Preparations

BP 2008: Etodolac Capsules; Etodolac Tablets;
USP 31: Etodolac Capsules; Etodolac Extended-Release Tablets; Etodolac Tablets.

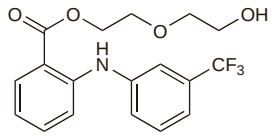
Proprietary Preparations (details are given in Part 3)
Austria: Lodine†; ***Braz.:*** Flancox; ***Canad.:*** Ultradol†; ***Denm.:*** Todolac; ***Fin.:*** Lodine; ***Fr.:*** Lodine; ***Gr.:*** Ecridoxan†; Lonine; ***Hong Kong:*** Lodine; ***Indon.:*** Lonene; ***Israel:*** Etopan; ***Ital.:*** Lodine†; ***Jpn:*** Hypen; ***Mex.:*** Lodine†; ***Port.:*** Acudor; Articulan; Dualgan; Lodine†; Lodot†; Metazin†; Sodolac; ***Switz.:*** Lodine; ***Thai.:*** Etonox; ***Turk.:*** Edolar; Etodin; Etol; Lodine; Tadolak; ***UK:*** Eccoxolac; Etopan; Lodine; ***USA:*** Lodine†; ***Venez.:*** Lodine†.

Etofenamate *(BAN, USAN, rINN)*

B-577; Bay-d-1107; Etofenamaatti; Etofenamát; Etofenamat; Etofenamatas; Étofénamate; Etofenamato; Etofenamatum; TV-485; TVX-485; WHR-5020. 2-(2-Hydroxyethoxy)ethyl *N*-(ααα-trifluoro-*m*-tolyl)anthranilate.

Этофенамат

$C_{18}H_{18}F_3NO_4$ = 369.3.
CAS — 30544-47-9.
ATC — M02AA06.
ATC Vet — QM02AA06.



Pharmacopoeias. In *Eur.* (see p.vii).
Ph. Eur. 6.2 (Etofenamate). A yellowish viscous liquid. Practically insoluble in water; miscible with alcohol and with ethyl acetate.

Profile

Etofenamate is an NSAID (p.96) that has been applied topically in a concentration of 5 or 10% for the relief of pain and inflammation associated with musculoskeletal, joint, and soft-tissue disorders. It has also been given by deep intramuscular injection in single doses of 1 g.

Preparations

Proprietary Preparations (details are given in Part 3)
Arg.: Bayrogel†; Contour†; Flogol; ***Austria:*** Rheumon; Traumon; ***Belg.:*** Flexium; ***Braz.:*** Bayro; ***Chile:*** Bayagel; Bayro†; Flogojet; Master-Gel†; Valorel; ***Cz.:*** Etogel†; Rheuma Denk†; Rheumon; Traumon; ***Ger.:*** Algesalona E†; Rheuma-Gel; Rheumon; Traumon; ***Gr.:*** Cimal†; Etofenol; Fenam†; Melferut; Pazergicel†; Radermin; Reumina; Roiplon; ***Hong Kong:*** Flogoprofen; ***Hung.:*** Activon Extra; Rheumon; Traumon†; ***Ital.:*** Bayro; ***Mex.:*** Bayro; ***Pol.:*** Rheumon; Traumon; ***Port.:*** Fenogel; Reumon; ***Spain:*** Aspitopic; Flogoprofen; Zenavan; ***Switz.:*** Activon†; Etofen†; Rheumon; Traumalix; ***Turk.:*** Doline; Flexo; Painex; Rheumon; ***Venez.:*** Traflan.

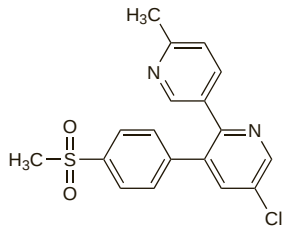
Multi-ingredient: ***Arg.:*** Bayagel; ***Austria:*** Thermo-Rheumon; ***Chile:*** Bayro-Therm†; ***Cz.:*** Thermo-Rheumon†; ***Ger.:*** Thermo-Rheumon†; ***Gr.:*** Thermo-Roiplon; ***Hung.:*** Thermo-Rheumon†; ***Mex.:*** Bayro Termo; ***Pol.:*** Thermo-Rheumon; ***Turk.:*** Thermo-Doline; Thermo-Rheumon; Thermoflex; ***Venez.:*** Reugel.

Etoricoxib *(BAN, USAN, rINN)*

Étoricoxib; Etoricoxibum; Etorikoksib; Etorikoksibi; Etorikoxib; L-791456; MK-0663; MK-663. 5-Chloro-6′-methyl-3-[*p*-(methylsulfonyl)phenyl]-2,3′-bipyridine.

Эторикоксиб

$C_{18}H_{15}ClN_2O_2S$ = 358.8.
CAS — 202409-33-4.
ATC — M01AH05.
ATC Vet — QM01AH05.



Adverse Effects, Treatment, and Precautions

As for NSAIDs in general, p.96.

Hypersensitivity reactions including anaphylaxis and angioedema have occurred in patients receiving etoricoxib; it should be stopped at the first signs of hypersensitivity. Etoricoxib should be avoided in patients with severe hepatic impairment (Child-Pugh score of 10 or more). Therapy should be stopped if persistently abnormal liver enzyme values are seen.

Etoricoxib should not be used in patients with ischaemic heart disease, peripheral arterial disease, or cerebrovascular disease. It should be used with caution in patients with significant risk factors for cardiovascular disease such as hypertension, hyperlipidaemia, and diabetes mellitus. Etoricoxib, particularly at high doses, may be associated with more frequent and severe hypertension compared with other NSAIDs and selective COX-2 inhibitors; blood pressure monitoring during etoricoxib treatment is recommended. Etoricoxib should not be used in patients with hypertension whose blood pressure is not controlled (see also Effects on the Cardiovascular System, below).

Etoricoxib is also contra-indicated in patients with inflammatory bowel disease, moderate to severe heart failure (NYHA class II to IV), and renal impairment associated with a creatinine clearance of less than 30 mL/minute. Caution is recommended when using etoricoxib in dehydrated patients; it may be advisable to rehydrate patients before giving etoricoxib.

Effects on the cardiovascular system. There have been concerns about the adverse cardiovascular effects of selective cyclo-oxygenase-2 (COX-2) inhibitors after the worldwide withdrawal of rofecoxib (see p.121). The cardiovascular safety of etoricoxib has been assessed in the MEDAL programme[1] which pooled data from 3 studies involving over 30 000 patients with either osteoarthritis or rheumatoid arthritis. Patients with osteoarthritis were given etoricoxib 60 or 90 mg daily; those with rheumatoid arthritis received 90 mg daily. In all studies, diclofenac 150 mg daily was given as the comparator; low-dose aspirin (100 mg daily or less) was also allowed where indicated. After an average treatment duration of 18 months, the rates of thrombotic events such as myocardial infarction, stroke, and sudden or unexplained death with etoricoxib were similar to those for diclofenac. (It has been suggested that diclofenac itself may increase the risk of some thrombotic events; for further details, see p.97.) The programme also found that the rate of some other non-thrombotic cardiovascular events was increased with etoricoxib: one of the 3 studies showed that there was a non-significant increase in the rate of heart failure with etoricoxib 90 mg daily compared to diclofenac; withdrawals due to oedema were also more frequent with high-dose etoricoxib than with diclofenac or etoricoxib 60 mg daily. In addition, the number of patients stopping treatment because of hypertension was higher with both doses of etoricoxib than with diclofenac. Similar results were seen in the other 2 studies.

In another study[2] that pooled pre-licensing data, the risk of thrombotic events with etoricoxib, given at a dose of at least 60 mg daily, was also found to be similar to that for placebo treatment, ibuprofen (2.4 g daily), diclofenac (150 mg daily), and naproxen (1 g daily), although there was a trend towards more events with etoricoxib than with naproxen. For details on the relative risk of thrombotic events associated with non-selective NSAIDs, see p.97.

The EMEA's Committee for Medicinal Products for Human Use (CHMP)[3] has recommended the inclusion of a warning in the labelling of etoricoxib that it must not be given to patients whose blood pressure is persistently above 140/90 mmHg and inadequately controlled; in addition, high blood pressure should be controlled before starting treatment and monitored for 2 weeks afterwards then regularly thereafter.

For discussion and advice on the use of selective COX-2 inhibitors in patients with cardiovascular or cerebrovascular disease, see under Celecoxib, p.34.

1. Cannon CP, *et al.* Cardiovascular outcomes with etoricoxib and diclofenac in patients with osteoarthritis and rheumatoid arthritis in the Multinational Etoricoxib and Diclofenac Arthritis Long-term (MEDAL) programme: a randomised comparison. *Lancet* 2006; **368:** 1771–81.
2. Curtis SP, *et al.* Pooled analysis of thrombotic cardiovascular events in clinical trials of the COX-2 selective inhibitor etoricoxib. *Curr Med Res Opin* 2006; **22:** 2365–74.
3. EMEA. EMEA recommends strengthening warnings and contraindications for etoricoxib-containing medicines used in the treatment of rheumatoid arthritis and ankylosing spondylitis (issued 26th June, 2008). Available at: http://www.emea.europa.eu/pdfs/human/press/pr/33363608en.pdf (accessed 16/07/08)

Effects on the gastrointestinal tract. It is generally accepted that the inhibition of cyclo-oxygenase-1 (COX-1) plays a role in the adverse gastrointestinal effects of the NSAIDs, and that the selective inhibition of the other isoform, COX-2, by NSAIDs such as etoricoxib may cause less gastrotoxicity than that seen with the non-selective inhibition of the traditional NSAIDs. However, licensed product information states that upper gastrointestinal perforation, ulceration, and bleeds, in some cases fatal, have occurred with etoricoxib treatment; consequently, it should be used with caution in patients with a history of, or at risk of developing, such events. In addition, etoricoxib should not be used in patients with active gastrointestinal ulceration or bleeding.

Results from controlled studies have suggested that NSAIDs selective for COX-2 were associated with a lower incidence of serious gastrointestinal effects. In a study[1] of the pooled data from 3 randomised clinical studies, etoricoxib (in doses of 60 or 90 mg daily) was associated with significantly less frequent upper gastrointestinal clinical events than diclofenac (150 mg daily). The result was attributed to the lower rate of uncomplicated ulcers with etoricoxib compared with diclofenac; there was no difference in the rate of complicated gastrointestinal events between the 2 drugs. The lower rate of uncomplicated events with etoricoxib compared with diclofenac was not affected by treatment with low-dose aspirin or proton pump inhibitors. An analysis[2] by the manufacturer, of pooled data from 10 randomised clinical studies, found that etoricoxib (in daily doses of 60, 90, or 120 mg) was associated with a lower combined risk of upper gastrointestinal perforations and bleeding, and symptomatic gastroduodenal ulcers when compared with non-selective NSAIDs (diclofenac 150 mg daily, ibuprofen 2.4 g daily, or naproxen 1 g daily) as a group. This reduced risk was seen even in patients with known risk factors for such complications such as the elderly and those with a history of gastrointestinal reactions.

1. Laine L, *et al.* Assessment of upper gastrointestinal safety of etoricoxib and diclofenac in patients with osteoarthritis and rheumatoid arthritis in the Multinational Etoricoxib and Diclofenac Arthritis Long-term (MEDAL) programme: a randomised comparison. *Lancet* 2007; **369:** 465–73.
2. Ramey DR, *et al.* The incidence of upper gastrointestinal adverse events in clinical trials of etoricoxib vs. non-selective NSAIDs: an updated combined analysis. *Curr Med Res Opin* 2005; **21:** 715–22.

Effects on the kidneys. Limited evidence of the renal toxicity of the selective cyclo-oxygenase-2 (COX-2) inhibitors such as etoricoxib suggests that such NSAIDs appear to have effects on renal function similar to those of the non-selective NSAIDs (see p.98).

Interactions

The metabolism of etoricoxib is mediated by the cytochrome P450 isoenzyme CYP3A4. Use with other drugs that inhibit or induce this isoenzyme may result in changes in plasma concentration of etoricoxib. In addition, *in vitro* studies suggest that several other isoenzymes may also mediate the main metabolic pathway of etoricoxib. Rifampicin, a potent inducer of CYP isoenzymes, has produced decreased plasma concentrations of etoricoxib.

Etoricoxib is an inhibitor of human sulfotransferase activity and has been shown to increase the plasma concentration of ethinylestradiol. Interactions with other drugs, such as oral salbutamol and minoxidil, also metabolised by this enzyme may be a possibility and licensed product information advises care with such combinations.

For interactions associated with NSAIDs in general, see p.99.

Pharmacokinetics

Etoricoxib is well absorbed from the gastrointestinal tract after oral doses. Peak plasma concentrations are reached in about 1 hour in fasted adults; food delays absorption by about 2 hours, although it has no effect on the extent of absorption. Plasma protein binding is about 92%. At steady state the half-life of etoricoxib is about 22 hours. Etoricoxib is extensively metabolised with less than 2% of a dose recovered in the urine as the parent drug. The major route of metabolism is via cytochrome P450 isoenzymes including CYP3A4 to form the 6′-hydroxymethyl derivative of etoricoxib, which is then oxidised to the 6′-carboxylic acid derivative, the major metabolite. Both are inactive or only weak cyclo-oxygenase-2 (COX-2) inhibitors. Excretion is mainly via the urine (70%) with only 20% of a dose appearing in the faeces. Studies in *animals* suggest that etoricoxib may cross the placenta and that some is distributed into breast milk.

◊ References.

1. Agrawal NGB, *et al.* Single- and multiple-dose pharmacokinetics of etoricoxib, a selective inhibitor of cyclooxygenase-2, in man. *J Clin Pharmacol* 2003; **43:** 268–76.
2. Agrawal NGB, *et al.* Pharmacokinetics of etoricoxib in patients with hepatic impairment. *J Clin Pharmacol* 2003; **43:** 1136–48.
3. Agrawal NGB, *et al.* Pharmacokinetics of etoricoxib in patients with renal impairment. *J Clin Pharmacol* 2004; **44:** 48–58.

Uses and Administration

Etoricoxib is an NSAID (p.96) reported to be a selective inhibitor of cyclo-oxygenase-2 (COX-2). It is used in the symptomatic relief of rheumatoid arthritis, osteoarthritis, and acute gouty arthritis.

In osteoarthritis, etoricoxib is given orally in a usual dose of 30 mg once daily, increased to 60 mg once daily if necessary. The recommended dose in rheumatoid arthritis is 90 mg once daily; higher doses of 120 mg once daily are used in gouty arthritis although such doses should only be used for the acute symptomatic period and for a maximum of 8 days. For dosage recommendations in patients with hepatic impairment, see below.

◊ References.

1. Patrignani P, *et al.* Clinical pharmacology of etoricoxib: a novel selective COX2 inhibitor. *Expert Opin Pharmacother* 2003; **4:** 265–84.
2. Dallob A, *et al.* Characterization of etoricoxib, a novel, selective COX-2 inhibitor. *J Clin Pharmacol* 2003 **43:** 573–85.
3. Martina SD, *et al.* Etoricoxib: a highly selective COX-2 inhibitor. *Ann Pharmacother* 2005; **39:** 854–62.

Administration in hepatic impairment. The maximum oral dose of etoricoxib in patients with mild hepatic impairment (Child-Pugh score of 5 to 6), regardless of indication, is 60 mg once daily; those with moderate impairment (Child-Pugh 7 to 9) should be given a maximum of 60 mg every other day or 30 mg once daily. Etoricoxib should not be given to patients with severe hepatic impairment (Child-Pugh 10 or more).

Musculoskeletal and joint disorders. The selective cyclo-oxygenase-2 (COX-2) inhibitor etoricoxib is used in the treatment of the musculoskeletal disorders osteoarthritis and rheumatoid arthritis (see p.11 and p.11, respectively). However, in the UK, it is recommended that the use of selective COX-2 inhibitors is limited to those patients considered to be at high risk of developing serious gastrointestinal problems if given a non-selective NSAID (see p.97).

Etoricoxib is also used in gouty arthritis (p.552) and has been tried in the treatment of ankylosing spondylitis (see Spondyloarthropathies, p.13).

References.

1. Cochrane DJ, *et al.* Etoricoxib. *Drugs* 2002; **62:** 2637–51.
2. Schumacher HR, *et al.* Randomised double blind trial of etoricoxib and indometacin in treatment of acute gouty arthritis. *BMJ* 2002; **324:** 1488–92.
3. Gottesdiener K, *et al.* Results of a randomized, dose-ranging trial of etoricoxib in patients with osteoarthritis. *Rheumatology (Oxford)* 2002; **41:** 1052–61.
4. Wiesenhutter CW, *et al.* Evaluation of the comparative efficacy of etoricoxib and ibuprofen for treatment of patients with osteoarthritis: a randomized, double-blind, placebo-controlled trial. *Mayo Clin Proc* 2005; **80:** 470–9.
5. van der Heijde D, *et al.* Evaluation of the efficacy of etoricoxib in ankylosing spondylitis: results of a fifty-two-week, randomized, controlled study. *Arthritis Rheum* 2005; **52:** 1205–15.
6. Curtis SP, *et al.* Etoricoxib in the treatment of osteoarthritis over 52-weeks: a double-blind, active-comparator controlled trial [NCT00242489]. *BMC Musculoskelet Disord* 2005; **6:** 58. Available at: http://www.biomedcentral.com/content/pdf/1471-2474-6-58.pdf (accessed 01/11/07)
7. Bingham CO, *et al.* Efficacy and safety of etoricoxib 30 mg and celecoxib 200 mg in the treatment of osteoarthritis in two identically designed, randomized, placebo-controlled, non-inferiority studies. *Rheumatology (Oxford)* 2007; **46:** 496–507.

Preparations

Proprietary Preparations (details are given in Part 3)

Arg.: Arcoxia; ***Austria:*** Arcoxia; Auxib; ***Belg.:*** Arcoxia; ***Braz.:*** Arcoxia; ***Cz.:*** Arcoxia; ***Denm.:*** Arcoxia; ***Fin.:*** Arcoxia; ***Fr.:*** Arcoxia; ***Ger.:*** Arcoxia; ***Gr.:*** Arcoxia; ***Hong Kong:*** Arcoxia; ***India:*** Ebov; Ecoxib†; Etoxib; Etozox; Kretos†; Nucoxia; ***Indon.:*** Arcoxia; ***Irl.:*** Arcoxia; ***Israel:*** Arcoxia; ***Ital.:*** Algix; Arcoxia; Tauxib; ***Malaysia:*** Arcoxia; ***Mex.:*** Arcoxia; ***Neth.:*** Arcoxia; Auxib; ***Norw.:*** Arcoxia; ***NZ:*** Arcoxia; ***Philipp.:*** Arcoxia; ***Port.:*** Arcoxia; Exxiv; Turox; ***Singapore:*** Arcoxia; ***Spain:*** Arcoxia; ***Swed.:*** Arcoxia; ***Thai.:*** Arcoxia; ***UK:*** Arcoxia; ***Venez.:*** Arcoxia.

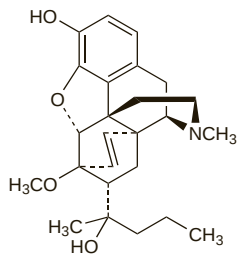
Etorphine Hydrochloride *(BANM, rINNM)*

Étorphine, Chlorhydrate d'; Etorphini Hydrochloridum; Hidrocloruro de etorfina; M-99; 19-Propylorvinol Hydrochloride. (6*R*,7*R*,14*R*)-7,8-Dihydro-7-[(1*R*)-1-hydroxy-1-methylbutyl]-6-*O*-methyl-6,14α-ethenomorphine hydrochloride; (2*R*)-2-[(–)-(5*R*,6*R*,7*R*,14*R*)-4,5-Epoxy-3-hydroxy-6-methoxy-9a-methyl-6,14-ethenomorphinan-7-yl]pentan-2-ol hydrochloride.

Эторфина Гидрохлорид

$C_{25}H_{33}NO_4$,HCl = 448.0.

CAS — 14521-96-1 (etorphine); 13764-49-3 (etorphine hydrochloride).



(etorphine)

Pharmacopoeias. In *BP(Vet)*.

BP(Vet) 2008 (Etorphine Hydrochloride). A white or almost white microcrystalline powder. Sparingly soluble in water and in alcohol; very slightly soluble in chloroform; practically insoluble in ether. A 2% solution in water has a pH of 4.0 to 5.5. Protect from light.

Dependence and Withdrawal

As for Opioid Analgesics, p.101.

Adverse Effects and Treatment

As for Opioid Analgesics in general, p.102. Etorphine is not used therapeutically in humans.

Etorphine hydrochloride is highly potent and rapid acting; minute amounts can exert serious effects leading to coma. It may be absorbed through skin and mucous membranes. It is thus advisable to inject an antagonist *immediately* after contamination of skin or mucous membranes with preparations containing etorphine hydrochloride and to wash the affected areas copiously. Accidental injection or needle scratch injuries should also be treated immediately by injecting an antagonist. Naloxone is preferred as the antagonist in medical treatment. However, veterinary preparations of etorphine are supplied with a preparation (*Revivon*) containing diprenorphine hydrochloride (p.1445) and this should be used for immediate first-aid antagonism if naloxone is not available.

Uses and Administration

Etorphine hydrochloride is a highly potent opioid analgesic (p.104) used for reversible neuroleptanalgesia (see Anaesthetic Techniques, p.1780) in veterinary medicine. It is given with acepromazine maleate or levomepromazine (*Immobilon*) to restrain animals and before minor veterinary surgery. The duration of action of etorphine is up to about 45 to 90 minutes depending on the species but it may be longer in man, especially if the large animal preparation is involved.

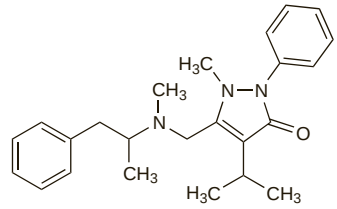
Famprofazone *(BAN, rINN)* ⊗

Famprofazona; Famprofazonum. 4-Isopropyl-2-methyl-3-[methyl(α-methylphenethyl)aminomethyl]-1-phenyl-3-pyrazolin-5-one.

Фампрофазон

$C_{24}H_{31}N_3O$ = 377.5.

CAS — 2288 1-35-2.



Profile

Famprofazone has analgesic and antipyretic properties and has been given orally, usually with other analgesics.

Pharmacokinetics. On ingestion, metabolic products of famprofazone include amfetamine and metamfetamine enantiomers,[1,2] which has led to false positive results on drug testing.[3] For sporting competition famprofazone was classified by the World Anti-Doping Agency as a stimulant.[4]

1. Greenhill B, *et al.* Metabolic profile of amphetamine and methamphetamine following administration of the drug famprofazone. *J Anal Toxicol* 2003; **27:** 479–84.
2. Rodriguez AT, *et al.* Metabolic profile of famprofazone following multidose administration. *J Anal Toxicol* 2004; **28:** 432–8.
3. Musshoff F, Kraemer T. Identification of famprofazone ingestion. *Int J Legal Med* 1998; **111:** 305–8.
4. World Anti-Doping Agency. The world anti-doping code: the 2008 prohibited list international standard. Available at: http://www.wada-ama.org/rtecontent/document/2008_List_En.pdf (accessed 24/07/08)

Felbinac *(BAN, USAN, rINN)*

CL-83544; Felbinaakki; Felbinaco; Felbinacum; Felbinak; LJC-10141. Biphenyl-4-ylacetic acid.

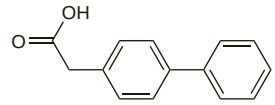
Фелбинак

$C_{14}H_{12}O_2$ = 212.2.

CAS — 5728-52-9.

ATC — M02AA08.

ATC Vet — QM02AA08.



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

Ph. Eur. 6.2 (Felbinac). A white or almost white, crystalline powder. Practically insoluble in water; sparingly soluble in alcohol; soluble in methyl alcohol.

Adverse Effects and Precautions

Mild local reactions such as erythema, dermatitis, and pruritus have occurred in patients using felbinac topically. More serious adverse effects including bullous dermatoses such as epidermal necrolysis and erythema multiforme, photosensitivity, anaphylaxis, and bronchospasm or wheeziness have also been reported. Gastrointestinal disturbances may occur.

Felbinac preparations should be avoided in patients with a history of hypersensitivity reactions to aspirin or other NSAIDs.

Incidence of adverse effects. The UK CSM had received 49 reports of adverse reactions associated with felbinac by October 1989, about 11 months after it was released on the UK market.[1] Bronchospasm or wheeziness was reported in 8 patients using felbinac gel. Four of these patients had a history of asthma of whom 3 were reported to have had a similar reaction to aspirin or other NSAIDs. Other reported reactions included skin rashes (17 cases), local application site reactions (7), and dyspepsia (6).

1. CSM. Felbinac (Traxam) and bronchospasm. *Current Problems 27* 1989. Also available at: http://www.mhra.gov.uk/home/idcplg?IdcService=GET_FILE&dDocName=CON2024444&RevisionSelectionMethod=LatestReleased (accessed 01/11/07)

Uses and Administration

Felbinac, an active metabolite of fenbufen (below), is an NSAID (p.99). It is used topically in the symptomatic treatment of musculoskeletal pain including that due to soft-tissue injuries. It is applied as a 3% gel or a 3.17% foam to unbroken skin over affected areas 2 to 4 times daily. The total daily dose of gel or foam should not exceed 25 g regardless of the size or number of affected areas. Therapy should be reviewed after 14 days.

Diisopropanolamine felbinac has been used similarly.

◊ References.

1. Hosie GAC. The topical NSAID, felbinac, versus oral ibuprofen: a comparison of efficacy in the treatment of acute lower back injury. *Br J Clin Res* 1993; **4:** 5–17.

Preparations

BP 2008: Felbinac Cutaneous Foam; Felbinac Gel.

Proprietary Preparations (details are given in Part 3)

Austria: Target†; ***Belg.:*** Flexfree; ***Ger.:*** Spalt Schmerz-Gel†; ***Irl.:*** Traxam; ***Ital.:*** Dolinac; Traxam; ***Jpn:*** Seltouch; ***Switz.:*** Dolo Target†; ***UK:*** Traxam.

Fenbufen *(BAN, USAN, rINN)*

CL-82204; Fenbufeeni; Fenbufén; Fenbufenas; Fenbufène; Fenbufenum. 4-(Biphenyl-4-yl)-4-oxobutyric acid.

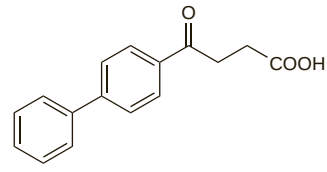
Фенбуфен

$C_{16}H_{14}O_3$ = 254.3.

CAS — 36330-85-5.

ATC — M01AE05.

ATC Vet — QM01AE05.



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

Ph. Eur. 6.2 (Fenbufen). A white or almost white, fine crystalline powder. Very slightly soluble in water; slightly soluble in alcohol, in acetone, and in dichloromethane.

Adverse Effects, Treatment, and Precautions

As for NSAIDs in general, p.96, although the commonest adverse effects of fenbufen are skin rashes,