFACTURER

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Review by Basic Manufacturer:

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