6. Lifshitz M, Gavrilov V. Central nervous system toxicity and early peripheral neuropathy following dermal exposure to methyl bromide. *J Toxicol Clin Toxicol* 2000; **38:** 799–801.
7. Yamano Y, *et al.* Three cases of acute methyl bromide poisoning in a seedling farm family. *Ind Health* 2001; **39:** 353–8.
8. Hoizey G, *et al.* An unusual case of methyl bromide poisoning. *J Toxicol Clin Toxicol* 2002; **40:** 817–21.
9. Geyer HL, *et al.* Methyl bromide intoxication causes reversible symmetric brainstem and cerebellar MRI lesions. *Neurology* 2005; **64:** 1279–81.

Uses

Methyl bromide has been used as an insecticidal fumigant for soil and some foodstuffs.

When supplied for fumigation it usually contains chloropicrin as a lachrymatory warning agent.

Methyl bromide has been used as a gaseous disinfectant; it has low antimicrobial activity but good penetrating power.

Methyl bromide was formerly used with carbon tetrachloride in some fire extinguishers. It has also been used as a refrigerant.

◊ General references.

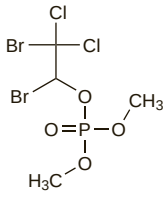
1. WHO. Methyl bromide (bromoethane) health and safety guide. *IPCS Health and Safety Guide 86*. Geneva: WHO, 1994. Available at: http://www.inchem.org/documents/hsg/hsg/hsg86_e.htm (accessed 26/04/04)
2. WHO. Methyl bromide. *Environmental Health Criteria 166*. Geneva: WHO, 1995. Available at: http://www.inchem.org/documents/ehc/ehc/ehc166.htm (accessed 26/04/04)

Naled

Bromchlophos. Dimethyl 1,2-dibromo-2,2-dichloroethyl phosphate.

$C_4H_7Br_2Cl_2O_4P$ = 380.8.

CAS — 300-76-5.



Profile

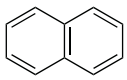
Naled is an organophosphorus insecticide used as a topical ectoparasiticide in veterinary practice.

Naphthalene

Naftalen; Naftaleno; Naphthalin.

$C_{10}H_8$ = 128.2.

CAS — 91-20-3.



Adverse Effects, Treatment, and Precautions

Ingestion of naphthalene can produce headache, nausea and vomiting, diarrhoea, profuse sweating, dysuria, coma, and convulsions. Less than one naphthalene mothball (200 to 500 mg) may cause haemolysis. Ingestion of 5 to 15 g is considered lethal for adults and a dose as low as 74 mg/kg has proved fatal. In children ingestion of 100 mg/kg is lethal. Doses as low as 2 g and 80 mg/kg ingested over 2 days have proved fatal in young children. Treatment is symptomatic; lavage may be considered if the patient presents within 1 hour of ingestion. The vapour is irritating to the eyes; chronic exposure has led to cataract formation. Haemolytic anaemia and haematuria leading to acute renal failure may occur particularly in persons with G6PD deficiency. Blood transfusions may be required.

Pregnancy. Haemolytic anaemia in a neonate has been attributed to inhalation of naphthalene by the mother during the twenty-eighth week of pregnancy.[1]

1. Athanasion M, *et al.* Hemolytic anemia in a female newborn infant whose mother inhaled naphthalene before delivery. *J Pediatr* 1997; **130:** 680–1.

Uses

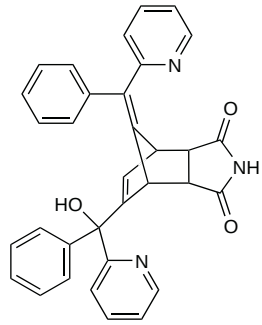
Naphthalene has been used in lavatory deodorant discs and in mothballs. It has also been used as a soil fumigant.

Norbormide

McN-1025; Norbormida. 5-[α-Hydroxy-α-(2-pyridyl)benzyl]-7-[α-(2-pyridyl)benzylidene]-8,9,10-trinorborn-5-ene-2,3-dicarboximide.

$C_{33}H_{25}N_3O_3$ = 511.6.

CAS — 991-42-4.



Profile

Norbormide is a selective rodenticide effective against most species of *rats*, in which it produces extreme irreversible peripheral vasoconstriction. It is not very toxic to other rodents.

Organophosphorus Insecticides

Insecticidas organofosforados; Organofosforlu İnsektisitler.

Description. The organophosphorus or organophosphate insecticides may be esters, amides, or thiol derivatives of phosphoric, phosphonic, phosphorothioic, or phosphonothioic acids.

◊ References.

1. WHO. Organophosphorus insecticides: a general introduction. *Environmental Health Criteria 63*. Geneva: WHO, 1986. Available at: http://www.inchem.org/documents/ehc/ehc/ehc63.htm (accessed 19/07/04)

Adverse Effects

Organophosphorus insecticides are potent cholinesterase inhibitors and can be very toxic. This inhibition results in both muscarinic and nicotinic effects with some central involvement.

Toxic effects may include abdominal cramps, nausea, vomiting, diarrhoea, pancreatitis, urinary incontinence, miosis or mydriasis, weakness, respiratory disturbances, lachrymation, increased salivation and sweating, bradycardia or tachycardia, hypotension or hypertension, cyanosis, muscular twitching, and convulsions. Some organophosphorus compounds cause delayed neuropathy. CNS symptoms include restlessness, anxiety, dizziness, confusion, coma, and depression of the respiratory or cardiovascular system. Patients may experience mental disturbances. Inhalation or external contact can cause local as well as systemic effects.

Repeated exposure may have a cumulative effect though the organophosphorus insecticides are, in contrast to the chlorinated insecticides, rapidly metabolised and excreted and are not appreciably stored in body tissues.

◊ References to the adverse effects and poisoning encountered with organophosphorus compounds such as insecticides (including sheepdips).

1. Minton NA, Murray VSG. A review of organophosphate poisoning. *Med Toxicol* 1988; **3:** 350–75.
2. Karalliedde L, Senanayake N. Organophosphorus insecticide poisoning. *Br J Anaesth* 1989; **63:** 736–50.
3. Öztürk MA, *et al.* Anticholinesterase poisoning in Turkey—clinical, laboratory and radiologic evaluation of 269 cases. *Hum Exp Toxicol* 1990; **9:** 273–9.
4. WHO. Safe use of pesticides: fourteenth report of the WHO expert committee on vector biology and control. *WHO Tech Rep Ser 813* 1991. Available at: http://libdoc.who.int/trs/WHO_TRS_813.pdf (accessed 24/07/08)
5. Casey P, Vale JA. Deaths from pesticide poisoning in England and Wales: 1945–1989. *Hum Exp Toxicol* 1994; **13:** 95–101.
6. Eyer P. Neuropsychopathological changes by organophosphorus compounds—a review. *Hum Exp Toxicol* 1995; **14:** 857–64.
7. Proudfoot A, ed. *Pesticide poisoning: notes for the guidance of medical practitioners*. 2nd ed. London: DoH, The Stationery Office, 1996.
8. Steenland K. Chronic neurological effects of organophosphate pesticides. *BMJ* 1996; **312:** 1312–13.
9. Brown AA, Brix KA. Review of health consequences from high-, intermediate- and low-level exposure to organophosphorus nerve agents. *J Appl Toxicol* 1998; **18:** 393–408.
10. Koksal N, *et al.* Organophosphate intoxication as a consequence of mouth-to-mouth breathing from an affected case. *Chest* 2002; **122:** 740–1.
11. Roberts DM, Aaron CK. Management of acute organophosphorus pesticide poisoning. *BMJ* 2007; **334:** 629–34.
12. Aardema H, *et al.* Organophosphorus pesticide poisoning: cases and developments. *Neth J Med* 2008; **66:** 149–53.

Treatment of Adverse Effects

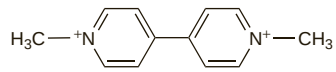
Rapid treatment for poisoning with organophosphorus insecticides is essential. Use of gastric lavage may be considered if a substantial amount has been ingested within 1 to 2 hours of presentation, unless the product is formulated in a hydrocarbon solvent (as there is a high risk of fatal aspiration pneumonia). Contaminated clothing should be removed and the skin, including any areas contaminated by vomiting or hypersecretion, should receive copious and prolonged washing with soap and water. Contamination of the eye is treated by washing of the conjunctiva. The patient should be treated with atropine (see Poisoning, p.1221) and either pralidoxime (p.1460) or obidoxime (p.1456) and symptomatic treatment should be instituted. Diazepam is sometimes given; it may be necessary to give it parenterally in moderate to severe poisoning to control muscle fasciculations and convulsions; oral dosage in mild poisoning may be helpful in relieving anxiety. The patient should be observed for signs of deterioration due to delayed absorption.

Paraquat

Parakuat. 1,1′-Dimethyl-4,4′-bipyridyldiylium ion.

$C_{12}H_{14}N_2$ = 186.3.

CAS — 4685-14-7.



Paraquat Dichloride

Paraquat, dicloruro de.

$C_{12}H_{14}Cl_2N_2$ = 257.2.

CAS — 1910-42-5.

Adverse Effects

Concentrated solutions of paraquat may cause irritation, inflammation, and possibly blistering of the skin, cracking and shedding of the nails, and delayed healing of cuts and wounds. It is not significantly absorbed from undamaged skin. A few fatalities have occurred after skin contact, but these appear to have been associated with prolonged contact and concentrated solutions.

Splashes in the eye cause severe inflammation, which may be delayed for 12 to 24 hours, corneal oedema, reduced visual acuity, and extensive superficial stripping of the corneal and conjunctival epithelium, which usually slowly heals. Inhalation of dust or spray may cause nasal bleeding.

Paraquat weedkillers available for use in domestic gardens contain 2.5% w/v paraquat, sometimes with other herbicides such as diquat. While this strength of paraquat can cause nausea and vomiting as well as some respiratory changes when ingested, it is not considered to be a lethal form.

Most of the cases of severe poisoning follow the ingestion or sometimes injection of the concentrated forms of paraquat herbicide (20% w/v), the distribution of which is restricted to agriculturalists and horticulturalists. Ingestion of, or dermal exposure to small amounts of concentrated paraquat may result in severe pulmonary fibrosis and death within 24 hours. A dose of 10 to 15 mL of the concentrate (20% w/v) is considered to be lethal. Most, but not all patients who ingest 20 to 40 mg of paraquat ion/kg (7.5 to 15 mL of the concentrate) die within 2 or 3 weeks after ingestion. Ingestion of doses of more than 40 mg of paraquat ion/kg (more than 15 mL of concentrate) usually results in death within 1 to 7 days after ingestion. The irritant effects of paraquat are reflected in oesophageal ulceration and gastrointestinal effects. There is widespread organ damage, most notably involving the kidneys and lungs. In such poisoning death is virtually certain and occurs rapidly.

Preparations of paraquat may contain an emetic or a laxative and some contain a malodorous agent to deter ingestion.

◊ General references concerning paraquat toxicity and its treatment.

1. WHO. Paraquat and diquat. *Environmental Health Criteria 39*. Geneva: WHO, 1984. Available at: http://www.inchem.org/documents/ehc/ehc/ehc39.htm (accessed 26/04/04)
2. Bismuth C, *et al.* Paraquat poisoning. *Drug Safety* 1990; **5:** 243–51.
3. Pond SM. Manifestations and management of paraquat poisoning. *Med J Aust* 1990; **152:** 256–9.
4. Paraquat health and safety guide. *Health and Safety Guide 51*. Geneva: WHO, 1991. Available at: http://www.inchem.org/documents/hsg/hsg/hsg051.htm (accessed 26/04/04)
5. WHO. Safe use of pesticides: fourteenth report of the WHO expert committee on vector biology and control. *WHO Tech Rep Ser 813* 1991. Available at: http://libdoc.who.int/trs/WHO_TRS_813.pdf (accessed 21/07/08)
6. Proudfoot A, ed. *Pesticide poisoning: notes for the guidance of medical practitioners*. 2nd ed. London: DoH, The Stationery Office, 1996.
7. Eddleston M, *et al.* Prospects for treatment of paraquat-induced lung fibrosis with immunosuppressive drugs and the need for better prediction of outcome: a systematic review. *QJM* 2003; **96:** 809–24.
8. Dinis-Oliveira RJ, *et al.* Paraquat poisonings: mechanisms of lung toxicity, clinical features, and treatment. *Crit Rev Toxicol* 2008; **38:** 13–71.

Treatment of Adverse Effects

After contact with paraquat, contaminated clothing should be removed and the skin washed with soap and water. The eyes, if splashed, should be irrigated; later topical therapy may involve the symptomatic use of antibacterials or corticosteroids.

There is no specific treatment for paraquat poisoning and the immediate aim is to remove or inactivate the paraquat. Although the benefit of gastric decontamination is uncertain, if the patient presents less than 6 hours after ingestion the initial management involves giving an oral adsorbent, preferably activated charcoal in a dose of 100 g and followed by 50 g every 2 hours for up to 6 hours post ingestion. Fuller's earth (often as a 15% suspension)