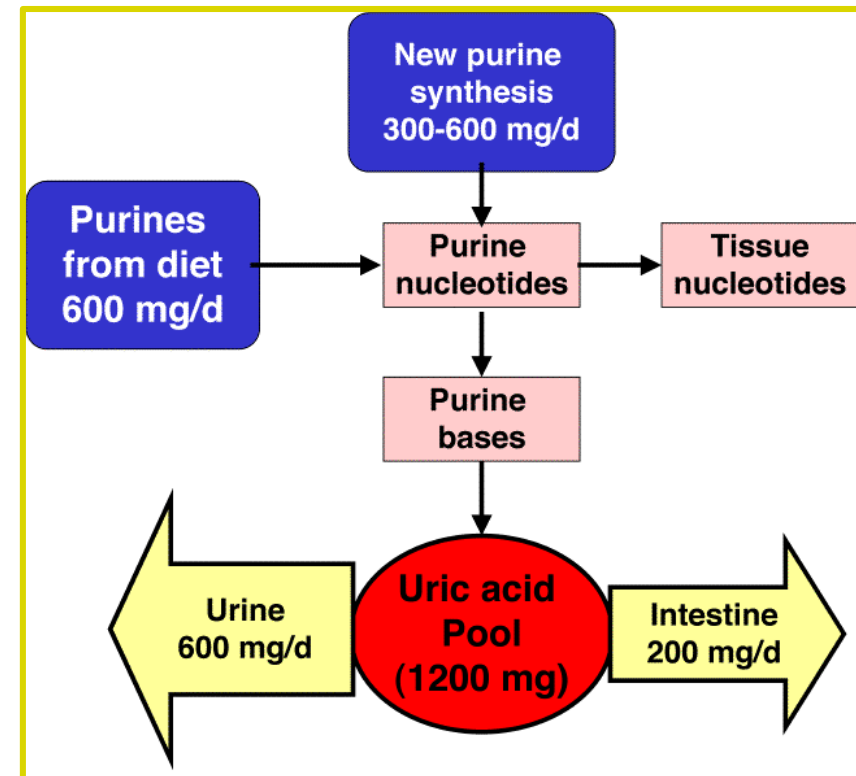
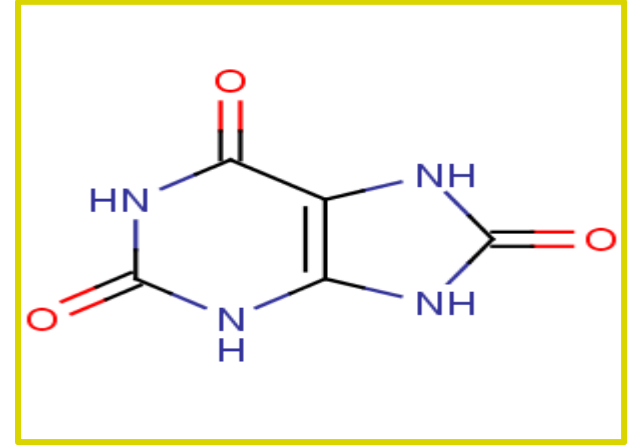


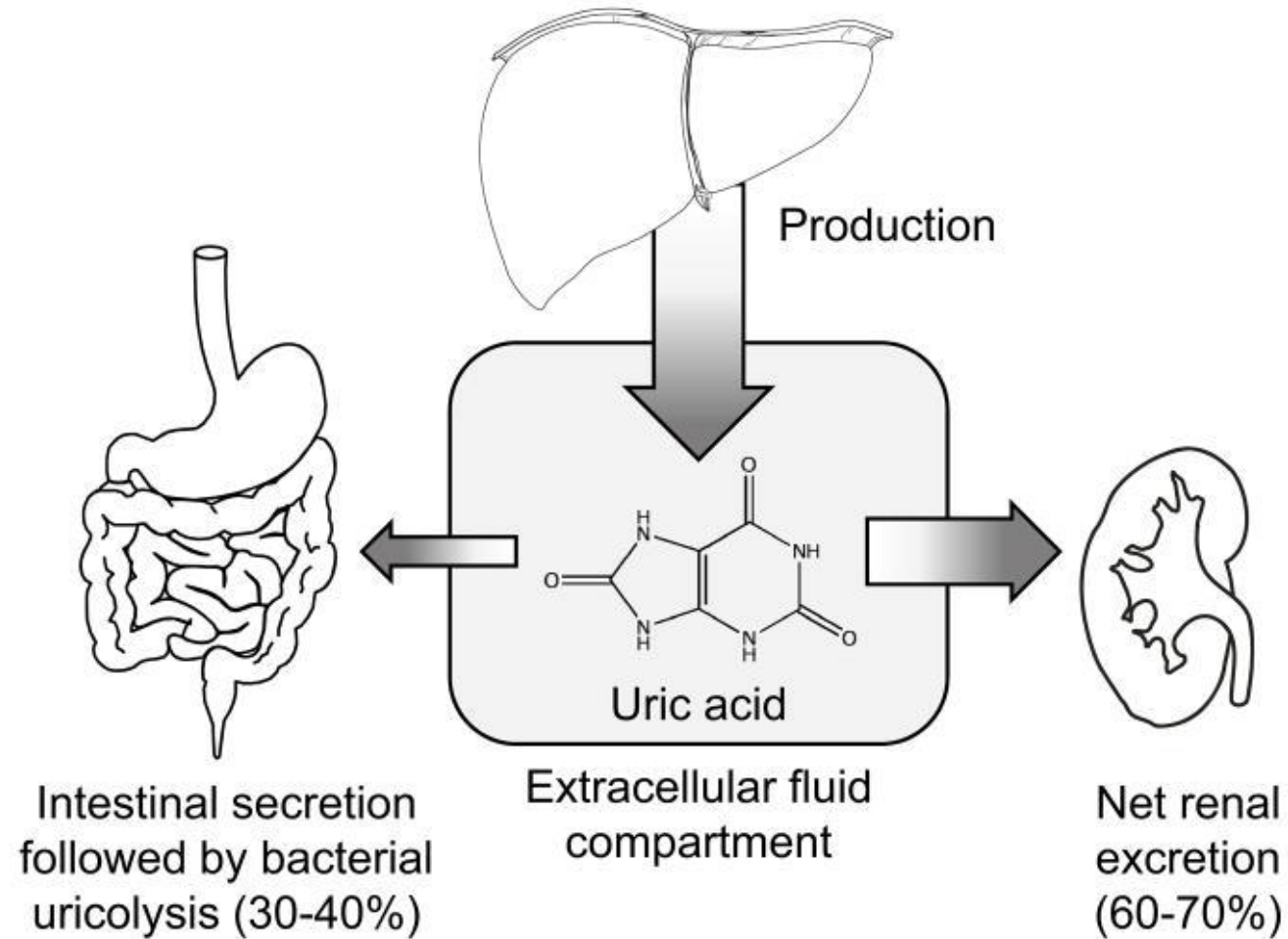
Estimation of Uric Acid in serum

-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 is transported by the plasma from the **liver** to the **kidney**, where it is filtered and where about **70% is excreted**. The remainder of uric acid is excreted into the GI tract and degraded.



-Uric acid excretion:



-Clinical application:

1. Uric acid is measured to assess inherited disorders of **purine metabolism**.
2. To conform diagnosis and monitor treatment of **gout**.
3. To assist the diagnosis of **renal calculi** (uric acid kidney stones).
4. To detect **kidney dysfunction**.
5. Evaluation of **leukemia**.

-Uric acid -Serum:

Case	Cause
Increased uric acid serum (Elevated uric acid levels (hyperuricemia))	<ul style="list-style-type: none">• Gout (the amount of increase is <u>not</u> directly related to the severity of the disease).
	<ul style="list-style-type: none">• Renal diseases and renal failure (decreased excretion of uric acid)
	<ul style="list-style-type: none">• Leukemia, multiple myeloma, lymphoma.
	<ul style="list-style-type: none">• Lesch-Nyhan syndrome (rare hereditary gout result from an enzyme defiance hypoxanthine-guanine phosphoribosyltransferase (HGPRT)).
Decreased uric acid serum (hypouricemia)	<ul style="list-style-type: none">• Liver disease (Decreased Production).
	<ul style="list-style-type: none">• Fanconi syndrom (Increased excretion).

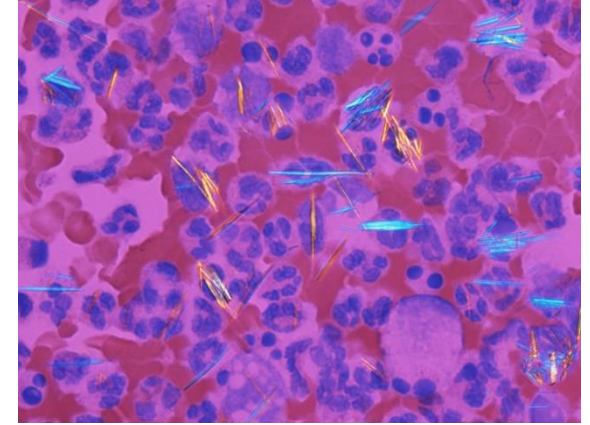
-Uric acid -Urine:

- 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	Cause
Increased urine uric acid (uricosuria)	Tubular reabsorption defect (fanconi syndrom)
	Multiple myeloma, lymphoma
	Lesh-Nayan syndrom
Decreased urine uric acid	Kidney disease

-Gout:

- Excess **monosodium urate crystallizes** and deposits (needle like crystals) in the joints, soft tissues, and organs.
- This will lead to inflammation of tissues → **This inflammation is responsible for the crisis symptoms acute gouty arthritis.**



monosodium urate crystals

Notes:

- Hyperuricemia **does not** always lead to gout. Less than 20% of cases develop into arthritic gout disease.
- Uric acid level is just one of several criteria necessary for diagnosis.
- 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.

Practical Part

-Objective:

- To estimate the amount of uric acid in blood by using uric acid liquicolor kit.

-Principle:

- Kit contains:

-The enzyme reagent used includes: buffer, uricase, peroxidase, 4-Aminophenazone and DCHBS.

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



2. Hydrogen peroxide reacts with 3,5-Dichloro-2-hydroxybenzene-sulfonic acid (DCHBS) and 4-aminophenazone (PAP) in the presence of **peroxidase** (Hydrogen peroxide oxidoreductase) to yield a red-violet quinoneimine dye (**chromogen**). The intensity of the dye is measured at 520nm and it is directly proportional to the concentration of uric acid present in the sample.



-Materials:

- Uric acid liquicolor (PAP-method) kit.

-Method:

	Blank	Standard	Test
Buffer	1ml	1ml	1ml
Standard	—	0.020 ml	—
Sample	—	—	0.025 ml

1. Water bath at 37°C for 5 min.
2. Read absorbance at 520nm.

-Calculations:

- $\frac{\text{Absorbance of sample}}{\text{Absorbance of standard}} \times \text{concentration of standard (5 mg/dl)}$
- **Reference value in serum:**
 - Men: 3.4–7.0 mg/dL or 202–416 mol/L
 - Women: 2.4–5.7 mg/dL or 143–357 mol/L

Homework:

- 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)?

References:

- Bobulescu, I. A., & Moe, O. W. (2012). Renal Transport of Uric Acid: Evolving Concepts and Uncertainties. *Advances in Chronic Kidney Disease*, 19(6), 358–371. <http://doi.org/10.1053/j.ackd.2012.07.009>.
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