UNITED NATIONS

RC

UNEP/FAO/RC/CRC.6/9/Add.2

Rotterdam Convention on the Prior Informed Consent Procedure for Certain Hazardous Chemicals and Pesticides in International Trade

Distr.: General
2 December 2009

English only

Chemical Review Committee
Sixth meeting
Geneva, 15–19 March 2010
Item 5 (b) (v) of the provisional agenda*

Listing of chemicals in Annex III to the Rotterdam Convention: review of notifications of final regulatory actions to ban or severely restrict a chemical: paraquat

Paraquat

Note by the Secretariat

Addendum

Supporting documentation provided by Sweden

The Secretariat has the honour to provide, in the annex to the present note, the English translation and additional documentation received from Sweden to support its notification of final regulatory action for paraquat as a pesticide. The documentation has been reproduced as received, without formal editing by the Secretariat.

* UNEP/FAO/RC/CRC.6/1.

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Annex

1. Comments from Sweden on the translated background material for the PIC notification on paraquat

2. Paraquat: A discussion of toxicity and human risk (English translation)
   The original document in Swedish and the summary in English were made available to the fifth session of the Committee and are reproduced in document UNEP/FAO/RC/CRC.6/9.Add.1.

3. Literature review concerning the presence of the herbicide paraquat in soil and water (English translation)
   The original document in Swedish and the summary in English were made available to the fifth session of the Committee and are reproduced in document UNEP/FAO/RC/CRC.6/9.Add.1.

4. Application for registration of the product Gramoxone 80 (original in Swedish and English translation)

Swedish comments on the translated background material for the PIC notification on paraquat

Paraquat was banned in Sweden in 1983 and the legal text refers to: "*its acute high toxicity, risk for irreversible effects on health and for accidents during handling and use of the substance*". The correspondence with the applying company further shows that environmental concerns also played a role in the decision. The translated background material supports these conclusions accordingly:

From the dossier *"PARAQUAT, a discussion of toxicity and human risk":*

The acute high toxicity – The acute high toxicity of paraquat is well documented. Mortality is high even at "such low doses as 1-5 grams orally". No effective treatment is known.

Risk for irreversible effects on health – The background document notes an Acceptable Daily Intake for humans at 0-0,002 mg/kg body weight. For a person of 70 kg BW this would mean 140 µg per day. The document also notes that dermal exposure may occur in the range of 300-3400 µg/h. A comparison between the ADI and exposure supports the conclusions in the regulatory action. Exposure through inhalation also occur (2µg/h or less). It is noted that not much information existed on long-term effects from low-dose exposure but it was deemed likely that lung damage would occur and further that "concentrated preparations have a high corrosive effect and can cause contact dermatitis and severe and lasting eye damage".

Risk for accidents during handling and use of the substance – The background document noted that around 300 cases of fatal accidental poisoning were known (60% of 500 cases). Cases of accidental poisoning were known from Sweden and the neighbouring country Denmark. "Single or repeated severe contamination of the skin" could also lead to fatal poisoning.

The dossier *"Litterature revue concerning the presence of paraquat in soil and water"* shows that the decision makers were aware of the high persistence of paraquat and that continuous use of the herbicide led to accumulation of the compound in the soil, especially in soils with high clay content. In the decision communication letter to the firm, it is stated that *"The pesticide Gramoxone 80 must therefore be regarded as having such inconvenient properties regarding the protection of health and the environment that it is not appropriate to use the product for the treatment of pests".*

13.05.82

PARAQUAT

A discussion of toxicity and human risk

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BACKGROUND

The following review and evaluation does not represent a complete review of the literature but is essentially based on review articles (Smith and Heath 1976; Pasi 1978; Haley 1979; Manzo et al 1979; Evers 1980; Pedersen et al 1981). In addition, supplementary material from the Product Control Office (Stecko 1978) and original papers etc. have been utilised.

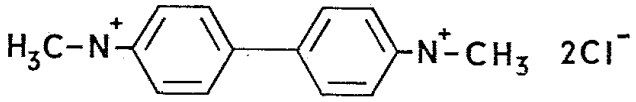
Paraquat is a quaternary ammonium compound which was synthesised as long ago as 1882 (Weidel and Russo 1882), but its herbicidal properties were not discovered until 1955. Preparations of paraquat were introduced as commercial herbicides in 1962 and in Sweden in 1964. On the other hand, paraquat has been generally used as a redox indicator (methyl viologen) in chemical laboratories since 1933 (Michaelis and Hill 1933 a,b).

Paraquat is reported to be the most common herbicide in the world (Turner 1982). A preparation containing 20% paraquat (Gramoxone) was registered in hazard class 2 in Sweden until 1978 but was reclassified in class 1 in 1978. The preparation is also a class 1 agent in other Nordic countries. Preparations of 2.5% paraquat are placed in hazard classes 2 or 3 in all the Nordic countries.

The world-wide use of paraquat has resulted in at least 500 registered fatal cases of poisoning since 1964 (Fairshter et al 1979; Russel et al 1981).

In Sweden paraquat has caused two serious incidents since 1975 (Poisons Information Centre, unpublished). These cases related to a 5-year-old boy who accidentally had paraquat splashed on his face, resulting in severe and permanent eye damage, and to a 30-year-old man who ingested paraquat for suicidal purposes and who despite treatment died 10 days following the exposure. A case of fatal accidental oral paraquat poisoning was reported from Denmark in 1981 (Pedersen et al 1981).

STRUCTURE, PHYSICAL AND CHEMICAL PROPERTIES

Structure: ..... 

Chemical name ..... 1,1[1]-dimethyl-4,4[1]-dipyridinium dichloride

Empirical formula ..... $C_{12}H_{14}N_2C_{12}$

Molecular weight	Paraquat ion	186.2
	Paraquat dichloride	257.2

Physical state ..... White (pure), yellow (technical grade) solid substance

Density 1.24-1.26 20/20

Melting point ..... 175-180°C

Disintegration temperature ..... 300°C

Vapour pressure ..... Non-volatile

Solubility	Water -	soluble
	Alcohol -	very low solubility
	Paraffin -	insoluble
	Acetone -	very low solubility

Corrosiveness ..... Highly corrosive to metals

Stability ..... Stable in neutral and acidic solution, non-stable in alkaline solution

EXPOSURE

Occupational exposure can take place in connection with manufacturing and formulation. No cases of poisoning have been reported in this connection (Howard 1979).

Exposure also takes place in connection with weed control. Total concentrations in the air of approx. 1 µg/m3 or less (Chester et al 1982) and up to 12 µg/m^3 (Hogarty 1975, cited in Howard 1980) have been reported. Measurement of inhalation exposure in another study showed total concentrations in the inhalatory air equivalent to 0.002 mg/h or less (Staiff et al 1975). Sprayers are reported only to a small extent to produce particles with an aerodynamic diameter which is so small that they reach the alveoli to any significant degree (Hogarty 1975, cited by Howard 1979).

Dermal exposure obviously also takes place in connection with spraying. Staiff et al. (1975) calculated that deposition on skin was 3.4 mg/h or less (mean value 0.3-0.4 mg/h). Most skin exposure occurred on the hands.

In weed control on rubber plantations (Swan 1969; Chester et al. 1982) the sprayer drivers (without protective equipment) excreted up to 0.3 mg/l in the urine, in the majority of cases less than 0.01 mg/l. Protective equipment (breathing mask, rubber boots and rubber gloves) led to a reduction in exposure. For comparison it may be mentioned that individuals who have been poisoned orally have had two orders of magnitude higher concentrations (or more) following exposure (e.g. Kerr et al. 1968).

Residual quantities may be found in certain agricultural products. Marijuana may be contaminated. In such cases the smoker may inhale paraquat, which has been reported to cause tracheal problems and expectoration of blood (Smith 1978). It should be noted that cannabis in itself is an irritant. The causal connection with paraquat is highly doubtful.

Available data do not suggest that residual quantities of significance occur in foods (FAO/WHO 1976). Paraquat residues have not been analysed in Swedish foods.

UPTAKE, DISTRIBUTION, DISTRIBUTION, BIOTRANSFORMATION AND ELIMINATION

Uptake

Bipyridyl compounds are generally absorbed poorly through biological membranes. Only 5-10% of an orally given dose of paraquat is taken up from the gastrointestinal tract. There is good agreement between humans and experimental animals (Conning et al 1969; Murray and Gibson 1974; Okonek and Hofmann 1975).

Dermal uptake via intact skin is low (McElligott 1972), and systemic toxic effects therefore ought not to be expected following dermal exposure. Despite this, cases of fatal poisoning in humans following single or repeated dermal exposure to paraquat have been reported (Howard 1978).

In animal experimental exposure via the lungs paraquat was only retained to a small extent in the lungs (Gage 1968). According to another study significant uptake via the lung can take place (Seidenfeld et al 1978).

Distribution

Following uptake from the gastrointestinal tract in rats a high concentration is initially obtained in the liver and kidneys, and the concentration then decreases gradually (Smith et al 1979). The plasma level remains relatively constant for nearly 30 hours, during which time the concentration in lung tissue rises. The accumulation in lung tissue takes place via an energy-dependent process, and paraquat which is accumulated is in free and non-bound form. The process is selective for paraquat, and no accumulation of the closely related bipyridyl compound diquat occurs in the lung (Rose et al 1980).

In rabbits it has been previously indicated that no increase in the concentration of paraquat occurs in the lung (Ilett et al 1974). However, in later aerosol exposure studies on rabbits it has been shown that lung changes of the same kind as following oral administration occur, and that repeated inhalation exposure to paraquat leads to a significant accumulation of paraquat in lung tissues (Seidenfeld et al 1978).

TOXICITY

Animals

Single-dose toxicity varies substantially for different species, strains and routes of administration (Table 1).

A large oral or parenteral dose causes central nervous system symptoms (excitation, incoordination, ataxia and convulsions), as well as respiratory symptoms. If the animals survive they stop eating and lose weight. They die within 10 days due to lung damage (Manzo et al 1979).

The lung damage has a destructive phase with damage first to the thin cells in the alveolar walls (type I cells), possibly with pulmonary oedema, and later the cells (type II cells) that produce a surface tension-reducing substance (surfactant). After 3 days the damage is extensive. Mesenchymal cells in the alveoli differentiate to fibroblasts, which cause pulmonary fibrosis, which may be very marked.

Subacute oral administration of 300 mg/kg diet to rats over 15-20 days causes lung damage (approx. 15 mg/kg/day; Conning et al. 1969). In other studies rats were given 170 mg/kg (approx. 8 mg/kg/day; Gage 1969, cited in Haley 1979) and 250 mg/kg (12.5 mg/kg per day; cited in Stecko 1978) for two years without showing signs of lung damage.

Dogs which received 36 mg/kg in their diet for two years (approx. 0.9 mg/kg/day; Gage 1969, cited in Haley 1979) or 50 mg/kg (approx. 1.25 mg/kg per day; cited in Stecko 1978) did not show signs of illness, while animals which received 125 mg/kg feed (approx. 3 mg/kg/day) displayed symptoms of lung damage (cited in Stecko 1978). Sheep were poisoned by 5-20 mg/kg body weight per day over a few weeks (cited in Stecko 1978).

Rabbits which received approximately 7 mg/kg per day by skin application showed signs of lung damage after 20 days, while 3 mg/kg per day did not cause illness (cited in Stecko 1978).

Inhalation of aerosol can also cause lung damage. A concentration of 0.7-1 mg/m^3 for 6 h caused approximately 50% mortality in rats (Gage 1968). Exposure to 0.4 mg/m^3 for 6 h/day over a period of 3 weeks caused inflammatory lung changes, which was not the case at 0.1 mg/m^3 (Gage 1968). Maximum effect was obtained at a particle size of 3 µm. Smaller and larger particles had lower toxicity, which shows that particle size may be very significant.

Inhalatory exposure of rats to paraquat aerosol with > 80% of the particles in the size range 2.5-5 µm at concentrations above 1-2 mg/m^3 for several hours results in blood stasis, oedema, bronchial irritation 2-3 days after exposure. If the animals survive there are no delayed effects (Conning et al 1969). Topical instillation of paraquat in the lung or muscle produces local fibrosis in rats (Kimbrough and Gaines 1970).

Toxic effects in the lung have also been induced by inhalatory exposure of mice, guinea-pigs, rabbits and dogs (Gage 1968).

There has been discussion of whether the effects on the lung seen in connection with inhalatory exposure are of the same nature as those seen after oral or parenteral administration. Gage (1968) interprets the inhalatory effects as purely due to irritation, separate from the more specific damage caused by paraquat accumulated in the lung from blood. The reasoning is that the damage does not contain proliferative components. On the other hand, studies on rabbits suggest that the effects are of the same type (Seidenfeld et al 1978).

100 mg paraquat/kg feed (approx. 5 mg/kg/day) given to rats in a multi-generation test did not affect offspring (Hathway 1975, cited in Haley 1979).

A single intraperitoneal (sic) dose of 6.5 mg/kg given on the 6th day of gestation to female rats caused abnormal embryos, while the same dose on the 7th-14th days did not cause damage (Khera et al. 1970, cited in Haley 1979). The suitability of administering such a corrosive and locally irritant substance as paraquat intraperitoneally is a matter for discussion. Foetotoxicity could not be detected in mice with intraperitoneal administration (Selypes et al 1980).

Testing has not provided evidence of mutagenicity for paraquat (Haley 1979; Selypes et al 1980). In rats 250 mg/kg in the feed (approx. 12.5 mg/kg/day) for 2 years did not cause tumours (cited in Stecko 1978). Intraperitoneal administration of 10 and 20 mg/kg every other week to rats did not cause lung tumours over a period of 112 days (Smith 1971, cited in Haley 1979).

Humans

Paraquat causes strong local irritation. Both concentrated and dilute solutions can cause damage to the eyes (splashes cause conjunctivitis and keratitis, sometimes uveitis), skin (yellow discoloration and contact dermatitis) and nails (paronychiae, destruction; Hearn and Keir 1971; Howard 1979).

Almost all cases of poisoning have occurred after oral ingestion. Suicide by injection has been reported (Almog and Tai 1967). A total of around 500 cases have been published. Around 60% of these were suicide. The clinical course depends on the dose (Hofmann and Frohberg 1972; Russel et al 1981).

A massive dose causes severe irritation in the mouth, throat, oesophagus and stomach, with vomiting (which may be bloody), abdominal pain and diarrhoea (which may be bloody). Kidney, liver and lung damage occur rapidly, the patient becomes unconscious within hours and dies from circulatory collapse within a few days. Damage to heart muscle, the adrenal cortex, the central nervous system and blood (methaemoglobinaemia) sometimes occurs.

A smaller dose only produces mild direct symptoms with insignificant irritation in the mouth and throat and nausea for about 24 hours. After a few days kidney (principally tubular necrosis) and liver damage (principally centrilobular necrosis) is manifested. These effects may be reversible, which may lead to underestimation of the risk, particularly as the paraquat concentration in the urine is low after about one day (particularly in the case of renal damage, the blood concentration is more informative but difficult to master analytically). After a few days or weeks, however, progressive lung damage generally (but not always) occurs with fibrosis. This lung damage often leads to death from respiratory failure. No studies of lung function in poisoned individuals who survive appear to have been reported. It is reasonable to assume, however, that severe poisoning leads to permanent pulmonary fibrosis. If the patient survives, impairment of lung function appears to remain.

Single oral doses of 5-10 g or more are always fatal. The majority appear to survive one g or less (Hofmann and Frohberg 1972). Accidents have occurred, for example, in connection with the cleaning of spray nozzles using the mouth (Fitzgerald et al. 1978). The lowest oral dose that causes systemic poisoning in humans is not known. About a teaspoon of concentrate is reported to have caused poisoning (Masterson and Roche 1971). The chance of survival only appears to a limited extent to be dependent on any treatment given (see below).

Several cases of fatal poisoning have been reported following single or repeated severe contamination of the skin with paraquat (Howard 1978; Fitzgerald et al 1978; Jaros 1978; Newhouse et al 1978; Levin et al 1979; Waight 1979). Dermatitis (not least that caused by concentrated paraquat solution) is said to increase the risk.

In connection with inhalation of spray aerosol, irritation phenomena from the eyes and upper airways (inflammation on lips and in mouth/throat, nose bleeds) and from the gastrointestinal tract have been reported (Garnier et al 1979).

Only a few cases of systemic poisoning that have been related to inhalation of spray aerosol have been reported. One concerned a person who sprayed with a particularly strong solution over a period of 4 h in a greenhouse (Fitzgerald et al 1978). He had initial symptoms in the upper airways. After 5 days he had signs of transient mild renal damage, but no signs of lung damage.

One man suffered vomiting after spraying for 4 h under unfavourable conditions (wind) (Malone et al 1971). After approximately six days he developed transient renal damage but did not show signs of lung damage.

Absence of lung damage in these cases means that some doubt must prevail on the relationship between exposure and disease, although cases of renal damage without lung affection have been reported (Hofmann and Frohberg 1972).

In another case of suspected paraquat intoxication due to inhalation the exposure had occurred through the inhaling of aerosol from a circular saw over which water was poured for stone sawing. The water had been kept in a drum that had previously contained paraquat (Adam 1980). On seven occasions 36-48 hours after exposure the operator had a general sensation of illness, cough and fever. The chest X-ray was normal, and the causal connection between paraquat and illness must be judged to be highly doubtful.

A woman was exposed for 10 min. in her garden to paraquat spray mist from a nearby field (George and Hedworth-Whitty 1980). She felt tightness in the chest. Over the course of a week she felt increasingly short of breath on physical exertion. She sought medical attention and was treated with steroids, after which she improved somewhat. After 2 months there was a normal chest X-ray but distinctly pathological lung function (of the restrictive type). Seven months after exposure her lung function parameters had improved substantially. Comments: The exposure should have been very slight. The combination of severe impairment of lung function and a normal chest X-ray is perplexing. Great doubt must therefore prevail on the causal connection between inhalatory exposure to paraquat and lung damage (Hart 1980).

In some cases of time connection between facial paralysis (Mourin 1967; Swan 1967), liver damage (Carrad 1969, cited in Pasi 1978) and muscle cramps (Guardscione et al 1969, cited in Pasi 1978) and occupational handling of paraquat attempts have been made to relate the symptoms to the exposure. The causal connections may be regarded as highly dubious.

In a report (Peoples et al 1977, cited in Howard 1979) symptoms such as dizziness, paraesthesia, chest pain and general weakness are said to have occurred following exposure. The phenomenon cannot be judged more closely.

Surprisingly, very little has been done with regard to studies of the incidence of lung disease among those chronically exposed to paraquat. In the reported studies on a small number of workers in a formulation factory (Howard 1979) and weed control work (Hearn and Keir 1971; Howard 1979) there is no mention of lung disease or lung function tests.

Toxicological mechanism

The delayed lung damage is the most distinctive effect of paraquat. The selective concentration of paraquat in lung tissue has been shown to be energy-dependent (Smith et al 1979; Hose et al 1980).

The biochemical basis for lung fibrosis has not been entirely clarified but is assumed to be related to conversion of paraquat to free radical. Bus et al (1976) have put forward a hypothesis that the toxicity of paraquat is caused by superoxide formation with accompanying formation of singlet oxygen and increased lipid peroxidation (Figure 1). This would among other things mean increased production of malondialdehyde. These mechanisms have been questioned by several studies but supported by others (Manzo et al 1979; Yasaka et al 1981). A Japanese study has recently pointed to raised levels of malondialdehyde in the plasma of an intoxicated patient. Equivalent observations were also made experimentally in guinea-pigs (Yasaka et al 1981).

Treatment

Several different kinds of treatments have been tried in systemic poisoning with paraquat. Optimal therapy is still the object of discussion. Treatment success is heavily dependent on therapy being initiated early (within two days; Hofmann and Frohberg 1972; Russel et al 1981).

Within hours after oral ingestion, gastric lavage is performed with instillation of Fuller's earth or bentonite (clays; activated charcoal ineffective) and laxatives. Vomiting is generally not advised owing to the corrosiveness of paraquat. A number of methods have been tried with the aim of increasing elimination: forced diuresis (if toxic pulmonary oedema is not present), haemodialysis (less than 2% is eliminated), haemoperfusion through an activated charcoal filter (probably better than haemodialysis) and plasmaphoresis. Oxygen therapy is reported to increase lung damage, and reduced oxygen tension has instead been tried during the stage before the lung damage has become so severe that the patient has been affected by severe hypoxaemia. Treatment with superoxide dismutase, which has been effective in animal experiments, has also been proposed. Steroids and immunosuppressant drugs, which in themselves pose risks, have also been tried. Even lung transplantation has been attempted without success.

Treatment success (possibly with exceptions for the initial attempts to inhibit resorption) at best is moderate. The mortality rate is around 50% (Hofmann and Frohberg 1972; Howard 1979).

LIMIT VALUES

Paraquat has been evaluated by FAO/WHO, most recently in 1976. On the basis of available documentation the zero-effect dose was estimated at 1.5 mg/kg body weight per day for rats and 1.25 mg/kg body weight per day for dogs. The acceptable daily intake for humans was set at 0-0.002 mg/kg body weight for paraquat dichloride (equivalent to 0-0.0014 mg/kg for paraquat ion).

The maximum permitted level in foods in Sweden is set at 0.05 mg/kg in fruit and berries and 0.1 mg/kg in potatoes.

In the United States, ACGIH (1981) has set the limit value for total occupational exposure at 0.5 mg/m^3 and 0.1 mg/m^3 for the respirable fraction.

Table 1

The LD50 for different species in oral (po), intraperitoreal (ip), subcutaneous (sc) and dermal (d) administration. Data compiled form Stecko (1978), Haley (1979) and Manzo et al (1979).

	Route of administration			
Species	po mg/kg	ip mg/kg	sc mg/kg	d mg/kg
Rat	40-200	14-34	19-26	80-90
Mouse	120	30	-	-
Guinea-pig	22-80	3	-	-
Rabbit	49-150	18	-	346
Sheep	50-75	-	-	-
Cat	26-50	-	-	-
Dog	25-50	-	-	-
Monkey	50	-	-	-
Man	40-60	-	4	-

SUMMARY

Paraquat is a highly toxic substance. Damage can occur principally in the kidney, liver and lung. The most interesting effect is the delayed lung damage which can follow a symptom-free interval of days to weeks.

Administration of sufficiently high doses can thus lead to severe lung fibrosis. Mortality is very high, even in the administration of such low doses as 1-5 grams orally. More than 500 cases of fatal poisoning have been reported. It is unclear whether survivors have permanent impairment of lung function. It may, however, be judged likely that this will be the case after severe poisoning.

Almost all cases of poisoning have occurred after oral ingestion of a single dose, accidentally or for suicidal purposes. The treatment options are limited. Non-seriously intended suicidal attempts can therefore to a large extent be expected to lead to death. Several cases of severe systemic poisoning have been reported following extensive skin contamination, including occupational contamination.

It has been shown in animal experiments that lung damage with fibrosis can occur following inhalation of high concentrations. Only a few cases have nevertheless been reported of human intoxication following particularly great exposure by inhalation. The causal link may also be regarded as doubtful in these cases. The explanations for the low risk may be firstly that the concentration in the air in spraying is generally low and secondly that the spray aerosols only contain a small amount of respirable particles.

However, in view of the extremely widespread use of paraquat it is remarkable that only a small number of studies of any chronic effects in spraying workers, methodologically unsatisfactory for the purpose, have been published. There appears to be a particularly pressing need for lung function studies. Data suggesting an accumulation of paraquat in lung tissue make such studies justified. On the other hand, it must be emphasised that the paraquat concentrations that lead to damage in the short-term exposure of animals as a rule are two to three orders of magnitude greater than those that occur in connection with occupational exposure.

Concentrated preparations have a high corrosive effect and can cause contact dermatitis and severe and lasting eye damage.

No teratogenic, mutagenic or carcinogenic effects of paraquat have been reported.

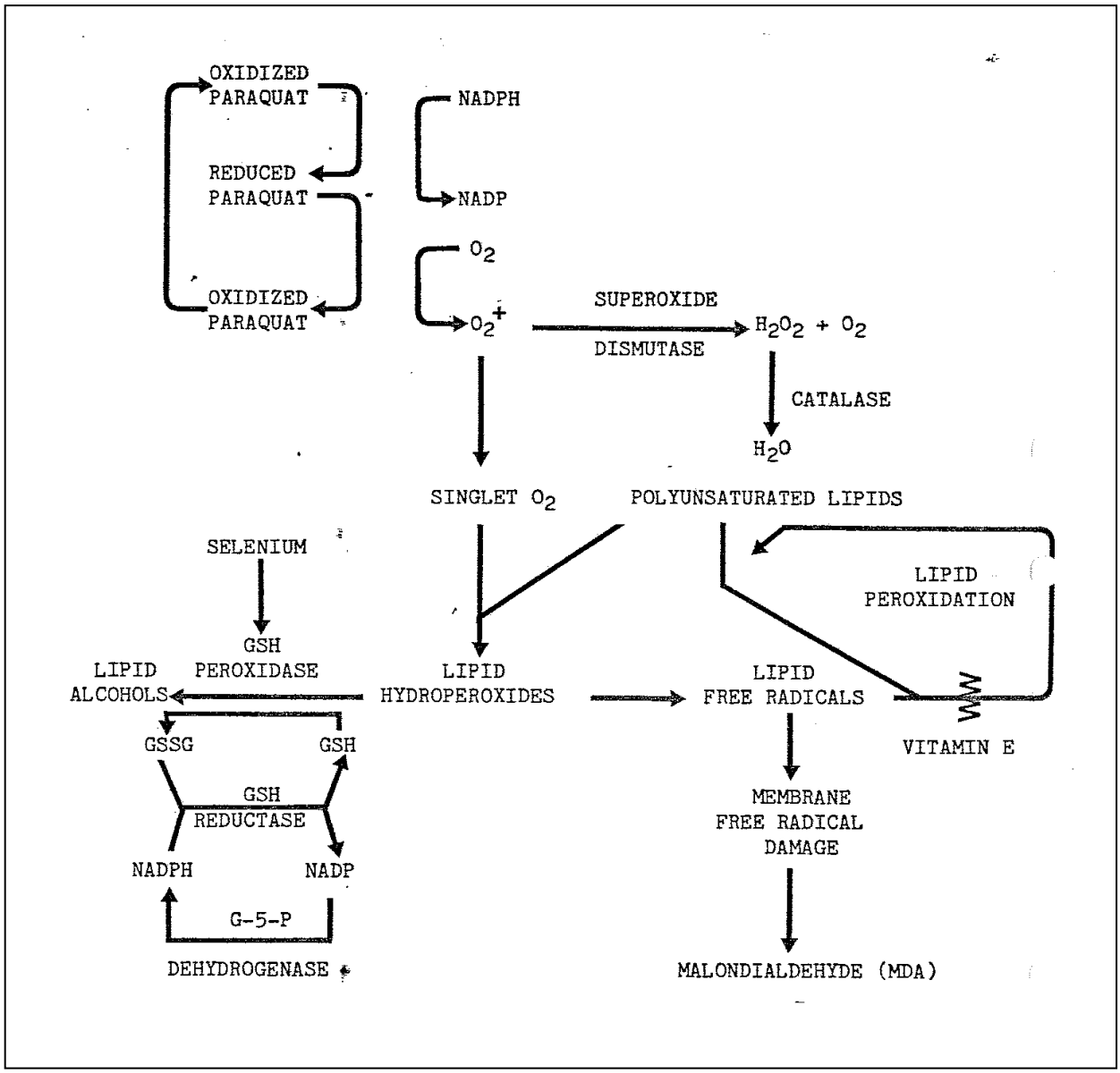


Figure 1. Mechanism for paraquat toxicity according to Bus et al. 1976

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LITERATURE REVIEW CONCERNING THE PRESENCE OF THE HERBICIDE PARAQUAT IN SOIL AND WATER

Between 100 and 200 references were found in a literature search in the above-mentioned area. As there are around ten review articles on various sub-areas, these have been primarily used, supplemented by data from original papers. The result of the literature review has been arranged as follows:

Introduction
Binding of paraquat in soil
- Adsorption of paraquat to clay
- Adsorption of paraquat to organic matter
- Quantity of paraquat adsorbed in soil

Transportation of paraquat in soil
Photochemical breakdown of paraquat Breakdown of paraquat in soil
Persistence of paraquat in soil
Persistence of paraquat in natural waters
Effects of paraquat on soil organisms
Summary
Literature

INTRODUCTION

Paraquat is normally used at doses of 0.1 - 2 kg a.s./ha. In normal spraying some of the preparation ends up on sprayed plants and some on the soil surface. Paraquat is not volatile (Haier-Bode, 1971) and is therefore not released to the atmosphere. Photochemical breakdown may, however, occur (Calderbank 1968). Paraquat that has ended up on plants enters the soil when the plants shrivel and are mixed into the soil (Riley et al., 1976). The quantity of paraquat that eventually reaches the soil varies depending on the thickness of the plant cover and the intensity of the UV radiation. In a study of 50 different spraying occasions it was found that between 10 and 100% of the sprayed quantity of paraquat entered the soil (Riley et al., 1976). In the soil, paraquat is adsorbed to organic matter (Khan, 1974) and to clay (Hayes et al., 1975). The adsorption to clay is very strong, while adsorption to organic matter is somewhat weaker. In the latter case desorption is sufficient to permit uptake of detectable quantities of paraquat in plants, soil animals and soil microorganisms, and to enable biological breakdown of the herbicide at a significant rate. The adsorption capacity of clay for paraquat is very

high and only permits insignificant desorption, resulting in long persistence for the herbicide but also unavailability for plants and soil organisms. Figure 1 shows in schematic form mechanisms of significance to the presence of paraquat in the soil. An account is given below of the adsorption, transportation, breakdown and effects on soil-living organisms of paraquat.

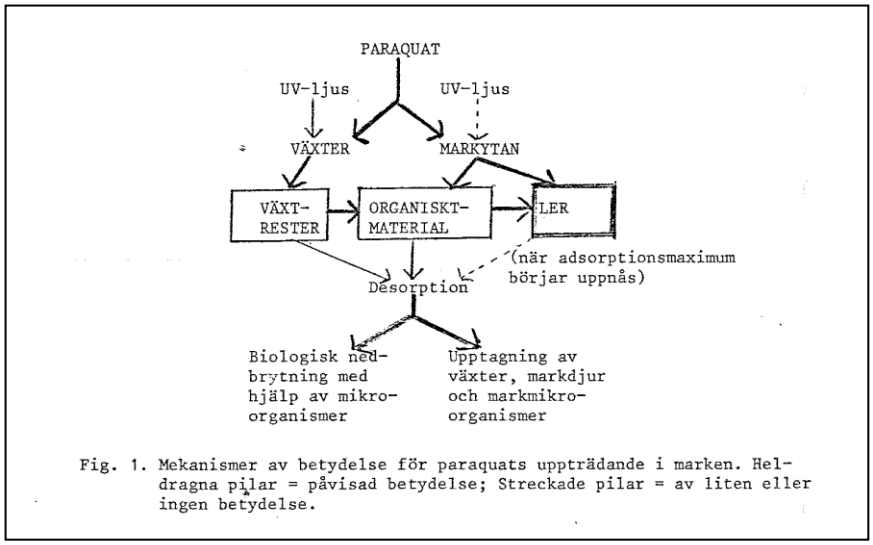


Fig. 1. Mekanismer av betydelse för paraquats uppträdande i marken. Heldragna pilar = påvisad betydelse; Streckade pilar = av liten eller ingen betydelse.

UV-ljus = UV light
VÄXTER = PLANTS
MARKYTAN = SOIL SURFACE
VÄXTRESTER = PLANT RESIDUES
ORGANISKT MATERIAL = ORGANIC MATTER
LER = CLAY
(när adsorptionsmaxium börjar uppås) = (when adsorption maximum starts to be reached)
Biologisk nedbrytning med hjälp av mikroorganismer = Biological breakdown by microorganisms
Upptagning av växter, markdjur och markmikroorganismer = Uptake by plants, soil animals and soil microorganisms

Fig. 1. Mechanisms of significance to the presence of paraquat in the soil. Solid arrows = proven significance; dashed arrows = of little or no significance.

BINDING OF PARAQUAT IN SOIL

The binding of paraquat in soil has been the subject of a considerable number of studies. In the following reference is principally made to review articles written by Akhavein et al. (1968), Calderbank (1968), Khan (1974), Hayes et al. (1975) and Riley et al. (1976).

Paraquat is adsorbed in soil by ion exchange (Calderbank, 1968). The positively charged paraquat ion is bound to negative charges in the soil. The adsorption capacity of paraquat in soil is smaller than for inorganic cations, for example ammonium ions. On the other hand, the tendency towards adsorption is greater for paraquat than for ammonium. Paraquat is adsorbed to soil in the presence of surplus ammonium. Even with a very large surplus of ammonium it is not possible to displace more than a part of adsorbed paraquat. NH_4^+ and K^+ in this respect are approximately equivalent, while Ca^{2+} and Na^+ displace less than the other two ions.

It is characteristic of paraquat that its phytotoxic persistence in soil is generally short: less than one week for mineral soils but 2 months or more for humus soils (Ogräsnyckeln, 1980). The difference in persistence is due to the herbicide being adsorbed at differing strength to clay and organic matter. This is clarified in Figure 2,

which presents adsorption isotherms for paraquat in a sandy soil (Calderbank, 1968, from Knight et al., 1967), firstly in a natural sandy soil and secondly after treatment of the soil with peroxide to destroy the organic matter.

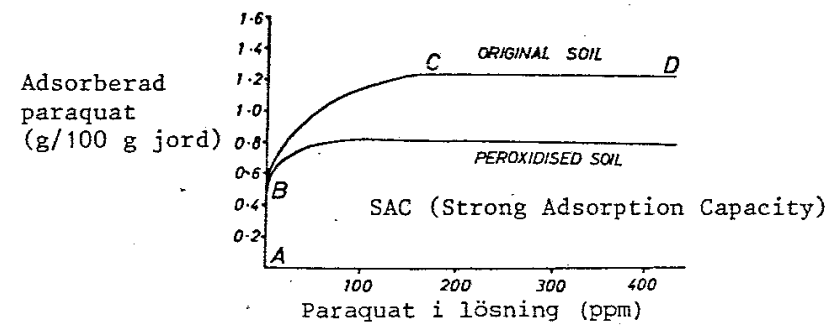


Fig. 2. Adsorptionsisotermer för paraquat i en sandjord med och utan peroxidbehandling (Knight et al., 1967).

Adsorberad paraquat = Adsorbed paraquat (g/100 g soil)
Paraquat I lösning = Paraquat in solution (ppm)

Fig. 2. Adsorption isotherms for paraquat in a sandy soil with and without peroxide treatment (Knight et al., 1967).

Within area A – B (Fig. 2) the quantity of paraquat in solution is so small that it usually cannot be detected in analysis. This area represents very powerful adsorption of the herbicide and is often referred to as SAC in the literature. Within the area B – C paraquat can be detected in solution. C – D represents saturation in adsorption. In the peroxide-treated soil the area B – C has decreased substantially, while the area A – B is approximately equal in size. The conclusion is that the strongest adsorption of paraquat takes place to clay.

It has also been shown that paraquat that has been adsorbed to organic matter is transferred to clay (Riley et al., 1976). The process is rapid in dilute solutions, but in damp soil paraquat is rapidly transferred from weak bonds in organic matter, including remnants of sprayed plants, to stronger bonds in clay. In organogenic soils with low clay content the transfer may take several weeks.

Adsorption of paraquat to clay

Adsorption of paraquat to clay constitutes the principal inactivation mechanism for the herbicide in soil. A review of knowledge of the mechanism and the impact of various factors on the process of adsorption has been written by Hayes et al. (1975).

Paraquat is adsorbed to non-expanding clay minerals, e.g. kaolinite, principally through ionic bonding. Paraquat is bound harder to kaolinite than diquat is, due to the paraquat molecule being more flexible and therefore causing closer contact with bonding charges. The mechanism for adsorption of paraquat to expanding clay minerals, e.g. montmorillonite, is more complicated. When paraquat is adsorbed to the intermediate layer of montmorrilonite, an equivalent quantity of inorganic cations is released. The inorganic cations contribute to the water being able to penetrate through the layers of montmorillonite and cause the swelling typical of this clay mineral. On the other hand, paraquat is readily dehydrated, and the clay mineral loses its swelling capacity through the departure of water from the layers. Paraquat is therefore almost inaccessibly bound for exchange with natural ion-exchange partners. It has been shown that 6 M ammonium acetatate can desorb only a very small proportion of paraquat adsorbed to montmorillonite. Under natural conditions the desorbent would be needed in such high concentrations as to be phytotoxic in itself. In this case no actual desorption of paraquat bound to montmorillonite can be anticipated. From kaolinite and also certain other clay

minerals, such as vermicullite and illite, a large surplus of inorganic cations can desorb at least a few per cent of adsorbed paraquat.

Adsorption of paraquat to organic matter

Reviews concerning reactions of humus substances with paraquat have been presented by Khan et al. (1974) and Burns et al. (1974). The adsorption of the herbicide to humus, as to clay, is principally an ion-exchange process. Ionisable H^+ in COOH and OH groups is exchanged with paraquat, and there is therefore an accompanying decrease in pH. Because of steric hindrance, however, only part of the cation exchange capacity of the humus can be utilised. The adsorption equilibrium is therefore also significantly slower than with regard to clay. The adsorption takes place to both weak and strong binding sites in the humus, and the adsorption to the weaker binding sites delays the binding to the stronger ones. The rate of establishment of the adsorption equilibrium is only insignificantly dependent on temperature. If inorganic cations bound to the humus are present, adsorption of paraquat takes place according to the sequence $Al^{3+} < Fe^{3+} < Cu^{2+} < Mn^{2+} < H^+ < Ca^{2+} < Mg^{2+}$.

Desorption of paraquat from organic matter depends on how much of the adsorption capacity can be utilised. Some release of paraquat can be obtained by washing with water. This means that in the soil, where organic matter is present in close contact with clay mineral, paraquat is desorbed from the organic matter to be adsorbed to clay. Damanakis et al. (1970) have shown that paraquat found to peat is rapidly transferred to clay when the two components are mixed.

Quantity of paraquat adsorbed in soil

Many authors have made estimates of how much paraquat can be adsorbed in different soils without the desorption being so great that phytotoxic concentrations are reached in the soil liquid. The SAC has been determined for the soil in question (see explanation in Fig. 2), and on this basis it has been calculated how many kg of paraquat can be added per ha without causing damage to sensitive test plants. The quantity of paraquat that can be bound is generally large. See two different calculations in Table 1. Damanakis et al. (1970) found that six peat soils could bind 88 – 165 kg paraquat/ha in the layer down to 2.5 cm. Summers (1980) states that seven clay soils can bind 56 – 2880 lb paraquat/acre down to a depth of 2.5 cm.

Table 1. Adsorption capacity for paraquat in some soils according to Riley et al. (1976) and Calderbank (1968).

Soil	Humus (%)	Clay (%)	SAC	Quantity of paraquat (kg/ha)
acc. to Riley et al			(µg paraquat/g soil)	In upper 15 cm layer of soil
1	1.7	11	400	720
2	1.9	17	300	540
2	4.8	21	800	1440
4	42.3	-	40	30
acc. to Calderbank			(me paraquat/g soil)	In upper 2.5 cm layer of soil
1	-	42	13.7	2880
2	-	29	4.7	1012
3	-	18	4.1	950
4	-	7.6	0.78	197
5	-	4	0.19	56

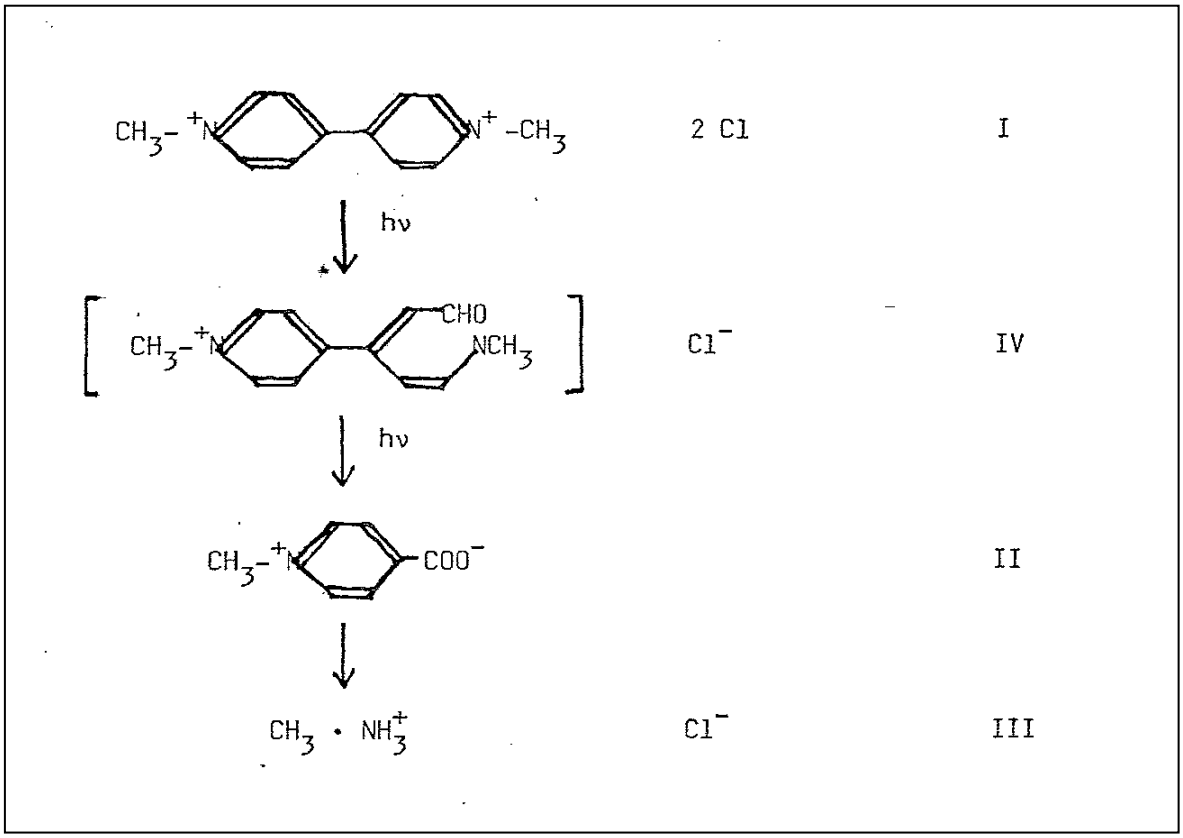
TRANSPORTATION OF PARAQUAT IN THE SOIL

Owing to its strong adsorption to clay and organic matter, paraquat is one of the least mobile pesticides in soil (Helling et al., 1971). Paraquat sprayed onto the surface of a peat soil at a dose of 36 kg/ha was found in the upper 7 mm layer after a 4-month period during which precipitation totalled 305 mm (Damanakis et al., 1970). Nor does transportation take place via the soil water in clay soils. Dilution of the herbicide in the soil can only be obtained through movement of soil particles, to which paraquat is adsorbed. The particles can drop down into natural cracks in the soil, move through water transportation or move through soil cultivation. In an experiment reported by Hance et al. (1980) traces of paraquat after annual application for 12 years at a dose of 4.48 kg/ha to a sandy clay soil could be detected in the 35-40 cm layer.

PHOTOCHEMICAL BREAKDOWN OF PARAQUAT

Breakdown of paraquat under the effect of UV light has been described in reviews by Summers (1980). Paraquat has an absorption maximum for UV radiation at a wavelength of 256 nm.

In an aqueous solution of paraquat (I) exposed to UV radiation two breakdown products were detected, II and III. After 3-4 days only minor quantities of I remained. Product II was slowly broken down to III. Product IV was not detected but was considered a likely intermediate stage.



Breakdown of paraquat by solar radiation appears only to occur to a noticeable extent when the herbicide is adsorbed to a plane surface. In aqueous solution there is little breakdown. Paraquat adsorbed on plants is broken down through the effect of sunlight (see Tab. 2). Products II and III are formed. The breakdown also continued after the plant

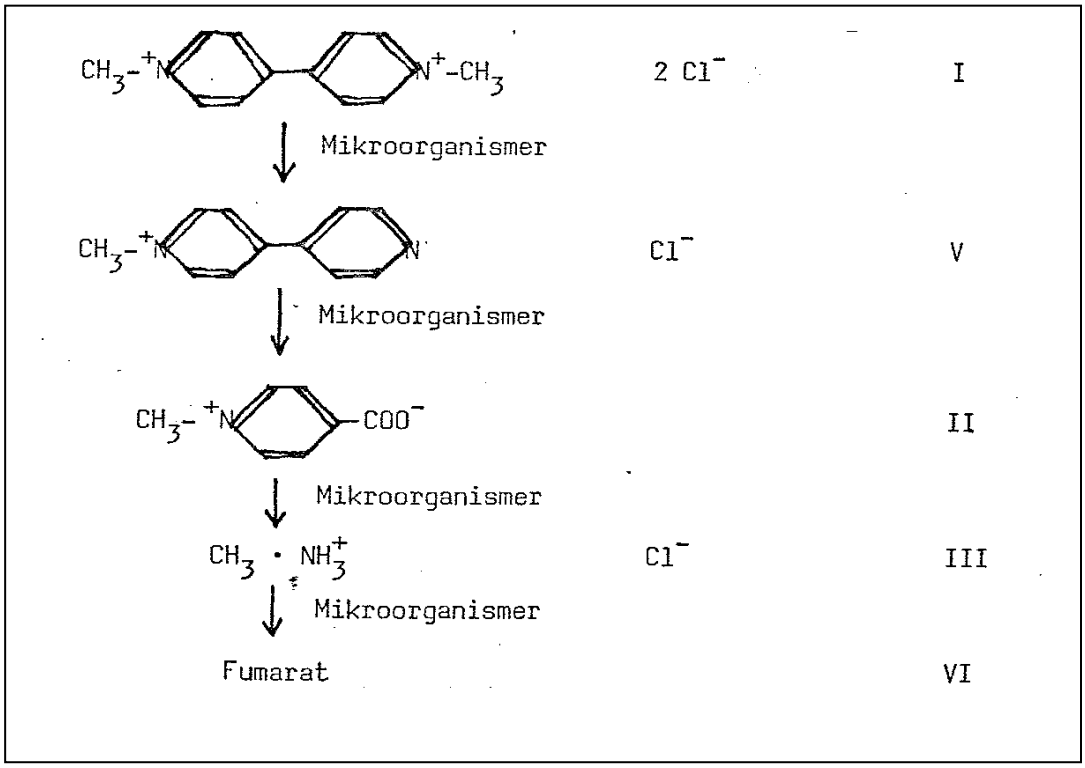
had withered as a result of the paraquat treatment. Paraquat adsorbed to soil does not appear to be broken down to a significant degree by the effect of sunlight.

Table 2. Approximate disappearance of paraquat under the effect of UV radiation or sunlight (Summers, 1980),

		Time (days)	Disappearance (%)
UV radiation,	aqueous solution	3	100
Sunlight,	aqueous solution		Insignificant
	on plants	21	66
	on soil		Insignificant

BREAKDOWN OF PARAQUAT IN SOIL

Breakdown of paraquat in soil is a microbial process. Purely chemical breakdown appears unlikely (Hance, 1967). A number of different microorganisms, both bacteria and fungi, have been isolated and have been shown to be capable of breaking down the herbicide. Reviews have been presented by Calderbank (1968), Suiraners (1980), Riley et al. (1976). The breakdown in most cases appears to be a cometabolic process which requires access to a suitable energy source for the microorganisms that produce enzymes needed for breakdown. Various breakdown products have been detected. II and III are the same as in photochemical breakdown. Demethylation of paraquat (V) has been shown in microbial breakdown.



It has additionally been shown that III by a bacterium through a hydroxylation mechanism is transferred to fumarate (VI). The microbial breakdown of products from photochemical breakdown of paraquat, II and III, is of significance as remnants of treated plants, where photochemical breakdown occurs, are mixed into the soil.

There is thus no doubt that the microorganisms can break paraquat down to products that are harmless from an environmental point of view. The question is, however, whether the desorption of paraquat in the soil is so great that any significant breakdown for that reason is possible. Several studies have been done (Summers, 1980). The results are not entirely clear, but point towards substantial breakdown of paraquat adsorbed to organic matter (see e.g. Burns et al., 1970), and also breakdown of the herbicide in adsorption to non-expanding clay material. On the other hand, it is doubtful whether any breakdown occurs of paraquat located between the layers of swelling clay mineral. The breakdown of paraquat that occurs in soil takes place principally in the time immediately after application. Remaining herbicide is the part most strongly adsorbed to clay.

PERSISTENCE OF PARAQUAT IN THE SOIL

The phytotoxic persistence of paraquat in mineral soil is short, < 1 week, in humus soil longer > 2 months (Ogräsnyckeln, 1980). Its chemical persistence, on the other hand, is very long. Riley & Wilkinson (1976) present the results of an outdoor pot experiment (Tab. 3).

Table 3. Analysis of paraquat in some soils various times after treatment (Riley et al., 1976).

Soil	Clay (%)	Org. matter (%)	Quantity of paraquat found (µg/g soil) after different times (years)				
			0	1	2	3	4.5
1	1	0.5	4.4	4.0	4.8	4.1	4.2
2	-	~100	47.0	46.7	-	-	-
3	16	1.3	14.5	13.7	13.2	17.4	18.8
4	12	3.3	14.0	15.0	17.1	13.9	16.6
5	26	3.8	4.3	4.9	3.8	5.2	3.9
5	26	3.8	19.4	15.4	14.3	18.3	17.0

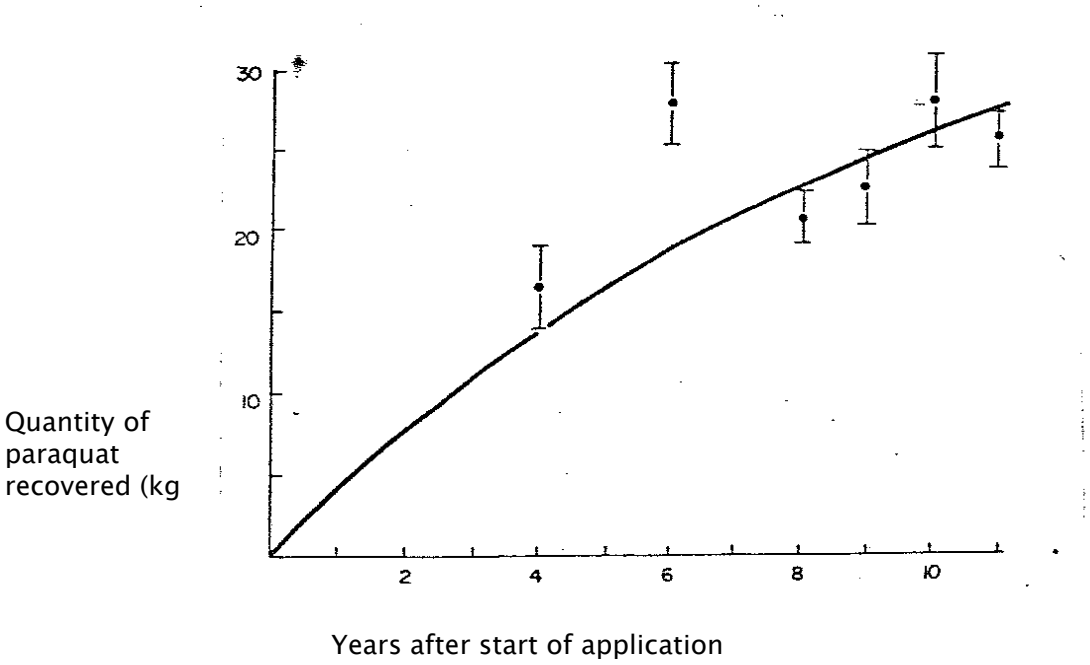
The analyses do not show any disappearance of paraquat in 4.5 years. Similar results were obtained in a Swedish experiment in which no breakdown was detected after 250 days (Stecko et.al., 1976). The transition from weaker bonding to stronger bonding in the soil for paraquat was, however, clearly apparent in this experiment.

Accumulation takes place in repeated application of paraquat over a succession of years, which is apparent from a study reported by Summers (1980), see Table 4. Fryer et al. (1975) also showed that an accumulation of the herbicide had occurred after six annual applications of paraquat. Almost 100% of the paraquat applied over the years could be detected after the sixth year. Further sampling suggested, however, that breakdown nevertheless occurs (Hance et al., 1980), see Fig. 3. A suggestion of breakdown of paraquat under field conditions is also given in Tab. 4. It may possibly be thought that when a soil has reached a certain level of adsorbed paraquat the strongest binding sites are occupied and adsorption takes place through weaker bonds so that desorption and therefore breakdown of the herbicide can take place.

Table 4. Applied quantities of paraquat during a sequence of years to a field experiment (sandy soil: sand 98%, clay 1%, organic matter < 1%) and residual quantities found in the upper 15 cm deep layer of soil. The experiment began in 1965 and was analysed once per year (Summers, 1980).

Analysis	Paraquat added up to time of analysis (kg/ha)	Paraquat recovered (kg/ha)	Paraquat recovered (% of added)
1966	2.2	0.5	23
1967	4.9	1.0	20
1968	6.6	2.7	41
1969	8.5	4.4	52
1970	9.9	4.5	45
1971	11.6	6.7	58
1972	13.0	6.4	49
1973	15.6	5.6	36

Figure 3. Paraquat recovered in soil from a field experiment after annual application of 4.48 kg herbicide/ha (Hance et al., 1980).



PERSISTENCE OF PARAQUAT IN NATURAL WATERS

The presence of paraquat in water has been the subject of many studies, summarised by Summers (1980), because the herbicide has been used for the control of aquatic vegetation. Paraquat disappears relatively rapidly from natural waters, principally due to adsorption to bottom sediment and suspended matter or uptake by aquatic plants and other living organisms. The herbicide tends to accumulate on sediment surfaces either directly or after absorption by aquatic plants and other organisms. The herbicide is transferred to the sediment and is bound there, to a differing extent depending on sediment type, but generally appears to be at least partially available for microbial breakdown.

Initial concentrations of 1-5 ppm paraquat in water are generally not detectable in water samples after one month. In bottom sediments, on the other hand, paraquat is recovered over a long period. In a study, 36% of applied herbicide was found in the upper 5 cm of

sediment after 6 months, and in another study detectable quantities were still found after four years.

EFFECTS OF PARAQUAT ON ORGANISMS IN THE SOIL

Paraquat in aqueous solution is readily taken up by the roots of plants (Damanakis et al., 1970), and the effects on plants have been described by many authors (summarised by Summer, 1980). However, as a result of the strong absorption of paraquat to clay and organic matter a soil may contain considerable quantities of paraquat without the roots of the plants being able to take up phytotoxic quantities. Most soils have an adsorption capacity for strong bonding of paraquat (SAC, see Fig. 2) which is far greater than the application of paraquat at normal doses over a period of many years (Riley & Wilkinson, 1976). Immediately after spraying with paraquat so much of the herbicide may be more weakly bound, however, that it can be taken up by and cause damage to plants which become established just after the treatment (Steckó et al., 1974, 1976).

Earthworms appear not to be affected by normal paraquat doses (Andrén & Steen, 1978). The earthworms eat soil particles containing paraquat, but uptake in the worm body does not appear to occur (Riley et al,, 1976). On the other hand, the quantity of paraquat bound to soil which is present in the worm's gut will be transferred to the higher animals that eat earthworms. Earthworms may have some significance as transporters of paraquat within the soil profile.

Adverse effects of paraquat on certain other soil-living lower animals, for example springtails and mites, have been detected in some cases (Andréen et al., 1978; Eijsackers et al., 1980). Changes in vegetation caused by the herbicide generally have a greater impact on the soil fauna than paraquat does directly (Riley & Wilkinson, 1976).

Several studies into the impact of paraquat on soil microorganisms have been conducted. Reviews are presented by Riley et al. (1976), Anderson (1978) and Torstensson (1979). Microorganisms active in such functions as the turnover of plant remnants and transformation of nitrogen are particularly affected. Breakdown of paraquat-treated straw, for example, is delayed (Grossbard et al., 1974; 1981). Paraquat generally acts more powerfully on fungi than on bacteria, which may possibly explain the effects on turnover of treated plant residues in the soil, as the fungi play a large role here. Some plant-pathogenic fungi are also inhibited somewhat (Grossbard et al., 1976), which is not a negative factor from the point of view of plant cultivation.

An assessment of the impact of paraquat on the growth and various activities of soil microorganisms shows that the herbicide causes disturbances, although generally to a minor extent, to a large number of different microbial processes in the soil, which may result in minor functional disturbances to the soil ecosystem (Torstensson, 1979). No studies of long-term effects in cultivation systems with reduced tillage, in which paraquat is often used, have been found in the literature.

SUMMARY

Well studied	The presence of paraquat in soil may be regarded as having been well studied, particularly with regard to its adsorption to clay and organic matter.
Adsorption	Paraquat is adsorbed very hard to clay, with regard to bonding in the intermediate layer of expanding clay mineral so hard that it is doubtful whether any desorption occurs. The adsorption to organic matter is not as strong. Transfer of paraquat bound in organic matter to stronger bonds on clay takes place in soil.
Adsorption capacity	Most soils have very large capacity to bind paraquat so hard (up to several hundred kg/ha in the upper soil layer) that the quantity dissolved in the soil liquid is below the limit to cause phytotoxic damage.
Mobility	Paraquat is immobile in the soil.
Photochemical breakdown	Paraquat bound in plants can be broken down by the action of sunlight. On the other hand, photochemical breakdown of paraquat bound on the soil surface does not appear to occur to any significant extent.
Microbial breakdown	In the soil paraquat can be broken down by the action of microorganisms. Purely chemical breakdown is unlikely. For breakdown to take place, paraquat must be available in the soil liquid. Desorption from organic matter takes place relatively rapidly, while desorption from clay takes place slowly or does not take place at all.
Persistence in soil	Persistence of paraquat in soil is principally dependent on its strong adsorption principally to clay but also to organic matter. Disappearance through evaporation is negligible, and leaching does not occur at all. The quantity of paraquat in the soil is reduced by microbial breakdown. The persistence of paraquat in soil is short (less than one week - up to a few months), while the chemical persistence is very long (on some clay soils paraquat will probably not disappear within a foreseeable time). On repeated application accumulation takes place at least up to the limit for hard-adsorbed paraquat. If this limit is exceeded desorption and therefore breakdown takes place.
Persistence in water	The persistence of paraquat in natural waters is short in the actual water (about a month) but significantly longer in bottom sediments (several years).

Availability for soil organisms	Paraquat, particularly on humus soils, is available to living organisms in the soil in the period immediately after application. Uptake through the plants' roots takes place and can damage plants that become established after the herbicide treatment. Microorganisms take up paraquat, which results either in breakdown of the herbicide or, in some cases, in inhibition of the microorganism. Earthworms consume soil particles with accompanying adsorbed paraquat. Animals that eat earthworms take over the herbicide.
Effects on soil organisms	Paraquat at normal doses causes no or only small negative effects on earthworms and other soil-living lower animals. The herbicide acts on the soil microorganisms, in some cases negatively (e.g. delayed turnover of plant remnants), in other cases positively (e.g. inhibited nitrification, inhibition of plant-pathogenic fungi). Disturbances to the function of the soil ecosystem may thus be anticipated, generally to a small extent.

Lennart Torstensson

PRODUKTKONTROLLNÄMNDEN
Byrådirektör
J Dich/BS

Arkivkopia

6/5

Datum
1983-11-03

PKN beteckning
Dnr PK-R-1577-341-

[redacted]
Box [redacted]
GÖTEBORG

Registreringsansökan för medlet Gramoxone 80

Er registreringsansökan för medlet Gramoxone 80, en herbicid för bekämpning av kvickrot framförallt i potatisodling inkom till produktkontrollnämnden 1983-07-20.

Gramoxone 80 är ett vattenlösligt granulat innehållande 8 % parakvat som verksam beståndsdel avsett att ersätta en av företaget återkallad flytande 20 %-ig formulering.

Den nu lämnade dokumentationen visar att det vattenlösliga granulatet "Gramoxone 80" innebär mindre förgiftningsrisk jämfört med den flytande parakvat 20 %-iga formulering som hittills funnits på marknaden. Likväl har vid användning av olika granulatformuleringar utomlands flera svåra olyckstillbud inträffat varav en del med dödlig utgång. Risker för allvarliga förgiftningar till följd av olycksfall kvarstår därför även med denna nya formulering.

Det finns flera alternativa medel med likartad användning på marknaden, vilka inte är behäftade med dessa risker.

Bekämpningsmedlet Gramoxone 80 får därför anses vara behäftat med sådan olägenhet från hälso- och miljöskyddssynpunkt att det ej lämpligen bör användas i bekämpningssyfte.

Produktkontrollnämnden har därför vid sammanträde 1983-11-03 beslutat att med stöd av § 27, 1 st kungörelsen (1973:334) om hälso- och miljöfarliga varor lämna registreringsansökan för medlet Gramoxone 80 utan bifall.

8

Detta beslut har fattats av - förutom ordföranden Paulsson - ledamöterna Lönngren, Bolinder, Brandt, Daglund, Engström, Freiwalds, Vegis och Westerholm. Vid ärendets handläggning i övrigt har närvarit byråchef Kökeritz, avdelningsdirektör Wahlström, byrådirektör Steckő och byrådirektör Dich den senare föredragande.

Talan mot detta beslut förs hos regeringen genom besvär. Besvärshandlingen skall ha inkommit till jordbruksdepartementet, 103 33 Stockholm, inom tre veckor från den dag Ni får del av beslutet.

För produktkontrollnämnden

Valfrid Paulsson

Jan Dich

Kopia för kännedom:

[redacted]
Att: [redacted]
Box [redacted]
[redacted]

Translation:

Page 1

Application for registration of the product Gramoxone 80

Your application for registration of the product Gramoxone 80, a herbicide for treatment of quackgrass, primarily in potato cultivation, was received by the Board for Product Control the 20th of July 1983.

Gramoxone 80 is a water-soluble granulate containing 8% of paraquat as active compound, intended to replace a 20% liquid formulation which has been withdrawn from the market on the initiative of the company.

The documentation presented shows that the water-soluble granulate "Gramoxone 80" has a lower risk of intoxication compared to the 20% liquid formulation of paraquat hitherto being marketed by the company. Nevertheless, the use of granulated formulations abroad, has led to several severe accidents, including fatal intoxications. The risks for severe accidental intoxications therefore still remain also with this new formulation.

There are several alternative products on the market with similar use, which are not associated with these risks.

The pesticide Gramoxone 80 must therefore be regarded as having such inconvenient properties regarding the protection of health and the environment that it is not appropriate to use the product for the treatment of pests.

The Board for Product Control has therefore at its meeting the 3rd of November 1983 decided, supported by the 27th §, first chapter of the communication (1973:344) about products dangerous for human health and the environment, not to accept the application for registration of the product Gramoxone 80.

Page 2

This decision has been taken – including the chairman Paulsson – by the members of the Board: Lönngren, Bolinder, Brandt, Daglund, Engström, Freiwalds, Vegis and Westerholm. Bureau director Kökeritz, department director Wahlström, bureau director Steckó and bureau director Dich (presenting the case) have also participated in the preparations for the decision.

Appeal against this decision may be made to the government. Documents where the appeal is made should be submitted to the ministry of agriculture, 103 33 Stockholm, within a period of three weeks from the day you take part of the decision.

Tor the Board of Product Control

Valfrid Paulsson , Jan Dich (copy to the firm)