USP 31 (Dibasic Sodium Phosphate). It is dried, or contains one, two, seven, or twelve molecules of water of hydration.
The dried substance is a white powder that readily absorbs moisture. It is soluble 1 in 8 of water; insoluble in alcohol.
The heptahydrate is a colourless or white, granular or caked salt that effloresces in warm, dry air. It is freely soluble in water; very slightly soluble in alcohol. Its solutions are alkaline to phenolphthalein, a 0.1M solution having a pH of about 9.
Store all forms in airtight containers.

Equivalence. Each g of dibasic sodium phosphate (anhydrous) represents about 14.1 mmol of sodium and 7.0 mmol of phosphate. Each g of dibasic sodium phosphate (dihydrate) represents about 11.2 mmol of sodium and 5.6 mmol of phosphate. Each g of dibasic sodium phosphate (heptahydrate) represents about 7.5 mmol of sodium and 3.7 mmol of phosphate. Each g of dibasic sodium phosphate (dodecahydrate) represents about 5.6 mmol of sodium and 2.8 mmol of phosphate.

Tribasic Sodium Phosphate

E339; Sodio, fosfato de; Trisodium Orthophosphate; Trisodium Phosphate.

Na_3PO_4 = 163.9.
CAS — 7601-54-9.
ATC — A06AD17; A06AG01.
ATC Vet — QA06AD17; QA06AG01.

Pharmacopoeias. In *USNF*.
USNF 26 (Tribasic Sodium Phosphate). It is anhydrous or contains 1 to 12 molecules of water of hydration. White, odourless crystals or granules, or a crystalline powder. Freely soluble in water; insoluble in alcohol. pH of a 1% solution in water is between 11.5 and 12.0. Store in airtight containers.

Equivalence. Each g of tribasic sodium phosphate (anhydrous) represents about 18.3 mmol of sodium and 6.1 mmol of phosphate.

Adverse Effects and Treatment

Excessive doses of intravenous phosphate cause hyperphosphataemia, particularly in patients with renal failure. Hyperphosphataemia leads in turn to hypocalcaemia, which may be severe, and to ectopic calcification, particularly in patients with initial hypercalcaemia. Tissue calcification may cause hypotension and organ damage and result in acute renal failure. Hyperphosphataemia, hypocalcaemia, and tissue calcification are rare after oral or rectal doses (but see Effects on Electrolytes, and Effects on the Kidneys, below).

Adverse effects of oral phosphates may include nausea, vomiting, diarrhoea, and abdominal pain. When they are being used for indications other than their laxative effects, diarrhoea may necessitate a reduction in dosage. Sodium phosphates given rectally for bowel evacuation may cause local irritation.

Phosphates are given as the potassium or sodium salts or both, and may thus be associated with hyperkalaemia, and hypernatraemia and dehydration. Sodium phosphate may cause hypokalaemia.

Treatment of adverse effects involves withdrawal of phosphate, general supportive measures, and correction of serum-electrolyte concentrations, especially calcium. Measures to remove excess phosphate such as oral phosphate binders and haemodialysis may be required (see also Hyperphosphataemia, p.1669).

Effects on electrolytes. Although less common than after intravenous therapy, hyperphosphataemia, accompanied by hypocalcaemia or other severe electrolyte disturbances and resulting in tetany[1,2] and even death,[2] has been reported after the use of phosphate enemas. Similar effects have also been reported with the use of oral phosphate laxatives,[3-7] and in the USA, the FDA has issued warnings of the risk of electrolyte disturbances after the use of high oral doses of sodium phosphate, particularly in vulnerable patients.[8] Infants or children,[2,9,10] the elderly,[4,11] and those with renal impairment,[1,4,11] or congestive heart failure[4] have often had these adverse effects. Licensed product information for one oral sodium phosphate bowel cleanser (*Visicol; Salix, USA*) states that there have been reports of generalised tonic-clonic seizures and/or loss of consciousness in patients with no history of seizures; these cases were associated with electrolyte abnormalities, and low serum osmolality.

Hyperphosphataemia may precipitate nephrocalcinosis, causing an acute phosphate nephropathy, see Effects on the Kidneys, below.

1. Haskell LP. Hypocalcaemic tetany induced by hypertonic-phosphate enema. *Lancet* 1985; **ii:** 1433.
2. Martin RR, *et al.* Fatal poisoning from sodium phosphate enema: case report and experimental study. *JAMA* 1987; **257:** 2190–2.
3. Peixoto Filho AJ, Lassman MN. Severe hyperphosphatemia induced by a phosphate-containing oral laxative. *Ann Pharmacother* 1996; **30:** 141–3.
4. Adverse Drug Reactions Advisory Committee (ADRAC). Electrolyte disturbances with oral phosphate bowel preparations. *Aust Adverse Drug React Bull* 1997; **16:** 2. Also available at: http://www.tga.gov.au/adr/aadrb/aadr9702.htm (accessed 04/08/08)
5. Ullah N, *et al.* Fatal hyperphosphatemia from a phosphosoda bowel preparation. *J Clin Gastroenterol* 2002; **34:** 457–8.
6. Woo YM, *et al.* A life threatening complication after ingestion of sodium phosphate bowel preparation. *BMJ* 2006; **333:** 589–90.
7. Domico MB, *et al.* Severe hyperphosphatemia and hypocalcemic tetany after oral laxative administration in a 3-month-old infant. *Pediatrics* 2006; **118:** e1580–e1583. Also available at: http://pediatrics.aappublications.org/cgi/reprint/118/5/e1580 (accessed 13/12/06)
8. FDA. Safety of Sodium Phosphates Oral Solution (issued 17th September, 2001). Available at: http://www.fda.gov/cder/drug/safety/sodiumphospate.htm (accessed 18/05/04)
9. McCabe M, *et al.* Phosphate enemas in childhood: cause for concern. *BMJ* 1991; **302:** 1074.
10. Harrington L, Schuh S. Complications of Fleet enema administration and suggested guidelines for use in the pediatric emergency department. *Pediatr Emerg Care* 1997; **13:** 225–6.
11. Boivin MA, Kahn SR. Symptomatic hypocalcemia from oral sodium phosphate: a report of two cases. *Am J Gastroenterol* 1998; **93:** 2577–9.

Effects on the kidneys. Acute renal failure and nephrocalcinosis have been reported after the use of oral phosphate-based cathartics for bowel cleansing.[1,2] Although relatively rare with oral preparations, this acute phosphate nephropathy is a serious adverse effect; most patients were left with chronic renal insufficiency, and some developed end-stage renal disease. Potential contributing factors include inadequate hydration, increased age, a history of hypertension and arteriosclerosis, and concurrent use of ACE inhibitors, angiotensin receptor antagonists, diuretics, or NSAIDs.[2] The FDA has issued warnings[3] about the use of oral sodium phosphate products, especially in patients with impaired renal function, dehydration, or uncorrected electrolyte abnormalities, or in those taking drugs likely to contribute to the risk of nephropathy. The patient should be advised to take the correct dose of oral sodium phosphate, to drink sufficient liquid during bowel cleansing, and to avoid other phosphate-containing laxatives. Patients at increased risk should have their electrolytes and renal function monitored.

Nephrocalcinosis has also been reported in children with hypophosphataemic rickets treated with calcitriol and phosphate supplements; this was found to be associated with the phosphate dose.[4]

1. Desmeules S, *et al.* Acute phosphate nephropathy and renal failure. *N Engl J Med* 2003; **349:** 1006–7.
2. Markowitz GS, *et al.* Acute phosphate nephropathy following oral sodium phosphate bowel purgative: an underrecognized cause of chronic renal failure. *J Am Soc Nephrol* 2005; **16:** 3389–96.
3. FDA. Oral sodium phosphate products for bowel cleansing (issued May 2006). Available at: http://www.fda.gov/cder/drug/InfoSheets/HCP/OSP_solutionHCP.pdf (accessed 11/12/06)
4. Verge CF, *et al.* Effects of therapy in X-linked hypophosphatemic rickets. *N Engl J Med* 1991; **325:** 1843–8.

Local toxicity. Rectal gangrene has been associated with the use of phosphate enemas in elderly patients and was believed to be due to a direct necrotising effect of the phosphate on the rectum.[1]

1. Sweeney JL, *et al.* Rectal gangrene: a complication of phosphate enema. *Med J Aust* 1986; **144:** 374–5.

Precautions

Phosphates should not generally be given to patients with severe renal impairment. They should be avoided in patients who may have low serum-calcium concentrations, as these may decrease further, and in patients with infected phosphate renal calculi. Potassium phosphates should be avoided in patients with hyperkalaemia and sodium phosphates should generally be avoided in patients with congestive heart failure, hypertension, and oedema. Serum electrolytes and renal function should be monitored during therapy, particularly if phosphates are given parenterally.

Oral or rectal sodium phosphate preparations for bowel evacuation should not be used in patients with gastrointestinal obstruction, inflammatory bowel disease, and conditions where there is likely to be increased colonic absorption. They should be used cautiously in elderly and debilitated patients, and in those with pre-existing electrolyte disturbances (see Effects on Electrolytes, above).

Interactions

Oral phosphate supplements should not be used with aluminium, calcium, or magnesium salts as these will bind phosphate and reduce its absorption. Vitamin D increases the gastrointestinal absorption of phosphates and therefore increases the potential for hyperphosphataemia.

Hyperphosphataemia, hypocalcaemia, and hypernatraemia are more likely to occur with phosphate enemas or oral laxatives if these are given to patients receiving diuretics or other drugs that may affect serum electrolytes. The risk of ectopic calcification may be increased by concurrent use of calcium supplements or calcium-containing antacids.

The risk of hyperkalaemia is increased if potassium phosphates are given with drugs that can increase serum-potassium concentrations.

Pharmacokinetics

About two-thirds of ingested phosphate is absorbed from the gastrointestinal tract. Excess phosphate is mainly excreted in the urine, the remainder being excreted in the faeces.

◊ References.

1. Larson JE, *et al.* Laxative phosphate poisoning: pharmacokinetics of serum phosphorus. *Hum Toxicol* 1986; **5:** 45–9.

Human Requirements

Phosphorus requirements are usually regarded as equal to those of calcium.

Most foods contain adequate amounts of phosphate, particularly meat and dairy products, hence deficiency is virtually unknown except in certain disease states, in patients receiving total parenteral nutrition, or in those who have received phosphate-binding drugs for prolonged periods; for further details see under Hypophosphataemia, p.1669.

UK and US recommended dietary intake. In the UK dietary reference values (DRV—see Human Requirements, p.1925)[1] and in the USA dietary reference intakes including recommended dietary allowances (RDA)[2] have been published for phosphorus. In the UK the reference nutrient intake (RNI) for adults is about 550 mg (17.5 mmol) daily; no additional amount is recommended for pregnancy although an additional amount of about 440 mg (14.3 mmol) daily is advised during lactation. In the USA the RDA is 1250 mg daily for those aged 9 to 18 years and 700 mg daily in adults; no increase in RDA is recommended during pregnancy and lactation. A tolerable upper intake level of 4 g daily has been set in adults aged up to 70 years; in those older than 70 a maximum of 3 g daily is recommended.[2]

1. DoH. Dietary reference values for food energy and nutrients for the United Kingdom: report of the panel on dietary reference values of the committee on medical aspects of food policy. *Report on health and social subjects 41.* London: HMSO, 1991.
2. Standing Committee on the Scientific Evaluation of Dietary Reference Intakes of the Food and Nutrition Board. *Dietary Reference Intakes for calcium, phosphorus, magnesium, vitamin D, and fluoride*. Washington, DC: National Academy Press, 1999. Also available at: http://www.nap.edu/openbook.php?isbn=0309063507 (accessed 21/07/08)

Uses and Administration

Phosphates are used in the management of **hypophosphataemia** caused by phosphate deficiency or hypophosphataemic states (p.1669). Doses of up to 100 mmol of phosphate daily may be given orally. The intravenous route is seldom necessary, but a dose of up to 9 mmol of phosphate as monobasic potassium phosphate may be given over 12 hours and repeated every 12 hours as necessary for severe hypophosphataemia. Alternatively, 0.2 to 0.5 mmol/kg phosphate, up to a maximum of 50 mmol, may be given over 6 to 12 hours (see also below). Plasma-electrolyte concentrations, especially phosphate and calcium, and renal function should be carefully monitored. Reduced doses may be necessary in patients with renal impairment. Phosphate supplements are used in total parenteral nutrition regimens; typical daily requirements are 20 to 30 mmol of phosphate.

Phosphates act as mild osmotic **laxatives** (p.1693) when given orally as dilute solutions or by the rectal route. Phosphate enemas or concentrated oral solutions are used for bowel cleansing before surgery or endoscopy procedures. Preparations typically combine monobasic and dibasic sodium phosphates but the composition and dosage do vary slightly. Phosphate enemas act within 2 to 5 minutes, whereas the oral solutions act within 30 minutes to 6 hours.

Phosphates also have **other uses**. They lower the pH of urine and have been given as adjuncts to urinary antibacterials that depend on an acid urine for their activity. Phosphates have also been used for the prophylaxis of calcium renal calculi; the phosphates reduce urinary excretion of calcium thus preventing calcium deposition. A suggested oral dose for both uses is 7.4 mmol of phosphate four times daily.

Butafosfan (1-butylamino-1-methylethylphosphinic acid) and the sodium salt of toldimfos (4-dimethylamino-*O*-tolylphosphinic acid) are used as phosphorus sources in veterinary medicine.

Bowel evacuation. A review concluded that the efficacy and tolerability of oral sodium phosphate solution was generally similar to, or significantly better than, that of polyethylene glycol-based or other colorectal cleansers in patients preparing for colorectal-related procedures.[1]

1. Curran MP, Plosker GL. Oral sodium phosphate solution: a review of its use as a colorectal cleanser. *Drugs* 2004; **64:** 1697–1714.

Hypercalcaemia. Intravenous phosphates have been used to lower plasma-calcium concentrations in hypercalcaemic emergencies (p.1668), but because of their potential to cause serious adverse effects other drugs are now preferred. Oral phosphates may be used to prevent gastrointestinal absorption of calcium in the treatment of hypercalcaemia. The dose in adults is up to 100 mmol phosphate daily adjusted according to response.

Hypophosphataemia. Phosphate salts are given in the management of hypophosphataemia when a phosphate deficiency is identified, as discussed in Uses and Administration, above. Intravenous phosphates are associated with serious adverse effects if hypophosphataemia is over-corrected, and the rise in serum-phosphorus concentration cannot be predicted from a given dose. Consequently, it has been recommended[1-4] that intravenous phosphate be used cautiously in the treatment of severe hypophosphataemia (for the standard rate and dose see Uses and Administration, above). However, some advocate a more aggressive fixed-dose regimen in critically ill patients.[5-7]

1. Vannatta JB, *et al.* Efficacy of intravenous phosphorus therapy in the severely hypophosphataemic patient. *Arch Intern Med* 1981; **141:** 885–7.
2. Anonymous. Treatment of severe hypophosphatemia. *Lancet* 1981; **ii:** 734.
3. Lloyd CW, Johnson CE. Management of hypophosphatemia. *Clin Pharm* 1988; **7:** 123–8.
4. Coyle S, *et al.* Treatment of hypophosphataemia. *Lancet* 1992; **340:** 977.
5. Perreault MM, *et al.* Efficacy and safety of intravenous phosphate replacement in critically ill patients. *Ann Pharmacother* 1997; **31:** 683–8.
6. Miller DW, Slovis CM. Hypophosphatemia in the emergency department therapeutics. *Am J Emerg Med* 2000; **18:** 457–61.
7. Charron T, *et al.* Intravenous phosphate in the intensive care unit: more aggressive repletion regimens for moderate and severe hypophosphatemia. *Intensive Care Med* 2003; **29:** 1273–8.

Osteomalacia. Vitamin D deficiency, or its abnormal metabolism, is the most usual cause of osteomalacia and rickets (p.1084); however, phosphate depletion may also contribute, and phosphate supplementation may be given as appropriate. A suggested oral dose for vitamin-D-resistant hypophosphataemic osteomalacia in adults is 65 to 100 mmol phosphate daily, and for vitamin D-resistant rickets in children is 32 to 48 mmol phosphate daily.

RICKETS OF PREMATURITY. Dietary deficiency of phosphorus is unusual, but can occur in small premature infants fed exclusively on human breast milk. The phosphate intake in these infants appears to be inadequate to meet the needs of bone mineralisation, and hypophosphataemic rickets can develop. It has been proposed that this condition, variably called metabolic bone disease of prematurity, or rickets of prematurity, could be prevented by giving phosphorus supplements to very low-birth-weight babies (less than about 1000 g) fed on breast milk alone.[1] A suggested regimen is to add 10 to 15 mg of phosphorus per 100 mL of feed (as buffered sodium phosphate) until the infant reached 2000 g. Concomitant calcium and vitamin D supplementation are also recommended.[1] A placebo-controlled study[2] in infants weighing less than 1250 g at birth confirmed that phosphate supplements (50 mg daily) could prevent the development of the bone defects of rickets of prematurity.

1. Brooke OG, Lucas A. Metabolic bone disease in preterm infants. *Arch Dis Child* 1985; **60:** 682–5.
2. Holland PC, *et al.* Prenatal deficiency of phosphate, phosphate supplementation, and rickets in very-low-birthweight infants. *Lancet* 1990; **335:** 697–701. Correction. *ibid.*; 1408–9.

Preparations

BP 2008: Dipotassium Hydrogen Phosphate Injection; Phosphates Enema; Sterile Potassium Dihydrogen Phosphate Concentrate;
Ph. Eur.: Anticoagulant Citrate-Phosphate-Glucose Solution (CPD);
USP 31: Anticoagulant Citrate Phosphate Dextrose Adenine Solution; Anticoagulant Citrate Phosphate Dextrose Solution; Potassium Phosphates Injection; Sodium Phosphates Injection; Sodium Phosphates Oral Solution; Sodium Phosphates Rectal Solution.

Proprietary Preparations (details are given in Part 3)
Arg.: Dicofan; Enemol; Fleet Enema†; Fosfacol; Fosfafarma; Fosfo-Dom; Fosfoadital; Fosfobarigraf; Gadolax; Kritel Enema; Prontonema; Silaxa; Tekfema; ***Austral.:*** Celloids PP 85; Celloids SP 96; Fleet Phospho-Soda; Fleet Ready-to-Use; Phosphate-Sandoz; Phosphoprep; ***Austria:*** Fleet Phospho-Soda; Relaxyl; ***Belg.:*** Colexklysma; Fleet Enema; Fleet Phospho-Soda; Practo-Clyss; ***Braz.:*** Fleet Enema; Phosfoenema; ***Canad.:*** Fleet Enema; Fleet Phospho-Soda; ***Chile:*** Fabulaxol; Fleet Enema; Fleet Fosfosoda; ***Denm.:*** Fleet; ***Fin.:*** K-Fosfosteril†; ***Fr.:*** Fleet Phospho-Soda; ***Ger.:*** Fleet Phospho-Soda; ***Gr.:*** Bioklysm; Enema Cooper; Fleet Enema; Fosfolax; Klysmol; ***Hong Kong:*** Fleet Enema; Fleet Phospho-Soda; Unima; ***Hung.:*** Fleet Phospho-Soda; Optacid; ***India:*** Exit; ***Indon.:*** Fleet Enema; Fleet Phosposoda; ***Irl.:*** Fleet; ***Israel:*** Fleet Enema; ***Ital.:*** Clisma Fleet; Fosfo-Soda Fleet; ***Malaysia:*** Fleet Enema; Fleet Phospho-Soda; ***Mex.:*** Deplecat†; Fleet Enema Fosf-Sodio; Fleet PS; ***Neth.:*** Fleet Gebruiksklaar Klysma; Phosphoral; ***NZ:*** Fleet Phosphate Enema; Fleet Phospho-Soda; ***Philipp.:*** Fleet Enema; Phospho-Soda; ***Pol.:*** Enema; Fleet Phospho-Soda; Phospho-Laxative; Rectanal; ***Port.:*** Fleet Enema; Fleet Phospho-Soda; ***Singapore:*** Fleet Enema†; Fleet Phospho-Soda†; ***Spain:*** Fosfoevac; Fosfosoda; ***Swed.:*** Phosphoral; ***Turk.:*** BT Enema; Fleet Enema; Fleet Fosfo Soda; ***UK:*** Fleet Phospho-Soda; Fleet Ready-to-Use; ***USA:*** Fleet Enema; Fleet Phospho-Soda; K-Phos Original; OsmoPrep; Visicol; ***Venez.:*** Fleet Enema; Fleet Fosfosoda.

Multi-ingredient: ***Arg.:*** Colonil; ***Austral.:*** Cal Alkyline; Celloid Compounds Magcal Plus; Celloid Compounds Sodical Plus; Duo Celloids PPIP; Duo Celloids PPMP; Duo Celloids SPCF; Duo Celloids SPCP; Duo Celloids SPIP; Duo Celloids SPMP; Duo Celloids SPPC; Duo Celloids SPPP; Duo Celloids SPPS; Duo Celloids SPS; Duo Celloids SPSS; Gingo A†; Ginkgo Plus Herbal Plus Formula 10†; Lifesystem Herbal Plus Formula 11 Ginkgo†; Lifesystem Herbal Plus Formula 2 Valerian†; Magnesium Plus†; ML 20†; Potasi; Travad†; Valerian Plus Herbal Plus Formula 12†; ***Austria:*** Clysmol; Prepacol; Reducto; ***Belg.:*** Lavement au Phosphate†; Prepacol; ***Braz.:*** Digestron†; ***Canad.:*** Enemol; Gent-L-Tip†; Normo Gastryl; Phosphate-Novartis; Phosphates; ***Cz.:*** Blend-a-Med†; Mopasol; Prepacol; ***Denm.:*** Phosphoral; ***Fin.:*** Phosphoral; ***Fr.:*** Bactident; Digedryl; Hepargitol; Normacol Lavement; Normogastryl†; Oxyboldine; Phosphoneuros; Phosphore Medifa; Prefagyl†; Prepacol; Tavag; ***Ger.:*** Isogutt†; Klistier; Klysma Salinisch†; Lecicarbon; Practo-Clyss; Prepacol; Reducto-spezial; ***Gr.:*** Enter-Out; Fleet Phospho-Soda; Kathargon; Mineralin; Phospho-Laxat; Phosphoclean; Trifalac; ***Hong Kong:*** PMS-Enemol†; ***Hung.:*** Nilacid; Vition; ***India:*** Cotaryl; ***Indon.:*** Fosen; Kalkurenal; ***Irl.:*** Fletchers Phosphate Enema; ***Israel:*** Calciless; Soffodex; ***Ital.:*** Clisflex; Clisma-Lax; Enemac; IperClean; Phospho-Lax; Pomag; ***Malaysia:*** Unima; ***Mex.:*** Travad†; ***Neth.:*** Colex; ***Norw.:*** Phosphoral; ***NZ:*** Phosphate-Sandoz; ***Pol.:*** Phosphor; Sal Ems Artificiale; Sal Ems Factitium; Sal Vichy Factitium; ***S.Afr.:*** Colo-Prep; Lenolax; Phosphate-Sandoz; Sabax Fosenema; ***Singapore:*** ENTsol; ***Spain:*** Alcalinos Gelos; Darmen Salt; Enema Casen; Eupeptina; Foslainco; Lebersal; ***Switz.:*** Colophos; Freka-Clyss; Lecicarbon; Practo-Clyss†; ***Thai.:*** Swiff; Uni-Ma; ***UK:*** Carbalax; Fletchers Phosphate Enema†; Phosphate-Sandoz; Salivix; ***USA:*** K-Phos MF; K-Phos Neutral; K-Phos No.2; MSP-Blu; Neutra-Phos; Neutra-Phos-K; Phos-NaK; Summers Eve Post-Menstrual; Urelle; Uretron; Urimar-T; Urimax; UriSym†; Uro Blue; Urogesic Blue; Utira; ***Venez.:*** Fisiolin; Polantac.

Potassium

Kalium; Potasio.
K = 39.0983.

Description. Potassium salts covered in this section are those principally given as a source of potassium ions, but consideration should also be given to the effect of the anion. Phosphate salts of potassium are covered under Phosphate, p.1682, and the bicarbonate and citrate salts under Bicarbonate, p.1673.

Potassium Acetate

E261; Kalii acetas; Kalio acetatas; Kaliumacetat; Kálium-acetát; Kaliumasetaatti; Octan draselný; Potasio, acetato de; Potassium, acétate de; Potasu octan.
$CH_3.CO_2K$ = 98.14.
CAS — 127-08-2.

Pharmacopoeias. In *Eur.* (see p.vii) and *US*.
Ph. Eur. 6.2 (Potassium Acetate). Deliquescent white or almost white, crystalline powder or colourless crystals. Very soluble in water; freely soluble in alcohol. A 5% solution in water has a pH of 7.5 to 9.0. Protect from moisture.
USP 31 (Potassium Acetate). Colourless, monoclinic crystals, or a white crystalline powder. It is odourless or has a faint acetous odour. Deliquesces on exposure to moist air. Soluble 1 in 0.5 of water, 1 in 0.2 of boiling water, and 1 in 3 of alcohol. pH of a 5% solution in water is between 7.5 and 8.5. Store in airtight containers.

Equivalence. Each g of potassium acetate (anhydrous) represents about 10.2 mmol of potassium. Potassium acetate (anhydrous) 2.51 g is equivalent to about 1 g of potassium.

Potassium Aspartate

Aspartate monopotassique hémihydraté; Kalii hydrogenoaspartas hemihydricus; Kalio-divandenilio aspartatas hemihidratas; Kalium-hydrogen-aspartát hemihydrát; Kaliumväteaspartathemihydrat; Kaliumvetyaspartaattihemihydraatti; Potassium Hydrogen Aspartate Hemihydrate. Potassium aminosuccinate hemihydrate.
$C_4H_6KNO_4,\ ½H_2O$ = 180.2.
CAS — 7259-25-8 (hemihydrate).

Pharmacopoeias. In *Eur.* (see p.vii).
Ph. Eur. 6.2 (Potassium Hydrogen Aspartate Hemihydrate). A white or almost white, powder or crystalline powder, or colourless crystals. Very soluble in water; practically insoluble in alcohol and in dichloromethane. pH of a 2.5% solution in water is between 6.0 and 7.5.

Equivalence. Each g of potassium aspartate represents about 5.5 mmol of potassium. Potassium aspartate 4.61 g is equivalent to about 1 g of potassium.

Potassium Chloride

Chlorid draselný; Cloreto de Potássio; E508; Kalii chloridum; Kalio chloridas; Kalium Chloratum; Kaliumklorid; Kálium-klorid; Kaliumkloridi; Potasio, cloruro de; Potassium, chlorure de; Potasu chlorek.
KCl = 74.55.
CAS — 7447-40-7.
ATC — A12BA01; B05XA01.
ATC Vet — QA12BA01; QB05XA01.

Pharmacopoeias. In *Chin.*, *Eur.* (see p.vii), *Int.*, *Jpn*, *US*, and *Viet.*
Ph. Eur. 6.2 (Potassium Chloride). A white or almost white, crystalline powder or colourless crystals. Freely soluble in water; practically insoluble in dehydrated alcohol.
USP 31 (Potassium Chloride). Colourless, elongated, prismatic, or cubical crystals, or a white, granular powder. Is odourless. Soluble 1 in 2.8 of water, and 1 in 2 of boiling water; insoluble in alcohol. Its solutions are neutral to litmus.

Equivalence. Each g of potassium chloride represents about 13.4 mmol of potassium. Potassium chloride 1.91 g is equivalent to about 1 g of potassium.

Potassium Gluconate

E577; Potasio, gluconato de. Potassium D-gluconate.
$CH_2OH.[CH(OH)]_4.CO_2K$ = 234.2.
CAS — 299-27-4 (anhydrous potassium gluconate); 35398-15-3 (potassium gluconate monohydrate).
ATC — A12BA05.
ATC Vet — QA12BA05.

Pharmacopoeias. In *Fr.*
US permits anhydrous or the monohydrate.
USP 31 (Potassium Gluconate). It is anhydrous or contains one molecule of water of hydration. A white or yellowish-white, odourless, crystalline powder or granules. Soluble 1 in 3 of water; practically insoluble in dehydrated alcohol, in chloroform, in ether, and in benzene. Its solutions are slightly alkaline to litmus. Store in airtight containers.

Equivalence. Each g of potassium gluconate (anhydrous) represents about 4.3 mmol of potassium. Each g of potassium gluconate (monohydrate) represents about 4 mmol of potassium. Potassium gluconate (anhydrous) 5.99 g and potassium gluconate (monohydrate) 6.45 g are each equivalent to about 1 g of potassium.

Potassium Sulfate

E515; Kalii sulfas; Kalio sulfatas; Kalium Sulfuricum; Kaliumsulfaatti; Kaliumsulfat; Potasio, sulfato de; Potassii Sulphas; Potassium, sulfate de; Potassium Sulphate; Síran draselný; Tartarus Vitriolatus.
K_2SO_4 = 174.3.
CAS — 7778-80-5.

Pharmacopoeias. In *Eur.* (see p.vii) and *Jpn*.
Ph. Eur. 6.2 (Potassium Sulphate). A white or almost white, crystalline powder or colourless crystals. Soluble in water; practically insoluble in dehydrated alcohol.

Equivalence. Each g of potassium sulfate represents about 11.5 mmol of potassium. Potassium sulfate 2.23 g is equivalent to about 1 g of potassium.

Potassium Tartrate

E336; Potasio, tartrato de; Potasu winian.
$C_4H_4K_2O_6,\ ½H_2O$ = 235.3.
CAS — 921-53-9 (anhydrous potassium tartrate).

Equivalence. Each g of potassium tartrate (hemihydrate) represents about 8.5 mmol of potassium. Potassium tartrate (hemihydrate) 3.00 g is equivalent to about 1 g of potassium.

Adverse Effects

Excessive doses of potassium may lead to the development of hyperkalaemia (p.1669), especially in patients with renal impairment. Symptoms include paraesthesia of the extremities, muscle weakness, paralysis, cardiac arrhythmias, heart block, cardiac arrest, and confusion. Cardiac toxicity is of particular concern after intravenous dosage.

Pain or phlebitis may occur when given intravenously via peripheral veins, particularly at higher concentrations.

Nausea, vomiting, diarrhoea, and abdominal cramps may occur with oral potassium salts. There have been numerous reports of gastrointestinal ulceration, sometimes with haemorrhage and perforation or with the late formation of strictures, after the use of enteric-