Sodium Tartrate

Disodium L-Tartrate; E335 (sodium tartrate or monosodium tartrate); Sodu winian; Tartrato de sodio.

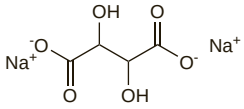
Виннокислый Натрий

$C_2H_4O_2(CO_2Na)_2,2H_2O$ = 230.1.

CAS — 868-18-8 (anhydrous sodium tartrate); 6106-24-7 (sodium tartrate dihydrate).

ATC — A06AD21.

ATC Vet — QA06AD21.



(anhydrous sodium tartrate)

Pharmacopoeias. In *USNF.*

USNF 26 (Sodium Tartrate). Transparent, colourless, odourless crystals. Freely soluble in water; insoluble in alcohol. pH of a 10% solution in water is between 7 and 9. Store in airtight containers.

Profile

Sodium tartrate has been used as an osmotic laxative. It is used as a food additive.

For the general properties of sodium salts, see p.1686.

Preparations

Proprietary Preparations (details are given in Part 3)
Canad.: Limonade Asepta.

Multi-ingredient: ***Arg.:*** Oral-B Enjuague Bucal Amosan†.

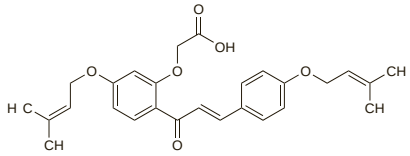
Sofalcone *(pINN)*

Sofalcona; Sofalconum; SU-88. {5-[(3-Methyl-2-butenyl)oxy]-2-{*p*-[(3-methyl-2-butenyl)oxy]cinnamoyl}phenoxy}acetic acid.

Софалькон

$C_{27}H_{30}O_6$ = 450.5.

CAS — 64506-49-6.



Profile

Sofalcone is reported to possess cytoprotective properties and is used in the treatment of gastritis and peptic ulcer disease (p.1702). An oral dose of 100 mg is given 3 times daily.

◊ References.

1. Isomoto H, *et al.* Sofalcone, a mucoprotective agent, increases the cure rate of Helicobacter pylori infection when combined with rabeprazole, amoxicillin and clarithromycin. *World J Gastroenterol* 2005; **11:** 1629–33.

Preparations

Proprietary Preparations (details are given in Part 3)
Jpn: Solon.

Sterculia

E416; Goma esterculia; Indian Tragacanth; Karaya; Karaya Gum; Sterculia Gum; Sterkülia.

Камедь Карайи; Стеркулия Жгучая (*Sterculia urens*)

CAS — 9000-36-6.

ATC — A06AC03.

ATC Vet — QA06AC03.

Pharmacopoeias. In *Br.* and *Fr.*

BP 2008 (Sterculia). The gum obtained from *Sterculia urens* and other species of *Sterculia*. Irregular or vermiform pieces, greyish-white with a brown or pink tinge, with an odour resembling that of acetic acid. It contains not less than 14.0% of volatile acid (or not less than 10.0% if supplied in powdered form), calculated as acetic acid. Sparingly soluble in water, but swells into a homogeneous, adhesive, gelatinous mass; practically insoluble in alcohol. Store at a temperature not exceeding 25°.

Adverse Effects and Precautions

As for Ispaghula, p.1737. There is a risk of intestinal or oesophageal obstruction and faecal impaction, especially if such compounds are swallowed dry. Therefore they should always be taken with sufficient fluid and should not be taken immediately before going to bed. They should be avoided by patients who have difficulty swallowing.

Uses and Administration

Sterculia is used similarly to ispaghula (p.1737) as a bulk laxative and for adjusting faecal consistency. It has also been used as an aid to appetite control in the management of obesity (p.2149) but there is little evidence of efficacy. It is usually taken in the form of granules containing sterculia 62%; the dose is 1 to 2 sachets or 1 to 2 heaped 5 mL spoonfuls orally once or twice daily after meals. (For a dose in children see below.) The granules are washed down without chewing with plenty of water. They may also be taken sprinkled onto soft foods such as yogurt.

Sterculia is used topically, as a paste or powder, for skin protection and sealing in the fitting of ileostomy and colostomy appliances. It has also been used in dental fixative powders, and as an emulsifier and stabiliser in foods.

Administration in children. In the UK, the recommended oral dose of granules, containing sterculia 62%, for constipation in children aged 6 to 12 years is ⁄ to 1 sachet, or ⁄ to one 5 mL spoonful, once or twice daily after meals. Children over 12 years may be dosed as for adults, see Uses and Administration, above. The granules are washed down without chewing with plenty of water. They may also be taken sprinkled onto soft foods such as yogurt.

Preparations

BP 2008: Sterculia Granules.

Proprietary Preparations (details are given in Part 3)
Austral.: Normafibe; ***Belg.:*** Normacol†; ***Braz.:*** Corega; ***Canad.:*** Normacol; ***Fr.:*** Inolaxine†; Normacol; ***Ger.:*** Decorpa†; Granamon†; ***Hong Kong:*** Normacol; ***Irl.:*** Normacol; ***Ital.:*** Normacol; ***Malaysia:*** Normacol†; ***Neth.:*** Normacol; ***NZ:*** Normacol; ***S.Afr.:*** Normacol; ***Singapore:*** Normacol; ***Swed.:*** Inolaxol; ***Switz.:*** Colosan mite; Inolaxine; Normacol; ***Thai.:*** Normacol†; ***UK:*** Normacol.

Multi-ingredient: ***Austral.:*** Alvercol†; Granocol†; Normacol Plus; ***Belg.:*** Normacol Antispasmodique†; Normacol Plus†; ***Fr.:*** Kaologeais; Karayal; Normacol a la Bourdaine†; Poly-Karaya; ***Hong Kong:*** Normacol Plus; ***India:*** Kanormal; ***Irl.:*** Normacol Plus; ***NZ:*** Granocol; Normacol Plus; ***Port.:*** Normacol Plus; ***S.Afr.:*** Alvercol†; Normacol Plus; ***Singapore:*** Normacol Plus; ***Spain:*** Normacol Forte; ***Switz.:*** Colosan plus; Normacol avec bourdaine nouvelle formule†; ***UK:*** Normacol Plus; Spasmonal Fibre†; ***Venez.:*** Polifix†.

Sucralfate *(BAN, USAN, rINN)*

Sucralfato; Sucralfatum; Sukralfaatti; Sükralfat; Sukralfat. Sucrose hydrogen sulphate basic aluminium salt; Sucrose octakis(hydrogen sulphate) aluminium complex; β-D-Fructofuranosyl-α-D-glucopyranoside octakis (hydrogen sulphate) aluminium complex.

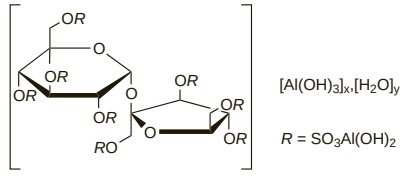
Сукральфат

$C_{12}H_mAl_{16}O_nS_8$.

CAS — 54182-58-0.

ATC — A02BX02.

ATC Vet — QA02BX02.



Pharmacopoeias. In *Chin., Jpn,* and *US.*

USP 31 (Sucralfate). The hydrous basic aluminium salt of sucrose octasulfate. Store in airtight containers.

Adverse Effects and Precautions

Constipation is the most frequently reported adverse effect of sucralfate although diarrhoea, nausea, vomiting, flatulence, or gastric discomfort may also occur. Other adverse effects include dry mouth, dizziness, drowsiness, headache, vertigo, back pain, and skin rashes. Hypersensitivity reactions such as pruritus, oedema, urticaria, respiratory difficulty, rhinitis, laryngospasm, and facial swelling have been reported.

Great caution is needed in patients with renal impairment (below) as absorption and accumulation of aluminium may cause adverse effects.

Bezoar formation. As of March 1999, the UK CSM was aware of 7 reports worldwide of bezoar formation associated with sucralfate use in intensive care patients.[1] It advised caution in the use of sucralfate in seriously ill patients because of the risks of bezoar formation and intestinal obstruction.[1] Patients with delayed gastric emptying or receiving concomitant enteral feeds may be at increased risk. A report by the French Pharmacovigilance System at about the same time made similar recommendations but also contra-indicated the use of sucralfate in premature and immature neonates.[2]

1. Committee on Safety of Medicines/Medicines Control Agency. Bezoar formation with sulcralfate [sic] (Antepsin). *Current Problems* 1999; **25:** 6. Also available at: http://www.mhra.gov.uk/home/idcplg?IdcService=GET_FILE&dDocName=CON2023235&RevisionSelectionMethod=LatestReleased (accessed 07/11/06)
2. Guy C, Ollagnier M. Sucralfate et bézoard: bilan de l'enquête officielle de pharmacovigilance et revue de la littérature. *Therapie* 1999; **54:** 55–8.

Renal impairment. Sucralfate under acid conditions can release aluminium ions that may be absorbed systemically. Significant increases in the urinary excretion of aluminium have been seen in healthy subjects given sucralfate 4 g daily,[1,2] reflecting gastrointestinal absorption of aluminium; aluminium concentrations in serum and urine were significantly higher in patients with chronic renal impairment than in subjects with normal renal function,[3] and similar serum increases have been seen in children with acute renal failure.[4] Aluminium toxicity in patients with normal renal function receiving sucralfate would not be expected, but seizures, muscle weakness, bone pain,[1] and severe aluminium encephalopathy[5] have been reported in patients with end-stage renal disease requiring dialysis. Sucralfate should be used with caution in patients with renal impairment, especially if other aluminium-containing agents are also taken, and such patients should be monitored for signs of aluminium toxicity.[4,6]

1. Robertson JA, *et al.* Sucralfate, intestinal aluminium absorption, and aluminium toxicity in a patient on dialysis. *Ann Intern Med* 1989; **111:** 179–81.
2. Allain P, *et al.* Plasma and urine aluminium concentrations in healthy subjects after administration of sucralfate. *Br J Clin Pharmacol* 1990; **29:** 391–5.
3. Burgess E, *et al.* Aluminum absorption and excretion following sucralfate therapy in chronic renal insufficiency. *Am J Med* 1992; **92:** 471–5.
4. Thorburn K, *et al.* Aluminum accumulation in critically ill children on sucralfate therapy. *Pediatr Crit Care Med* 2001; **2:** 247–9.
5. Withers DJ, *et al.* Encephalopathy in patient taking aluminium-containing agents, including sucralfate. *Lancet* 1989; **ii:** 674.
6. Hemstreet BA. Use of sucralfate in renal failure. *Ann Pharmacother* 2001; **35:** 360–4.

Interactions

Sucralfate may interfere with the absorption of other drugs and it has been suggested that there should be an interval of 2 hours between giving sucralfate and other non-antacid medication. Some of the drugs reported to be affected by sucralfate include cimetidine, ranitidine, digoxin, fluoroquinolone antibacterials, ketoconazole, levothyroxine, phenytoin, tetracycline, quinidine, theophylline, and possibly warfarin. The recommended interval between sucralfate and antacids is 30 minutes. An interval of 1 hour should elapse between giving sucralfate and enteral feeding.

Pharmacokinetics

Sucralfate is only slightly absorbed from the gastrointestinal tract after oral doses. However, there can be some release of aluminium ions and of sucrose sulfate; small quantities of sucrose sulfate may then be absorbed and excreted, primarily in the urine; some absorption of aluminium may also occur (see Renal Impairment, above).

Uses and Administration

Sucralfate is a cytoprotective drug that, under acid gastrointestinal conditions, forms an adherent complex with proteins which coats the gastric mucosa and is reported to have a special affinity for ulcer sites. It also inhibits the action of pepsin and adsorbs bile salts.

Sucralfate has been used in the treatment of peptic ulcer disease (p.1702) and chronic gastritis. It is given orally and should be taken on an empty stomach before meals and at bedtime. The usual dose is 1 g four times daily or 2 g twice daily for 4 to 8 weeks; if necessary the dose may be increased to a maximum of 8 g daily. If longer-term therapy is required sucralfate may be given for up to 12 weeks. Where appropriate a maintenance dose of 1 g twice daily may be given to prevent the recurrence of duodenal ulcers.

For prophylaxis of gastrointestinal haemorrhage from stress ulceration the usual dose of sucralfate is 1 g six times daily; a dose of 8 g daily should not be exceeded. For children's doses see below.

Administration in children. Although sucralfate is not licensed in the UK for use in children under 15 years, the *BNFC* recommends the following oral doses for the treatment of peptic

ulcer disease, or the prophylaxis of stress ulceration in children in intensive care (but see also under Bezoar Formation, above):

- 1 month to 2 years: 250 mg four to six times daily
- 2 to 12 years: 500 mg four to six times daily
- 12 to 18 years: 1 g four to six times daily

The oral suspension blocks fine-bore feeding tubes and tablets should be crushed and dispersed in water instead.

Gastrointestinal bleeding. Sucralfate is an effective drug for the prophylaxis and management of stress-induced gastrointestinal bleeding in severely ill patients but whether it should be chosen over an H_2-antagonist has been subject to debate. One study suggested it might reduce the risk of late-onset pneumonia compared with ranitidine.[1] Another study[2] found ranitidine to be more effective than sucralfate in reducing the risk of gastrointestinal bleeding; while there was a trend towards a lower rate of pneumonia among patients receiving sucralfate, the difference was not significant. However, meta-analyses[3] found an increased risk of pneumonia with ranitidine compared with sucralfate, but no difference in the rate of pneumonia with sucralfate and placebo; insufficient data were available to conduct a meta-analysis of sucralfate's efficacy in terms of rates of bleeding, and a re-assessment of recommendations for the prophylaxis of stress ulcers was called for. Later guidelines[4] concluded that the use of sucralfate did not influence the incidence of ventilator-associated pneumonia compared with placebo. For further discussion of stress ulceration and bleeding, including the use of sucralfate, see under Peptic Ulcer Disease, p.1702. There is also some evidence from a study[5] that sucralfate reduces gastrointestinal bleeding associated with NSAID use, although it does not prevent drug-induced gastric erosion.

In a study to assess whether oral prophylactic sucralfate could ameliorate the symptoms of acute radiation proctitis, sucralfate was found to increase the incidence of rectal bleeding compared with placebo; the cause of this increased bleeding was unclear.[6]

1. Prod'hom G, *et al.* Nosocomial pneumonia in mechanically ventilated patients receiving antacid, ranitidine, or sucralfate as prophylaxis for stress ulcer: a randomized controlled trial. *Ann Intern Med* 1994; **120:** 653–62.
2. Cook D, *et al.* A comparison of sucralfate and ranitidine for the prevention of upper gastrointestinal bleeding in patients requiring mechanical ventilation. *N Engl J Med* 1998; **338:** 791–7.
3. Messori A, *et al.* Bleeding and pneumonia in intensive care patients given ranitidine and sucralfate for prevention of stress ulcer: meta-analysis of randomised controlled trials. *BMJ* 2000; **321:** 1103–6.
4. Dodek P, *et al.* Evidence-based clinical practice guideline for the prevention of ventilator-associated pneumonia. *Ann Intern Med* 2004; **141:** 305–13.
5. Hudson N, *et al.* Effect of sucralfate on aspirin induced mucosal injury and impaired haemostasis in humans. *Gut* 1997; **41:** 19–23.
6. Kneebone A, *et al.* The effect of oral sucralfate on the acute proctitis associated with prostate radiotherapy: a double-blind, randomized trial. *Int J Radiat Oncol Biol Phys* 2001; **51:** 628–35.

Gastro-oesophageal reflux disease. Although sucralfate has been tried for gastro-oesophageal reflux disease (p.1696) the results of studies have been inconsistent.[1-3] However, if lifestyle or dietary measures prove insufficient for the management of heartburn in pregnancy, sucralfate may be considered for first-line therapy.

1. Orlando RC. Sucralfate therapy and reflux esophagitis: an overview. *Am J Med* 1991; **91** (suppl 2A); 123S–124S.
2. Klinkenberg-Knol EC, *et al.* Pharmacological management of gastro-oesophageal reflux disease. *Drugs* 1995; **49:** 695–710.
3. Simon B, *et al.* Sucralfate gel versus placebo in patients with non-erosive gastro-oesophageal reflux disease. *Aliment Pharmacol Ther* 1996; **10:** 441–6.

Mouth ulceration. Sucralfate has been investigated as a mouth rinse in the treatment and prophylaxis of stomatitis induced by cancer chemotherapy[1-3] although evidence of benefit for any drug is ambiguous (see Mucositis, p.640). One study[2] in 40 patients found a significant reduction in symptoms among 23 evaluable patients given sucralfate prophylactically. Seven patients withdrew due to aggravation of chemotherapy-induced nausea. It was suggested that to overcome this problem, the suspension should have a neutral taste, should not be swallowed after rinsing, and that rinsing should not be started until nausea had stopped. However, another study[4] involving 80 patients treated with fluorouracil for colorectal cancer found no significant difference in self-reported mucositis symptoms between patients given sucralfate suspension and those given placebo.

Sucralfate has also been reported to be of benefit in patients with recurrent aphthous stomatitis (mouth ulceration—p.1700). A study involving 21 such patients over 2 years found that topical application of sucralfate suspension 4 times daily was superior to treatment with an antacid (aluminium hydroxide with magnesium hydroxide) or placebo.[5]

In patients with Behçet's syndrome (p.1499), topical sucralfate suspension significantly decreased the frequency, healing time, and pain of oral ulceration, as well as the healing time and pain of genital ulceration,[6] when compared with placebo.

1. Pfeiffer P, *et al.* A prospective pilot study on the effect of sucralfate mouth-swishing in reducing stomatitis during radiotherapy of the oral cavity. *Acta Oncol* 1990; **29:** 471–3.
2. Pfeiffer P, *et al.* Effect of prophylactic sucralfate suspension on stomatitis induced by cancer chemotherapy: a randomized, double-blind cross-over study. *Acta Oncol* 1990; **29:** 171–3.
3. Allison RR, *et al.* Symptomatic acute mucositis can be minimized or prophylaxed [sic] by the combination of sucralfate and fluconazole. *Cancer Invest* 1995; **13:** 16–22.
4. Nottage M, *et al.* Sucralfate mouthwash for prevention and treatment of 5-fluorouracil-induced mucositis: a randomized, placebo-controlled trial. *Support Care Cancer* 2003; **11:** 41–7.
5. Rattan J, *et al.* Sucralfate suspension as a treatment of recurrent aphthous stomatitis. *J Intern Med* 1994; **236:** 341–3.
6. Alpsoy E, *et al.* The use of sucralfate suspension in the treatment of oral and genital ulceration of Behçet disease: a randomized, placebo-controlled, double-blind study. *Arch Dermatol* 1999; **135:** 529–32.

Skin ulceration. Sucralfate has reportedly been applied topically with some success to treat bleeding skin ulcers (p.1585) associated with malignancy,[1] and to promote the healing of venous stasis ulcers.[2] It has been suggested that sucralfate promotes angiogenesis by binding to, and preventing degradation of, basic fibroblast growth factor (bFGF).[2] Topical sucralfate 7% cream was also reported to decrease pain and speed healing of the wound after open surgical removal of haemorrhoids.[3]

1. Regnard CFB. Control of bleeding in advanced cancer. *Lancet* 1991; **337:** 974.
2. Tsakayannis D, *et al.* Sucralfate and chronic venous stasis ulcers. *Lancet* 1994; **343:** 424–5.
3. Gupta PJ, *et al.* Topical sucralfate decreases pain after hemorrhoidectomy and improves healing: a randomized, blinded, controlled study. *Dis Colon Rectum* 2008; **51:** 231–4.

Preparations

USP 31: Sucralfate Tablets.

Proprietary Preparations (details are given in Part 3)
Arg.: Antepsin; Netunal; Sucralmax; ***Austral.:*** Carafate; Ulcyte; ***Austria:*** Citogel; Sucralan; Sucralbene; Sucralstad†; Sucramed; Ulceral; Ulcogant; ***Belg.:*** Ulcogant; ***Braz.:*** Sucrafilm; ***Canad.:*** Novo-Sucralate; Sulcrate; ***Chile:*** Gastrocol†; Mulcatel; Sulcran; ***Cz.:*** Sucralan; Ulcogant; Venter; ***Denm.:*** Antepsin; Hexagastron; ***Fin.:*** Alsucral; Antepsin; ***Fr.:*** Keal; Ulcar; ***Ger.:*** Sucrabest; Sucraphil†; Ulcogant; ***Gr.:*** Peptonorm; Sucrate†; ***Hong Kong:*** Sucari; Ulsanic; ***Hung.:*** Alusulin; Ulcogant; Venter; ***India:*** Alfate; Sucrase; Ulcekon†; ***Indon.:*** Inpepsa; Musin; Neciblok; Ulcumaag; Ulsafate; Ulsicral; Ulsidex; ***Irl.:*** Antepsin; ***Israel:*** Ulsanic; ***Ital.:*** Antepsin; Citogel; Crafilm; Escudo; Gastrogel; Ipagastril†; Sucrager†; Sucral†; Sucralfin; Sucramal; Sucrate; Sucroril; Sugar; Sugast; Sunil; Ulcrast; Zenodian†; ***Jpn:*** Ulcerlmin; ***Malaysia:*** Alsucral; Ulcertec; ***Mex.:*** Apo-Lato; Unival; ***Neth.:*** Ulcogant; ***Norw.:*** Antepsin; ***NZ:*** Carafate; ***Philipp.:*** Iselpin; ***Pol.:*** Ulgastran; Venter; ***Port.:*** Calfate; Cinebil; Sucralum†; Ulcermate; Ulcermin; Ulcimer; ***Rus.:*** Venter (Вентер); ***S.Afr.:*** Ulcetab; Ulsanic; ***Singapore:*** Alsucral; Ulcertec; ***Spain:*** Gastral; Urbal; ***Swed.:*** Andapsin; ***Switz.:*** Ulcogant; ***Thai.:*** Sucraf-en†; Sucral; Sucrate†; Ulcefate; Ulcrafate; Ulsanic; ***Turk.:*** Antepsin; ***UAE:*** Sucralose; ***UK:*** Antepsin; ***USA:*** Carafate; ***Venez.:*** Dip; Exinol; Ulciram; Ulcon.

Multi-ingredient: ***Chile:*** Cicalfate; ***Fr.:*** Cicalfate; ***Gr.:*** Profenil†.

Used as an adjunct in: ***Ital.:*** Ketodol.

Sulfasalazine *(BAN, USAN, rINN)*

Salatsosulfapyridiini; Salazosulfapyridin; Salazosulfapyridine; Salazosulfapyridinum; Salicylazosulfapyridine; SI-88; Sulfasalatsiini; Sülfasalazin; Sulfasalazin; Sulfasalazina; Sulfasalazinas; Sulfasalazinum; Sulfasalazyna; Sulphasalazine; Szulfaszalazin. 4-Hydroxy-4′-(2-pyridylsulphamoyl)azobenzene-3-carboxylic acid.

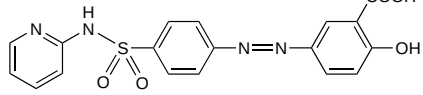
Сульфасалазин

$C_{18}H_{14}N_4O_5S$ = 398.4.

CAS — 599-79-1.

ATC — A07EC01.

ATC Vet — QA07EC01.



Pharmacopoeias. In *Chin.*, *Eur.* (see p.vii), *Int.*, *Jpn*, and *US*.
Ph. Eur. 6.2 (Sulfasalazine). A bright yellow or brownish-yellow, fine powder. Practically insoluble in water and in dichloromethane; very slightly soluble in alcohol; dissolves in dilute solutions of alkali hydroxides. Protect from light.
USP 31 (Sulfasalazine). A fine odourless bright yellow or brownish-yellow powder. Practically insoluble in water, in chloroform, in ether, and in benzene; soluble 1 in 2900 of alcohol, and 1 in 1500 of methyl alcohol; soluble in aqueous solutions of alkali hydroxides. Store in airtight containers. Protect from light.

Adverse Effects and Precautions

Since sulfasalazine is metabolised to sulfapyridine and 5-aminosalicylic acid (mesalazine), its adverse effects and precautions are similar to those of sulfonamides (see Sulfamethoxazole, p.340) and of mesalazine (p.1745). Many adverse effects have been attributed to the sulfapyridine moiety and appear to be more common if serum-sulfapyridine concentrations are greater than 50 micrograms/mL, if the daily dose of sulfasalazine is 4 g or more, or in slow acetylators of sulfapyridine.

The most commonly reported adverse effects include nausea and vomiting, abdominal discomfort, headache, fever, and skin rash.

Adverse effects can be broadly divided into 2 groups:

- dose-related effects are dependent on acetylator phenotype, and largely predictable; this group includes nausea and vomiting, headache, haemolytic anaemia, and methaemoglobinaemia
- hypersensitivity reactions are essentially unpredictable and usually occur at the start of treatment; this group includes skin rash, aplastic anaemia, hepatic and pulmonary dysfunction, and auto-immune haemolysis

Oligospermia, reversible on withdrawal of sulfasalazine, has also been reported. Sulfasalazine treatment may result in yellow-orange discoloration of skin, urine, and other body fluids. Some soft contact lenses may be stained.

Sulfasalazine should not be given to patients with a history of sensitivity to sulfonamides or salicylates. Use in children under 2 years of age is contra-indicated because of the risk of kernicterus.

Blood counts should be performed at the start of therapy and at least once a month for a minimum of the first 3 months of treatment. If a blood dyscrasia is suspected treatment should be stopped immediately and a blood count performed. Patients or their carers should be told how to recognise signs of blood toxicity and should be advised to seek immediate medical attention if symptoms such as fever, sore throat, mouth ulcers, bruising or bleeding develop. Care is advisable in patients with G6PD deficiency because of the risk of haemolytic anaemia.

Sulfasalazine should be used with caution in hepatic or renal impairment. Liver function tests should be carried out at monthly intervals for the first 3 months of treatment. Periodic monitoring of kidney function has also been recommended.

◊ The adverse effects associated with the use of sulfasalazine in patients with inflammatory bowel disease[1] or rheumatoid arthritis have been reviewed.[2,3] The type and incidence of adverse effects appear to be similar in both groups of patients.[2] Although most reactions are minor and patients may continue therapy at the same or reduced dosage, some patients stop treatment because of adverse effects and in these cases a hyposensitisation regimen may be considered.[1,4,5] **Hyposensitisation** should not be attempted in patients with a history of a serious adverse effect such as agranulocytosis, toxic epidermal necrolysis, erythema multiforme, frank haemolysis, or a severe hypersensitivity reaction.[1,4,5] An alternative to hyposensitisation in patients with inflammatory bowel disease who cannot tolerate sulfasalazine is to try a drug that supplies the active 5-aminosalicylic acid component without sulfapyridine, as the latter is thought to be responsible for many of the adverse effects. Examples include balsalazide, mesalazine, and olsalazine; however, some patients still experience hypersensitivity, see under Mesalazine, p.1745.

1. Taffet SL, Das KM. Sulfasalazine: adverse effects and desensitization. *Dig Dis Sci* 1983; **28:** 833–42.
2. Amos RS, *et al.* Sulphasalazine for rheumatoid arthritis: toxicity in 774 patients monitored for one to 11 years. *BMJ* 1986; **293:** 420–3.
3. Farr M, *et al.* Side effect profile of 200 patients with inflammatory arthritides treated with sulphasalazine. *Drugs* 1986; **32** (suppl 1): 49–53.
4. Purdy BH, *et al.* Desensitization for sulfasalazine skin rash. *Ann Intern Med* 1984; **100:** 512–14.
5. Bax DE, Amos RS. Sulphasalazine in rheumatoid arthritis: desensitising the patient with a skin rash. *Ann Rheum Dis* 1986; **45:** 139–40.

Breast feeding. Small amounts of sulfasalazine and its sulfapyridine metabolites are excreted in breast milk; the concentrations of sulfasalazine and total sulfapyridine may be up to 30% and 50% of maternal serum concentrations respectively.[1] Bloody diarrhoea in a breast-fed infant whose mother was taking sulfasalazine 3 g daily has been reported.[2] The mother was a slow acetylator with a relatively high blood concentration of sulfapyridine, which contributed to the appearance of the drug in the infant's blood. Based on this report, the American Academy of Pediatrics[3] considers that sulfasalazine should be given with caution to breast-feeding mothers. However, others consider that continued treatment with sulfasalazine can generally be recommended to breast-feeding mothers of healthy infants.[4] A small study initially involving 17 mother-child pairs concluded sulfasalazine treatment could continue throughout pregnancy and lactation. The study found negligible amounts of sulfasalazine, and its main metabolite sulfapyridine, were transferred to the