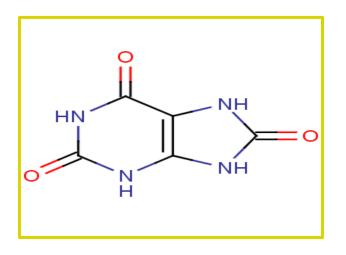
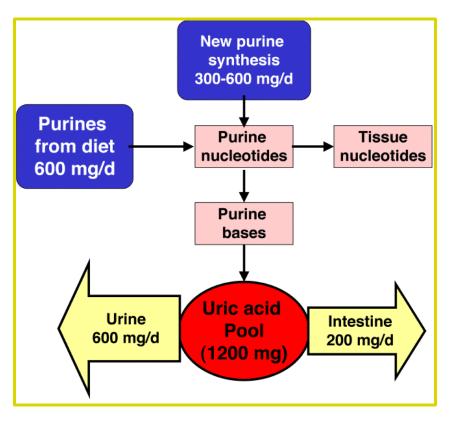
Estimation of Uric Acid in serum

-Uric acid production:

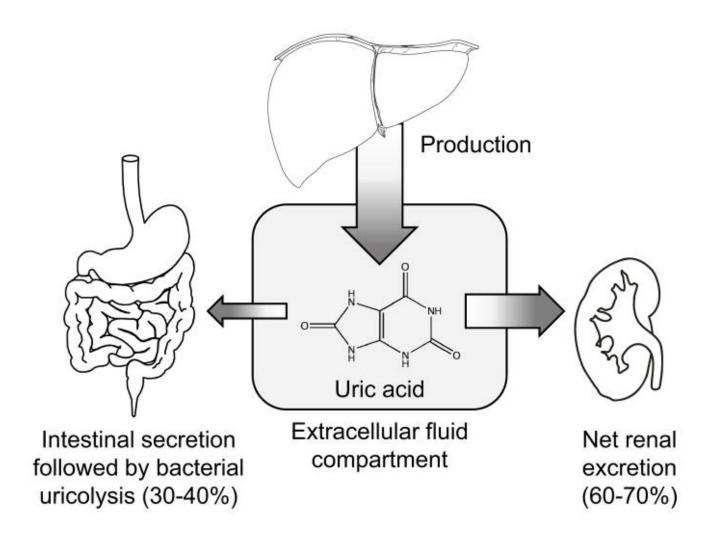
• Uric acid is the product of catabolism of the purine (adenosine and guanine) that result from the <u>break down of ingested nucleic acid</u> (exogenous) or from <u>tissue destruction</u> (endogenous).



• 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:

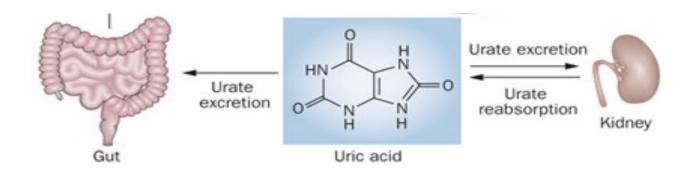


-Uric acid excretion:

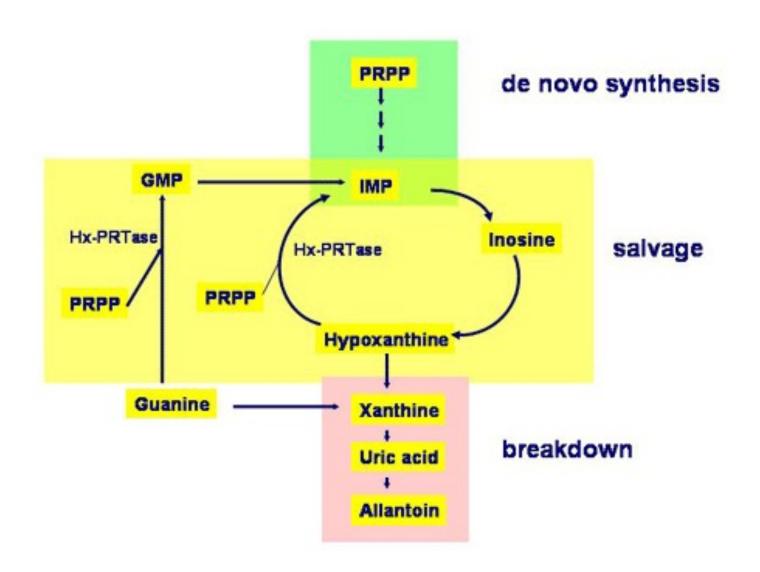
Uric acid excretion

The rest pass into the gastrointestinal tract 30%

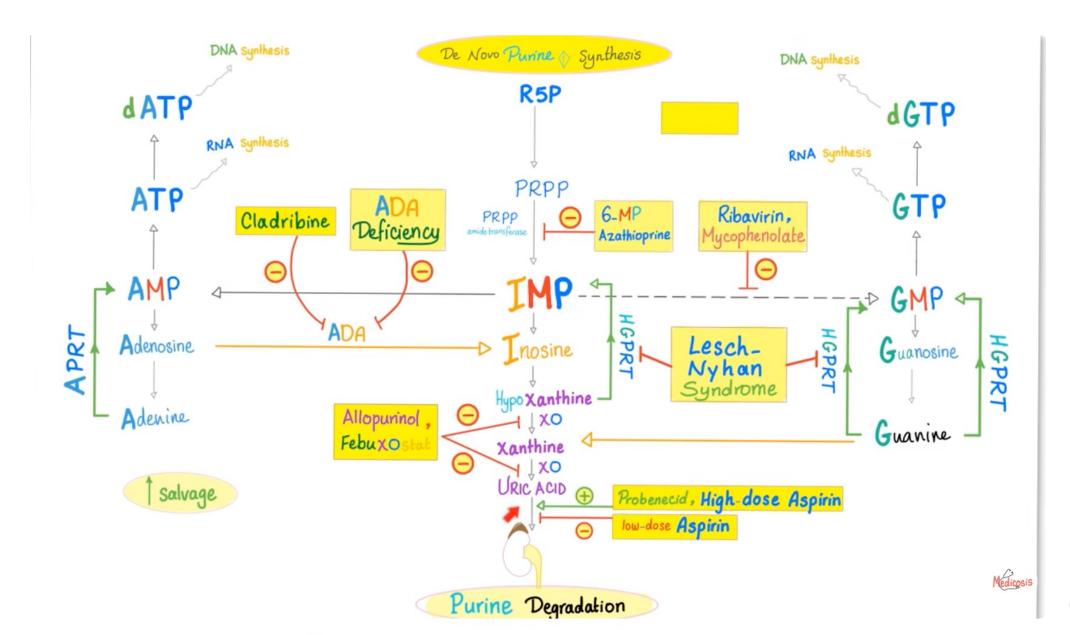
Renal excretion account for 70 %



- Purine metabolism:



- Purine metabolism:



-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.

Serum Uric acid:

Case	Cause		
Increased uric acid serum (Elevated uric acid levels (hyperuricemia)	• Gout (the amount of increase is <u>not</u> directly related to the severity of the disease).		
	Renal diseases and renal failure (decreased excretion of uric acid)		
	 Leukemia, multiple myeloma, lymphoma. (over-production) 		
	• Lesch-Nyhan syndrome (rare hereditary gout result from an enzyme defiance hypoxanthine-guanine phosphoribosyltransferase (HGPRT)). (over-production)		
Decreased uric acid serum (hypourecemia)	• Liver disease (Decreased Production).		
	Fanconi syndrome (Increased excretion).		

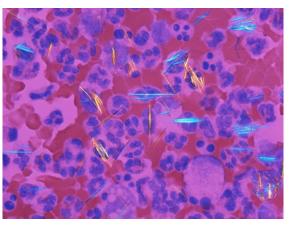
<u>Urine</u> Uric acid:

- ➤ This test evaluates uric acid <u>metabolism in gout and renal calculus formation</u>.
- The uric acid urine test measured in a sample of urine collected <u>over 24 hours</u>.
- 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 syndrome)		
	Multiple myeloma, lymphoma		
	Lesch-Nayhan 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 Stanbio 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.

Uric acid +
$$O_2$$
 +2 H_2O Uricase Allantoin + CO_2 + H_2O_2

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:

• Stanbio Uric Acid LiquiColor kit.

-Method:

	Blank	Standard	Test
Buffer	1ml	1ml	1ml
Standard		0.02 ml / 20 μl	
Sample			0.02 ml/ 20 μl

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

-Calculations:

• Absorbance of sample Absorbance of standard x concentration of standard (8 mg/dl)

• Normal Range in serum: Men (3.4 - 7 mg/dl). Women (2.4 - 5.7 mg/dl)

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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