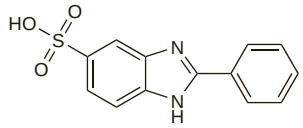
Ensulizole *(USAN, rINN)*

Ensulizol; Ensulizolum; Phenylbenzimidazole Sulphonic Acid. 2-Phenyl-1*H*-benzimidazole-5-sulphonic acid.

Энсулизол

$C_{13}H_{10}N_2O_3S$ = 274.3.

CAS — 27503-81-7.



NOTE. Eusolex 232 and Neo-Heliopan Hydro are trade names that have been used for ensulizole.

Pharmacopoeias. In *US.*

USP 31 (Ensulizole). A white to ivory-coloured, odourless powder. Practically insoluble in water and in oily solvents; soluble in alcohol; its salts are freely soluble in water. Store in airtight containers at a temperature of 8° to 15°.

Profile

Ensulizole is used topically as a sunscreen (p.1576). It is effective against UVB light (for definitions, see p.1580).

Preparations

Proprietary Preparations some preparations are listed in Part 3.

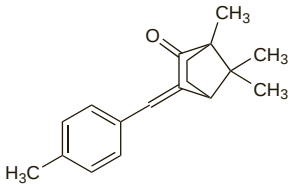
Enzacamene *(USAN, rINN)*

Enzacamène; Enzacameno; Enzacamenum; Methyl Benzylidene Camphor; 3-(4-Methylbenzylidene)bornan-2-one; 3-(4-Methylbenzylidene)camphor. 1,7,7-Trimethyl-3-[(4-methylphenyl)methylene]bicyclo[2.2.1]heptan-2-one.

Энзакамен

$C_{18}H_{22}O$ = 254.4.

CAS — 36861-47-9 (D,L-form); 38102-62-4 (form unspecified).



NOTE. Eusolex 6300, Neo-Heliopan MBC, and Parsol 5000 are trade names that have been used for enzacamene.

Pharmacopoeias. In *US.*

USP 31 (Enzacamene). A white, fine crystalline powder. M.p. between 66° and 68°. Practically insoluble in water; freely soluble in alcohol; very soluble in chloroform. Store in airtight containers.

Profile

Enzacamene is a camphor derivative used as a sunscreen (p.1576). It is effective against UVB light (for definitions, see p.1580).

Preparations

Proprietary Preparations numerous preparations are listed in Part 3.

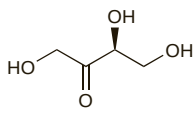
Erythrulose

DL-Glycero-tetrulose. 1,3,4-Trihydroxy-2-butanone.

Эритрулаза

$C_4H_8O_4$ = 120.1.

CAS — 40031-31-0 (DL-erythrulose); 496-55-9 (D-erythrulose); 533-50-6 (L-erythrulose).



Profile

Application to the skin of preparations containing L-erythrulose slowly produces a brown coloration similar to that caused by exposure to the sun, probably due to a reaction with the amino acids of the skin. It is often used with dihydroxyacetone (p.1594) in artificial suntan preparations. Recommended concentrations are up to 5% when used alone or up to 4% with dihydroxyacetone.

Preparations

Proprietary Preparations (details are given in Part 3)

Multi-ingredient: Braz.: Sunmax Autobronzeador; ***UK:*** ViTicolor.

Etretinate *(BAN, USAN, rINN)*

Etretinaatti; Etretinat; Étrétinate; Etretinato; Etretinatum; Ro-10-9359. Ethyl 3-methoxy-15-apo-ϕ-caroten-15-oate; Ethyl (*all-trans*)-9-(4-methoxy-2,3,6-trimethylphenyl)-3,7-dimethylnona-2,4,6,8-tetra-enoate.

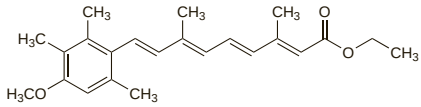
Этретинат

$C_{23}H_{30}O_3$ = 354.5.

CAS — 54350-48-0.

ATC — D05BB01.

ATC Vet — QD05BB01.



Adverse Effects and Precautions

As for Isotretinoin, p.1599.

Donation of blood should be avoided for at least 2 years after cessation of treatment. The period of time during which pregnancy must be avoided after stopping treatment has not been determined; detectable plasma-etretinate concentrations have been reported nearly 3 years after the last dose.

◊ In addition to the references cited below, further references to the adverse effects of etretinate can be found in Isotretinoin, p.1599, under Effects on the Blood, Cardiovascular System, Eyes, Liver, Musculoskeletal System, Serum Lipids, and the Skin, as well as under Vasculitic Syndromes.

Carcinogenicity. A report of 2 patients who developed lymphomas while receiving etretinate[1] prompted a report of 3 other malignancies in patients taking etretinate.[2]

1. Woll PJ, *et al.* Lymphoma in patients taking etretinate. *Lancet* 1987; **ii:** 563–4.
2. Harrison PV. Retinoids and malignancy. *Lancet* 1987; **ii:** 801.

Effects on the kidneys. Rare cases of impaired renal function associated with etretinate have been described.[1,2] In one report[1] it was also noted that in manufacturer-sponsored studies the mean serum-creatinine concentration had been raised in patients receiving etretinate.

1. Horber FF, *et al.* Impaired renal function and hypercalcaemia associated with etretinate. *Lancet* 1984; **ii:** 1093.
2. Cribier B, *et al.* Renal impairment probably induced by etretinate. *Dermatology* 1992; **185:** 266–8.

Oedema. A report of generalised oedema after treatment with etretinate.[1] Five other cases had been reported in the literature and rechallenge in 4 patients had provoked a recurrence. Generalised oedema as part of the capillary leak syndrome has been reported with acitretin (p.1586).

1. Allan S, Christmas T. Severe edema associated with etretinate. *J Am Acad Dermatol* 1988; **19:** 140.

Pregnancy. For further information on the teratogenicity of etretinate, see under Acitretin, p.1586.

Interactions

As for Isotretinoin, p.1602.

Anticoagulants. Etretinate has been reported to reduce the therapeutic efficacy of *warfarin* (see p.1430).

Antiepileptics. Etretinate was ineffective and none of its characteristic mucocutaneous adverse effects occurred in a patient with pityriasis rubra pilaris who was already taking *carbamazepine* and *valproate* for epilepsy. However, there was a clinical response after the carbamazepine had been withdrawn, suggesting that it may have reduced the bioavailability or increased the metabolism of etretinate.[1]

1. Mohammed KN. Unresponsiveness to etretinate during anticonvulsant therapy. *Dermatology* 1992; **185:** 79.

Antineoplastics. The risk of developing hepatotoxicity may be increased when etretinate is used with *methotrexate* (see Retinoids, p.748).

Hormonal contraceptives. For discussion of the potential interactions of retinoids with oral hormonal contraceptives, and the effect this might have on contraceptive choice during retinoid treatment, see p.2068.

Pharmacokinetics

The mean bioavailability of etretinate is about 40% after oral doses but there is a large interindividual variation. Absorption can be increased if taken with milk or fatty food. Etretinate undergoes significant first-pass metabolism and plasma concentrations of the active carboxylic acid metabolite, acitretin (p.1586), may be detected before those of the parent drug; acitretin may itself be metabolised to etretinate (see p.1586). Both etretinate and acitretin are extensively bound to plasma protein. Etretinate appears to accumulate in adipose tissue after repeated dosing and has a prolonged elimination half-life of about 120 days; detectable serum concentrations have been observed up to 3 years after stopping therapy. Up to 75% of a dose is excreted in the faeces mainly as unchanged drug. Etretinate is also excreted in the urine as metabolites. Etretinate crosses the placenta. Although it is not known whether etretinate is distributed into breast milk, this would be expected because of its lipophilicity; acitretin, a metabolite of etretinate, has been found in breast milk when it was given to a lactating woman (see p.1586).

◊ References.

1. Lucek RW, Colburn WA. Clinical pharmacokinetics of the retinoids. *Clin Pharmacokinet* 1985; **10:** 38–62.
2. DiGiovanna JJ, *et al.* Etretinate: persistent serum levels after long-term therapy. *Arch Dermatol* 1989; **125:** 246–51.
3. Larsen FG. Pharmacokinetics of etretinate and acitretin with special reference to treatment of psoriasis. *Acta Derm Venereol (Stockh)* 1994; **190** (suppl): 1–33.
4. Wiegand UW, Chou RC. Pharmacokinetics of acitretin and etretinate. *J Am Acad Dermatol* 1998; **39** (suppl): S25–S33.

Uses and Administration

Etretinate is a retinoid and is a derivative of tretinoin (p.1618). It has been given orally for the treatment of severe, extensive psoriasis that has not responded to other treatment, especially generalised and palmo-plantar pustular psoriasis. It has also been used in severe congenital ichthyosis, severe Darier's disease (keratosis follicularis) as well as other disorders of keratinisation, and oral lichen planus. Acitretin (p.1586) is now preferred to etretinate.

Therapy is generally started at doses of 0.75 to 1 mg/kg daily in divided oral doses. A maximum dose of 1.5 mg/kg daily should not be exceeded (some licensed product information has suggested a maximum of 75 mg daily). Erythrodermic psoriasis may respond to lower initial doses of 250 micrograms/kg daily, increased at weekly intervals by 250 micrograms/kg daily until optimal response occurs. After the initial response, generally after 8 to 16 weeks of therapy, maintenance doses of 500 to 750 micrograms/kg daily have been given. Therapy should be stopped once lesions have sufficiently resolved.

◊ References.

1. Magis NLJ, *et al.* The treatment of psoriasis with etretinate and acitretin: a follow up of actual use. *Eur J Dermatol* 2000; **10:** 517–21.
2. Katugampola RP, Finlay AY. Oral retinoid therapy for disorders of keratinization: single-centre retrospective 25 years' experience on 23 patients. *Br J Dermatol* 2006; **154:** 267–76.

Preparations

Proprietary Preparations (details are given in Part 3)

Jpn: Tigason.

Fumaric Acid

Acidum Fumaricum; Allomalenic Acid; Boletic Acid; E297; Fumárico, ácido; Kwas fumarowy. *trans*-Butenedioic acid.

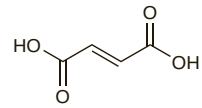
Фумаровая Кислота

$C_2H_2(CO_2H)_2$ = 116.1.

CAS — 110-17-8 (fumaric acid); 624-49-7 (dimethyl fumarate).

ATC — D05AX01.

ATC Vet — QD05AX01.



Pharmacopoeias. In *Pol.* Also in *USNF.*

USNF 26 (Fumaric Acid). White, odourless granules or crystalline powder. Slightly soluble in water and in ether; soluble in alcohol; very slightly soluble in chloroform.

Profile

Fumaric acid and some of its derivatives have been used in the treatment of psoriasis and other skin disorders.

Fumaric acid is also used as an acidifier and flavouring agent in foods.

Skin disorders. Fumaric acid, its sodium salts, and derivatives such as dimethyl fumarate, monoethyl fumarate (ethyl hydrogen fumarate), and octil hydrogen fumarate have been used, both topically and systemically, in the treatment of psoriasis (p.1583) and other skin disorders. Dimethyl fumarate appears to be the most active compound given orally but combination with various salts of monoethyl fumarate has been claimed to improve efficacy.[1-6] However, there have been reports of acute renal failure associated with treatment and the German Federal Office of Health has expressed the opinion that the available evidence did not establish the value of fumaric acid derivatives in psoriasis or other skin disorders.[7] A subsequent retrospective analysis of 41 patients who received fumaric acid esters orally, for between 1 and 14 years, suggested that these drugs might be effective,[8] and a later review suggested that they may be of value in refractory psoriasis.[9] Reported adverse effects in the earlier analysis[8] were generally mild, with only 1 case of elevated serum creatinine; however, lymphocytopenia was noted in 76% of patients and treatment consequently stopped in 4 patients. Other adverse effects with oral therapy have included disturbances of liver func-

tion,[3,10] gastrointestinal effects,[2-4,10,11] and flushing.[2-4,10,11] There has been a report of exanthema in a patient receiving dimethyl fumarate for lichen planus.[12]

1. van Loenen AC, *et al.* Fumaarzuurtherapie: van fictie tot werkelijkheid? *Pharm Weekbl* 1989; **124:** 894–900.
2. Kolbach DN, Nieboer C. Fumaric acid therapy in psoriasis: a long-term retrospective study on the effect of fumaric acid combination (FAC-EC) therapy and dimethyl-fumaric acid ester (DMFAE) monotherapy. *Br J Dermatol* 1990; **123:** 534–5.
3. Nugteren-Huying WM, *et al.* Fumaric acid therapy for psoriasis: a randomized, double-blind, placebo-controlled study. *J Am Acad Dermatol* 1990; **22:** 311–12.
4. Altmeyer PJ, *et al.* Antipsoriatic effect of fumaric acid derivatives: results of a multicenter double-blind study in 100 patients. *J Am Acad Dermatol* 1994; **30:** 977–81.
5. Mrowietz U, *et al.* Treatment of severe psoriasis with fumaric acid esters: scientific background and guidelines for therapeutic use. *Br J Dermatol* 1999; **141:** 424–9.
6. Ständer H, *et al.* Efficacy of fumaric acid ester monotherapy in psoriasis pustulosa palmoplantaris. *Br J Dermatol* 2003; **149:** 220–2.
7. Anonymous. Fumaric acid derivatives and nephrotoxicity. *WHO Drug Inf* 1990; **4:** 28.
8. Hoefnagel JJ, *et al.* Long-term safety aspects of systemic therapy with fumaric acid esters in severe psoriasis. *Br J Dermatol* 2003; **149:** 363–9.
9. Harries MJ, *et al.* Fumaric acid esters for severe psoriasis: a retrospective review of 58 cases. *Br J Dermatol* 2005; **153:** 549–51.
10. Nieboer C, *et al.* Systemic therapy with fumaric acid derivates: new possibilities in the treatment of psoriasis. *J Am Acad Dermatol* 1989; **20:** 601–8.
11. Mrowietz U, *et al.* Treatment of psoriasis with fumaric acid esters: results of a prospective multicentre study. *Br J Dermatol* 1998; **138:** 456–60.
12. Guenther CH, *et al.* Macular exanthema due to fumaric acid esters. *Ann Pharmacother* 2003; **37:** 234–6.

Preparations

Proprietary Preparations (details are given in Part 3)
Arg.: Ingepsor; ***Ger.:*** Psoriasis-Solution†; Psoriasis-Tabletten†.

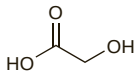
Multi-ingredient: ***Arg.:*** Noqueral†; ***Austral.:*** Pro-PS†; ***Ger.:*** Fumaderm; Psoriasis-Bad†; Psoriasis-Salbe S†.

Glycolic Acid

Glicólico, ácido; Hydroxyacetic Acid. Hydroxyethanoic acid.

Гидроксиуксусная Кислота; Гликолевая Кислота

$C_2H_4O_3$ = 76.05.
CAS — 79-14-1.



Profile
Glycolic acid is an alpha hydroxy organic acid that has been used in topical preparations for hyperpigmentation (see Pigmentation Disorders, p.1582) and photodamaged skin (see Photoageing, p.1581).

Preparations

Proprietary Preparations (details are given in Part 3)
Arg.: Alfabase 8; Geloforte†; Glicoisdin; Gligel; Lactrime†; Lipomax†; Loxidil; Vansame G; ***Canad.:*** Reversa; ***Chile:*** Alastik†; Neosolets; Teen Derm†; ***Hong Kong:*** Glyderm; ***Indon.:*** Exfoliac; Glycare; ***Ital.:*** Neostrata; Revitalizing†; ***Malaysia:*** Glyderm†; ***Mex.:*** Glicoderm; Glicolic; Nova Derm; ***Philipp.:*** Teranex; ***Singapore:*** Glyderm; Sunsense Anti-Ageing; ***Venez.:*** Glyco-A†; Teen Derm†.

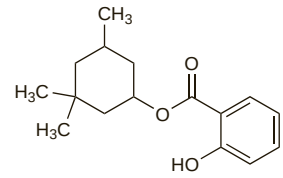
Multi-ingredient: ***Arg.:*** Cellskinlab C + AHA; Controlacne; Diacneal; Effalpha†; Hidroskin; Hydragenic†; Keracnyl; Melacler†; Negacne; Neoquin; Neoquin Forte; Neostrata; Neostrata Gel Despigmentante; Purasoft; Revital; Vansame GS; Vansame Plus; ***Austral.:*** Neostrata; ***Braz.:*** Glyquin; ***Canad.:*** Biobase-G; Dilusol AHA†; Glyquin XM; Neostrata; NeoStrata Blemish Spot Gel; NeoStrata Daytime; NeoStrata HQ; Reversa UV; Viquin Forte†; ***Chile:*** Alastik†; D 4†; Diacneal; Neostrata; Neutrogena Healthy Skin; Neutrogena Limpiadora; Primacy C+AHA†; Ureadin Forte; ***Fr.:*** Alpha 5 DS†; Aniospray 29; Body Peel; Cleanance K; Correcteur Anti-Taches; Cosmodex Uniwhite†; Day Peel; Hyfac soin keratolytique†; Item Alphakeptol; Kelual DS; Keracnyl; Keracnyl eau nettoyante; Keracnyl stop bouton; Kertyol-S; Night Peel; Photakne†; Seborheane; ***Hong Kong:*** Glyquin; ***Indon.:*** Exfoliac; Interquin Plus; ***Ital.:*** Acnesan†; Biophase Shampoo; Lightening; Neoceuticals Spot Treatment; Phytic Acid; Same-Seb Beta; Sebacnol†; ***Mex.:*** Nova Derm; ***Port.:*** Bioclin Sebo Care; Ureadin; Ureadin Forte; ***Singapore:*** Glyquin; Glyquin XM; Percutalfa; ***USA:*** Glyquin XM; ***Venez.:*** Diacneal; Photoderm AKN.

Homosalate *(USAN, rINN)*

Homomenthyl Salicylate; Homosalato; Homosalatum. 3,3,5-Trimethylcyclohexyl salicylate.

Гомосалат

$C_{16}H_{22}O_3$ = 262.3.
CAS — 118-56-9.



NOTE. Eusolex HMS and Neo-Heliopan HMS are trade names that have been used for homosalate.

Pharmacopoeias. In *US.*
USP 31 (Homosalate). Store in airtight containers.

Profile
Homosalate, a substituted salicylate, is a sunscreen (p.1576) with actions similar to those of octisalate (p.1608). It is effective against UVB light (for definitions, see p.1580).

Preparations

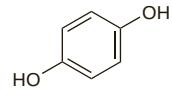
Proprietary Preparations numerous preparations are listed in Part 3.

Hydroquinone

Hidrokinon; Hidroquinona; Hydrochinon; Hydrochinonum; Quinol. 1,4-Benzenediol.

Гидрохинон

$C_6H_6O_2$ = 110.1.
CAS — 123-31-9.
ATC — D11AX11.
ATC Vet — QD11AX11.



NOTE. Do not confuse with Hydroquinine (p.2322).

Pharmacopoeias. In *US.*
USP 31 (Hydroquinone). Fine white needles which darken on exposure to light and air. Soluble 1 in 17 of water, 1 in 4 of alcohol, 1 in 51 of chloroform, and 1 in 16.5 of ether. Store in airtight containers. Protect from light.

Adverse Effects, Treatment, and Precautions
Topical hydroquinone may cause transient erythema and a mild burning sensation. Occasionally hypersensitivity has occurred and US licensed product information recommends skin testing before use. Hydroquinone should not be applied to abraded or sunburnt skin. It should not be used to bleach eyelashes or eyebrows and contact with the eyes should be avoided as it may produce staining and corneal opacities. High concentrations or prolonged use may produce a blue-black hyperpigmentation (ochronosis) or pigmented colloid milium. The systemic effects of hydroquinone and their treatment are similar to those of phenol (see p.1656) but tremors and convulsions may also occur.

Carcinogenicity. There is some evidence from *animal* studies that hydroquinone might be carcinogenic (see Effects on the Skin, below).

Effects on the liver. Toxic hepatitis in a radiographer was attributed to occupational exposure to hydroquinone fumes from the developing medium used in the darkroom.[1] However, it has been pointed out[2] that hydroquinone is not volatile under normal conditions of use and that surveillance of 879 people engaged in the manufacture and use of hydroquinone from 1942 to 1990 found no association between toxic hepatitis and hydroquinone exposure.

1. Nowak AK, *et al.* Darkroom hepatitis after exposure to hydroquinone. *Lancet* 1995; **345:** 1187.
2. O'Donaghue JL, *et al.* Hydroquinone and hepatitis. *Lancet* 1995; **346:** 1427–8.

Effects on the skin. The incidence of exogenous ochronosis (blue-black hyperpigmentation) in a survey of black South African patients was found to be 15% in males and 42% in females with 69% of affected individuals admitting to using hydroquinone-containing preparations.[1] This was considered to be more consistent with a toxic effect of a drug with a low therapeutic index, rather than an idiosyncratic reaction. The data revealed that even preparations with hydroquinone 2% or less with a sunscreen produced ochronosis. Ochronosis usually became apparent after about 6 months of use and, once established, was probably irreversible. Patients may initially use skin lighteners for cosmetic purposes but once ochronosis develops they may fall into the 'skin lightener trap' as they use other hydroquinone preparations to remove the disfigurement.[1] Treatment of exogenous ochronosis is based on stopping the use of hydroquinone, but it may take years for any improvement to be apparent. There are a few reports of benefit from topical tretinoin, dermabrasion, and laser therapy, but these are far from established therapies.[2] Reversible brown discoloration of the nails has also been reported after the use of skin lighteners containing hydroquinone.[3-5]

In addition to the risk of ochronosis it has been suggested that, based on *animal* studies, long-term use of hydroquinone might be carcinogenic.[6] In the USA, preparations containing up to 2% hydroquinone may be sold without prescription, but in 2006, based on data regarding potential carcinogenicity and reports of ochronosis, the FDA proposed to reclassify these products as drugs and make them available by prescription only.[7] In Europe the use of hydroquinone in cosmetic preparations for skin lightening is already banned, but it is still available for prescription as a medicine.[6]

1. Hardwick N, *et al.* Exogenous ochronosis: an epidemiological study. *Br J Dermatol* 1989; **120:** 229–38.
2. Levin CY, Maibach H. Exogenous ochronosis: an update on clinical features, causative agents and treatment options. *Am J Clin Dermatol* 2001; **2:** 213–17.
3. Mann RJ, Harman RRM. Nail staining due to hydroquinone skin-lightening creams. *Br J Dermatol* 1983; **108:** 363–5.
4. Ozluer SM, Muir J. Nail staining from hydroquinone cream. *Australas J Dermatol* 2000; **41:** 255–6.
5. Parlak AH, *et al.* Discolouration of the fingernails from using hydroquinone skin-lightening cream. *J Cosmet Dermatol* 2003; **2:** 199–201.
6. Kooyers TJ, Westerhof W. Toxicology and health risks of hydroquinone in skin lightening formulations. *J Eur Acad Dermatol Venereol* 2006; **20:** 777–80.
7. FDA. Skin bleaching drug products for over-the-counter human use: proposed rule. *Fed Regist* 2006; **71:** 51146–55. Available at: http://a257.g.akamaitech.net/7/257/2422/01jan20061800/edocket.access.gpo.gov/2006/pdf/E6-14263.pdf (accessed 27/09/07)

Uses and Administration
Hydroquinone increases melanin excretion from melanocytes and may also prevent its production. Hydroquinone is used topically as a depigmenting agent for the skin in hyperpigmentation conditions (p.1582) such as chloasma (melasma), freckles, and lentigines (liver spots). Concentrations of 2 to 4% are commonly used; higher concentrations may be very irritant and increase the risk of ochronosis. It may be several weeks before any effect is apparent but depigmentation may last for 2 to 6 months after stopping. Application of hydroquinone should stop if there is no improvement after 2 months. Hydroquinone should be applied twice daily only to intact skin which should be protected from sunlight to reduce repigmentation. A preparation containing hydroquinone 4%, tretinoin 0.05%, and fluocinolone acetonide 0.01% may be applied once daily at night in the treatment of chloasma (melasma). Hydroquinone preparations often include a sunscreen or a sunblocking basis.

Hydroquinone is also used as an antoxidant in topical preparations and in photographic developers.

Preparations

USP 31: Hydroquinone Cream; Hydroquinone Topical Solution.

Proprietary Preparations (details are given in Part 3)
Arg.: Claripel; ***Braz.:*** Claripel; Solaquin; ***Canad.:*** African Gold†; Banishing Cream; Eldopaque; Eldoquin; Esoterica Regular; Esoterica Unscented; Lustra; Nadinola†; NeoStrata Canada HQ Plus; Porcelana Nighttime Formula†; Ultraquin Plain; ***Chile:*** Etnoderm; Unitone 4; ***Hong Kong:*** Derma-Rx Lightener; Eldopaque; Eldoquin; Solaquin; ***Indon.:*** Bioquin; Mediquin; Melanox; Melaskin; Pigmet; Pylaquin; Qutifar; Vitaquin; ***Israel:*** Esomed; ***Malaysia:*** Eldopaque; Eldoquin; ***Mex.:*** Crema Blanca; Eldopaque; Eldoquin; Hidroquin; Melanex; Quinoret Forte; ***NZ:*** Eldoquin; ***Singapore:*** Eldopaque; Eldoquin; Polyquin; Solaquin; ***Spain:*** Hidroquilaude; Licostrata; Melanasa; Nadona; Pigmentasa; ***Thai.:*** Clariderm; ***Turk.:*** Expigment; ***UK:*** Eldopaque; Eldoquin; Solaquin; ***USA:*** Aclaro; Claripel; Eldopaque; Eldoquin; EpiQuin; Esoterica Regular; Lustra; Solaquin; ***Venez.:*** Pharquinon†.

Multi-ingredient: ***Arg.:*** Melacler†; Melasmax; Neoceuticals Crema Despigmentante de Dia†; Neoquin; Neoquin Forte; Neostrata Gel Despigmentante; Solaquin Forte; Tri-Luma; ***Austral.:*** Superfade; ***Braz.:*** Glyquin; Tri-Luma; Vitacid Plus; ***Canad.:*** Esoterica; Glyquin XM; Lustra-AF; NeoStrata Canada HQ Plus; NeoStrata HQ; Porcelana Daytime Formula†; Solaquin Forte†; Ultraquin; Viquin Forte†; ***Chile:*** Alastik†; Clasifel; D 4†; Neostrata; Tri-Luma; Trio-D†; ***Ger.:*** Pigmanorm; ***Hong Kong:*** Glyquin; Superfade; Tri-Luma; ***India:*** Melalite 15; ***Indon.:*** Hidrogel; Interquin; Interquin Plus; Nu-Derm Sunblock; ***Malaysia:*** Solaquin Forte; Tri-Luma; ***Mex.:*** Clasifel; Nova Derm; Quinoret; Solaquin; Tri-Luma; ***Philipp.:*** Tri-Luma; ***Singapore:*** Glyquin; Glyquin XM; Tri-Luma; ***Switz.:*** Pigmanorm; ***Thai.:*** Tri-Luma; ***Turk.:*** Metamorfoz; ***USA:*** Esoterica Facial and Sunscreen; Glyquin XM; Solaquin Forte; Tri-Luma; ***Venez.:*** Tri-Luma.