2. Debontridder O. Extrapyramidal reactions due to domperidone. *Lancet* 1980; **ii:** 802. Correction, *ibid.*; 1259.
3. Casteels-Van Daele M, *et al.* Refusal of further cancer chemotherapy due to antiemetic drug. *Lancet* 1984; **i:** 57.
4. Spirt MJ, *et al.* Neuroleptic malignant syndrome induced by domperidone. *Dig Dis Sci* 1992; **37:** 946–8.

Precautions

Domperidone is not recommended for chronic use or for the routine prophylaxis of postoperative nausea and vomiting. Domperidone should be used with great caution if given intravenously, because of the risk of arrhythmias, especially in patients predisposed to cardiac arrhythmias or hypokalaemia.

Breast feeding. No adverse effects have been seen in breast-fed infants whose mothers were given domperidone, and the American Academy of Pediatrics considers[1] that it is therefore usually compatible with breast feeding. However, the FDA in the USA has issued a warning against the use of domperidone to increase milk production because of the possibility of serious adverse effects.[2] Others have commented that these warnings were based on data from patients with malignant disease receiving high doses of intravenous domperidone, and that if the mother were taking smaller oral doses, the total amount of drug ingested by an infant would be extremely small. They recommend that low-dose domperidone should still be considered for lactating women with decreased milk supply who are unresponsive to non-pharmacological measures to enhance lactation. However, patients should be warned of the risk of arrhythmias at high doses, and women with known cardiac disease should not take domperidone.[3]

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 07/05/04)
2. FDA. FDA warns against women using unapproved drug, domperidone, to increase milk production (June 7, 2004). Available at: http://www.fda.gov/bbs/topics/ANSWERS/2004/ANS01292.html (accessed 30/06/04)
3. da Silva OP, Knoppert DC. Domperidone for lactating women. *Can Med Assoc J* 2004; **171:** 725–6.

Interactions

As with other dopamine antagonists (see Metoclopramide, p.1749), there is a theoretical potential that domperidone may antagonise the hypoprolactinaemic effect of drugs such as bromocriptine. In addition, the prokinetic effects of domperidone may alter the absorption of some drugs. Opioid analgesics and antimuscarinics may antagonise the prokinetic effects of domperidone.

Domperidone is metabolised via the cytochrome P450 isoenzyme CYP3A4; use with ketoconazole has been reported to produce a threefold increase in plasma concentrations of domperidone, and an associated slight prolongation in QT interval. Similar increases in domperidone concentrations might theoretically be seen with other potent inhibitors of CYP3A4 such as erythromycin or ritonavir, and such combinations may be best avoided.

Pharmacokinetics

Although absorption is rapid, the systemic bioavailability of domperidone is only about 15% in fasting subjects given an oral dose; this is increased when domperidone is given after food. The low bioavailability is thought to be due to first-pass hepatic and intestinal metabolism. The bioavailability of rectal domperidone is similar to that after oral doses, although peak plasma concentrations are only about one-third that of an oral dose and are achieved after about an hour, compared with 30 minutes after an oral dose.

Domperidone is more than 90% bound to plasma proteins, and has a terminal elimination half-life of about 7.5 hours. It undergoes rapid and extensive hepatic metabolism. The main metabolic pathways are *N*-dealkylation by cytochrome P450 isoenzyme CYP3A4, and aromatic hydroxylation by CYP3A4, CYP1A2, and CYP2E1. About 30% of an oral dose is excreted in urine within 24 hours, almost entirely as metabolites; the remainder of a dose is excreted in faeces over several days, about 10% as unchanged drug. It does not readily cross the blood-brain barrier.

Small amounts of domperidone are distributed into breast milk; concentrations are 10 to 50% of those in maternal serum.

Uses and Administration

Domperidone is a dopamine antagonist with actions and uses similar to those of metoclopramide (p.1749). It is used as an antiemetic for the short-term treatment of nausea and vomiting of various aetiologies (p.1700). It is not considered suitable for chronic nausea and vomiting, nor for the routine prophylaxis of postoperative vomiting.

Domperidone is also used for its prokinetic actions in dyspepsia (p.1695) and has been tried in diabetic gastroparesis (see Diabetic Complications, p.433). It has been given with paracetamol in the symptomatic treatment of migraine (p.616).

Domperidone is used as the maleate in tablet preparations and as the base in suppositories and the oral suspension; doses are expressed in terms of the base. Domperidone maleate 12.73 mg is equivalent to about 10 mg of domperidone. Domperidone has been given parenterally, but this route has been associated with severe adverse effects (see above).

For the treatment of **nausea and vomiting** domperidone may be given in oral doses of 10 to 20 mg three or four times daily up to a maximum daily dose of 80 mg or it may be given rectally in a dose of 60 mg twice daily. For doses in children see below.

For the symptomatic management of non-ulcer **dyspepsia** similar oral doses of 10 mg taken up to four times daily (the last dose to be taken at night) have been recommended; if necessary, an increase in the dose to 20 mg may be prescribed. An initial course of treatment should not normally exceed 2 to 4 weeks. In **migraine**, a dose of 20 mg has been given orally up to every 4 hours, with paracetamol, as required, up to a maximum of 4 doses in 24 hours.

◊ Reviews.

1. Prakash A, Wagstaff AJ. Domperidone: a review of its use in diabetic gastropathy. *Drugs* 1998; **56:** 429–45.
2. Barone JA. Domperidone: a peripherally acting dopamine -receptor antagonist. *Ann Pharmacother* 1999; **33:** 429–40.
3. Ahmad N, *et al.* Making a case for domperidone in the treatment of gastrointestinal motility disorders. *Curr Opin Pharmacol* 2006; **6:** 571–6.
4. Reddymasu SC, *et al.* Domperidone: review of pharmacology and clinical applications in gastroenterology. *Am J Gastroenterol* 2007; **102:** 2036–45.

Administration in children. UK licensed product information states that children may be given domperidone in oral doses equivalent to 250 to 500 micrograms/kg three or four times daily; a total daily dose of 2.4 mg/kg or 80 mg, whichever is less, should not be exceeded. Alternatively, children weighing more than 15 kg may be given a rectal dose of 30 mg twice daily. The *BNFC* gives similar doses, but specifies use in children over 2 years; in those children over 35 kg, it allows an oral dose of 10 to 20 mg three or four times daily (maximum 80 mg daily) or a rectal dose of 60 mg twice daily.

Gastro-oesophageal reflux disease. A systematic review of the use of domperidone in infants and young children with gastro-oesophageal reflux (p.1696), which identified 4 randomised controlled studies of such use, considered that there was very little evidence of its efficacy in reducing symptoms.[1] Some suggest that it has been overused because of the lack of a suitable alternative after withdrawal of cisapride in many countries.[2]

1. Pritchard DS, *et al.* Should domperidone be used for the treatment of gastro-oesophageal reflux in children? Systematic review of randomized controlled trials in children aged 1 month to 11 years old. *Br J Clin Pharmacol* 2005; **59:** 725–9.
2. Vandenplas Y, *et al.* The diagnosis and management of gastro-oesophageal reflux in infants. *Early Hum Dev* 2005; **81:** 1011–24.

Parkinsonism. Domperidone is used to control gastrointestinal effects of dopaminergic drugs given in the management of parkinsonism (p.791). It may be of use in those patients who experience peripheral effects with levodopa despite the use of peripheral dopa-decarboxylase inhibitors and for patients using dopamine agonists such as bromocriptine or apomorphine since peripheral dopa-decarboxylase inhibitors are ineffective for preventing the peripheral effects of these drugs. Although domperidone does not readily cross the blood-brain barrier there have been isolated reports of extrapyramidal effects associated with its use (see above). Consequently there has been concern over its potential to produce central effects and some consider that domperidone should only be used in patients with parkinsonism when safer antiemetic measures have failed.[1,2] However, this view has been contested both by the manufacturers and other authors.[3,4] In a subsequent review of the use of domperidone in Parkinson's disease it was considered[5] that domperidone might produce central blockade of the therapeutic effects of levodopa if given at a high oral dosage such as 120 mg daily for prolonged periods but also noted that such high doses were rarely required to control levodopa-induced vomiting.

Domperidone was found to significantly improve anorexia, nausea, vomiting, abdominal bloating, and regurgitation in patients taking levodopa.[6] Dysphagia and constipation were unaffected; these are thought to be more likely a reflection of the disease process. Doses ranged from 50 to 120 mg daily, with most patients responding to 80 mg daily. No central effects were noted.

1. Leeser J, Bateman DN. Domperidone. *BMJ* 1985; **290:** 241.
2. Bateman DN. Domperidone. *BMJ* 1985; **290:** 1079.
3. Lake-Bakaar G, Cameron HA. Domperidone. *BMJ* 1985; **290:** 241–2.
4. Critchley P, *et al.* Domperidone. *BMJ* 1985; **290:** 788.
5. Parkes JD. Domperidone and Parkinson's disease. *Clin Neuropharmacol* 1986; **9:** 517–32.
6. Soykan I, *et al.* Effect of chronic oral domperidone therapy on gastrointestinal symptoms and gastric emptying in patients with Parkinson's disease. *Mov Disord* 1997; **12:** 952–7.

Preparations

BP 2008: Domperidone Tablets.

Proprietary Preparations (details are given in Part 3)
Arg.: Ecuamon; Euciton; Moperidona; Motilium; Peridon; ***Austral.:*** Motilium; ***Austria:*** Motilium; ***Belg.:*** Docdomperi; Domperitop; Motilium; Zilium; ***Braz.:*** Domperol†; Motilium; Peridal; ***Canad.:*** Motilium†; ***Chile:*** Donegal; Dosin; Gasdol; Idon; Restol; Siligaz; ***Cz.:*** Motilium; ***Denm.:*** Motilium; ***Fr.:*** Biperidys; Motilium; Motilyo; Peridys; ***Ger.:*** Domidon; Motilium; ***Gr.:*** Cilroton; ***Hong Kong:*** Costi; Dompeon; Doridone; Motilium; Qualidom; Rabugen; ***Hung.:*** Motilium; ***India:*** Domperi; Domperon; Domstal; Nautigo; Stopvom; Vomistop; ***Indon.:*** Costil; DOM; Domedon; Domet; Dometa; Dometic; Galflux; Gerdilium; Monell; Motilium; Novotil; Regit; Tilidon; Vometa; Vomidone; Vomistop; Vomitas; Vosedon; ***Irl.:*** Domerid; Motilium; ***Israel:*** Motilium; ***Ital.:*** Digestivo Giuliani; Fobidon†; Gastronorm; Motilium; Peridon; Permod; Permotil; Riges; Stalcare; ***Jpn:*** Nauzelin; ***Malaysia:*** Domper; Motilium; Rabugen; ***Mex.:*** Biolix; Emiken; Motilium; Seronex; ***Neth.:*** Gastrocure; Motilium; ***NZ:*** Motilium; ***Philipp.:*** Dompenyl; Gilax; Motilium; ***Port.:*** Cinet; Mogasinte; Motilium; Nausidone; Nefius; Nordonil†; Remotil; ***Rus.:*** Motilak (Мотилак); Motilium (Мотилиум); Motonium (Мотониум); Passagix (Пассажикс); ***S.Afr.:*** Motilium; Vomidon; ***Singapore:*** Dompel; Dompenyl†; Domper; Doridone; Mirax; Motilium; ***Spain:*** Motilium; ***Switz.:*** Motilium; ***Thai.:*** Dany; Dolium; Domerdon; Domidone; Domper-M; Domperdone; Donum; Mirax; Mocydone; Modomed; Molax; Moticon; Motidom; Motilium; Movelium; Ninlium; Peptomet†; Peridom-M; Pondperdone; Rabugen-M; ***Turk.:*** Motilium; ***UK:*** Motilium; Vivadone; ***Venez.:*** Agilam; Tilium; Tonun.

Multi-ingredient: ***Arg.:*** Alplax Net; Ansielix Digest; Bigetric; Bilagol; Dom-Polienzim; Euciton Complex; Euciton Reflux; Euciton Stress; Faradil Novo; Megalex; Moperidona AF; Moperidona Enzimatica; Praxis; Sidomal; Tensium Gastric; Tetralgin Novo; Vegestabil Digest; ***Belg.:*** Touristil; ***Braz.:*** Lansodom; ***India:*** Aciloc RD; Domcet; Esoz-D; Nogacid D; Okacid D; Okalan D; Pantosec D; Praize-D; Vertigil; ***UK:*** Domperamol†.

Dosmalfate *(rINN)*

Dosmalfato; Dosmalfatum; F-3616; F-3616. {μ_7-[(Diosmin heptasulfato)(7-)]}tetracontahydroxytetradecaaluminium.

Дозмальфат

$C_{28}H_{60}Al_{14}O_{71}S_7$ = 2134.9.
CAS — 122312-55-4.

Profile

Dosmalfate is a cytoprotective drug derived from diosmin (p.2304), that is used for the prevention and treatment of NSAID-associated peptic ulcer disease (p.1702) in an oral dose of 1.5 g twice daily.

Preparations

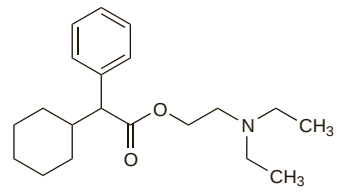
Proprietary Preparations (details are given in Part 3)
Spain: Diotul.

Drofenine Hydrochloride *(plNNM)*

Droféninе, Chlorhydrate de; Drofenini Hydrochloridum; Hexahydroadiphenine Hydrochloride; Hidrocloruro de drofenina. 2-(Diethylamino)ethyl α-phenylcyclohexaneacetate hydrochloride.

Дрофенина Гидрохлорид

$C_{20}H_{31}NO_2.HCl$ = 353.9.
CAS — 1679-76-1 (drofenine); 548-66-3 (drofenine hydrochloride).



(drofenine)

Pharmacopoeias. In *Swiss*.

Profile
Drofenine hydrochloride is an antimuscarinic available in preparations for the treatment of visceral spasms.

Preparations
Proprietary Preparations (details are given in Part 3)

Multi-ingredient: ***Arg.:*** Espasmo Cibalena; ***Austria:*** Spasmoplus; ***Belg.:*** Spasmoplus†; ***Chile:*** Espasmo Cibalgina; Espasmo Cibalgina Compuesta; ***Ger.:*** Spasmo-Cibalgin S†; ***Ital.:*** Spasmo-Cibalgina†; ***Mex.:*** Espasmo Cibalgina; ***Switz.:*** Lunadon; Spasmo-Cibalgin comp†; Spasmo-Cibalgin†.

Dronabinol *(USAN, rINN)* ⊗

Dronabinolum; NSC-134454; Δ^9-Tetrahydrocannabinol; Δ^9-THC. (6a*R*,10a*R*)-6a,7,8,10a-Tetrahydro-6,6,9-trimethyl-3-pentyl-6*H*-dibenzo[*b*,*d*]pyran-1-ol.

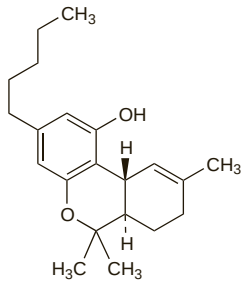
Дронабинол

$C_{21}H_{30}O_2$ = 314.5.

CAS — 1972-08-3.

ATC — A04AD10.

ATC Vet — QA04AD10.



Pharmacopoeias. In *US*.

USP 31 (Dronabinol). Store at a temperature between 8° and 15° in airtight glass containers in an inert atmosphere. Protect from light.

Adverse Effects and Precautions
As for Nabilone, p.1750. The most frequent adverse effects of dronabinol include abdominal pain, nausea and vomiting, dizziness, euphoria, paranoid reactions, and somnolence. Seizures and seizure-like activity have been reported; dronabinol should be used with caution in those with a history of seizure disorders, and therapy should be stopped if seizures occur.

Abuse. The abuse liability of dronabinol was rated as being substantially lower than that of cannabis.[1]

1. WHO. WHO expert committee on drug dependence: thirty-third report. *WHO Tech Rep Ser 915* 2003. Available at: http://libdoc.who.int/trs/WHO_TRS_915.pdf (accessed 03/07/08)

Breast feeding. US licensed product information states that dronabinol is concentrated in breast milk and recommends that it should not be used in breast-feeding mothers.

Pharmacokinetics
After oral doses dronabinol is slowly and erratically absorbed from the gastrointestinal tract; the bioavailability of an oral dose is about 10 to 20%, due to extensive first-pass metabolism. Peak plasma concentrations of dronabinol and its 11-hydroxy metabolite are achieved about 2 to 4 hours after a dose by mouth. It is widely distributed and is extensively protein bound, with a volume of distribution of about 10 litres/kg. Elimination is biphasic, with an initial half-life of about 4 hours, and a terminal half-life of about 25 to 36 hours.

Dronabinol is extensively metabolised, mainly in the liver by cytochrome P450 isoenzymes; the primary metabolite, 11-hydroxydronabinol is also active. The 11-hydroxy metabolite is converted to other, more polar and acidic compounds which are excreted in faeces via the bile, and in the urine. About 50% of an oral dose is recovered in faeces within 72 hours and 10 to 15% in urine. Many of the metabolites have relatively prolonged half-lives, and accumulation may occur with repeated dosage.

Dronabinol is distributed into breast milk and crosses the placenta.

◊ References.

1. Grotenhermen F. Pharmacokinetics and pharmacodynamics of cannabinoids. *Clin Pharmacokinet* 2003; **42:** 327–60.
2. McGilveray IJ. Pharmacokinetics of cannabinoids. *Pain Res Manag* 2005; **10:** 15A–22A.

Uses and Administration
Dronabinol, the major psychoactive constituent of cannabis (p.2274), has antiemetic properties and is used for the control of **nausea and vomiting** associated with cancer chemotherapy (p.1700) in patients who have failed to respond adequately to conventional antiemetics.

The usual initial oral dose of dronabinol is 5 mg/m^2 given 1 to 3 hours before the first dose of the antineoplastic drug with subsequent doses being given every 2 to 4 hours after chemotherapy to a maximum of 4 to 6 doses daily. If necessary, the dose may be increased by increments of 2.5 mg/m^2 to a maximum dose of 15 mg/m^2, if adverse effects permit.

Dronabinol also has appetite-stimulant effects and is used in the treatment of **anorexia** associated with weight loss in patients with AIDS. For this purpose 2.5 mg may be taken twice daily, before lunch and supper, reduced to a single 2.5-mg dose in the evening in patients who tolerate the drug poorly. If necessary, and if adverse effects permit, doses may also be increased up to 20 mg daily in divided doses.

Dronabinol is used with cannabidiol, another cannabinoid, in a buccal spray preparation as adjunctive treatment for the symptomatic relief of **neuropathic pain** in multiple sclerosis in adults; this combination is also used as adjunctive analgesic treatment in adult patients with advanced cancer and is under investigation for a number of other conditions (see under Cannabis, p.2275).

◊ General references.

1. Voth EA, Schwartz RH. Medicinal applications of delta-9-tetrahydrocannabinol and marijuana. *Ann Intern Med* 1997; **126:** 791–8.
2. Williamson EM, Evans FJ. Cannabinoids in clinical practice. *Drugs* 2000; **60:** 1303–14.
3. Tramer MR, *et al.* Cannabinoids for control of chemotherapy induced nausea and vomiting: quantitative systematic review. *BMJ* 2001; **323:** 16–21.
4. Berman JS, *et al.* Efficacy of two cannabis based medicinal extracts for relief of central neuropathic pain from brachial plexus avulsion: results of a randomised controlled trial. *Pain* 2004; **112:** 299–306.
5. Costa B. On the pharmacological properties of Delta9-tetrahydrocannabinol (THC). *Chem Biodivers* 2007; **4:** 1664–77.
6. Beaulieu P, Ware M. Reassessment of the role of cannabinoids in the management of pain. *Curr Opin Anaesthesiol* 2007; **20:** 473–7.

Alzheimer's disease. There is some suggestion[1] that dronabinol may decrease agitation in patients with Alzheimer's disease.

1. Volicer L, *et al.* Effects of dronabinol on anorexia and disturbed behavior in patients with Alzheimer's disease. *Int J Geriatr Psychiatry* 1997; **12:** 913–19.

Anorexia. Dronabinol is used for the management of anorexia in patients with HIV-associated wasting (p.858). However, although dronabinol may stimulate appetite and prevent weight loss,[1] it does not appear to produce significant weight gain, and may produce less benefit than megestrol acetate.[2] Benefits were also less than those of megestrol in patients with anorexia associated with malignant disease.[3]

1. Beal JE, *et al.* Dronabinol as a treatment for anorexia associated with weight loss in patients with AIDS. *J Pain Symptom Manage* 1995; **10:** 89–97.
2. Timpone JG, *et al.* The safety and pharmacokinetics of single-agent and combination therapy with megestrol acetate and dronabinol for the treatment of HIV wasting syndrome. *AIDS Res Hum Retroviruses* 1997; **13:** 305–15.
3. Jatoi A, *et al.* Dronabinol versus megestrol acetate versus combination therapy for cancer-associated anorexia: a North Central Cancer Treatment Group study. *J Clin Oncol* 2002; **20:** 567–73.

Multiple sclerosis. Anecdotal evidence has suggested that cannabinoids might improve symptoms in patients with multiple sclerosis (p.892); a review[1] considered evidence of effectiveness to be lacking. In a large placebo-controlled study, treatment with dronabinol or oral cannabis extract had no benefit on objective assessment of spasticity;[2] however, there were improvements in walking time, and subjective improvements in both spasticity and pain. A subsequent small controlled study found dronabinol to have a modest but clinically relevant effect on central neuropathic pain in patients with multiple sclerosis.[3]

Dronabinol is used with cannabidiol, another cannabinoid, in a buccal spray preparation for the symptomatic relief of neuropathic pain in multiple sclerosis in adults.

1. Killestein J, *et al.* Cannabinoids in multiple sclerosis: do they have a therapeutic role? *Drugs* 2004; **64:** 1–11.
2. Zajicek J, *et al.* Cannabinoids for treatment of spasticity and other symptoms related to multiple sclerosis (CAMS study): multicentre randomised placebo-controlled trial. *Lancet* 2003; **362:** 1517–26.
3. Svendsen KB, *et al.* Does the cannabinoid dronabinol reduce central pain in multiple sclerosis? Randomised double blind placebo controlled crossover trial. *BMJ* 2004; **329:** 253–7.

Tourette's syndrome. Preliminary studies[1,2] indicate that dronabinol may reduce tic severity in Tourette's syndrome (see Tics, p.954).

1. Müller-Vahl KR, *et al.* Treatment of Tourette's syndrome with Δ -tetrahydrocannabinol (THC): a randomized crossover trial. *Pharmacopsychiatry* 2002; **35:** 57–61.
2. Müller-Vahl KR, *et al.* Δ -Tetrahydrocannabinol (THC) is effective in the treatment of tics in Tourette syndrome: a 6-week randomized trial. *J Clin Psychiatry* 2003; **64:** 459–65.

Preparations
USP 31: Dronabinol Capsules.

Proprietary Preparations (details are given in Part 3)

Canad.: Marinol; ***Israel:*** Ronabin†; ***S.Afr.:*** Elevat†; ***USA:*** Marinol.

Multi-ingredient: ***Canad.:*** Sativex.

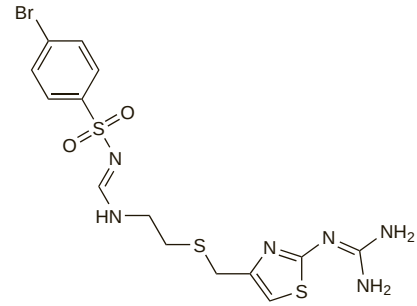
Ebrotidine *(rINN)*

Ebrotidina; Ébrotidine; Ebrotidinum. *p*-Bromo-*N*-[(*E*)-({2-[({2-[(diaminomethylene)amino]-4-thiazolyl}methyl)thio]ethyl}amino)methylene]benzenesulfonamide.

Эбротидин

$C_{14}H_{17}BrN_6O_2S_3$ = 477.4.

CAS — 100981-43-9.



Profile
Ebrotidine is a histamine H_2-antagonist with general properties similar to those of cimetidine (p.1716), but which also has cytoprotective actions. It has been used in peptic ulcer disease. Serious liver damage has been reported.

◊ References.

1. Patel SS, Wilde MI. Ebrotidine. *Drugs* 1996; **51:** 974–80.
2. Various. Ebrotidine: a new generation H -receptor antagonist and gastroprotective agent. *Arzneimittelforschung* 1997; **47:** 427–590.
3. Andrade RJ, *et al.* Acute liver injury associated with the use of ebrotidine, a new H -receptor antagonist. *J Hepatol* 1999; **31:** 641–6.

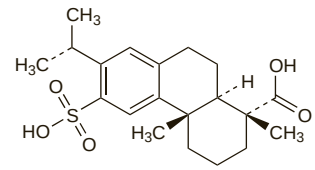
Ecabet Sodium *(rINNM)*

Ecabet sódico; Écabet Sodique; Natrii Ecabetum; 12-Sulphodehydroabietic Acid, Monosodium Salt; TA-2711. 13-Isopropyl-12-sulphopodocarpa-8,11,13-trien-15-oic acid pentahydrate, sodium salt.

Экабет Натрий

$C_{20}H_{27}NaO_5S,5H_2O$ = 492.6.

CAS — 33159-27-2 (ecabet); 86408-72-2 (ecabet sodium).



(ecabet)

Profile
Ecabet sodium is a cytoprotective drug used in the treatment of peptic ulcer disease (p.1702). The suggested oral dose is 1 g of ecabet sodium twice daily.

It is also under investigation as eye drops in the management of dry eye.

◊ References.

1. Murata H, *et al.* Combination therapy of ecabet sodium and cimetidine compared with cimetidine alone for gastric ulcer: prospective randomized multicenter study. *J Gastroenterol Hepatol* 2003; **18:** 1029–33.
2. Lee JH, *et al.* Efficacy and safety of ecabet sodium on functional dyspepsia: a prospective, double-blinded, randomized, multicenter controlled trial. *World J Gastroenterol* 2006; **12:** 2756–61.

Administration. The use of ecabet sodium as a rectal enema has been investigated in patients with ulcerative colitis.[1,2]

1. Kono T, *et al.* Effect of ecabet sodium enema on mildly to moderately active ulcerative proctosigmoiditis: an open-label study. *Am J Gastroenterol* 2001; **96:** 793–7.
2. Iizuka M, *et al.* Efficacy of ecabet sodium enema on steroid resistant or steroid dependent ulcerative colitis. *Gut* 2006; **55:** 1523.

Preparations
Proprietary Preparations (details are given in Part 3)

Jpn: Gastrom.