Uses and Administration

Temsirolimus is an ester analogue of sirolimus (p.1841) that is given intravenously for the treatment of advanced renal cell carcinoma in a dose of 25 mg once weekly. Treatment may continue until disease progression or toxicity occurs. Temsirolimus is given in 250 mL sodium chloride 0.9% and infused over 30 to 60 minutes, preferably via an infusion pump.

Treatment should be interrupted if the absolute neutrophil count falls below 1000 cells/mm^3, or the platelet count falls below 75 000 cells/mm^3, or if grade 3 toxicity occurs. Once these have resolved to grade 2 or less, temsirolimus may be restarted with the dose reduced by 5 mg weekly, to a dose no lower than 15 mg weekly.

Use of strong inhibitors or inducers of CYP3A4 may increase or decrease exposure to sirolimus, the active metabolite of temsirolimus. If no alternative is available, a dose reduction of temsirolimus to 12.5 mg weekly should be considered if it is given with a strong CYP3A4 inhibitor. Once the inhibitor is stopped, a washout period of about 1 week should be allowed before the dose of temsirolimus is increased back to the original dose. A dose increase of temsirolimus to 50 mg weekly should be considered if it is given with a strong CYP3A4 inducer; once the inducer is stopped, the dose of temsirolimus should be decreased to the original dose.

Temsirolimus is also under investigation for the treatment of mantle cell lymphoma.

◊ References.

1. Anonymous. Temsirolimus: CCI 779, CCI-779, cell cycle inhibitor-779. *Drugs R D* 2004; **5:** 363–7.
2. Mounier N, *et al.* Activitié clinique du CCI779 (temsirolimus), inhibiteur de mTOR. *Bull Cancer* 2006; **93:** 1139–43.
3. Hudes G, *et al.* Temsirolimus, interferon alfa, or both for advanced renal-cell carcinoma. *N Engl J Med* 2007; **356:** 2271–81.
4. Simpson D, Curran MP. Temsirolimus: in advanced renal cell carcinoma. *Drugs* 2008; **68:** 631–8.

Preparations

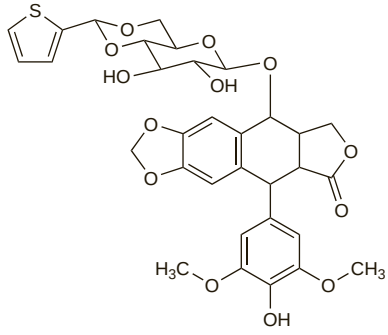
Proprietary Preparations (details are given in Part 3)
Cz.: Torisel; ***Fr.:*** Torisel; ***Port.:*** Torisel; ***USA:*** Torisel.

Teniposide *(BAN, USAN, rINN)*

ETP; NSC-122819; Teniposid; Téniposide; Teniposidi; Tenipósido; Teniposidum; VM-26. (5*S*,5a*R*,8a*S*,9*R*)-5,8,8a,9-Tetrahydro-5-(4-hydroxy-3,5-dimethoxyphenyl)-9-(4,6-*O*-thenylidene-β-D-glucopyranosyloxy)isobenzofuro[5,6-*f*][1,3]benzodioxol-6(5a*H*)-one.

Тенипозид

$C_{32}H_{32}O_{13}S$ = 656.7.
CAS — 29767-20-2.
ATC — L01CB02.
ATC Vet — QL01CB02.



Stability. Precipitation occurred repeatedly in preparations for infusion containing teniposide 200 micrograms/mL in either glucose 5% or sodium chloride 0.9% injection, although previously such preparations had been used uneventfully.[1] Dilution of teniposide solutions to 100 micrograms/mL or less reduced the frequency of the problem, which could not be attributed to a change in formulation and remained unexplained.

1. Strong DK, Morris LA. Precipitation of teniposide during infusion. *Am J Hosp Pharm* 1990; **47:** 512,518.

Adverse Effects, Treatment, and Precautions

As for Etoposide, p.718. There is some evidence that teniposide may be a more potent mutagen and carcinogen than etoposide.

Hypersensitivity. Haemolytic anaemia and acute renal failure with tubular necrosis has been reported in a patient who developed an antibody to teniposide.[1] As with etoposide (p.718) hypersensitivity or infusion reactions occur, sometimes with the first dose, and may be severe;[2,3] the frequency may be as high as 13% in neuroblastoma patients.[2] Although it has been suggested that hypersensitivity reactions might be due to the polyoxyl castor oil in the injection vehicle,[2] studies *in vitro* suggest that it is the drug rather than the vehicle that is responsible.[3]

1. Habibi B, *et al.* Immune hemolytic anemia and renal failure due to teniposide. *N Engl J Med* 1982; **306:** 1091–3.
2. Siddall SJ, *et al.* Anaphylactic reactions to teniposide. *Lancet* 1989; **i:** 394.
3. Carstensen H, *et al.* Teniposide-induced hypersensitivity reactions in children. *Lancet* 1989; **ii:** 55.

Interactions

For a general outline of antineoplastic drug interactions, see p.642.

Antiepileptics. Clearance of teniposide was markedly increased by *phenytoin* or *phenobarbital*; the resultant decrease in systemic exposure to the antineoplastic might reduce its efficacy, and increased dosage would be needed in patients receiving these drugs to guarantee equivalent exposure.[1]

1. Baker DK, *et al.* Increased teniposide clearance with concomitant anticonvulsant therapy. *J Clin Oncol* 1992; **10:** 311–15.

Ciclosporin. Use of ciclosporin with teniposide has been reported[1] to produce a decrease in the clearance of the latter, with increased terminal half-life, peak plasma concentrations, and toxicity.

1. Toffoli G, *et al.* Cyclosporin A as a multidrug-resistant modulator in patients with renal cell carcinoma treated with teniposide. *Br J Cancer* 1997; **75:** 715–21.

Uses and Administration

Teniposide is an antineoplastic agent with general properties similar to those of etoposide (p.718). It has been given alone or with other antineoplastic agents in the treatment of refractory acute lymphoblastic leukaemia (p.651). Teniposide has been tried in solid tumours including neuroblastoma (p.674), and retinoblastoma (p.675).

Teniposide is given by slow intravenous infusion over at least 30 to 60 minutes, as a solution of up to 1 mg/mL in sodium chloride 0.9% injection or glucose 5% injection. Dosage regimens have ranged from 30 mg/m^2 every 5 days, to 180 mg/m^2 weekly, as a single agent. Doses of 165 mg/m^2 twice weekly for 8 or 9 doses with cytarabine, or up to 250 mg/m^2 weekly for 4 to 8 weeks with vincristine and prednisone have been given in the treatment of refractory acute lymphoblastic leukaemia.

Preparations

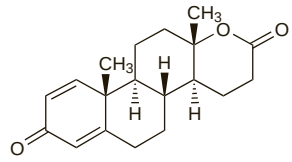
Proprietary Preparations (details are given in Part 3)
Arg.: Vumon; ***Austral.:*** Vumon; ***Austria:*** Vumon; ***Belg.:*** Vumon; ***Braz.:*** Vumon; ***Canad.:*** Vumon; ***Chile:*** Vumon; ***Cz.:*** Vumon†; ***Ger.:*** VM 26; ***Gr.:*** Vumon; ***Hong Kong:*** Vumon; ***Israel:*** Vumon†; ***Ital.:*** Vumon; ***Malaysia:*** Vumon†; ***Mex.:*** Vumon; ***Neth.:*** Vumon; ***NZ:*** Vumon; ***Pol.:*** Vumon; ***Port.:*** Vumon; ***S.Afr.:*** Vumon; ***Singapore:*** Vumon†; ***Spain:*** Vumon; ***USA:*** Vumon.

Testolactone *(USAN, rINN)* ⊗

1-Dehydrotestololactone; NSC-23759; SQ-9538; Testolactona; Testolactonum; Testolakton; Testolaktoni. D-Homo-17a-oxaandrosta-1,4-diene-3,17-dione.

Тестолактон

$C_{19}H_{24}O_3$ = 300.4.
CAS — 968-93-4.



Pharmacopoeias. In *US.*
USP 31 (Testolactone). A white to off-white, practically odourless, crystalline powder. Soluble 1 in 4050 of water; soluble in alcohol and in chloroform; slightly soluble in benzyl alcohol; insoluble in ether and in petroleum spirit. Store in airtight containers.

Profile

Testolactone is a derivative of testosterone (see p.2129). It is reported to be an aromatase inhibitor that reduces peripheral oestrogen synthesis but has no significant androgenic activity. It has been used in the palliative treatment of advanced breast cancer in postmenopausal women (p.661).

The usual oral dose is 250 mg four times daily.

It should not be given to men with breast cancer.

Peripheral neuropathies have occurred in patients given testolactone; gastrointestinal disturbances, pain or oedema of the extremities, hypertension, malaise, maculopapular erythema, and glossitis have also been reported.

Congenital adrenal hyperplasia. For mention of the use of testolactone with flutamide to block androgenic effects in congenital adrenal hyperplasia, see p.1502.

Precocious puberty. Encouraging results have been reported using testolactone in the treatment of 5 girls with precocious puberty (p.2081) due to the McCune-Albright syndrome.[1] Testolactone is an aromatase inhibitor and blocks the synthesis of oestrogens from androgens. Long-term therapy (for up to 5 years) was associated with continued benefit in many patients; however, signs of puberty were not always completely suppressed, in some cases perhaps because of difficulties in maintaining the dosage regimen.[2] Encouraging results were also obtained using testolactone with spironolactone in the treatment of familial precocious puberty in boys, although neither agent was successful when used alone.[3] Again, signs of a reduced response to longer-term therapy have occurred; in this case control was restored by addition of a gonadorelin analogue.[4] Another study[5] in 10 boys who were treated for at least 6 years with spironolactone and testolactone, with deslorelin added at the onset of secondary central precocious puberty, found normalisation in growth rate and bone maturation, and improvements in predicted adult height.

1. Feuillan PP, *et al.* Treatment of precocious puberty in the McCune-Albright syndrome with the aromatase inhibitor testolactone. *N Engl J Med* 1986; **315:** 1115–19.
2. Feuillan PP, *et al.* Long term testolactone therapy for precocious puberty in girls with the McCune-Albright syndrome. *J Clin Endocrinol Metab* 1993; **77:** 647–51.
3. Laue L, *et al.* Treatment of familial male precocious puberty with spironolactone and testolactone. *N Engl J Med* 1989; **320:** 496–502.
4. Laue L, *et al.* Treatment of familial male precocious puberty with spironolactone, testolactone, and deslorelin. *J Clin Endocrinol Metab* 1993; **76:** 151–5.
5. Leschek EW, *et al.* Six-year results of spironolactone and testolactone treatment of familial male-limited precocious puberty with addition of deslorelin after central puberty onset. *J Clin Endocrinol Metab* 1999; **84:** 175–8.

Preparations

USP 31: Testolactone Tablets.

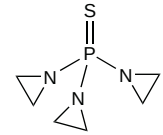
Proprietary Preparations (details are given in Part 3)
Chile: Teslac†; ***Ger.:*** Fludestrin†; ***USA:*** Teslac†.

Thiotepa *(BAN, rINN)*

NSC-6396; TESPA; Thiophosphamide; Thiotépa; Thiotepum; Tiotepa; Triethylenethiophosphoramide; TSPA; WR-45312. Phosphorothioic tri(ethyleneamide); Tris(aziridin-1-yl)phosphine sulphide.

Тиотепа

$C_6H_{12}N_3PS$ = 189.2.
CAS — 52-24-4.
ATC — L01AC01.
ATC Vet — QL01AC01.



Pharmacopoeias. In *Br.*, *Chin.*, *Fr.*, *Jpn*, and *US.*
BP 2008 (Thiotepa). Fine white, crystalline flakes. M.p. 52° to 57°. Freely soluble in water, in alcohol, and in chloroform. Store at 2° to 8°. At higher temperatures it polymerises and becomes inactive.
USP 31 (Thiotepa). Fine white, crystalline flakes, having a faint odour. M.p. 52° to 57°. Soluble 1 in 13 of water, 1 in about 8 of alcohol, 1 in about 2 of chloroform, and 1 in about 4 of ether. Store at 2° to 8° in airtight containers. Protect from light.

Incompatibility. Lyophilised thiotepa 1 mg/mL in glucose 5% was incompatible when mixed with solutions of cisplatin or minocycline hydrochloride.[1]

1. Trissel LA, Martinez JF. Compatibility of thiotepa (lyophilized) with selected drugs during simulated Y-site administration. *Am J Health-Syst Pharm* 1996; **53:** 1041–5.

Stability. A solution of a lyophilised thiotepa preparation 0.5 mg/mL in glucose 5% was considered to be stable (less than 10% loss of thiotepa) for 8 hours at both 4° and 23°.[1] After 24 hours losses ranged between about 10 and 17%. A higher thiotepa concentration (5 mg/mL) was stable for 3 days at 23° and 14 days at 4°. Another study found that solutions containing 1 or 3 mg/mL of thiotepa in sodium chloride 0.9% were stable for 24 hours at 25° and 48 hours at 8°, but solutions containing 0.5% thiotepa needed to be used immediately.[2]

1. Xu QA, *et al.* Stability of thiotepa (lyophilized) in 5% dextrose injection at 4 and 23°C. *Am J Health-Syst Pharm* 1996; **53:** 2728–30.
2. Murray KM, *et al.* Stability of thiotepa (lyophilized) in 0.9% sodium chloride injection. *Am J Health-Syst Pharm* 1997; **54:** 2588–91.

Adverse Effects, Treatment, and Precautions

For general discussions, see Antineoplastics, p.635, p.639, and p.641.

Bone-marrow depression may be delayed; the nadir of white cell and platelet counts may occur up to 30 days after therapy has been stopped. Bone-marrow depression has been reported after intravesical as well as parenteral use, and has occasionally been prolonged or fatal.

Gastrointestinal disturbances, fatigue, weakness, headache and dizziness, hypersensitivity reactions, blurred vision and conjunctivitis may occur. Amenorrhoea and impaired fertility have also been reported. Local irritation, and rarely frank chemical or haemorrhagic cystitis may follow intravesical instillation. Depigmentation of periorbital skin has occurred after the use of thi-

otepa eye drops. As with other alkylating agents, thiotepa is potentially mutagenic, teratogenic, and carcinogenic.

Thiotepa should be given with extreme care, if at all, to patients with pre-existing impairment of hepatic, renal, or bone-marrow function.

Pharmacokinetics

The absorption of thiotepa from the gastrointestinal tract is incomplete and unreliable; variable absorption also occurs from intramuscular injection sites. Absorption through serous membranes such as the bladder and pleura occurs to some extent. After intravenous doses it is rapidly cleared from plasma, with an elimination half-life of about 2.4 hours. It is extensively metabolised: triethylenephosphoramide (TEPA), the primary metabolite, and some of the other metabolites have cytotoxic activity and are eliminated more slowly than the parent compound. It is excreted in the urine: less than 2% of a dose is reported to be present as unchanged drug or its primary metabolite.

Uses and Administration

Thiotepa is an ethyleneimine compound whose antineoplastic effect is related to its alkylating action. It has generally been replaced by cyclophosphamide (p.703) or other drugs. It is not a vesicant and may be given by all parenteral routes, as well as directly into tumour masses.

Instillations of thiotepa may be used in the adjuvant treatment of superficial tumours of the bladder (p.659) and in the control of malignant effusions (p.659). It has been given parenterally in the palliative treatment of various solid tumours, including those of breast and ovary (p.661 and p.670). It has also been given intrathecally to patients with malignant meningeal disease, and has been used, in the form of eye drops, as an adjunct to the surgical removal of pterygium, to prevent recurrence (see p.753).

Thiotepa is given in a variety of dosage schedules. In general, initial doses to suit the individual patient are followed by maintenance doses given at intervals of 1 to 4 weeks. Blood counts are recommended before and during therapy and should continue for at least 3 weeks after stopping. Thiotepa should not be given if the white cell or platelet counts fall below acceptable levels (see also Bone-marrow Depression, p.639) and treatment should be stopped if the white cell count falls rapidly. Dosage should be reduced in patients with lesser degrees of leucopenia.

In the treatment of bladder cancer thiotepa in doses up to 60 mg may be instilled in 30 to 60 mL of sterile water or sodium chloride 0.9% into the bladder of a patient previously dehydrated for 8 to 12 hours, and retained if possible for 2 hours. The instillation may be repeated weekly for up to 4 weeks. Similar instillations have been given at intervals of 1 to 2 weeks, for up to 8 instillations in the prophylaxis of recurrence after surgical removal of bladder cancer. Single doses of 90 mg in 100 mL of sterile water have also been used prophylactically. For malignant effusions, doses of up to 60 mg of thiotepa in 20 to 60 mL of sterile water may be instilled after aspiration; in the USA the licensed dose is 600 to 800 micrograms/kg, a dose similar to that suggested for injection directly into tumours. Thiotepa for local use may be mixed with solutions of procaine and adrenaline.

Intramuscular and intravenous dosage regimens vary considerably; several regimens have used courses of 15 mg daily for 4 days. In the USA a licensed dose is 300 to 400 micrograms/kg given at 1- to 4-week intervals. A solution containing 1 mg/mL in sterile water has been tried intrathecally in doses of up to 10 mg given on alternate days, for up to 4 doses.

Thiotepa 0.05% in sterile Ringer's solution has been instilled as eye drops every 3 hours for up to 6 weeks after surgical removal of pterygium in order to reduce the likelihood of recurrence.

A dose of 60 mg weekly has been instilled into the urethra for the treatment of condylomata acuminata (genital warts). Topical application of thiotepa has also been used for condylomata.

Thiotepa is under investigation for use as conditioning therapy before haematopoietic stem cell transplantation.

Preparations

BP 2008: Thiotepa Injection;
USP 31: Thiotepa for Injection.

Proprietary Preparations (details are given in Part 3)
Gr.: Ledertepa†; ***Ital.:*** Thioplex; ***Neth.:*** Ledertepa; ***Spain:*** Onco Tiotepa†; ***USA:*** Thioplex.

Tioguanine *(BAN, rINN)*

NSC-752; 6-TG; Thioguanine *(USAN)*; 6-Thioguanine; Tioguaniini; Tioguanin; Tioguanina; Tioguaninum; WR-1141. 2-Aminopurine-6(1*H*)-thione; 2-Amino-6-mercaptopurine; 2-Aminopurine-6-thiol.

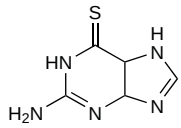
Тиогуанин

$C_5H_5N_5S$ = 167.2.

CAS — 154-42-7 (anhydrous tioguanine); 5580-03-0 (tioguanine hemihydrate).

ATC — L01BB03.

ATC Vet — QL01BB03.



Pharmacopoeias. In *Br.*, *Chin.*, and *US.*
BP 2008 (Tioguanine). A pale yellow, crystalline powder. Practically insoluble in water, in alcohol, and in chloroform; dissolves in dilute solutions of alkali hydroxides.
USP 31 (Thioguanine). It is anhydrous or contains one-half molecule of water of hydration. A pale yellow, odourless or practically odourless, crystalline powder. Insoluble in water and in chloroform; soluble 1 in 7700 of alcohol; freely soluble in dilute solutions of alkali hydroxides. Store in airtight containers.

Adverse Effects, Treatment, and Precautions

As for Mercaptopurine, p.744.

In some patients, gastrointestinal reactions are reported to be less frequent than with mercaptopurine.

Effects on the blood. For the view that it may be possible to predict those individuals likely to have severe bone-marrow depression with tioguanine based on measurement of the activity of thiopurine methyltransferase or the concentration of tioguanine nucleotide, see under Azathioprine, p.1819.

Effects on the liver. The use of tioguanine has been limited by reports of hepatic veno-occlusive disease attributed to the drug.[1-4] The manufacturer (*GlaxoSmithKline*) has stated that, in most cases, liver toxicity is reversible upon withdrawal of chemotherapy. A comparison with mercaptopurine in the maintenance treatment of children with acute lymphoblastic leukaemia found that of 95 patients who developed veno-occlusive disease, 82 were receiving tioguanine, representing about 11% of all patients assigned to the drug.[5] In addition, although tioguanine was associated with fewer CNS relapses, patients in the tioguanine arm were more likely to develop fatal infections.

Centrilobular hepatic necrosis has also been reported; reports are confounded by the use of high doses of tioguanine, other antineoplastics, oral contraceptives, and chronic alcohol abuse.

1. Gill RA, *et al.* Hepatic veno-occlusive disease caused by 6-thioguanine. *Ann Intern Med* 1982; **96:** 58–60.
2. Krivoy N, *et al.* Reversible hepatic veno-occlusive disease and 6-thioguanine. *Ann Intern Med* 1982; **96:** 788.
3. Kao NL, Rosenblate HJ. 6-Thioguanine therapy for psoriasis causing toxic hepatic venoocclusive disease. *J Am Acad Dermatol* 1993; **28:** 1017–18.
4. Romagosa R, *et al.* Treatment of psoriasis with 6-thioguanine and hepatic venoocclusive disease. *J Am Acad Dermatol* 2002; **47:** 970–2.
5. Vora A, *et al.* Medical Research Council/National Cancer Research Network Childhood Leukaemia Working Party. Toxicity and efficacy of 6-thioguanine versus 6-mercaptopurine in childhood lymphoblastic leukaemia: a randomised trial. *Lancet* 2006; **368:** 1339–48.

Handling and disposal. For reference to a method for the destruction of tioguanine in wastes, see Mercaptopurine, p.744.

Interactions

Unlike mercaptopurine (p.744), normal doses of tioguanine may be used with allopurinol.

A number of cases of portal hypertension with hepatic nodular regenerative hyperplasia have been reported in patients who received tioguanine with busulfan (see p.691).

It has been suggested that daunorubicin might enhance the hepatotoxicity of tioguanine (see p.709).

Pharmacokinetics

Tioguanine is incompletely and variably absorbed from the gastrointestinal tract; on average about 30% of a dose is absorbed after oral doses. It is rapidly activated in the body by intracellular conversion to its nucleotide, thioguanylic acid and its thioguanosine phosphate derivatives. With repeated doses increasing amounts of the nucleotide are incorporated into DNA. Very little unchanged tioguanine has been detected circulating in the blood but the half-life of the nucleotide in the tissues is prolonged. Tioguanine is inactivated primarily by methylation to aminomethylthiopurine; small amounts are deaminated to thioxanthine, and may go on to be oxidised by xanthine oxidase to thiouric acid, but inactivation is essentially independent of xanthine oxidase and is not affected by inhibition of the enzyme.

It is excreted in the urine almost entirely as metabolites; only negligible amounts of tioguanine have been detected. Tioguanine does not appear to cross the blood-brain barrier to a significant extent; very little is found in CSF after normal clinical doses. It crosses the placenta.

Uses and Administration

Tioguanine is an analogue of the naturally occurring purine, guanine, and is an antineoplastic with actions and uses similar to those of mercaptopurine (p.744). It appears to cause fewer gastrointestinal reactions but cross-resistance exists so that patients who do not respond to one are unlikely to respond to the other.

Tioguanine may be given orally, usually with other antineoplastics, in the induction of remissions in acute myeloid leukaemia (p.652). It has also been used in other malignancies including acute lymphoblastic leukaemia (p.651) and chronic myeloid leukaemia (p.653).

Doses of between 100 and 200 mg/m^2 daily have been given at various stages of treatment for short term cycles; similar doses have been used in children. A dose of 2 mg/kg daily increased after 4 weeks, if there is no response or toxicity allows, to 3 mg/kg daily may be given to adults and children in those rare cases when single agent therapy is considered appropriate.

Blood counts should be made frequently, particularly during induction and when tioguanine is given with other antineoplastics. Therapy should be withdrawn at the first sign of severe bone-marrow depression. Tioguanine is not recommended for long-term continuous therapy because of the high risk of hepatotoxicity (see Effects on the Liver, above).

Tioguanine has been given intravenously as the sodium salt.

Psoriasis. A report of the use of tioguanine, in doses ranging from 20 mg twice weekly to 120 mg daily, in the management of patients with refractory psoriasis.[1] Dramatic improvement occurred in 14 of 18 patients, but a further 2 were unable to tolerate the drug. Myelosuppression was the principal toxic effect and it was suggested that thiopurine methyltransferase activity could be measured as a basis to determine initial dosage and the risk of toxicity. For the conventional management of psoriasis see p.1583.

1. Mason C, Krueger GG. Thioguanine for refractory psoriasis. *J Am Acad Dermatol* 2001; **44:** 67–72.

Preparations

BP 2008: Tioguanine Tablets;
USP 31: Thioguanine Tablets.

Proprietary Preparations (details are given in Part 3)
Arg.: Lanvis; ***Austral.:*** Lanvis; ***Belg.:*** Lanvis; ***Braz.:*** Lanvis; ***Canad.:*** Lanvis; ***Chile:*** Lanvis; ***Cz.:*** Lanvis; ***Fr.:*** Lanvis; ***Gr.:*** Lanvis; ***Hong Kong:*** Lanvis; ***Irl.:*** Lanvis; ***Israel:*** Lanvis; ***Malaysia:*** Lanvis; ***Neth.:*** Lanvis; ***NZ:*** Lanvis; ***Pol.:*** Lanvis; ***S.Afr.:*** Lanvis; ***Singapore:*** Lanvis†; ***Swed.:*** Lanvis; ***Switz.:*** Lanvis; ***Thai.:*** Lanvis; ***UK:*** Lanvis; ***USA:*** Tabloid.

Tipifarnib *(USAN, rINN)*

R-115777; Tipifarnibum. (+)-6-[(*R*)-Amino(4-chlorophenyl)(1-methyl-1*H*-imidazol-5-yl)methyl]-4-(3-chlorophenyl)-1-methyl-2(1*H*)-quinolinone.

Типифарниб

$C_{27}H_{22}Cl_2N_4O$ = 489.4.

CAS — 192185-72-1.