zyme values, and there have been some reports of nephrotic syndrome. Nervousness and, sometimes, hallucinations, have also been reported.

**Intolerance.** Three patients with nephropathic cystinosis developed fever, maculopapular eruption, leucopenia, or headache within 2 weeks of starting mercaptamine at doses of 53, 67, and 75 mg/kg daily by mouth, respectively.[1] These adverse effects resolved within 48 hours of drug withdrawal and all 3 patients were able to tolerate mercaptamine when restarted at a dose of 10 mg/kg daily, slowly increased to therapeutic levels over 2 to 3 months. Higher doses of mercaptamine had been associated with lethargy and seizures.

1. Schneider JA, *et al.* Cysteamine therapy in nephropathic cystinosis. *N Engl J Med* 1981; **304:** 1172.

### Pharmacokinetics

◊ Results of a pharmacokinetic-pharmacodynamic study[1] in paediatric patients with nephropathic cystinosis showed that although mercaptamine is rapidly cleared from plasma, dosing every 6 hours was sufficient to maintain the content of cystine in the white blood cells below the target value (see below).

1. Belldina EB, *et al.* Steady-state pharmacokinetics and pharmacodynamics of cysteamine bitartrate in paediatric nephropathic cystinosis patients. *Br J Clin Pharmacol* 2003; **56:** 520–5.

### Uses and Administration

Mercaptamine reduces intracellular cystine levels and is given orally as the bitartrate in the treatment of cystinosis (see below); it has also been given as the hydrochloride. Doses are expressed in terms of the base; 2.94 g of the bitartrate or 1.47 g of the hydrochloride are each equivalent to 1 g of mercaptamine. Mercaptamine bitartrate is given in an initial dose that is one-sixth to one-quarter of the expected maintenance dose, and is then increased gradually over 4 to 6 weeks. The usual maintenance dose in adults weighing over 50 kg is 2 g daily in 4 divided doses with or after food. Children up to 12 years of age are given 1.3 g/m$^2$ (approximately 50 mg/kg) daily in 4 divided doses. Doses are given in conjunction with monitoring of leucocyte-cystine levels which should be kept below 1 nanomol of hemicystine per mg of protein.

Phosphocysteamine, a phosphorothioester of mercaptamine, has been said to be more palatable, and is used similarly.

Mercaptamine facilitates glutathione synthesis and was formerly used intravenously in the treatment of severe paracetamol poisoning to prevent hepatic damage, but other forms of treatment are now preferred (see p.108).

**Cystinosis.** Mercaptamine and phosphocysteamine (which appears to be rapidly hydrolysed to mercaptamine after ingestion) have been reported to be of benefit in children with cystinosis, a rare autosomal recessive metabolic disorder characterised by the intracellular accumulation of cystine. Cystinosis is marked by growth retardation, rickets, Fanconi syndrome, and renal failure; acute episodes of acidosis and dehydration may develop, and there may be photophobia associated with deposition of cystine in the eye.[1] Use of mercaptamine, which results in a reduction in the concentrations of cystine in leucocytes, has been shown to be effective in controlling many of the symptoms,[2-5] especially if begun early, although it has not been clear from contradictory results[4,6] how much benefit would be seen on renal function. Excellent clinical outcomes were reported[7] on long-term follow-up of 2 siblings with severe nephropathic cystinosis, reinforcing the need for early and diligent treatment in order to avoid progressive renal disease. Compliance may be a problem, because of the taste and odour of mercaptamine, and the more palatable prodrug phosphocysteamine has been developed as an alternative;[8,9] more palatable formulations of mercaptamine are also being investigated. Mercaptamine eye drops are reportedly of benefit in reversing or preventing deposition of corneal cystine crystals.[10] A strength of 0.11% has been used. Renal transplantation may be necessary if renal failure develops.

1. Gahl WA, *et al.* Cystinosis. *N Engl J Med* 2002; **347:** 111–21.
2. Yudkoff M, *et al.* Effects of cysteamine therapy in nephropathic cystinosis. *N Engl J Med* 1981; **304:** 141–5.
3. Gahl WA, *et al.* Cysteamine therapy for children with nephropathic cystinosis. *N Engl J Med* 1987; **316:** 971–7.
4. Reznik VM, *et al.* Treatment of cystinosis with cysteamine from early infancy. *J Pediatr* 1991; **119:** 491–3.
5. Gahl WA, *et al.* Nephropathic cystinosis in adults: natural history and effects of oral cysteamine therapy. *Ann Intern Med* 2007; **147:** 242–50.
6. Markello TC, *et al.* Improved renal function in children with cystinosis treated with cysteamine. *N Engl J Med* 1993; **328:** 1157–62.
7. Kleta R, *et al.* Long-term follow-up of well-treated nephropathic cystinosis patients. *J Pediatr* 2004; **145:** 555–60.
8. Gahl WA, *et al.* Cystinosis: progress in a prototypic disease. *Ann Intern Med* 1988; **109:** 557–69.
9. van't Hoff WG, *et al.* Effects of oral phosphocysteamine and rectal cysteamine in cystinosis. *Arch Dis Child* 1991; **66:** 1434–7.
10. Kaiser-Kupfer MI, *et al.* A randomized placebo-controlled trial of cysteamine eye drops in nephropathic cystinosis. *Arch Ophthalmol* 1990; **108;** 689–93.

### Preparations

**Proprietary Preparations** (details are given in Part 3)
***Austral.:*** Cystagon; ***Belg.:*** Cystagon; ***Cz.:*** Cystagon; ***Denm.:*** Cystagon; ***Fin.:*** Cystagon; ***Fr.:*** Cystagon; ***Ger.:*** Cystagon; ***Ital.:*** Cystagon; ***Neth.:*** Cystagon; ***Pol.:*** Cystagon; ***Port.:*** Cystagon; ***Spain:*** Cystagon; ***Swed.:*** Cystagon; ***UK:*** Cystagon.

## Mercuric Chloride

Bicloruro de Mercurio; Chlorid rtut'natý; Cloreto Mercúrico; Corrosive Sublimate; Gyvsidabrio dichloridas; Higany(II)-klorid; Hydrarg. Perchlor.; Hydrargyri dichloridum; Hydrargyri Perchloridum; Hydrargyrum Bichloratum; Kvicksilverdiklorid; Mercuric Chlor.; Mercúrico, cloruro; Mercurique (Chlorure); Mercurique, chlorure; Mercury Bichloride; Mercury Perchloride; Merkuridikloridi; Quecksilberchlorid; Rtęci(II) chlorek.

$HgCl_2$ = 271.5.
*CAS — 7487-94-7.*
*ATC — D08AK03.*
*ATC Vet — QD08AK03.*

**Pharmacopoeias.** In *Eur.* (see p.vii).
**Ph. Eur. 6.2** (Mercuric Chloride). A white or almost white, crystalline powder, or colourless or white or almost white crystals or heavy crystalline masses. Soluble in water and in glycerol; freely soluble in alcohol. Protect from light.

### Profile

The use of mercuric chloride as an antibacterial substance is limited by its toxicity, its precipitating action on proteins, its irritant action on raw surfaces, its corrosive action on metals, and by the fact that its activity is greatly reduced in the presence of excreta or body fluids.

Details of the adverse effects of inorganic mercury compounds are provided under Mercury, below.

### Preparations

**Proprietary Preparations** (details are given in Part 3)
**Multi-ingredient:** ***Spain:*** Pantenil.

## Yellow Mercuric Oxide

Gelbes Quecksilberoxyd; Hydrargyri Oxidum Flavum; Hydrargyri Oxydum Flavum; Mercúrico amarillo, óxido; Mercurique (Oxyde) Jaune; Oxido Amarillo de Mercurio; Yellow Precipitate.

HgO = 216.6.
*CAS — 21908-53-2.*

**Pharmacopoeias.** In *Fr.* and *It.*

### Profile

Yellow mercuric oxide has been used in eye ointments for the local treatment of minor infections including the eradication of pubic lice from the eyelashes. Absorption can occur and produce the adverse effects of inorganic mercury (see Mercury, below).

**Pediculosis.** Yellow mercuric oxide 1% eye ointment was considered to be a safe and effective treatment in pediculosis (p.2034) of the eyelashes caused by pubic lice (pthiriasis palpebrarum).[1]

1. Ashkenazi I, *et al.* Yellow mercuric oxide: a treatment of choice for phthiriasis palpebrarum. *Br J Ophthalmol* 1991; **75:** 356–8.

**Porphyria.** Mercuric oxide has been associated with acute attacks of porphyria and is considered unsafe in porphyric patients.

### Preparations

**Proprietary Preparations** (details are given in Part 3)
***Austral.:*** Golden Eye Ointment†; ***Fr.:*** Pommade Maurice.
**Multi-ingredient:** ***Hung.:*** Dermaforine†; ***India:*** Bell Diono Resolvent; Bell Resolvent.

## Mercurous Chloride

Calomel; Calomelanos; Cloreto Mercuroso; Hydrarg. Subchlor.; Hydrargyri Subchloridum; Hydrargyrosi Chloridum; Hydrargyrum Chloratum (Mite); Mercureux (Chlorure); Mercurioso, cloruro; Mercurius Dulcis; Mercury Monochloride; Mercury Subchloride; Mild Mercurous Chloride; Protocloruro de Mercurio; Quecksilberchlorür.

HgCl = 236.0; $Hg_2Cl_2$ = 472.1.
*CAS — 7546-30-7 (HgCl); 10112-91-1 ($Hg_2Cl_2$).*

NOTE. Precipitated Mercurous Chloride (Hydrargyri Subchloridum Praecipitatum), is a white amorphous powder, for which the synonym 'White Precipitate' (Praecipitatum Album) has been used. White Precipitate has also been used as a name for Ammoniated Mercury.

**Pharmacopoeias.** In *Chin.* as $Hg_2Cl_2$.

### Profile

Mercurous chloride was formerly given as a laxative and was applied topically as an antibacterial. It was one of the mercury compounds employed in the management of syphilis in the pre-antibiotic era.

The mercurous form of mercury does not possess the corrosive properties of the mercuric form and is not absorbed to any great extent. However, the mercurous form can be converted to the mercuric, with consequent toxicity as described under Mercury (see below).

### Preparations

**Proprietary Preparations** (details are given in Part 3)
**Multi-ingredient:** ***Hung.:*** Dermaforine†.

## Mercury

Hydrarg.; Hydrargyrum; Hydrargyrum Depuratum; Mercure; Mercurio; Quecksilber; Quicksilver; Rtęć.

Hg = 200.59.
*CAS — 7439-97-6.*
*ATC — D08AK05.*

**Description.** Mercury is a shining, silvery white, very mobile liquid, easily divisible into globules, which readily volatilises on heating.

### Adverse Effects

Poisoning with liquid mercury or inorganic mercury salts has arisen from sources such as batteries, cosmetics, dental materials, medical equipment, and jewellery manufacture. Barometers, sphygmomanometers, and thermometers are still sources of liquid mercury. Trace amounts of organic and inorganic mercury may also be ingested in the diet.

The effects of **acute** exposure depend on the nature of the compound.

- *Elemental (liquid) mercury* if ingested is poorly absorbed and, unless there is aspiration or pre-existing gastrointestinal disorders, is not considered to be a severe toxicological hazard. The greatest dangers from elemental mercury arise from the inhalation of mercury vapour, which can cause gastrointestinal effects including nausea, vomiting, and diarrhoea; more importantly it is toxic to the respiratory system and this effect can be fatal. Some CNS involvement has also been reported. Adverse effects have also been reported after accidental or intentional parenteral dosage.
- *Inorganic salts* such as mercuric chloride are corrosive when ingested causing severe nausea, vomiting, pain, bloody diarrhoea, and necrosis. The kidney is also involved and tubular necrosis may develop. Mercurous salts are considered to be less hazardous, but the mercurous form can be converted to the mercuric.
- *Organic mercurial compounds* produce similar toxic effects to inorganic compounds, but they have a more selective action on the CNS that has proved difficult to treat. The degree of toxicity varies; those used as preservatives or disinfectants are less toxic than the ethyl or methyl compounds that are not used pharmaceutically or clinically. Methylmercury is notorious for its toxicity; there have been cases of fetal neurotoxicity during outbreaks of methylmercury poisoning.

**Chronic mercury poisoning** may result from inhalation of mercury vapour, skin contact with mercury or mercury compounds, or ingestion of mercury salts over prolonged periods. It is characterised by many symptoms including tremor, motor and sensory disturbances, mental deterioration, gastrointestinal symptoms, dermatitis, kidney damage, salivation, and gingivitis. A blue line may be present on the gums. There is little difference between acute and chronic poisoning with organic mercurials.

The syndrome of *acrodynia* (pink disease), with symptoms of sweat, rash, erythema of the extremities, photophobia, wasting, weakness, hypertension, tachycardia, and diminished reflexes, occurred in children given mercury in teething powders or in ointments or dusting powders. Such preparations have long since been withdrawn from use. However, the syndrome is still a feature of mercury poisoning from other sources.

Hypersensitivity to mercury and mercurial compounds has been reported.

Mercurialentis has been reported in patients treated with eye drops containing an organomercurial preservative.

**Chronic exposure.** Acute *occupational exposure* to mercury vapour in 53 men resulted in an initial phase described as metal fume fever, an intermediate phase of severe symptoms with CNS, gastrointestinal, respiratory, and urological involvement, and a late phase with persistent CNS symptoms, dysuria, and pain on ejaculation.[1,2] Although persistent hyperchloraemia was noted in the 11 patients with the highest mercury levels, renal impairment tended to be temporary.[2]

Long-term follow-up of a patient who had an intravenous *injection* of mercury 12 years previously also revealed no persistent renal impairment,[3] despite the presence of mercury microemboli in lungs, kidneys, liver, and subcutaneous tissues and high concentrations in the urine. At this time, the patient had residual reductions in respiratory function, polyneuropathy, and marked asthenozoospermia. Spermatozoal abnormalities may also have contributed to his wife's miscarriage. Mercury deposits mimicking gallbladder cancer were found in the gallbladder of a patient 35 years after he had injected himself with elemental mercury.[4]

Fetal neurotoxicity after *maternal exposure* to methylmercury is well recognised, and there has been widespread concern about the effect of maternal diets on the developing fetus because of mercury concentrations in freshwater and marine organisms. Results from a study in the Faroe Islands showed an association between delays in neurological development in children and maternal consumption of pilot whale meat.[5] Follow-up studies of this population when they reached 14 years of age suggest that the neurotoxic effects of methylmercury might compromise brain development into the teenage years,[6] as well as affecting autonomic regulation of heart function.[7] However, data from a study conducted in a fish-consuming population in the Seychelles failed to find a similar connection.[8]

There has been considerable concern over the systemic absorption of mercury from *dental amalgam*, which typically contains between 40 and 70% of mercury. However, the quantity absorbed from amalgam fillings is reported to be relatively small[9,10] and current evidence suggests that the use of dental amalgam for tooth restoration is both safe and effective.[11-14] The main risks appear to be occupational exposure of dental staff and environmental pollution. Some patients with hypersensitivity to mercury (manifest most commonly as local lichenoid reactions) may benefit from removal of amalgam fillings.[15-17]

Ethylmercury is contained in *thiomersal*, which is used as a preservative in some vaccines for infants and children, thus representing a potential source of mercury exposure. The safety of these vaccines has been a matter of considerable debate worldwide for many years although it is now usually accepted that there is no evidence of neurotoxicity. However, some countries have phased out vaccines containing thiomersal in favour of alternative preservatives. For further details and references, see Thiomersal p.1664.

The symptoms of *acrodynia* have been mistaken for those of phaeochromocytoma.[18-21]


1. Bluhm RE, *et al.* Elemental mercury vapour toxicity, treatment, and prognosis after acute, intensive exposure in chloralkali plant workers part I: history, neuropsychological findings and chelator effects. *Hum Exp Toxicol* 1992; **11:** 201–10.
2. Bluhm RE, *et al.* Elemental mercury vapour toxicity, treatment, and prognosis after acute, intensive exposure in chloralkali plant workers part II: hyperchloraemia and genitourinary symptoms. *Hum Exp Toxicol* 1992; **11:** 211–15.
3. dell'Omo M, *et al.* Long-term toxicity of intravenous mercury injection. *Lancet* 1996; **348:** 64.
4. Zippel D, *et al.* Quicksilver cholecystitis. *Ann Intern Med* 2006; **144:** 941.
5. Grandjean P, *et al.* Cognitive deficit in 7-year-old children with prenatal exposure to methylmercury. *Neurotoxicol Teratol* 1997; **19:** 417–28.
6. Murata K, *et al.* Delayed brainstem auditory evoked potential latencies in 14-year-old children exposed to methylmercury. *J Pediatr* 2004; **144:** 177–83.
7. Grandjean P, *et al.* Cardiac autonomic activity in methylmercury neurotoxicity: 14-year follow-up of a Faroese birth cohort. *J Pediatr* 2004; **144:** 169–76.
8. Myers GJ, *et al.* Prenatal methylmercury exposure from ocean fish consumption in the Seychelles child development study. *Lancet* 2003; **361:** 1686–92.
9. Eley BM. The future of dental amalgam: a review of the literature. Part 3: mercury exposure from amalgam restorations in dental patients. *Br Dent J* 1997; **182:** 333–8.
10. Eley BM. The future of dental amalgam: a review of the literature. Part 4: mercury exposure hazards and risk assessment. *Br Dent J* 1997; **182:** 373–81.
11. FDI/WHO. Consensus statement on dental amalgam. *FDI World* 1995; **4** (July/Aug): 9–10.
12. Eley BM. The future of dental amalgam: a review of the literature. Part 6: possible harmful effects of mercury from dental amalgam. *Br Dent J* 1997; **182:** 455–9.
13. Bellinger DC, *et al.* Neuropsychological and renal effects of dental amalgam in children: a randomized clinical trial. *JAMA* 2006; **295:** 1775–83.
14. DeRouen TA, *et al.* Neurobehavioral effects of dental amalgam in children: a randomized clinical trial. *JAMA* 2006; **295:** 1784–92.
15. Ibbotson SH, *et al.* The relevance and effect of amalgam replacement in subjects with oral lichenoid reactions. *Br J Dermatol* 1996; **134:** 420–3.
16. McGivern B, *et al.* Delayed and immediate hypersensitivity reactions associated with the use of amalgam. *Br Dent J* 2000; **188:** 73–6.
17. Laeijendecker R, *et al.* Oral lichen planus and allergy to dental amalgam restorations. *Arch Dermatol* 2004; **140:** 1434–8.
18. Henningsson C, *et al.* Acute mercury poisoning (acrodynia) mimicking pheochromocytoma in an adolescent. *J Pediatr* 1993; **122:** 252–3.
19. Velzeboer SCJM, *et al.* A hypertensive toddler. *Lancet* 1997; **349:** 1810.
20. Wößmann W, *et al.* Mercury intoxication presenting with hypertension and tachycardia. *Arch Dis Child* 1999; **80:** 556–7.
21. Torres AD, *et al.* Mercury intoxication and arterial hypertension: report of two patients and review of the literature. Abstract: *Pediatrics* 2000; **105:** 627. Full version: http://pediatrics.aappublications.org/cgi/reprint/105/3/e34.pdf (accessed 14/07/06)


**Effects on the kidneys.** The kidneys are one of the primary sites for the accumulation of mercury in the body. All forms of mercury (liquid mercury, inorganic mercury, and organic mercury) may be toxic to the kidney although the inorganic forms are the most nephrotoxic.[1]


1. Zalups RK. Molecular interactions with mercury in the kidney. *Pharmacol Rev* 2000; **52:** 113–43.


**Hypersensitivity.** Systemic contact dermatitis in a patient apparently sensitised by red, mercury-based tattoo pigments, developed when he ate raw swordfish and alfonsino, both renowned for high mercury levels.[1]


1. Tsuruta D, *et al.* A red tattoo and a swordfish supper. *Lancet* 2004; **364:** 730.


### Treatment of Adverse Effects

The treatment of **acute** mercury toxicity depends on the form of mercury, the route of exposure, and the dose. Supportive measures may be needed with all types of toxicity. Ingestion of elemental mercury seldom requires active treatment since it is poorly absorbed by this route, although inhalation or aspiration of elemental mercury vapour is a potentially serious problem. Poisoning due to organic mercury is difficult to treat and supportive measures are the mainstay of treatment. Acute exposure to mercury by injection requires mainly supportive therapy, although excision of the affected area has been recommended after subcutaneous or intramuscular injection. Mercurials on the skin should be removed by copious washing with soap and water; for contamination in the eye, irrigation with water at room-temperature for at least 15 minutes should be performed.

Acute oral poisoning due to inorganic mercury salts should be treated if appropriate by activated charcoal or gastric lavage to reduce absorption if within 1 hour of ingestion of a potentially life-threatening dose. Decontamination is not usually necessary after ingestion of small amounts such as elemental mercury from a thermometer. Some inorganic mercury compounds may be corrosive to the gastrointestinal tract when ingested, which can make gastrointestinal decontamination problematic; induction of emesis is not recommended. Gastric decontamination may be tried for organic mercury compounds, although the benefits are uncertain.

In severe cases of toxicity with all forms of mercury poisoning, chelation therapy may be required to facilitate the removal of mercury from the body. Unithiol (p.1468) is the chelating agent of choice in the UK and should be considered in symptomatic patients with a blood-mercury concentration of 100 micrograms/litre, and in asymptomatic patients with a blood-mercury concentration of 200 micrograms/litre. Other chelating agents that may be used are succimer (p.1466), dimercaprol (p.1444), and penicillamine (p.1456). The treatment of choice may vary in other countries depending on local policies and availability of the above chelating agents. Penicillamine may be associated with more adverse effects than other chelating agents and should be reserved for use when others are unavailable or not tolerated. Dimercaprol should be avoided in poisoning with metallic mercury or methylmercury because it may exacerbate neurological effects through redistribution of mercury to the brain from other sites.

Some centres start haemodialysis early in the course of treatment; others wait until renal failure develops. Giving a thiol resin complex to prevent the reabsorption of mercury from the bile has also been tried.

The management of **chronic** toxicity is generally symptomatic although chelation therapy has been used in some patients if the blood-mercury concentration is raised or the patient is symptomatic.

◊ References.


1. Kostyniak PJ, *et al.* Extracorporeal regional complexing haemodialysis treatment of acute inorganic mercury intoxication. *Hum Exp Toxicol* 1990; **9:** 137–41.
2. Ferguson L, Cantilena LR. Enhanced mercury clearance during hemodialysis with chelating agents. *Clin Pharmacol Ther* 1991; **49:** 131.
3. Florentine MJ, Sanfilippo DJ. Elemental mercury poisoning. *Clin Pharm* 1991; **10:** 213–21.
4. Bluhm RE, *et al.* Elemental mercury vapour toxicity, treatment, and prognosis after acute, intensive exposure in chloralkali plant workers part I: history, neuropsychological findings and chelator effects. *Hum Exp Toxicol* 1992; **11:** 201–10.
5. Toet AE, *et al.* Mercury kinetics in a case of severe mercuric chloride poisoning treated with dimercapto-1-propane sulphonate (DMPS). *Hum Exp Toxicol* 1994; **13:** 11–16.
6. Houeto P, *et al.* Elemental mercury vapour toxicity: treatment and levels in plasma and urine. *Hum Exp Toxicol* 1994; **13:** 848–52.
7. Aaseth J, *et al.* Treatment of mercury and lead poisonings with dimercaptosuccinic acid and sodium dimercaptopropanesulfonate: a review. *Analyst* 1995; **120:** 853–4.
8. Isik S, *et al.* Subcutaneous metallic mercury injection: early, massive excision. *Ann Plast Surg* 1997; **38:** 645–8.
9. Baum CR. Treatment of mercury intoxication. *Curr Opin Pediatr* 1999; **11:** 265–8.
10. Satar S, *et al.* Intoxication with 100 grams of mercury: a case report and importance of supportive therapy. *Eur J Emerg Med* 2001; **8:** 245–8.
11. Risher JF, Amler SN. Mercury exposure: evaluation and intervention — the inappropriate use of chelating agents in the diagnosis and treatment of putative mercury poisoning. *Neurotoxicology* 2005; **26:** 691–9.


### Pharmacokinetics

There is little absorption of elemental mercury from globules in the gastrointestinal tract. The main hazard of elemental mercury is from absorption after inhalation of mercury vapour; this mercury is widely distributed before being oxidised to the mercuric form. Concentrations can be detected in the brain.

Soluble inorganic mercuric salts are absorbed from the gastrointestinal tract and can also be absorbed through the skin. The mercury is distributed throughout the soft tissues with high concentrations in the kidneys; it is mainly excreted in the urine and faeces with an elimination half-life of about 60 days, although it may take years to eliminate mercury from the brain; elimination from other tissues can take several months.

Organic alkyl mercury compounds are more readily absorbed from both the gastrointestinal and the respiratory tracts. They are widely distributed and can produce high concentrations in the brain. Alkyl mercury compounds are excreted in urine and in the faeces with extensive enterohepatic recycling. The biological half-life varies but is longer than that of inorganic mercury.

Organic mercury, and to some extent inorganic mercury, diffuse across the placenta and are distributed into breast milk.

### Uses and Administration

The hazards associated with mercury generally outweigh any therapeutic benefit and its clinical use has largely been abandoned. The use of mercurial diuretics such as mersalyl (p.1333) has generally been superseded by other diuretics. Ointments containing mercurials, such as ammoniated mercury (p.1604) have also generally been replaced by less toxic preparations. Mercurials were formerly used as spermicides.

Some ionisable inorganic mercury salts and certain organic compounds of mercury have been used as disinfectants, and some mercury salts are effective parasiticides and fungicides. Organic mercurials such as phenylmercuric acetate, borate, and nitrate are also used as preservatives (p.1657). Mercury is a component of dental amalgams.

Other mercury salts that have been used for their antibacterial activity include mercuric chloride, yellow mercuric oxide, and mercurous chloride (above).

**Homoeopathy.** Mercury and some mercury compounds have been used in homoeopathic medicines under the following names:

- Mercury: Hydrargyrum metallicum; Mercurius vivus; Merc. viv.
- Mercuric cyanide: Hydrargyrum bicyanatum; Mercurius cyanatus
- Mercuric iodide: Mercurius iodatus flavus; Merc. i. f.
- Red mercuric iodide: Hydrargyrum biiodatum; Mercurius bijodatus; Mercurius iodatus ruber; Merc. i. r.
- Mercuric nitrate: Hydrargyrum nitricum oxydulatum; Mercurius nitricus oxydulatus; Mercurius nitricus; Merc. nit.
- Red mercuric sulfide: Hydrargyrum sulfuratum rubrum; Cinnabaris; Cinbar.
- Ammoniated mercuric nitrate: Mercurius solubilis; Mere. sol.
- Potassium mercuric iodide: Mercurius et kali iodatus; Merc. ki.

See also Ammoniated Mercury (p.1604), Mercuric Chloride (above), and Mercurous Chloride (above).

### Preparations

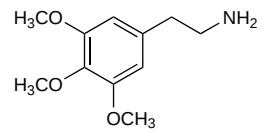
**Proprietary Preparations** (details are given in Part 3)
***Arg.:*** Lagrimas de Santa Lucia†.

**Multi-ingredient:** ***Austria:*** Coldophthal; ***Belg.:*** Ocal; ***Spain:*** Oftalmol Ocular.

## Mescaline

Mescalina; Mescalinum; Meskaliini; Meskalin. 3,4,5-Trimethoxyphenethylamine.

$C_{11}H_{17}NO_3$ = 211.3.
*CAS — 54-04-6.*




NOTE. The following terms have been used as 'street names' (see p.vi) or slang names for mescaline or peyote:
Bad seed; Beans; Big chief; Black button; Blue caps; Britton; Buttons; Cactus; Cactus buttons; Cactus head; Chief; Crystal; Dusty; Full moon; Green button; Half moon; Hikori; Hikula; Hikuli; Hyatari; Indians; M; Mesc; Mesca; Mescal; Mescalito; Mescap; Mescy; Mese; Mess; Mezc; Moon; Musk; Nubs; P; Peyote; Peyotl; Pixie sticks; San Pedro; Seni; Shaman; Topi; Tops.

### Profile

Mescaline is an alkaloid obtained from the cactus *Lophophora williamsii* (*Anhalonium williamsii*, *A. lewinii*) (Cactaceae), which grows in the northern regions of Mexico. The cactus is known in those areas by the Aztec name 'peyote' or 'peyotl' and dried slices of the cactus are called 'mescal buttons'.

Mescaline produces hallucinogenic and sympathomimetic effects similar to those produced by lysergide (see p.2335), but it is less potent. Its effects last for up to 12 hours. It has no therapeutic use. Both Mexican and North American Indians have used peyote in religious ceremonies on account of its hallucinogenic activity.

**Botulism.** Peyote consumed during a ceremonial ritual was believed to have caused botulism in three men.[1] The sample was found to contain type B botulinum toxin when assayed.


1. Hashimoto H, *et al.* Botulism from peyote. *N Engl J Med* 1998; **339:** 203–4.


## Mesoglycan Sodium

Mesoglicano sódico.

### Profile

Mesoglycan sodium is a mucopolysaccharide complex (glycosaminoglycan) extracted from calf aorta, containing mainly suleparoid (heparan sulfate) (p.1406) and dermatan sulfate (p.1256). It has been claimed to have antithrombotic, antiplatelet, and antihyperlipidaemic properties.

◊ References.


1. Forconi S, *et al.* A randomized, ASA-controlled trial of mesoglycan in secondary prevention after cerebral ischemic events. *Cerebrovasc Dis* 1995; **5:** 334–41.