

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

Piricarbato; Pyricarbatus; Pyridinolcarbamate. 2,6-Pyridinediyl-dimethylene bis(methylcarbamate).

Пирикарбат

C₁₁H₁₅N₃O₄ = 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.:** Angin†.**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.

ProfileQuassia is the dried stem wood of Jamaica quassia, *Picrasma excelsa* (*Aeschlin 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 I; Picutec.**Multi-ingredient:** **Arg.:** Aulo Repelente De Piojos; Cuassicum; Fuera Bicho; Uze Active; Yaluf; **Braz.:** Camomila; **Fr.:** Quinonine; Skin Nail; **Ital.:** Dekar 2; **S.Afr.:** Essens Amara de Groen Amara; Versterkdruppels; **Switz.:** Stomacine; **UK:** Sanderson's Throat Specific.**Quebracho**

Quebracho Blanco; White Quebracho.

Квбрахо 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 Elixir; Bronchicum†; **Rus.:** Afrodor (Афродор); Bronchicum (Бронхикум); **S.Afr.:** Bronchicough†; Bronchicum†; **Spain:** Broncovital†.**Quinagolide Hydrochloride** (BANM, rINN)

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

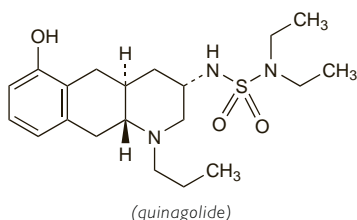
Хинаголида Гидрохлорид

C₂₀H₃₃N₃O₃·HCl = 432.0.

CAS — 87056-78-8 (quinagolide); 94424-50-7 (quinagolide hydrochloride).

ATC — G02CB04.

ATC Vet — QG02CB04.

**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.

PharmacokineticsQuinagolide 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 D₂-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 study¹ 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 study¹ 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; **Canada:** 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; **Sweden:** Norprolac; **Switz.:** Norprolac; **UK:** Norprolac.**Quinine and Urea Hydrochloride**Carbamidated Quinine Dihydrochloride; Chininum Dihydrochloricum Carbamidatum; Quinina γ urea, hidrocloruro de; Urea-Quinine.C₂₀H₂₄N₂O₂·CH₂N₂O₂·2HCl·5H₂O = 547.5.

CAS — 549-52-0 (anhydrous quinine and urea hydrochloride).

Profile

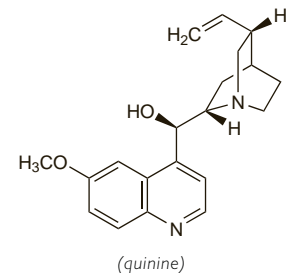
Quinine and urea hydrochloride has been used for the treatment of haemorrhoidal bleeding and anal fissure. It was formerly used as a local anaesthetic and for the therapeutic actions of quinine.

Preparations**Proprietary Preparations** (details are given in Part 3)**Fr.:** Kinurea H.**Quinine Ascorbate** (USAN)

Quinina, ascorbato de; Quinine Biscarbonate.

C₂₀H₂₄N₂O₂·2C₆H₈O₆ = 676.7.

CAS — 146-40-7.

**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.

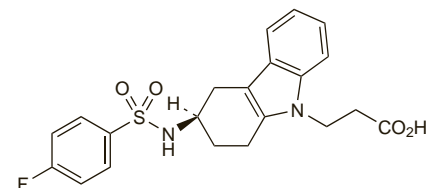
Ramatroban (BAN, rINN)

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

РАМАТРОБАН

C₂₁H₂₁FN₂O₄S = 416.5.

CAS — 116649-85-5.

**Profile**Ramatroban is a thromboxane A₂ 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 γ 1-chain), disulfide with human-mouse monoclonal rhuFAB V2 κ -chain.

Ранибизумаб

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 periorbital 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