Colfosceril Palmitate *(BAN, USAN, rINN)*

Colfoscéril, Palmitate de; Colfoscerili Palmitas; Dipalmitoylphosphatidylcholine; DPPC; Palmitato de colfoscerilo; 129Y83. 1,2-Dipalmitoyl-*sn*-glycero(3)phosphocholine.

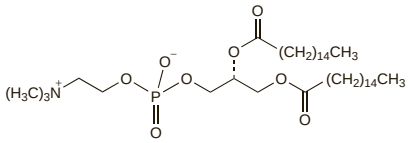
Колфосцерила Пальмитат

$C_{40}H_{80}NO_8P$ = 734.0.

CAS — 63-89-8.

ATC — R07AA01.

ATC Vet — QR07AA01.



Description. Colfosceril palmitate is a phospholipid which forms an important constituent of natural and many synthetic pulmonary surfactant compounds.

Lucinactant *(USAN)*

ATI-02; KL_4-surfactant.

Description. Lucinactant is a mixture of sinapultide, colfosceril palmitate, sodium palmitoyloleaylphosphatidyl glycerol, and palmitic acid.

Poractant Alfa *(BAN)*

CAS — 129069-19-8.

Description. Poractant alfa is an extract of porcine lung containing not less than 90% of phospholipids, about 1% of hydrophobic proteins (SP-B and SP-C), and about 9% of other lipids.

Pumactant *(BAN)*

Artificial Lung Expanding Compound.

Description. Pumactant is a mixture of colfosceril palmitate and phosphatidyl glycerol (2-oleoyl-1-palmitoyl-*sn*-glycero(3)phospho(1)-*sn*-glycerol) in the proportion 7:3.

Sinapultide *(USAN, rINN)*

ATI-01; Sinapultida; Sinapultidum.

Синапультид

CAS — 138531-07-4.

Description. Sinapultide is a synthetic peptide that mimics the actions of human surfactant protein B, an important constituent of natural pulmonary surfactant compounds.

Adverse Effects and Precautions

Surfactant therapy may be associated with an increased risk of pulmonary haemorrhage, especially in more premature infants. Therapy should only be given where there are adequate facilities for ventilation and monitoring. Rapid chest expansion and improvement of oxygenation may follow successful treatment, and peak ventilatory pressure and inspired oxygen concentration may need to be reduced promptly to avoid the risk of pneumothorax and hyperoxaemia. A transient decrease in brain electrical activity has been reported in neonates given surfactant but its significance is unknown. Transient bradycardia has also been reported. Giving surfactant has occasionally been associated with obstruction of the endotracheal tube by mucus.

◊ While surfactant therapy is clearly associated with an increased risk of pulmonary haemorrhage,[1-4] meta-analysis suggests that the risk is small compared with the benefits.[1] However, neonates who do develop moderate or severe pulmonary haemorrhage after surfactant therapy are at increased risk of death or short-term morbidity.[5] Haemodynamic changes associated with surfactant therapy or consequent pulmonary haemorrhage may also predispose premature infants to intracranial (periventricular) haemorrhage.[5,6] Early preventive use of surfactant in very low birthweight infants may be associated with a poorer neurodevelopmental outcome,[7] although a long-term follow-up study[8] of premature infants born in the surfactant era concluded that these children had similar neurodevelopmental outcomes to such children born before the introduction of surfactant therapy. Decreased brain electrical activity has been reported after surfactant treatment.[9]

The rate of instillation of surfactant may be significant: one study,[10] in which the apparatus was adapted so that mechanical ventilation could continue while giving surfactant, found that rapid instillation over a 5-minute period provoked a transient increase in cerebral blood flow velocity associated with an increase in carbon dioxide tension, compared with slow instillation over 15 minutes. Although the authors acknowledged that such changes were likely to be related to several factors, particularly the type of surfactant, they recommended that, until further data were available, instillation should take place slowly, over at least 15 to 20 minutes.

1. Raju TNK, Langenberg P. Pulmonary hemorrhage and exogenous surfactant therapy: a metaanalysis. *J Pediatr* 1993; **123:** 603–10.
2. Majeed-Saidan MA, *et al.* Pulmonary haemorrhage in low-birthweight babies. *Lancet* 1993; **341:** 120.
3. Rogers D. Pulmonary haemorrhage, surfactant, and low-birthweight babies. *Lancet* 1993; **341:** 698.
4. Pappin A, *et al.* Extensive intraalveolar pulmonary hemorrhage in infants dying after surfactant therapy. *J Pediatr* 1994; **124:** 621–6.
5. Pandit PB, *et al.* Outcome following pulmonary haemorrhage in very low birthweight neonates treated with surfactant. *Arch Dis Child Fetal Neonatal Ed* 1999; **81:** F40–F44.
6. Gunkel JH, Banks PLC. Surfactant therapy and intracranial hemorrhage: review of the literature and results of new analyses. *Pediatrics* 1993; **92:** 775–86.
7. Vaucher YE, *et al.* Outcome at twelve months of adjusted age in very low birthweight infants with lung immaturity: a randomized placebo-controlled trial of human surfactant. *J Pediatr* 1993; **122:** 126–32.
8. D'Angio CT, *et al.* Longitudinal, 15-year follow-up of children born at less than 29 weeks' gestation after introduction of surfactant therapy into a region: neurologic, cognitive, and educational outcomes. *Pediatrics* 2002; **110:** 1094–1102.
9. Hellström-Westas L, *et al.* Cerebroelectrical depression following surfactant treatment in preterm neonates. *Pediatrics* 1992; **89:** 643–7.
10. Saliba E, *et al.* Instillation rate effects of Exosurf on cerebral and cardiovascular haemodynamics in preterm neonates. *Arch Dis Child* 1994; **71:** F174–8.

Uses and Administration

Pulmonary surfactants are compounds with surface active properties similar to those natural substances in the lung that help to maintain the patency of the airways by reducing the surface tension of pulmonary fluids. Exogenous pulmonary surfactants are used in the treatment of neonatal respiratory distress syndrome (p.1508) in premature infants, and may also be given for prevention in infants considered to be at risk of developing the syndrome. Doses vary, but most pulmonary surfactants are given in recommended doses of 100 to 200 mg phospholipids per kg birth-weight; a suggested dose for colfosceril palmitate is 67.5 mg/kg. For the treatment of overt neonatal respiratory distress syndrome, the initial dose is given as soon as possible after diagnosis, while for prevention it is given as soon as possible after birth. It is given as a suspension via an endotracheal tube to intubated neonates receiving mechanical ventilation. Manufacturers may recommend regimens with or without disconnection from the ventilator. Repeat doses may be given if necessary, although the number of doses and the dosage interval varies.

Pulmonary surfactants have also been tried in bronchopulmonary dysplasia in premature infants, meconium aspiration syndrome in newborn infants, and acute respiratory distress syndrome in adults. A similar compound lusupultide is also under investigation for aspiration pneumonitis.

Acute respiratory distress syndrome. Pulmonary surfactants have been investigated for acute respiratory distress syndrome (p.1498). In adults, they have been given by intrabronchial instillation[1] or nebulisation[2-4] but results have been largely disappointing. Sequential bronchopulmonary segmental lavage with a synthetic surfactant has also been tried[5] and appeared to be well tolerated. Endotracheal poractant alfa moderately improved oxygenation in some children with severe acute respiratory distress syndrome secondary to pulmonary or systemic disease.[6]

1. Haslam PL, *et al.* Surfactant replacement therapy in late-stage adult respiratory distress syndrome. *Lancet* 1994; **343:** 1009–11.
2. do Campo JL, *et al.* Natural surfactant aerosolisation in adult respiratory distress syndrome. *Lancet* 1994; **344:** 413–14.
3. Weg JG, *et al.* Safety and potential efficacy of an aerosolized surfactant in human sepsis-induced adult respiratory distress syndrome. *JAMA* 1994; **272:** 1433–8.
4. Anzueto A, *et al.* Aerosolized surfactant in adults with sepsis-induced respiratory distress syndrome. *N Engl J Med* 1996; **334:** 1417–21.
5. Wiswell TE, *et al.* Bronchopulmonary segmental lavage with Surfaxin (KL -Surfactant) for acute respiratory distress syndrome. *Am J Respir Crit Care Med* 1999; **160:** 1188–95.
6. López-Herce J, *et al.* Surfactant treatment for acute respiratory distress syndrome. *Arch Dis Child* 1999; **80:** 248–52.

Drowning. Reference to the use of colfosceril palmitate in the management of a 9-year-old rescued after near drowning.[1]

1. McBrien M, *et al.* Artificial surfactant in the treatment of near drowning. *Lancet* 1993; **342:** 1485–6.

Meconium aspiration syndrome. Meconium aspiration syndrome produces respiratory distress in infants born at term or later and is a consequence of disturbances of the pulmonary surfactant system. Bolus doses of exogenous pulmonary surfactant are of benefit in some ventilated infants, although lung lavage with dilute surfactant is also under investigation.[1] Results from a pilot study[2] of beractant as a tracheobronchial lavage fluid for the treatment of infants with severe meconium aspiration syndrome were promising, and a small comparative trial[3] found that bronchoalveolar lavage with diluted beractant, with or without intravenous dexamethasone, significantly improved oxygenation in neonates when compared with standard therapy. Systematic review[4] of 4 randomised controlled trials evaluating the effect of pulmonary surfactants also found encouraging results, although comparison with other established treatments for meconium aspiration syndrome remains to be done.

1. Dargaville PA, Mills JF. Surfactant therapy for meconium aspiration syndrome: current status. *Drugs* 2005; **65:** 2569–91.
2. Lam BCC, Yeung CY. Surfactant lavage for meconium aspiration syndrome: a pilot study. *Pediatrics* 1999; **103:** 1014–18.
3. Salvia-Roigés MD, *et al.* Efficacy of three treatment schedules in severe meconium aspiration syndrome. *Acta Paediatr* 2004; **93:** 60–5.
4. El Shahed AI, *et al.* Surfactant for meconium aspiration syndrome in full term/near term infants. Available in The Cochrane Database of Systematic Reviews; Issue 3. Chichester: John Wiley; 2007 (accessed 13/06/08).

Preparations

Proprietary Preparations (details are given in Part 3)
Arg.: Baby Fact B; Exosurf†; Natsurf†; Surfactante B; Survanta; ***Austral.:*** Curosurf; Exosurf; Survanta; ***Austria:*** Alveofact†; Curosurf; Exosurf; Survanta; ***Belg.:*** Alvofact†; Curosurf; Survanta; ***Braz.:*** Alveofact; Curosurf; Exosurf†; Survanta; ***Canad.:*** BLES; Exosurf†; Survanta; ***Chile:*** Exosurf†; Survanta; ***Cz.:*** Alveofact†; Curosurf; Exosurf†; Survanta; ***Denm.:*** Curosurf; ***Fin.:*** Curosurf; ***Fr.:*** Curosurf; Survanta; ***Ger.:*** Alveofact; Curosurf; Survanta; ***Gr.:*** Alvofact; Curosurf; Exosurf†; Survanta; ***Hong Kong:*** Exosurf†; Survanta; ***Hung.:*** Curosurf; Survanta; ***Indon.:*** Survanta; ***Irl.:*** Curosurf; Exosurf†; ***Israel:*** Curosurf; Exosurf†; Infasurf; ***Ital.:*** Curosurf; Exosurf†; ***Jpn:*** Surfacten; ***Malaysia:*** Survanta; ***Mex.:*** Exosurf; Survanta; ***Neth.:*** Alvofact†; Curosurf; Exosurf†; Survanta; ***Norw.:*** Curosurf; Survanta-Vent; ***NZ:*** Curosurf; Survanta; ***Philipp.:*** Survanta; ***Pol.:*** Alveofact; Curosurf; Survanta; ***Port.:*** Curosurf; ***Rus.:*** Curosurf (Куросурф); ***S.Afr.:*** Curosurf; Survanta; ***Singapore:*** Survanta; ***Spain:*** Curosurf; Survanta; ***Swed.:*** Curosurf; Survanta-Vent†; ***Switz.:*** Curosurf; Survanta; ***Thai.:*** Curosurf; Survanta; ***UK:*** Curosurf; Survanta; ***USA:*** Curosurf; Exosurf†; Infasurf; Survanta; ***Venez.:*** Survanta.

Pulsatilla

Anémone pulsatille; Meadow Anemone; Pasque Flower.

CAS — 62887-80-3.

Profile

Pulsatilla is the whole flowering plant of *Pulsatilla vulgaris* (*Anemone pulsatilla*) or *Pulsatilla pratensis* (Ranunculaceae). It has been used in herbal preparations for the treatment of conditions including nervous disorders, circulatory disorders, and gynaecological disorders and benign prostatic hyperplasia.

Homoeopathy. Pulsatilla has been used in homoeopathic medicines under the following names: Pulsatilla pratensis; Pulsatilla vulgaris; Pulsatilla nigricans; Puls.

Preparations

Proprietary Preparations (details are given in Part 3)
USA: Yeast-X†.

Multi-ingredient: ***Austral.:*** Bioglan Cirflo†; Calmo; Lifesystem Herbal Formula 4 Women's Formula†; Proflo†; Women's Formula Herbal Formula 3†; ***Braz.:*** Eviprostat†; ***Cz.:*** Cicaderma; ***Fr.:*** Cicaderma; Hepatoum; Histo-Fluine P; ***Ger.:*** Eviprostat N; ***Indon.:*** Eviprostat; ***Jpn:*** Eviprostat; ***Port.:*** Cicaderma; ***S.Afr.:*** Cough Elixir; ***Singapore:*** Eviprostat; ***UK:*** Anased; Menopause Relief; Period Pain Relief; Prementaid.

Pumilio Pine Oil

Dwarf Pine Needle Oil; Dwarf Pine Oil; Essence de Pin de Montagne; Latschenöl; Oleum Pini Pumilionis; Olio di Mugo; Pin de montagne, huile essentielle de; Pini pumilionis aetheroleum; Pino mugo, aceite esencial de.

CAS — 8016-46-4.

Pharmacopoeias. In *Eur.* (see p.vii).

Ph. Eur. 6.2 (Dwarf Pine Oil). An essential oil obtained by steam distillation of the fresh leaves and twigs of *Pinus mugo*. A suitable antoxidant may be added. Relative density 0.857 to 0.868. A clear, colourless or pale yellow liquid. Store in inert, well-filled, airtight containers at a temperature not exceeding 25°. Protect from light.

Profile

Pumilio pine oil is a volatile oil obtained by distillation from the fresh leaves of *Pinus mugo* var. *pumilio* (Pinaceae). It has been inhaled with steam, sometimes with other essential oils, to relieve cough and nasal congestion and has been applied externally as a rubefacient. It has also been used as a perfume.

P. mugo is a source of pine needle oil (see Pine Oil, p.2368).

Preparations

Proprietary Preparations (details are given in Part 3)
Multi-ingredient: ***Austral.:*** Biosal Arthritis; Biosal†; Goanna Heat Cream; Goanna Salve; Karvol†; Menalation†; Vicks Inhaler; ***Austria:*** Berggeist; Bronchostop; Colda; Emser Nasensalbe; Erkaltungsbalsam; Expectal-Balsam; Leukona-Rheuma-Bad; Luuf Balsam; Mentopin; Nasanal; Opino; Piniment; ***Cz.:*** Thrombocid; Transpulmin; ***Ger.:*** Aerosol Spitzner N†; Dolo-cyl; Em-eukal†; Emser Nasensalbe N†; Euflux; Franzbranntwein; Hevertopect N†; Klosterfrau Franzbranntwein Latschenkiefer; Nasentropfen-ratiopharm†; Ner-fluid S; polio-elan; Rosarthron†; Thrombocid; ***Gr.:*** Opino-jel; ***Irl.:*** Karvol; ***Israel:*** Karvol; Mentholatum Balm; ***Ital.:*** Altuss; Antipulmina†; Broncosedina; Pinedrin†; Pumilene Vapo; ***Malaysia:*** Purporent†; ***Neth.:*** Luuf Verkoudheidsbalsem; ***NZ:*** Vicks Inhaler; ***Port.:*** Thrombocid; ***Switz.:*** Eau-de-vie de France avec huile de pin nain du Tirol†; Eucapinol; Liberol Baby N; Liberol Bain†; Liberol N; Makaphyt Baume†; Pinimenthol†; Piniol Pommade Speciale†; Thrombocid; ***UK:*** Allens Pine & Honey; Karvol; Mentholatum Rub; Original Cabdrivers Expectorant; Potter's Catarrh Pastilles.

Punarnava

Punarnaba.

Profile

Punarnava is the fresh or dried plant *Boerhaavia diffusa* (*B. repens*) (Nyctaginaceae), containing an alkaloid, punarnavine. It has been used as a diuretic and for liver disorders, usually in the form of a liquid extract.

Preparations

Proprietary Preparations (details are given in Part 3)
Multi-ingredient: ***Rus.:*** Dipana (Дипана).

Pyricarbate *(rINN)*

Piricarbato; Pyricarbatum; Pyridinolcarbamate. 2,6-Pyridinediyldimethylene bis(methylcarbamate).

Пирикарбат

$C_{11}H_{15}N_3O_4$ = 253.3.
CAS — 1882-26-4.

Profile

Pyricarbate has been given by mouth in the treatment of atherosclerosis and other vascular disorders, hyperlipidaemias, and thromboembolic disorders. Adverse effects have included gastrointestinal disturbances and liver damage.

Preparations

Proprietary Preparations (details are given in Part 3)
Ital.: Cicloven; ***Port.:*** Anginin†.

Quassia

Bitter Wood; Cuasia; Leño de Cuasia; Quassia Wood; Quassiae Lignum; Quassiaholz.

CAS — 76-78-8 (quassin); 76-77-7 (neoquassin).
ATC — P03AX04.
ATC Vet — QP53AX03.

Profile

Quassia is the dried stem wood of Jamaica quassia, *Picrasma excelsa* (*Aeschrion excelsa; Picraena excelsa*) (Simaroubaceae), or of Surinam quassia, *Quassia amara* (Simaroubaceae). It has been used as a bitter. It was formerly given as an enema for the expulsion of threadworms and was applied for pediculosis. It may also be used as a flavour in food, drinks, and confectionery. Extracts of quassia or preparations containing its triterpenoid bitter principle quassin are used to denature alcohol.

Preparations

Proprietary Preparations (details are given in Part 3)
Arg.: Cuassicum Prevent 2 en 1; Picutex.
Multi-ingredient: ***Arg.:*** Aulo Repelente De Piojos; Cuassicum; Fuera Bicho; Uze Active; Yalu†; ***Braz.:*** Camomila; ***Fr.:*** Quintonine; Skin Nail; ***Ital.:*** Dekar 2; ***S.Afr.:*** Essens Amara of Groen Amara; Versterkdruppels; ***Switz.:*** Stomacine; ***UK:*** Sanderson's Throat Specific.

Quebracho

Quebracho Blanco; White Quebracho.

Квебрахо Белое

NOTE. Do not confuse with trees belonging to the genus *Schinopsis* that may also be referred to as quebracho.

Profile

The bark of white quebracho, *Aspidosperma quebracho-blanco* (Apocynaceae) is used in herbal medicine for the treatment of respiratory disorders. It has also been used to reduce fever, as an antihypertensive, and as a flavouring agent.

Preparations

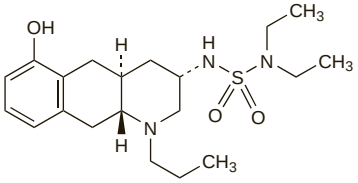
Proprietary Preparations (details are given in Part 3)
Multi-ingredient: ***Cz.:*** Afrodor; Bronchicum Elixir†; Bronchicum Tropfen†; ***Ger.:*** Afrodor; Bronchicum Elixir N†; Bronchicum Tropfen N†; ***Hung.:*** Afrodor†; ***Pol.:*** Bronchicum Eliksir; Bronchicum†; ***Rus.:*** Afrodor (Афродор); Bronchicum (Бронхикум); ***S.Afr.:*** Bronchicough†; Bronchicum†; ***Spain:*** Broncovital†.

Quinagolide Hydrochloride *(BANM, rINNM)*

CV-205-502 (quinagolide); Hidrocloruro de quinagolida; Quinagolide, Chlorhydrate de; Quinagolidi Hydrochloridum; SD2-CV-205-502 (quinagolide). (±)-*N,N*-Diethyl-N′-[(3*R**,4a*R**,10a*S**)-1,2,3,4,4a,5,10,10a-octahydro-6-hydroxy-1-propylbenzo[*g*]quinolin-3-yl]sulfamide hydrochloride.

Хинаголида Гидрохлорид

$C_{20}H_{33}N_3O_3S,HCl$ = 432.0.
CAS — 87056-78-8 (quinagolide); 94424-50-7 (quinagolide hydrochloride).
ATC — G02CB04.
ATC Vet — QG02CB04.



(quinagolide)

Adverse Effects and Precautions

As for Bromocriptine, p.798, although it is not an ergot derivative and does not seem to be associated with fibrotic reactions or vasoconstriction. Licensed product information contra-indicates the use of quinagolide in patients with hepatic or renal impairment; however, this is based on a lack of data in such patients.

Effects on mental function. For reports of daytime somnolence occurring in patients receiving dopamine agonists including quinagolide, see Effects on Mental Function, under Adverse Effects of Levodopa, p.805.

Interactions

As for Bromocriptine, p.800.

Pharmacokinetics

Quinagolide is rapidly absorbed from the gastrointestinal tract and undergoes extensive first-pass metabolism to the *N*-desethyl analogue which is biologically active and the *N,N*-didesethyl analogue. Roughly equal amounts of a dose appear in the urine and the faeces; it is excreted in the urine as sulfate or glucuronide conjugates of quinagolide and its metabolites and in the faeces as the unconjugated forms. Protein binding has been reported to be about 90%. The elimination half-life of quinagolide at steady state is about 17 hours.

Uses and Administration

Quinagolide is a non-ergot dopamine D_2-agonist that has actions and uses similar to those of bromocriptine (p.800). It is used in the treatment of disorders associated with hyperprolactinaemia.

Quinagolide is given as the hydrochloride, but doses are described in terms of the base; 27.3 micrograms of quinagolide hydrochloride is equivalent to about 25 micrograms of quinagolide. The initial dose, given once daily with food at bedtime, is 25 micrograms daily for 3 days. Doses are increased thereafter at 3-day intervals in steps of 25 micrograms until the optimal response is achieved, which is usually within the range of 75 to 150 micrograms daily. If doses greater than 300 micrograms daily are required, the daily dosage may be increased in steps of 75 to 150 micrograms at intervals of not less than 4 weeks.

Quinagolide has also been investigated in the treatment of acromegaly and lactation inhibition.

Acromegaly. Dopaminergics can produce a paradoxical reduction in growth hormone secretion and may be used in the treatment of acromegaly as adjunctive therapy to surgery, radiotherapy, or somatostatin analogues to reduce circulating growth hormone levels, although they are less effective than somatostatin analogues (p.1798). Bromocriptine has been the main dopamine agonist used, but quinagolide has been tried and results of an open study[1] in which quinagolide was given to 17 patients with acromegaly suggest that quinagolide has a more prolonged effect on suppression of growth hormone secretion than bromocriptine. However, it was ineffective in bromocriptine-resistant patients. In another study involving 34 patients, quinagolide was more effective than either cabergoline or a depot preparation of bromocriptine in normalising circulating growth hormone and insulin-like growth factor.[2]

1. Chiodini PG, *et al.* CV 205-502 in acromegaly. *Acta Endocrinol (Copenh)* 1993; **128:** 389–93.
2. Colao A, *et al.* Effect of different dopaminergic agents in the treatment of acromegaly. *J Clin Endocrinol Metab* 1997; **82:** 518–23.

Hyperprolactinaemia and prolactinomas. Dopamine agonists such as quinagolide are widely used for the treatment of hyperprolactinaemia secondary to a prolactinoma (see p.2079).

References.

1. Rohmer V, *et al.* Efficacy of quinagolide in resistance to dopamine agonists: results of a multicenter study. *Ann Endocrinol (Paris)* 2000; **61:** 411–17.
2. Schultz PN, *et al.* Quinagolide in the management of prolactinoma. *Pituitary* 2000; **3:** 239–49.
3. Barlier A, Jaquet P. Quinagolide—a valuable treatment option for hyperprolactinaemia. *Eur J Endocrinol* 2006; **154:** 187–95.

Lactation inhibition. A small preliminary study[1] has suggested that quinagolide is of similar efficacy to bromocriptine for prevention of puerperal lactation. However, the routine use of dopaminergics is not recommended for the suppression of physiological lactation (see p.2003).

1. van der Heijden PFM, *et al.* Lactation inhibition by the dopamine agonist CV 205-502. *Br J Obstet Gynaecol* 1991; **98:** 270–6.

Preparations

Proprietary Preparations (details are given in Part 3)
Austria: Norprolac; ***Canad.:*** Norprolac; ***Cz.:*** Norprolac; ***Fin.:*** Norprolac; ***Fr.:*** Norprolac; ***Ger.:*** Norprolac; ***Gr.:*** Norprolac; ***Hong Kong:*** Norprolac†; ***Hung.:*** Norprolac; ***Israel:*** Norprolac; ***Mex.:*** Norprolac; ***Neth.:*** Norprolac; ***Norw.:*** Norprolac; ***Pol.:*** Norprolac; ***Port.:*** Norprolac; ***Rus.:*** Norprolac (Норпролак); ***S.Afr.:*** Norprolac; ***Spain:*** Norprolac; Prodelion; ***Swed.:*** Norprolac; ***Switz.:*** Norprolac; ***UK:*** Norprolac.

Quinine and Urea Hydrochloride

Carbamidated Quinine Dihydrochloride; Chininum Dihydrochloricum Carbamidatum; Quinina y urea, hidrocloruro de; Urea-Quinine.

$C_{20}H_{24}N_2O_2,CH_4N_2O,2HCl,5H_2O$ = 547.5.
CAS — 549-52-0 (anhydrous quinine and urea hydrochloride).

Profile

Quinine and urea hydrochloride has been used for the treatment of haemorrhoidal bleeding and anal fissure. It was formerly used as a local anaesthetic and for the therapeutic actions of quinine.

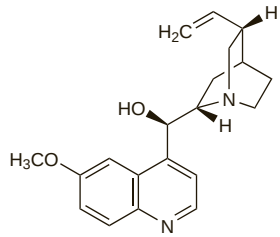
Preparations

Proprietary Preparations (details are given in Part 3)
Fr.: Kinurea H.

Quinine Ascorbate *(USAN)*

Quinina, ascorbato de; Quinine Biascorbate.

$C_{20}H_{24}N_2O_2,2C_6H_8O_6$ = 676.7.
CAS — 146-40-7.



(quinine)

Profile

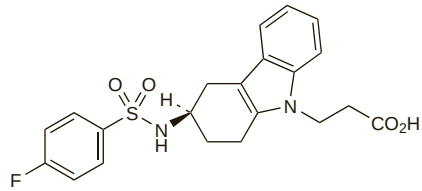
Quinine ascorbate is a compound (2 : 1) of ascorbic acid with quinine. It has been used as an ingredient of preparations promoted as smoking deterrents.

Ramatroban *(BAN, rINN)*

BAY-U-3405; EN-137774; Ramatrobán; Ramatrobanum. (*R*)-3-[3-(4-Fluorophenylsulphonylamino)-1,2,3,4-tetrahydrocarbazol-9-yl]propionic acid.

Рематробан

$C_{21}H_{21}FN_2O_4S$ = 416.5.
CAS — 116649-85-5.



Profile

Ramatroban is a thromboxane A_2 inhibitor that has been used for the treatment of allergic rhinitis.

Preparations

Proprietary Preparations (details are given in Part 3)
Jpn: Baynas.

Ranibizumab *(BAN, USAN, rINN)*

Ranibizumabum; rhuFab V2. Immunoglobulin G1, anti-(human vascular endothelial growth factor) Fab fragment (human-mouse monoclonal rhuFAB V2 γ1-chain), disulfide with human-mouse monoclonal rhuFAB V2 κ-chain.

Ранибицумаб

CAS — 347396-82-1.
ATC — S01LA04.
ATC Vet — QS01LA04.

Adverse Effects and Precautions

Intra-ocular inflammation and raised intra-ocular pressure may occur with ranibizumab. Adverse effects relating to the injection procedure include endophthalmitis, rhegmatogenous retinal detachment, retinal tear, and iatrogenic traumatic cataract. Patients should be monitored for signs of infections for a week after the procedure. Common but less serious ocular adverse effects include red eye, eye pain, vitreous floaters, eye irritation, increased lachrymation, and the sensation of a foreign body in the eye.

Non-ocular adverse effects that have been reported include headache, nausea, arthralgia, back pain, bronchitis, anaemia, and hypertension. Arterial thromboembolic events are a theoretical possibility with vascular endothelial growth factor inhibitors.

Ranibizumab is contra-indicated in patients with active or suspected ocular or periocular infections, or active severe intra-ocular inflammation.

Stroke. The manufacturers of ranibizumab have reported that interim analysis of results from an ongoing study have revealed