Pharmacopoeias. In *US*.
USP 31 (Valganciclovir Hydrochloride). A white to off-white powder. Freely soluble in alcohol; practically insoluble in acetone or in ethyl acetate; slightly soluble in hexane; very soluble in isopropyl alcohol. Store in airtight containers at a temperature of 25°, excursions permitted between 15° and 30°.

Stability. References.

1. Anaizi NH, *et al.* Stability of valganciclovir in an extemporaneously compounded oral liquid. *Am J Health-Syst Pharm* 2002; **59:** 1267–70.
2. Henkin CC, *et al.* Stability of valganciclovir in extemporaneously compounded liquid formulations. *Am J Health-Syst Pharm* 2003; **60:** 687–90.

Adverse Effects, Treatment, and Precautions

As for Ganciclovir, p.879.

In the USA, valganciclovir is not indicated for use in liver transplant recipients, because of reports of a higher incidence of tissue-invasive CMV infection compared with patients treated with ganciclovir (although see Cytomegalovirus Infections, below). In the UK, valganciclovir is licensed for use in the prevention of CMV disease in all solid organ graft recipients.

Interactions

As for Ganciclovir, p.879.

Antiviral Action

As for Ganciclovir, p.880.

Pharmacokinetics

Valganciclovir is well absorbed from the gastrointestinal tract after oral doses and is rapidly converted to ganciclovir by first-pass intestinal or hepatic metabolism. The bioavailability of ganciclovir after an oral dose with food is reported to be about 60% and peak plasma concentrations of ganciclovir are achieved after 1 to 3 hours. Valganciclovir is eliminated in the urine as ganciclovir (see p.880).

◊ References.

1. Brown F, *et al.* Pharmacokinetics of valganciclovir and ganciclovir following multiple oral dosages of valganciclovir in HIV- and CMV-seropositive volunteers. *Clin Pharmacokinet* 1999; **37:** 167–76.
2. Jung D, Dorr A. Single-dose pharmacokinetics of valganciclovir in HIV- and CMV-seropositive subjects. *J Clin Pharmacol* 1999; **39:** 800–4.
3. Pescovitz MD, *et al.* Valganciclovir results in improved oral absorption of ganciclovir in liver transplant recipients. *Antimicrob Agents Chemother* 2000; **44:** 2811–15.
4. Wiltshire H, *et al.* Pharmacokinetic profile of ganciclovir after its oral administration and from its prodrug, valganciclovir, in solid organ transplant recipients. *Clin Pharmacokinet* 2005; **44:** 495–507.
5. Winston DJ, *et al.* Pharmacokinetics of ganciclovir after oral valganciclovir versus intravenous ganciclovir in allogeneic stem cell transplant patients with graft-versus-host disease of the gastrointestinal tract. *Biol Blood Marrow Transplant* 2006; **12:** 635–40.

Uses and Administration

Valganciclovir is a prodrug of the antiviral ganciclovir (p.880) that is used for the treatment of CMV retinitis in patients with AIDS, and for the prevention of CMV disease in transplant recipients who have received an organ from a CMV-positive donor (see below).

Valganciclovir is given orally with food as the hydrochloride; doses are expressed in terms of the base. Valganciclovir hydrochloride 1.1 g is equivalent to about 1 g of valganciclovir.

For *induction* in patients with active CMV retinitis, the dose is 900 mg twice daily for 21 days. For *maintenance* following induction, or in patients with inactive CMV retinitis, the dose is 900 mg daily. Patients whose retinitis deteriorates during maintenance may repeat induction but the possibility of viral resistance should be considered. For *prevention* of CMV disease in organ transplant recipients, the dose is 900 mg daily starting within 10 days and continuing until 100 days after transplantation.

Doses of valganciclovir should be reduced in renal impairment (see Administration in Renal Impairment, below).

◊ Reviews.

1. Freeman RB. Valganciclovir: oral prevention and treatment of cytomegalovirus in the immunocompromised host. *Expert Opin Pharmacother* 2004; **5:** 2007–16.
2. Cvetković RS, Wellington K. Valganciclovir: a review of its use in the management of CMV infection and disease in immunocompromised patients. *Drugs* 2005; **65:** 859–78.

Administration in renal impairment. Doses of oral valganciclovir should be reduced in renal impairment according to creatinine clearance (CC). Licensed product information recommends the following doses:

- CC 40 to 59 mL/minute: 450 mg twice daily for *induction* and 450 mg daily for *maintenance* or *prevention*
- CC 25 to 39 mL/minute: 450 mg daily for *induction* and 450 mg every two days for *maintenance* or *prevention*
- CC 10 to 24 mL/minute: 450 mg every two days for *induction* and 450 mg twice weekly for *maintenance* or *prevention*
- haemodialysis patients: not recommended

Cytomegalovirus infections. Valganciclovir produces high systemic concentrations of ganciclovir after oral doses; exposure may be higher than with intravenous ganciclovir regimens.[1] It is therefore active against CMV infections (p.853). It has been shown to be of benefit for both induction therapy and maintenance treatment of CMV retinitis in patients with AIDS,[2,3] and although this has become less widespread in the developed world with the advent of HAART, it continues to be a problem in resource-poor settings in particular; there have been calls for valganciclovir to be made more widely available for treatment in preference to less effective and convenient drugs.[3]

Valganciclovir is also used in the prophylaxis and preemptive treatment of CMV infections in transplant recipients,[4-8] and many centres consider it to be the standard of care for this indication (including in liver transplantation although it is contra-indicated for such use in the USA—see Adverse Effects, Treatment, and Precautions, above).[9]

1. Einsele H, *et al.* Oral valganciclovir leads to higher exposure to ganciclovir than intravenous ganciclovir in patients following allogeneic stem cell transplantation. *Blood* 2006; **107:** 3002–8.
2. Martin DF, *et al.* Valganciclovir Study Group. A controlled trial of valganciclovir as induction therapy for cytomegalovirus retinitis. *N Engl J Med* 2002; **346:** 1119–26. Correction. *ibid.*; **347:** 862.
3. Heiden D, *et al.* Cytomegalovirus retinitis: the neglected disease of the AIDS pandemic. *PLoS Med* 2007; **4:** e334. Available at: http://medicine.plosjournals.org/archive/1549-1676/4/12/pdf/10.1371_journal.pmed.0040334-S.pdf (accessed 28/08/08)
4. Paya C, *et al.* Valganciclovir Solid Organ Transplant Study Group. Efficacy and safety of valganciclovir vs. oral ganciclovir for prevention of cytomegalovirus disease in solid organ transplant recipients. *Am J Transplant* 2004; **4:** 611–20.
5. Khoury JA, *et al.* Prophylactic versus preemptive oral valganciclovir for the management of cytomegalovirus infection in adult renal transplant recipients. *Am J Transplant* 2006; **6:** 2134–43.
6. Said T, *et al.* Oral valgancyclovir versus intravenous gancyclovir for cytomegalovirus prophylaxis in kidney transplant recipients. *Transplant Proc* 2007; **39:** 997–9.
7. Asberg A, *et al.* VICTOR Study Group. Oral valganciclovir is noninferior to intravenous ganciclovir for the treatment of cytomegalovirus disease in solid organ transplant recipients. *Am J Transplant* 2007; **7:** 2106–13.
8. Len O, *et al.* RESITRA. Valganciclovir as treatment for cytomegalovirus disease in solid organ transplant recipients. *Clin Infect Dis* 2008; **46:** 20–7.
9. Snydman DR. Use of valganciclovir for prevention and treatment of cytomegalovirus disease. *Clin Infect Dis* 2008; **46:** 28–9.

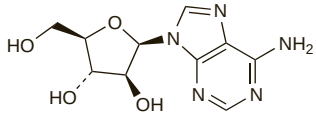
Preparations

Proprietary Preparations (details are given in Part 3)
Arg.: Valixa; ***Austral.:*** Valcyte; ***Austria:*** Valcyte; ***Belg.:*** Valcyte; ***Braz.:*** Valcyte; ***Canad.:*** Valcyte; ***Chile:*** Valixa; ***Cz.:*** Valcyte; ***Denm.:*** Valcyte; ***Fin.:*** Valcyte; ***Fr.:*** Rovalcyte; ***Ger.:*** Valcyte; ***Gr.:*** Valcyte; ***Hong Kong:*** Valcyte; ***Hung.:*** Valcyte; ***Indon.:*** Valcyte; ***Irl.:*** Valcyte; ***Israel:*** Valcyte; ***Ital.:*** Darilin; Valcyte; ***Mex.:*** Valcyte; ***Neth.:*** Valcyte; Valixa; ***Norw.:*** Valcyte; ***NZ:*** Valcyte; ***Philipp.:*** Valcyte; ***Pol.:*** Valcyte; ***Port.:*** Rovalcyte; ***S.Afr.:*** Valcyte; ***Singapore:*** Valcyte; ***Spain:*** Valcyte; ***Swed.:*** Valcyte; ***Switz.:*** Valcyte; ***Thai.:*** Valcyte; ***UK:*** Valcyte; ***USA:*** Valcyte; ***Venez.:*** Valixa.

Vidarabine *(BAN, USAN, rINN)*

Adenine Arabinoside; Ara-A; CI-673; Vidarabiini; Vidarabin; Vidarabina; Vidarabinum. 9-β-D-Arabinofuranosyladenine monohydrate.

Видарабин
$C_{10}H_{13}N_5O_4,H_2O$ = 285.3.
CAS — 5536-17-4 (anhydrous vidarabine); 24356-66-9 (vidarabine monohydrate).
ATC — J05AB03; S01AD06.
ATC Vet — QJ05AB03; QS01AD06.



(anhydrous vidarabine)

Pharmacopoeias. In *US*.
USP 31 (Vidarabine). A white to off-white powder. Very slightly soluble in water; slightly soluble in dimethylformamide. Store in airtight containers.

Vidarabine Phosphate *(BANM, USAN, rINNM)*

Ara-AMP; Arabinosyladenine Monophosphate; CI-808; Fosfato de vidarabina; Vidarabine 5′-Monophosphate; Vidarabine, Phosphate de; Vidarabini Phosphas. 9-β-D-Arabinofuranosyladenine 5′-(dihydrogen phosphate).

Видарабина Фосфат
$C_{10}H_{14}N_5O_7P$ = 347.2.
CAS — 29984-33-6.
ATC — J05AB03; S01AD06.
ATC Vet — QJ05AB03; QS01AD06.

Vidarabine Sodium Phosphate *(BANM, USAN, rINNM)*

CI-808 Sodium; Fosfato sódico de vidarabina; Natrii Vidarabini Phosphas; Vidarabine, Phosphate Sodique de. 9-β-D-Arabinofuranosyladenine 5′-(dihydrogen phosphate) disodium.

Натрия Видарабина Фосфат
$C_{10}H_{12}N_5Na_2O_7P$ = 391.2.
CAS — 71002-10-3.
ATC — J05AB03; S01AD06.
ATC Vet — QJ05AB03; QS01AD06.

Adverse Effects

Adverse effects that may occur when vidarabine is applied to the eyes include irritation, pain, superficial punctate keratitis, photophobia, lachrymation, and occlusion of the lachrymal duct.

Pharmacokinetics

Systemic absorption does not occur after application of vidarabine to the eye; trace amounts of its principal metabolite hypoxanthine arabinoside (arabinosyl hypoxanthine), and vidarabine, if the cornea is damaged, may be found in the aqueous humour.

Uses and Administration

Vidarabine is a purine nucleoside obtained from *Streptomyces antibioticus*. It has been used in the treatment of herpes simplex and varicella-zoster infections (p.854 and p.855), although aciclovir and related drugs are generally preferred.

Vidarabine has been used topically in the treatment of herpes simplex keratitis and keratoconjunctivitis as a 3% ophthalmic ointment.

It has also been used as the sodium phosphate as a 10% gel for the treatment of genital herpes.

Vidarabine was formerly used intravenously in the treatment of severe and disseminated herpes simplex infections and herpes zoster but aciclovir is preferred.

Preparations

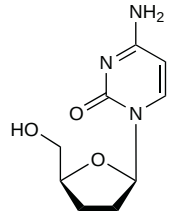
USP 31: Vidarabine Ophthalmic Ointment.

Proprietary Preparations (details are given in Part 3)
Gr.: Erpimycin; Tekarin; Virerpin; ***Jpn:*** Arasena-A.

Zalcitabine *(BAN, USAN, rINN)*

ddC; ddCyd; Dideoxycytidine; NSC-606170; Ro-24-2027; Ro-24-2027/000; Tsalsitabiini; Zalcitabin; Zalcitabina; Zalcitabinum; Zalsitabin. 2′,3′-Dideoxycytidine.

Зальцитабин
$C_9H_{13}N_3O_3$ = 211.2.
CAS — 7481-89-2.
ATC — J05AF03.
ATC Vet — QJ05AF03.



Pharmacopoeias. In *US*.
USP 31 (Zalcitabine). A white to off-white, crystalline powder. Soluble in water and in methyl alcohol; sparingly soluble in alcohol, in acetonitrile, in chloroform, and in dichloromethane; slightly soluble in cyclohexane. Store in airtight containers. Protect from light.

Adverse Effects

The most serious adverse effects of zalcitabine are peripheral neuropathy, which can affect up to one-third of patients, and pancreatitis which is rare, affecting up to about 1% of patients, but which can be fatal. Other severe adverse effects include oral and oesophageal ulceration, hypersensitivity reactions including anaphylaxis, cardiomyopathy and heart failure, lactic acidosis and severe hepatomegaly with steatosis (both potentially life-threatening), and hepatic failure.

Precautions
Zalcitabine should be interrupted or stopped if peripheral neuropathy develops. Neuropathy is usually slowly reversible if treatment is stopped promptly but may be irreversible if treatment is continued after symptoms develop. Zalcitabine should be avoided in patients who already have peripheral neuropathy and used with caution in patients at risk of developing it (especially those with a low CD4+ cell count) or taking other drugs that may cause it (see Interactions, below).

Treatment should be interrupted in patients who develop abdominal pain, nausea, or vomiting or with abnormal biochemical test results until pancreatitis has been excluded. Zalcitabine should be permanently withdrawn if pancreatitis develops. Patients with a history of pancreatitis or of raised serum amylase should be monitored closely. Zalcitabine should not be used with other drugs known to cause pancreatitis (see Interactions, below).

Zalcitabine should be used with caution in patients with hepatic impairment and treatment interrupted or stopped if hepatic function deteriorates or there are signs of hepatic damage or lactic acidosis. It should be used with caution in patients with renal impairment, and dosage reductions may be necessary. It should also be used with caution in patients with cardiomyopathy or heart failure.

Complete blood count and biochemical tests should be carried out before treatment starts and at regular intervals throughout therapy.

Handling. Exposure of the skin to zalcitabine and inhalation of zalcitabine powder should be avoided.

Interactions
Zalcitabine should not be used with other drugs known to cause pancreatitis (for example intravenous pentamidine). Caution is necessary when zalcitabine is given with other drugs that may cause peripheral neuropathy, such as other nucleoside reverse transcriptase inhibitors, chloramphenicol, dapsone, ethionamide, isoniazid (the clearance of which may also be affected—see p.290), metronidazole, nitrofurantoin, ribavirin, and vincristine. Use of zalcitabine with didanosine is not recommended.

The absorption of zalcitabine is reduced by about 25% when given with aluminium- or magnesium-containing antacids.

Cimetidine, probenecid, or trimethoprim can reduce the renal excretion of zalcitabine, resulting in elevated plasma concentrations. Renal excretion of zalcitabine may also be reduced by amphotericin B, aminoglycosides, or foscarnet, potentially increasing its toxicity.

The antiviral action of zalcitabine may be antagonised by lamivudine and the two drugs should not be used together.

Antiviral Action
Zalcitabine is converted intracellularly in stages to the triphosphate. This triphosphate halts the DNA synthesis of retroviruses, including HIV, through competitive inhibition of reverse transcriptase and incorporation into viral DNA.

The emergence of zalcitabine-resistant strains of HIV has been reported.

◊ References.

1. Jeffries DJ. The antiviral activity of dideoxycytidine. *J Antimicrob Chemother* 1989; **23** (suppl A): 29–34.

Pharmacokinetics
Zalcitabine is absorbed from the gastrointestinal tract with a bioavailability of greater than 80%. The rate of absorption is reduced if given with food. Peak plasma concentrations in the fasting state are achieved within about 1 hour. Zalcitabine crosses the blood-brain barrier producing CSF concentrations ranging from 9 to 37% of those in plasma. Binding to plasma proteins is negligible. The plasma elimination half-life is about 2 hours.

Zalcitabine is metabolised intracellularly to the active antiviral triphosphate. It does not appear to undergo any substantial hepatic metabolism and is excreted mainly in the urine, in part by active tubular secretion.

Uses and Administration
Zalcitabine is a nucleoside reverse transcriptase inhibitor derived from cytidine with antiviral activity against HIV. It is used in the treatment of HIV infection and AIDS (p.856). Viral resistance emerges rapidly when zalcitabine is used alone in the treatment of HIV infection, and it is therefore used with other antiretrovirals.

Zalcitabine is given orally in a dose of 750 micrograms every 8 hours. Doses should be reduced in patients with renal impairment (see below).

Administration in renal impairment. Doses of zalcitabine should be reduced for patients with renal impairment according to creatinine clearance (CC):

- CC 10 to 40 mL/minute: 750 micrograms every 12 hours
- CC less than 10 mL/minute: 750 micrograms every 24 hours

Preparations
USP 31: Zalcitabine Tablets.

Proprietary Preparations (details are given in Part 3)
Arg.: Hivid†; Inxibir†; ***Austral.:*** Hivid; ***Austria:*** Hivid; ***Belg.:*** Hivid; ***Braz.:*** Hivid†; ***Canad.:*** Hivid; ***Chile:*** Hivid†; ***Cz.:*** Hivid†; ***Denm.:*** Hivid†; ***Fin.:*** Hivid; ***Fr.:*** Hivid†; ***Ger.:*** Hivid; ***Gr.:*** Hivid; ***Hong Kong:*** Hivid†; ***Irl.:*** Hivid†; ***Israel:*** Hivid; ***Ital.:*** Hivid; ***Jpn:*** Hivid; ***Mex.:*** Arlevid; Hivid; ***Neth.:*** Hivid; ***Port.:*** Hivid†; ***S.Afr.:*** Hivid†; ***Singapore:*** Hivid†; ***Spain:*** Hivid; ***Swed.:*** Hivid†; ***Switz.:*** Hivid†; ***Thai.:*** Hivid; ***Turk.:*** Hivid; ***UK:*** Hivid†; ***USA:*** Hivid†; ***Venez.:*** Hivid.

Zanamivir *(BAN, USAN, rINN)*

GG-167; GR-121167X; 4-Guanidino-2,4-dideoxy-2,3-dehydro-*N*-acetylneuraminic Acid; Tsanamiviiri; Zanamivirum. 5-Acetamido-2,6-anhydro-3,4,5-trideoxy-4-guanidino-D-*glycero*-D-*galacto*-non-2-enonic acid.

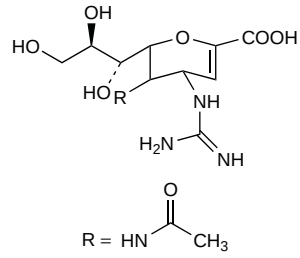
Занамивир

$C_{12}H_{20}N_4O_7$ = 332.3.

CAS — 139110-80-8.

ATC — J05AH01.

ATC Vet — QJ05AH01.



Adverse Effects
Inhaled zanamivir has generally been well tolerated. Acute bronchospasm or decline in respiratory function, with some fatalities, has been reported rarely in patients with a history of respiratory disease and very rarely in those with no such history. Other effects that have been noted include nasal symptoms, headache, gastrointestinal symptoms, cough, and bronchitis, but they may be difficult to distinguish from the symptoms of influenza. There have also been rare reports of hypersensitivity reactions, including oropharyngeal oedema and severe skin rashes.

There have been postmarketing reports (mostly from Japan) of neuropsychiatric adverse effects, such as delirium and abnormal behaviour, in patients taking neuraminidase inhibitors such as zanamivir.

◊ Reviews.

1. Freund B, *et al.* Zanamivir: a review of clinical safety. *Drug Safety* 1999; **21:** 267–81.
2. Gravenstein S, *et al.* Zanamivir: a review of clinical safety in individuals at high risk of developing influenza-related complications. *Drug Safety* 2001; **24:** 1113–25.

Precautions
Zanamivir should be used with caution in patients with chronic respiratory diseases as they may be at increased risk of bronchospasm; if zanamivir use is considered appropriate, patients with asthma or chronic obstructive pulmonary disease should have a fast-acting bronchodilator available during treatment. Patients on maintenance therapy with inhaled bronchodilators should inhale the bronchodilator before zanamivir. Patients experiencing bronchospasm should be advised to stop zanamivir and seek medical attention.

Patients should be monitored for abnormal behaviour throughout the treatment period.

Antiviral Action
Zanamivir inhibits the viral surface enzyme neuraminidase (sialidase) which is essential for the release of newly formed viral particles from infected cells, and may facilitate access of virus through mucus to the cell surface. Zanamivir is active against influenza A and B virus replication.

Resistance. For information on the development of resistance to zanamivir and other neuramidase inhibitors, see under Oseltamivir, p.901.

Pharmacokinetics
Zanamivir is poorly absorbed after oral doses with a bioavailability of about 2%. Inhaled doses produce high local concentrations in the respiratory tract. About 4 to 20% of the inhaled dose is absorbed producing peak serum concentrations at about 1 to 2 hours. Zanamivir is less than 10% bound to plasma protein. It is not metabolized and the absorbed portion is excreted unchanged in the urine with a serum half-life of 2.6 to 5 hours; unabsorbed drug is excreted in the faeces.

◊ References.

1. Aoki FY, Hayden FG (eds.). The pharmacokinetics of zanamivir: a new inhaled antiviral for influenza. *Clin Pharmacokinet* 1999; **36** (suppl 1): 1–58.

Uses and Administration
Zanamivir is a neuraminidase inhibitor used by inhalation for the treatment and prophylaxis (postexposure and seasonal) of influenza A and B (p.859). For *treatment*, it is given to adults in a dose of 10 mg twice daily for 5 days, starting as soon as possible (within 48 hours) after the onset of symptoms.

Zanamivir is given by inhalation for *postexposure prophylaxis* of influenza A and B in household or close contacts and should be started within 36 hours of exposure. The dose for adults is 10 mg once daily for 10 days. For *seasonal prophylaxis* in a community setting 10 mg once daily may be given to adults and adolescents for 28 days and treatment should start within 5 days of an outbreak.

For details of doses in children, see below.

Administration in children. Zanamivir is given by inhalation for the treatment and postexposure prophylaxis of influenza A and B. For *treatment*, children may be given the same dose as adults (10 mg twice daily for 5 days), starting within 36 to 48 hours after the onset of symptoms. In the USA it is approved for those from 7 years of age whereas UK licensed product information permits use from 5 years of age.

Postexposure prophylaxis after close contact with infected patients should be started within 36 hours of exposure. Children from 5 years of age may be given the same dose as adults (10 mg once daily for 10 days).

Influenza. Reviews.[1,2] For further reviews on neuraminidase inhibitors (including zanamivir), see Influenza, under Oseltamivir, p.901.

1. Cheer SM, Wagstaff AJ. Zanamivir: an update of its use in influenza. *Drugs* 2002; **62:** 71–106.
2. Fleming DM. Zanamivir in the treatment of influenza. *Expert Opin Pharmacother* 2003; **4:** 799–805.

Preparations
Proprietary Preparations (details are given in Part 3)
Arg.: Relenza†; ***Austral.:*** Relenza; ***Austria:*** Relenza; ***Belg.:*** Relenza; ***Braz.:*** Relenza; ***Canad.:*** Relenza; ***Chile:*** Relenza†; ***Cz.:*** Relenza; ***Denm.:*** Relenza†; ***Fin.:*** Relenza; ***Fr.:*** Relenza; ***Ger.:*** Relenza; ***Gr.:*** Relenza; ***Hong Kong:*** Relenza; ***Hung.:*** Relenza; ***Irl.:*** Relenza†; ***Israel:*** Relenza†; ***Ital.:*** Relenza; ***Malaysia:*** Relenza; ***Mex.:*** Relenza; ***Neth.:*** Relenza; ***Norw.:*** Relenza; ***NZ:*** Relenza; ***Port.:*** Relenza; ***S.Afr.:*** Relenza; ***Singapore:*** Relenza; ***Spain:*** Relenza; ***Swed.:*** Relenza; ***Switz.:*** Relenza; ***Turk.:*** Relenza; ***UK:*** Relenza; ***USA:*** Relenza.

Zidovudine *(BAN, USAN, rINN)*

Azidodeoxythymidine; Azidothymidine; AZT; BW-A509U; BW-509U; Compound-S; Tsidovudiini; Zidovudin; Zidovudina; Zidovudinas; Zidovudinum; Zydowudyna. 3′-Azido-3′-deoxythymidine.

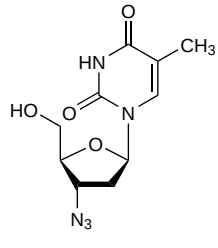
Зидовудин

$C_{10}H_{13}N_5O_4$ = 267.2.

CAS — 30516-87-1.

ATC — J05AF01.

ATC Vet — QJ05AF01.



NOTE. The abbreviation AZT has also been used for azathioprine.

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

Ph. Eur. 6.2 (Zidovudine). A white or brownish powder. It shows polymorphism. Sparingly soluble in water; soluble in dehydrated alcohol. Protect from light.

USP 31 (Zidovudine). A white to yellowish powder. Exhibits polymorphism. Sparingly soluble in water; soluble in alcohol.