in; Cleocin T; Clindagel; ClindaMax; Clindesse; Clindets; Evoclin; ***Venez.:*** Bioclindax; Clindaval; Clindox; Clinfol; Dalacin; Felisep†.

Multi-ingredient: ***Arg.:*** Clindacur; Clindoxyl; CP-Acne; Dermaclean; Duo Clindacin; Ovogin; Perclin; Torgyn Duo; ***Austral.:*** Duac; ***Austria:*** Clindoxyl; ***Braz.:*** Clindoxyl; ***Canad.:*** Benzaclin; Clindoxyl; ***Chile:*** Indoxyl; Klina; ***Cz.:*** Duac; ***Ger.:*** Copal; ***Gr.:*** Indoxyl; ***Hong Kong:*** Duac; ***India:*** Deriva-C; ***Indon.:*** Benzolac Cl; Climadan; Medi-Klin TR; ***Irl.:*** Duac; ***Mex.:*** Benzaclin; Clindapack; Femisan; Gynoclin-V; Indoxyl; Trexen Duo; ***Neth.:*** Duac; ***NZ:*** Duac; ***Pol.:*** Duac; ***Port.:*** Duac; ***Spain:*** Duac; ***Swed.:*** Duac; ***Turk.:*** Cleocin; ***UK:*** Duac Once Daily; ***USA:*** Benzaclin; Duac; Ziana.

Clioquinol *(BAN, rINN)*

Chinoform; Chloroiodoquine; Cliochinolum; Clioquinolum; Iodochlorhydroxyquin; Iodochlorhydroxyquinoline; Kliochinol; Kliokinol; Kliokinoli; Kliokvinolis; PBT-1; Quiniodochlor. 5-Chloro-7-iodoquinolin-8-ol.

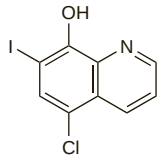
Клиохинол

C_9H_5ClINO = 305.5.

CAS — 130-26-7.

ATC — D08AH30; D09AA10; G01AC02; P01AA02; S02AA05.

ATC Vet — QD08AH30; QD09AA10; QG01AC02; QS02AA05.



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

Ph. Eur. 6.2 (Clioquinol). An almost white, light yellow, brownish-yellow, or yellowish-grey powder. Practically insoluble in water; very slightly soluble or slightly soluble in alcohol; sparingly soluble in dichloromethane. Protect from light.

USP 31 (Clioquinol). A voluminous, spongy, yellowish-white to brownish-yellow powder having a slight characteristic odour. It darkens on exposure to light. Practically insoluble in water; soluble 1 in 3500 of alcohol, 1 in 120 of chloroform, and 1 in 4500 of ether; soluble in hot ethyl acetate and in hot glacial acetic acid. Store in airtight containers. Protect from light.

Adverse Effects and Precautions

Clioquinol may rarely cause iodism in sensitive patients. Local application of clioquinol in ointments or creams may occasionally cause severe irritation or hypersensitivity and there may be cross-sensitivity with other halogenated hydroxyquinolines.

Clioquinol stains clothing and linen yellow on contact and may stain the skin and discolour fair hair.

Clioquinol given by mouth has been associated with severe neurotoxicity. In Japan, the epidemic development of subacute myelo-opticoneuropathy (SMON) in the 1960s was associated with the ingestion of normal or high doses of clioquinol for prolonged periods, and the sale of clioquinol and related hydroxyquinolines was subsequently banned there. Symptoms of SMON are principally those of peripheral neuropathy, including optic atrophy, and myelopathy. Abdominal pain and diarrhoea often precede neurological symptoms, such as paraesthesias in the legs progressing to paraplegia in some patients, and loss of visual acuity sometimes leading to blindness. A characteristic green pigment, a chelate of clioquinol with iron, is often seen on the tongue and in the urine and faeces. Cerebral disturbances, including confusion and retrograde amnesia, have also been reported. Although many patients improved when clioquinol was withdrawn, others had residual disablement.

It was suggested that the Japanese epidemic might be due to genetic susceptibility, but a few similar cases of SMON associated with clioquinol or related hydroxyquinoline derivatives, such as broxyquinoline or diiodohydroxyquinoline have been reported from other countries. Oral preparations of clioquinol have now been banned in most countries.

Hypersensitivity. Clioquinol is classified as a contact allergen which can commonly cause sensitisation, especially when applied to eczematous skin; chlorquinaldol can also cause sensitisation, although less frequently.[1] It is important to include clioquinol and chlorquinaldol in routine patch testing since the clinical reaction may be relatively mild and sensitivity easily missed, particularly in the presence of a corticosteroid which suppresses or attenuates the reaction.

1. Anonymous. Skin sensitisers in topical corticosteroids. *Drug Ther Bull* 1986; **24:** 57–9.

Topical application. Absorption of clioquinol through the skin has been noted on topical application.[1,2] The Committee on Drugs of the American Academy of Pediatrics[3] considered that there was a potential risk of toxicity to infants and children from clioquinol and diiodohydroxyquinoline applied topically. Since alternative effective preparations are available for dermatitis, the Committee recommended that products containing either of these compounds should not be used.

1. Fischer T, Hartvig P. Skin absorption of 8-hydroxyquinolines. *Lancet* 1977; **i:** 603.
2. Stohs SJ, *et al.* Percutaneous absorption of iodochlorhydroxyquin in humans. *J Invest Dermatol* 1984; **82:** 195–8.
3. Kauffman RE, *et al.* Clioquinol (iodochlorhydroxyquin, Vioform) and iodoquinol (diiodohydroxyquin): blindness and neuropathy. *Pediatrics* 1990; **86:** 797–8.

Uses and Administration

Clioquinol is a halogenated hydroxyquinoline with antibacterial and antifungal activity and is used in creams and ointments, usually containing 3%, in the treatment of skin infections. It is applied with a corticosteroid in inflammatory skin conditions complicated by bacterial or fungal infections. It is also used in ear drops for otitis externa. The treatment of bacterial and of fungal skin infections is described on p.194 and p.521 respectively.

For a discussion of the risks from topical application of clioquinol, see Adverse Effects and Precautions, above.

Clioquinol was formerly given by mouth in the treatment of intestinal amoebiasis. It was also formerly used for the prophylaxis and treatment of traveller's diarrhoea and similar infections but was of doubtful value. Oral preparations have now been withdrawn because of neurotoxicity (see Adverse Effects and Precautions, above). However, clioquinol by mouth has been investigated for its action as a chelator of copper and zinc in the treatment of Alzheimer's disease (see below).

Alzheimer's disease. A systematic review[1] to evaluate the efficacy of metal protein attenuating compounds, such as clioquinol, for the treatment of cognitive impairment due to Alzheimer's disease, evaluated only one small randomised controlled study comparing clioquinol and placebo; no significant differences were found. Further studies with clioquinol have now been stopped, but studies are on-going with a successor compound, PBT2.

1. Sampson E, *et al.* Metal protein attenuating compounds for the treatment of Alzheimer's disease. Available in The Cochrane Database of Systematic Reviews; Issue 1. Chichester: John Wiley; 2008 (accessed 14/05/08).

Preparations

BP 2008: Betamethasone and Clioquinol Cream; Betamethasone and Clioquinol Ointment; Hydrocortisone and Clioquinol Cream; Hydrocortisone and Clioquinol Ointment;
USP 31: Clioquinol and Hydrocortisone Cream; Clioquinol and Hydrocortisone Ointment; Clioquinol Cream; Clioquinol Ointment; Compound Clioquinol Topical Powder.

Proprietary Preparations (details are given in Part 3)

Ger.: Linola-sept; ***Hung.:*** Linola-sept†; ***India:*** Dermoquinol; Entero-Quinol; Entrozyme Plain; ***Mex.:*** Bagton; Bionder-C; Cortifung-C; Lasalar-Y Simple; Luzolona Simple; Nolil; Quindoleina†; Vioformo; ***Port.:*** Quinodermil†.

Multi-ingredient: ***Arg.:*** Betnovate-C; Locorten Vioformo†; Quadriderm†; ***Austral.:*** Hydroform; Locacorten Vioform; Quinaband†; ***Austria:*** Betnovate-C; Locacorten Vioform; ***Belg.:*** Betnelan-VC†; Locacortene Vioforme†; ***Braz.:*** Betnovate-Q; Cremederme; Dreniformio; Hidrocorte; Locorten Vioformio; Permut; Policderms; Predmicin; Quadriderm; Quadrikin; Quadrilon; Quadriplus; Qualiderm; Tetraderm; Vioformio-Hidrocortisona; ***Canad.:*** Locacorten Vioform; Phenoris; Vioform-Hydrocortisone; ***Cz.:*** Lorinden C†; Prednisolon J†; ***Denm.:*** Betnovat med Chinoform; Celeston med Chinoform; Locacorten Vioform; Synalar med Chinoform; ***Fin.:*** Bemetson-K; Betnovat-C; Celestoderm cum Chinoform†; Locacorten Vioform; ***Fr.:*** Diprosept†; Locacortene Vioforme; ***Ger.:*** Locacorten Vioform; ***Gr.:*** Betnovate-C; Myco-Synalar; ***Hong Kong:*** Betnovate-C†; Clobeta-G; Dermafacte; Quadriderm; ***Hung.:*** Lorinden C; Prednisolon J; ***India:*** Beclate-C; Betnederm C; Betnovate-C; Cortoquinol; Fourderm; Millicorten-Vioform; Polyderm†; Quiss; ***Indon.:*** Benoson V; Krimbeson; Viohydrocort; Visancort; ***Irl.:*** Betnovate-C; Synalar C†; Vioform-Hydrocortisone; ***Israel:*** Betnovate-C; Topicorten V; ***Ital.:*** Diproform; Locorten; Locorten Vioformio; ***Mex.:*** Bentix; Cetoquina Y; Clio-Betnovate; Cliooderm-H; Contefur†; Cortifung-Y; Cortilona Compuesta; Dealan; Diprosone Y; Ditayod; Farmacorti YC; Fluccinol C†; Flunal†; Lasalar-Y; Luzolona Y; Sebryl; Sebryl Plus; Sebstopp; Solfurol; Sultroquin†; Suyodil; Synalar C; Taliviform†; Topsyn-Y; Trilor†; Ultracortin; Vioformo-Cort; Yderm; Yodozona; ***Neth.:*** Locacorten Vioform; ***Norw.:*** Betnovat med Chinoform; Synalar med Chinoform; ***NZ:*** Betnovate-C; Locorten Vioform; ***Philipp.:*** Aplosyn C; Betnovate-C; Dermalin; Diproform; Quadriderm; Quadrotopic; ***Pol.:*** Betnovate-C; Lorinden C; Viosept; ***Port.:*** Betnovate-C; Dexaval V; Locorten Vioformio†; Quinodermil-AS; ***Rus.:*** Dermosolon (Дермозолон); Lorinden C (Лоринден С); ***S.Afr.:*** Betnovate-C; Cortoderm-C; Locacorten Vioform; Quadriderm; Synalar C; ***Singapore:*** Dermanol-C; Hydroderm-C; Quadriderm†; ***Spain:*** Cuatroderm; Menaderm Clio; Menaderm Otologico; Synobel†; ***Swed.:*** Betnovat med Chinoform; Celeston valerat med chinoform; Locacorten Vioform; ***Switz.:*** Betnovate-C; Quadriderm; ***Thai.:*** Banocin; Beta-C; Betnovate-C; Betosone-CE; Chlorotracin; Endothalyl; Genquin; ***Turk.:*** Betnovate-C; Locacortene Vioform; Prednol-A; ***UK:*** Betnovate-C; Locorten Vioform; Quinaband†; Synalar C; Vioform-Hydrocortisone; ***USA:*** I + I-F; Corque; Hysone; ***Venez.:*** Dermosupril C; Diproformo; Locorten Vioformo; Neo-Synalar con Yodoclorohidroxiquina†; Propioformo†; Quadriderm; Tridetarmon; Vio Celestoderm†.

Clofazimine *(BAN, USAN, rINN)*

B-663; Clofazimina; Clofaziminum; G-30320; Klofatsimiini; Klofatzimin; Klofazimin; Klofaziminas; NSC-141046. 3-(4-Chloroanilino)-10-(4-chlorophenyl)-2,10-dihydro-2-phenazin-2-ylideneisopropylamine.

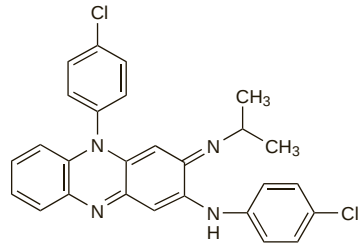
Клофазимин

$C_{27}H_{22}Cl_2N_4$ = 473.4.

CAS — 2030-63-9.

ATC — J04BA01.

ATC Vet — QJ04BA01.



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

Ph. Eur. 6.2 (Clofazimine). A fine reddish-brown powder. It exhibits polymorphism. Practically insoluble in water; very slightly soluble in alcohol; soluble in dichloromethane.

USP 31 (Clofazimine). Dark red crystals. Practically insoluble in water; sparingly soluble in alcohol, in acetone, and in ethyl acetate; soluble in chloroform and in benzene. Store in airtight containers. Protect from light.

Adverse Effects

Adverse effects to clofazimine are dose related, the most common being red to brown discoloration of the skin especially on areas exposed to sunlight; leprotic lesions may become mauve to black. These changes are more noticeable in light-skinned people and may limit its acceptance. The conjunctiva and cornea may also show some signs of red to brown pigmentation. The generalised discoloration may take months to years to disappear after stopping therapy. Discoloration of hair, tears, sweat, sputum, breast milk, urine, and faeces may occur, as may nail discoloration with high doses of 300 mg daily. Severe depression related to skin discoloration has been reported rarely.

Gastrointestinal effects are uncommon for doses of clofazimine less than 100 mg daily and usually are not severe. Symptoms of nausea, vomiting, and abdominal pain experienced shortly after the start of treatment may be due to direct irritation of the gastrointestinal tract and such symptoms usually disappear on dose reduction. Use of doses of 300 mg daily or more for several months sometimes produces abdominal pain, diarrhoea, weight loss, gastrointestinal bleeding, and in severe cases the small bowel may become oedematous and symptoms of bowel obstruction may develop. This may be due to deposition of crystals of clofazimine in the wall of the small bowel and in the mesenteric lymph nodes. Crystal deposition may also occur in other organs including the liver and spleen and there have been rare reports of splenic infarction. Symptoms usually regress on withdrawal of treatment although fatalities have been reported.

Clofazimine may produce a dryness of the skin and ichthyosis as well as decreased sweat production and rashes. Pruritus, acneiform eruptions, and photosensitivity reactions have also been reported.

Eye irritation and decreased tear production may occur.

Headache, drowsiness, dizziness, taste disorders, and elevation of blood glucose levels have been reported rarely.

Incidence of adverse effects. The incidence of adverse effects was reviewed in 65 patients[1] who were receiving, or had received, clofazimine in weekly doses of either 700 mg or less as antimycobacterial therapy, or more than 700 mg as anti-inflammatory therapy. Length of treatment ranged from 1 to 83 months. Adverse effects on the skin included discoloration (20% of patients), pigmentation (64.6%), dry skin (35.4%), and pruritus (5%). Ocular adverse effects were conjunctival pigmentation (49.2%), subjective dimness of vision (12.3%), and dry eyes, burning, and other ocular irritation (24.6%). Gastrointestinal adverse effects included abdominal pain (33.8%), nausea (9.2%), diarrhoea (9.2%), and weight loss, vomiting, or loss of appetite (13.8%). The different dose regimens for antimycobacterial therapy or anti-inflammatory effect had similar incidences of adverse effects. Skin pigmentation in 8 patients disappeared on average 8.5 months after stopping therapy with clofazimine, the maximum time required being one year. Adverse effects of clofazimine were considered to be well tolerated.

In another report covering 540 patients receiving clofazimine 100 mg on alternate days or 300 mg daily, the most common adverse effect was skin pigmentation, which occurred in 77.8% of

the patients. Ichthyotic changes were reported in 66.7% and pruritus in 20.2%. Gastrointestinal symptoms occurred in 20 patients (about 4%); other effects such as discoloration of sweat, urine, and tears were minor.[2]

1. Moore VJ. A review of side-effects experienced by patients taking clofazimine. *Lepr Rev* 1983; **54:** 327–35.
2. Kumar B, *et al*. More about clofazimine—3 years experience and review of the literature. *Indian J Lepr* 1987; **59:** 63–74.

Effects on the eyes. Accumulation of clofazimine crystals in the eye can lead to pigmentation of the cornea and conjunctiva. Degeneration of the retinal pigment epithelium has also been attributed to clofazimine therapy in a patient.[1] Slight repigmentation was observed after withdrawal of clofazimine.

1. Forster DJ, *et al*. Bull's eye retinopathy and clofazimine. *Ann Intern Med* 1992; **116:** 876–7.

Effects on the gastrointestinal tract. Gastrointestinal effects are uncommon at doses of clofazimine less than 100 mg daily. However, there have been some reports of severe gastrointestinal adverse events, including fatalities, in patients taking clofazimine.[1-4] An 11-year-old child given clofazimine (150 mg daily) for graft-versus-host disease developed severe enteropathy 2 years after starting treatment.[1] Clofazimine was stopped and symptoms resolved after 5 weeks. Enteropathy has also been reported in a 20-year-old patient who had taken 200 mg of clofazimine daily for 4 years.[2] Clofazimine was stopped but his symptoms did not resolve; he developed peripheral oedema and hypoalbuminaemia and died 2 years later due to cerebral thrombosis. In another report,[4] partial intestinal obstruction developed in a patient after 12 months of treatment with clofazimine 100 mg daily for the treatment of multidrug-resistant tuberculosis. The patient recovered 3 weeks after stopping clofazimine. Splenic infarction has been reported after 11 months treatment with high-dose clofazimine for the management of pyoderma gangrenosum.[5] Chronic abdominal pain due to crystal-storing histiocytosis of mesenteric lymph nodes is well recognised, and may mimic the symptoms of gastrointestinal lymphoma or myeloma.[3]

1. Parizhskaya M, *et al*. Clofazimine enteropathy in a pediatric bone marrow transplant recipient. *J Pediatr* 2001; **138:** 574–6.
2. Hameed A, *et al*. A case of clofazimine enteropathy. *Int J Clin Pract* 1998; **52:** 439–40.
3. Sukpanichnant S, *et al*. Clofazimine-induced crystal-storing histiocytosis producing chronic abdominal pain in a leprosy patient. *Am J Surg Pathol* 2000; **24:** 129–35.
4. Üsküdar O, *et al*. Partial intestinal obstruction due to clofazimine in a patient with multidrug-resistant tuberculosis. *Int J Tuberc Lung Dis* 2005; **9:** 703–4.
5. McDougall AC, *et al*. Splenic infarction and tissue accumulation of crystals associated with the use of clofazimine (Lamprene; B663) in the treatment of pyoderma gangrenosum. *Br J Dermatol* 1980; **102:** 227–30.

Effects on the heart. Ventricular tachycardia, thought to be probably torsade de pointes, was reported to be associated with clofazimine.[1]

1. Choudhri SH, *et al*. Clofazimine induced cardiotoxicity—a case report. *Lepr Rev* 1995; **66:** 63–8.

Precautions

Clofazimine should be used with caution in patients with gastrointestinal symptoms such as abdominal pain and diarrhoea. If gastrointestinal symptoms develop during treatment, the dose should be reduced and, if necessary, the interval between doses increased, or the drug should be stopped. Daily doses of more than 100 mg should not be used for more than 3 months because of dose-related adverse effects on the gastrointestinal tract; patients receiving doses greater than 100 mg daily should be under medical supervision.

Patients should be warned that clofazimine may cause a reddish-brown discoloration of breast milk, hair, skin, conjunctiva, tears, sputum, sweat, urine, and faeces. Nails may be discoloured at higher doses.

As clofazimine crosses the placental barrier, neonates of women receiving clofazimine may have skin discoloration at birth.

Breast feeding. The American Academy of Pediatrics[1] considers that the use of clofazimine by mothers during breast feeding may be of concern, since there is the possibility of transfer of a high percentage of the maternal dose and a possible increase in skin pigmentation in the infant. A small study in 8 women calculated that up to 30% of a maternal dose may be ingested by a breast-fed infant.[2]

1. American Academy of Pediatrics. The transfer of drugs and other chemicals into human milk. *Pediatrics* 2001; **108:** 776–89. Correction. *ibid.*; 1029. Also available at: http://aappolicy.aappublications.org/cgi/content/full/pediatrics%3b108/3/776 (accessed 03/10/07)
2. Venkatesan K, *et al*. Excretion of clofazimine in human milk in leprosy patients. *Lepr Rev* 1997; **68:** 242–6.

Pregnancy. Two successful pregnancies in women who received clofazimine throughout pregnancy have been reported[1] but a literature review revealed 3 neonatal deaths in 13 pregnancies, although the deaths could not be directly attributed to clofazimine. However, WHO[2] states that its recommended multiple drug therapy regimens for leprosy, which may include clofazimine, are safe during pregnancy.

1. Farb H, *et al*. Clofazimine in pregnancy complicated by leprosy. *Obstet Gynecol* 1982; **59:** 122–3.
2. WHO. *Guide to eliminate leprosy as a public health problem*. 1st ed. Geneva: WHO, 2000. Also available at: http://www.who.int/lep/resources/Guide_Int_E.pdf (accessed 28/07/08)

Interactions

Some preliminary data have suggested that the anti-inflammatory action of clofazimine in Type 2 lepra reactions may be reduced by dapsone, although US licensed product information (*Lamprene; Novartis, USA*) states that these findings have not been confirmed; the antimycobacterial effect was not affected.

Elevated plasma and urine concentrations of clofazimine have been detected in patients receiving high doses of clofazimine with isoniazid, although skin concentrations were found to be lower.

For a report of the effect of clofazimine on rifampicin absorption, see p.327.

Antimicrobial Action

Clofazimine is bacteriostatic and weakly bactericidal against *Mycobacterium leprae*. Tissue antimicrobial activity in humans cannot be found until after about 50 days of therapy. Clofazimine is active *in vitro* against various other species of *Mycobacterium*. Resistance has been reported rarely and no cross-resistance occurs with rifampicin or dapsone.

Pharmacokinetics

Clofazimine is absorbed from the gastrointestinal tract in amounts varying from 45 to 70%. Absorption is greatest when clofazimine is given in microcrystalline formulations and when it is taken immediately after food. The time to steady-state plasma concentrations has not been determined but exceeds 42 days.

Average plasma concentrations in leprosy patients receiving 100 or 300 mg daily are reported as 0.7 micrograms/mL and 1.0 microgram/mL, respectively.

Because of its lipophilic nature, clofazimine is mainly distributed to fatty tissue and reticuloendothelial cells, including macrophages. Clofazimine is distributed to most organs and tissues and into breast milk; it crosses the placenta but not the blood-brain barrier.

The tissue half-life after a single dose has been reported to be about 10 days; that after multiple oral doses has been variously estimated to be between 25 and 90 days. Clofazimine accumulates in the body and is largely excreted unchanged in the faeces, both as unabsorbed drug and via biliary excretion. About 1% of the dose is excreted in 24 hours in the urine as unchanged clofazimine and metabolites. A small amount of clofazimine is also excreted through sebaceous and sweat glands, and in sputum.

◊ References.

1. Holdiness MR. Clinical pharmacokinetics of clofazimine: a review. *Clin Pharmacokinet* 1989; **16:** 74–85.

Uses and Administration

Clofazimine is an antimycobacterial and is used as part of multidrug regimens for the treatment of multibacillary leprosy (p.176). It has anti-inflammatory properties and has been given in chronic Type 2 lepra reactions (erythema nodosum leprosum) and in a variety of skin disorders.

Clofazimine is given orally with, or immediately after, food or milk for optimum absorption.

For multibacillary leprosy the most common regimen is that recommended by WHO, in which rifampicin 600 mg and clofazimine 300 mg are both given *once a month*, together with *daily* doses of clofazimine 50 mg and dapsone 100 mg; this treatment continues for 12 months.

For details of doses in children, see below.

Clofazimine 50 mg daily is given with ofloxacin and minocycline in patients unable to take rifampicin. Clofazimine is not usually given in paucibacillary leprosy. However, it may be used with rifampicin instead of dapsone when the latter has caused severe toxicity.

Clofazimine has been used in the treatment of chronic Type 2 lepra reactions, although the effect may not be evident for 4 to 6 weeks. A dose of up to 300 mg daily has been suggested but it should not be given for longer than 3 months. Corticosteroids may be given with clofazimine, and standard antileprosy treatment should be continued. Clofazimine is not used in Type 1 lepra reactions.

Administration in children. For the treatment of multibacillary leprosy in children WHO recommends that children aged 10 to 14 years may be given oral clofazimine 150 mg plus rifampicin 450 mg and dapsone 50 mg *once a month*, together with dapsone 50 mg *daily* and clofazimine 50 mg on *alternate days*. For children less than 10 years of age the dose should be adjusted according to body weight. As for adults, treatment is given for 12 months.

Preparations

BP 2008: Clofazimine Capsules;
USP 31: Clofazimine Capsules.

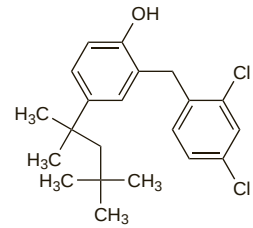
Proprietary Preparations (details are given in Part 3)
Austral.: Lamprene; ***Braz.:*** Neozimina; ***Cz.:*** Lamprene; ***Fr.:*** Lamprene; ***Gr.:*** Lamprene; ***Hong Kong:*** Lamprene†; ***India:*** Clofozine; Hansepran; ***Jpn:*** Lampren; ***Malaysia:*** Lamprene†; ***Neth.:*** Lampren; ***NZ:*** Lamprene; ***S.Afr.:*** Lamprene†; ***Spain:*** Lampren; ***Switz.:*** Lamprene†; ***Thai.:*** Lamcoin; ***UK:*** Lamprene†; ***USA:*** Lamprene.

Clofoctol *(rINN)*

Clofoctolum. 2-(2,4-Dichlorobenzyl)-4-(1,1,3,3-tetramethylbutyl)phenol.

Клофоктол

$C_{21}H_{26}Cl_2O$ = 365.3.
CAS — 37693-01-9.
ATC — J01XX03.
ATC Vet — QJ01XX03.



Profile

Clofoctol has bacteriostatic or bactericidal activity against Gram-positive organisms such as staphylococci and streptococci. It is given in doses of 750 mg twice daily rectally in the treatment of respiratory-tract infections.

Preparations

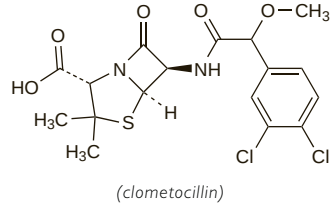
Proprietary Preparations (details are given in Part 3)
Fr.: Octofene†; ***Ital.:*** Gramplus; Octofene†; ***Port.:*** Octofene†.

Clometocillin Potassium *(rINNM)*

Clometocilina potásica; Clométocilline Potassique; 3,4-Dichloro-α-methoxybenzylpenicillin Potassium; Kalii Clometocillinum; Penicillin 356 (clometocillin). Potassium (6*R*)-6-[2-(3,4-dichlorophenyl)-2-methoxyacetamido]penicillanate.

Калия Клометоциллин

$C_{17}H_{17}Cl_2KN_2O_5S$ = 471.4.
CAS — 1926-49-4 (clometocillin); 15433-28-0 (clometocillin potassium).
ATC — J01CE07.
ATC Vet — QJ01CE07.



(clometocillin)

Profile

Clometocillin is a penicillin given orally as the potassium salt in the treatment of susceptible bacterial infections. Doses are ex-