# Iron

- Iron absorption
  - Converted to ferric in the mucosal cells
  - Iron is absorbed in ferrous form
  - absorbed from upper part of small intestine by active transport
  - ↑ absorption
    - fructos
    - acidic medium
    - Vitamin C
    - amino acids
  - ↓ absorption
    - tetracyclines
    - Antacids
- Iron Excretion
  - by exfoliation (peeling off) of intestinal mucosal cells containing ferritin into stool
  - lost in saliva, sweat
  - no efficient way for excretion
- Uses of iron therapy
  - Usually given orally
  - Microcytic or hypochromic anemia.
- forms of iron
  - Stored forms
    - Non available Forms
      - myoglobin
      - cytochromes
      - various enzymes
    - Available Forms
      - haemosiderin
      - Ferritin
  - Hemoglobin
  - Transferrin
- Daily requirement
  - 5 mg for male.
  - 15 mg for female & children.
  - available in meat, grain and vegetables
- Parenteral Iron therapy
  - Drugs
    - Rare use
    - Iron dextran
    - Iron sucrose
    - Iron sodium gluconate
  - Uses
    - Non compliance of oral iron therapy
    - Severe malabsorption syndrome
    - Marked blood loss
  - Side Effect
    - Hypersensitivity reactions.
      - Urticaria up to anaphylactic reactions I.M. , I.V
    - Local pain & tissue staining ( I.M. )
    - Headache, fever
- Oral iron preparations
  - Drugs
    - Ferrous sulphate
    - Ferrous gluconate
    - Ferrous succinate
    - Ferrous fumarate
  - Side Effect
    - Black stool
    - Teeth staining
    - GIT disturbances
      - Nausea, abdominal cramps and diarrhea or
- Iron toxicity
  - Acute
  - Chronic

Done by: Norah Alsyefi

# Vitamin B12 (cobalamin)

- deficiency
  - pernicious anemia
    - characterized by
      - Megaloblastic anaemia
      - Neuropathy
    - caused by
      - Lack of intrinsic factor
      - Diseases that interfere its absorption in the ileum
        - *e.g. inflammatory bowel disease*
- It is absorbed from GIT in presence of intrinsic factor.
  - Intrinsic factor is a protein secreted by parietal cells of the stomach
    - It is stored mainly in the liver (3- 5 mg)
    - It may be absent in patients having pernicious anemia
    - Full absorption of vitamin B12 requires intrinsic factor (gastric mucosa)+ receptors mediated transport system in ileum
- Should be supplied by dietary sources as eggs, dairy products & liver
- Function
  - Conversion of methylmalonyl CoA to succinyl-CoA
    - *essential for lipid metabolism & normal neuronal function of the brain*
  - Conversion of homocysteine into methionine
    - *this is required in folic acid metabolism and DNA synthesis (necessary for red blood cells production).*
- Vitamin B12 is not synthesized in human
- Preparations available
  - Cyanocobalamin
  - Hydroxocobalamin (preferable)
    - because it is more bound to plasma protein + ↑ t ½ longer half life
- USES
  - Naturally occurring pernicious anemia (life long therapy, I.M.)
  - Cyanide poisoning (hydroxocobal amin)
  - Gastrectomy
  - Neurological abnormalities

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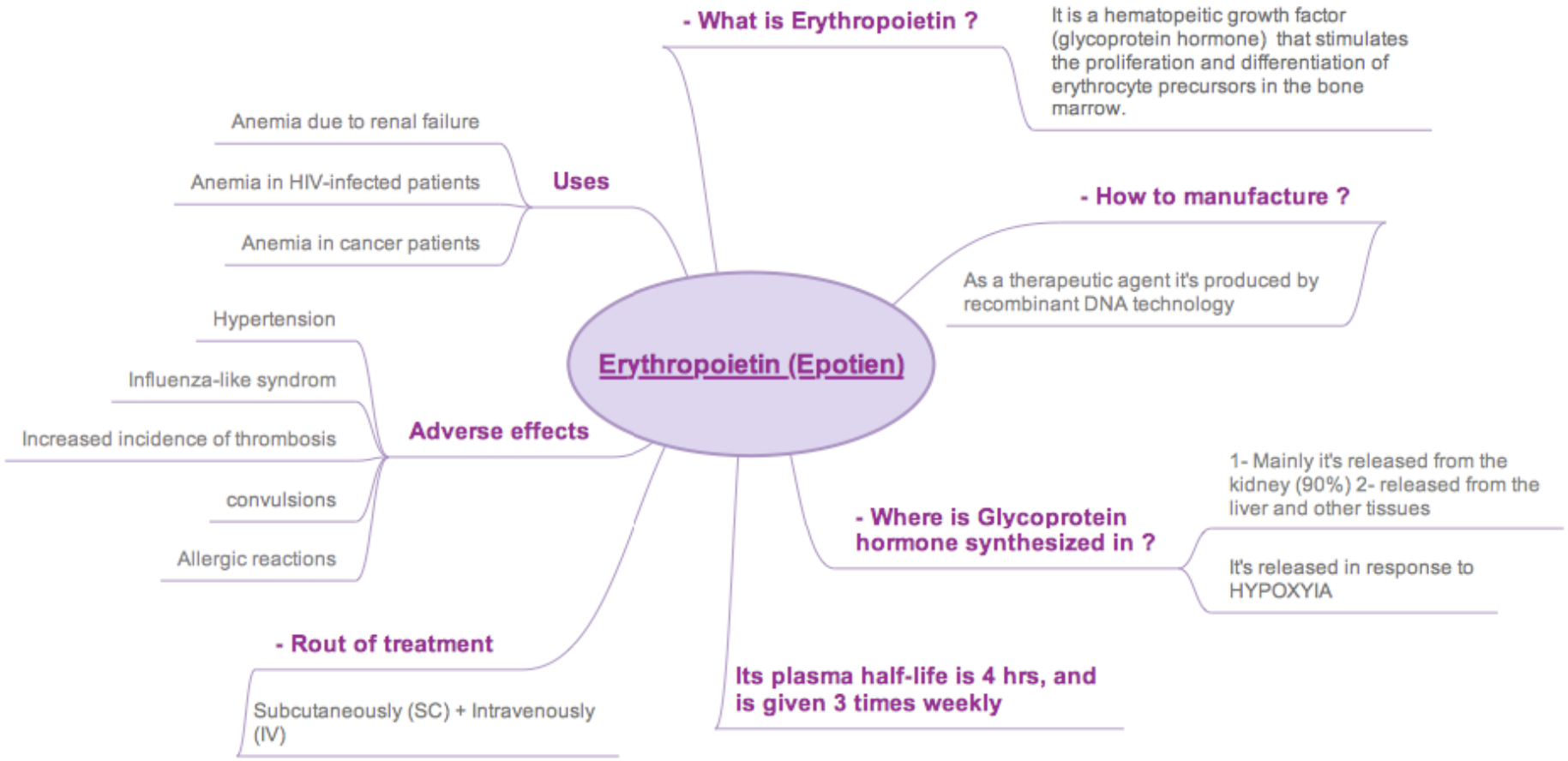
Erythropoietin (Epotien)
- What is Erythropoietin ?
It is a hematopeitic growth factor (glycoprotein hormone) that stimulates the proliferation and differentiation of erythrocyte precursors in the bone marrow.
- How to manufacture ?
As a therapeutic agent it's produced by recombinant DNA technology
- Where is Glycoprotein hormone synthesized in ?
1- Mainly it's released from the kidney (90%) 2- released from the liver and other tissues
It's released in response to HYPOXYIA
Its plasma half-life is 4 hrs, and is given 3 times weekly
- Rout of treatment
Subcutaneously (SC) + Intravenously (IV)
Adverse effects
Hypertension
Influenza-like syndrom
Increased incidence of thrombosis
convulsions
Allergic reactions
Uses
Anemia due to renal failure
Anemia in HIV-infected patients
Anemia in cancer patients

# Folic Acid

**Sources**: 1-Green vegetables, 2-Liver, 3-Kidneys, 4-Yeasts "Folic acid in these sources is in the inactive form METHYL tetrahydrofolates so as it gets inside the cell it needs to be activated by reducing methyl tetrahydrofolate to tetrahydrofolate polyglutamate "

**Deficiency:** can be caused by

- **Poor diet;** alcoholics is common (EXTRA INFO: alcohol prevents absorption of several nutrients especially the B vitamins )
- **Pregnancy**; increases the risk of neural tube defect in fetus
- **Drugs**; Phenytoin, Phenobarbitone
- **Premature infants** (EXTRA INFO: It is now widely recognized that folate supplements are necessary and best started before pregnancy occurs since closure of the neural tube occurs by day 28 of pregnancy.)
- **Hemolytic anemia**
- **Folate antagonist** ( Suplphonamides, methotrexate)

**Function**: Folic acid is necessary for:

- 1-Amino acids metabolism
- 2- Synthesis of purines and pyrimidines (EXTRA INFO: Purines and pyrimidines make up the two groups of nitrogenous bases, including the two groups of nucleotide bases. Two of the four deoxyribonucleotides and two of the four ribonucleotides, the respective building blocks of DNA and RNA, are purines.)

**Preparations:** Synthetic folic acid (tablets & parenteral)

**Kinetics:**

- **In Cells**: Folic acid is reduced first to cihydrofolate then to tetrahydrofolate by cihydrofolate reductase enzyme. So folate is demrthylated "taking methyl group off" to tetrahydrofolate to be activated. Vitamin B12 is required for activation of folic acid to bind to the methyl group.
- **In intestine**: Folic acid or dietary folates are absorped COMPLETELY by active transport system; needs carrier and energy

**Contraindications:** Pernicious anemia (Vitamin B12 deficiency). Folic acid can aggravates the neurological symptoms although blood picture may improve. (EXTRA INFO : Folic Acid is contraindicated in patients who have shown previous intolerance to the drug)

**Uses:**

- **Megaloblastic anemia** ( Folic acid deficiency) (EXTRA INFO : megaloblastic anemia can be caused by either folic acid deficiency or B12 deficiency. When it's caused by vitamin B12 deficiency and the patient has been given folic acid the main problem is not cured it's only covered (WHY?)
- **Pregnancy**; to avoid the risk of neural tube defect in newborns
- **During cytotoxic therapy** (Methotrexate) (EXTRA INFO : Methotrexate (MTX) is a chemotherapy drug that prevents cell reproduction by interfering with folic acid activation. Prolonged treatment with MTX may lead to folic acid deficiency. To reduce the risk of this side effect, without fear of interfering with its efficacy, a maximum daily dose of 5mg of folic acid is recommended for those patients taking MTX)