Purine Metabolism

Second stage

DR . RASHAD AL - TUUAMAH

Medical Biochemistry

2026

PURINE METABOLISM INTRODUCTION AND BASICS:

the three pillars of PURINE METABOLISM are DE NOVO synthesis (creating new purines)

degradation (breaking down purines into waste), and SALVAGE (recycling and reusing purines). understanding these processes is v

ital for diagnosing and managing conditions like GOUT. regarding DNA vs RNA basics, the sugar

component in DNA uses deoxyribose sugar, while RNA uses ribose.

2026

The nitrogenous bases include PURINES, which are ADENINE (A) and

GUANINE (G) containing double rings, and PYRIMIDINES, which are

CYTOSINE (C) and THYMINE (T) (or URACIL in RNA) containing a single

ring. in terms of terminology, a NUCLEOSIDE is sugar + base, while a NUCLEOTIDE is

sugar + base + phosphate.

2026

THE ROLE OF FOLATE AND DE NOVO SYNTHESIS:

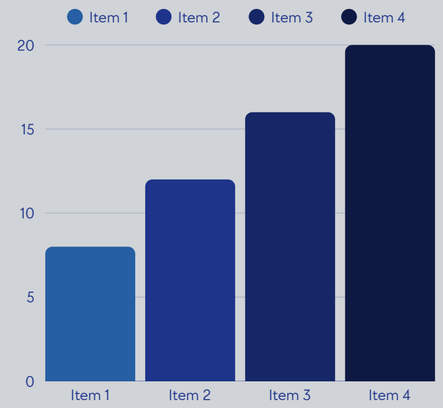
in the synthesis process, bacteria and plants start with PABA to create DIHYDROFOLATE

(DHF) and then TETRAHYDROFOLATE (THF). the function of THF is to help create the

purines and pyrimidines necessary for DNA and RNA. clinical correlations show that

SULFONAMIDES block the conversion of PABA, while TRIMETHOPRIM,

PYRIMETHAMINE, and METHOTREXATE inhibit the enzyme DIHYDROFOLATE REDUCTASE (DHFR).



2026

these drugs can cause folate deficiency and megaloblastic

anemia. the DE NOVO PURINE SYNTHESIS PATHWAY starts with

RIBOSE 5-PHOSPHATE (from the HMP shunt) being converted into

PRPP. then, PRPP is converted into INOSINE MONOPHOSPHATE

(IMP). the product IMP serves as the branch point to create AMP

(ADENINE) and GMP (GUANINE). finally, these must be converted

to deoxy forms to participate in DNA synthesis.

2026

PURINE DEGRADATION, URIC ACID, AND SALVAGE :

the goal of degradation is to break down purines into a form that can be excreted in urine. the pathway follows: IMP to INOSINE,

INOSINE to HYPOXANTHINE, HYPOXANTHINE to XANTHINE (via XANTHINE OXIDASE), and XANTHINE to URIC ACID (via XANTHINE

OXIDASE). URIC ACID is excreted by the kidneys; excess can lead to kidney stones or GOUT.

2026

the PURINE SALVAGE PATHWAY is the "redeeming" or recycling of

bases instead of wasting them as URIC ACID. key enzymes include

APRT, which converts ADENINE back into AMP, and HGPRT,

which converts HYPOXANTHINE or GUANINE back into IMP or GMP.

a deficiency in HGPRT leads to LESCH-NYHAN syndrome, causing hyperuricemia and GOUT.

PHARMACOLOGY AND GOUT MANAGEMENT:

2026

synthesis inhibitors include 6-MERCAPTOPURINE (6-MP) and AZATHIOPRINE, which block the pathway

from PRPP to IMP, while MYCOPHENOLATE and RIBAVIRIN inhibit the conversion of IMP to GMP.

XANTHINE OXIDASE INHIBITORS like ALLOPURINOL and FEBUXOSTAT block URIC ACID production. ADENOSINE

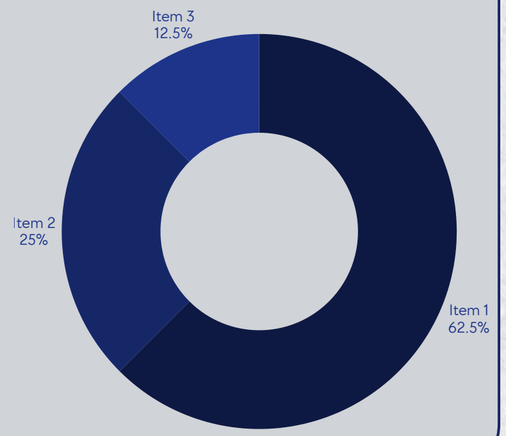
DEAMINASE (ADA) deficiency prevents ADENOSINE from becoming INOSINE.

2026

for GOUT management, URICOSURIC DRUGS like PROBENECID stimulate the excretion of URIC ACID in urine. there is also the ASPIRIN

PARADOX: low-dose ASPIRIN inhibits URIC ACID secretion (can be "pro-gout"), while high-dose ASPIRIN acts as an anti-inflammatory and

stimulates URIC ACID excretion (helps in gout). safety note: never give ASPIRIN to babies due to the risk of REYE syndrome.



2026

THANK YOU FOR YOUR ATTENTION !