METHYL PARATHION MONOGRAPh

SUMMARY

Background

Methyl parathion is an extremely toxic organophosphate insecticide, listed in the World Health Organisation’s highest category for toxicity, IA. It has been in use since the 1950s on a wide variety of crops in many countries. Commonly it has been sold as Folidol, manufactured by Bayer, but it is also manufactured by other companies such as Cheminova and is sold under a variety of names.

Methyl parathion has been banned, restricted or cancelled in at least 21 countries because of health effects. It is included on the FAO/UNEP list for Prior Informed Consent because of its impact on health in developing countries, and 43 countries have not given consent to its import.

Overall Risk

In November 2006 the FAO called for a worldwide ban of methyl parathion because of the high risk of rural workers in tropical climates being poisoned, and stated that its use should be considered unethical. Individual countries should take responsibility for stopping its use (Copenhagen Post 2006).

A comprehensive assessment of the risk posed by methyl parathion, carried out by the US EPA (2003), expressed concern about the potential of the chemical to cause cholinesterase inhibition and peripheral neuropathy, from residues in food, drinking water, and operator exposure. Methyl parathion also poses unacceptable risks to all aquatic and terrestrial species (US EPA 2003).

A recognition of the extreme toxicity of this pesticide to children has led the US government to prohibit its use on food crops that are commonly consumed by children, such as apples, peaches, pears and carrots (ATSDR 2001).

Poisonings

Methyl parathion can cause death by oral, dermal or inhalation exposure and many deaths are known to have occurred. It is highly persistent in the indoor environment, and it is very difficult to remove residues from surfaces and workers clothing. As a result non-occupational poisonings have occurred as well as occupational poisonings. Children are especially vulnerable.

Exposure has caused cardiac arrhythmia, pulmonary oedema, respiratory difficulties, cardiovascular lesions, headache, insomnia, muscle weakness, dizziness, impaired memory, sweating, involuntary muscle contractions, vomiting, diarrhoea, abdominal cramps, effects on the eyes, salivation, sweating, weakness, fatigue, acute kidney nephrosis, liver lesions, neuropsychiatric disorders, tremors, convulsions, coma, paralysis, and death.
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**Acute Toxicity**

Methyl parathion is extremely acutely toxic and its metabolite, methyl paraoxon, appears to be even more toxic, but there is little data available on the rate of formation or half-life of this chemical, and it is usually not included in monitoring data, so little is known about its environmental fate.

Males may be more susceptible than females to acute effects, and children more susceptible than adults.

Death is due to respiratory failure or cardiac arrest.

Methyl parathion interacts with a number of other pesticides, increasing the toxicity of either those chemicals or of the methyl parathion.

**Long-term Toxicity**

Long-term effects include degeneration of the sciatic nerve, behavioural changes, decreased ability to fight infections, and reproductive and developmental effects. Methyl parathion appears to be weakly oestrogenic. It is generally believed to be non-carcinogenic, but there is evidence it may cause cancer.

Methyl parathion can cross the placenta and also contaminate breast milk, affecting both the unborn foetus and the newborn who are most vulnerable to its effects.

**Environmental Effects**

Methyl parathion is very highly toxic to freshwater and marine invertebrates and fish, and to bees, birds, beneficial insects and small mammals. It is moderately toxic to amphibians. At least 48 insect species have developed resistance to methyl parathion.

Methyl parathion has been detected in ambient air, surface water, groundwater, rainfall, coastal fog, soil, sediments, fish, human breast milk, umbilical cord blood, and foods.

**Chemical Profile**

**Common names**
Methyl parathion, parathion-methyl

**Common trade name**
Metacid (previously Folidol)

**Other related chemicals**
Ethyl parathion, parathion

**Chemical name**
0,0-dimethyl 0-p-nitrophenyl phosphorothioate

**Chemical group**
Organophosphate

**CAS number**
289-00-0

**Inerts**
Technical grade methyl parathion contains xylene (Edwards & Tchounwou 2005).

**Metabolites**
Methyl parathion is rapidly degraded in the environment by bacteria in soil and water, and by sunlight and water (ATSDR 2001).

In animals it is rapidly metabolised to methyl paraoxon. This chemical is regarded by the US EPA (2003) as having the same toxicity as the parent, methyl parathion, but the ATSDR (2001) regards it as being more toxic, citing evidence that it can be 5 times as toxic as methyl parathion in laboratory studies.

Methyl paraoxon is then degraded to:
- 4-nitrophenol - highly toxic
- dimethyl phosphate - no data on toxicity.

**Trade names include**
Methyl parathion may also be found in compounds with other insecticides such as acephate, camphorchlor, carbaryl, carbophenothion, cypermethrin, dichlorophen, endosulfan, ethyl parathion, ethion, lindane, malathion, methomyl, methoxychlor, monocrotophos, phosalone, propargite, petroleum oils, tetrachlorfuran; and with the fungicides chlorothalonil, omethoate and sulphur (Kidd & James 1991; US EPA 2003; Environmental Defense 2004).

Uses
Methyl parathion is a non-systemic insecticide, nematicide, acaricide/miticide, with contact, stomach, and some respiratory action (Kidd & James 1991).

It is used for the control of chewing and sucking insects and mites, including thrips, weevils, aphids and leafhoppers, in a very wide range of crops including cereals, fruit, nuts, vines, vegetables, ornamentals, cotton, and field crops (Kidd & James 1991; US EPA 2003).

It is available as a wettable powder, emulsifiable concentrate, dustable powder, ULV liquid, and microencapsulated product (FAO 1997; US EPA 2003).

It is applied aerially, by groundboom and airblast sprayers, and through chemigation (US EPA 2003). It is also applied by handheld or backpack sprayers contrary to the advice of the WHO (FAO/UNEP 1996).

Manufacturers
Bayer has long been the ‘parent’ company for methyl parathion, with its well-known brand ‘Foliod’. However there have also been a number of other manufacturers globally. In 2003 Cheminova (Denmark) took over the registrations of methyl parathion from US company Griffin (Cheminova 2003). It has stated that it will phase out production for developing countries between 2007 and 2010 (FAO 2006). Currently Cheminova supplies methyl parathion manufactured in Denmark to USA, Australia, Brazil, Colombia, Mexico, Cuba, Taiwan and Uruguay. It plans to phase out sales to Mexico, Cuba and Colombia by 2009, but not to the other countries (Cheminova 2007; AGROW 509 2006).

China’s recent ban on production and use of Methyl parathion has reduced the number of manufacturers to 3 who hold permits to produce it for emergency purposes only (AGROW 526 2007).

India produced 464 tonnes of methyl parathion in 2006, as well as importing considerable quantities (AGROW 508 2006).

Regulatory status
Methyl parathion is banned, restricted or cancelled in at least 21 countries and there is no consent to import it into an additional 43 countries (Orme & Kegley 2006; PAN AP 2006).

In the Asia Pacific region it is banned in Cambodia, Indonesia (extreme toxicity to humans and animals), Sri Lanka (fatal and non-fatal poisoning of farmers), Laos, the Philippines, and Thailand. It was been restricted in China (not permitted for use on fruit, vegetables, herbs and tobacco), but was banned from July 2007. It is restricted in Australia, Japan and India. It is still approved in New Zealand but there are no longer any products registered for use. There is no consent to import it in Malaysia, Myanmar, Samoa, South Korea, Vanuatu and Viet Nam. (Orme & Kegley 2006; PAN AP 2006; ERMA 2006; AGROW 494 2006; CIBRC 2007).

Toxicological Assessment

Absorption
Methyl parathion is lipid soluble and can penetrate the skin. It is also absorbed through the respiratory and gastrointestinal tracts. It may be stored in adipose tissue, and the primary mechanism for toxicity is its slow release into the blood stream and subsequently to the nervous system. Some also enters the liver where it is changed into...
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the more harmful methyl paraxoxon. Methyl parathion and its metabolite may be transferred via the placenta to the developing foetus (Edwards & Tchounwou 2005).

**Acute toxicity**
Methyl parathion interferes with the normal functioning of the nervous system and brain, primarily by inhibiting the enzyme acetylcholinesterase, which causes the accumulation of the neurotransmitter acetylcholine at nerve endings. This results in over stimulation of the nervous system (ATSDR 2001).

- WHO Recommended Classification by Acute Hazard: Class 1a, extremely hazardous (FAO/UNEP 1996).
  - Acute toxicity from oral intake – Category 1, very toxic.
  - Acute toxicity from dermal absorption – Category I, very toxic.
  - Acute toxicity by inhalation – Category 1, very toxic.

**Lethal doses**
Lethal dose, LD$_{50}$, is the dose that kills 50 per cent of test animals:
- Oral LD$_{50}$ (rat) = 4.5-24mg/kg body weight (US EPA 2003)
  = 3mg/kg bw (FAO/UNEP 1996)
- Dermal LD$_{50}$ (rat) = 40mg/kg bw (FAO/UNEP 1996)
  = 67-120mg/kg (ATSDR 2001)
- Inhalation LC$_{50}$ (rat) = <0.163mg/L or <7mg/kg (US EPA 2003).

**Gender differences**
Tests indicate that male rodents tend to be more susceptible than females to acute effects of methyl parathion. This is attributed to a more efficient conversion of the parent compound to the more toxic metabolite, methyl paraxoxon (ATSDR 2001).

**Age differences**
Detoxification pathways appear to be more effective in adult rats than in young rats, causing a decrease in susceptibility to methyl parathion with age. Methyl parathion is most toxic to newborn animals (ATSDR 2001).

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**No Observed Adverse Effects Levels**
- Acute NOAEL (inhalation) = 0.11mg/kg/day based on plasma, brain and red blood cell cholinesterase inhibition, and neuropathology at 0.53mg/kg/day, the lowest observed adverse effect level (US EPA 2003).
- Chronic NOAEL = 0.02mg/kg/day based on red blood cell cholinesterase inhibition, neuropathology and haematologic effects seen at 0.21mg/kg/day, the lowest observed adverse effect level (US EPA 2003).

**Skin and eye irritation**
The US EPA (2003) classified methyl parathion as “not a strong eye or dermal irritant” and “not a skin sensitiser”.
- Primary eye irritation: irritation cleared by 7 days – Toxicity Category III.
- Primary skin irritation: score of 0.5 at 72h (maximum score is 2) – Toxicity Category IV.

Allergic dermatitis has been reported in a farmer (ATSDR 2001).

**Chronic toxicity**
Chronic effects noted by the US EPA (2003) include systemic toxicity (decreased hematocrit and erythrocyte levels), retinal degeneration, and sciatic nerve degeneration.

Sub-chronic exposure to low doses of methyl parathion caused effects on enzymes in the liver and plasma indicative of cellular toxicity (Kaur & Dhanju 2004).

**Cardiovascular effects**
Abnormalities in heart rate and electrocardiograms have been reported from animal studies, as well as increased heart-to-body ratio in female rats at high dietary exposure rates (Edwards & Tchounwou 2005).

**Immune system**
Repetto & Baliga (1996) reported the following effects of methyl parathion on the immune system:
- decreased weight of the thymus gland in rats and rabbits;
• decreased secondary antibody response in rats;  
• decreased T-cell proliferative response in humans;  
• decreased resistance to bacteria and fungi in mouse; and  
• decreased overall host resistance in mouse and rabbit (Repetto & Baliga 1996).  

ATSDR (2001) reported that methyl parathion decreased the ability of animals to fight infections in some studies but not in others. Exposure to a mixture of methyl parathion and chlorpyrifos at 1/30th the LD$_{50}$ affected immune function in rats (Liu et al 2006).

**Endocrine disruption**
Methyl parathion is an endocrine disruptor. The US EPA (2003) reported possible endocrine disruption in mammals. The ATSDR (2001) also reported indications of weak oestrogenic activity. It has shown oestrogenic potential similar to 17b-estradiol (the primary natural oestrogen) in trout cells (Petit et al 1997), and it induces activity of aromatase, an enzyme that converts androgens to oestrogen thus increasing breast cancer risk (Laville et al 2006). Exposure to a mixture of methyl parathion and chlorpyrifos at 1/30th the LD$_{50}$ affected endocrine hormone levels in rats, increasing oestradiol in both males and females (Liu et al 2006). It caused increased testosterone and decreased luteinizing hormone in testes (Narayana et al 2006a).

**Nervous system**
Effects noted by the US EPA (2003) included:
• neuropathology and acetylcholinesterase inhibition in the brain, red blood cells and plasma;  
• behavioural effects;  
• sciatic nerve degeneration;  
• peripheral nerve demyelination (after a single oral dose of 7.5mg in rodents).  

Tremor, irritating and purposeless chewing, lacrimation, reduced spontaneous locomotor activity and neuromuscular coordination, and impaired memory have been observed (Edwards & Tchounwou 2005).

Repeated low-dose dermal treatment of rats resulted in inhibition of acetylcholinesterase activity and impairment of both motor function and memory (Zhu et al. 2001). The inhibition of acetylcholinesterase activity was prolonged when exposure was by the dermal route, whereas recovery was much quicker with oral or intravenous exposure (Edwards & Tchounwou 2005).

Repeated exposure to methyl parathion caused greater reduction in acetylcholinesterase activity and muscarinic receptor binding in the brains of neonates than it did in adult rats (Edwards & Tchounwou 2005).

**Reproductive & developmental effects**
Detrimental effects on the reproductive organs of both male and female have been reported, as well as degeneration of placental cells (Edwards & Tchounwou 2005).

Methyl parathion damages the endometrium (lining of the uterus) (Güney et al 2007). It has caused fibrosis and haemorrhage of the endometrium during pregnancy, and significant changes in the duration of oestrus cycle (Edwards & Tchounwou 2005). It decreased uterus weight and reduced the number of healthy follicles (Bretveld et al 2006), and caused degenerative changes in the ovaries of rats (Kaur & Dhanju 2005).

It is described as a reproductive toxicant in the male rat, causing deterioration in the structural integrity of the reproductive organs and in the biochemical parameters in the epididymis (Prashanthi et al 2006). It damaged DNA in mouse sperm cells though oxidative stress (Pina-Guzman et al 2006). Neonatal exposure affected the growth and function of the male reproductive system, increasing abnormal sperm and testosterone, and decreasing epididymal sperm count, luteinizing hormone and the number of seminiferous tubules in adult rats (Narayana et al 2006a). It has also caused cytotoxic damage and tubular atrophy in the testis of rats (Narayana et al 2006b).

Other reported effects include:
• postnatal functional toxicity following prenatal exposure, including inhibition of acetyl cholinesterase and other biochemical markers, and impaired behaviour (ATSDR 2001; US EPA 2003);
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• newborn animals more sensitive to acute lethality than adults (Liu et al 1999; US EPA 2003);
• decreased foetal survival and convulsions in surviving rat pups (US EPA 2003);
• embryotoxicity or foetotoxicity observed at non-maternally toxic levels (US EPA 2003);
• retarded growth which included reduced body weight and reduced body length and length of leg bones, short neck, muscular hypoplasia of legs, abdominal hernias and haemorrhagic spots in brain and upper body – in developing chick embryos exposed to doses of methyl parathion via injection into the yolk sac (Kumar & Devi 1992);
• malformations of the spinal column (scoliosis) and/or limbs (short and thick long bones with the epiphyses grossly twisted) in tadpoles kept for 2 weeks in sublethal levels of methyl parathion, the defects in bone formation being caused by changes in composition of the connective tissue (Alvarez 1995).

The ATSDR (2001) reported the potential for non-linearity in the dose-response for behavioural effects and that no dose-response relationship can be established for developmental toxicity from available data.

Birth defects (teratogenicity)
None have been observed in laboratory studies or humans according to FAO/UNEP (1996).

Genotoxicity / mutagenicity
Results of most in vitro genotoxicity studies on both mammalian and bacterial cells were positive. IARC (1987) concluded there is sufficient evidence of mutagenicity in some cellular systems. Results of in vivo studies were equivocal (FAO/UNEP 1996). A 1974 study of 31 patients exposed to methyl parathion found significant increases in chromosomal aberrations in lymphocytes, but a later smaller study of 5 patients did not (Edwards & Tchounwou 2005).

Cancer
Methyl parathion has the potential to cause cancer, based on evidence of mutagenicity, although some agencies have reached different conclusions on the basis of industry-generated data. For example the US EPA decided there was no evidence of carcinogenicity seen in any study submitted to the Agency, so classified it as Group E or “Not Likely” (US EPA 2003). The International Agency for Research on Cancer (IARC 1983) was unable to provide a classification on cancer for methyl parathion, although this decision has not been revisited since 1987 despite more recent evidence of mutagenicity. For example it has been found to damage DNA in human lymphocytes in vitro (Undeger & Basaran 2005).

According to Edwards & Tchounwou (2005), chronic low-level exposure to parathion and methyl parathion from early infancy may lead to cancer later in life in laboratory animals.

Toxic interactions
A number of studies have demonstrated that methyl parathion can have either cumulative or synergistic interactions with other chemicals, increasing the toxicity of either those chemicals or the methyl parathion:

• Rats exposed to chlorpyrifos before being exposed to methyl parathion, suffered dramatically increased toxic effects from the methyl parathion, because the chlorpyrifos blocked the detoxification of the methyl parathion (Karanth et al 2004).
• Methyl parathion in combination with permethrin considerably increased the toxicity of the permethrin when fed to rats (Ortiz et al 1995).
• Synergistic effects were seen with methyl parathion and alpha-cypermethrin (Sun et al 2000).
• Both methyl parathion and its metabolite methyl paraoxon have been found to increase the mutagenicity of some dietary heterocyclic aromatic amines (2-acetoxyacetylamino fluorene; 2-amino-1-methyl-6-phenylimidazo (4,5-b)pyridine; 2-amino-3-methylimidazo-(4,5-f)quinoline) (Wagner et al 2003).
• *Hyalella azteca* (invertebrates) that were pre-treated with atrazine were much more sensitive to methyl parathion compared with *H. azteca* that were not pre-treated with atrazine before being tested (Anderson & Lydy 2002).
• A low, non-toxic, dose of methyl parathion was found to increase the toxicity of propoxur (Institoris et al 2004).
• Methyl parathion and endosulfan, when administered individually, were found to
have marginal effects on the behaviour of rats, but can produce behavioural alterations when given in combination (Castillo et al 2002).

Health Effects and Poisonings

Exposure

Exposure guidelines
The reference dose (RfD) is an estimate of the amount of a given chemical a person can consume each day over a lifetime without incurring 'appreciable risk' of negative effects.

- Acute oral RfD: 0.0011mg/kg/day (US EPA 2003).
- Chronic oral RfD: 0.0002mg/kg/day (US EPA 2003).
- Acceptable Daily Intake (Codex): 0.003mg/kg diet (FAO/UNEP 1996).

In the USA, a re-entry period of 4-5 days has been established for areas where methyl parathion has been applied (US EPA 1999).

Occupational
Workers may be exposed when mixing, loading, and/or applying methyl parathion, and on entering previously treated sites for insect scouting, harvesting, weeding, thinning, irrigation, etc.

Protective equipment required in the USA when applying methyl parathion includes coveralls over long sleeved shirt and long pants, waterproof or chemical resistant gloves, chemical resistant footwear plus socks, protective eye wear, and chemical resistant headgear to protect against overhead exposure. For outdoor spraying a dust/mist filtering respirator, and for enclosed areas a respirator with an organic vapour removing cartridge with a prefilter or canister approved for pesticides are required (US EPA 2003).

In a study conducted in the Philippines, it was demonstrated that in the course of a normal spraying operation farmers are exposed to contamination of their clothing and potential dermal absorption (FAO/UNEP 1996).

The World Health Organisation recommends that methyl parathion should be handled and applied only by competently supervised and well-trained applicators, who must follow adequate safety measures and use the chemical according to good application practices. Regularly exposed workers should receive appropriate monitoring and health evaluation. A respirator should be worn. Hand held ULV spraying equipment should not be used. Unprotected workers should be kept out of the area treated for 48 hours (FAO/UNEP 1996).

Non-occupational
Non-workers may be exposed through drift, emissions from nearby production facilities, by living near landfills where methyl parathion has been dumped, or near water containing methyl parathion that washes off nearby land. Others may be exposed through residues in food, drinking water, bathing water, and contact with contaminated clothing or equipment. Laundry workers may be exposed through residues on protective clothing before and after washing (ATSDR 2001).

Children playing in the soil in the vicinity of crops on which methyl parathion is used may be exposed (ATSDR 2001).

Methyl parathion is not degraded indoors, so that when it is intentionally sprayed in homes, drifts in from nearby operations, or is introduced on users' clothes or equipment, exposure can be an ongoing problem. People in homes sprayed in the USA were still regarded as being highly exposed more than a year after the spraying occurred (Rubin et al 2002b), and detectable levels were still found 3 years after spraying (Clark et al 2002). It is also resistant to being removed from indoor surfaces - in some homes wallboard and carpeting had to be removed when repeated surface cleaning failed to remove trace amounts of the methyl parathion (Rubin et al 2002b).

Food
Residues of methyl parathion have been found in many foods, even occasionally in milk (ATSDR 2001), and in drinking water sources such as in China (Na et al 2006).

When sprayed on apples methyl parathion has been found to decompose fastest in fruit left on the tree:  
- 8 day half-life in apples left on tree;  
- 45 day half-life for apples stored at ambient temperature;
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- 68 day half-life for apples in a controlled-atmosphere storage room (ATSDR 2001).

Absorption
Methyl parathion is readily absorbed via all routes of exposure (oral, dermal, inhalation) and is rapidly distributed to the tissues of the body (IPCS 1993; ATSDR 2001).

It is transferred across the placenta to the unborn foetus (ATSDR 2001).

It has been found in human breast milk in Turkmenistan, Tajikistan and Kazakhstan and can be transferred to the newborn infant through breast feeding (ATSDR 2001).

Treatment
People who have been poisoned should be immediately taken to hospital and placed under surveillance by properly trained medical staff. General surveillance and cardiac monitoring should continue for 14 days. Antidotes are atropine sulfate and pralidoxime chloride (FAO/UNEP 1996).

Symptoms and consequences of poisonings
Cases of acute methyl parathion poisoning show symptoms characteristic of poisoning by other cholinesterase-inhibiting organophosphate compounds:
- inhalation causes bloody or runny nose, coughing, chest discomfort, difficult or short breath, and wheezing due to constriction or excess fluid in bronchial tubes;
- skin contact causes localised sweating, involuntary muscle contractions;
- eye contact causes pain, bleeding, tears, pupil constriction, and blurred vision (FAO 1997).

Systemic effects may follow within a few minutes or be delayed up to 12 hours:
- pallor, nausea, vomiting, diarrhoea, abdominal cramps, headache, dizziness, eye pain, blurred vision, constriction or dilation of pupils, tears, salivation, sweating, confusion (FAO 1997).
- acute nephrosis of the kidney, liver lesions (Edwards & Tchounwou 2005).

Severe poisoning can affect the central nervous system causing:
- incoordination, slurred speech, loss of reflexes, weakness, fatigue, involuntary muscle contractions, twitching, tremors of the tongue or eyelids;
- involuntary defecation or urination, psychosis, irregular heart beat, unconsciousness, convulsions, coma;
- and eventually paralysis of the body extremities and respiratory muscles;
- death is due to respiratory failure or cardiac arrest (FAO 1997).

Patients surviving 24 hours after intoxication were found to have degeneration of the heart muscle (Edwards & Tchounwou 2005).

Long-term effects
Chronic effects of exposure have been less well characterised (Wasley et al 2002). An increase in chromosomal aberrations has been reported following acute poisoning (IPCS 1993).

No cases of delayed peripheral neuropathy have been reported according to the ATSDR (2001). One report of long-term neuropsychiatric illness in two aerial application pilots has been associated with exposure to methyl parathion. Effects included dizziness, anxiety, emotional lability, frequent and severe disagreements with the family, and inability to perform familiar tasks (ATSDR 2001).

Susceptible populations
People who have increased susceptibility to methyl parathion include those with organic central nervous system disease, mental disorders, epilepsy, pronounced endocrine disorders, respiratory conditions, cardiovascular disease, circulatory disorders, gastroenteric diseases, liver or kidney disease, chronic conjunctivitis and keratitis, and pregnant women (ATSDR 2001).

Cases of Poisonings
Deaths have occurred from oral, dermal and inhalation exposure (ATSDR 2001).

Six cases of methyl parathion poisoning were reported in Bulgaria between 1965-1968 (FAO/UNEP 1996).
Sixteen cases of methyl parathion poisoning were reported in the lower Rio Grande Valley (Texas, USA) in 1968, mainly following dermal exposure (FAO/UNEP 1996).

A 1971 report details the deaths of 20 men and 10 women from methyl parathion, 26 of which were from intentional ingestion, and 4 from a combination of inhalation and dermal exposure during spraying. The deaths occurred from 2 hours to 9 days after exposure (ATSDR 2001).

Two sisters, aged 4 and 11 years, died when methyl parathion was sprayed indoors in a Mississippi home, according to a 1984 report (ATSDR 2001). The main exposure route was thought to be contaminated drinking water. Five other children presented with lethargy, pinpoint pupils, increased salivation, and respiratory secretions. Adults in the house showed no symptoms.

Methyl parathion has been one of the 12 most frequently reported pesticides causing acute pesticide poisoning in Central America (Kishi 2002).

In Parana State, Brazil, 1,243 poisoning incidents involving methyl parathion were reported between 1982 and 1991 (Dinham 1993).

Acute symptoms have been reported from non-occupational exposure in agricultural communities in El Salvador, through living with a farmer who had applied methyl parathion (exposure occurred via contact with the farmers skin, clothing and contaminated equipment). Symptoms included cramps in limbs, chest pressure, change in defecation, feeling dazed, and eyes tearing (Azaroff & Neas 1999).

In September 2002, 227 women were admitted to hospital in Kerala, India, after being exposed to methyl parathion applied by a broom in a cashew nut peeling shed. Symptoms included nausea, giddiness, eye irritation, diarrhoea, vomiting, headache, and loss of consciousness (Thanal 2003).

In 1999, 24 children in the remote Andean village of Taucamarca were killed and 18 more severely poisoned when they drank a powdered milk substitute that had been contaminated with the pesticide methyl parathion (Rosenthal 2003).

In a series of incidents in the USA over 5-7 years in the 1990s, many thousands of people were exposed to methyl parathion when it was sprayed indoors to control cockroaches in an estimated 9,000 homes and businesses across nine States (Clark et al 2002; Rubin et al 2002a; Imitaz & Haugh 2002; Zeitz et al 2002). 1,014 homes had to be decontaminated (Clark et al 2002). Residents reported headaches, nausea, vomiting, flu-like symptoms, night waking, diarrhoea, restlessness, difficulty breathing, dizziness, abdominal cramps, incoordination, excess salivation, skin rash, and mental confusion. Many were hospitalised, some died (Rubin et al 2002b; McCann et al 2002).

Of 54 homes with pets, 35 reported that at least one had died, including dogs, cats, birds, and fish (Rubin et al 2002b).

More than half the victims interviewed in one study reported symptoms of clinical depression. Those at greatest risk of depressive symptoms were people who had been exposed to the neurotoxin for the longest period of time, among whom there was an overrepresentation of women and African Americans (Rehner et al 2000).

In a subsequent investigation of long-term neurological effects some of the exposed children were found to have impaired short-term memory and attention, and more behavioural and motor skill problems. Behavioural problems included, anger, misbehaving, acting on impulse, sadness, shyness, and difficulty relating to other children (Ruckart et al 2004).

Children appear to be more sensitive to methyl parathion than adults (Wasley et al 2002): in 1 home, 2 adults remained symptom-free whilst 7 siblings were hospitalised and one died (Rubin et al 2002b).
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Environmental Effects

Aquatic toxicity

Freshwater fish
Methyl parathion is highly toxic to freshwater fish. It causes chronic effects in fish at concentrations less than 80ppb, including deceased weight and length. Other effects include behavioural changes, growth reduction, and indirect mortality (US EPA 2003).

Amphibians
It is moderately toxic to amphibians, including larval stages of developing frogs (US EPA 2003).

Freshwater invertebrates
It is very highly toxic to freshwater invertebrates. Chronic effects include decreased number of young, decreased survival of young and decreased growth. Residues in the surface waters of lakes Beasley and Deep Hollow in the Mississippi Delta have significantly impaired growth of the amphipod *Hyalella azteca* (a shrimp-like crustacean) (Moore et al 2007). Large decreases in invertebrate populations can lead to algal blooms and subsequent fish kills by depleting dissolved oxygen (US EPA 2003).

Estuarine/marine
It is very highly toxic to estuarine/marine fish and invertebrates, and causes chronic effects to the later at low concentrations, including to the survival and number of offspring. In fish it causes behavioural changes, cholinesterase inhibition, and ovarian damage (US EPA 2003). It is also very highly toxic to crustaceans and moderately toxic to zooplankton (Orme & Kegley 2006).

Aquatic plants
It is moderately toxic to aquatic plants (marine diatoms).

Toxic doses
- Bluegill sunfish (96hr LC$_{50}$) – 1.0ppm
- Chorus frog (96hr LC$_{50}$) – 3.7ppm
- *Daphnia*, waterflea (48hr LC$_{50}$) – 0.14ppb
- Marine diatom (EC$_{50}$) – 5.3ppm. (US EPA 2003).

Terrestrial ecotoxicity

Birds
Methyl parathion is very highly toxic to birds, from single oral doses, dermal and short-term dietary exposures, posing significant acute and chronic risks. Effects include direct mortality, decreased egg production, changes in maternal care and viability of young birds, anorexia, increased susceptibility to predation, and greater sensitivity to environmental stress. It can pose a risk to the maintenance of viable populations of bird species where its use is widespread (US EPA 2003).

Bees
It is very highly toxic to bees, and likely to cause bee mortality under field conditions of use, including as foliar residues (US EPA 2003). Sublethal concentrations alter foraging behaviour (Guez et al 2005).

Mammals
It is very highly toxic to small mammals, which are adversely affected through oral, dermal and inhalation exposure. Effects include significant decrease in survival of young, and reduction in maternal bodyweight during lactation (US EPA 2003).

- Acute oral LD$_{50}$ (Mallard duck) – 6.6mg/kg
- Acute dermal LD$_{50}$ (Northern bobwhite quail) – 2.9-9.1mg/kg
- LD$_{50}$ (honey bee) – 0.111-0.214ug/bee (US EPA 2003).

Beneficial insects
Methyl parathion is highly toxic to beneficial insects, as it is a broad-spectrum insecticide. One survey found 98-100 percent mortality of predators of mealybugs on citrus (Wakgari & Giliomee 2003). In laboratory conditions methyl parathion 50 EC (0.05 percent), had a direct knockdown effect on the ecologically important soil micro-arthropod *Cyphoderus* sp (Joy & Chakravorty 1991).

Soil Micro-organisms
Long-term contamination of soil with methyl parathion altered the composition of microbial communities including increasing the presence of gamma-proteobacteria (Zhang et al 2006), a group of bacterial pathogens responsible for
a number of human diseases such as typhoid, cholera, plague, etc.

**Insecticide resistance**
At least 48 insect species are known to have developed resistance to methyl parathion, making the insecticide ineffective. These include pests of cotton, apples, potato and other vegetables, rape, corn, sorghum, chickpea, tobacco, cereals, rice, flowers, soybean, and sugarcane, as well as mosquitoes, house flies and sheep blowfly (Whalon et al 2004).

**Environmental fate**
- **Soil**: degrades rapidly in soil under non-aerobic conditions (flooded soils) with an average half-life of 7 days, but has an average half-life of 64 days in non-flooded aerobic soils (ATSDR 2001). It also degrades faster in alkaline soils. It is relatively mobile in soils (US EPA 2003).

- **Water**: degrades in water (half-life is 24-28 days). It has been detected in rivers in US cotton- and rice-growing regions ranging from 0.42ppb to 6ppb. It has also been detected in high concentrations in river samples in Nicaragua (Castilho et al 2000), and the Ganges in India (Rehana et al 1996).

- **Groundwater contamination**: has been detected, for example it was found in 5.45 percent of wells sampled in cotton growing districts of Punjab, Pakistan (Tariq et al 2004).

- **Ice**: residues from historical use have been found in ice cores taken from the Austfonna ice cap in the Svalbard archipelago in the arctic (Hermanson et al 2005).

- **Air/rain**: can volatilise from plants and soil and has been detected in air, rain and fog samples (ATSDR 2001). It has been detected vaporizing from treated cotton fields 24 hours after spraying (FAO/UNEP 1996). Weekly sampling of air in the Mississippi River valley found methyl parathion in 70 percent of the samples and at the highest concentration (62 ng/m3 air) of any insecticide measured in the study (Foreman et al 2000). It also had the highest concentrations of all pesticides measured in rain (Coupe et al 2000). It has been measured in ambient air breathed by farmers in Tambon Bang Rieng, Thailand (Jirachaiyabhas et al 2004). It has been measured in the air in the vicinity of methyl parathion producing factories (ATSDR 2001).

- **Plants**: phytotoxic effects have been observed in cotton and lettuce, and a reduction of growth has been caused in sorghum (US EPA 2003).

- **Bioaccumulation**: reviews generally state that methyl parathion does not bioaccumulate (e.g. FAO 1997; ATSDR 2001). However De La Vega Salazar et al (1997) found that both methyl parathion and its metabolite 4-nitrophenol did bioaccumulate in the tissues of aquatic organisms in response to environmental stress. A significant concentration in reproductive tissues (plants) and unborn progeny (animals) was found in all samples. ATSDR (2001) suggests that methyl parathion sometimes persists long enough for uptake by fish to occur, and thus could possibly play a role in bioaccumulation.

**Alternatives to Methyl Parathion**

**Alternative insecticides**
There are many other synthetic chemical insecticides on the market, but most of these also have a range of adverse health and environmental effects, such as endocrine disruption, cancer, neurological damage, groundwater contamination, persistence, etc. Hence their use is not recommended as replacements for methyl parathion.

There are some insecticides derived from natural plant extracts that can kill or repel insects; some deter insects feeding, or inhibit their growth. Natural soaps and minerals can also be used, as can naturally occurring pathogens like *Bacillus thuringiensis* (Bt) used as a spray – NOT as a genetically engineered part of the crop itself.

Care must be taken even with natural plant extracts as some, such as pyrethrum, can have toxic effects on beneficial insects, animals, and humans. Other plant extracts include neem, lemon grass, and galanga.
Generally an insecticide, even a natural one, should be regarded as the choice of last resort, with the primary focus being placed on alternative pest management practices that prevent the need for a spray.

**Alternative or ecological pest management**

Alternative or ecological pest management focuses on sustainable ecological solutions that prevent pest build up. It takes a holistic approach to crop management that recognise pests as an integral part of the whole agroecosystem, forming a complex with beneficial insects, weeds, diseases and crops. The self-regulatory mechanisms of a highly biodiverse farming system help keep pest species in balance.

**Elements of alternative or ecological pest management:**

- designing a farm ecosystem that encourages biodiversity, providing habitats for beneficial insects;
- using resistant, often indigenous, crop varieties;
- diversifying crops by intercropping, rotation, and use of multiple varieties;
- cultural practices that encourage healthy soils and hence healthy plants, such as fallowing, appropriate tillage, water management, mulching, and use of animal manures, green manures, vermicasts, composts, liquid bio-fertilisers, and enhanced indigenous micro-organisms;
- cultural practices that contribute to the suppression of pest populations such as varying times of sowing, planting and harvesting, adjusting row width, and use of trap crops;
- companion planting to deter pests;
- enhancing the habitats and hence populations of natural enemies such as parasitoids like the *Encarsia* wasp and predators like the damselfly and spiders, as well as birds and snakes where appropriate;
- accurate identification of both pests and beneficial insects and knowledge of their life cycles, habitats, and periods of population expansion and vulnerability;
- field sanitation – removing infested plant material including crop residues to reduce carryover of pests from one planting to the next;
- systematic scouting of crops for pests and natural enemies, either regularly or at susceptible times, sometimes involving the use of sweep nets, sticky traps, and pheromone traps;
- use of mechanical methods such as light traps, trenches (e.g. to prevent migration of rice molluscs into paddy fields), nets, reflective ribbon, bird perches, pheromone traps, sticky board traps, soil baits, and plant ash.

(SIBAT 1999a, 1999b; OISAT 2004).

The Online Information Service for Pesticide Management in the Tropics (http://www.OISAT.orgl), established by PAN Germany, provides information on managing particular pests in specific crops without the use of methyl parathion.

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Pesticide Action Network Asia and the Pacific (PAN AP) is one of five regional centres of PAN, a global network working to eliminate the human and environmental harm caused by pesticides, and to promote biodiversity-based ecological agriculture.

“Our vision is a society that is truly democratic, equal, just, culturally diverse, and based on food sovereignty, gender justice and environmental sustainability”. Thus PAN AP asserts people’s food sovereignty based on the right to food for all, founded on the right to land and productive resources and the right of communities to decide on our own food and agriculture policies. We are committed to protect the safety and health of people and the environment from pesticide use, and genetic engineering in food and agriculture. We strive to protect and promote the rights, equality and dignity of women. We will promote and protect biodiversity based ecological agriculture. Our goal is to strengthen people’s movements to eliminate hunger and achieve food sovereignty. We endeavour to achieve these goals by empowering people within effective networks at the Asia and the Pacific, and global levels.