Uric Acid

Synonyms	Urate
Clinical Indication	Diagnosis and monitoring of gout and other disturbances of uric acid metabolism. The presence of hyperuricaemia on its own is insufficient to establish a diagnosis of gout. Hyperuricaemia may be primary (genetic) or secondary. Primary hyperuricaemia may be due to reduced tubular secretion of uric acid, a smaller proportion is due to 1% of an enzymatic defect in purine metabolism which results in overproduction of uric acid. Secondary causes of hyperuricaemia include CKD, myeloproliferative diseases, haemolytic diseases, excess alcohol consumption and chemotherapy (tumour lysis syndrome). Hypouricaemia may result from decreased uric acid production, or due to increased renal uric acid excretion. Hypouricaemia may result from treatment with uricosuric agents.
Part of Profile / See Also	
Request Form	Combined Pathology manual Blood form or ICE request
Availability / Frequency of Analysis	On request.
Turnaround Time	Same day
Patient Preparation	None required.
Sample Requirements	
Specimen Type	Serum and plasma
Volume	2 ml
Acceptable Containers	 Yellow top (SST) tube  Green top (lithium-heparin) tube  paediatric orange top (lithium-heparin)  paediatric green top (lithium-heparin)

Plain serum samples may also be used.

Reference Range & Units	Male: 200 - 430 umol/L Female: 140 - 360 umol/L Reference: Pathology Harmony Group, Clinical Biochemistry Outcomes, January 2011 (www.pathologyharmony.co.uk).
Interferences	Elitek (rasburicase) which causes enzymatic degradation of uric acid in blood samples, causing spuriously low uric acid results. Dicynone (etamsylate), a haemostatic agent, interferes with the assay and may lead to spuriously low uric acid results. Patients treated with N-Acetyl Cysteine (NAC) for a Paracetamol overdose may generate a false low result for uric acid. In very rare cases gammopathy, especially IgM (Waldenström's macroglobulinemia), may cause unreliable results. Interference less than 12% or 35 µmol/L up to 20 mg/dL or 342 µmol/L conjugated bilirubin.
Interpretation & Clinical Decision Value (if applicable)	Sustained hyperuricaemia is the single most important risk factor for the development of gout. Hyperuricaemia occurs secondarily to reduced fractional clearance of uric acid in > 90% of patients with gout If on treatment for gout, management guidelines (BSR) suggest uric acid levels should be maintained at <300 µmol/L to prevent further urate crystal formation and to dissolve away existing crystals. The less stringent serum uric acid target of 360 µmol/l is recommended after some years of successful treatment when tophi have resolved and the patient remains symptom free. The initial aim of ULT is to reduce and maintain the sUA at or below a target level of 300 µmol/l
References	Hui M. et al. (2016) The British Society for Rheumatology Guideline for the Management of Gout. Rheumatology, Volume 56, Issue 7, 1 July 2017, Pages e1–e20.
Test code	UA
Lab Handling	Analysed from primary tube and stored at 4°C. Serum and plasma stable for 7 days at 4°C.

UKAS MEDICAL
7880
Accredited to ISO 15189:2012