6. Morgner A, *et al.* Esomeprazole: prevention and treatment of NSAID-induced symptoms and ulcers. *Expert Opin Pharmacother* 2007; **8:** 975–88.
7. Blandizzi C, *et al.* Clinical efficacy of esomeprazole in the prevention and healing of gastrointestinal toxicity associated with NSAIDs in elderly patients. *Drugs Aging* 2008; **25:** 197–208.

Administration. *In-vitro* studies found that almost the entire contents of an esomeprazole capsule is deliverable through small calibre and standard sizes of nasogastric and gastrostomy tubes.[1,2]

1. White CM, *et al.* Delivery of esomeprazole magnesium enteric-coated pellets through small caliber and standard nasogastric tubes and gastrostomy tubes in vitro. *Am J Health-Syst Pharm* 2002; **59:** 2085–8.
2. Shah SA, *et al.* Delivery of esomeprazole magnesium through nasogastric and gastrostomy tubes using an oral liquid vehicle as a suspending agent in vitro. *Am J Health-Syst Pharm* 2006; **63:** 1882–7.

Administration in children. UK licensed product information allows for the use of adult doses of esomeprazole (see Uses and Administration, above) in children over 12 years.

In the USA licensed doses, which may be given once daily for up to 8 weeks for the treatment of gastro-oesophageal reflux in children, are:

- 1 to 11 years: 10 mg
- 12 to 17 years: 20 or 40 mg

For healing erosive oesophagitis in children, the following doses based on body-weight are licensed in the USA to be given once daily for up to 8 weeks:

- less than 20 kg: 10 mg
- 20 kg or over: 10 or 20 mg

Administration in hepatic impairment. No dosage adjustment of esomeprazole is considered necessary for patients with mild to moderate hepatic impairment (Child-Pugh Classes A and B, respectively). For patients with severe hepatic impairment (Child-Pugh Class C), a daily dose of 20 mg should not be exceeded.

Administration in renal impairment. Although no dosage adjustment is considered necessary in patients with renal impairment, UK licensed product information advises caution in those with severe renal impairment, as experience in these patients is limited.

Preparations

Proprietary Preparations (details are given in Part 3)
Arg.: Esomax; Nexium; ***Austral.:*** Nexium; ***Austria:*** Nexium; ***Belg.:*** Nexiam; ***Braz.:*** Nexium; ***Canad.:*** Nexium; ***Chile:*** Nexium; Ulcratex; ***Cz.:*** Nexium; ***Denm.:*** Nexium; ***Fin.:*** Nexium; ***Fr.:*** Inexium; ***Ger.:*** Nexium; ***Gr.:*** Nexium; ***Hong Kong:*** Nexium; ***Hung.:*** Nexium; ***India:*** Esomac; Esoz; Sompraz; ***Indon.:*** Nexium; ***Irl.:*** Nexium; ***Israel:*** Nexium; ***Ital.:*** Axagon; Esopral; Lucen; Nexium; ***Malaysia:*** Nexium; ***Mex.:*** Nexium; ***Neth.:*** Esopral; Nexium; ***Norw.:*** Nexium; ***Philipp.:*** Nexium; ***Pol.:*** Nexium; ***Port.:*** Nexium; ***Rus.:*** Nexium (Нексиум); ***S.Afr.:*** Nexiam; ***Singapore:*** Nexium; ***Spain:*** Axiago; Nexium; ***Swed.:*** Nexium; ***Switz.:*** Nexium; ***Thai.:*** Nexium; ***Turk.:*** Nexium; ***UK:*** Nexium; ***USA:*** Nexium; ***Venez.:*** Esoz; Nexium.

Multi-ingredient: ***Austral.:*** Nexium Hp; ***India:*** Esoz-D; ***Swed.:*** Nexium Hp.

Euonymus

Evónimo; Fusain Noir Pourpré; Spindle Tree Bark; Wahoo Bark.

Бересклетова Кора

Profile

Euonymus is the dried root-bark of *Euonymus atropurpureus* (=*Evonymus atropurpurea*) (Celastraceae). It is reported to have laxative, choleretic, and diuretic activity.

Preparations

Proprietary Preparations (details are given in Part 3)
Multi-ingredient: ***Fr.:*** Jecopeptol; ***UK:*** Acidosis; GB Tablets; Indigestion Mixture.

Famotidine *(BAN, USAN, rINN)*

Famotidiini; Famotidin; Famotidina; Famotidinas; Famotidinum; L-643341; MK-208; YM-11170. 3-[2-(Diaminomethyleneamino)-thiazol-4-ylmethylthio]-*N*-sulphamoylpropionamidine.

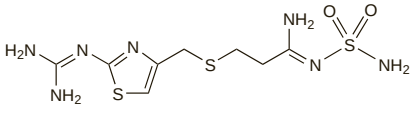
Фамотидин

$C_8H_{15}N_7O_2S_3$ = 337.4.

CAS — 76824-35-6.

ATC — A02BA03.

ATC Vet — QA02BA03.



Pharmacopoeias. In *Chin.*, *Eur.* (see p.vii), *Jpn*, and *US.*

Ph. Eur. 6.2 (Famotidine). A white or yellowish-white, crystalline powder or crystals. It exhibits polymorphism. Very slightly soluble in water and in dehydrated alcohol; freely soluble in glacial acetic acid; practically insoluble in ethyl acetate. It dissolves in dilute mineral acids. Protect from light.

USP 31 (Famotidine). A white to pale yellowish-white crystalline powder. Very slightly soluble in water; practically insoluble in alcohol, in acetone, in chloroform, in ether, and in ethyl acetate; freely soluble in dimethylformamide and in glacial acetic acid; slightly soluble in methyl alcohol. Protect from light.

Stability. References.

1. Quercia RA, *et al.* Stability of famotidine in an extemporaneously prepared oral liquid. *Am J Hosp Pharm* 1993; **50:** 691–3.
2. Dentinger PJ, *et al.* Stability of famotidine in an extemporaneously compounded oral liquid. *Am J Health-Syst Pharm* 2000; 1340–2.

Adverse Effects

As for Cimetidine, p.1716. Unlike cimetidine, famotidine is reported to have little or no anti-androgenic effect, although there are isolated reports of gynaecomastia and impotence.

◊ General references.

1. Howden CW, Tytgat GNJ. The tolerability and safety profile of famotidine. *Clin Ther* 1996; **18:** 36–54.

Effects on the blood. For reports of blood dyscrasias, some serious, occurring with famotidine, see under Cimetidine p.1717.

Effects on the cardiovascular system. Famotidine 40 mg daily by mouth reduced cardiac output and stroke volume, compared with placebo, cimetidine, or ranitidine in healthy subjects.[1] Similar effects seen in another study[2] were delayed by pretreatment with ranitidine. However, other workers have found that oral famotidine had no effect on exercise capacity or left ventricular systolic function in healthy subjects,[3] and that famotidine 20 mg intravenously had no effect on any of the haemodynamic parameters measured in 11 critically ill patients.[4] As with other H_2-antagonists (p.1717), bradycardia and AV block has been reported with famotidine,[5] as has a case of QT prolongation.[6]

1. Hinrichsen H, *et al.* Hemodynamic effects of different H -receptor antagonists. *Clin Pharmacol Ther* 1990; **48:** 302–8.
2. Mescheder A, *et al.* Changes in the effects of nizatidine and famotidine on cardiac performance after pretreatment with ranitidine. *Eur J Clin Pharmacol* 1993; **45:** 151–6.
3. Hillermann DE, *et al.* Impact of chronic oral H -antagonist therapy of left ventricular systolic function and exercise capacity. *J Clin Pharmacol* 1992; **32:** 1033–7.
4. Heiselman DE, *et al.* Hemodynamic status during famotidine infusion. *DICP Ann Pharmacother* 1990; **24:** 1163–5.
5. Schoenwald PK, *et al.* Complete atrioventricular block and cardiac arrest following intravenous famotidine administration. *Anesthesiology* 1999; **90:** 623–6.
6. Endo T, *et al.* Famotidine and acquired long QT syndrome. *Am J Med* 2000; **108:** 438–9.

Effects on the endocrine system. Hyperprolactinaemia and breast engorgement occurred in a woman during the fourth month of treatment with famotidine 80 mg daily;[1] she had mistakenly been given twice the usual maximum dose. Recovery occurred when famotidine was withdrawn. Transient hyperprolactinaemia and galactorrhoea have also been reported in a woman after standard doses (40 mg daily) of famotidine.[2] There have been a few instances of impotence.[3]

1. Delpre G, *et al.* Hyperprolactinaemia during famotidine therapy. *Lancet* 1993; **342:** 868.
2. Güven K. Hyperprolactinemia and galactorrhea with standard-dose famotidine therapy. *Ann Pharmacother* 1995; **29:** 788.
3. Kassianos GC. Impotence and nizatidine. *Lancet* 1989; **i:** 963.

Effects on the kidneys. For mention of acute interstitial nephritis associated with H_2-antagonists, including famotidine, see under Cimetidine, p.1717.

Effects on the liver. Mixed hepatocellular jaundice[1] and acute hepatitis[2] have been associated with use of famotidine; in the latter case hepatitis later recurred when the patient was given cimetidine.

1. Ament PW, *et al.* Famotidine-induced mixed hepatocellular jaundice. *Ann Pharmacother* 1994; **28:** 40–2.
2. Hashimoto F, *et al.* Hepatitis following treatments with famotidine and then cimetidine. *Ann Pharmacother* 1994; **28:** 37–9.

Effects on the nervous system. Similarly to other H_2-antagonists (p.1717), CNS reactions have occurred with famotidine, particularly in the elderly and those with renal failure.[1-3] In one report,[1] convulsions and mental deterioration in 2 elderly patients with renal failure were associated with grossly elevated plasma and CSF concentrations of the drug; symptoms resolved within 3 days of withdrawing famotidine. In another elderly patient with renal impairment, delirium was associated with use of famotidine but did not occur with cimetidine.[4]

1. Yoshimoto K, *et al.* Famotidine-associated central nervous system reactions and plasma and cerebrospinal drug concentrations in neurosurgical patients with renal failure. *Clin Pharmacol Ther* 1994; **55:** 693–700.
2. Catalano G, *et al.* Famotidine-associated delirium: a series of six cases. *Psychosomatics* 1996; **37:** 349–55.
3. Odeh M, Oliven A. Central nervous system reactions associated with famotidine: report of five cases. *J Clin Gastroenterol* 1998; **27:** 253–4.
4. Yuan R-Y, *et al.* Delirium following a switch from cimetidine to famotidine. *Ann Pharmacother* 2001; **35:** 1045–8.

Effects on the skin. Toxic epidermal necrolysis or erythema multiforme have been reported after use of famotidine;[1,2] the second patient had a recurrence with cimetidine.

1. Brunner M, *et al.* Toxic epidermal necrolysis (Lyell syndrome) following famotidine administration. *Br J Dermatol* 1995; **133:** 814–15.
2. Horiuchi Y, Ikezawa K. Famotidine-induced erythema multiforme: cross-sensitivity with cimetidine. *Ann Intern Med* 1999; **131:** 795.

Fever. Famotidine 20 mg intravenously every 12 hours was associated with hyperpyrexia in a patient with facial and cranial trauma.[1] Rectal temperature in the 24 hours after starting famotidine was 40.5° and remained elevated for the 5 days of famotidine treatment, despite use of antipyretics. Withdrawal of famotidine resulted in a return to normal temperature within 24 hours.

1. Norwood J, *et al.* Famotidine and hyperpyrexia. *Ann Intern Med* 1990; **112:** 632.

Precautions

As for Cimetidine, p.1718.

Hepatic impairment. For a report of increased resistance to H_2-antagonists in patients with liver cirrhosis, see Ranitidine, p.1766.

Renal impairment. In patients with renal impairment, famotidine clearance is reduced and the elimination half-life increased, resulting in increased serum-drug concentrations and in some cases clinical sequelae (see Effects on the Nervous System, above). The half-life of famotidine in healthy subjects is about 3 hours, but in patients with a creatinine clearance less than 38 mL/minute[1] or those with end-stage renal disease[2] it has been reported to be 19.3 hours and 27.2 hours respectively. A 50% reduction in the dose of famotidine in patients with renal impairment has therefore been recommended. However, it may not be sufficient to adjust the dose only on the basis of creatinine clearance since famotidine is partly eliminated by tubular secretion, which may also be diminished.[1] A chart review[3] in patients with end-stage renal disease suggested that most patients would tolerate a dose of 20 mg daily, although a few might need further adjustment to prevent mental status changes.

Haemodialysis does not effectively remove famotidine from the systemic circulation. The proportion removed depends on the type of membrane used; with a high flux polysulfone membrane about 16% is reported to be removed, but only 6% with a cuprophan membrane.[2] Continuous ambulatory peritoneal dialysis is reported to remove about 5% of a dose.[2] Continuous haemofiltration may remove about 16% of a dose;[2] intermittent haemofiltration is reported to remove about 4%[4] or 8%.[2] Dosage supplements of famotidine are not required during or after dialysis or filtration procedures.

1. Inotsume N, *et al.* Pharmacokinetics of famotidine in elderly patients with and without renal insufficiency and in healthy young volunteers. *Eur J Clin Pharmacol* 1989; **36:** 517–20.
2. Gladziwa U, *et al.* Pharmacokinetics and dynamics of famotidine in patients with renal failure. *Br J Clin Pharmacol* 1988; **26:** 315–21.
3. Redmond AM, *et al.* Use of famotidine in adult patients with end-stage renal disease: assessment of dosing and mental status changes. *Am J Med Sci* 2005; **330:** 8–10.
4. Saima S, *et al.* Hemofiltrability of H -receptor antagonist, famotidine, in renal failure patients. *J Clin Pharmacol* 1990; **30:** 159–62.

Interactions

Unlike cimetidine (see p.1718) famotidine does not inhibit cytochrome P450, and therefore is considered to have little effect on the metabolism of other drugs. However, like other H_2-antagonists its effects on gastric pH may affect the absorption of some other drugs.

Antacids. Giving famotidine 40 mg with a 10-mL dose of antacid containing 800 mg *aluminium hydroxide* with 800 mg *magnesium hydroxide*,[1] resulted in a decrease in the bioavailability of famotidine that was considered clinically insignificant. Giving famotidine with a 30-mL dose of the same antacid resulted in a greater reduction in the absorption of famotidine from the gastrointestinal tract, but the interaction could be minimised by separating ingestion by 2 hours.[2]

1. Lin JH, *et al.* Effects of antacids and food on absorption of famotidine. *Br J Clin Pharmacol* 1987; **24:** 551–3.
2. Barzaghi N, *et al.* Impaired bioavailability of famotidine given concurrently with a potent antacid. *J Clin Pharmacol* 1989; **29:** 670–2.

Probenecid. Probenecid in a total dose of 1500 mg had a significant effect on the pharmacokinetics of famotidine 20 mg in 8 healthy subjects.[1] The maximum serum concentration of famotidine and the area under the concentration/time curve were significantly increased and renal clearance significantly reduced.

These effects were explained by inhibition of the renal tubular secretion of famotidine by probenecid.

1. Inotsume N, *et al.* The inhibitory effect of probenecid on renal excretion of famotidine in young, healthy volunteers. *J Clin Pharmacol* 1990; **30:** 50–6.

Theophylline. Although famotidine is considered not to interfere with the metabolism of other drugs there is a report of a clinically significant interaction with theophylline—see p.1145.

Pharmacokinetics

Famotidine is readily but incompletely absorbed from the gastrointestinal tract with peak concentrations in plasma occurring 1 to 3 hours after oral doses. The bioavailability of oral famotidine is about 40 to 45% and is not significantly affected by the presence of food.

The elimination half-life from plasma is reported to be about 3 hours and is prolonged in renal impairment. Famotidine is weakly bound, about 15 to 20%, to plasma proteins. A small proportion of famotidine is metabolised in the liver to famotidine *S*-oxide. About 25 to 30% of an oral dose, and 65 to 70% of an intravenous dose, is excreted unchanged in the urine in 24 hours, primarily by active tubular secretion. Famotidine is also found in breast milk.

◊ Reviews.

1. Echizen H, Ishizaki T. Clinical pharmacokinetics of famotidine. *Clin Pharmacokinet* 1991; **21:** 178–94.

Children. Famotidine 300 micrograms/kg intravenously was given to 10 children aged 2 to 7 years, after cardiac surgery and before extubation, to prevent aspiration.[1] This dose (equivalent to about 20 mg in adults) induced a rise in the intragastric pH within 1 hour of being given and the pH remained above 3.5 for about 9 hours. The mean elimination half-life was 3.3 hours, similar to the value in healthy adults and it was considered that doses in children need therefore only be adjusted according to bodyweight and renal function. This conclusion was supported by a review of 8 studies in children over 1 year of age.[2] Conversely, in infants aged 5 to 19 days, the mean elimination half-life was prolonged (10.5 hours) secondary to reduced renal clearance.[3] This was confirmed by another study,[4] which indicated that reduced clearance was found in infants under 3 months of age, but that pharmacokinetics in older infants were similar to those previously reported for children and adults.

1. Kraus G, *et al.* Famotidine: pharmacokinetic properties and suppression of acid secretion in paediatric patients following cardiac surgery. *Clin Pharmacokinet* 1990; **18:** 77–81.
2. James LP, Kearns GL. Pharmacokinetics and pharmacodynamics of famotidine in paediatric patients. *Clin Pharmacokinet* 1996; **31:** 103–10.
3. James LP, *et al.* Pharmacokinetics and pharmacodynamics of famotidine in infants. *J Clin Pharmacol* 1998; **38:** 1089–95. Correction. *ibid.* 2000; **40:** 1298.
4. Wenning LA, *et al.* Pharmacokinetics of famotidine in infants. *Clin Pharmacokinet* 2005; **44:** 395–406.

Distribution into breast milk. The peak concentration of famotidine in breast milk, which occurred in 8 women 6 hours after an oral dose of 40 mg, was similar to the peak plasma concentration which occurred 2 hours after the dose.[1]

1. Courtney TP, *et al.* Excretion of famotidine in breast milk. *Br J Clin Pharmacol* 1988; **26:** 639P.

Enterohepatic recirculation. Some individuals have a second peak in the plasma concentration of famotidine, which could be due to enterohepatic recirculation. However, a maximum of 0.43% of a dose of famotidine was excreted in the bile of 2 patients following single doses of 20 mg intravenously or 40 mg by mouth indicating that significant recirculation had not occurred.[1]

1. Klotz U, Walker S. Biliary excretion of H -receptor antagonists. *Eur J Clin Pharmacol* 1990; **39:** 91–2.

Uses and Administration

Famotidine is a histamine H_2-antagonist with actions and uses similar to those of cimetidine (see p.1719).

Famotidine may be given orally or intravenously.

In the management of benign **gastric** and **duodenal ulceration** (p.1702) the dose is 40 mg daily orally at bedtime, for 4 to 8 weeks. A dose of 20 mg twice daily has also been given. A maintenance dose of 20 mg at bedtime may be given to prevent recurrence of duodenal ulceration. In **gastro-oesophageal reflux disease** (p.1696) the recommended oral dose is 20 mg twice daily for 6 to 12 weeks, or up to 40 mg twice daily if there is oesophageal ulceration. A maintenance dose of 20 mg twice daily may be given to prevent recurrence. For the short-term symptomatic relief of heartburn or non-ulcer **dyspepsia** (p.1695) a dose of 10 mg up to twice daily is suggested. In the **Zollinger-Ellison syndrome** (p.1704) the initial oral dose is 20 mg every 6 hours, increased as necessary; doses up to 800 mg daily have been used.

The usual dose of famotidine by the intravenous route is 20 mg and may be given by injection over at least 2 minutes or as an infusion over 15 to 30 minutes; the dose may be repeated every 12 hours.

Doses of famotidine should be reduced in patients with renal impairment (see below).

Administration. Although famotidine is most usually given as a film-coated tablet, an alternative wafer formulation, designed to dissolve on the tongue without the need for water, has also been developed.[1]

Parenteral formulations of famotidine are also available in some countries. Although licensed product information recommends that intravenous injections be given over at least 2 minutes, a study that compared rapid intravenous injection (over up to 1 minute) with slow intravenous infusion found both to be safe.[2] Continuous infusion has however been reported by others[3] to be more effective in the prevention of stress ulceration than bolus injection.

1. Schwartz JI, *et al.* Novel oral medication delivery system for famotidine. *J Clin Pharmacol* 1995; **35:** 362–7.
2. Fish DN. Safety and cost of rapid iv injection of famotidine in critically ill patients. *Am J Health-Syst Pharm* 1995; **52:** 1889–94.
3. Baghaie AA, *et al.* Comparison of the effect of intermittent administration and continuous infusion of famotidine on gastric pH in critically ill patients: results of a prospective, randomized, crossover study. *Crit Care Med* 1995; **23:** 687–91.

Administration in renal impairment. The dosage of famotidine should be reduced in patients with renal impairment. In the UK, a 50% reduction is suggested for patients whose creatinine clearance is less than 10 mL/minute; in the USA this reduction is recommended in all those with creatinine clearance less than 50 mL/minute. Alternatively, the dosage interval may be prolonged to 36 to 48 hours.

Immunomodulation. *MALIGNANT NEOPLASMS.* References[1-3] to the use of adjuvant famotidine in patients with malignant neoplasms, including use with interleukin-2 infusions.[2,3]

1. Parshad R, *et al.* Effect of preoperative short course famotidine on TILs and survival in breast cancer. *Indian J Cancer* 2005; **42:** 185–90.
2. Quan WD, *et al.* Continuous infusion interleukin-2 and famotidine in metastatic kidney cancer. *Cancer Biother Radiopharm* 2006; **21:** 515–19.
3. Quan WD, *et al.* Continuous infusion interleukin-2 and intravenous famotidine in metastatic melanoma. *Cancer Biother Radiopharm* 2006; **21:** 607–12.

Schizophrenia. There are reports of improvement in schizophrenic symptoms (p.955) in patients given famotidine.[1-4]

1. Kaminsky R, *et al.* Effect of famotidine on deficit symptoms of schizophrenia. *Lancet* 1990; **335:** 1351–2.
2. Rosse RB, *et al.* Famotidine adjunctive pharmacotherapy of schizophrenia: a case report. *Clin Neuropharmacol* 1995; **18:** 369–74.
3. Rosse RB, *et al.* An open-label study of the therapeutic efficacy of high-dose famotidine adjuvant pharmacotherapy in schizophrenia: preliminary evidence for treatment efficacy. *Clin Neuropharmacol* 1996; **19:** 341–8.
4. Martinez MC. Famotidine in the management of schizophrenia. *Ann Pharmacother* 1999; **33:** 742–7.

Preparations

BP 2008: Famotidine Tablets;
USP 31: Famotidine for Oral Suspension; Famotidine Injection; Famotidine Tablets.

Proprietary Preparations (details are given in Part 3)
Arg.: Ulcelac; ***Austral.:*** Amfamox†; Ausfam; Famohexal; Pamacid; Pepcid†; Pepcidine; Pepzan; ***Austria:*** Eradix; Famohexal; Famosin; Pepcid; Sodexx Famotidine; Tetacid; Ulcusan; ***Belg.:*** Pepcidine†; ***Braz.:*** Famodine†; Famoset; Famotid; Famotil†; Famox; Famoxil†; ***Canad.:*** Acid Control; Acid Halt; Maalox H Acid Controller; Pepcid; Peptic Guard; Ulcidine†; ***Chile:*** Anulbet†; Fibonel†; Gastrium; ***Cz.:*** Famosan; Quamatel; Ulceran†; Ulfamid; ***Denm.:*** Pepcidin†; ***Fin.:*** Pepcid; Pepcidin; ***Fr.:*** Pepcidac; Pepdine; ***Ger.:*** Fadul; Famo; Famobeta; Famonerton; Pepcid†; Pepdul; ***Gr.:*** Ansilan; Banatin; Cepal†; Esseldon; Gasterogen; Imposergon; Mostrelan; Panalba; Peptan; Rosagenus; Sedanium-R; Vexurat; ***Hong Kong:*** Ausfam; Beilande; Fadine; Famine; Famocid; Famodine; Famolta; Famopsin; Famotin; Famox; Gastrodomina; LAfamo; Marmodine; Motidine; Pepcidine; Phyzidine; Quamatel; Servipep†; Ulceran†; Vida Famodine; ***Hung.:*** Motidin; Peptigal†; Quamatel; Servipep†; ***India:*** Blocacid†; Fadine; Famodin; Famonite; Famowal; Famtac; Fudone; Ulcimax; ***Indon.:*** Antidine; Corocyd; Denufam; Faberdin; Facid; Famocid; Fluktan; Gasfamin; Gaster; Gestofam; Ifamul; Interfam; Lexmodine; Nulcefam; Pompaton; Promocid; Purifam; Regastin; Renapepsa; Tismafam; Ulcerid; Ulfam; Ulmo; ***Irl.:*** Pepcid; ***Israel:*** Apogastine; Famo; Gastro; Rogasti; Zarex†; ***Ital.:*** Famodil; Gastridin; Motiax; ***Jpn:*** Gaster; ***Malaysia:*** Acidine†; Fadine†; Famopsin; Pepcidine; Pepzan; Ulceran; Voker; ***Mex.:*** Adiatin; Amofat†; Androtin; Durater; Eufatin†; Fabutin; Fagatrim†; Famoxal; Fatoril†; Fawodin†; Ludex; Pepcidine; Sertidine; Sigafam†; Ultidin; ***Neth.:*** Pepcid; Pepcidin; ***Norw.:*** Famotal; Pepcid; Pepcidin; ***NZ:*** Famox; Pepcid†; Pepcidine†; Pepzan; ***Philipp.:*** Famorila; Famtine; H2 Bloc; Hista-Bloc; Motid; Pepcidine; Ulcefam; ***Pol.:*** Famidyna; Famogast; Quamatel; Ulfamid; ***Port.:*** Digeslit†; Dinul; Dipsin; Fatidin; Gastopride†; Gastrifam†; Lasa; Mensoma; Nulceran†; Pepcidina; ***Rus.:*** Famocid (Фамоцид); Famonit (Фамонит); Famosan (Фамосан); Gastrosidin (Гастросидин); Quamatel (Квамател); Ulceran (Ульцеран); Ulfamid (Ульфамид); ***Singapore:*** Blocacid†; Famoc; Famopril; Famopsin†; Famotin; Famox†; Motidine; Pepcidine; Pepzan; Ulceran; ***Spain:*** Brolin†; Confobos; Cronol; Digervin; Dispromil†; Eviantrina; Fagastril; Famokey; Famulcer; Fanosin†; Fanox†; Gastenin; Gastrion; Gastrodomina; Gastropen†; Ingastri; Invigan; Nos†; Nulcerin; Pepcid; Rubacina; Tairal; Tamin; Tipodex; Ulcetrax†; Ulgarine; Vagostal; ***Swed.:*** Pepcid; Pepcidin; ***Switz.:*** Pepcid†; Pepcidine†; ***Thai.:*** Agufam; Fadine†; Famoc; Famocid†; Famonox; Famopsin; Famosia; Famotab; Famotin†; Fasidine; Motidine; Pepcidine; Pepcine; Pepdenal; Pepfamin; Peptoci; Pepzan†; Pharmotidine†; Ulceran; Ulcofam†; Ulfamet; ***Turk.:*** Duovel; Famec; Famo; Famodin; Famogast; Famoser; Famotep; Famotsan; Gasterol; Gastifam; Gastrofam; Gastrosidin; Neotab; Nevofam; Notidin; Pepdif; ***UAE:*** Famotec; ***UK:*** Pepcid; Ultra Heartburn Relief; ***USA:*** Mylanta AR Acid Reducer†; Pepcid; ***Venez.:*** Dinamot; Fadipina†; Famogel; Famulcer; Isomina; Klinotal; Medalin; Neutracid; Pepcidine†; Ulcenol.

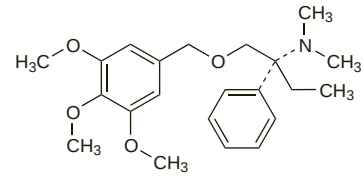
Multi-ingredient: ***Arg.:*** Actual; Megalex Antiacido; Mylanta Extra; ***Canad.:*** Pepcid Complete; ***Fin.:*** Pepcid Duo; ***Fr.:*** Pepcidduo; ***Ger.:*** Pepciddual; ***Indon.:*** Neosanmag Fast; Promag Double Action; ***Ital.:*** Pepciddual; ***Mex.:*** Facidex Total; ***Norw.:*** Pepcidduo; ***Spain:*** Pepdual; ***Swed.:*** Pepcid Duo; ***UK:*** Pepcidtwo; ***USA:*** Pepcid Complete.

Fedotozine *(rINN)*

Fedotozina; Fédotozine; Fedotozinum; JO-1196 (tartrate). (+)-(*R*)-α-Ethyl-*N*,*N*-dimethyl-α-{[(3,4,5-trimethoxybenzyl)oxy]methyl}benzylamine.

Федотозин

$C_{22}H_{31}NO_4$ = 373.5.
CAS — 123618-00-8 (fedotozine); 133267-27-3 (fedotozine tartrate).



Profile

Fedotozine is a peripherally acting selective agonist of opioid κ-receptors that has been investigated in dyspepsia and the irritable bowel syndrome.

◊ References.

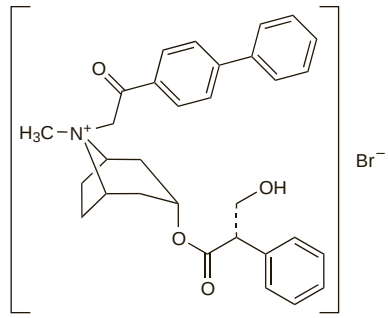
1. Delvaux M. Pharmacology and clinical experience with fedotozine. *Expert Opin Invest Drugs* 2001; **10:** 97–110.

Fentonium Bromide *(rINN)*

Bromuro de fentonio; Fa-402; Fentonii Bromidum; Fentonium, Bromure de; Ketoscilium; *N*-(4-Phenylphenacyl)-1-hyoscyaminium Bromide; Z-326. (−)-(1*R*,3*r*,5*S*)-8-(4-Phenylphenacyl)-3-[(*S*)-tropoloxy]tropanium bromide.

Фентония Бромид

$C_{31}H_{34}BrNO_4$ = 564.5.
CAS — 5868-06-4.
ATC — A03BB04.
ATC Vet — QA03BB04.



Profile

Fentonium bromide is a quaternary ammonium antimuscarinic with peripheral effects similar to those of atropine (p.1219). It has been used to relieve visceral spasms.

Fig

Carica; Ficus; Higo.

Инжир; Фикус

Pharmacopoeias. In *Br.* and *Swiss*.
BP 2008 (Fig). The sun-dried succulent fruit of *Ficus carica* containing not less than 60.0% of water-soluble extractive. Store in a dry place.

Profile

Fig is a mild laxative and demulcent usually used with other laxatives.