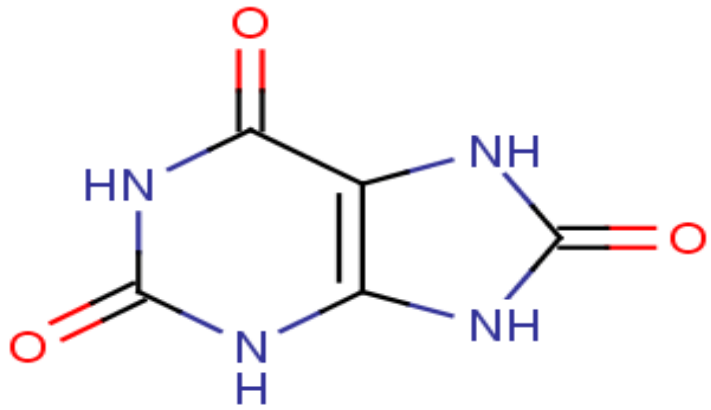
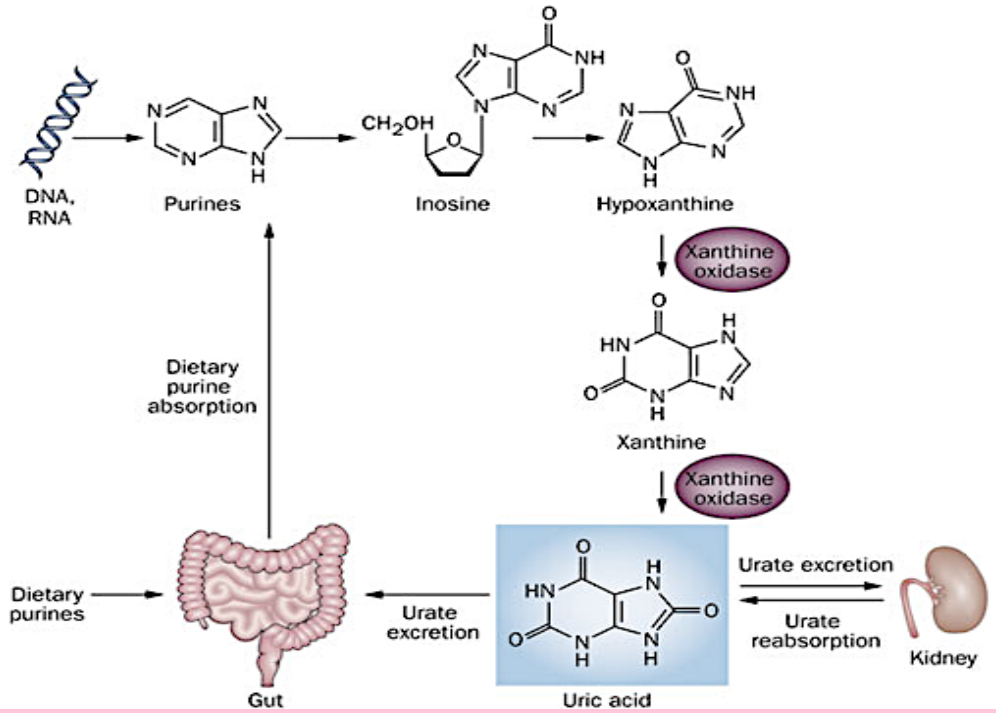
Estimation of Uric Acid in serum

BCH 472

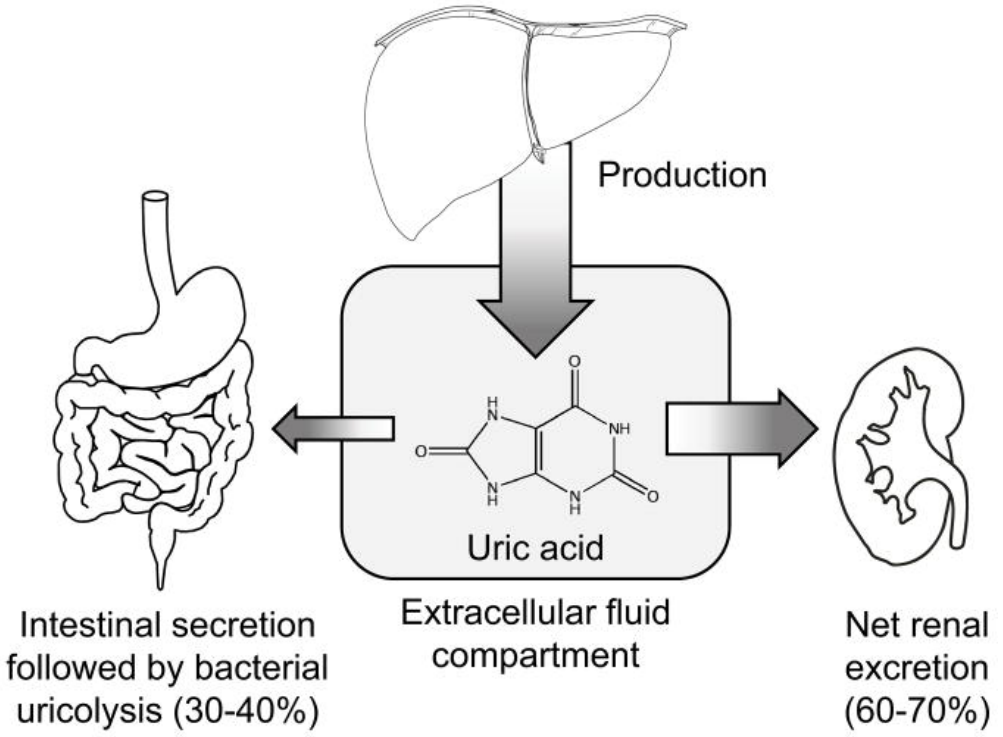


Uric acid production:

Uric acid is the product of **catabolism of the purine** (adenosine and guanine) that result from the break down of ingested nucleic acid (exogenous) or from tissue destruction (endogenous).



Uric acid excretion



Clinical application:

- To assess inherited disorders of **purine metabolism**.
- To confirm diagnosis and monitor treatment of **gout**.
- To assist the diagnosis of **renal calculi (**uric acid kidney stones).
- To detect **kidney dysfunction**.

Uric acid –Serum:

Case	Cause
Increased uric acid in serum (**hyperuricemia**)	• **Gout**
	• **Renal diseases and renal failure**, (decreased excretion of uric acid)
	• **Leukemia, multiple myeloma, lymphoma** • Over production of uric acid occurs after chemotherapy, due to the rapid amount of cellular destruction
	• **Lesch-Nyhan syndrome** (rare hereditary gout result from an enzyme defiance hypoxanthine-guanine phosphoribosyltransferase (HGPRT)). • This enzyme functions primarily to salvage purines from degraded DNA to reintroduce into purine synthetic pathways.
Decreased uric acid in serum (**hypourecemia**)	• **Liver disease** (Decreased Production)
	• **Fanconi syndrome** (Increased excretion, due to defect in the reabsorption)

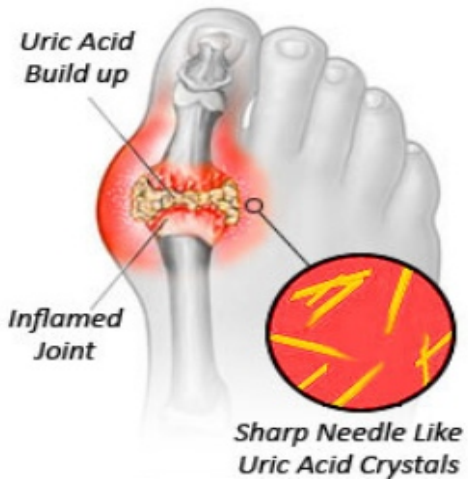
Uric acid –urine:

- This test evaluates uric acid metabolism in gout and renal calculus formation.
- The uric acid urine test measured in a sample of **urine collected over 24 hours**.
- A **high level of uric acid** in the urine means that the patient is more likely to develop **uric acid kidney stones**.

Case	
Increased urine uric acid (**uricosuria**) occurs in:	Tubular reabsorption defect (Fanconi syndrome)
	multiple myeloma, lymphoma
	Lesch-Nyhan syndrome
Decreased urine uric acid	Kidney disease

Gout:

- **Excess uric acid crystals** in the joints, soft tissues, and organs.
- Caused by an increased conversion of purine bases to uric acid or a decreased excretion of uric acid by the kidney.
- This will lead to **inflammation of tissues.** This inflammation is responsible for the crisis symptoms acute gouty arthritis



Notes:

- **Hyperuricemia** does not always lead to gout. Less than 20% of cases develop into arthritic gout disease.
- Blood test results can be misleading, though. Some people have high uric acid levels, but never experience gout. And some people have signs and symptoms of gout, but don't have unusual levels of uric acid in their blood.
- Definitive diagnosis can be made by observing the presence of urate crystals in synovial fluid removed from affected joints.

Practical Part

Objective:

To estimate the amount of uric acid in blood.

Principle:

Kit contains:

The **reagent** used includes: uricase, peroxidase, 4-Aminoantipyrine.

URIZYME BUFFER: Polyhalogenated benzoic acid in Tris buffer at pH 7.5.

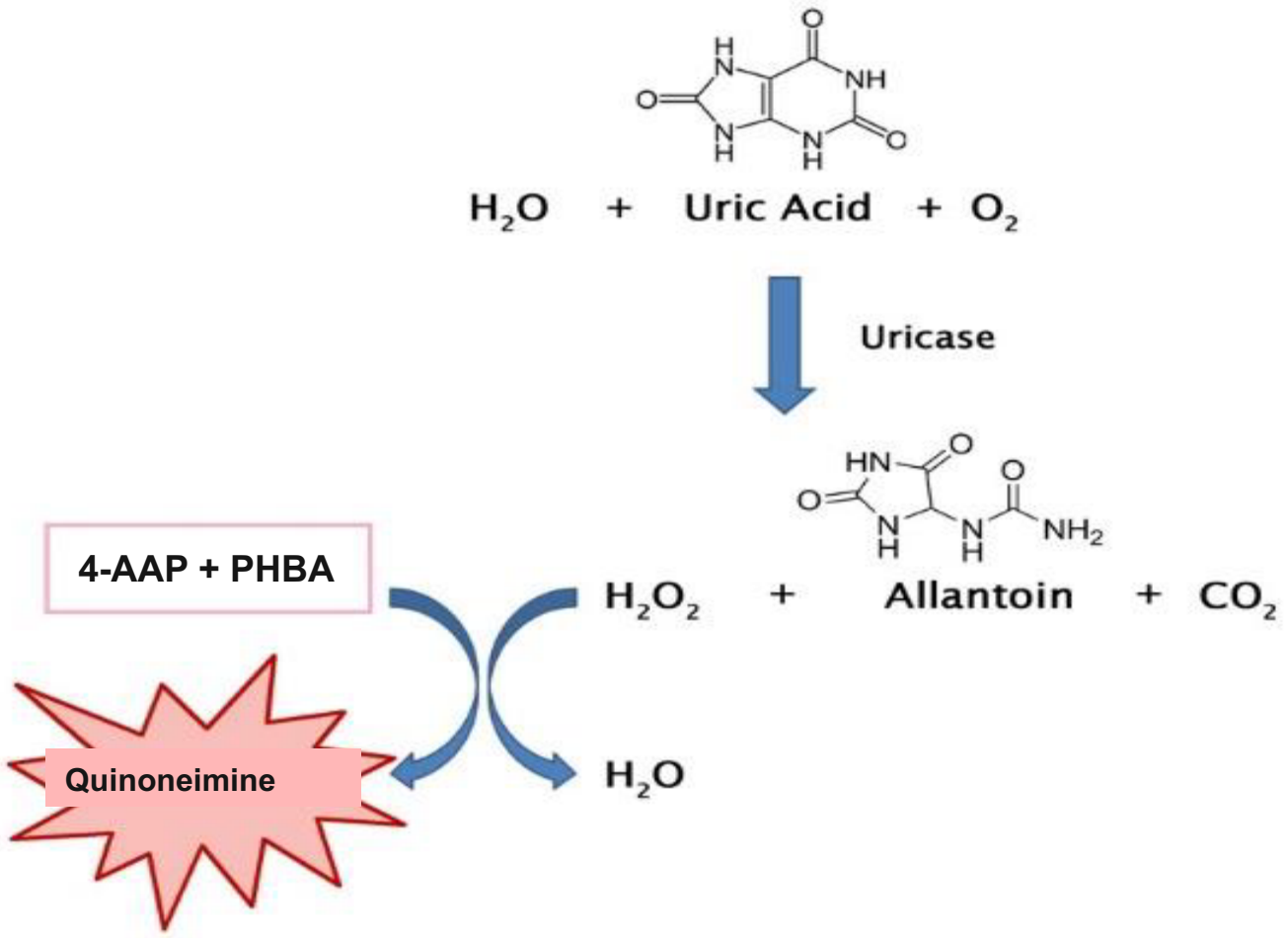
1. Uric acid in the sample oxidized by **uricase** to allantoin and hydrogen peroxide.

   Uric acid + O_2 +$2H_2O$ $\xrightarrow{\textbf{Uricase}}$ Allantoin + CO_2 + H_2O_2

2. Hydrogen peroxide reacts with polyhalogenated benzoic acid (PHBA) and 4-aminoantipyrine (4-AAP) in the presence of **peroxidase** to yield a **quinoneimine dye** (chromogen).

   $2H_2O_2$ +4-AAP + PHBA $\xrightarrow{\textbf{Peroxidase}}$ **Quinoneimine** +$4H_2O$

* The (intensity of the dye) absorbance is measured and is **directly proportional** to the concentration of uric acid present in the sample.


H_2O + Uric Acid + O_2
Uricase
4-AAP + PHBA
H_2O_2 + Allantoin + CO_2
Quinoneimine
H_2O

Method:

	Blank	Standard	Test
Reagent	1 ml	1 ml	1 ml
Standard	—	0.02 ml	—
Sample	—	—	0.02 ml

-Water bath at 37ºC for 5 min.

-Read absorbance at **520 nm** .

Calculations:

- $\frac{\text{Absorbance of sample}}{\text{Absorbance of standard}}$ x concentration of standard (**8 mg/dl**)

Reference value in serum:

Men: 3.4–7.0 mg/dL

Women: 2.4–5.7 mg/dL

Discussion :

Comment on your result and mention if there are any abnormalities.

Questions

Supposed that you have a plasma uric acid estimation result for a patient with (Phosphoribosylpyrophosphate synthetase superactivity), what do you think the result would be (high or low) ? Why (explain)?