tory-tract RSV infection, days of hospitalisation and short-term outcomes have not been affected.[2,15] Due to the small number of patients enrolled in these studies, evaluation of the effects has been difficult. Also, there are some difficulties in giving the drug, and concerns about occupational health and safety, and the high cost. Routine use is not recommended;[6,7] but it may be used for selected infants and children at risk of severe disease and complications. If used, ribavirin should be started early in the course of the disease.[2,3,6]

Antibacterials, although often used in the management of bronchiolitis, are not routinely recommended.[6,7] The results from three small studies[16] suggest that *surfactant* may reduce duration of ventilation and length of intensive care stay.

Prevention of RSV infection involves good infection control practices and use of *RSV immunoglobulin* and a human monoclonal antibody to RSV, *palivizumab*. Both RSV immunoglobulin and palivizumab can be given during an RSV outbreak to prevent serious complications of infection in infants and children considered at high risk. The effectiveness of RSV immunoglobulin[17] and palivizumab[18] were tested in randomised, placebo-controlled clinical studies involving high-risk infants and children (history of prematurity or with bronchopulmonary dysplasia). A 41% overall reduction in hospital admissions was reported in those given RSV immunoglobulin prophylaxis. Prophylaxis with palivizumab resulted in a 55% overall reduction in hospitalisation; reduction rates were 39% and 78% in those with and without bronchopulmonary dysplasia respectively. Respiratory severity scores, hospital days, days of oxygen requirement, and the rate of intensive care admission were also significantly lower in the palivizumab group than for the placebo group. Prophylaxis with palivizumab was also found to reduce post-bronchiolitic wheezing in premature infants.[19] It is recommended by some expert groups for prophylaxis in infants and children at high risk of severe RSV infections.[6,7,20] Vaccines to prevent RSV infection are currently under development.

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5. Fitzgerald DA, Kilham HA. Bronchiolitis: assessment and evidence-based management. *Med J Aust* 2004; **180:** 399–404.
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10. Hartling L, *et al.* Epinephrine for bronchiolitis. Available in The Cochrane Database of Systematic Reviews; Issue 1. Chichester: John Wiley; 2004 (accessed 03/04/08).
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12. Csonka P, *et al.* Oral prednisolone in the acute management of children age 6 to 35 months with viral respiratory infection-induced lower airway disease: a randomized, placebo-controlled trial. *J Pediatr* 2003; **143:** 725–30.
13. Corneli HM, *et al.* Bronchiolitis Study Group of the Pediatric Emergency Care Applied Research Network (PECARN). A multicenter, randomized, controlled trial of dexamethasone for bronchiolitis. *N Engl J Med* 2007; **357:** 331–9.
14. Blom D, *et al.* Inhaled corticosteroids during acute bronchiolitis in the prevention of post-bronchiolitic wheezing. Available in The Cochrane Database of Systematic Reviews; Issue 1. Chichester: John Wiley; 2007 (accessed 03/04/08).
15. Ventre K, Randolph AG. Ribavirin for respiratory syncytial virus infection of the lower respiratory tract in infants and young children. Available in The Cochrane Database of Systematic Reviews; Issue 1. Chichester: John Wiley; 2007 (accessed 03/04/08).
16. Ventre K, *et al.* Surfactant therapy for bronchiolitis in critically ill infants. Available in The Cochrane Database of Systematic Reviews; Issue 3. Chichester: John Wiley; 2006 (accessed 03/04/08).
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18. The IMpact-RSV Study Group. Palivizumab, a humanized respiratory syncytial virus monoclonal antibody, reduces hospitalization from respiratory syncytial virus infection in high-risk infants. *Pediatrics* 1998; **102:** 531–7.
19. Simoes EA, *et al.* Palivizumab prophylaxis, respiratory syncytial virus, and subsequent recurrent wheezing. *J Pediatr* 2007; **151:** 34–42.
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SARS

Severe acute respiratory syndrome (SARS)[1,2] is a respiratory illness caused by a newly identified coronavirus (SARS-CoV). SARS presents primarily in previously healthy adults although there have been some cases reported in children. SARS-CoV is transmitted by contact or droplets and transmission mainly occurs during the second week of illness. The incubation period for SARS is usually 2 to 10 days but may be as long as 16 days. The disease manifests initially as flu-like prodromal symptoms, usually characterised by fever, malaise, myalgia, headache, and rigors. Cough (initially dry), dyspnoea, and diarrhoea may be present in the first week but are more commonly present in the second week of illness. Severe cases develop rapidly progressive respiratory distress and hypoxia and up to about 20% of patients may require intubation or mechanical ventilation. About 20% of patients develop large volume, watery diarrhoea. The overall fatality rate during the 2002–2003 SARS outbreak was about 9.5%.

There is currently no consensus on the optimal treatment for SARS and treatment recommendations are based on the experience gained during the 2002–2003 SARS outbreak. Guidelines for the **surveillance and management** of SARS have been developed by WHO.[3] In the UK guidelines[4] have been issued for the hospital management of adults with SARS, and others have also been developed by clinicians involved in the SARS outbreak in Hong Kong.[5] Because SARS is indistinguishable from pneumonia caused by viral and bacterial pathogens, empirical antibacterial treatment in accordance with local guidelines for severe community-acquired pneumonia (p.186) is recommended. Fluids and oxygen therapy should be given as required. Other treatments tried have included corticosteroids, ribavirin, interferons, normal immunoglobulins, and the co-formulated HIV-protease inhibitor ritonavir-boosted lopinavir. Corticosteroids, usually with ribavirin, were widely used and the timely use of high-dose corticosteroids may decrease fever, improve radiographic appearances, and reduce oxygen requirements.[6-8] There is, however, concern that high-dose and long-term use of corticosteroids may suppress the patient's immune system resulting in increased viral replication and possible bacterial or fungal superinfection. The UK guidelines recommend that their use be considered in moderate doses in severely ill patients with increased oxygen requirements.[4] Additionally there is no convincing clinical evidence that the use of ribavirin alters clinical outcome and the UK guidelines state that its routine use is not recommended.[4] Although interferon beta shows greater *in-vitro* antiviral activity against SARS-CoV, most experience during the 2002–2003 outbreak was with interferon alfa with or without normal immunoglobulins.[6] An open study[9] using interferon-alfacon-1 and high-dose pulse methylprednisolone reported more rapid improvement in radiographic appearance and oxygenation than corticosteroids alone. Better clinical improvement was reported in patients treated with daily interferon alfa plus high-dose corticosteroids than in those given interferon plus low-dose or limited corticosteroids.[8] The UK guidelines state that no recommendation can be given regarding the use of interferons.[4] Although normal immunoglobulins have been used in SARS their effectiveness cannot be established as they were usually given with other therapies.[6] A preliminary open study[10] with ritonavir-boosted lopinavir in 41 patients with probable SARS and receiving the local standard treatment of ribavirin and corticosteroids, reported an improved outcome at 21 days and reductions in viral load, corticosteroid dose, and the incidence of nosocomial infections.

1. Peiris JSM, *et al.* The severe acute respiratory syndrome. *N Engl J Med* 2003; **349:** 2431–41.
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4. Lim WS, *et al.* The British Thoracic Society, the British Infection Society, and the Health Protection Agency. Hospital management of adults with severe acute respiratory syndrome (SARS) if SARS re-emerges—updated 10 February 2004. *J Infect* 2004; **49:** 1–7. Also available at: http://www.brit-thoracic.org.uk/Portals/0/Clinical%20Information/Severe%20Acute%20Resp%20Syndrome/Guidelines/sars0304.pdf (accessed 03/04/08)
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8. Zhao Z, *et al.* Description and clinical treatment of an early outbreak of severe acute respiratory syndrome (SARS) in Guangzhou, PR China. *J Med Microbiol* 2003; **52:** 715–20.
9. Loutfy MR, *et al.* Interferon alfacon-1 plus corticosteroids in severe acute respiratory syndrome: a preliminary study. *JAMA* 2003; **290:** 3222–8.
10. Chu CM, *et al.* Role of lopinavir/ritonavir in the treatment of SARS: initial virological and clinical findings. *Thorax* 2004; **59:** 252–6.

Warts

Warts are caused by human papillomaviruses. The lesions present in several different forms and can affect any skin site although the hands, feet, and anogenital areas are most frequently affected. Anogenital warts are known as condylomata acuminata. Treatment generally relies on some form of local tissue destruction (see p.1584). Interferons have also been used (see p.891).

Abacavir *(BAN, rINN)*

Abacavirum; Abakaviiri; Abakavir. {(1*S*,4*R*)-4-[2-Amino-6-(cyclopropylamino)-9*H*-purin-9-yl]cyclopent-2-enyl}methanol.

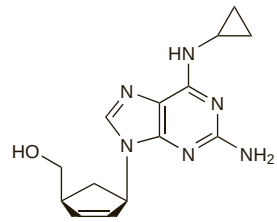
Абакавир

$C_{14}H_{18}N_6O$ = 286.3.

CAS — 136470-78-5.

ATC — J05AF06.

ATC Vet — QJ05AF06.



NOTE. The code 1592U89 has been applied to abacavir but is more properly reserved for abacavir sulfate.

Abacavir Succinate *(BANM, USAN, rINNM)*

Abacavir, Succinate d'; Abacaviri Succinas; Succinato de abacavir.

Абакавира Суксинат

$C_{14}H_{18}N_6O,C_4H_6O_4$ = 404.4.

CAS — 168146-84-7.

ATC — J05AF06.

ATC Vet — QJ05AF06.

NOTE. The code 1592U89 has been applied to abacavir succinate but is more properly reserved for abacavir sulfate.

Abacavir Sulfate *(USAN, rINNM)*

Abacavir, Sulfate d'; Abacavir Sulphate *(BANM)*; Abacaviri Sulfas; Sulfato de abacavir; 1592U89.

Абакавира Сульфат

$(C_{14}H_{18}N_6O)_2,H_2SO_4$ = 670.7.

CAS — 188062-50-2.

ATC — J05AF06.

ATC Vet — QJ05AF06.

NOTE. The code 1592U89 and its abbreviated form, 1592, have also been applied to abacavir and abacavir succinate.

Adverse Effects

The most significant adverse effects associated with antiretroviral regimens containing abacavir are severe hypersensitivity reactions, sometimes fatal, that may occur in up to 9% of patients given abacavir, especially (but not exclusively) during the first 6 weeks of treatment, or during intermittent therapy. Symptoms of hypersensitivity often include fever, rash, cough, dyspnoea, lethargy, malaise, headache, myalgia, and gastrointestinal disturbances, particularly nausea and vomiting, diarrhoea, and abdominal pain. Anaphylaxis has occurred. Caution is needed as hypersensitivity

may be misdiagnosed as influenza, respiratory disease, or gastroenteritis. Erythema multiforme, Stevens-Johnson syndrome, and toxic epidermal necrolysis have occurred rarely. Other adverse effects associated with abacavir include pancreatitis and raised liver enzyme values. Lactic acidosis, sometimes fatal and usually associated with severe hepatomegaly and steatosis, has been reported in patients receiving NRTIs.

Immune reconstitution syndrome (an inflammatory immune response resulting in clinical deterioration) has been reported during the initial phase of treatment with combination antiretroviral therapy, including abacavir, in HIV-infected patients with severe immune deficiency. Accumulation or redistribution of body fat (lipodystrophy) including central obesity, dorsocervical fat enlargement (buffalo hump), peripheral wasting, facial wasting, breast enlargement, and cushingoid appearance have been observed in patients receiving antiretroviral therapy, including abacavir. Metabolic abnormalities such as hypertriglyceridaemia, hypercholesterolaemia, insulin resistance, hyperglycaemia, and hyperlactatemia have also been reported. NRTIs have also been associated with mitochondrial dysfunction such as abnormal behaviour, anaemia, convulsions, hyperlipasaemia, hypertonia, and neutropenia. Elevated creatine phosphokinase, myalgia, myositis, and rarely rhabdomyolysis have been reported, particularly when nucleoside analogues have been given with HIV-protease inhibitors. Osteonecrosis has been reported, particularly in patients with advanced HIV disease or long-term exposure to combination antiretroviral therapy. For further information on adverse effects associated with NRTIs see Zidovudine, p.914.

Effects on the heart. For the possible risk of myocardial infarction in patients taking abacavir, see Effects on the Heart under Adverse Effects of Zidovudine, p.914.

Effects on the skin. Stevens-Johnson syndrome occurring in a patient receiving antiretroviral therapy with abacavir, lamivudine, and zidovudine was probably associated with abacavir.[1] Resolution occurred upon stopping antiretroviral therapy and the condition did not recur upon rechallenge with an alternative regimen also containing lamivudine and zidovudine.

1. Bossi P, *et al.* Stevens-Johnson syndrome associated with abacavir therapy. *Clin Infect Dis* 2002; **35:** 902.

Hypersensitivity. Reviews of hypersensitivity associated with abacavir.[1,2]

1. Hewitt RG. Abacavir hypersensitivity reaction. *Clin Infect Dis* 2002; **34:** 1137–42.
2. Hughes CA, *et al.* Abacavir hypersensitivity reaction: an update. *Ann Pharmacother* 2008; **42:** 387–96.

Precautions

Patients considered to be at increased risk for an abacavir hypersensitivity reaction are those that carry the human leucocyte antigen (HLA) HLA-B(*)5701 allele; screening patients for HLA-B(*)5701 allele before starting treatment with abacavir has been shown to reduce the risk of hypersensitivity reactions. Routine screening of all patients before starting treatment with an abacavir-containing product is therefore recommended. Abacavir should be stopped immediately if symptoms associated with hypersensitivity occur and should *never be restarted* in patients who have stopped therapy due to a hypersensitivity reaction. Patients should be closely monitored for signs of hypersensitivity during the first 2 months of treatment, although hypersensitivity reactions can occur at any time. Patients restarting therapy after an interruption are at particular risk even if they have not previously had symptoms of hypersensitivity. Since intermittent therapy may increase the risk of hypersensitivity developing, patients should be advised of the importance of regular dosing.

Abacavir should not be used in patients with moderate to severe hepatic impairment, and should be used with caution and reduced doses in those with lesser degrees of impairment and those with risk factors for liver disease. Treatment should be stopped if liver function deteriorates rapidly or if hepatomegaly or unexplained metabolic acidosis develop.

Abacavir should be avoided in patients with end-stage renal disease.

Interactions

Use of alcohol with abacavir may result in decreased elimination of abacavir and consequent increases in exposure. Abacavir increases the systemic clearance of oral methadone and patients should be monitored for signs of withdrawal symptoms. The dose of methadone may need to be increased in some patients.

Alcohol. References.

1. McDowell JA, *et al.* Pharmacokinetic interaction of abacavir (1592U89) and ethanol in human immunodeficiency virus-infected adults. *Antimicrob Agents Chemother* 2000; **44:** 1686–90.

Antiviral Action

Abacavir is converted intracellularly in stages to its active form carbovir triphosphate. This halts the DNA synthesis of retroviruses, including HIV, through competitive inhibition of reverse transcriptase and incorporation into viral DNA.

◊ References.

1. Faletto MB, *et al.* Unique intracellular activation of the potent anti-human immunodeficiency virus agent 1592U89. *Antimicrob Agents Chemother* 1997; **41:** 1099–1107.

Pharmacokinetics

Abacavir is rapidly absorbed after oral doses with a bioavailability of about 80%. Absorption is delayed slightly by food but the extent is unaffected. Abacavir crosses the blood-brain barrier. It is about 50% bound to plasma proteins. The elimination half-life is about 1.5 hours after a single dose. Abacavir undergoes intracellular metabolism to the active antiviral metabolite carbovir triphosphate. Elimination is via hepatic metabolism primarily by alcohol dehydrogenase and by glucuronidation and the metabolites are excreted mainly in the urine. There is no significant metabolism by hepatic cytochrome P450 isoenzymes.

◊ References.

1. Kumar PN, *et al.* Safety and pharmacokinetics of abacavir (1592U89) following oral administration of escalating single doses in human immunodeficiency virus type 1-infected adults. *Antimicrob Agents Chemother* 1999; **43:** 603–8.
2. Hughes W, *et al.* Safety and single-dose pharmacokinetics of abacavir (1592U89) in human immunodeficiency virus type 1-infected children. *Antimicrob Agents Chemother* 1999; **43:** 609–15.
3. McDowell JA, *et al.* Multiple-dose pharmacokinetics and pharmacodynamics of abacavir alone and in combination with zidovudine in human immunodeficiency virus-infected adults. *Antimicrob Agents Chemother* 2000; **44:** 2061–7.
4. Izzedine H, *et al.* Pharmacokinetics of abacavir in HIV-1-infected patients with impaired renal function. *Nephron* 2001; **89:** 62–7.
5. Jullien V, *et al.* Abacavir pharmacokinetics in human immunodeficiency virus-infected children ranging in age from 1 month to 16 years: a population analysis. *J Clin Pharmacol* 2005; **45:** 257–64.
6. Yuen GJ, *et al.* A review of the pharmacokinetics of abacavir. *Clin Pharmacokinet* 2008; **47:** 351–71.

Uses and Administration

Abacavir is a nucleoside reverse transcriptase inhibitor with antiretroviral activity against HIV. It is used in the treatment of HIV infection and AIDS (p.856). Viral resistance emerges rapidly when abacavir is used alone, and it is therefore used with other antiretrovirals.

Abacavir is given orally as the sulfate but doses are expressed in terms of the base; 1.17 g of abacavir sulfate is equivalent to about 1 g of abacavir. The adult dose is 300 mg twice daily or 600 mg once daily. For details of doses in children, see below. Doses should be reduced in patients with hepatic impairment (see below).

Fixed-dose combination products have been developed in order to improve patient adherence and avoid monotherapy, thereby decreasing the risk of acquired drug resistance. Products containing abacavir in combination with lamivudine and with lamivudine and zidovudine are available in some countries.

◊ Reviews.

1. Hervey PS, Perry CM. Abacavir: a review of its clinical potential in patients with HIV infection. *Drugs* 2000; **60:** 447–79.
2. Dando TM, Scott LJ. Abacavir plus lamivudine: a review of their combined use in the management of HIV infection. *Drugs* 2005; **65:** 285–302.
3. Castillo SA, *et al.* Long-term safety and tolerability of the lamivudine/abacavir combination as components of highly active antiretroviral therapy. *Drug Safety* 2006; **29:** 811–26.

Administration in children. For the treatment of HIV infection in children 3 months of age and older, abacavir may be given orally as a tablet or solution with other antiretroviral drugs. Doses are based on body-weight:

- 14 to 21 kg: 150 mg (half a tablet) twice daily
- 22 to 29 kg: 150 mg (half a tablet) in the morning and 300 mg (1 tablet) in the evening
- 30 kg or more: 300 mg (1 tablet) twice daily

or

- the solution may be given in a dose of 8 mg/kg twice daily to a maximum dose of 300 mg twice daily

Administration in hepatic impairment. Abacavir should not be used in patients with moderate to severe hepatic impairment, although reduced oral doses of 200 mg twice daily may be given to patients with mild impairment (Child-Pugh score 5 to 6).

Preparations

Proprietary Preparations (details are given in Part 3)
Arg.: Filabac; Finecil; Plusabcir; Zepril; Ziagenavir; ***Austral.:*** Ziagen; ***Austria:*** Ziagen; ***Belg.:*** Ziagen; ***Braz.:*** Ziagenavir; ***Canad.:*** Ziagen; ***Chile:*** Ziagen; ***Cz.:*** Ziagen; ***Denm.:*** Ziagen; ***Fin.:*** Ziagen; ***Fr.:*** Ziagen; ***Ger.:*** Kivexa; Ziagen; ***Gr.:*** Ziagen; ***Hong Kong:*** Ziagen; ***Hung.:*** Ziagen; ***India:*** Abamune; ***Irl.:*** Ziagen; ***Israel:*** Ziagen; ***Ital.:*** Ziagen; ***Mex.:*** Ziagenavir; ***Neth.:*** Ziagen; ***Norw.:*** Ziagen; ***NZ:*** Ziagen; ***Pol.:*** Ziagen; ***Port.:*** Ziagen; ***Rus.:*** Ziagen (Зиаген); ***S.Afr.:*** Ziagen; ***Singapore:*** Ziagen; ***Spain:*** Ziagen; ***Swed.:*** Ziagen; ***Switz.:*** Ziagen; ***Thai.:*** Ziagenavir; ***Turk.:*** Ziagen; ***UK:*** Ziagen; ***USA:*** Ziagen; ***Venez.:*** Ziagen.

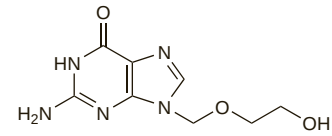
Multi-ingredient: ***Arg.:*** Kivexa; Tricivir; Trivudin; ***Austral.:*** Kivexa; Trizivir; ***Austria:*** Trizivir; ***Belg.:*** Kivexa; Trizivir; ***Canad.:*** Kivexa; Trizivir; ***Chile:*** Kivexa; Tricivir; ***Cz.:*** Kivexa; Trizivir; ***Denm.:*** Kivexa; Trizivir; ***Fin.:*** Kivexa; Trizivir; ***Fr.:*** Kivexa; Trizivir; ***Ger.:*** Trizivir; ***Gr.:*** Kivexa; Trizivir; ***Hong Kong:*** Trizivir; ***Hung.:*** Kivexa; Trizivir; ***Irl.:*** Kivexa; Trizivir; ***Israel:*** Trizivir; ***Ital.:*** Kivexa; Trizivir; ***Mex.:*** Kivexa; Trizivir; ***Neth.:*** Kivexa; Trizivir; ***Norw.:*** Kivexa; Trizivir; ***NZ:*** Kivexa; Trizivir; ***Pol.:*** Kivexa; Trizivir; ***Port.:*** Kivexa; Trizivir; ***Rus.:*** Trizivir (Тризивир); ***S.Afr.:*** Trizivar; ***Singapore:*** Trizivir†; ***Spain:*** Kivexa; Trizivir; ***Swed.:*** Kivexa; Trizivir; ***Switz.:*** Trizivir; ***UK:*** Kivexa; Trizivir; ***USA:*** Epzicom; Trizivir; ***Venez.:*** Trizivir.

Aciclovir *(BAN, rINN)*

Acicloguanosina; Aciclovirum; Aciklovír; Aciklovir; Acikloviras; Acycloguanosine; Acyclovir *(USAN)*; Acyklowir; Asikloviiri; Asiklovir; BW-248U. 9-[(2-Hydroxyethoxy)methyl]guanine; 2-Amino-1,9-dihydro-9-(2-hydroxyethoxymethyl)-6*H*-purin-6-one.

Ацикловир

$C_8H_{11}N_5O_3$ = 225.2.
CAS — 59277-89-3.
ATC — D06BB03; J05AB01; S01AD03.
ATC Vet — QD06BB03; QJ05AB01; QS01AD03.



Pharmacopoeias. In *Chin.*, *Eur.* (see p.vii), and *US.*
Ph. Eur. 6.2 (Aciclovir). A white to almost white crystalline powder. Slightly soluble in water; very slightly soluble in alcohol; freely soluble in dimethyl sulfoxide; soluble in dilute solutions of alkali hydroxides and mineral acids.
USP 31 (Acyclovir). A white to off-white crystalline powder. Slightly soluble in water; insoluble in alcohol; soluble in dilute hydrochloric acid. Store in airtight containers. Protect from light and moisture.

Aciclovir Sodium *(BANM, rINNM)*

Aciclovir sódico; Aciclovir Sodique; Acyclovir Sodium *(USAN)*; Natrii Aciclovirum.

Натрий Ацикловир

$C_8H_{10}N_5NaO_3$ = 247.2.
CAS — 69657-51-8.
ATC — D06BB03; J05AB01; S01AD03.
ATC Vet — QD06BB03; QJ05AB01; QS01AD03.

Incompatibility. Aciclovir is reported to be incompatible with foscarnet.[1,2]

1. Lor E, Takagi J. Visual compatibility of foscarnet with other injectable drugs. *Am J Hosp Pharm* 1990; **47:** 157–9.
2. Baltz JK, *et al.* Visual compatibility of foscarnet with other injectable drugs during simulated Y-site administration. *Am J Hosp Pharm* 1990; **47:** 2075–7.

Stability. A study[1] found that aciclovir sodium solutions prepared with sodium chloride 0.9% and with dextrose 5% were stable for 7 and 21 days respectively when stored at 23°. Solutions stored at 4° were found to be stable for 35 days although subsequent storage at room temperature produced irreversible precipitation. Precipitation may also occur when freshly prepared solutions are refrigerated but the precipitate redissolves at room temperature. US licensed product information recommends that diluted solutions be used within 24 hours of preparation.

1. Zhang Y, *et al.* Stability of acyclovir sodium 1, 7, and 10 mg/mL in 5% dextrose injection and 0.9% sodium chloride injection. *Am J Health-Syst Pharm* 1998; **55:** 574–7.