commercially available tablets found them to be stable for at least 90 days when stored at 3 to 5° and at 23 to 25°.

1. Johnson CE, *et al.* Stability of dolasetron in two oral liquid vehicles. *Am J Health-Syst Pharm* 2003; **60:** 2242–4.

Adverse Effects and Precautions

As for Ondansetron, p.1757. Diarrhoea, anorexia, and abdominal pain may also occur. Various ECG changes have been noted with dolasetron. Dolasetron should be used with caution in patients who have, or may develop, prolongation of the QT interval or other alterations in cardiac conduction intervals, and in those with electrolyte imbalances. Other adverse effects include dyspepsia, flatulence, taste disturbances, fever, chills or shivering, sleep disorders, fatigue, and drowsiness. There have been rare reports of intestinal obstruction, pancreatitis, jaundice, seizures, bronchospasm, cardiac arrhythmias, and oedema. Local reactions may occur on intravenous use. No dosage reduction is considered necessary in renal or hepatic impairment, despite possible reductions in clearance.

Effects on the cardiovascular system. For a discussion of the effects of 5-HT_3 antagonists on the cardiovascular system, see under Ondansetron, p.1757.

Phlebitis. Venous irritation has been reported after intravenous use of dolasetron; diluting subsequent doses with sodium chloride 0.9% and infusing it more slowly markedly reduced the frequency of phlebitis.[1]

1. Oshiro MM. Dolasetron-associated venous irritation. *Am J Health-Syst Pharm* 2000; **57:** 1533–4.

Interactions

Plasma concentrations of hydrodolasetron, the active metabolite of dolasetron, are increased by cimetidine and atenolol and decreased by rifampicin. Dolasetron should be used with caution in patients taking drugs that prolong the QT interval, including those likely to cause electrolyte disturbances (see also p.1757).

Pharmacokinetics

Dolasetron given orally or intravenously is rapidly converted to the active metabolite hydrodolasetron by carbonyl reductase, a ubiquitous enzyme. Peak plasma concentrations of hydrodolasetron occur 1 hour after oral, and 0.6 hours after intravenous, doses of dolasetron. The apparent oral bioavailability of dolasetron determined as hydrodolasetron is about 75%. It has a mean elimination half-life of about 7 to 8 hours.

Hydrodolasetron is partially metabolised by the cytochrome P450 isoenzymes CYP2D6 and CYP3A and about 50 to 60% is eliminated unchanged in the urine. Two thirds of a dose of dolasetron is recovered in the urine and one third in the faeces.

Clearance of hydrodolasetron is increased in children, but is not altered in the elderly. Clearance is reduced in severe hepatic impairment (Child-Pugh category B or C) and in severe renal impairment (creatinine clearance less than 10 mL/min) after oral use. After intravenous use, clearance is reduced in severe renal impairment but apparently unchanged in severe hepatic impairment.

◊ References.

1. Lerman J, *et al.* Pharmacokinetics of the active metabolite (MDL 74,156) of dolasetron mesylate after oral or intravenous administration to anesthetized children. *Clin Pharmacol Ther* 1996; **60:** 485–92.
2. Dempsey E, *et al.* Pharmacokinetics of single intravenous and oral doses of dolasetron mesylate in healthy elderly volunteers. *J Clin Pharmacol* 1996; **36:** 903–10.
3. Stubbs K, *et al.* Pharmacokinetics of dolasetron after oral and intravenous administration of dolasetron mesylate in healthy volunteers and patients with hepatic dysfunction. *J Clin Pharmacol* 1997; **37:** 926–36.
4. Dimmitt DC, *et al.* Pharmacokinetics of oral and intravenous dolasetron mesylate in patients with renal impairment. *J Clin Pharmacol* 1998; **38:** 798–806.
5. Dimmitt DC, *et al.* Effect of infusion rate on the pharmacokinetics and tolerance of intravenous dolasetron mesylate. *Ann Pharmacother* 1998; **32:** 39–44.

Uses and Administration

Dolasetron is a 5-HT_3 antagonist with antiemetic actions similar to those of ondansetron (see p.1757). It is used as the mesilate in the prevention of nausea and vomiting (p.1700) associated with chemotherapy, and in the prevention and treatment of postoperative nausea and vomiting.

For *prevention* of acute **nausea and vomiting** associated with **chemotherapy** dolasetron mesilate may be given orally in a dose of 100 mg (in the USA) or 200 mg (in most other countries including the UK) within 1 hour before treatment. Alternatively, it may be given in a dose of 1.8 mg/kg, or 100 mg, by intravenous injection at a rate of up to 100 mg over 30 seconds about 30 minutes before chemotherapy; the same dose may be diluted to 50 mL with a suitable infusion solution and given intravenously over up to 15 minutes. To protect against delayed emesis, a further 200-mg dose may be given orally once daily; in Europe and the UK dolasetron may not normally be given for more than 4 consecutive days per chemotherapy cycle although some countries permit use for up to 7 days.

When given for the *prevention* of **postoperative** nausea and vomiting the recommended dose is usually 50 mg of dolasetron mesilate orally before induction of anaesthesia or 12.5 mg intravenously at the end of anaesthesia. In the USA, it is given as a 100-mg oral dose within 2 hours before surgery, or 12.5 mg intravenously about 15 minutes before the end of anaesthesia. The same intravenous dose may be given for the *treatment* of postoperative nausea and vomiting.

The use of dolasetron mesilate in **children** is licensed in some countries, including the USA. However, others have not licensed such use and in the UK it is contraindicated in children and adolescents under 18 years of age because they may be at increased risk of acute changes in the QT interval, and there have been reports of cardiac conduction disorders, cardiac arrest, and myocardial infarction in children treated with dolasetron. In the USA, children over 2 years of age may be given dolasetron mesilate 1.8 mg/kg orally (within 1 hour before chemotherapy) or intravenously (about 30 minutes before chemotherapy), up to a maximum dose of 100 mg, to prevent acute chemotherapy-induced nausea and vomiting. For prevention of postoperative nausea and vomiting, 1.2 mg/kg by mouth, up to a maximum of 100 mg, may be given within 2 hours before surgery; or 350 micrograms/kg, up to a maximum of 12.5 mg, may be given intravenously 15 minutes before the end of anaesthesia. The same intravenous dose may be given to treat established postoperative nausea and vomiting.

◊ Reviews.

1. Balfour JA, Goa KL. Dolasetron: a review of its pharmacology and therapeutic potential in the management of nausea and vomiting induced by chemotherapy, radiotherapy or surgery. *Drugs* 1997; **54:** 273–98.
2. Anonymous. Dolasetron for prevention of nausea and vomiting due to cancer chemotherapy. *Med Lett Drug Ther* 1998; **40:** 53–4.

Pruritus. Dolasetron and other 5-HT_3 antagonists have been investigated for the management of pruritus (see under Ondansetron, p.1758).

Preparations

USP 31: Dolasetron Mesylate Injection; Dolasetron Mesylate Oral Solution; Dolasetron Mesylate Oral Suspension; Dolasetron Mesylate Tablets.

Proprietary Preparations (details are given in Part 3)

Arg.: Anzemet†; ***Austral.:*** Anzemet; ***Austria:*** Anzemet; ***Braz.:*** Anzemet; ***Canad.:*** Anzemet; ***Cz.:*** Anzemet†; ***Fin.:*** Anzemet†; ***Fr.:*** Anzemet; ***Ger.:*** Anemet; ***Gr.:*** Anzemet; ***Hung.:*** Anemet†; ***Ital.:*** Anzemet; ***Mex.:*** Anzemet; ***Neth.:*** Anzemet; ***S.Afr.:*** Zamanon; ***Switz.:*** Anzemet; ***UK:*** Anzemet; ***USA:*** Anzemet; ***Venez.:*** Anzemet.

Domperidone *(BAN, USAN, rINN)*

Domperidon; Domperidona; Domperidonas; Dompéridone; Domperidoni; Domperidonum; R-33812. 5-Chloro-1-{1-[3-(2-oxobenzimidazolin-1-yl)propyl]-4-piperidyl}benzimidazolin-2-one.

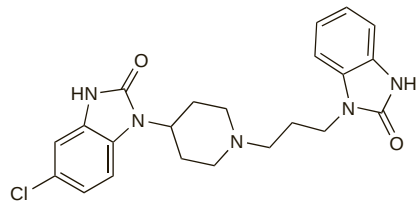
Домперидон

$C_{22}H_{24}ClN_5O_2$ = 425.9.

CAS — 57808-66-9.

ATC — A03FA03.

ATC Vet — QA03FA03.



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

Ph. Eur. 6.2 (Domperidone). A white or almost white powder. Practically insoluble in water; slightly soluble in alcohol and in methyl alcohol; soluble in dimethylformamide. Protect from light.

Domperidone Maleate *(BANM, rINNM)*

Dompéridone, maléate de; Domperidoni maleas; Domperidon-imaleaatti; Domperidonmaleat; Domperidon-maleát; Domperidon-maleinát; Domperidono maleatas; Maleato de domperidona.

Домперидона Малеат

$C_{22}H_{24}ClN_5O_2.C_4H_4O_4$ = 542.0.

CAS — 99497-03-7.

ATC — A03FA03.

ATC Vet — QA03FA03.

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

Ph. Eur. 6.2 (Domperidone Maleate). A white or almost white powder; it exhibits polymorphism. Very slightly soluble in water and in alcohol; sparingly soluble in dimethylformamide; slightly soluble in methyl alcohol. Protect from light.

Adverse Effects

Plasma-prolactin concentrations may be increased, which may lead to galactorrhoea or gynaecomastia. There have been reports of reduced libido, and rashes and other allergic reactions. Domperidone does not readily cross the blood-brain barrier and the incidence of central effects such as extrapyramidal reactions or drowsiness may be lower than with metoclopramide (p.1748); however, there have been reports of dystonic reactions.

Domperidone by injection has been associated with convulsions, arrhythmias, and cardiac arrest. Fatalities have restricted use by this route.

Effects on the cardiovascular system. Sudden death has occurred in cancer patients given domperidone *intravenously* in high doses.[1-3] Four cancer patients experienced cardiac arrest after high intravenous doses[4] and 2 of 4 similar patients had ventricular arrhythmias.[5] After such reports the injection has been withdrawn from general use in many countries, including the UK.

Prolongation of the QT interval has been reported in an infant given *oral* domperidone, with normalisation after the drug was stopped.[6]

1. Joss RA, *et al.* Sudden death in cancer patient on high-dose domperidone. *Lancet* 1982; **i:** 1019.
2. Giaccone G, *et al.* Two sudden deaths during prophylactic antiemetic treatment with high doses of domperidone and methylprednisolone. *Lancet* 1984; **ii:** 1336–7.
3. Weaving A, *et al.* Seizures after antiemetic treatment with high dose domperidone: report of four cases. *BMJ* 1984; **288:** 1728.
4. Roussak JB, *et al.* Cardiac arrest after treatment with intravenous domperidone. *BMJ* 1984; **289:** 1579.
5. Osborne RJ, *et al.* Cardiotoxicity of intravenous domperidone. *Lancet* 1985; **ii:** 385.
6. Rocha CMG, Barbosa MM. QT interval prolongation associated with the oral use of domperidone in an infant. *Pediatr Cardiol* 2005; **26:** 720–3.

Effects on the endocrine system. There have been reports of galactorrhoea with gynaecomastia[1] or mastalgia[2,3] generally associated with raised serum-prolactin concentrations. Gynaecomastia without galactorrhoea has also been reported.[4]

1. Van der Steen M, *et al.* Gynaecomastia in a male infant given domperidone. *Lancet* 1982; **ii:** 884–5.
2. Cann PA, *et al.* Galactorrhoea as side effect of domperidone. *BMJ* 1983; **286:** 1395–6.
3. Cann PA, *et al.* Oral domperidone: double blind comparison with placebo in irritable bowel syndrome. *Gut* 1983; **24:** 1135–40.
4. Keating JP, Rees M. Gynaecomastia after long-term administration of domperidone. *Postgrad Med J* 1991; **67:** 401–2.

Extrapyramidal effects. There are reports of extrapyramidal symptoms,[1,2] including acute dystonic reactions[3] and neuroleptic malignant syndrome[4] in individual patients given domperidone.

1. Sol P, *et al.* Extrapyramidal reactions due to domperidone. *Lancet* 1980; **ii:** 802.

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

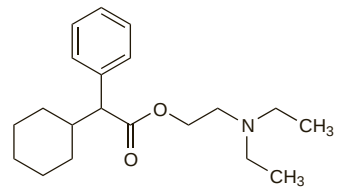
Proprietary Preparations (details are given in Part 3)
Spain: Diotul.

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)