

Uric Acid, Serum

## **Overview**

#### **Useful For**

Diagnosis and treatment of renal failure

Monitoring patients receiving cytotoxic drugs and a variety of other disorders, including gout, leukemia, psoriasis, starvation and other wasting conditions

#### **Method Name**

Photometric, Uricase/Quinone-Imine Dye Formation

#### **NY State Available**

Yes

## Specimen

## **Specimen Type**

Serum

#### **Necessary Information**

Patient's age and sex are required.

#### Specimen Required

**Collection Container/Tube:** 

Preferred: Serum gel
Acceptable: Red top
Specimen Volume: 0.5 mL

Submission Container/Tube: Plastic vial

**Collection Instructions:** 

- 1. Serum gel tubes should be centrifuged within 2 hours of collection.
- 2. Red-top tubes should be centrifuged and aliquoted within 2 hours of collection.

#### **Forms**

If not ordering electronically, complete, print, and send a Renal Diagnostics Test Request (T830) with the specimen.

#### **Specimen Minimum Volume**

0.25 mL

## **Reject Due To**

Gross	Reject
hemolysis	



Uric Acid, Serum

#### **Specimen Stability Information**

Specimen Type	Temperature	Time	Special Container
Serum	Refrigerated (preferred)	7 days	
	Frozen	180 days	

## **Clinical & Interpretive**

#### **Clinical Information**

Uric acid is the final product of purine metabolism in humans. Purines, compounds that are vital components of nucleic acids and coenzymes, may be synthesized in the body or they may be obtained by ingesting foods rich in nucleic material (eg, liver, sweetbreads). Approximately 75% of the uric acid excreted is lost in the urine; most of the remainder is secreted into the gastrointestinal tract where it is degraded to allantoin and other compounds by bacterial enzymes.

Asymptomatic hyperuricemia is frequently detected through biochemical screening. The major causes of hyperuricemia are increased purine synthesis, inherited metabolic disorder, excess dietary purine intake, increased nucleic acid turnover, malignancy, cytotoxic drugs, and decreased excretion due to chronic renal failure or increased renal reabsorption. Long-term follow-up of these patients is undertaken because many are at risk of developing renal disease; few of these patients ever develop the clinical syndrome of gout.

Hypouricemia, often defined as serum urate below 2.0 mg/dL, is much less common than hyperuricemia. It may be secondary to severe hepatocellular disease with reduced purine synthesis, defective renal tubular reabsorption, overtreatment of hyperuricemia with allopurinol, as well as some cancer therapies (eg, 6-mercaptopurine).

#### **Reference Values**

#### Males

1-10 years: 2.4-5.4 mg/dL 11 years: 2.7-5.9 mg/dL 12 years: 3.1-6.4 mg/dL 13 years: 3.4-6.9 mg/dL 14 years: 3.7-7.4 mg/dL 15 years: 4.0-7.8 mg/dL > or =16 years: 3.7-8.0 mg/dL

Reference values have not been established for patients who are <12 months of age.

#### Females

1 year: 2.1-4.9 mg/dL 2 years: 2.1-5.0 mg/dL 3 years: 2.2-5.1 mg/dL 4 years: 2.3-5.2 mg/dL 5 years: 2.3-5.3 mg/dL 6 years: 2.3-5.4 mg/dL 7-8 years: 2.3-5.5 mg/dL



Uric Acid, Serum

9-10 years: 2.3-5.7 mg/dL 11 years: 2.3-5.8 mg/dL 12 years: 2.3-5.9 mg/dL > or =13 years: 2.7-6.1 mg/dL

Reference values have not been established for patients who are <12 months of age.

#### Interpretation

Hyperuricemia is most commonly defined by serum or plasma uric acid concentrations above 8.0 mg/dL in males or above 6.1 mg/dL in females.

#### **Cautions**

The following drugs cause interference (falsely decreased levels) at therapeutic concentrations:

- -Alpha-methyldopa
- -Desferoxamine
- -Calcimdobesilate

Results can be falsely decreased in patients with elevated levels of N-acetyl-p-benzoquinone imine (NAPQI, a metabolite of acetaminophen), N-acetylcysteine (NAC), and metamizole.

#### **Clinical Reference**

Tietz Textbook of Clinical Chemistry. Chapter 24: Fourth edition, Edited by CA Burtis, ER Ashwood, WS Bruns. WB Saunders Company, Philadelphia, 2006, pp 803-807

#### **Performance**

## **Method Description**

Uric acid is oxidized by uricase to form allantoin and hydrogen peroxide. The hydrogen peroxide reacts with TOOS (N-ethyl-N-[2-hydroxy-3-sulfopropyl]-3-methylaniline) and 4-aminophenazone in the presence of peroxidase to form a quinone-imine dye. The intensity of the red color formed is proportional to the uric acid concentration. Prior to the start of the reaction, the sample is initially incubated with a reagent mixture containing ascorbate oxidase and a clearing system. This eliminates any ascorbic acid present in the sample which could interfere with the peroxidase indicator reaction. (Package insert: Roche Uric Acid reagent, Roche Diagnostic Corp., Indianapolis IN, V10. 12/2018)

#### PDF Report

No

## Day(s) Performed

Monday through Sunday

#### Report Available

Same day/1 to 2 days

#### **Specimen Retention Time**

1 week



Uric Acid, Serum

#### **Performing Laboratory Location**

Mayo Clinic Laboratories - Rochester Main Campus

#### **Fees & Codes**

#### **Fees**

- Authorized users can sign in to <u>Test Prices</u> for detailed fee information.
- Clients without access to Test Prices can contact <u>Customer Service</u> 24 hours a day, seven days a week.
- Prospective clients should contact their account representative. For assistance, contact <u>Customer Service</u>.

#### **Test Classification**

This test has been cleared, approved, or is exempt by the US Food and Drug Administration and is used per manufacturer's instructions. Performance characteristics were verified by Mayo Clinic in a manner consistent with CLIA requirements.

#### **CPT Code Information**

84550

#### **LOINC®** Information

Test ID	Test Order Name	Order LOINC® Value
URIC	Uric Acid, S	3084-1

Result ID	Test Result Name	Result LOINC® Value
URIC	Uric Acid, S	3084-1