
بسم الله الرحمن الرحيم

WATER SOLUBLE VITAMINS

Non B-complex

Vitamin C

B-complex

Energy Yeleasing

Thiamine (B_1)

Riboflavin (B_2)

Niacin(B_3)

Pantothenic Acid(B_5)

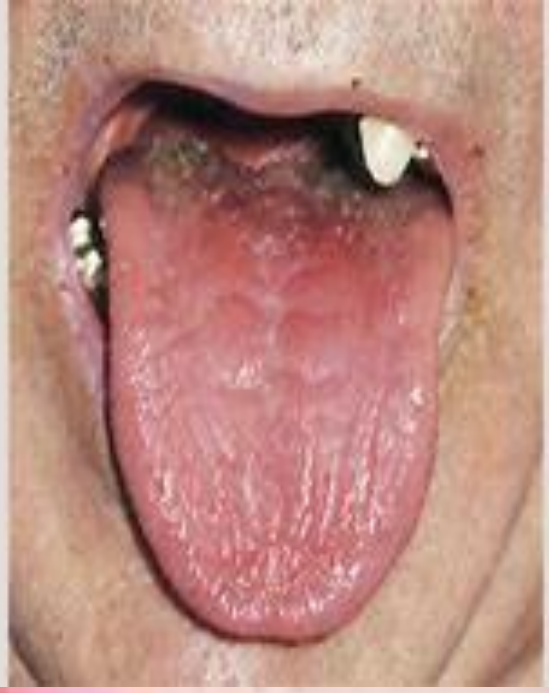
Pyridoxine(B_6)

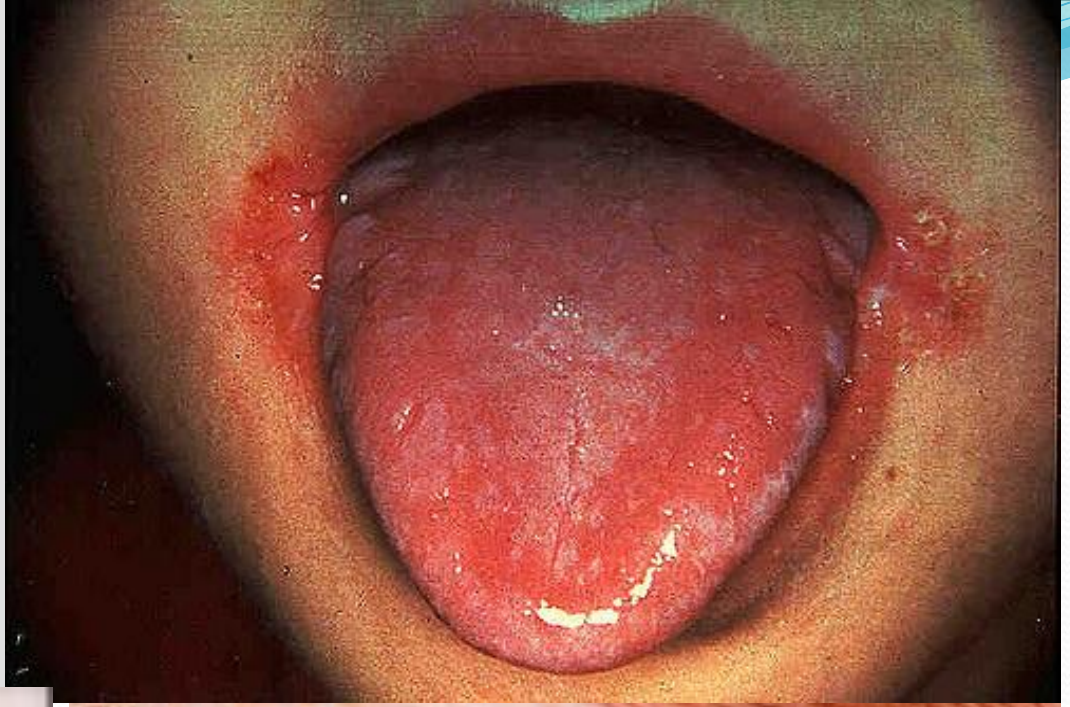
Biotin(B_7)

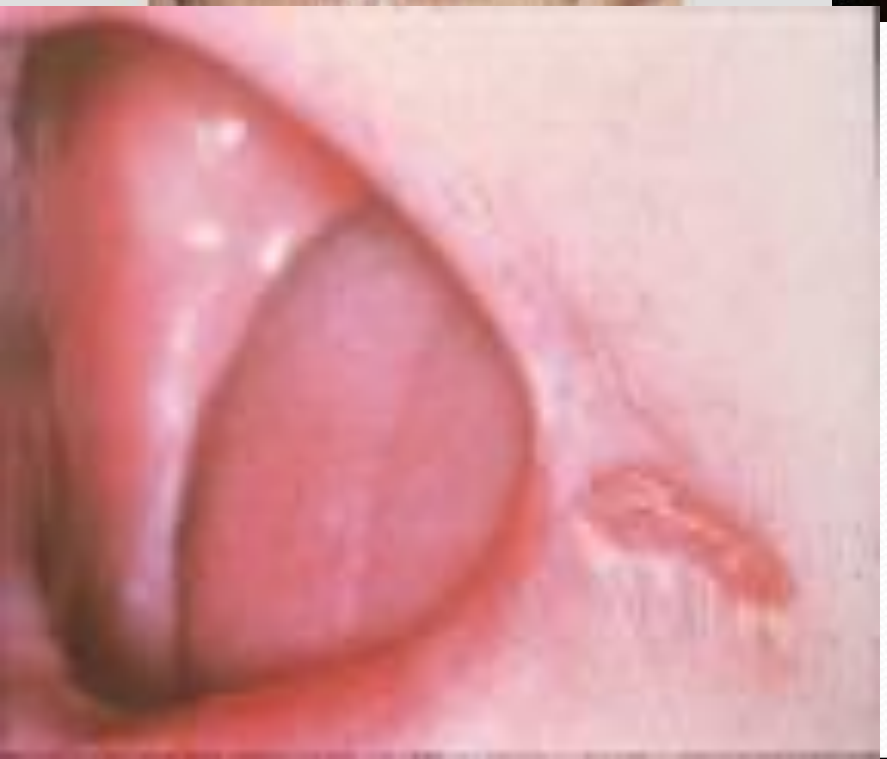
Hematopoietic

Folic Acid

Vitamin B_{12} / Cyanocobalamin







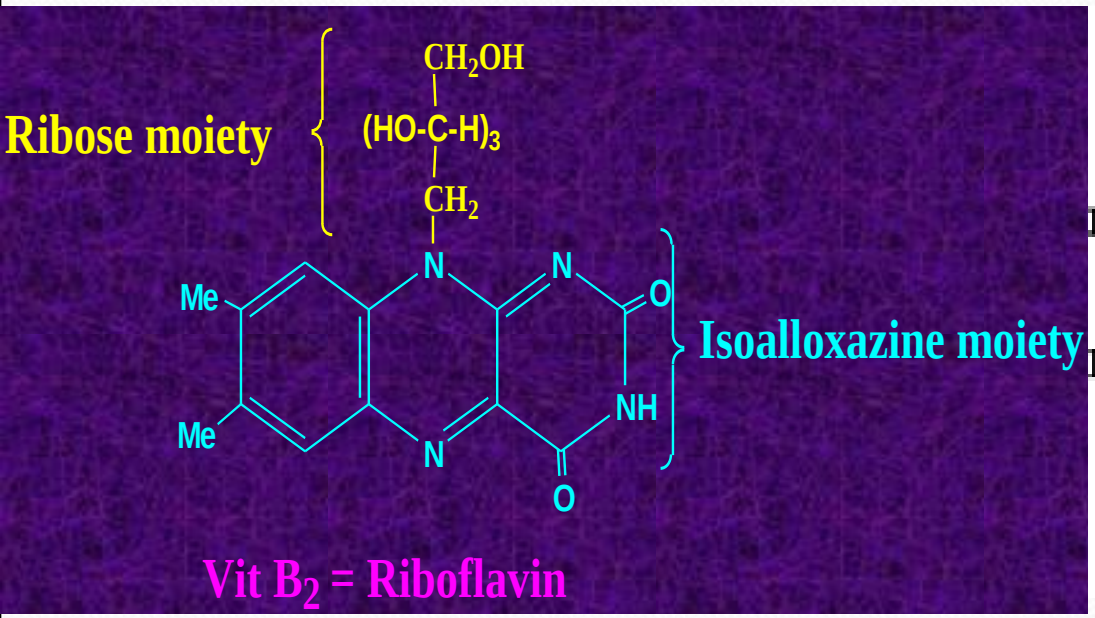

VITAMIN
B2

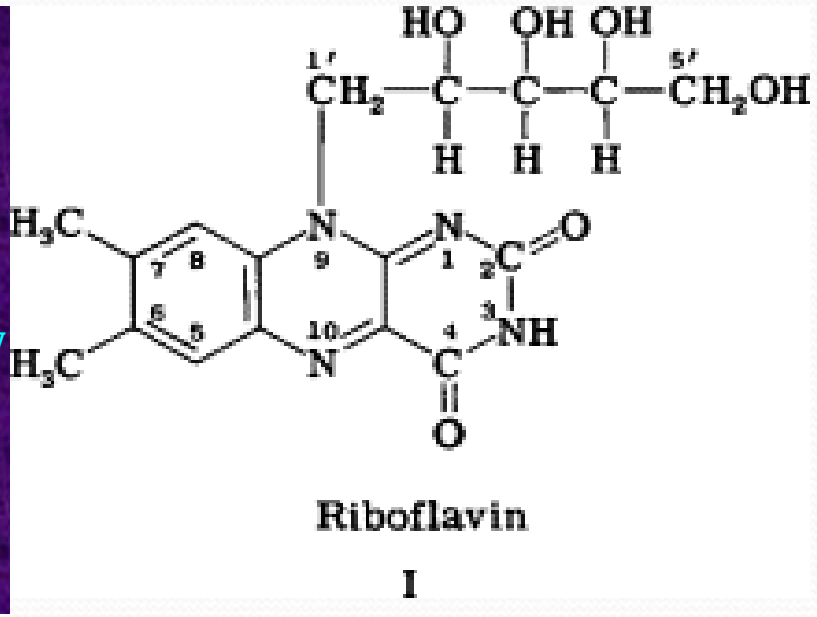
Objectives

- Chemical structure of Riboflavin
- Sources of Riboflavin
- Absorption of Riboflavin
- Biologically active form of Riboflavin
- Biochemical role of Riboflavin
- Deficiency manifestation & causes of deficiency of Riboflavin

Vitamin B_2

Riboflavin, lactoflavin, Vitamin G





➢**It chemically has a three rings structure (isoalloxazine) linked to ribityl moiety. I carbon of ribityl attached to 9 position of isoalloxazine.**

➢**Riboflavin is a yellow to orange- yellow powder, soluble in water. Heat stable.**

Stability

When exposed to UV light, converted to lumiflavin which emits yellow fluorescence.

Vitamin B_2 is unstable to light in both acidic and basic medium.

- **Under acidic condition light produce lumichrome.**
- **In alkaline PH light produce lumiflavin.**

Both are inactive biologically.

Natural Sources

- **Milk, kidney, eggs, liver and meat.**
- **Whole grains, peas,nuts, germinating seeds and green leafy vegetables**
- **Yeast**

RDA

1.2- 1.8 mg/day.

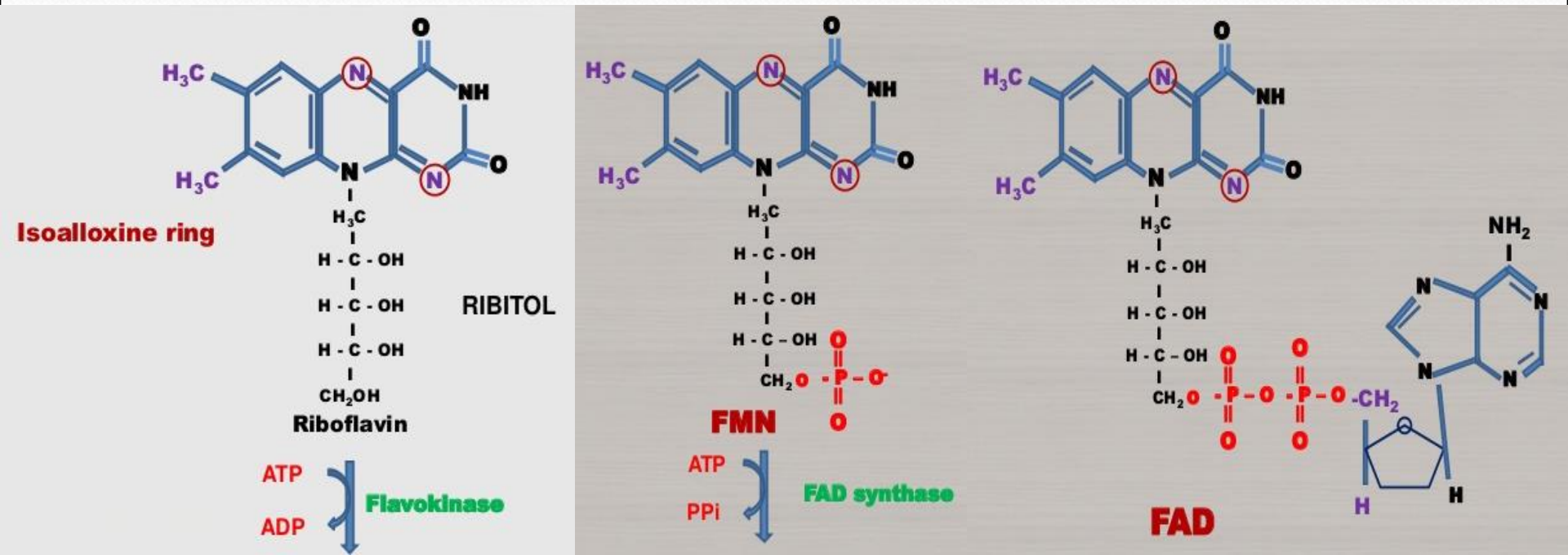
Absorption

- **Riboflavin is absorbed in the proximal intestine.**
- **Passes to all tissues through general circulation**
- **Riboflavin is not stored, mainly present in the liver, kidney and heart in the form of FAD (70-90%) or FMN or Riboflavin.**

Biologically active forms

Riboflavin is converted to the active forms by enzyme flavo-kinase

- Riboflavin-Mononucleotide (FMN) ,
- Riboflavin-Monophosphate .
- Riboflavin-Adenine Dinucleotide (FAD) ,
- Riboflavin-Adenine Diphosphate



Role of Vitamin B_2

Flavoproteins

Enzymes which use Flavin as coenzymes are called flavoproteins.

Metalloflavoproteins

Many flavoproteins contain metal atoms (iron, molybdenum) which are known as metalloflavoproteins.

The Active forms work as co-enzymes for about 150 oxidation-reduction reactions involved in:

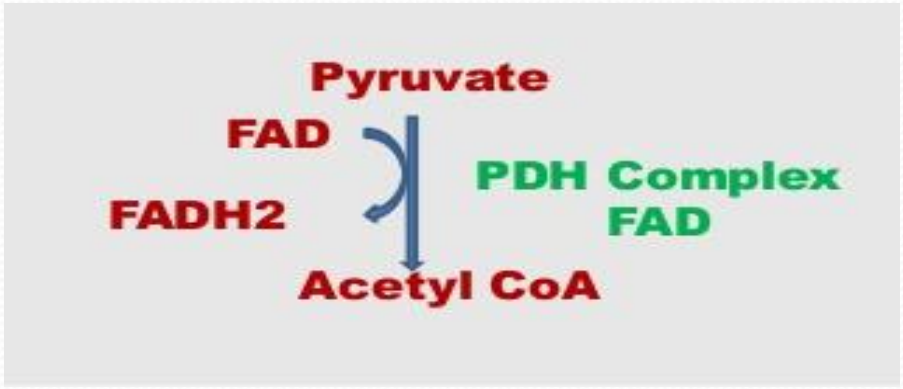
- Carbohydrate, Proteins and fat metabolism
- Activation of vitamin B_{12} and folate.
- Protection of erythrocytes and other cells from oxidative stress.

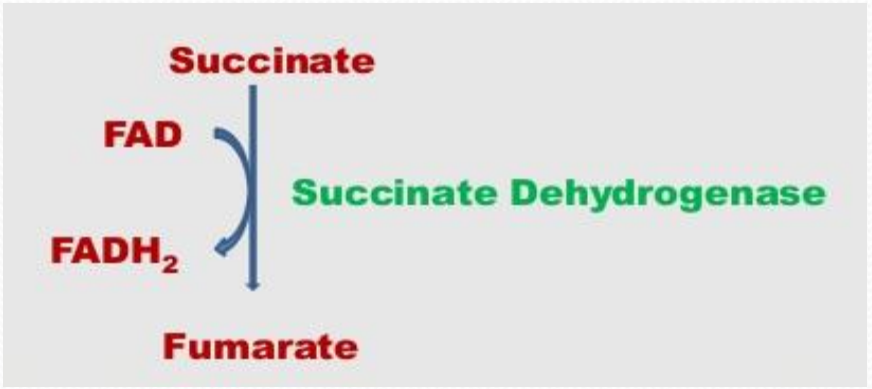
Reactions requiring FMN

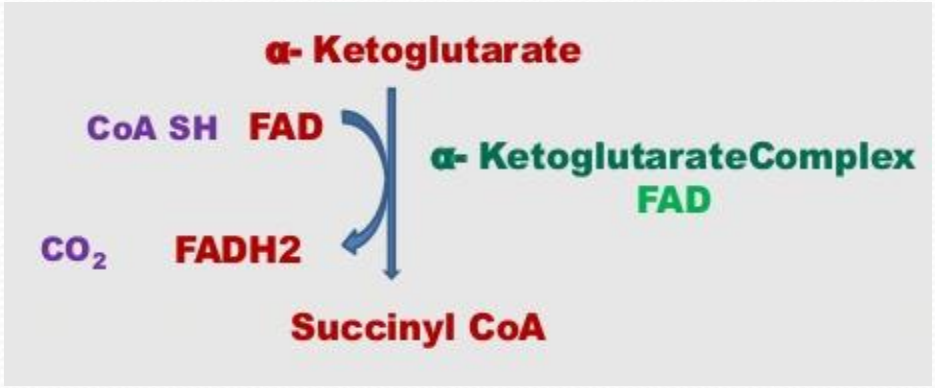
- Coenzyme for

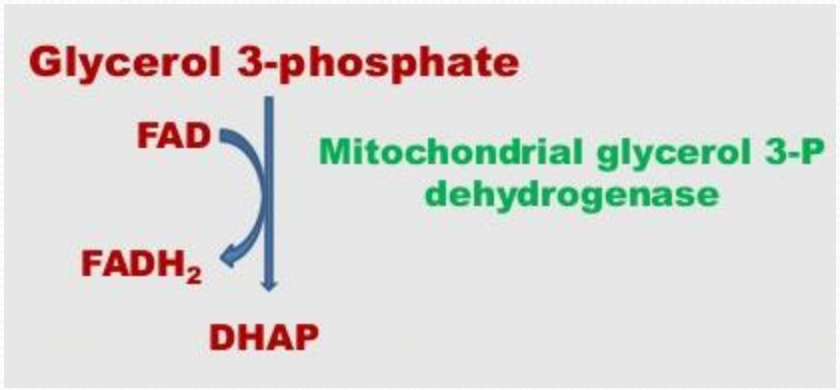
Reactions requiring FAD

- Carbohydrate metabolism





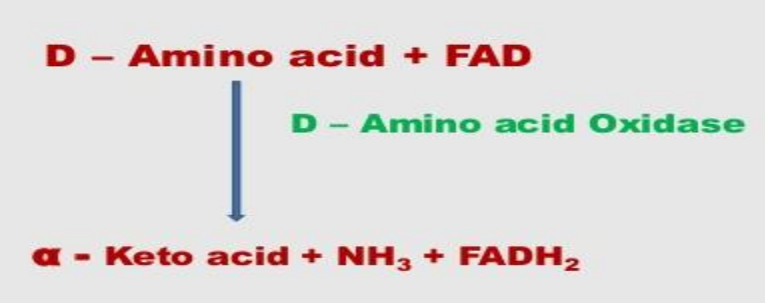




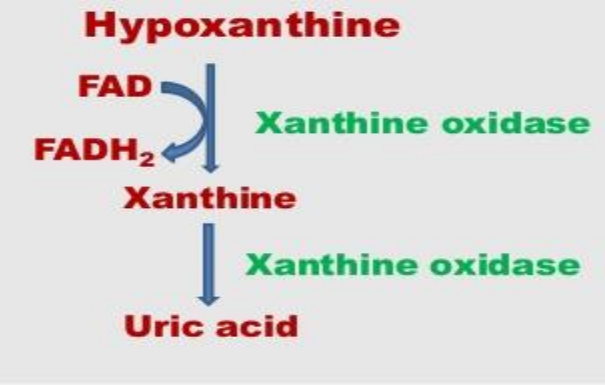
Protein metabolism

- Glycine cleavage system require FAD.

- α-aminoacid oxidase: conversion of D-aminoacid to α ketoacid.



- Xanthine oxidase : conversion of hypoxanthine to xanthine.



Lipid metabolism

- Coenzyme for Acyl-CoA dehydrogenase in fatty acids oxidation.

- It also helps in maintaining mucosal epithelial cell and ocular tissues.
- Involved in the protection against peroxidation in the metabolism of xenobiotics.
- It regenerate glutathione.

Causes of Riboflavin Deficiency

- **Not getting enough of the vitamin from the diet.**
- **A result of conditions that affect absorption in the intestine.**
- **The body not being able to use the vitamin.**
- **An increase in the excretion of the vitamin from the body.**

Risk Factors for Deficiency

- **People under high stress, including those experiencing surgery, chronic illnesses, liver disease, or poor nutritional status.**
- **Diabetics have a tendency to be low on riboflavin as a result of increased urinary excretion.**
- **Athletes, and anyone else with a high-energy output will need additional vitamin B_2.**
- **The elderly due to nutritional inadequacy as well as problems with absorption.**

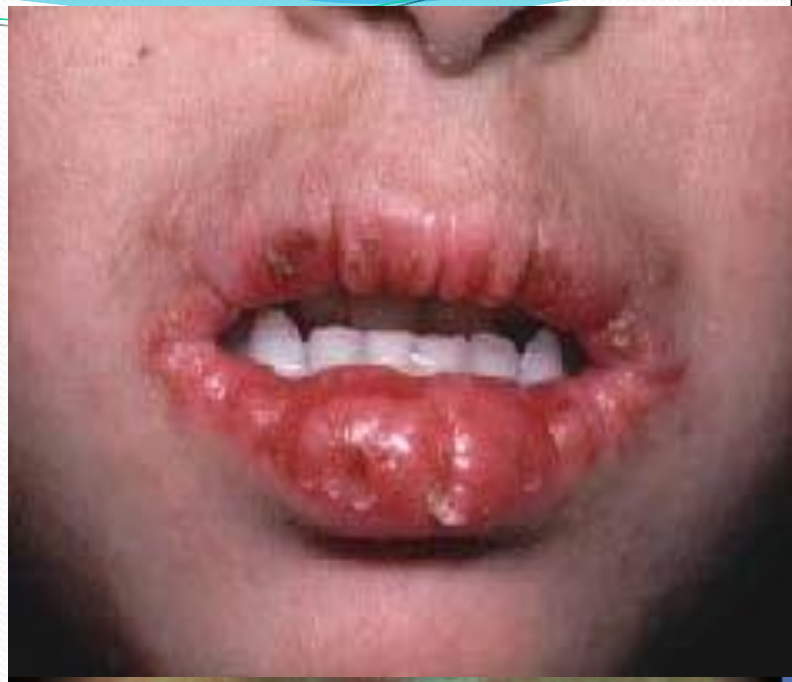
- **Smokers and alcoholics are at higher risk for deficiency as tobacco and alcohol suppress absorption.**

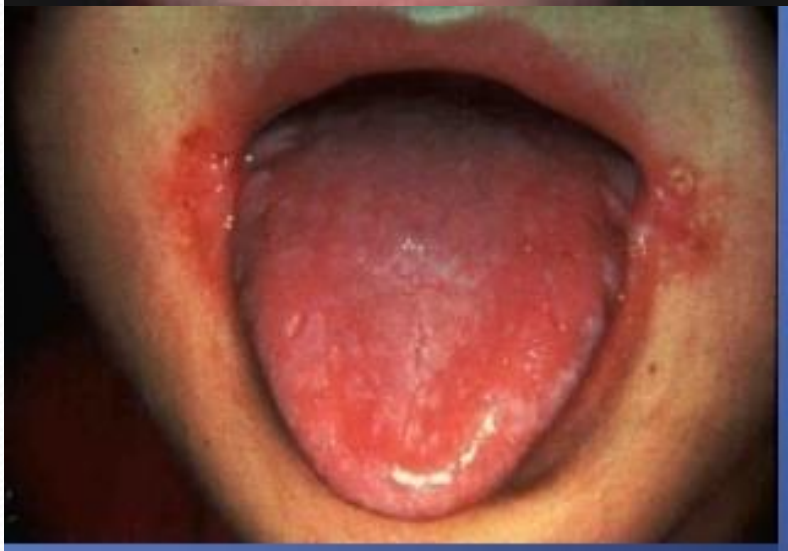
- **Birth control pills may possibly reduce riboflavin levels, as can phenothiazine tranquilizers, tricyclic antidepressants, and probenecid.**

DEFICIENCY

Symptoms of riboflavin deficiency:

- **Cracked and red lips.**
- **Inflammation of the lining of mouth and tongue(glositis).**





cheilitis and glossitis

- **<u>Angular cheilitis:</u>**

  is an inflammatory lesion at the corner of the mouth. Usually associated with a fungal (*Candidal*) or bacterial(*Staphylococcal*) infection. The condition manifests as deep cracks or splits. In severe cases, the splits can bleed when the mouth is opened.

DEFICIENCY

Symptoms of riboflavin deficiency:

Seborrheic dermatitis

- **Dry and scaling skin (dermititis) specially about the naso-labial fold.**
- **Iron-deficiency anemia.**

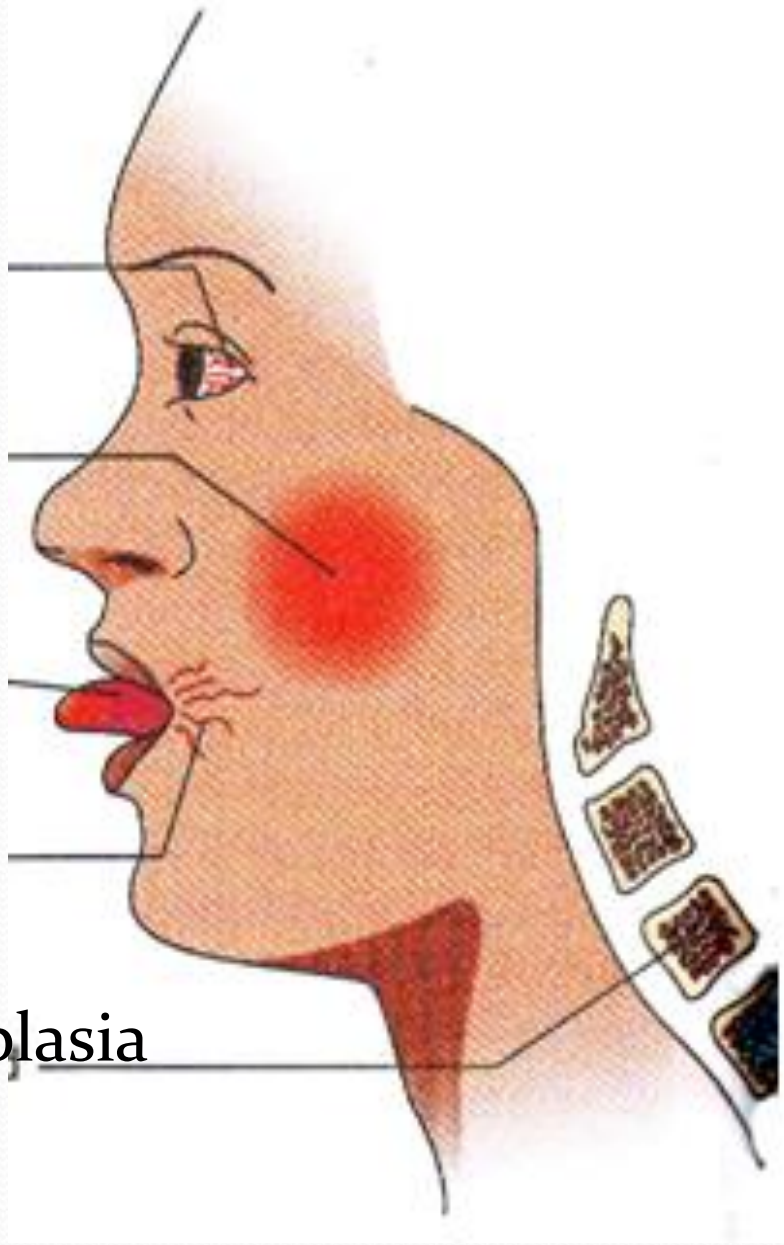
- **Deficiency leads to corneal vascularisation & inflammation. The eyes become bloodshot, itchy and sensitive to bright light.**

- Corneal vascularisation
- Dermatitis

Glossitis

Cheilosis

Anemia,erytheroid hypoplasia



Diagnostic Testing of B_2 Deficiency

1. **A positive diagnostic test of serum riboflavin by measuring glutathione reductase levels of erythrocytes.**

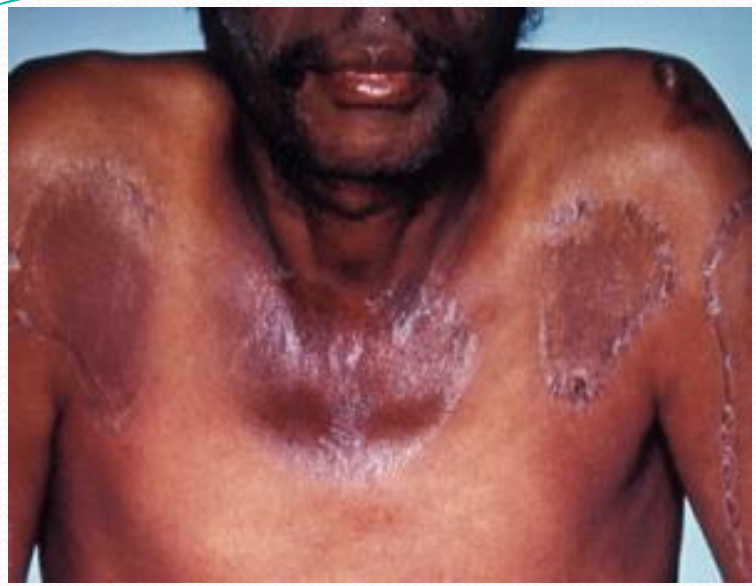
2. **Flourimetric assay of riboflavin in RBCs(15-30 µgm/dl).**

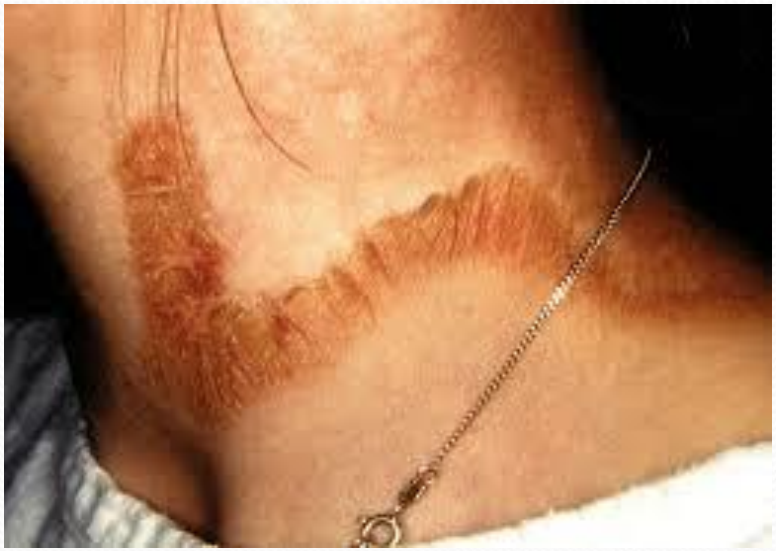
3. **Excretion in urine.**

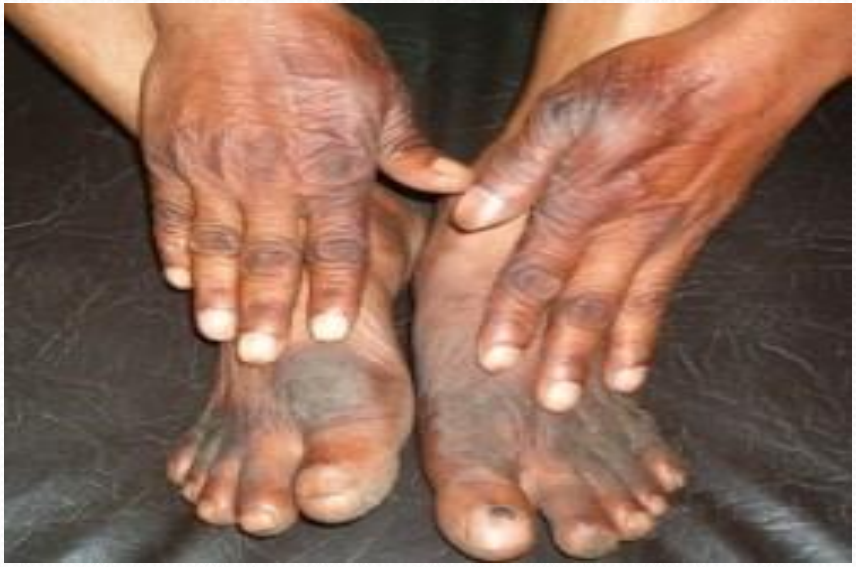
4. **Riboflavin content of blood plasma(2.5-4µgm/dl).**

Uses

- **High doses of riboflavin(400 mg/day) have been shown to reduce the frequency and severity of migraine headaches by half in susceptible people.**
- **Riboflavin help decrease the incidence of cataracts.**
- **Improve memory.**
- **Riboflavin and vitamin C both help boost the body's level of glutathione which is an antioxidant.**
- **Healthy development of the fetus.**







B3

Niacin

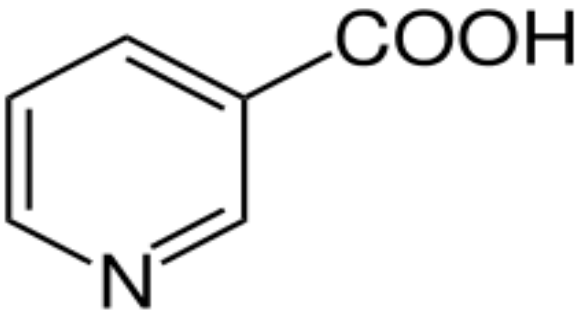
Objectives

- Chemical structure & properties of Niacin
- History of niacin discovery
- Biosynthesis of Niacin
- Sources , daily requirements of Niacin
- Absorption and transport of Niacin
- Biologically active form of Niacin
- Biochemical roles of Niacin
- Deficiency disease of Niacin

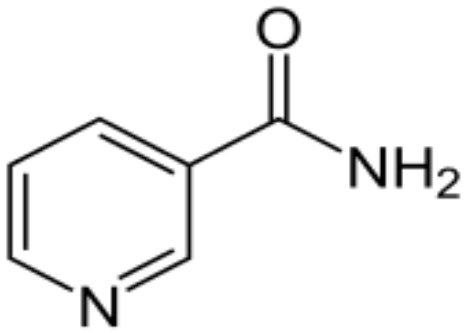
Vitamin B_3

(Niacin, Nicotinic acid, Nicotinamide, Vitamin P, VitaminPP)

pyridine 3-carboxylic acid



Niacin or nicotinic acid



Nicotinamide

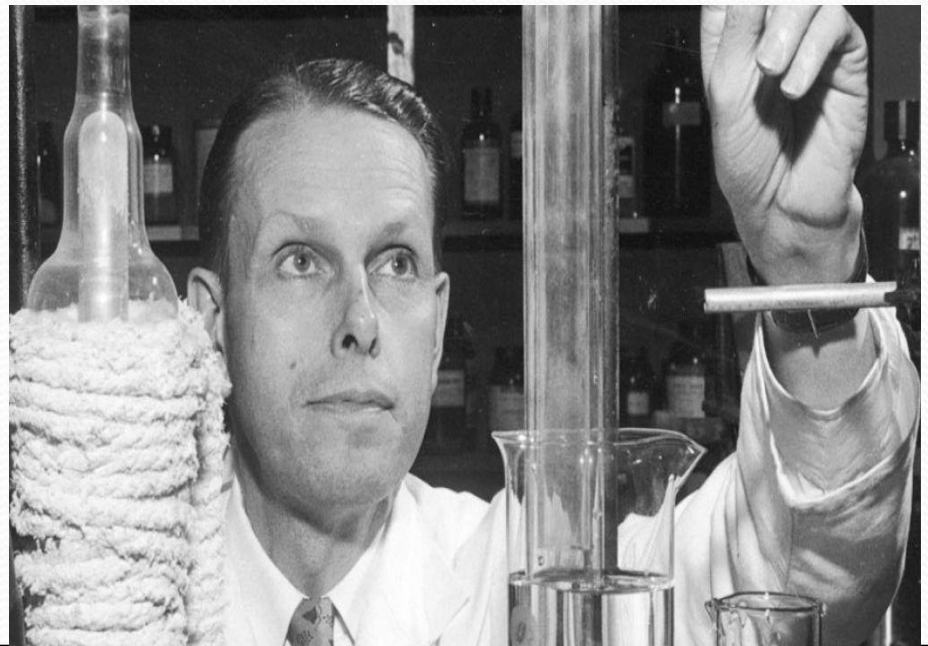
Properties

- Niacin is an odorless white, crystalline substance.
- Soluble in water
- Resistant to heat, oxidation and alkalis.
- It is one of the stable vitamin
- Cooking causes little destruction.

History

- First identified by Joseph Goldberger a researcher in 1928.

- The chemical structure of the Niacin was subsequently **discovered** in 1937 by the American biochemist Conrad Arnold



- Biosynthesis:

The liver can synthesize *Niacin* from the essential **amino acid** Tryptophan, but the synthesis is extremely slow and requires vitamin B_6 (60 mg of Tryptophan= 1mg of niacin).

Bacteria in the gut may also perform the conversion but are inefficient.

Natural Sources

- **Liver, fish, kidney, meat, legumes (peas, beans), nuts & unpolished rice are one of the best sources of niacin.**
- **Cheese, milk are highest in Tryptophan and about half of the Tryptophan consumed is used to make niacin.**

Required Daily Amount

About 7 mg/ 1000 calories.

- **15- 20 mg/day.**
- **Requirement increase in high corn diet(protein zein deficient in tryptophan).**

Absorption

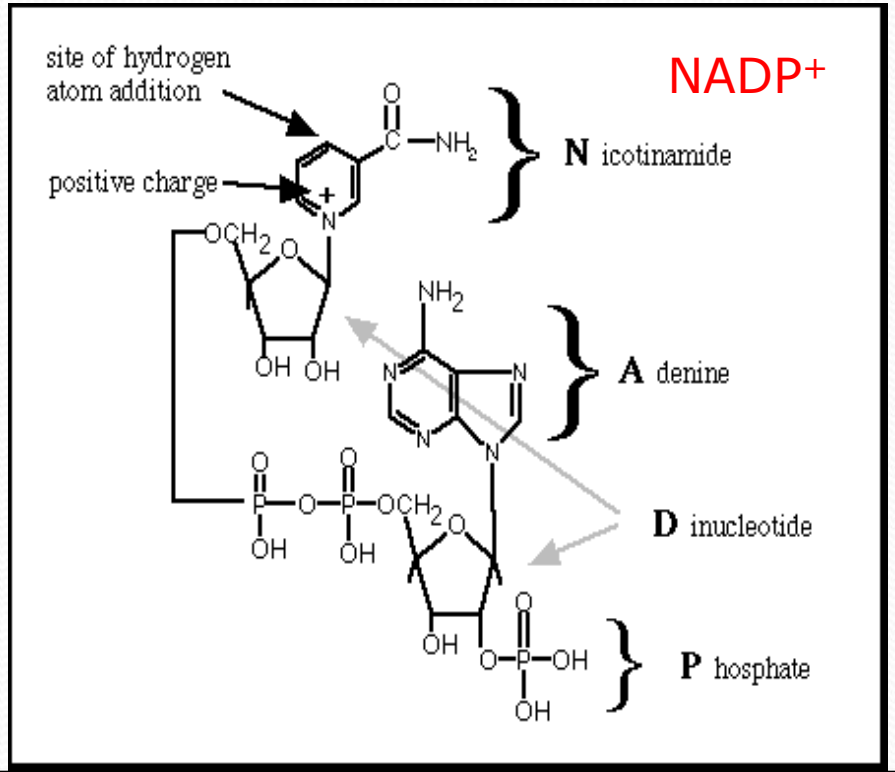
- Absorption:
- Niacin hydrolyzed in the intestinal tract, and both Nicotinic acid (NA) and Nicotinamide (NAM) are absorbed readily.

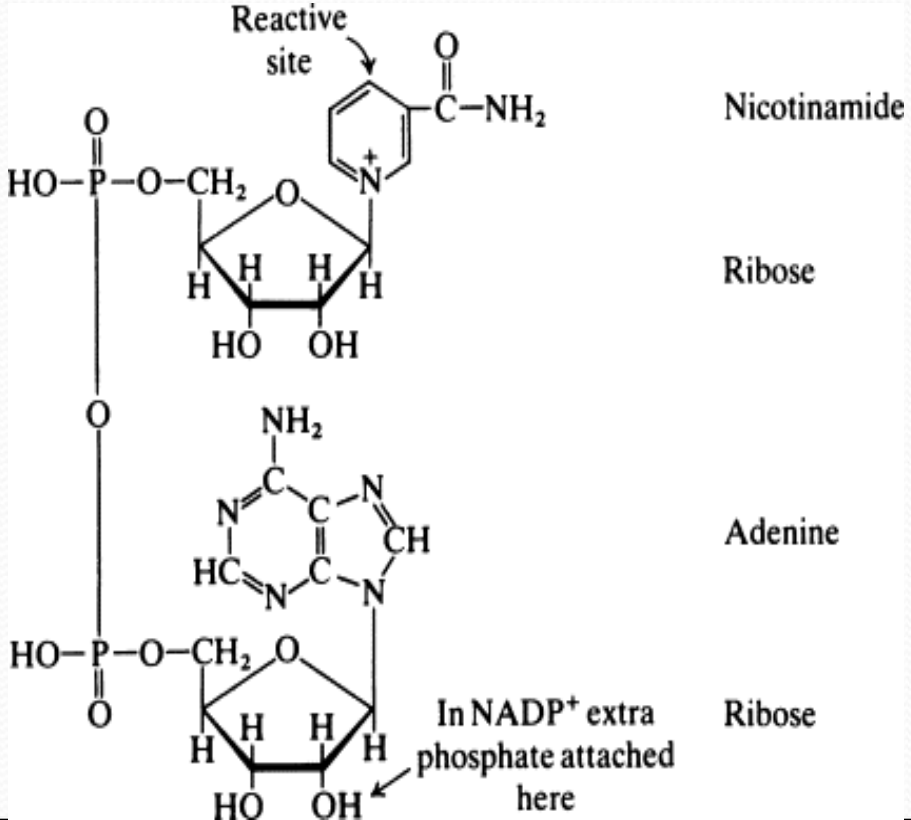
- Both compounds converted to coenzyme form in the blood cells, kidney, brain and liver.

- Excretion :
- Nicotinic acid & its amide are excreted in urine.
- Methylated derivative (N-metyl nicotinamide) is also excreted in urine.

Biological active forms

- NAD^+ consist of
- One molecule of nicotinamide
- Two molecules of D-ribose
- Two molecules of phosphoric acid
- One molecule of adenine





Biological active forms

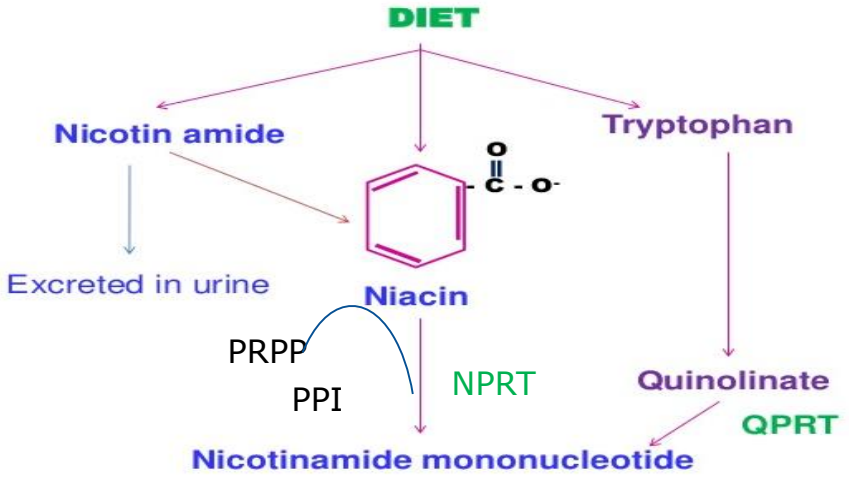
PRPP (phosphoribosylpyrophosphate) and ATP provide ribose and phosphate group.

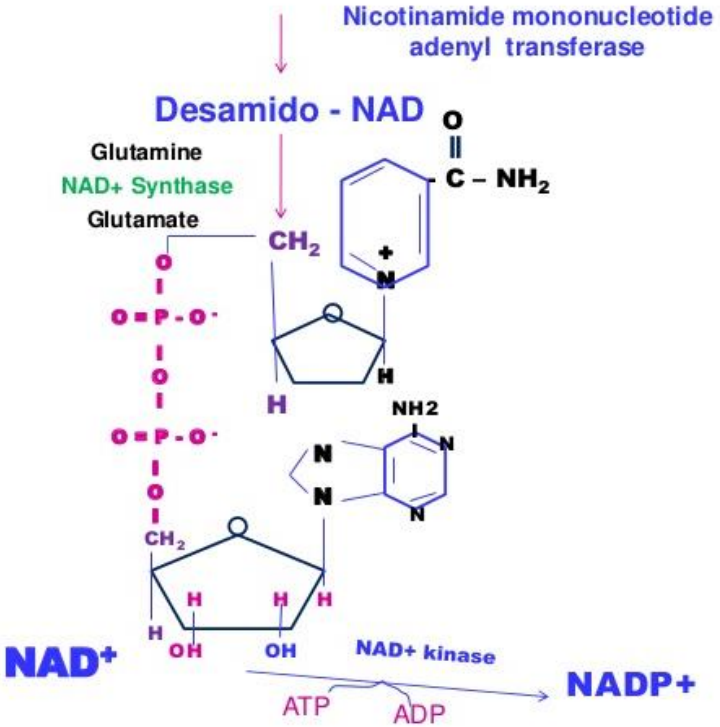
Glutamine donate amide group.

$NADP^+$

Is formed in the presence of NAD^+ kinase.

Positive charge on nitrogen atom is due to formation of extra bond .





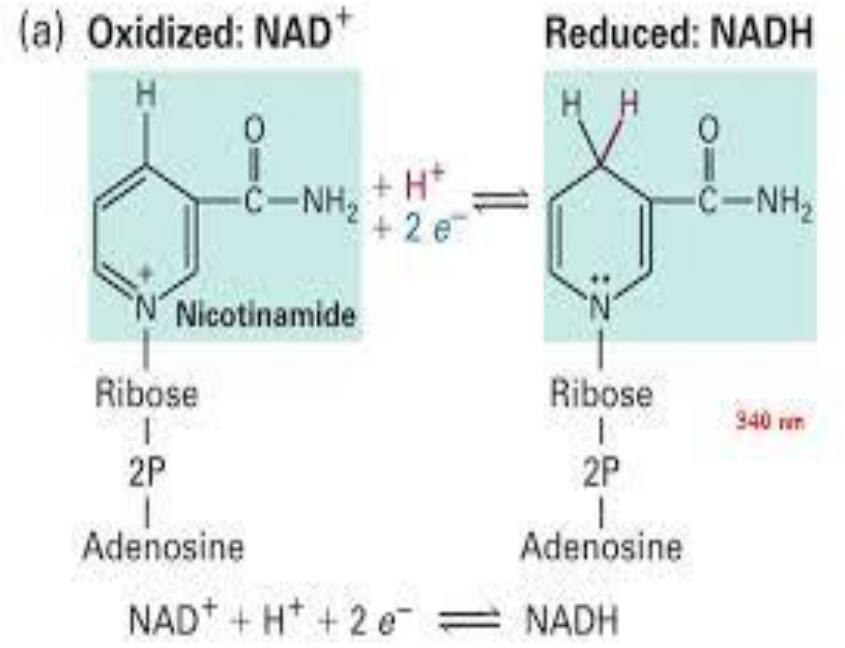
Role of Vitamin B_3

- It is act as co-enzyme in oxidation-reduction reactions:
  - Catabolic reactions:
  - NAD^+/NADH
  - Anabolic reactions:
  - $NADP^+$/NADPH

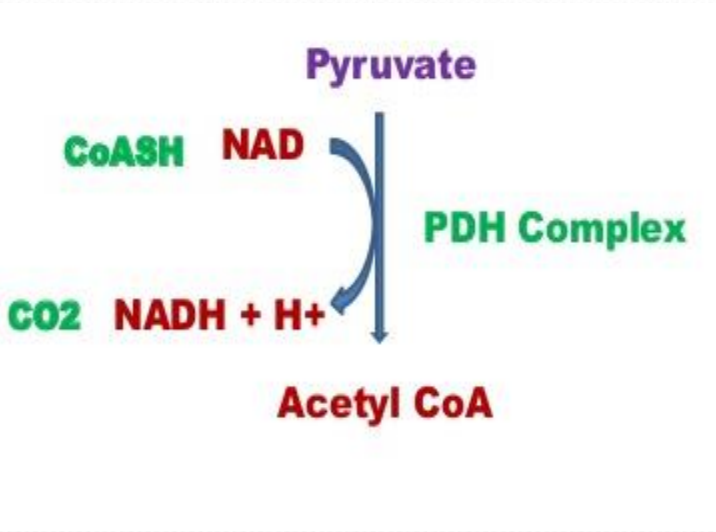
Metabolic function

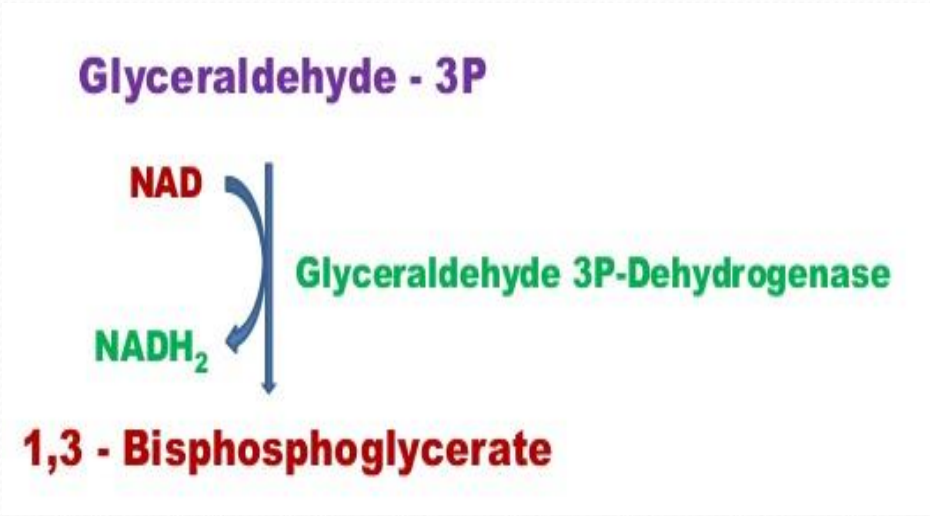
- They Accept hydroid ion(H atom and one electron H-) undergo reduction in pyridine ring. This result in neutralization of positive charges.
- One atom of H is accepted and other(H^+) released into surrounding.

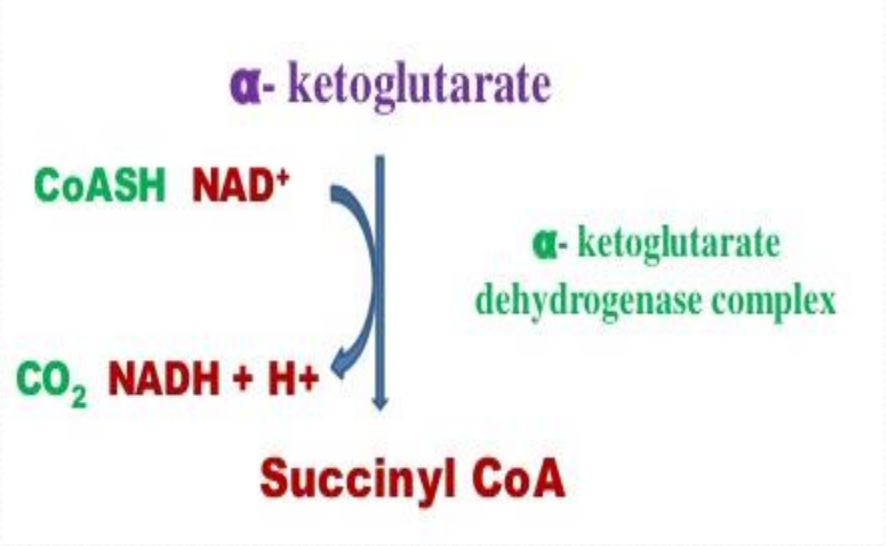
- Oxidoreductases
- (more than 40) depend on NAD^+ $NADP^+$.
- NADH is oxidized in ETC.



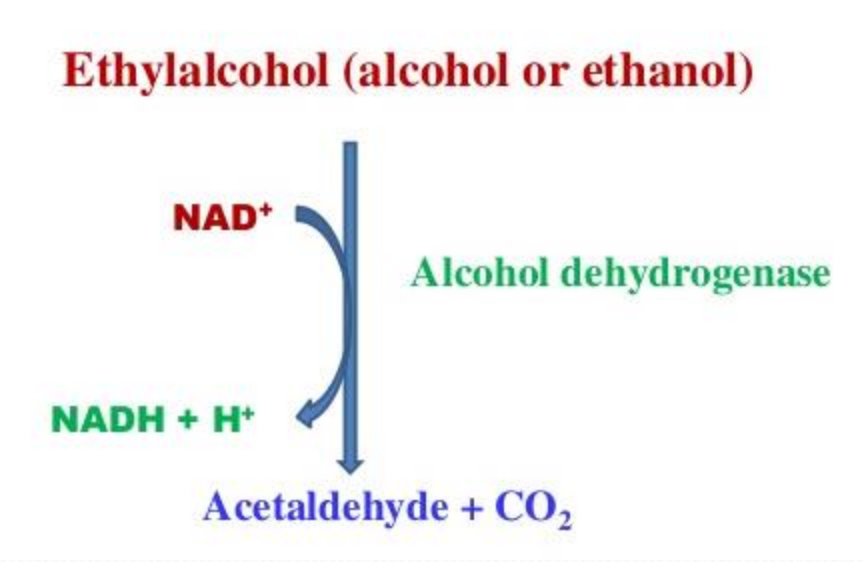
Carbohydrate metabolism

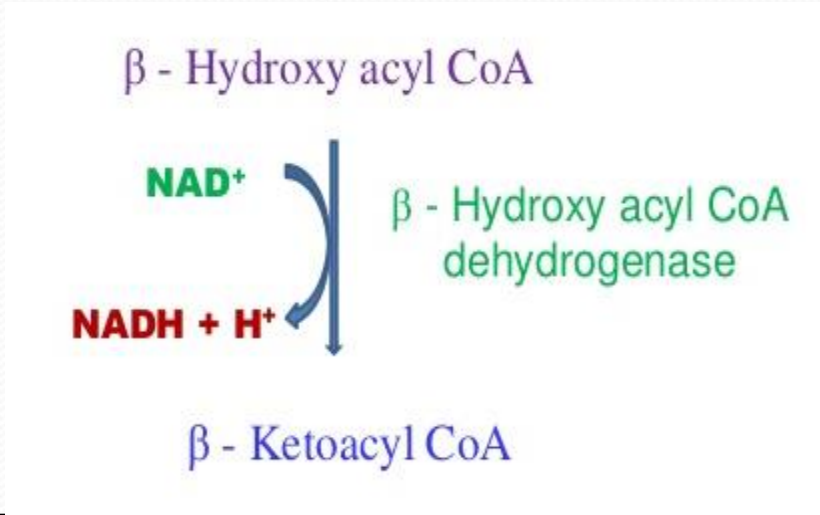




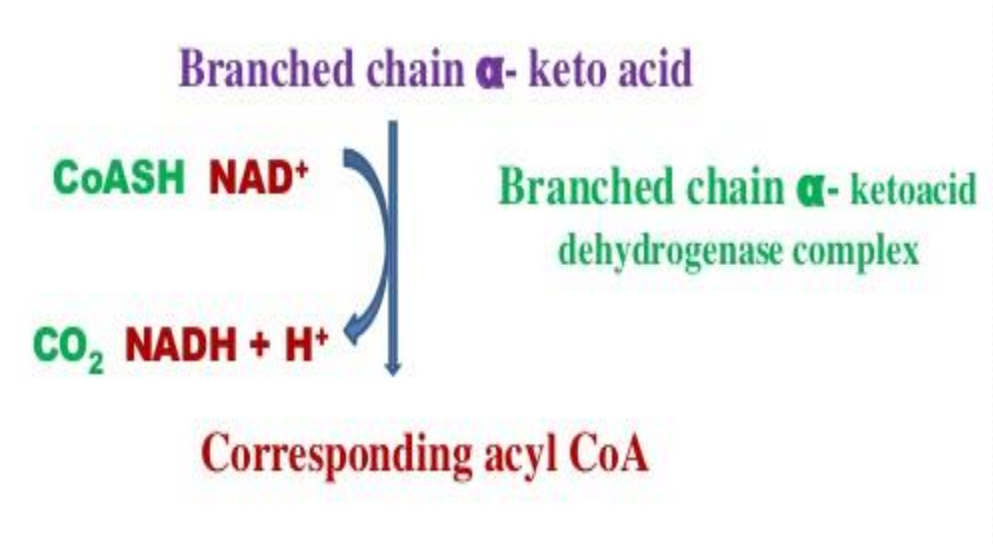


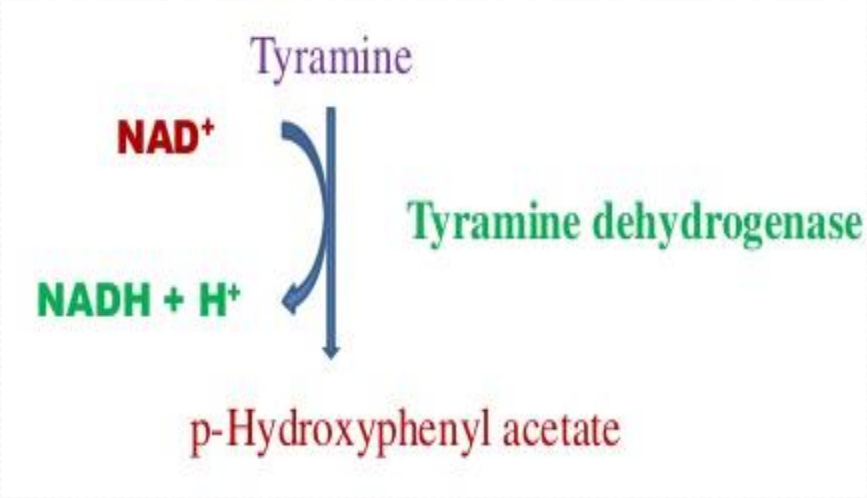
Lipid metabolism



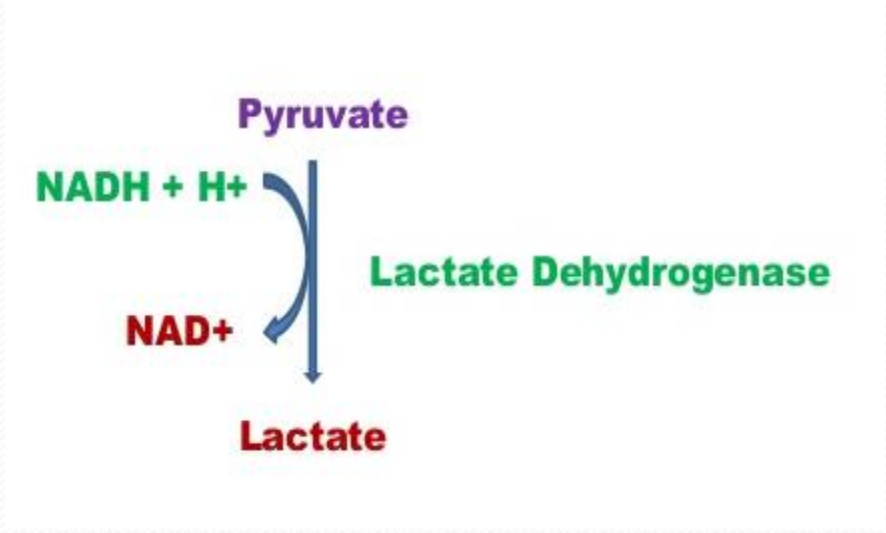


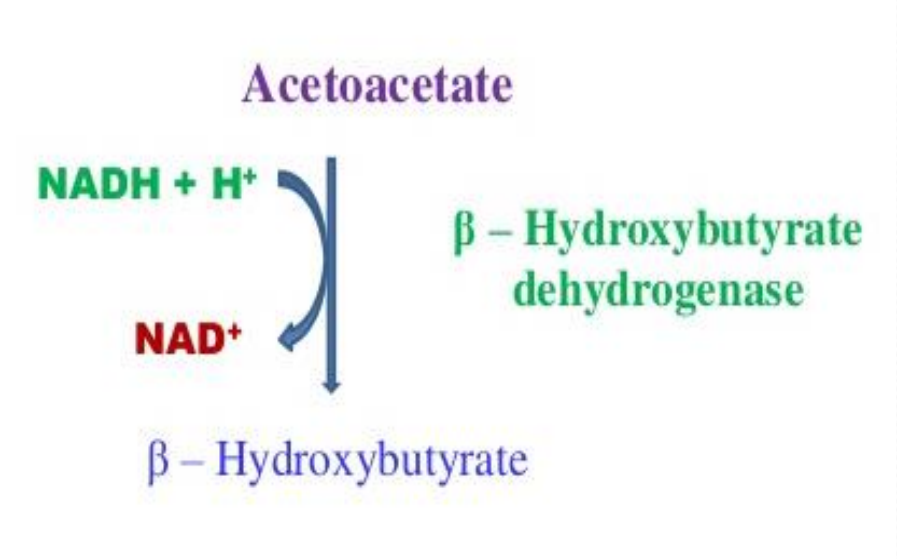
Protein metabolism



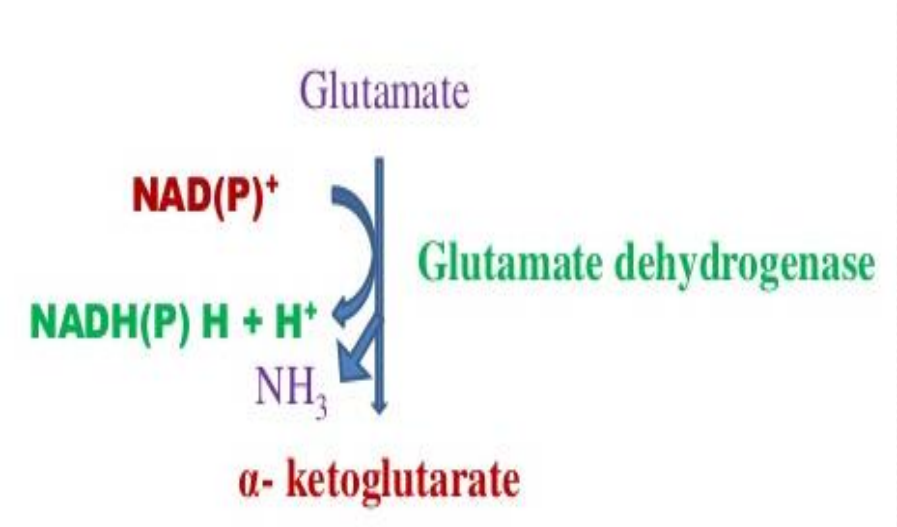


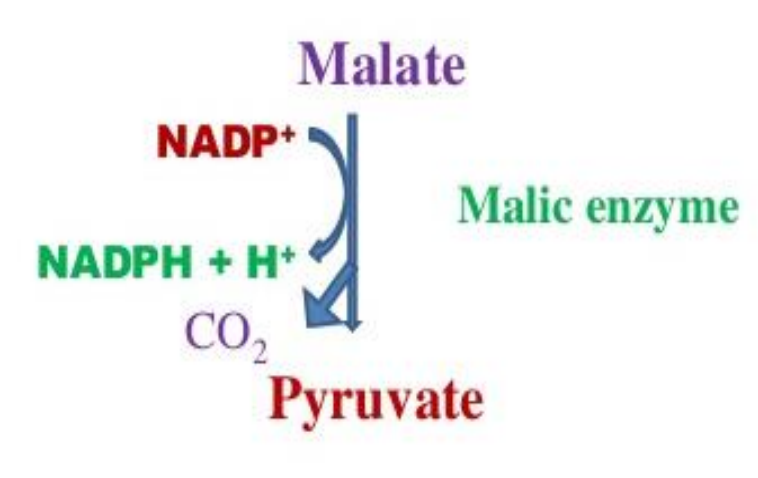
NADH dependent reactions

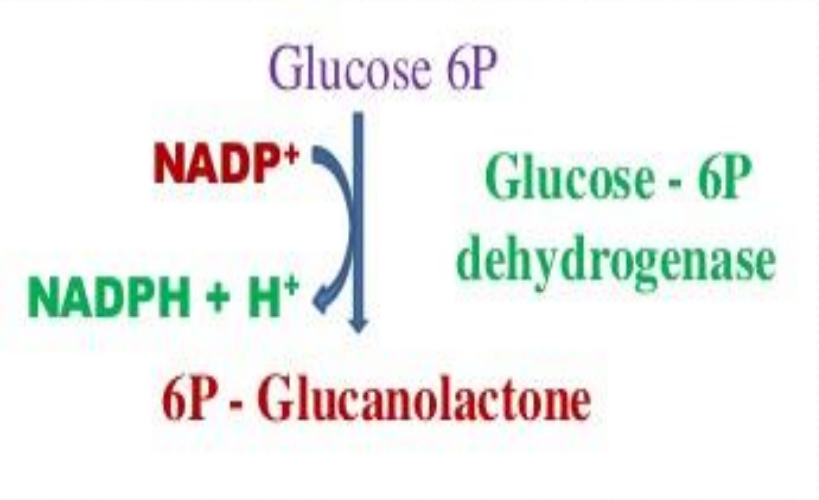




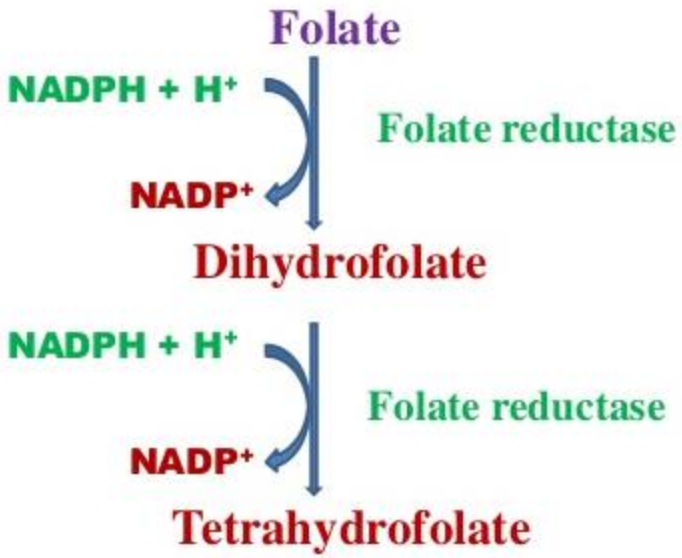
$NADP^+$ dependent reactions

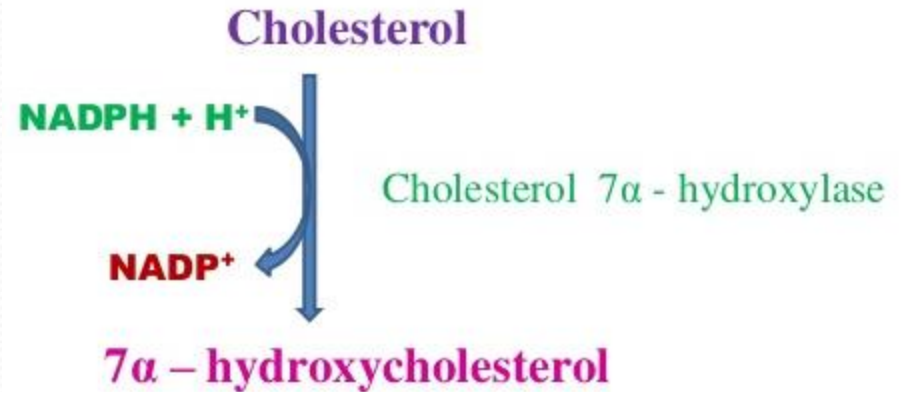


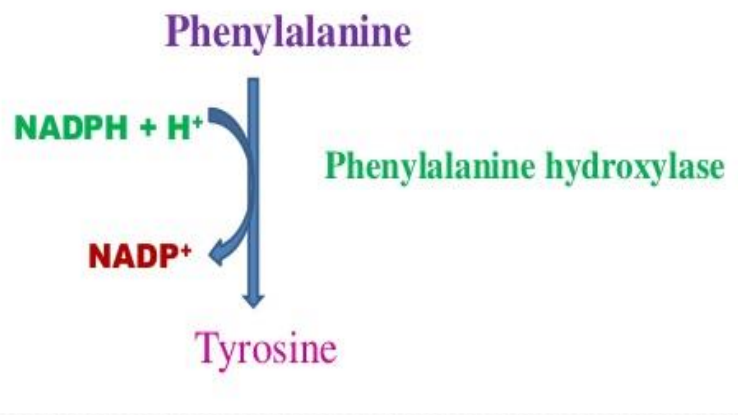


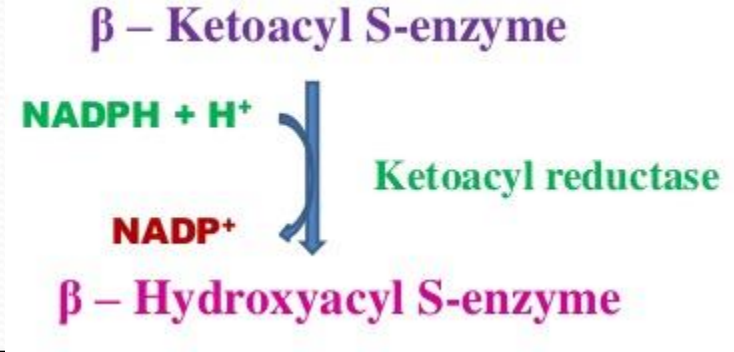


NADPH dependent reactions









Deficiency

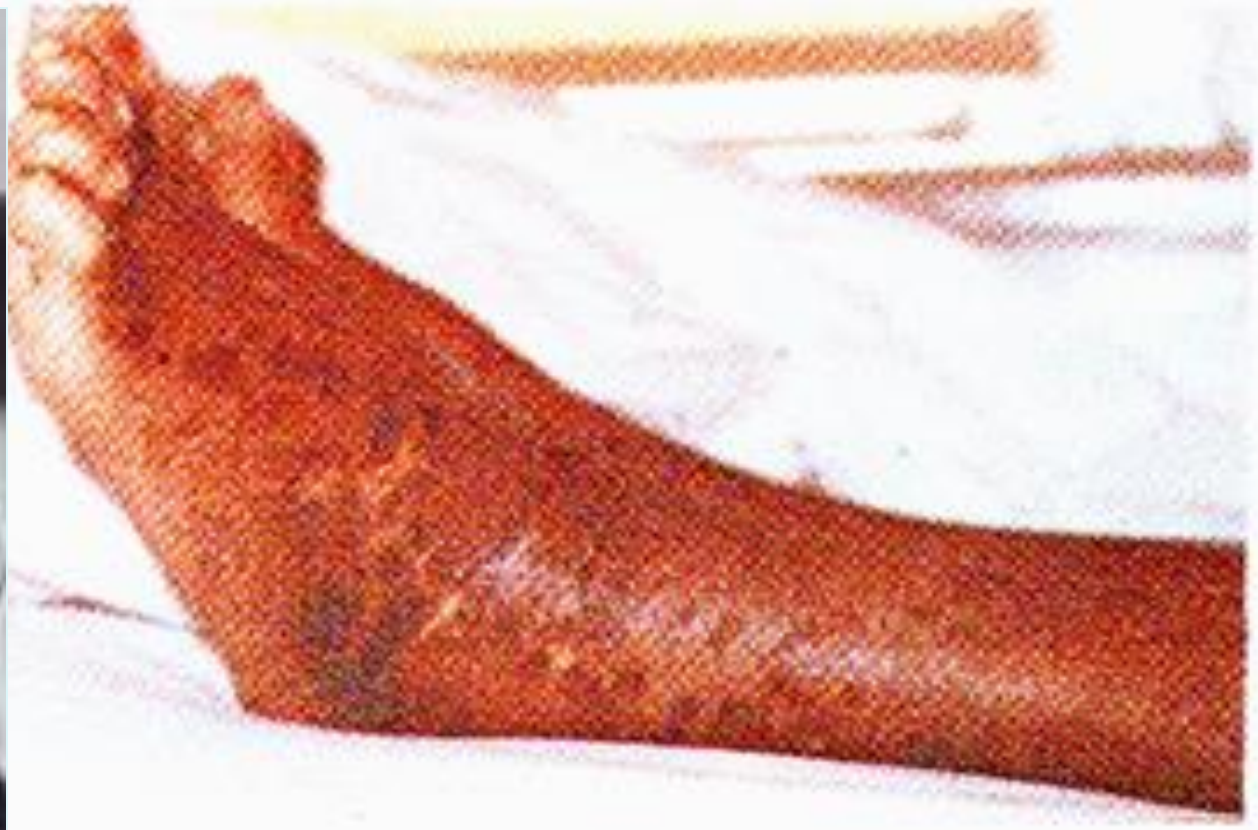
Pellagra:

A serious deficiency of niacin. The main results of pellagra can easily be remembered as "the four D's": diarrhea, dermatitis, dementia, and death. It is very rare now, except in alcoholics, strict vegetarians, and people in areas of the world with very poor nutrition.

Dermatitis ppted on exposure to sunlight,is due to role of NAD in DNA repair reactions following damage through exposure to UV-light.

Pellagra

The typical dermatitis of pellagra develops on skin that is exposed to light.



- Milder deficiencies of niacin can cause Dermatitis
- skin reddened thickened and scaly and rough.
- Diarrhea
- Nausea,vomiting,abdominal pain & diarrhea . Gingivitis & stomatitis is also reported.
- Dementia
- Headache, insomnia, depression & psychosis.

Causes of deficiency

- Dietary deficiency of tryptophan.
- Lack of vitamin pyridoxin ..... Kynureninase depends on pyridoxal phosphate.
- Anti tubercular drugs causes B6 deficiency.

General Uses

- **Niacin in very large doses (2-3g/d) is used to decrease blood cholesterol levels and reduce the risk of heart attack.**
- **In certain conditions (gout,diabetes, peptic ulcer, liver or kidney disease, and high blood pressure).**
- **Niacinamide used on a long-term basis to prevent the onset of juvenile diabetes.**
- **Treatment of Pellagra.**

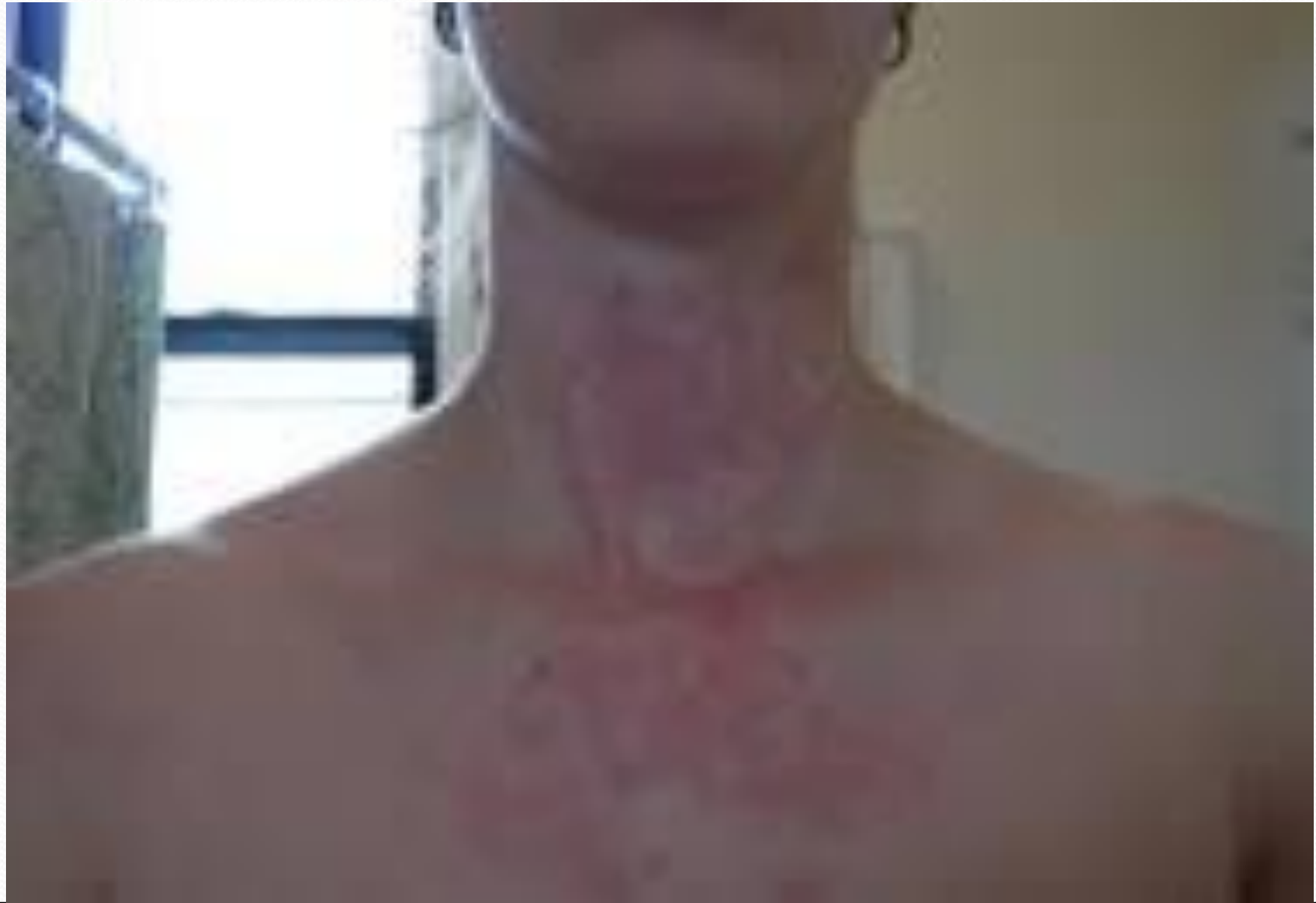
Side Effects

In large amounts Niacin commonly causes flushing and headache. Skin irritation and liver damage.

Glycogen and fat reserves of muscles depleted (cardiac).

Increase level of glucose ,uric acid and certain enzymes.

Niacin rash



Niacin flush

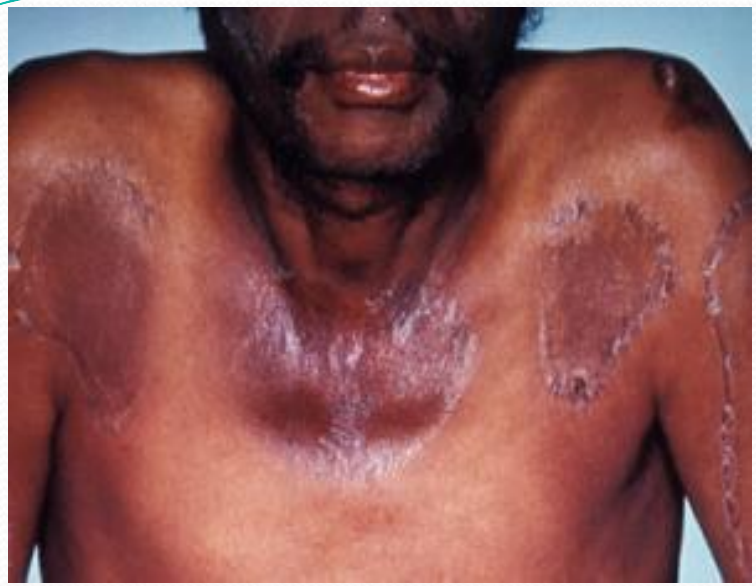
Pellagra like conditions

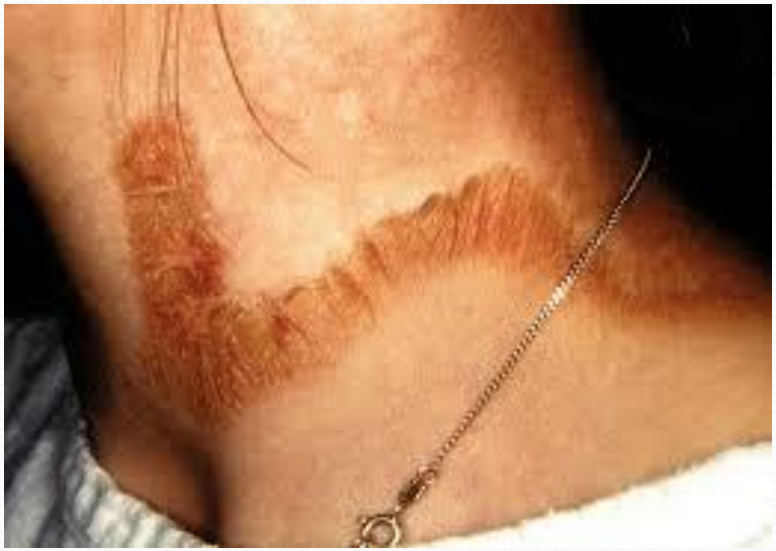
- Carcinoid syndrome

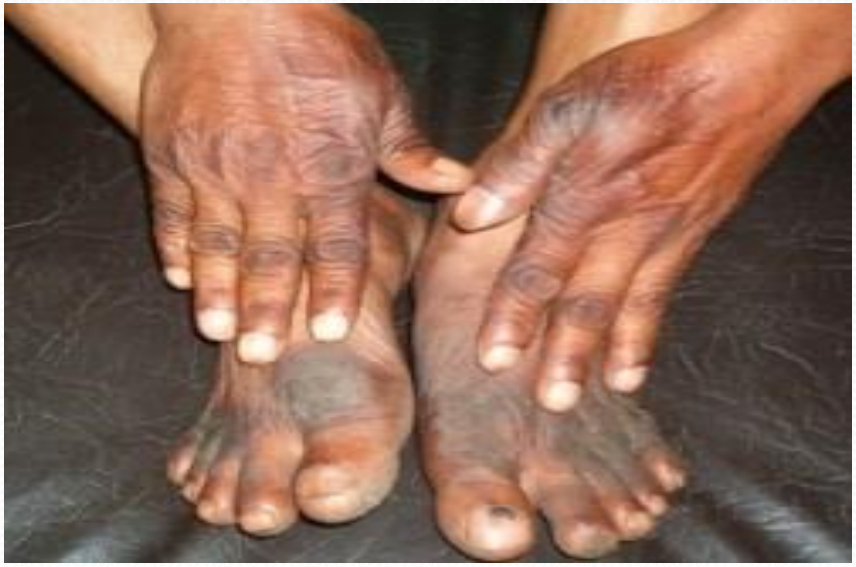
(over production of 5-OH tryptamine).

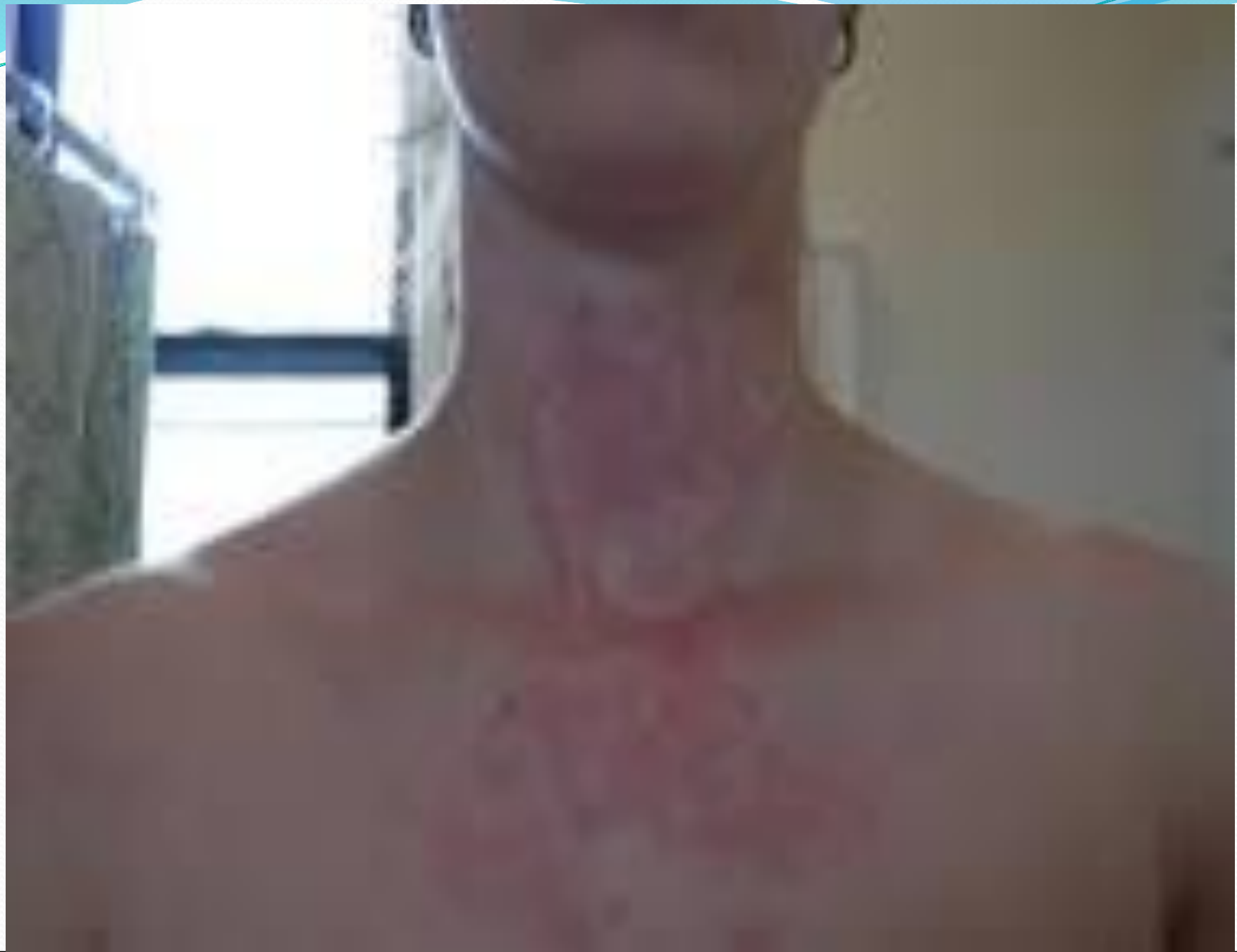
- Hartnup disease

Genetic defect in memberane transport mechanism(mal absorption).









Refrences

- Chatterjea
- Satyanarayana


Thank you!