

Uric Acid

WHAT IS URIC ACID?

Uric acid is a normal waste product in the body that forms as a result of purine breakdown. Purines are organic molecules that include nucleotides - the building blocks of DNA and RNA (1). They are found in high concentrations in meat and meat products (2), while plant-based diets are generally low in purines (3). Uric acid circulates in the body in the ionized form of urate. It is filtered by the kidneys and excreted in urine and feces (4).

NORMAL URIC ACID LEVELS

The normal reference ranges for blood uric acid levels are 3.7 – 7.7 mg/dL for adult males and 2.5 – 6.2 mg/dL for adult females (5). However, some references indicate that anything above 7 mg/dL is classed as elevated uric acid (hyperuricemia) (4).

PURPOSE OF A BLOOD URIC ACID TEST

Measurements of uric acid levels in a blood sample are beneficial for the diagnosis and treatment of gout, renal dysfunction, and a variety of other disorders, including leukemia, psoriasis, atherosclerosis, diabetes, and hypothyroidism.

ELEVATED URIC ACID

Elevated blood uric acid (hyperuricemia) is a common disorder that can affect people of all ages and genders, and it is estimated that up to 21% of the general population have asymptomatic hyperuricemia. It is usually due to decreased uric acid excretion, but can also be caused by increased uric acid production, or a combination of both processes (4).

Kidney disease, specific medications, Down syndrome, reduced thyroid function, and heavy metal poisoning can inhibit the normal excretion of uric acid. Increased uric acid production may be due to a purine-rich diet (e.g. high intake of red meat, organ meat), increased alcohol consumption (particularly beer), errors in purine metabolism, or increased cell breakdown due to various diseases (e.g. psoriasis or cancer) or chemotherapy treatments (4).

The most common complication of hyperuricemia is gout, an inflammatory arthritis. Gout occurs when uric acid accumulates in the blood and tissues, leading to the formation of crystals within the joints. This results in joint pain and swelling, particularly in peripheral joints, such as the toes. Gout is significantly more common in males than females (6).

Hyperuricemia can also lead to uric acid nephrolithiasis (kidney stones), which account for about 7% of all kidney stones. Symptoms can include severe pain in the lower back, nausea and vomiting, blood in the urine, and urgent need to urinate (7).

Hyperuricemia usually does not require any medical treatment, as the majority of affected people will never develop gout or nephrolithiasis. However, if health complications do occur, following a low-purine diet along with certain medications (e.g. allopurinol and probenecid) can effectively lower uric acid levels (4).

REDUCED URIC ACID

Hypouricemia is the term to describe lower than normal blood uric acid levels. It is usually benign, but may be a useful medical sign for other medical conditions. Hypouricemia can be caused by drugs that reduce uric acid production or increase the excretion of uric acid from the blood

into the urine (8). Vegetarians are more likely to have hypouricemia, due to the low purine content of most vegetarian diets (9). Medical conditions that can cause hypouricemia include Fanconi syndrome, hyperthyroidism, multiple sclerosis, nephritis, and Wilson's disease (10).

Idiopathic hypouricemia itself usually doesn't require any treatment. However, if it is a sign of another medical condition, the underlying condition may require treatment. For example, hypouricemia may be due to increased excretion of uric acid from the blood into the urine, resulting in high uric acid levels in the urine (hyperuricosuria), which can lead to uric acid nephrolithiasis (11).

TEST PROCEDURE

Correct specimen collection and handling is required for optimal assay performance. At least a four-hour fast is preferred prior to specimen collection.

This test requires a blood sample from a finger prick. All supplies for sample collection are provided in this kit. First wash and dry hands. Warm hands aid in blood collection. Clean the finger prick site with the alcohol swab and allow to air dry. Use the provided lancet to puncture the skin in one quick, continuous and deliberate stroke. Wipe away the first drop of blood. Massage hand and finger to increase blood flow to the puncture site. Angle arm and hand downwards to facilitate blood collection on the fingertip. Drip blood onto the blood collection card or into the microtainer tube.

Avoid squeezing or 'milking' the finger excessively. If blood flow stops, perform a second skin puncture on another finger, if more blood is required. Do not touch the fingertip.

Dispose of all sharps safely and return sample to the laboratory in the provided prepaid return shipping envelope.

Upon receipt at the laboratory, the blood sample is analyzed by the fully automated Alinity c Uric Acid assay on the Alinity ci series analyzer. This assay measures blood uric acid levels using uricase methodology.

TEST INTERPRETATION

This assay will provide an accurate uric acid level for the tested blood specimen. High levels can cause gout or uric acid nephrolithiasis, while low levels may be a sign of other underlying medical complications. Additional testing may be required to determine the cause of abnormal blood uric acid levels.

DISCLAIMERS/LIMITATIONS

These results should be interpreted in conjunction with other laboratory and clinical information.

Additional testing is recommended if uric acid levels are inconsistent with clinical evidence.

Correct specimen collection and handling is required for optimal assay performance.

Interferences from medication or endogenous substances may affect results, including aspirin, levodopa, and diuretics.

Vigorous exercise, chemotherapy and radiation therapy, and high-purine foods can affect results.

REFERENCES

- (1) Rosemeyer H. (2004). The chemodiversity of purine as a constituent of natural products. *Chem Biodivers*. 1(3), 361-401.
- (2) Zhang Y, et al. (2012). Purine-rich foods intake and recurrent gout attacks. *Ann Rheum Dis*. 71(9), 1448-1453.
- (3) Jakše, B. (2019). Uric Acid and Plant-Based Nutrition. *Nutrients*. 11(8), 1736.
- (4) George C & Minter DA. (2021). Hyperuricemia. StatPearls [Internet]. Treasure Island : StatPearls Publishing.
- (5) Rifai N, Horvath AR, & Wittwer C. (2018). Tietz textbook of clinical chemistry and molecular diagnostics (Sixth edition.). St. Louis, Missouri: Elsevier.
- (6) Williams LA. (2019). The History, Symptoms, Causes, Risk Factors, Types, Diagnosis, Treatments, and Prevention of Gout, Part 2. *Int J Pharm Compd*, 23(1), 14-21.
- (7) Worcester EM & Coe FL. (2008). Nephrolithiasis. *Prim Care*, 35(2), 369-vii.
- (8) Ramsdell CM & Kelley WN. (1973). The clinical significance of hypouricemia. *Ann Int Med*, 78(2), 239-242.
- (9) Siener R & Hesse A. (2003). The effect of a vegetarian and different omnivorous diets on urinary risk factors for uric acid stone formation. *Eur J Nutr*, 42(6), 332-337.
- (10) Son CN, et al. (2016). Prevalence and possible causes of hypouricemia at a tertiary care hospital. *Korean J Intern Med*, 31(5), 971-976.
- (11) Martin HE, et al. (2001) [Vesical uric acid lithiasis in a child with renal hypouricemia]. *An Esp Pediatr*, 55(3), 273-276.