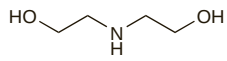
Diolamine *(pINN)*

Diaethanolamin; Dietanoloamina; Diethanolamine; Diolamina; Diolaminum. Bis(2-hydroxyethyl)amine; 2,2′-Iminobisethanol.

Диоламин

$C_4H_{11}NO_2$ = 105.1.

CAS — 111-42-2.



Pharmacopoeias. In *USNF.*

USNF 26 (Diethanolamine). It is a mixture of olamines, consisting largely of diolamine. White or clear, colourless crystals, deliquescing in moist air, or a colourless liquid. Miscible with water, with alcohol, with acetone, with chloroform, and with glycerol; slightly soluble to insoluble in ether, in petroleum spirit, and in benzene. Store in airtight containers. Protect from light.

Profile

Diolamine is an organic base that is used as an emulsifier and dispersant.

It is used to solubilise fusidic acid and sulfafurazole by the formation of the diolamine salt. It has been used for the preparation of salts of iodinated organic acids used as contrast media. It may be irritating to the skin and mucous membranes.

Dioxins

Dioxinas.

NOTE. The name Dioxin has also been applied to dimethoxane.

Profile

The term 'dioxins' encompasses a large group of closely related chemicals known as polychlorinated dibenzo-*p*-dioxins (PCDDs) and polychlorinated dibenzofurans (PCDFs). The most toxic is 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD).

Dioxins are byproducts in the manufacture of commercial chemical products such as chlorinated phenols and polychlorinated biphenyls (PCBs), and can also be produced in smaller quantities by combustion processes and industrial waste. They first came to public attention during the Vietnam war, when they were found to be present in the herbicide Agent Orange used as a defoliant. They are incriminated as causing chloracne (a severe and persistent acne caused by chlorinated compounds). They are potent teratogens and carcinogens in *animals*. An increased incidence of cancer at different organs due to dioxins has been claimed but this has not been substantiated by clinical and follow-up studies. An effect on cell-mediated immunity has been observed.

Exposure should be limited to the lowest feasible concentration.

Adverse effects. The impact of dioxins in food and the environment has been reviewed.[1-4]

An excess of soft tissue sarcomas was found in workers exposed to chlorophenoxy herbicides including those contaminated with TCDD,[5] but cautious interpretation of these results was advised.[6] In Vietnam veterans the risk of non-Hodgkin's lymphoma was about 50% higher than control subjects, but was not related to exposure to Agent Orange, nor was there evidence for an increase in other cancers.[7] Exposure to TCDD was implicated in an increase in cancer mortality in chemical workers,[8,9] but confounding factors such as smoking may have been present.[9,10] Other studies[11,12] have not shown an association between dioxin exposure and an increase in the incidence of human cancer, and epidemiological studies after occupational or accidental exposures have found no clear persistent systemic effects, except for chloracne, and no clear association with carcinogenesis or reproductive disorders.[1,2] Decreased plasma immunoglobulin G concentrations were measured in people after exposure to TCDD 20 years earlier as a result of accidental environmental contamination in Seveso, Italy.[13] A statistically significant increase in the incidence of breast cancer related to serum levels of TCDD was observed in a cohort of 981 women who ranged in age from infancy to 40 years in 1976 at the time of the Seveso accident.[14] The authors pointed out that this cohort is relatively young and continued follow-up would clarify any possible pathogenic role of TCDD.

In the USA, the National Academy of Sciences' Institute of Medicine is reported to have carried out an evaluation of publications on herbicide exposure, largely in industrial and agricultural workers.[15] They concluded that exposure to herbicides or dioxin was associated with soft-tissue sarcomas, Hodgkin's disease, non-Hodgkin lymphoma, chloracne, and porphyria cutanea tarda, and that there was limited evidence of an association with respiratory and prostate cancers and multiple myeloma. An update to the report has also suggested a link between Agent Orange exposure and spina bifida in veterans' offspring.[16] There is some evidence that exposure of men to TCDD is associated with a decreased male to female sex ratio in their offspring.[17] Results from studies[18-20] suggest that prenatal exposure to PCBs has an effect on mental and motor development in early childhood, although this may be counteracted by an advantageous home environment. However, virtually no adverse effects in relation to postnatal exposure to PCBs present in breast milk were demonstrated.[20]

1. Food Standards Agency UK. Dioxins and PCBs in the UK diet: 1997 Total Diet Study (Number 04/00) (issued September 2000). Available at: http://www.food.gov.uk/science/surveillance/fsis2000/4diox (accessed 24/07/08)
2. DEFRA. Dioxins and dioxin-like PCBs in the UK environment (issued October 2002). Available at: http://www.scotland.gov.uk/Resource/Doc/1052/0002248.pdf (accessed 24/07/08)
3. WHO. Dioxins and their effects on human health (issued November 2007). Available at: http://www.who.int/mediacentre/factsheets/fs225/en/print.html (accessed 24/07/08)
4. FAO. Dioxins in the food chain: prevention and control of contamination (issued April 2008). Available at: http://www.fao.org/ag/agn/agns/files/Dioxin_fact%20sheet.pdf (accessed 24/07/08)
5. Saracci R, *et al.* Cancer mortality in workers exposed to chlorophenoxy herbicides and chlorophenols. *Lancet* 1991; **338**: 1027–32.
6. Peto R. Occupational exposure to chlorophenoxy herbicides and chlorophenols. *Lancet* 1991; **338**: 1392.
7. Suskind R. The association of selected cancers with service in the US military in Vietnam. *Arch Intern Med* 1990; **150**: 2449–50.
8. Manz A, *et al.* Cancer mortality among workers in chemical plant contaminated with dioxin. *Lancet* 1991; **338**: 959–64.
9. Fingerhut MA, *et al.* Cancer mortality in workers exposed to 2,3,7,8-tetrachlorodibenzo-p-dioxin. *N Engl J Med* 1991; **324**: 212–18.
10. Triebig G. Is dioxin carcinogenic? *Lancet* 1991; **338:** 1592.
11. Coggon O, *et al.* Mortality and incidence of cancer at four factories making phenoxy herbicides. *Br J Ind Med* 1991; **48:** 173–8.
12. Green LM. A cohort mortality study of forestry workers exposed to phenoxy acid herbicides. *Br J Ind Med* 1991; **48:** 234–8.
13. Baccarelli A, *et al.* Immunologic effects of dioxin: new results from Seveso and comparison with other studies. *Environ Health Perspect* 2002; **110:** 1169–73.
14. Warner M, *et al.* Serum dioxin concentrations and breast cancer risk in the Seveso Women's Health Study. *Environ Health Perspect* 2002; **110:** 625–8.
15. McCarthy M. Agent Orange. *Lancet* 1993; **342:** 362.
16. Stephenson J. New IOM report links Agent Orange Exposure to risk of birth defect in Vietnam vets' children. *JAMA* 1996; **275:** 1066–7.
17. Mocarelli P, *et al.* Paternal concentrations of dioxin and sex ratio of offspring. *Lancet* 2000; **355:** 1858–63.
18. Walkowiak J, *et al.* Environmental exposure to polychlorinated biphenyls and quality of the home environment: effects on psychodevelopment in early childhood. *Lancet* 2001; **358:** 1602–7.
19. Vreugdenhil HJ, *et al.* Effects of prenatal PCB and dioxin background exposure on cognitive and motor abilities in Dutch children at school age. *J Pediatr* 2002; **140:** 48–56.
20. Jacobson JL, Jacobson SW. Association of prenatal exposure to an environmental contaminant with intellectual function in childhood. *J Toxicol Clin Toxicol* 2002; **40:** 467–75.

Diphemanil Metilsulfate *(BAN, rINN)*

Diphemanil Methylsulfate; Diphemanil Methylsulphate; Diphémanil, Métilsulfate de; Diphemanili Metilsulfas; Diphenmethanil Methylsulphate; Metilsulfato de difemanilo; Vagophemanil Methylsulphate. 4-Benzhydrylidene-1,1-dimethylpiperidinium methylsulphate.

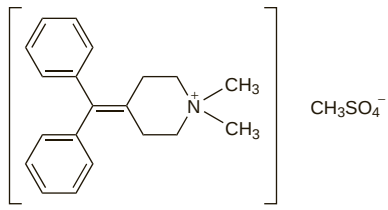
Дифеманила Метилсульфат

$C_{20}H_{24}N,CH_3SO_4$ = 389.5.

CAS — 62-97-5.

ATC — A03AB15.

ATC Vet — QA03AB15.



Profile

Diphemanil metilsulfate is a quaternary ammonium antimuscarinic with peripheral effects similar to those of atropine (p.1219). It is used topically as a 2% cream or powder to treat hyperhidrosis (p.1580).

Diphemanil metilsulfate, given orally, has been used for the treatment of symptomatic bradycardia in infants.

◊ References.

1. Vidal AM, *et al.* Pharmacokinetics of diphemanil methylsulphate in healthy subjects. *Eur J Clin Pharmacol* 1992; **42:** 689–91.
2. Vidal AM, *et al.* Pharmacokinetics of diphemanil methylsulphate in infants. *Eur J Clin Pharmacol* 1993; **45:** 89–91.
3. Pariente-Khayat A, *et al.* Pharmacokinetics of diphemanil methylsulphate in neonates and in premature infants. *Eur J Clin Pharmacol* 1996; **50:** 429–30.

Preparations

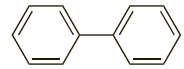
Proprietary Preparations (details are given in Part 3)
Austral.: Prantal; ***Chile:*** Nivelon†; ***Ital.:*** Prantal†; ***NZ:*** Prantal.

Diphenyl

Difenilo; E230; Phenylbenzene. Biphenyl.

$C_{12}H_{10}$ = 154.2.

CAS — 92-52-4.



Profile

Diphenyl is fungistatic against a limited number of moulds and has been employed for impregnating the material used for wrapping citrus fruits.

Adverse effects. Workers exposed to high concentrations of diphenyl (up to 128 mg/m^3) developed toxic symptoms that included irritation of the throat and eyes, headache, nausea, diffuse abdominal pain, numbness, aching of limbs, and general fatigue.[1] One of the workers, who also had somnolence, icterus, ascites, and oedema of the legs, died; at autopsy, the liver showed necrosis. Chronic hepatitis was reported in a woman exposed over a 25-year period to diphenyl in the paper used to pack citrus fruit.[2]

1. Häkkinen I, *et al.* Diphenyl poisoning in fruit paper production. *Arch Environ Health* 1973; **26:** 70–4.
2. Carella G, Bettolo PM. Reversible hepatotoxic effects of diphenyl: report of a case and a review of the literature. *J Occup Med* 1994; **36:** 575–6.

Dipivefrine *(BAN, rINN)* ⊗

Dipivalyl Epinephrine; Dipivefriini; Dipivefrin *(USAN)*; Dipivefrina; Dipivéfrine; Dipivefrinum; DPE.

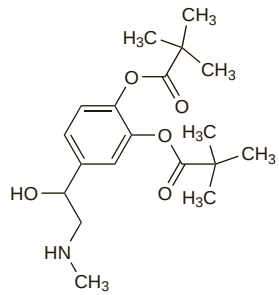
Дипивефрин

$C_{19}H_{29}NO_5$ = 351.4.

CAS — 52365-63-6.

ATC — S01EA02.

ATC Vet — QS01EA02.



Dipivefrine Hydrochloride *(BANM, rINNM)* ⊗

Dipivalyl Adrenaline Hydrochloride; Dipivalyl Epinephrine Hydrochloride; Dipivefriinihydrokloridi; Dipivefrin Hydrochloride; Dipivéfrine, chlorhydrate de; Dipivefrin-hydrochlorid; Dipivefrin-hydroklorid; Dipivefrini hydrochloridum; Dipivefrino hidrochloridas; Dipiwefryny chlorowodorek; Hidrocloruro de dipivefrina. (*RS*)-4-[1-Hydroxy-2-(methylamino)ethyl]-*o*-phenylene dipivalate hydrochloride.

Дипивефрина Гидрохлорид

$C_{19}H_{29}NO_5$,HCl = 387.9.

CAS — 64019-93-8.

ATC — S01EA02.

ATC Vet — QS01EA02.

Pharmacopoeias. In *Chin.*, *Eur.* (see p.vii), and *US.*

Ph. Eur. 6.2 (Dipivefrine Hydrochloride). A white or almost white crystalline powder. Freely soluble in water, in alcohol, and in dichloromethane; very soluble in methyl alcohol.

USP 31 (Dipivefrin Hydrochloride). White, crystalline powder or small crystals, having a faint odour. Very soluble in water. Store in airtight containers.

Profile

Dipivefrine is an ester and prodrug of adrenaline (p.1203). A 0.1% solution of the hydrochloride is used topically as eye drops to reduce intra-ocular pressure in patients with open-angle glaucoma or ocular hypertension (p.1873).

◊ References.

1. Parrow KA, *et al.* Is it worthwhile to add dipivefrin HCl 0.1% to topical β -, β -blocker therapy? *Ophthalmology* 1989; **96:** 1338–41.

2. Drake MV, *et al*. Levobunolol compared to dipivefrin in African American patients with open angle glaucoma. *J Ocul Pharmacol* 1993; **9:** 91–5. Correction. *ibid*.; 385.
3. Albracht DC, *et al*. A double-masked comparison of betaxolol and dipivefrin for the treatment of increased intraocular pressure. *Am J Ophthalmol* 1993; **116:** 307–13.
4. Widengard I, *et al*. Effects of latanoprost and dipivefrin, alone or combined, on intraocular pressure and on blood-aqueous barrier permeability. *Br J Ophthalmol* 1998; **82:** 404–6.

Preparations

BP 2008: Dipivefrine Eye Drops;
USP 31: Dipivefrin Hydrochloride Ophthalmic Solution.

Proprietary Preparations (details are given in Part 3)

Arg.: Propine†; ***Austral.:*** Dipoquin; Propine; ***Austria:*** Glaucothil†; ***Belg.:*** Propine; ***Braz.:*** Propine; ***Canad.:*** Propine†; ***Cz.:*** d Epifrin†; Oftanex†; ***Denm.:*** Oftapinex†; Propine; ***Fin.:*** Oftapinex†; Propine; ***Fr.:*** Propine; ***Ger.:*** d Epifrin; Glaucothil; ***Gr.:*** Diopine†; Glaucodose; Prodren†; Thilodrin; ***Hong Kong:*** Propine; ***Irl.:*** Propine; ***Israel:*** Difrin; ***Ital.:*** Propine; ***Jpn:*** Pivalephrine; ***Malaysia:*** Propine†; ***Mex.:*** Diopine†; ***Neth.:*** Diopine; ***Norw.:*** Oftapinex†; Propine; ***NZ:*** Dipoquin†; Propine; ***Port.:*** Propine†; ***S.Afr.:*** Propine†; ***Singapore:*** Propine†; ***Spain:*** Diopine; Glaudrops†; ***Swed.:*** Oftapinex†; Propine; ***Switz.:*** Diopine†; ***Thai.:*** Propine†; ***UK:*** Propine; ***USA:*** AkPro; Propine.

Multi-ingredient: ***Austria:*** Thiloadren; Thilodigon†; ***Canad.:*** Probeta†; ***Ger.:*** Thiloadren N; Thilodigon; ***Gr.:*** Ryvina†; Thilocombin†.

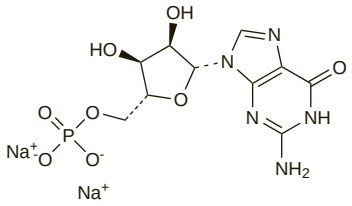
Disodium Guanylate

Disodium Guanosine-5′-monophosphate; E627; Guanilato disódico; Sodium 5′-Guanylate. Guanosine 5′-(disodium phosphate).

Гуанилат Натрия Двузамещенный

$C_{10}H_{12}N_5Na_2O_8P,xH_2O$ = 407.2 (anhydrous).

CAS — 5550-12-9 (anhydrous disodium guanylate).



Profile

Disodium guanylate is used in preparations containing other nucleosides in the treatment of corneal damage. Disodium guanylate has been used as a flavour enhancer in foods. The term sodium 5′-ribonucleotide (disodium 5′-ribonucleotide) has been used to refer to a mixture of disodium guanylate with disodium inosinate (see below).

Preparations

Proprietary Preparations (details are given in Part 3)

Multi-ingredient: ***Belg.:*** Vitacic; ***Cz.:*** Laevadosin†; ***Hung.:*** Vitacic†; ***Mon.:*** Vitacic; ***Rus.:*** Vitacic (Витасик)†.

Disodium Inosinate

Disodium Inosine-5′-monophosphate; E631; Inosinato disódico; Sodium 5′-Inosinate. Inosine 5′-(disodium phosphate).

$C_{10}H_{11}N_4Na_2O_8P,xH_2O$ = 392.2 (anhydrous).

CAS — 4691-65-0 (anhydrous disodium inosinate).

Profile

Disodium inosinate has been used as a flavour enhancer in foods. It has also been given by mouth and been applied topically in the treatment of visual disturbance. The term sodium 5′-ribonucleotide has been used to refer to a mixture of disodium inosinate with disodium guanylate (above).

Preparations

Proprietary Preparations (details are given in Part 3)

Arg.: Lumiclar; Opacout; ***Fr.:*** Catacol; Correctol; ***Ger.:*** Antikataraktikum N.

Multi-ingredient: ***Arg.:*** Antikatarata†.

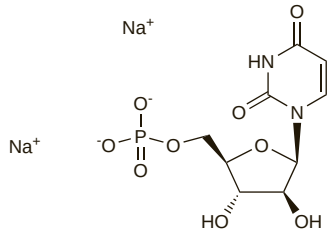
Disodium Uridine Monophosphate

Disodium UMP. 5′-Uridylic acid, disodium salt; disodium 5′ uridylate.

Уридин Монофосфат Динатрия

$C_9H_{11}N_2O_9PNa_2$ = 368.1.

CAS — 3387-36-8.



Profile

Uridine monophosphate is an endogenous uracil nucleotide involved in many biological processes. Disodium uridine monophosphate is included in preparations for neuralgia, neuritis, and myopathies and has also been used for peripheral and cerebral vascular disorders; disodium uridine diphosphate has also been used.

Preparations

Proprietary Preparations (details are given in Part 3)

Multi-ingredient: ***Cz.:*** Laevadosin†; ***Ger.:*** Keltican N; ***Spain:*** Nucleo CMP.

Disulfiram *(BAN, rINN)*

Dissulfiramo; Disulfiraami; Disulfiramas; Disulfirame; Disulfiramum; Diszulfirám; Éthyldithiourame; TTD. Tetraethylthiuram disulphide; Bis(diethylthiocarbamoyl) disulfide.

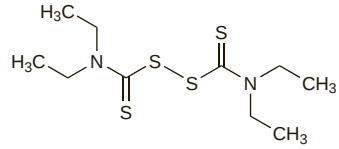
Дисульфирам

$C_{10}H_{20}N_2S_4$ = 296.5.

CAS — 97-77-8.

ATC — N07BB01; P03AA04.

ATC Vet — QN07BB01; QV03AA01.



Pharmacopoeias. In *Eur.* (see p.vii), *Jpn*, and *US*.

Ph. Eur. 6.2 (Disulfiram). A white or almost white, crystalline powder. M.p. 70° to 73°. Practically insoluble in water; sparingly soluble in alcohol; freely soluble in dichloromethane. Protect from light.

USP 31 (Disulfiram). A white to off-white, odourless crystalline powder. M.p. 69° to 72°. Very slightly soluble in water; soluble 1 in 30 of alcohol and 1 in 15 of ether; soluble in acetone, in carbon disulfide, and in chloroform. Store in airtight containers. Protect from light.

Stability. Studies[1,2] on the stability of disulfiram preparations.

1. Gupta VD. Stability of aqueous suspensions of disulfiram. *Am J Hosp Pharm* 1981; **38:** 363–4.
2. Philips M, *et al*. Stability of an injectable disulfiram formulation sterilized by gamma irradiation. *Am J Hosp Pharm* 1985; **42:** 343–5.

Adverse Effects and Treatment

Drowsiness and fatigue are common during initial treatment with disulfiram. Other adverse effects reported include a garlic-like or metallic aftertaste, gastrointestinal upsets, body odour, bad breath, headache, impotence, and allergic dermatitis. Peripheral and optic neuropathies, psychotic reactions, and hepatotoxicity may occur.

Disulfiram-alcohol reaction. The use of disulfiram in the management of alcoholism is based on the extremely unpleasant, but generally self-limiting, systemic effects which occur when a patient receiving the drug ingests alcohol. These effects begin with flushing of the face and, as vasodilatation spreads, throbbing in the head and neck and a pulsating headache may develop. Respiratory difficulties, nausea, copious vomiting, sweating, thirst, chest pain, tachycardia, palpitations, marked hypotension, giddiness, weakness, blurred vision, and confusion may follow. The intensity and duration of symptoms is very variable and even small quantities of alcohol may result in alarming reactions. In addition to the above effects, severe reactions have included respiratory depression, cardiovascular collapse, cardiac arrhythmias, myocardial infarction, acute heart failure, unconsciousness, convulsions, and sudden death.

Severe reactions require intensive supportive therapy; oxygen and intravenous fluids may be necessary. Potassium concentrations should be monitored. Giving intravenous ascorbic acid, ephedrine sulfate, or antihistamines has been suggested.

◊ Reviews.

1. Chick J. Safety issues concerning the use of disulfiram in treating alcohol dependence. *Drug Safety* 1999; **20:** 427–35.

Effects on the blood. There were isolated reports of blood dyscrasias associated with disulfiram in the 1960s. US licensed product information recommends that blood counts should be performed during treatment.

Effects on the liver. A review of 18 cases of hepatitis in patients receiving disulfiram.[1] Symptoms have appeared between 10 days and 6 months after starting disulfiram, and clinical improvement has been seen within 2 weeks of stopping the drug, although liver enzyme values may not return to normal for several months. Fatal hepatic coma had been reported in 7 patients. The clinical picture of disulfiram-induced hepatitis is consistent with a hypersensitivity reaction. Another review[2] evaluated 82 cases of liver injury thought to be due to disulfiram and reported to the Swedish Adverse Drug Reactions Advisory Committee between 1966 and 2002. All but one of the cases were of hepatocellular liver damage, and 4 patients died and 4 underwent liver transplantation. Although there was some evidence that hypersensitivity played a role, it might not be the only mechanism of disulfiram-induced liver disease.

1. Mason NA. Disulfiram-induced hepatitis: case report and review of the literature. *DICP Ann Pharmacother* 1989; **23:** 872–4.
2. Björnsson E, *et al*. Clinical characteristics and prognostic markers in disulfiram-induced liver injury. *J Hepatol* 2006; **44:** 791–7.

Effects on the nervous system. ***ENCEPHALOPATHY.*** A 2% incidence of reversible toxic encephalopathy has been reported in patients receiving disulfiram.[1] Onset varies from days to months following the start of therapy and early signs include impaired concentration, memory deficits, anxiety, depression, and somnolence. Confusion and disorientation follow, often accompanied by paranoid delusions and sometimes hallucinations. Other symptoms may include ataxia, loss of fine motor coordination, slurred speech, and intention tremor. The encephalopathy usually resolves within 3 days to 2 weeks of stopping disulfiram, although symptoms may persist for 6 weeks. There are conflicting opinions on whether this psychosis is a toxic reaction to disulfiram or a response to abstinence from alcohol, but the authors suspected that most cases represent a toxic encephalopathy. However, psychosis without any suggestion of encephalopathy has been reported.[2]

1. Hotson JR, Langston JW. Disulfiram-induced encephalopathy. *Arch Neurol* 1976; **33:** 141–2.
2. Rossiter SK. Psychosis with disulfiram prescribed under probation order. *BMJ* 1992; **305:** 763.

PERIPHERAL NEUROPATHY. Reports of peripheral neuropathy associated with disulfiram and reference to previously reported cases.[1,2] Onset of neuropathy varied from days to months after starting disulfiram treatment and could develop with doses of 250 or 500 mg daily. The most common symptom reported was pins and needles, but numbness, pain/burning, and weakness were frequently described; usually both muscle weakness and sensory loss were noted. Optic atrophy has also been described. Although there might be some improvement immediately after disulfiram withdrawal, the neurological deficit only improved slowly and symptoms might persist for as long as 2 years.[1]

1. Watson CP, *et al*. Disulfiram neuropathy. *Can Med Assoc J* 1980; **123:** 123–6.
2. Frisoni GB, Di Monda V. Disulfiram neuropathy: a review (1971-1988) and report of a case. *Alcohol Alcohol* 1989; **24:** 429–37.

Effects on the respiratory tract. Bronchospasm and hypertension were observed in an asthmatic patient taking disulfiram after an alcohol challenge test.[1]

1. Zapata E, Orwin A. Severe hypertension and bronchospasm during disulfiram-ethanol test reaction. *BMJ* 1992; **305:** 870.

Effects on the skin. Orange-coloured palms and soles, provoking an initial diagnosis of jaundice, developed in a 55-year-old man who had been taking disulfiram for about 2 months.[1] It was postulated that the discoloration was due to accumulation of carotenes in the skin as a result of inhibition of vitamin A metabolism by disulfiram. The discoloration disappeared soon after disulfiram was stopped.

1. Santonastaso M, *et al*. Yellow palms with disulfiram. *Lancet* 1997; **350:** 266.

Overdosage. There has been a report of a 6-year-old boy who experienced disulfiram intoxication after receiving disulfiram 250 mg four times daily to a total of 13 doses but who later recovered.[1] Of 6 previous reports one child died and 3 had moderate or severe brain damage. The syndrome of disulfiram intoxication in children is distinct from the disulfiram-alcohol interaction or acute disulfiram intoxication in adults. It is characterised by lethargy or somnolence, weakness, hypotonia, and vomiting, beginning about 12 hours after ingestion and progressing to stupor or coma. Dehydration, moderate tachycardia, and marked tachypnoea occur frequently, muscle tone is greatly decreased, and deep-tendon reflexes may be weak or absent.

Severe neurological damage has also been reported[2] in a 5-year-old girl after acute disulfiram intoxication which was initially diagnosed as diabetic ketoacidosis.

1. Benitz WE, Tatro DS. Disulfiram intoxication in a child. *J Pediatr* 1984; **105:** 487–9.
2. Mahajan P, *et al*. Basal ganglion infarction in a child with disulfiram poisoning. *Pediatrics* 1997; **99:** 605–8.