and as a tonic. Uridine has been tried in patients with hereditary orotic aciduria.

Preparations

Proprietary Preparations (details are given in Part 3)

Multi-ingredient: ***Belg.:*** Vitacic; ***Cz.:*** Laevadosin†; ***Hung.:*** Vitacic†; ***Ital.:*** Centrum; ***Mon.:*** Vitacic; ***Rus.:*** Vitacic (Витасик)†.

Uridine Triphosphate

Ins-316; Trifosfato de uridina; Uridina trifosfato; Uridine Triphosphoric Acid; UTP. Uridine 5′-(tetrahydrogen triphosphate).

Уридин Трифосфат

$C_9H_{15}N_2O_{15}P_3$ = 484.1.

CAS — 63-39-8.

Profile

Uridine triphosphate is an endogenous uracil nucleotide involved in many biological processes. It has been claimed to be of value in muscular atrophy and muscular weakness, and has been included in preparations for neuralgia, neuritis, and muscular disorders; the disodium and trisodium salts have also been used.

◊ Uridine triphosphate has been reported to have beneficial effects on mucociliary clearance in chronic respiratory disease such as cystic fibrosis, asthma, and chronic bronchitis, although it may not be suitable for such therapy because it is rapidly degraded by enzymatic activity within sputum and by airway epithelial cells.[1,2] It may, however, have a role as a method of producing deep-lung sputum specimens for cytological evaluation and is being investigated as an aid to the diagnosis of for example, lung cancer[1,2] or airway inflammation in asthma.[3]

1. Bennett WD, *et al.* Effect of aerosolized uridine 5′-triphosphate on mucociliary clearance in mild chronic bronchitis. *Am J Respir Crit Care Med* 2001; **164:** 302–6.
2. Johnson FL, *et al.* Improved sputum expectoration following a single dose of INS316 in patients with chronic bronchitis. *Chest* 2002; **122:** 2021–9.
3. Tamaoki J, *et al.* Validity and safety of sputum induction by inhaled uridine 5′-triphosphate. *Am J Respir Crit Care Med* 2001; **164:** 378–81.

Preparations

Proprietary Preparations (details are given in Part 3)

Fr.: Uteplex; ***Ital.:*** Miocuril†.

Multi-ingredient: ***Arg.:*** Nucleo CMP†; ***Braz.:*** Nucleo CMP; ***Chile:*** Citoneuron; ***Ital.:*** Fosfoutipi Vitaminico†; ***Mex.:*** Nucleo CMP; ***Spain:*** Cefabol; Nucleo CMP; Taurobetina†.

Ursodeoxycholic Acid *(BAN, rINN)*

Acide Ursodéoxycholique; Acide ursodésoxycholique; Ácido ursodeoxicólico; Acidum ursodeoxycholicum; Kyselina ursodeoxycholová; UDCA; Ursodeoksicholio rūgštis; Ursodeoksikolik Asit; Ursodeoksikoolihappo; Ursodeoxicholsyra; Ursodesoxycholic Acid; Ursodiol *(USAN)*; Urzodezoxikólsav. 3α,7β-Dihydroxy-5β-cholan-24-oic acid.

Урсодезоксихолевая Кислота

$C_{24}H_{40}O_4$ = 392.6.

CAS — 128-13-2.

ATC — A05AA02.

ATC Vet — QA05AA02.

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

Ph. Eur. 6.2 (Ursodeoxycholic Acid). A white or almost white powder. Practically insoluble in water and in dichloromethane; freely soluble in alcohol; slightly soluble in acetone.

USP 31 (Ursodiol). A white or almost white, crystalline powder. Practically insoluble in water; freely soluble in alcohol and in glacial acetic acid; sparingly soluble in chloroform; slightly soluble in ether. Store in airtight containers.

Stability. References.

1. Mallett MS, *et al.* Stability of ursodiol 25 mg/mL in an extemporaneously prepared oral liquid. *Am J Health-Syst Pharm* 1997; **54:** 1401–4.
2. Johnson CE, Streetman DD. Stability of oral suspensions of ursodiol made from tablets. *Am J Health-Syst Pharm* 2002; **59:** 361–3.

Adverse Effects and Precautions

Ursodeoxycholic acid may cause nausea, vomiting, and other gastrointestinal disturbances; diarrhoea is reported to occur less frequently than with chenodeoxycholic acid. Increased liver enzyme values are also less likely. Pruritus may occur. Treatment with ursodeoxycholic acid may cause more calcification of cholesterol stones than chenodeoxycholic acid.

Ursodeoxycholic acid should not be given to patients with intestinal and hepatic disorders that interfere with entero-hepatic circulation of bile salts (but see Chronic Liver Disease below). It is ineffective for the dissolution of calcified and pigment gallstones and is of no value in patients without a patent and functioning gallbladder. Licensed product information recommends that its use should be avoided in pregnancy.

◊ References.

1. Hempfling W, *et al.* Systematic review: ursodeoxycholic acid—adverse effects and drug interactions. *Aliment Pharmacol Ther* 2003; **18:** 963–72.

Interactions

Ursodeoxycholic acid should not be used with drugs that increase bile cholesterol such as oestrogenic hormones and cholesterol-lowering drugs such as clofibrate. Use with bile-acid binding drugs including antacids, charcoal, and colestyramine should be avoided since this may reduce the effectiveness of therapy with ursodeoxycholic acid.

For references to the possible effects of ursodeoxycholic acid on ciclosporin, see p.1828.

Pharmacokinetics

Ursodeoxycholic acid is absorbed from the gastrointestinal tract and undergoes enterohepatic recycling. It is partly conjugated in the liver before being excreted into the bile. Under the influence of intestinal bacteria the free and conjugated forms undergo 7α-dehydroxylation to lithocholic acid, some of which is excreted directly in the faeces and the rest absorbed and mainly conjugated and sulfated by the liver before excretion in the faeces. However, in comparison with chenodeoxycholic acid, less ursodeoxycholic acid undergoes such bacterial degradation.

◊ References.

1. Crosignani A, *et al.* Clinical pharmacokinetics of therapeutic bile acids. *Clin Pharmacokinet* 1996; **30:** 333–58.

Uses and Administration

Ursodeoxycholic acid is a naturally occurring bile acid (see p.2266) present in small quantities in human bile. Ursodeoxycholic acid suppresses the synthesis and secretion of cholesterol by the liver and inhibits intestinal absorption of cholesterol. It is used for the dissolution of cholesterol-rich **gallstones** in patients with functioning gallbladders (see below). The licensed dose is 6 to 12 mg/kg daily as a single bedtime dose or in 2 or 3 divided doses; obese patients may require up to 15 mg/kg daily. The daily dose may be divided unequally and the larger dose given before bedtime to counteract the increase in biliary cholesterol concentration seen overnight. The time required for dissolution of gallstones is likely to be between 6 and 24 months depending on stone size and composition. Treatment should be continued for 3 to 4 months after radiological disappearance of the stones. A dose of 300 mg twice daily has been suggested for the *prevention* of gallstones in patients undergoing rapid weight loss. Ursodeoxycholic acid has also been given in reduced doses in combination with chenodeoxycholic acid (p.2280).

Ursodeoxycholic acid is also used in **primary biliary cirrhosis**. The usual dose is 10 to 15 mg/kg daily in 2 to 4 divided doses.

Ursodeoxycholic acid has been tried in the treatment of primary sclerosing cholangitis.

The more hydrophilic derivative, tauroursodeoxycholic acid, has also been used.

Chronic liver disease. The use of ursodeoxycholic acid in chronic liver diseases has been summarised.[1-5] There have been differing opinions of its value in primary biliary cirrhosis (below). Response has been reported in obstetric cholestasis of pregnancy,[6-10] primary sclerosing cholangitis,[11] chronic active hepatitis,[12] and viral hepatitis.[13,14] Although ursodeoxycholic acid is widely used in the treatment of obstetric cholestasis, data on its safety and efficacy in this indication are lacking, and its possible role therefore remains unclear until further larger studies have been done.[15] See also Adverse Effects and Precautions, above. Ursodeoxycholic acid appears to be of benefit in liver disease associated with cystic fibrosis[16-18] and in the UK the *BNFC* recommends a dose of 10 to 15 mg/kg twice daily in children and adolescents with this condition; alternatively the total daily dose may be given in 3 divided doses. Ursodeoxycholic acid had initially shown some promise in the treatment of nonalcoholic steatohepatitis,[19] but a randomised, controlled study[20] failed to confirm this. There has also been some interest in the use of ursodeoxycholic acid to treat refractory graft-versus-host disease of the liver in transplant patients,[21] and possibly as an adjuvant to immunosuppressant therapy[22-24] after orthotopic liver transplantation (p.1815). It may also be of benefit in the prevention of hepatic complications following allogeneic bone marrow transplantation (p.1811).[25,26] Ursodeoxycholic acid has shortened the clinical course of parenteral nutrition-associated cholestasis in very-low-birth-weight infants,[27] and the *BNFC* recommends a dose of 10 mg/kg three times daily for this purpose. Benefit has also been reported[28] in parenteral-nutrition associated cholestasis in children and adults, although the optimal timing and duration of therapy remains to be evaluated. Ursodeoxycholic acid has also been associated with a lower incidence of colonic dysplasia in patients with ulcerative colitis and primary sclerosing cholangitis.[29,30] For sclerosing cholangitis in children and adolescents, the *BNFC* recommends a dose of 5 to 10 mg/kg 2 or 3 times daily up to a maximum of 15 mg/kg 3 times daily depending on response. In children with biliary atresia, ursodeoxycholic acid may be used to treat associated cholestasis. The *BNFC* suggests a dose of 5 mg/kg three times daily in neonates and children up to 2 years of age, the dose and frequency adjusted according to response up to a maximum of 10 mg/kg three times daily.

1. de Caestecker JS, *et al.* Ursodeoxycholic acid in chronic liver disease. *Gut* 1991; **32:** 1061–5.
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PRIMARY BILIARY CIRRHOSIS. Primary biliary cirrhosis (PBC) is a chronic liver disease of unknown aetiology that develops due to progressive destruction of small and intermediate bile ducts within the liver, subsequently evolving to fibrosis and cirrhosis. Over 90% of patients are female, usually aged between 40 and 60 years. The disease is thought to be auto-immune in nature. Most patients develop antimitochondrial antibodies that may be evident even before disease is clinically apparent.[1,2] Genetic factors and hormonal stimulation may play a role in precipitating PBC.[1,2] Infectious agents such as *Chlamydophila pneumoniae* (*Chlamydia pneumoniae*)[2] or retroviruses[3] may be involved in the pathogenesis.

Clinical manifestations include pruritus, fatigue, jaundice, hepatomegaly, and hypercholesterolaemia leading to xanthoma formation. In late disease portal hypertension, bleeding oesophageal varices and liver failure may develop. Impaired calcium and vitamin D absorption may result in osteomalacia or osteoporosis, and fat-soluble vitamin deficiencies may occur. There may be accumulation of copper in the liver.[4] Other disorders including rheumatoid arthritis, scleroderma, thyroiditis, and Sjögren's syndrome may be associated with PBC.[1,5]

The disease is slowly progressive, with a mean survival of 8 years for symptomatic patients, and 16 years for asymptomatic individuals.[1,6] Despite its presumed auto-immune aetiology, few immunosuppressive drugs have shown any benefit,[5,7] although newer drugs such as mycophenolate mofetil, sirolimus, and tacrolimus have yet to be fully evaluated.[7]

The best-studied treatment for PBC is ursodeoxycholic acid, which is thought to replace toxic endogenous bile acids, stimulate bile acid secretion, and exert local immunosuppressive and cytoprotective effects.[1,4,5,7-9] The value of ursodeoxycholic acid is controversial: its reported therapeutic benefits in terms of delaying disease progression and the need for liver transplantation[5,10] have not been confirmed by meta-analysis or systematic review.[11,12] Both of these were, however, criticised[13,14] on the grounds that most of the studies included had follow-up periods of only 2 years. In consequence, some do not recommend its use,[12,15] but others still believe it to be the treatment of choice.[1,7,13,14,16] Its advocates consider that it appears to be efficacious for about 10 years and improves long-term survival by delaying the progression of hepatic fibrosis, development of oesophageal varices, and the need for liver transplantation. However, it is not effective in the presence of extensive fibrosis or cirrhosis in advanced disease.[13,14,16]

Both penicillamine and azathioprine have been used in PBC, but trials have failed to show any benefit from treatment[5-7] and their use has declined. A systematic review[17] identified a significant increase in the occurrence of adverse effects with penicillamine and concluded that its use could not be supported for patients with PBC. Corticosteroids, colchicine, ciclosporin, and chlorambucil have also been tried, but toxicity has restricted their use.[5,7] They may be of benefit[2,4,6,7] when used with ursodeoxycholic acid, although some guidelines[5] do not recommend their use. A systematic review[18] of studies with methotrexate concluded that it tended to increase mortality or the need for liver transplantation and should not therefore be used in patients with PBC outside clinical trials. Budesonide[7] and bezafibrate[1,7] have also been tried.

Symptomatic treatment includes the use of bile acid sequestrants, such as colestyramine, to treat both pruritus and hypercholesterolaemia. Ursodeoxycholic acid may also improve pruritus in up to 40% of patients, and rifampicin, phenobarbital, and opioid antagonists are used as second-line therapies.[1,4,5,8] Vitamin D and calcium supplementation will prevent osteomalacia; supplementation with vitamins A, E, and K may also be necessary.[1,4,5,8] Liver transplantation is recommended for liver failure, although PBC can recur in the allograft.[5,13,14,16]

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Gallstones. Gallstones (cholelithiasis) occur when mechanisms for the solubilisation of cholesterol or bilirubin fail or are overcome. They may be divided into those formed of pure cholesterol, which are usually solitary; pigment stones, largely made up of bilirubin or its derivatives; and mixed stones of cholesterol, bile pigment, and calcium salts, which form the great majority of cases seen in the West.

Gallstones are generally more common in women than in men. The prevalence also increases with age and obesity, although rapid weight loss as a result of dieting or surgery is associated with an increased risk.

As many as two-thirds of patients with gallstones are asymptomatic. Symptoms usually relate to the site of the stone although biliary colic is often present regardless of whether the stone is in the gallbladder or biliary tract. If the stone blocks the exit from the gallbladder, inflammation and bacterial infection may follow (acute cholecystitis), sometimes leading to perforation and subsequent peritonitis. Less commonly, obstruction of the common bile duct by gallstones (choledocholithiasis) may lead to cholestasis and jaundice; infection of the bile ducts and septicaemia may follow. Pancreatitis may also be associated with gallstone disease, and there may be an increased risk of developing malignant neoplasms of the gallbladder.

Treatment. Asymptomatic gallstones discovered during other investigations should not be treated, and even mildly symptomatic patients may be managed with analgesics and subsequent observation. Potent analgesics such as morphine may be needed in more severe cases (see Biliary and Renal Colic, p.5). In symptomatic patients the preferred treatment for gallstones is surgical removal of the gallbladder; laparoscopic cholecystectomy causes less postoperative morbidity than open surgery, and has largely replaced other methods of treatment.

In patients unsuited to, or unwilling to undergo, surgery for gallbladder stones, drug therapy, alone or with lithotripsy, may be considered.

Exogenous bile acids have been tried in an attempt to dissolve the cholesterol component of gallstones. Ursodeoxycholic acid is more effective and is associated with fewer adverse effects than chenodeoxycholic acid. Combination therapy has also been tried but this is no more effective than ursodeoxycholic acid alone. Dissolution of gallstones is slow but can be achieved in about one-third of cases with the best results seen with small stones. However, about half of all successfully treated patients will develop further gallstones within 10 years. Studies of prophylactic bile acid therapy have mostly yielded disappointing results, although such therapy may be of benefit in patients on very-low-calorie diets, after surgery for weight loss, and in those receiving treatment with octreotide.

Somewhat larger stones may respond to extracorporeal shock-wave lithotripsy, or fluoroscopically guided laser lithotripsy, which may be more effective. Oral bile acids should then be given to dissolve the stone fragments.

Another method that has been used is the direct instillation of a solvent (usually methyl *tert*-butyl ether) into the gallbladder, which dissolves stones within a matter of hours, and is effective against almost all cholesterol-based stones regardless of size and number. Care is required to avoid overflow of the solvent into the common bile duct or the duodenum, where it can cause inflammation. Other solvents, such as ethyl propionate have been investigated as potentially less toxic alternatives, and edetic acid has been suggested as a possible solvent for non-cholesterol gallstones. As with all non-surgical methods, recurrence is likely.

Patients with **stones in the common bile duct** or **acute cholecystitis** require prompt therapy because of the risk of serious complications; endoscopic sphincterotomy and physical retrieval of the stones with a basket or balloon appears to be the preferred treatment, with open surgery as an alternative. A biliary stent to allow bile flow around the stone has been used as a temporary measure in patients with stones too large to remove by endoscopic sphincterotomy. Lithotripsy or infusion of a solvent such as monoctanoin or methyl *tert*-butyl ether are possible alternatives in patients unfit for surgery.

In patients who develop cholecystitis or cholangitis antibacterial therapy may be required (see Biliary-tract Infections, p.164).

References.

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Preparations

BP 2008: Ursodeoxycholic Acid Capsules; Ursodeoxycholic Acid Tablets; ***USP 31:*** Ursodiol Capsules.

Proprietary Preparations (details are given in Part 3)
Arg.: Dexo; Solutrat; UDCA; Ursidesox; Ursomax; Urzac; ***Austral.:*** Ursofalk; ***Austria:*** Ursofalk; ***Belg.:*** Ursochol; Ursofalk; ***Braz.:*** Ursacol; ***Canad.:*** Urso; ***Chile:*** Solvobil; Ursofalk; ***Cz.:*** Ursochol; Ursofalk; Ursosan; ***Fin.:*** Adursal; ***Fr.:*** Delursan; Ursolvan; ***Ger.:*** Cholit-Ursan; Cholofalk; UDC; Urso†; Ursochol; Ursofalk; ***Gr.:*** Ursofalk; ***Hong Kong:*** Ursofalk; Ursosan; ***Hung.:*** Ursofalk; ***India:*** Udiliv; ***Indon.:*** Estazor; Pramur; Urdafalk; Urdahex; ***Irl.:*** Ursofalk; ***Israel:*** Ursofalk; Ursolit; ***Ital.:*** Benursil†; Biliepar; Coledos†; Desocol; Desoxil; Deursil; Dissolursil; Epasol†; Fraurs; Galmax†; Lentorsil†; Litoff; Liturso; Tauro; Tudcabil†; Urdes; Ursacol; Ursilon; Ursobil; Ursodamor; Ursodexil†; Ursodiol; Ursofalk†; Ursoflor; Ursolac†; Ursolisin; Ursoprogé†; ***Jpn:*** Urso; Ursosan†; ***Malaysia:*** Ursofalk; ***Mex.:*** Ursofalk; ***Neth.:*** Ursochol; Ursofalk; ***Norw.:*** Ursofalk; ***NZ:*** Actigall; Ursofalk; ***Philipp.:*** Ursofalk; ***Pol.:*** Proursan; Ursocam; Ursofalk; Ursopol; ***Port.:*** Destolit; Ursofalk; ***Rus.:*** Ursosan (Урсосан); ***S.Afr.:*** Ursotan†; ***Singapore:*** Ursofalk; ***Spain:*** Ursobilane; Ursochol; ***Swed.:*** Ursofalk; ***Switz.:*** De-ursil; Ursochol; Ursofalk; ***Thai.:*** Udihep; Ursofalk; Ursolin; ***Turk.:*** Ursofalk; ***UK:*** Destolit; Urdox; Ursofalk; Ursogal; ***USA:*** Actigall; Urso.

Multi-ingredient: ***Austria:*** Lithofalk†; ***Ger.:*** Lithofalk; Urso Mix†; ***Gr.:*** Lithiofalk†; ***Ital.:*** Bilenor; ***Jpn:*** Cabe 2; Eki Cabe.

Urtica

Brännässleblad (nettle leaf); Brennessel; Dilgėlių lapai (nettle leaf); Kopřivový list (nettle leaf); Liść pokrzywy (nettle leaf); Nokkosenlehti (nettle leaf); Ortie; Ortie dioïque; Ortie, feuille d' (nettle leaf); Ortiga; Pokrzywa zwyczajna; Stinging Nettle; Urtica dioica; Urticae folium (nettle leaf).

Pharmacopoeias. In *Ger.* and *US* (both specify the root and rhizome of *Urtica dioica*).
Eur. (see p.vii) includes the leaf of *Urtica* spp. and also a form of *Urtica dioica* for homoeopathic preparations.
Br. includes a form of *Urtica urens* for homeopathic preparations.
Ph. Eur. 6.2 (Nettle Leaf; Urticae Folium). The whole or cut dried leaves of *Urtica dioica*, *Urtica urens*, or a mixture of the 2 species. It contains a minimum of 0.3% for the sum of caffeoylmalic acid and chlorogenic acid expressed as chlorogenic acid ($C_{16}H_{18}O_9$ = 354.3), calculated on the dried basis.
Ph. Eur. 6.2 (Common Stinging Nettle for Homoeopathic Preparations). The whole, fresh, flowering plant of *Urtica dioica*. Protect from light.
BP 2008 (Urtica Urens Herb for Homeopathic Preparations). Fresh leaves and flowers of *Urtica urens*. The plant produces an itchy, burning sensation.
USP 31 (Stinging Nettle). The dried roots and rhizomes of *Urtica dioica* (Urticaceae), and may contain *Urtica urens*, known in commerce as dwarf nettle, as a minor component. It contains not less than 0.8% of total amino acids, not less than 0.05% of sitosterol, and not less than 0.003% scopoletin ($C_{10}H_8O_4$ = 192.2), calculated on the dried basis. Store in airtight containers. Protect from light.

Profile

Urtica (*Urtica dioica*) has been used in herbal medicine, mainly for urinary-tract and rheumatic disorders. *Urtica urens* has been used similarly.

Homoeopathy. Urtica has been used in homoeopathic medicines under the following names: Common stinging nettle; Urtica dioica; Urtica urens; Urt. u.

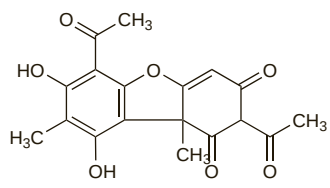
Preparations

Proprietary Preparations (details are given in Part 3)
Austria: Uro-POS; ***Braz.:*** Imuno Max; ***Cz.:*** Koprivovy Caj, Koprivova Nat; Zihlava†; ***Ger.:*** Arthrodynat N†; Asendra†; Azuprostat Urtica†; Bazoton; Flexal Brennessel; Hox Alpha; Natu-lind; Natu-prosta; Pro-Sabona Uno†; Prosta-Truw; Prostaforton; Prostagalen; Prostaherb N; Prostamed Urtica; Prostaneurin†; Prostata; Prostawern; Rheuma-Hek; Rheuma-Kapseln; Rheuma-Stada†; Serless†; Uro-POS; Urol pros; Urticaprostat uno†; Urtipret†; Urtivit; utk; ***Pol.:*** Prostaherb N; Urtix; ***Switz.:*** Valverde Prostate capsules.

Multi-ingredient: ***Austral.:*** Cough Relief†; Extralife Flow-Care; Haemo-Red Formula; Infant Tonic†; Irontona; Urapro†; Vitatona; ***Austria:*** Anaemodoron; Berggeist; Florissamin†; Florissamol†; Menodoron; Mentopin; Prostagutt; Prostatonin; Species Carvi comp†; ***Braz.:*** Prostem Plus; ***Canad.:*** Allercept†; Ultra Quercitin; ***Cz.:*** Abfuhr-Heilkrautertee†; Diabeticka Cajova Smes-Megadiabetin; Nephrosal†; Perospir†; Prostakan Forte; Prostatonin†; Pulmoran; Species Urologicae Planta; Stoffwechseltee N†; ***Fr.:*** Fitacnol†; ***Ger.:*** Combudoron; Presselin Nieren-Blasen K 3†; Prostagutt forte; Prostatin F†; Uvirgan N†; Vollmers praparierter gruner N; Winar†; ***Hong Kong:*** Calmiderm; ***Ital.:*** Biothymus DS; Pluvio; Prostaplant; Sebacnol†; Shamday Antiforfora†; ***Malaysia:*** Cleansa Plus†; Prostakan†; ***Mex.:*** Prosgutt; ***Pol.:*** Alliofil; Herbaton; Immunofort; Naturapia Prostata; Nefrobonisol; Seboren; Urofort; ***Rus.:*** Herbion Urtica (Гербион Уртика); Prostagutt Forte (Простагут Форте); ***S.Afr.:*** Combudoron; Enzian Anaemodoron Drops; Menodoron; ***Spain:*** Natusor Artilane†; ***Switz.:*** Combudoron; Prostagutt-F; Prostatonin; The a l'avoine sauvage de Vollmer; Tisane Diuretique; Tisane pour les problemes de prostate; ***UK:*** Sinose.

Usnea Barbata

Barba de capuchino.
CAS — 125-46-2 (usnic acid).



(usnic acid)

Profile

Usnea barbata is a lichen. It contains usnic acid, which is reported to have antimicrobial activity. *Usnea barbata* extract, usnic acid, and copper usnate have been used in topical preparations.

Adverse effects. Two patients developed severe hepatotoxicity believed to be associated with usnic acid contained in a multi-