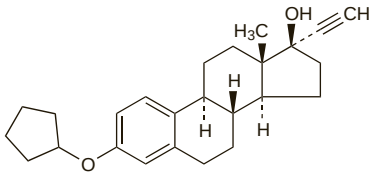
Quinestrol *(BAN, USAN, rINN)*

17α-Ethinyloestradiol 3-cyclopentyl Ether; Quinestrolum; W-3566. 3-Cyclopentyloxy-19-nor-17α-pregna-1,3,5(10)-trien-20-yn-17β-ol.

Хинэстрол

$C_{25}H_{32}O_2$ = 364.5.

CAS — 152-43-2.



Pharmacopoeias. In *Chin.*

Profile

Quinestrol is a synthetic oestrogen that has a prolonged duration of action and is metabolised to ethinylestradiol (p.2101). Quinestrol has been given orally for the treatment of menopausal symptoms and other conditions arising from oestrogen deficiency. It has also been used as the oestrogen component of combined oral contraceptive preparations.

Preparations

Proprietary Preparations (details are given in Part 3)
Arg.: Qui-Lea.
Multi-ingredient: ***Arg.:*** Soluna.

Raloxifene Hydrochloride

(BANM, USAN, rINNM) ⊗

Hidrocloruro de keoxifeno; Hidrocloruro de raloxifeno; Keoxifene Hydrochloride; LY-156758; LY-139481 (raloxifene); Raloksifen Hidroklorür; Raloxifène, chlorhydrate de; Raloxifeni hydrochloridum. 6-Hydroxy-2-(*p*-hydroxyphenyl)benzo[*b*]thien-3-yl-*p*-(2-piperidinoethoxy)phenyl ketone hydrochloride.

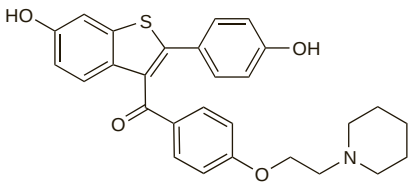
Ралоксифена Гидрохлорид

$C_{28}H_{27}NO_4S,HCl$ = 510.0.

CAS — 84449-90-1 (raloxifene); 82640-04-8 (raloxifene hydrochloride).

ATC — G03XC01.

ATC Vet — QG03XC01.



(raloxifene)

Pharmacopoeias. In *US.*

USP 31 (Raloxifene Hydrochloride). An almost white to pale yellow powder. Very slightly soluble in water, in isopropyl alcohol, and in octanol; slightly soluble in alcohol; sparingly soluble in methyl alcohol; freely soluble in dimethyl sulfoxide; practically insoluble in ether and in ethyl acetate.

Adverse Effects

The most common adverse effects of raloxifene are hot flushes, leg cramps, and a flu-like syndrome. Raloxifene is associated with an increased risk of venous thromboembolic events, particularly during the first 4 months of treatment. Peripheral oedema has also been reported. Rashes, gastrointestinal disturbances, thrombocytopenia, increased blood pressure, headache including migraine, and mild breast symptoms such as pain, enlargement, and tenderness have occurred very rarely.

Incidence of adverse effects. An observational cohort study[1] examined postmarketing adverse events that occurred during raloxifene use in primary care in England. The cohort of 13 987 patients consisted largely of women aged about 62 years, who were receiving raloxifene for the prevention or treatment of osteoporosis. Of the 461 events reported, the most common included flushing, headache or migraine, malaise or lassitude, cramp, oedema, sweating, depression, weight gain, and gastrointestinal disturbances such as nausea, vomiting, dyspepsia, and diarrhoea. Other less common effects included mastalgia and other breast symptoms, vaginal bleeding, thrombophlebitis, and visual disturbances. Rare events included cerebrovascular attack, transient ischaemic attack, pulmonary embolus, deep-vein thrombosis, bullous eruption, leucopenia, thrombocytopenia, upper gastrointestinal haemorrhage, and perforated duodenal ulcer.

The incidence of *cardiovascular* effects associated with raloxifene treatment was examined in the 4-year Multiple Outcomes of Raloxifene Evaluation (MORE), which studied its effects in postmenopausal women with osteoporosis, and in a subsequent 4-year follow-up (Continuing Outcomes Relevant to Evista; CORE).[2] There were 7705 women in the MORE study, of whom 4011 were enrolled in CORE. Overall no significant differences were seen between active treatment and placebo for any cardiovascular event over the 8 years; the calculated incidence was 72 per 10 000 woman-years in those taking raloxifene and 62 per 10 000 in the placebo group. Another large placebo-controlled study[3] (Raloxifene Use for The Heart: RUTH) investigated the cardiovascular effects of raloxifene in postmenopausal women with, or at increased risk of, ischaemic heart disease. After treatment for about 5 years there was no significant difference between the groups for overall cardiovascular events and related deaths. However, in those given raloxifene there was an increased risk of fatal stroke (absolute risk increase, 0.7 per 1000 women-years) and venous thromboembolism (1.2 per 1000 women-years).

1. Layton D, *et al.* Safety profile of raloxifene as used in general practice in England: results of a prescription-event monitoring study. *Osteoporosis Int* 2005; **16:** 490–500.
2. Ensrud K, *et al.* Effect of raloxifene on cardiovascular adverse events in postmenopausal women with osteoporosis. *Am J Cardiol* 2006; **97:** 520–7.
3. Barrett-Connor E, *et al.* Effects of raloxifene on cardiovascular events and breast cancer in postmenopausal women. *N Engl J Med* 2006; **355:** 125–37.

Effects on the liver. Hepatitis, probably associated with the drug, occurred in a woman a month after starting raloxifene.[1] Non-alcoholic steatohepatitis was associated with raloxifene in a woman with minor liver dysfunction, a fatty liver, and a family history of cryptogenic liver cirrhosis.[2] Her liver function worsened during the 3 months after starting the drug, and had returned to baseline 3 months after stopping.

1. Vilches AR, *et al.* Raloxifene-associated hepatitis. *Lancet* 1998; **352:** 1524–5.
2. Takamura T, *et al.* Selective estrogen receptor modulator raloxifene-associated aggravation of nonalcoholic steatohepatitis. *Intern Med* 2007; **46:** 579–81.

Precautions

Raloxifene should be avoided in women with active venous thromboembolism, or a history of thromboembolic disorders. It should be stopped at least 72 hours before periods of prolonged immobilisation, such as post-surgical recovery. Raloxifene should be used with caution in women with risk factors for venous thromboembolism including congestive heart failure or active malignancy, or risk factors for stroke such as transient ischaemic attack or atrial fibrillation. It should be avoided in hepatic and severe renal impairment, and used with caution in moderate renal impairment (but see also Administration in Renal Impairment, below).

Raloxifene had adverse effects in *animal* teratogenicity studies and should not be used in women who are or may become pregnant. It should not be given to women with undiagnosed uterine bleeding. An increase in triglycerides has been reported in some women with a history of hypertriglyceridaemia caused by oestrogen therapy.

Interactions

Colestyramine reduces the absorption and enterohepatic recycling of raloxifene, and they should not be given together. Raloxifene may decrease the efficacy of warfarin.

Fibrates. Cholestasis developed when *fenofibrate* was given to a woman who had been on raloxifene therapy for about 3 years.[1] The authors reviewed other rare reports of liver reactions to either raloxifene or fibrates and suggested that the reaction was likely to be due to an interaction, although the mechanism was not clear.

1. Lucena MI, *et al.* Prolonged cholestasis after raloxifene and fenofibrate interaction: a case report. *World J Gastroenterol* 2006; **12:** 5244–6.

Pharmacokinetics

Raloxifene is absorbed from the gastrointestinal tract and undergoes extensive first-pass hepatic metabolism to the glucuronide conjugates. It is highly bound to plasma proteins, principally albumin and α_1-acid glycoprotein. Raloxifene undergoes enterohepatic recycling, and has a half-life of about 27 hours. It is excreted almost entirely in the faeces.

Renal impairment. The pharmacokinetics of raloxifene are not expected to be affected by renal impairment because the renally excreted fraction is only about 6% in healthy subjects. However, a study[1] in male subjects found the clearance of a single oral dose of 120 mg to be significantly reduced in 10 men with renal impairment (creatinine clearance 24 to 51 mL/minute) compared with a group of 10 with normal renal function. It was calculated that steady-state concentrations could be about 2.3 times higher in patients with renal impairment. The authors suggested that this unexpected observation might be caused by reduced metabolic clearance in the renal tubules or by impairment of biliary excretion of raloxifene glucuronides by uraemic toxins.

1. Czock D, *et al.* Raloxifene pharmacokinetics in males with normal and impaired renal function. *Br J Clin Pharmacol* 2005; **59:** 479–82.

Uses and Administration

Raloxifene hydrochloride is a selective oestrogen receptor modulator; it is a benzothiophene that appears to have oestrogen agonist effects on bone and antagonist effects in uterine and breast tissue. It is used, in oral doses of 60 mg daily, for the prevention and treatment of postmenopausal osteoporosis (below). The same dose is also used to reduce the risk of invasive breast cancer in postmenopausal women who have osteoporosis or are at high risk of invasive breast cancer (below).

◊ Reviews.

1. Khovidhunkit W, Shoback DM. Clinical effects of raloxifene hydrochloride in women. *Ann Intern Med* 1999; **130:** 431–9.
2. Snyder KR, *et al.* Raloxifene hydrochloride. *Am J Health-Syst Pharm* 2000; **57:** 1669–75.
3. Barrett-Connor E. Raloxifene: risks and benefits. *Ann N Y Acad Sci* 2001; **949:** 295–303.
4. Heringa M. Review on raloxifene: profile of a selective estrogen receptor modulator. *Int J Clin Pharmacol Ther* 2003; **41:** 331–45.
5. Trémollières F, Ribot C. Indications du raloxifène chez la femme ménopausée. *Gynecol Obstet Fertil* 2006; **34:** 147–53.

Administration in renal impairment. Although unexpected, renal impairment reduced raloxifene clearance in a pharmacokinetic study (see above). Licensed product information in the UK contra-indicates the use of raloxifene in severe renal impairment, and in the USA it advises caution in moderate and severe impairment. Nevertheless, raloxifene has been studied in postmenopausal women with renal impairment and severe osteopenia or osteoporosis. An oral dose of 60 mg daily for 1 year was given to 25 women on haemodialysis and found to improve bone mineral density of the lumbar spine, compared with 25 women given placebo; there were no reported adverse effects.[1]

1. Hernández E, *et al.* Effects of raloxifene on bone metabolism and serum lipids in postmenopausal women on chronic hemodialysis. *Kidney Int* 2003; **63:** 2269–74.

Hyperparathyroidism. The effects of raloxifene have been reported in postmenopausal women with mild primary hyperparathyroidism (p.1087). Reductions in markers of bone turnover and plasma concentrations of calcium have been described in an observational report[1] and a small, short-term, placebo-controlled study.[2] An increase in bone mineral density was also detected after 12 months of treatment with raloxifene.[1]

1. Zanchetta JR, Bogado CE. Raloxifene reverses bone loss in postmenopausal women with mild asymptomatic primary hyperparathyroidism. *J Bone Miner Res* 2001; **16:** 189–90.
2. Rubin MR, *et al.* Raloxifene lowers serum calcium and markers of bone turnover in postmenopausal women with primary hyperparathyroidism. *J Clin Endocrinol Metab* 2003; **88:** 1174–8.

Malignant neoplasms of the breast. Studies have found raloxifene to be effective for the prophylaxis of breast cancer (p.662). In a placebo-controlled study[1] of postmenopausal women with osteoporosis and no history of breast cancer (MORE), the use of raloxifene for about 3 years reduced the risk of developing breast cancer. This was seen as a reduction in the risk of invasive oestrogen-receptor positive breast cancer, as there was no effect on the risk of oestrogen-receptor negative disease. The reduction in risk was maintained in an extension[2] of this study (CORE) to a total of 8 years of treatment. Similar results were reported in a large placebo-controlled study (RUTH)[3] of postmenopausal women who were treated for about 5 years. Both MORE/CORE[4] and RUTH[3] reported risk reduction in women at either high or low risk, but the MORE study[4] found the effect to be greater in women with a family history of breast cancer.

In a study[5] of postmenopausal women with a predicted increased risk of breast cancer (STAR), prophylactic raloxifene for up to 5 years was found to be as effective as tamoxifen in reducing the risk of invasive breast cancer.

1. Cummings SR, *et al.* The effect of raloxifene on risk of breast cancer in postmenopausal women: results from the MORE randomized trial. *JAMA* 1999; **281:** 2189–97. Correction. *ibid.*; **282:** 2124.
2. Martino S, *et al.* Continuing outcomes relevant to Evista: breast cancer incidence in postmenopausal osteoporotic women in a randomized trial of raloxifene. *J Natl Cancer Inst* 2004; **96:** 1751–61.

3. Barrett-Connor E, *et al.* Effects of raloxifene on cardiovascular events and breast cancer in postmenopausal women. *N Engl J Med* 2006; **355:** 125–37.
4. Lippman ME, *et al.* Effect of raloxifene on the incidence of invasive breast cancer in postmenopausal women with osteoporosis categorized by breast cancer risk. *Clin Cancer Res* 2006; **12:** 5242–7.
5. Vogel VG, *et al.* Effects of tamoxifen vs raloxifene on the risk of developing invasive breast cancer and other disease outcomes: the NSABP Study of Tamoxifen and Raloxifene (STAR) P-2 trial. *JAMA* 2006; **295:** 2727–41. Corrections. *ibid.*; **296:** 2926 and *ibid.* 2007; **298:** 973.

Osteoporosis. Raloxifene partially mimics the effects of oestrogens in bone to increase bone mineral density in postmenopausal women.[1,2] The MORE study in 7705 postmenopausal women with osteoporosis (p.1084) found that up to 4 years of raloxifene treatment reduced the risk of vertebral fracture,[3-6] but the risk of nonvertebral fracture did not differ significantly from that in women given placebo.[3,5] A meta-analysis[7] of data from this and other smaller studies concluded that raloxifene reduced the risk of vertebral fracture by between 40 and 49% in postmenopausal women with osteoporosis.

Small studies suggest that raloxifene may be effective as 'add-back' therapy to prevent the loss of bone mineral density associated with gonadorelin analogue therapy in women[8,9] and men.[10]

1. Clemett D, Spencer CM. Raloxifene: a review of its use in postmenopausal osteoporosis. *Drugs* 2000; **60:** 379–411.
2. Cranney A, Adachi JD. Benefit-risk assessment of raloxifene in postmenopausal osteoporosis. *Drug Safety* 2005; **28:** 721–30.
3. Ettinger B, *et al.* Multiple Outcomes of Raloxifene Evaluation Investigators. Reduction of vertebral fracture risk in postmenopausal women with osteoporosis treated with raloxifene: results from a 3-year randomized clinical trial. *JAMA* 1999; **282:** 637–45.
4. Maricic M, *et al.* Early effects of raloxifene on clinical vertebral fractures at 12 months in postmenopausal women with osteoporosis. *Arch Intern Med* 2002; **162:** 1140–3.
5. Delmas PD, *et al.* Multiple Outcomes of Raloxifene Evaluation Investigators. Efficacy of raloxifene on vertebral fracture risk reduction in postmenopausal women with osteoporosis: four-year results from a randomized clinical trial. *J Clin Endocrinol Metab* 2002; **87:** 3609–17.
6. Qu Y, *et al.* The effect of raloxifene therapy on the risk of new clinical vertebral fractures at three and six months: a secondary analysis of the MORE trial. *Curr Med Res Opin* 2005; **21:** 1955–9.
7. Seeman E, *et al.* Anti-vertebral fracture efficacy of raloxifene: a meta-analysis. *Osteoporos Int* 2006; **17:** 313–16.
8. Palomba S, *et al.* Raloxifene administration in women treated with gonadotropin-releasing hormone agonist for uterine leiomyomas: effects on bone metabolism. *J Clin Endocrinol Metab* 2002; **87:** 4476–81.
9. Palomba S, *et al.* Long-term effectiveness and safety of GnRH agonist plus raloxifene administration in women with uterine leiomyomas. *Hum Reprod* 2004; **19:** 1308–14.
10. Smith MR, *et al.* Raloxifene to prevent gonadotropin-releasing hormone agonist-induced bone loss in men with prostate cancer: a randomized controlled trial. *J Clin Endocrinol Metab* 2004; **89:** 3841–6.

Preparations

USP 31: Raloxifene Hydrochloride Tablets.

Proprietary Preparations (details are given in Part 3)
Arg.: Biofem†; Ciclotran†; Evista; Ketidin; Loxifen†; Oseofem; Raxeto; ***Austral.:*** Evista; ***Austria:*** Evista; ***Belg.:*** Evista; ***Braz.:*** Evista; ***Canad.:*** Evista; ***Chile:*** Evista; ***Cz.:*** Evista; Optruma; ***Denm.:*** Evista; ***Fin.:*** Evista; Optruma†; ***Fr.:*** Evista; Optruma; ***Ger.:*** Evista; Optruma; ***Gr.:*** Evista; ***Hong Kong:*** Evista; ***Hung.:*** Evista; ***India:*** Bonmax; Estroact; Ralista; ***Indon.:*** Evista; ***Irl.:*** Evista; ***Israel:*** Evista; ***Ital.:*** Evista; Optruma; ***Jpn:*** Evista; ***Malaysia:*** Evista; ***Mex.:*** Evista; ***Neth.:*** Evista; Optruma; ***Norw.:*** Evista; ***NZ:*** Evista; ***Philipp.:*** Evista; ***Pol.:*** Evista; ***Port.:*** Evista; Optruma; ***S.Afr.:*** Evista; ***Singapore:*** Evista; ***Spain:*** Evista; Optruma; ***Swed.:*** Evista; ***Switz.:*** Evista; ***Thai.:*** Celvista; ***Turk.:*** Evista; ***UK:*** Evista; ***USA:*** Evista; ***Venez.:*** Evista.

Stanozolol *(BAN, USAN, rINN)* ⊗

Androstanazol; Androstanazole; Estanazol; Estanozolol; Methylstanazole; Metistanazol; NSC-43193; Stanotsololi; Stanozololis; Stanozololum; Stanozolum; Sztanozolol; Win-14833. 17α-Methyl-2′*H*-5α-androst-2-eno[3,2-*c*]pyrazol-17β-ol.

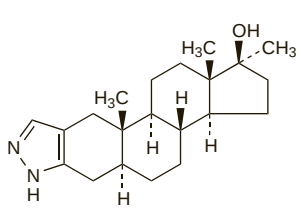
Станозолол

$C_{21}H_{32}N_2O$ = 328.5.

CAS — 10418-03-8.

ATC — A14AA02.

ATC Vet — QA14AA02.



NOTE. The following terms have been used as 'street names' (see p.vi) or slang names for various forms of stanozolol:
Iron Brew.

Pharmacopoeias. In *Chin.* and *US.*

USP 31 (Stanozolol). An odourless crystalline powder occurring in 2 forms; needles melt at about 155° and prisms at about 235°. Insoluble in water; soluble 1 in 41 of alcohol, 1 in 74 of chloroform, and 1 in 370 of ether; slightly soluble in acetone and in ethyl acetate; soluble in dimethylformamide; very slightly soluble in benzene. Store in airtight containers. Protect from light.

Adverse Effects and Precautions

As for androgens and anabolic steroids in general (see Testosterone, p.2130). As with other 17α-alkylated compounds stanozolol may produce hepatotoxicity, and liver function should be monitored. It is probably best avoided in patients with hepatic impairment, and certainly if this is severe. Haematocrit and haemoglobin concentrations should also be monitored.

Because of its androgenic effects it has been recommended that stanozolol should not be used to treat hereditary angioedema in premenopausal women except in life-threatening situations.

Effects on the kidney. Renal failure with cholestatic jaundice has been reported with stanozolol (see below).

Effects on the liver. Cholestatic jaundice has been reported with stanozolol,[1-3] in some cases with acute tubular necrosis and renal failure.[4]

1. Slater SD, *et al.* Jaundice induced by stanozolol hypersensitivity. *Postgrad Med J* 1976; **52:** 229–32.
2. Evely RS, *et al.* Severe cholestasis associated with stanozolol. *BMJ* 1987; **294:** 612–13.
3. Martínez B, *et al.* Colestasis inducida por consumo de estanozolol. *Rev Esp Enferm Dig* 2006; **98:** 219–20.
4. Yoshida EM, *et al.* At what price, glory? Severe cholestasis and acute renal failure in an athlete abusing stanozolol. *Can Med Assoc J* 1994; **151:** 791–3.

Effects on the nervous system. Benign intracranial hypertension developed in an elderly woman receiving stanozolol; CSF pressure returned to normal after stanozolol was stopped.[1]

1. Tully MP, *et al.* Intracranial hypertension associated with stanozolol. *DICP Ann Pharmacother* 1990; **24:** 1234.

Porphyria. Stanozolol is considered to be unsafe in patients with porphyria because it has been shown to be porphyrinogenic in *animals*.

Interactions

As mentioned under Testosterone, p.2131, anabolic steroids may enhance the activity of a number of drugs. For the effect of stanozolol on some anticoagulants, see p.1431.

Uses and Administration

Stanozolol has anabolic and androgenic properties (see Testosterone, p.2131). As with other anabolic steroids, stanozolol has been used for breast cancer in postmenopausal women, and for anaemias, osteoporosis, and catabolic disorders. It has been given in oral doses of 2 mg every 8 to 12 hours, or 50 mg by intramuscular injection every 2 or 3 weeks.

In the management of hereditary angioedema, an initial oral dose of 2.5 to 10 mg daily has been given to prevent attacks. The dosage may then be reduced, according to the patient's response; maintenance doses of 2 mg daily or on alternate days, or 2.5 mg three times weekly have been used successfully. For doses that have been used in children, see below.

Administration in children. Androgens are usually avoided in children with hereditary angioedema (below) because of their adverse effects, but they have been used when other treatments are ineffective. In the USA oral doses of stanozolol 1 mg daily, given only during an attack, have been used in children under 6 years of age, and up to 2 mg daily in those aged 6 to 12 years. Slightly higher doses have been permitted in children in the UK.

Hereditary angioedema. Stanozolol raises serum concentrations of C1 esterase inhibitor and has been used successfully to prevent attacks of hereditary angioedema (p.1081).

References.

1. Bowen T, *et al.* Canadian 2003 international consensus algorithm for the diagnosis, therapy, and management of hereditary angioedema. *J Allergy Clin Immunol* 2004; **114:** 629–37.
2. Zuraw BL. Current and future therapy for hereditary angioedema. *Clin Immunol* 2005; **114:** 10–16.
3. Gompels MM, *et al.* C1 inhibitor deficiency: consensus document. *Clin Exp Immunol* 2005; **139:** 379–94. Correction. *ibid.*; **141:** 189–90. [dose]

Vascular disorders. Stanozolol has been used in the treatment of vascular manifestations of Behçet's syndrome. It has also been reported to promote fibrinolysis in vascular disorders, and has been tried in various conditions. However, most studies have been noncomparative and in small numbers of patients, and results have been variable.

Preparations

BP 2008: Stanozolol Tablets;
USP 31: Stanozolol Tablets.

Proprietary Preparations (details are given in Part 3)
Gr.: Stromba; ***India:*** Menabol; Neurabol; ***Irl.:*** Stromba†; ***Spain:*** Winstrol; ***Thai.:*** Stanol†; ***USA:*** Winstrol†.

Multi-ingredient: ***Thai.:*** Cetabon.

Testis Extracts ⊗

Extractos testiculares; Testicular Extracts.

Тестикулярный Экстракт

Profile

Testis extracts are usually of bovine origin and have been used in a variety of disorders. They have been given to elderly men as androgenic supplements. They have also been used topically, often in preparations containing other mammalian tissue extracts, in the treatment of peripheral circulatory or musculoskeletal disorders.

Preparations

Proprietary Preparations (details are given in Part 3)
Ger.: Orchibion†.

Multi-ingredient: ***Canad.:*** ratio-Heracline; ***Ger.:*** poliomyelan†; tactunerval†; ***Hong Kong:*** Wari-Procomil; ***Thai.:*** Wari-Procomil†.

Testosterone *(BAN, rINN)* ⊗

Testosteron; Testosterona; Testosteronas; Testostérone; Testosteroni; Testosteronum; Tesztoszteron. 17β-Hydroxyandrost-4-en-3-one.

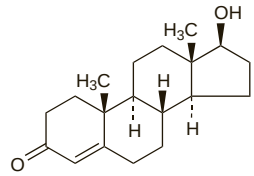
Тестостерон

$C_{19}H_{28}O_2$ = 288.4.

CAS — 58-22-0.

ATC — G03BA03.

ATC Vet — QG03BA03.



NOTE. The following terms have been used as 'street names' (see p.vi) or slang names for various forms of testosterone:
T-Stoff.

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

Ph. Eur. 6.2 (Testosterone). A white or almost white, crystalline powder, or colourless or yellowish-white crystals. Practically insoluble in water and in fatty oils; freely soluble in alcohol and in dichloromethane. Protect from light.

USP 31 (Testosterone). White or slightly creamy-white, odourless, crystals or crystalline powder. Practically insoluble in water; soluble 1 in 6 of dehydrated alcohol, 1 in 2 of chloroform, and 1 in 100 of ether; soluble in dioxan and in vegetable oils. Store at a temperature of 25°, excursions permitted between 15° and 30°.

Testosterone Cipionate *(BANM, rINNM)* ⊗

Cipionato de testosterona; Testostérone, Cipionate de; Testosterone Cyclopentylpropionate; Testosterone Cypionate; Testosteroni Cipionas. 3-Oxoandrost-4-en-17β-yl 3-cyclopentylpropionate; 17β-Hydroxyandrost-4-en-3-one cyclopentanepropionate; 17β-(3-Cyclopentyl-1-oxopropoxy)androst-4-en-3-one.

Тестостерона Ципионат

$C_{27}H_{40}O_3$ = 412.6.

CAS — 58-20-8.

ATC — G03BA03.

ATC Vet — QG03BA03.

Pharmacopoeias. In *US.*

USP 31 (Testosterone Cypionate). A white or creamy-white, crystalline powder, odourless or has a slight odour. Insoluble in water; freely soluble in alcohol, in chloroform, in dioxan, and in ether; soluble in vegetable oils. Protect from light.