

# B complex vitamins



- Include:
  - Thiamine
  - Riboflavin
  - Niacin
  - Pyridoxine
  - Pantothenic acid
  - Biotin
  - Folic acid &
  - Vitamin B<sub>12</sub>.



# All vitamins will be discussed under following headings

1. Chemistry
2. Sources
3. Co-enzyme forms
4. Metabolic role
5. RDA
6. Deficiency
  - a. causes diagnosis/treatment
  - b. C/F
  - c. biochemical basis of C/F
  - d.



# Thiamine☒

- 1. Chemistry:
- Other names : aneurine
  - : antineuritic factor
  - : anti beriberi factor
  - : Vitamin B<sub>1</sub>

(vitamin containing Sulphur)



## 2. Sources

- **Plant sources**

cereals - whole wheat flour, unpolished hand pound rice, parboiled rice.

### **Animal sources**

**liver, egg , meat**



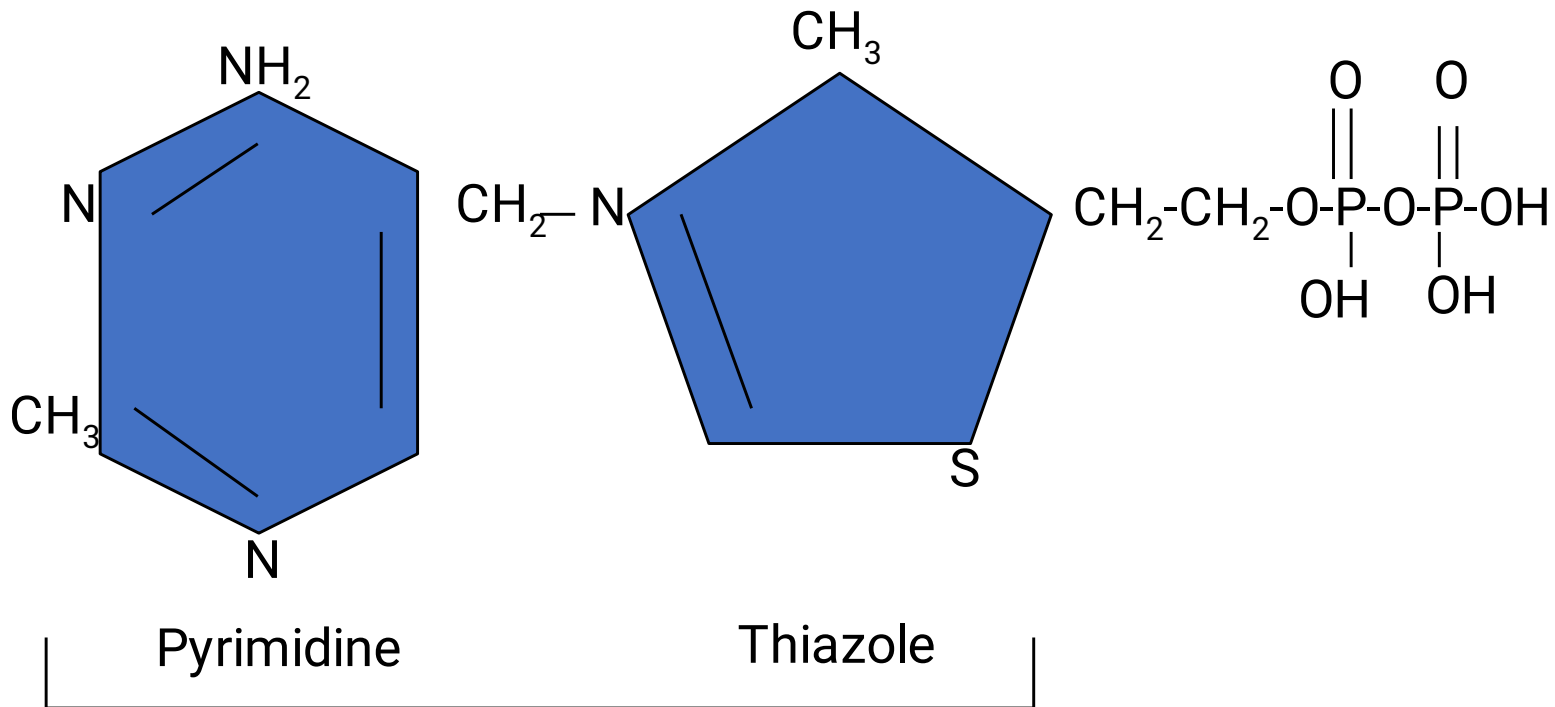
3. Coenzyme form: ☒☒

☒

Thiamine pyrophosphate (TPP) ☒



# 3. Coenzyme form of Thiamine

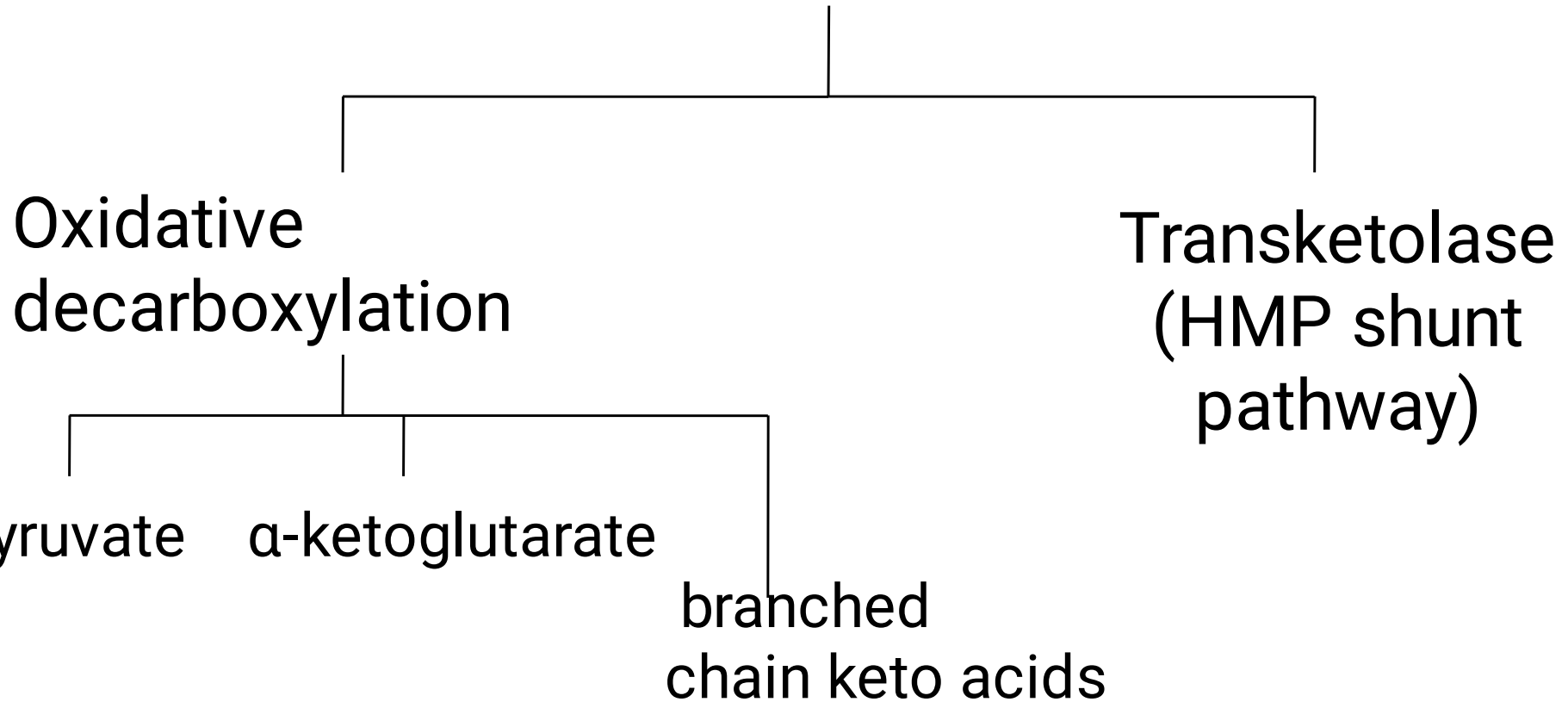


Thiamine  Edit with PDF Office

TPP

Pyrophosphate

## 4. Metabolic role



main role of TPP is in carbohydrate metabolism



# Recall: PYRUVATE DEHYDROGENASE

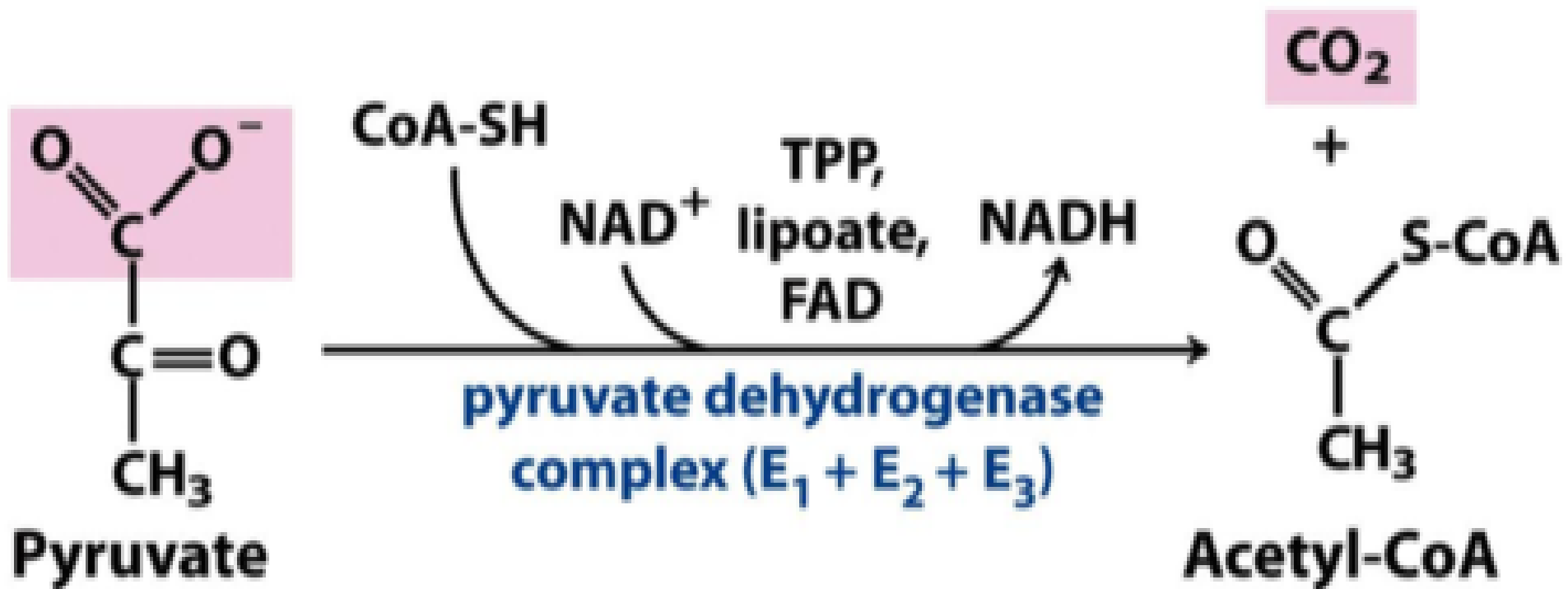
## 3 COMPONENTS :

- ✓ Pyruvate decarboxylase / dehydrogenase
- ✓ Dihydrolipoyl transacetylase
- ✓ Dihydro lipoyl dehydrogenase

## Co-factors / co-enzymes:

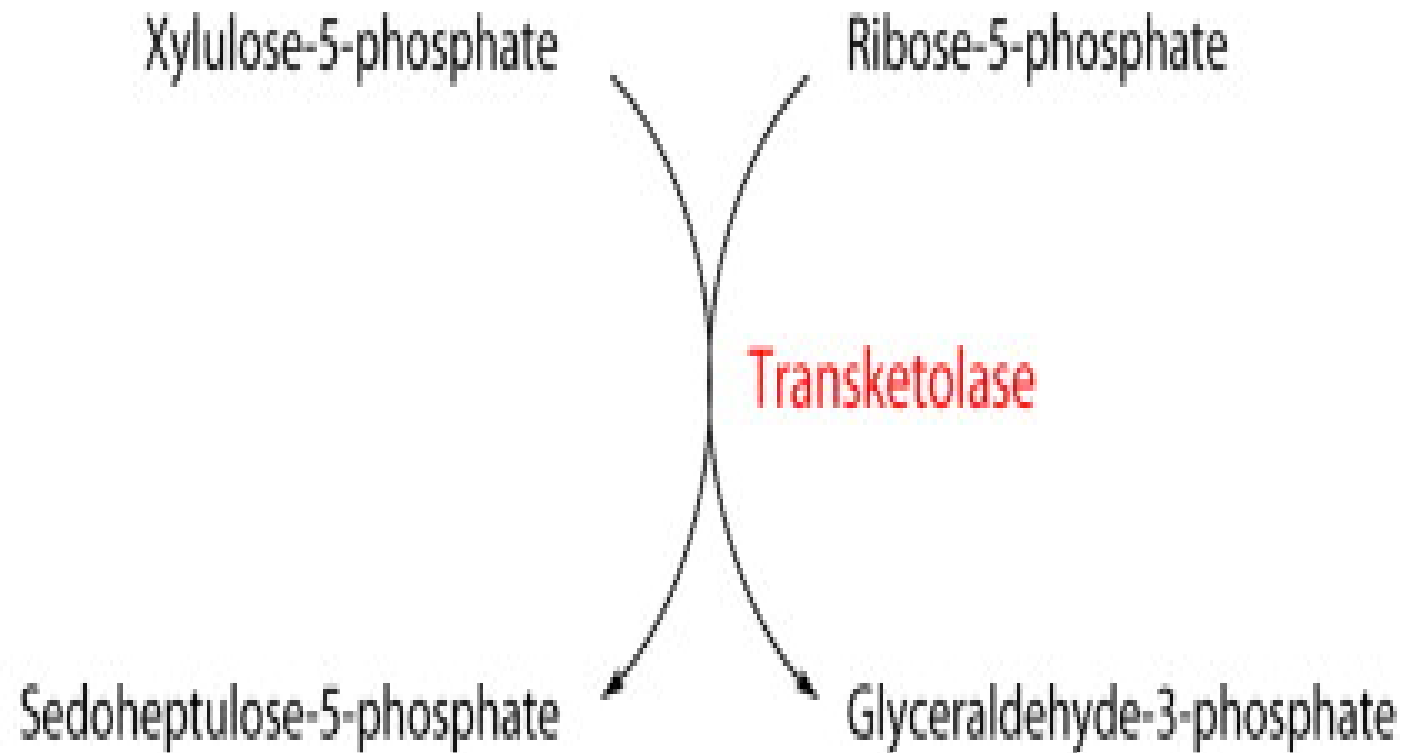
- ✓ Thiamine pyrophosphate
- ✓ lipoamide
- ✓ CoA
- ✓ FAD
- ✓ NAD





$\Delta G'^{\circ} = -33.4 \text{ kJ/mol}$

- **Transketolase**



- Role in **transmission of nerve impulse-** acetylcholine synthesis and ion translocation of neural tissue

- **5. RDA**

- **1-1.5mg/day**



# 6. Thiamine deficiency

## Beri beri:

Definition: It is a clinical condition arising from deficiency of vitamin B1/thiamine.

## Causes:

1. Dietary deficiency of thiamine: when polished rice is the staple food,
2. Alcoholism: absorption and storage of thiamine is low
3. Pregnancy and lactation increase the demand for thiamine
4. Prolonged diarrhoea can lead to thiamine deficiency
5. Dialysis can cause thiamine store depletion.

Biochemical changes: accumulation of pyruvate, alpha keto glutarate and branched chain keto acids



# Beriberi

Classification: mainly of two types:

wet beri beri and dry beri beri

- Wet beriberi – blood related – edema , heart failure etc
- Dry – nerve related - peripheral neuritis – sensation in limbs less etc



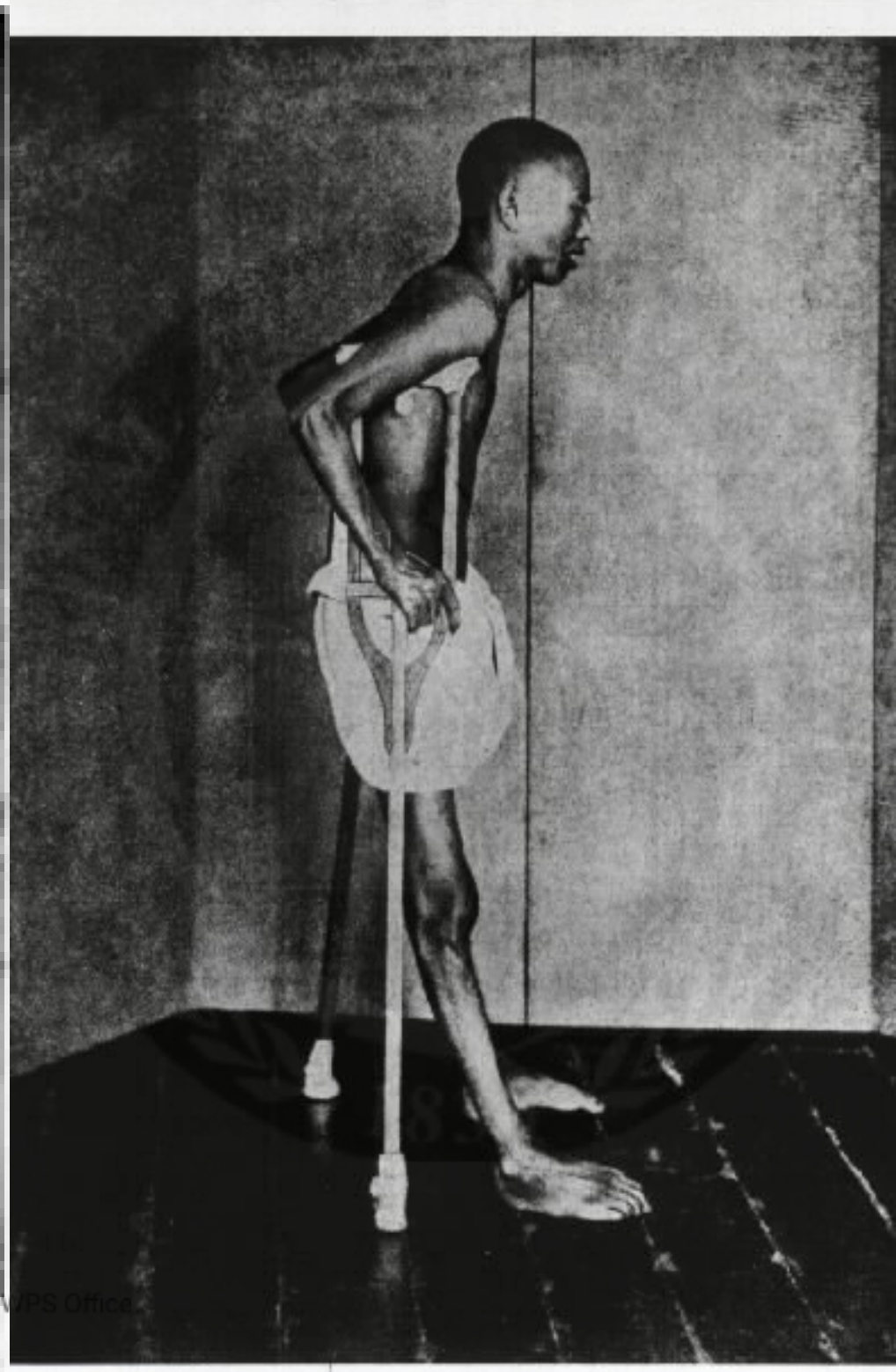
# Wet beriberi

- Cardiovascular manifestations
- Edema of legs, face & trunk
- Calf muscle swelling
- palpitation
- Breathlessness
- tachycardia
- Heart failure
- ↑ systolic BP, ↓ diastolic BP.

# Dry beriberi

- CNS manifestations
- Muscle wasting
- Peripheral neuropathy
- No edema





- **C. Biochemical basis of C/F:**
- glucose metabolism impaired
  - affects CNS
  - In wet beri-beri – peripheral vasodilation – high cardiac output, retention of Na & water.



# Infantile beriberi

- Seen in infants born to mothers suffering from thiamine deficiency
- C/F – restlessness, sleeplessness, vomiting, convulsions
- cardiac failure leads to death



# Wernicke-Korsakoff syndrome

- in chronic alcoholics
- Alcohol
  - inhibits intestinal absorption of thiamine
  - Interferes with TPP synthesis



# Wernicke-Korsakoff syndrome

- Clinical features

- Ophthalmoplegia – no eye movement due to paralysis of extraocular muscles
- Nystagmus- to & fro involuntary movement of eyeball
- Cerebellar ataxia- drunken people like gait
- Psychosis – psychiatric disorders



## Diagnosis

- blood thiamine level is low
- Erythrocyte transketolase activity is low
- Urinary thiamine excretion is low
- High levels of pyruvate and lactate in blood



Thiamine deficiency can also be caused by

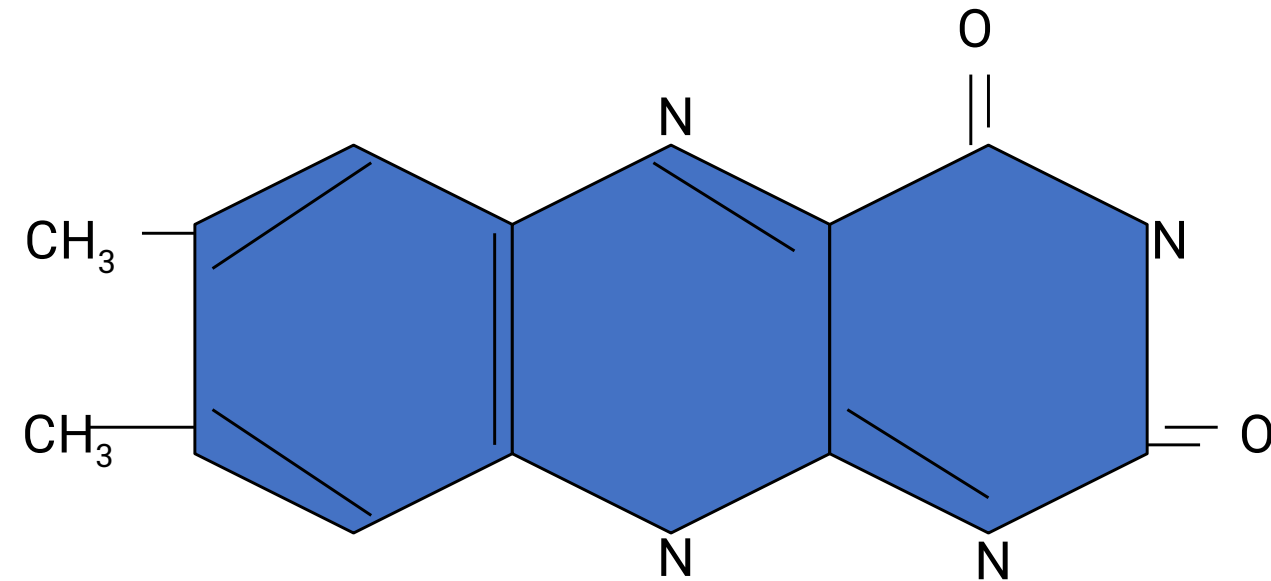
- Certain seafoods( fishes) –Thiaminase present in some fishes can lead to breakdown of thiamine
- Antimetabolites: interfere with thiamine activity:

Pyriithiamine

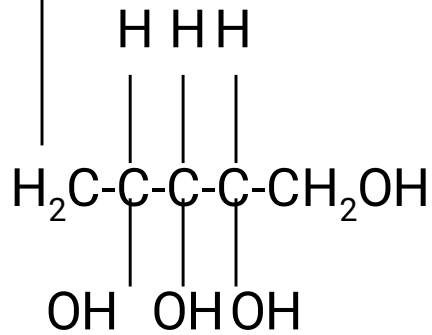
Oxythiamine



# Riboflavin



ISO-ALLOXAZINE



D-RIBITOL



## 2. sources

- Whole cereals, green leafy vegetables
- Whole Milk
- Germinating seeds
- Dried yeasts
- Cheese, yoghurt
- Liver

Light sensitive vitamin, easily destroyed by UV light and radiation



### 3. Coenzyme forms

- FMN – Flavin mononucleotide
- FAD – Flavin adenine dinucleotide

**Enzymes that require FMN/FAD as coenzyme are called Flavoproteins,**

**in addition if they have metal as cofactors - metalloflavoproteins**



## 4. Metabolic role ☒

FMN & FAD dependent enzymes ☒  
(take part in oxdn-redn reactions)

### FMN

- L- amino acid oxidase
- NADH dehydrogenase

### FAD

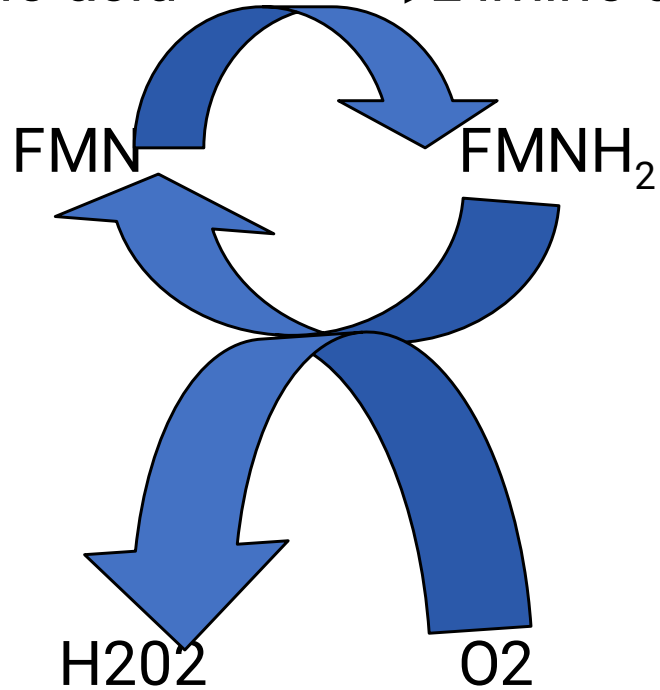
- Succinate dehydrogenase
- Acyl CoA dehydrogenase
- Xanthine oxidase
- PDH
- $\alpha$ -ketoglutarate dehydrogenase



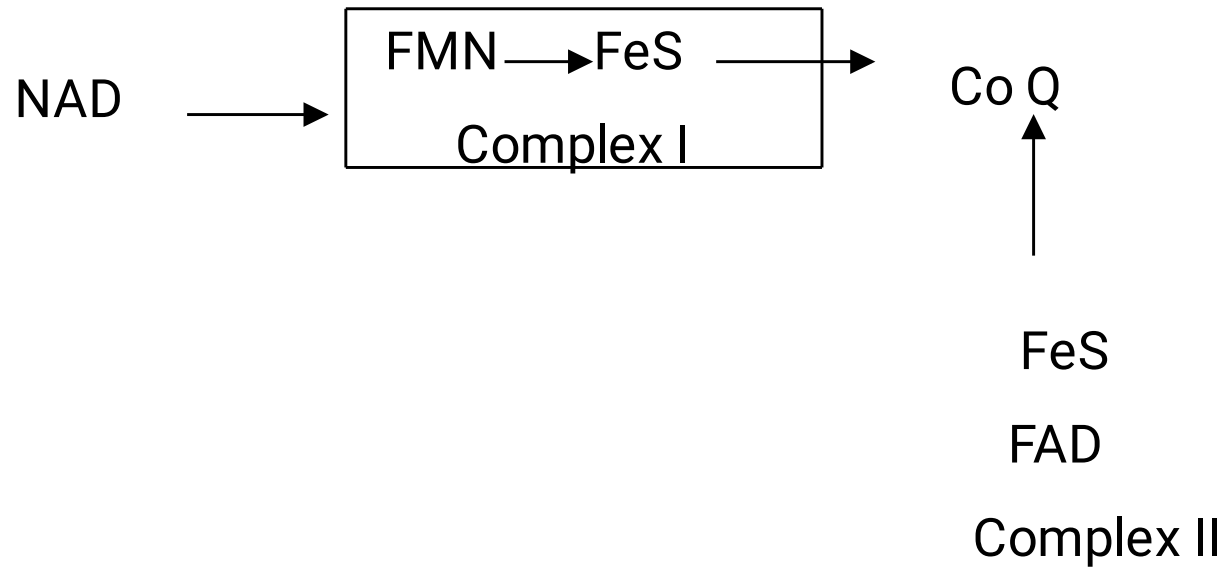
# FMN dependent enzymes

## 1. L amino acid oxidase

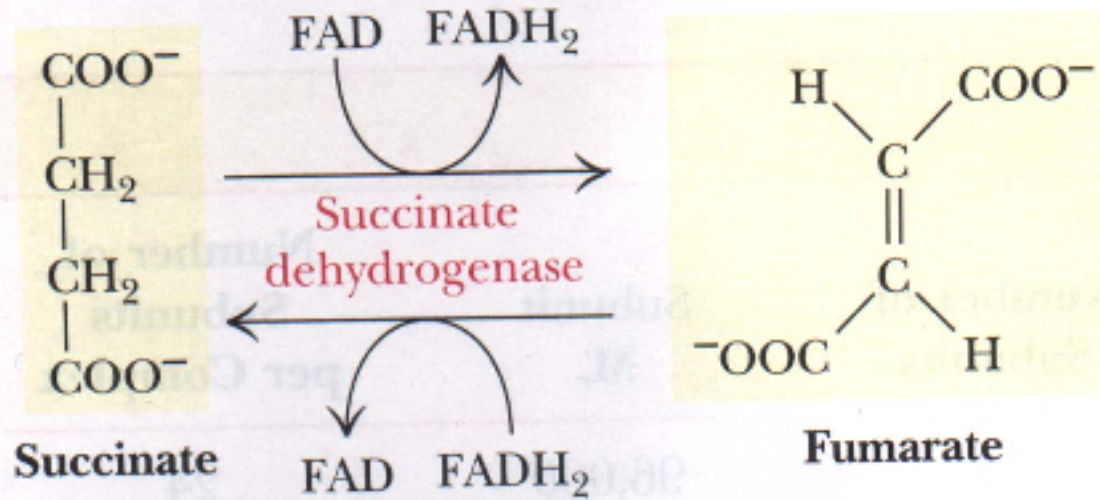
• L amino acid  $\xrightarrow{\quad}$  L imino acid  $\xrightarrow{\quad}$  ketoacid



- In electron transport chain: NADH dehydrogenase requires FMN



# FAD dependent enzymes



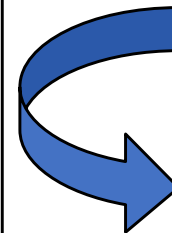
Pyruvate dehydrogenase complex  
 $\alpha$ -KG dehydrogenase complex



Acyl CoA

Acyl CoA dehydrogenase

FAD



FADH<sub>2</sub>

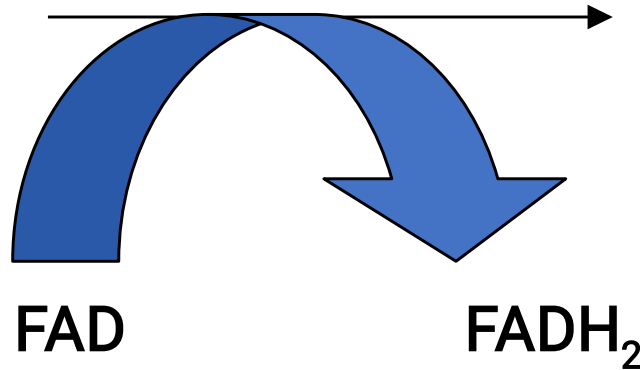
$\alpha\beta$  unsaturated acyl CoA



• *Xanthine oxidase*

• Xanthine

Uric acid



• Erythrocyte glutathione reductase

• Dihydrolipoate dehydrogenase



## 6. Riboflavin deficiency

a. Causes: along with other deficiency disorders like beriberi, pellagra & Kwashiorkor



- **b. C/F**
- **Glossitis**
  - Magenta colored tongue
- **Cheilosis**
  - Redness and shiny lips
- **Angular stomatitis**
  - Lesions at the angle of mouth
- **Genital dermatitis**





Glossitis of Riboflavin Deficiency





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# NIACIN



# 1. Chemistry

- Niacin =Nicotinic acid
- Nicotinamide
- Pellagra preventing factor

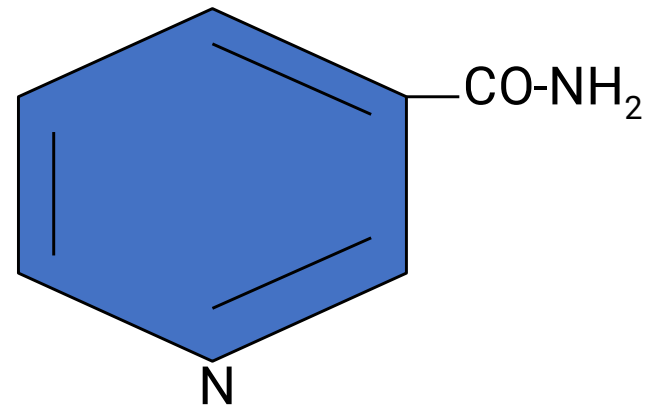
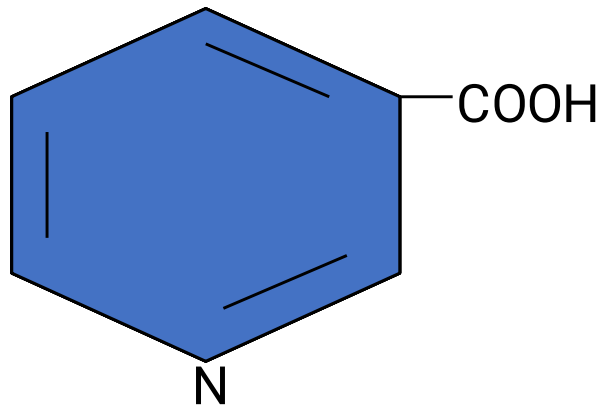


# Biologically active form

- Nicotinamide is present largely as dinucleotide.
- **NAD<sup>+</sup>**
- **NADP<sup>+</sup>** : It contains an additional phosphoric acid.



- Niacin term used to represent nicotinic acid & nicotinamide



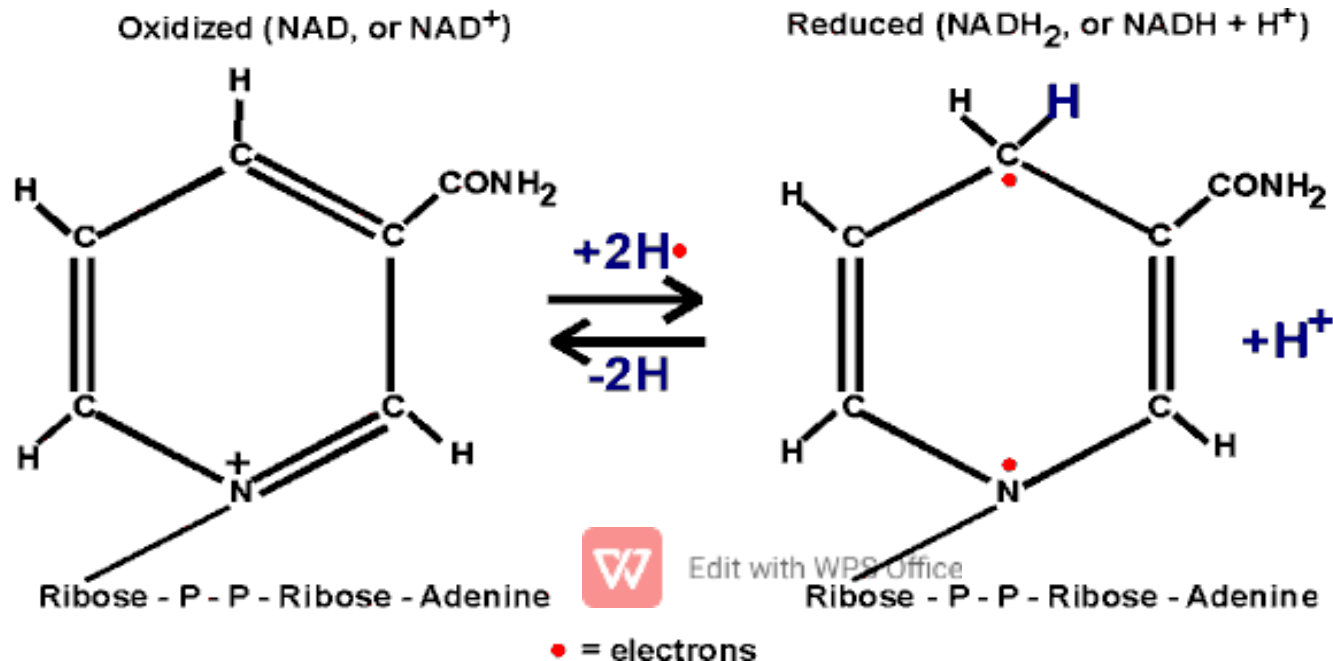
## 2. Dietary sources

- Dried yeast, legumes
- Liver, meat, fish
- About 50% requirement is met by conversion of Tryptophan to niacin.  
60mg of Trp gives 1mg of niacin (niacin equivalent)



# Biochemical functions

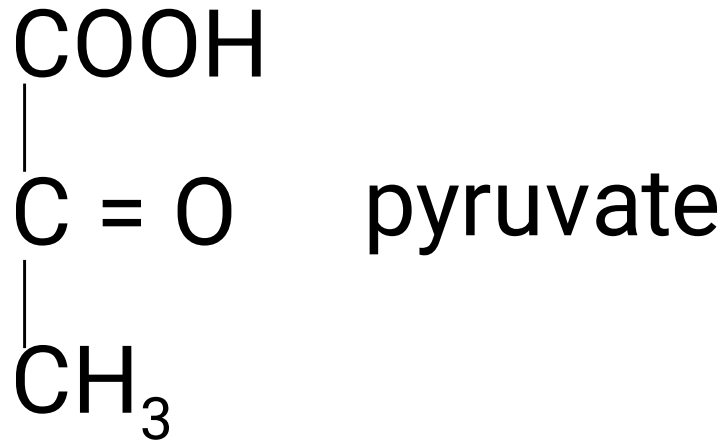
- The coenzymes  $\text{NAD}^+$  and  $\text{NADP}^+$  are involved in oxidation reduction reactions.
- Enzymes belonging to class oxido-reductases are dependent on them.



# NAD<sup>+</sup> dependent enzymes

- Lactate dehydrogenase
- Glyceraldehyde-3-P- dehydrogenase
- Malate dehydrogenase
- Pyruvate dehydrogenase
- $\alpha$ -keto glutarate dehydrogenase
- Iso citrate dehydrogenase (mitochondrial)

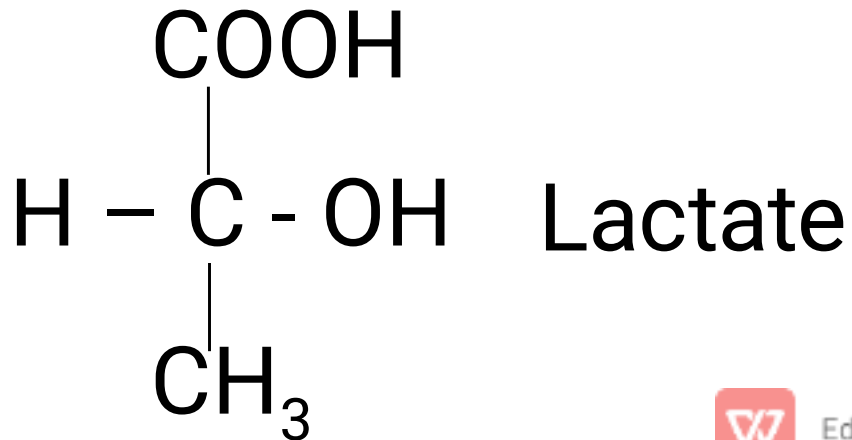




**LDH**

NADH+H<sup>+</sup>

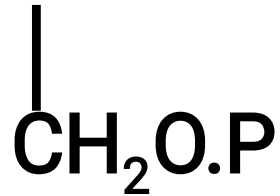
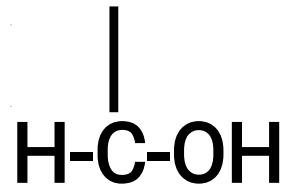
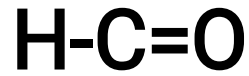
NAD<sup>+</sup>



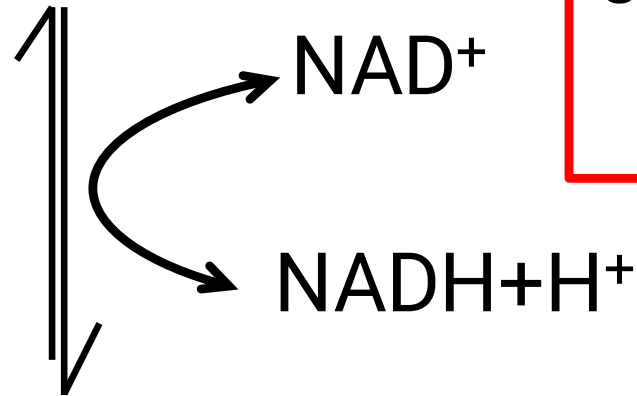
pyruvate

**PDH**

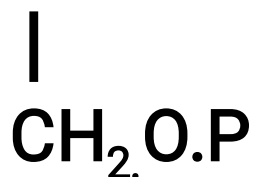
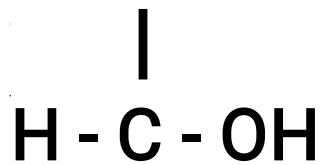
Acetyl CoA



Glyceraldehyde-3-phosphate

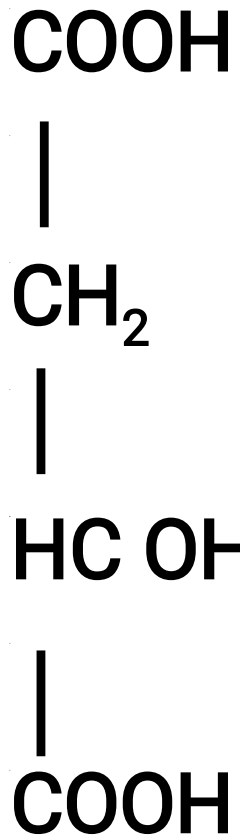


GLYCERALDEHYDE-3- PHOSPHATE  
DEHYDROGENASE

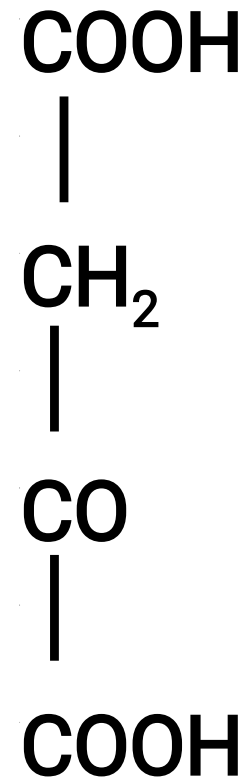
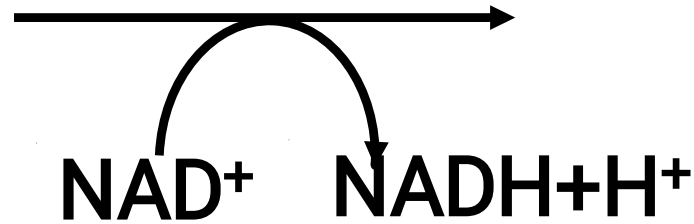


1, 3 bisphosphoglycerate



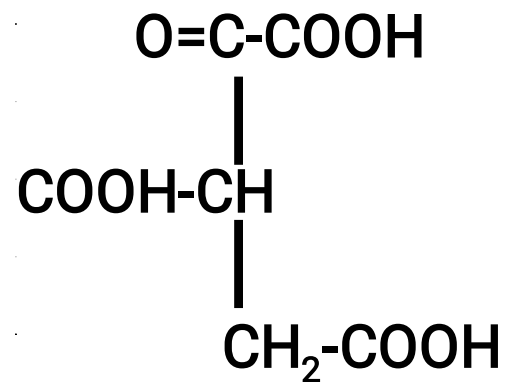
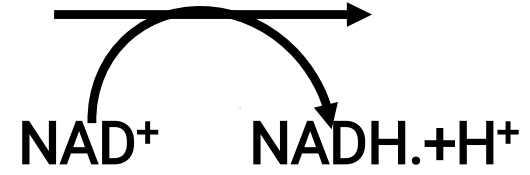
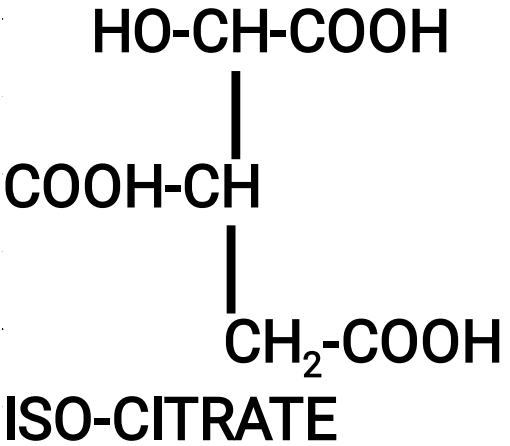


MALATE

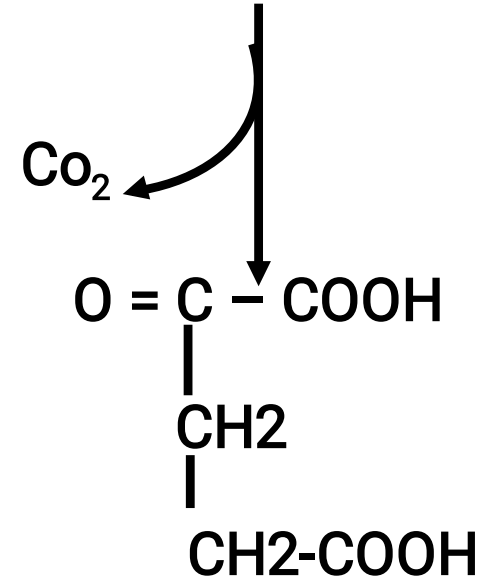


OXALO - ACETATE

**ENZYME: MALATE DEHYDROGENASE**

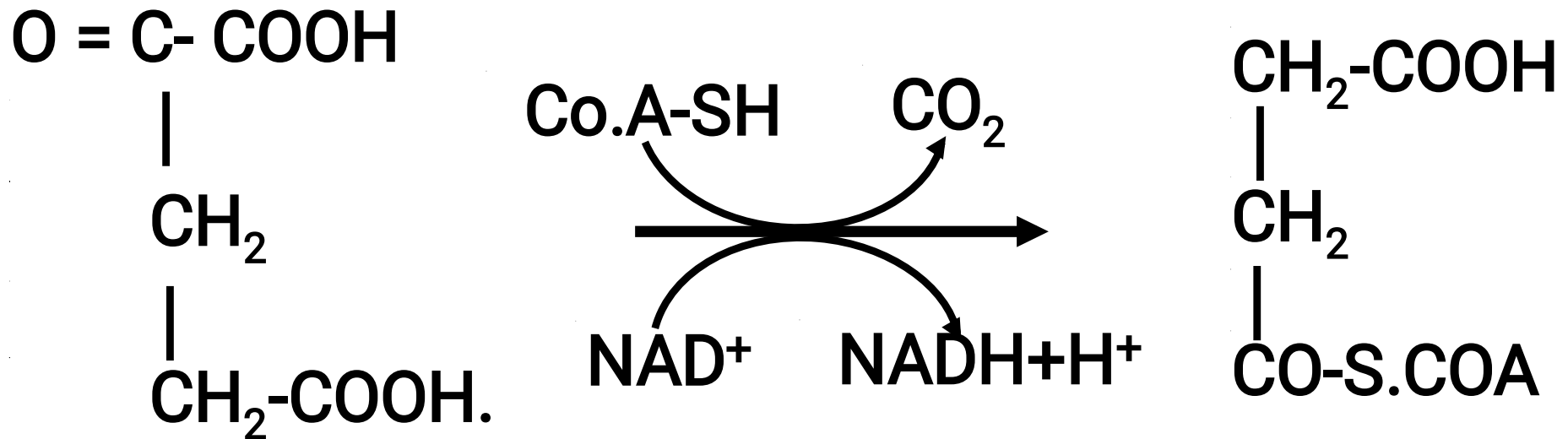


OXALO - SUCCINATE



ALPHA KETO - GLUTARATE

**ENZYME: ISOCITRATE DEHYDROGENASE**



ALPHA KETO - GLUTARATE

SUCCINYL CoA.

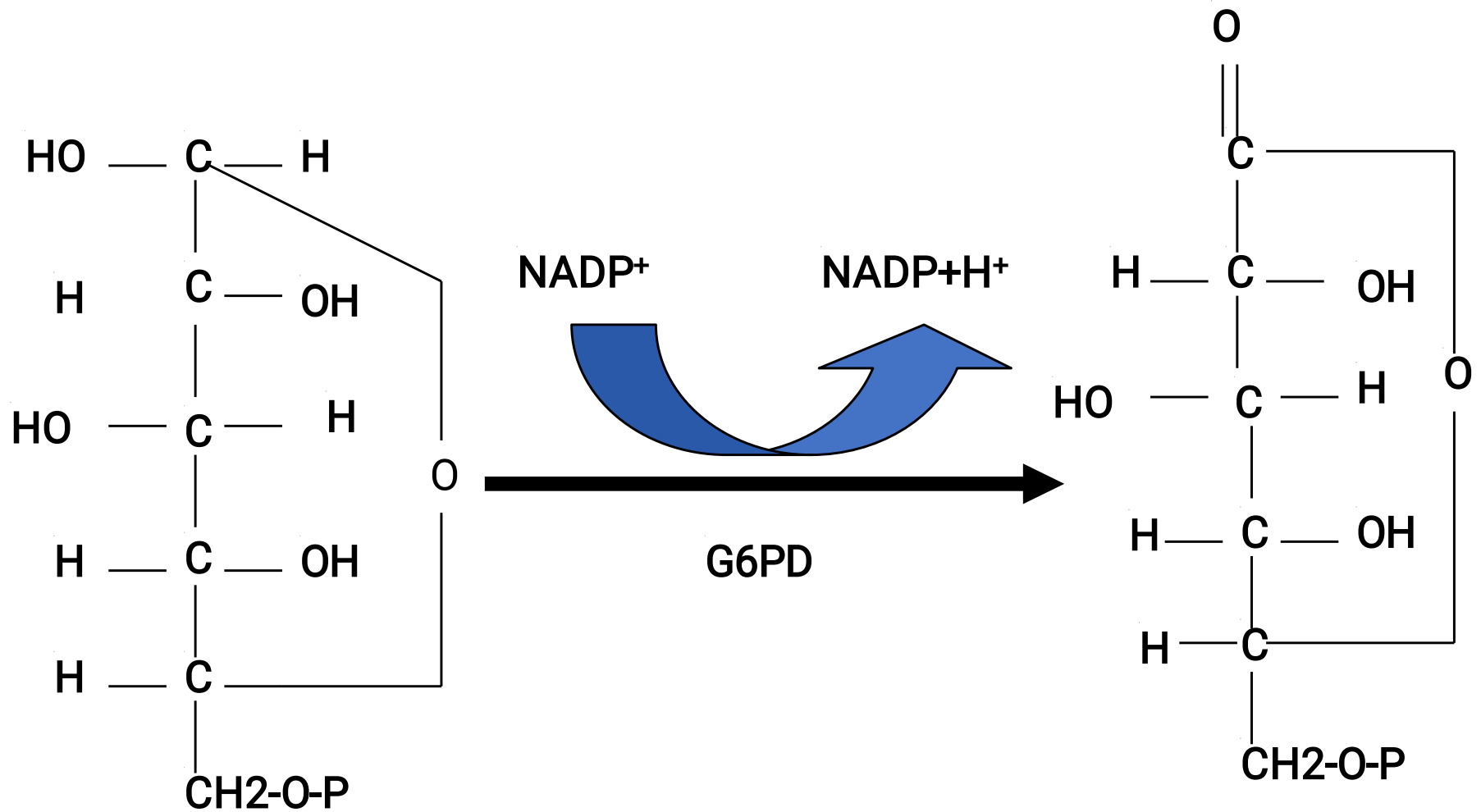
**ENZYME : ALPHA KETO – GLUTARATE  
DEHYDROGENASE**



# NADPH<sup>+</sup> generating reactions

- Glucose –6-phosphate dehydrogenase
- 6 phosphogluconate dehydrogenase
- Malic enzyme
- Cytoplasmic isocitrate dehydrogenase





Glucose 6 P

6 phosphogluconolactone

# NADPH utilizing reactions

- **Folate reductase**

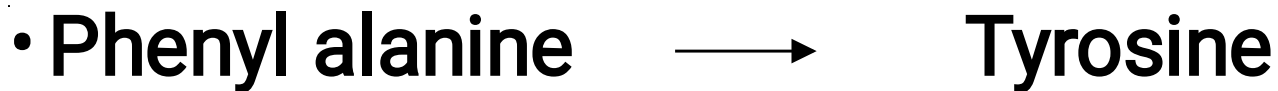


- **Methemoglobin reductase**



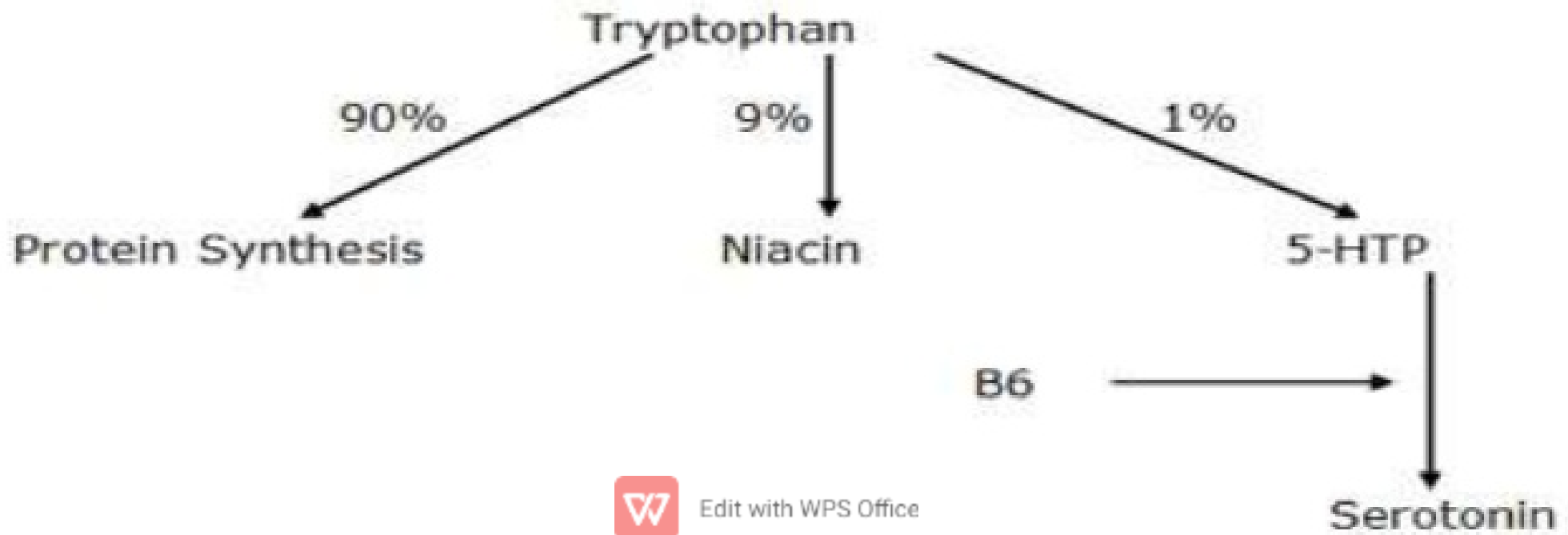
- **Phenylalanine hydroxylase**

- **HMGCoA reductase**



# DEFINITION

- Cellular deficiency of niacin, resulting from an inadequate dietary supply of **Niacin** (Vit B<sub>3</sub> / Nicotinic Acid) and tryptophan, or high Leucine, is termed Pellagra



## 6. Niacin Deficiency - Pellagra

### a. Causes:

#### 1. dietary deficiency of Trp

- When maize is staple diet – deficient in Trptophan, niacin present in bound form, so unavailable.
- When sorghum is staple diet – leucine rich, leucine inhibits QPRTase,  $\text{NAD}^+$  not synthesized





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## 2. Deficient synthesis :

In pyridoxal deficiency,

Tryptophan  $\xrightarrow{\text{X}}$  NAD<sup>+</sup>

(Kynureninase is PLP dependent enzyme)



3. Drugs - Some drugs inhibit vitamin B6  
can cause pellagra

Examples: INH, Oral contraceptives, alcohol,  
phenytoin



#### 4. Hartnup disease:

Congenital disease

Trp absorption from intestine & reabsorption from renal tubules defective – lack of Trp – nicotinamide deficiency

#### 5. Carcinoid syndrome:

Major portion of Trp diverted towards serotonin, so Trp unavailable

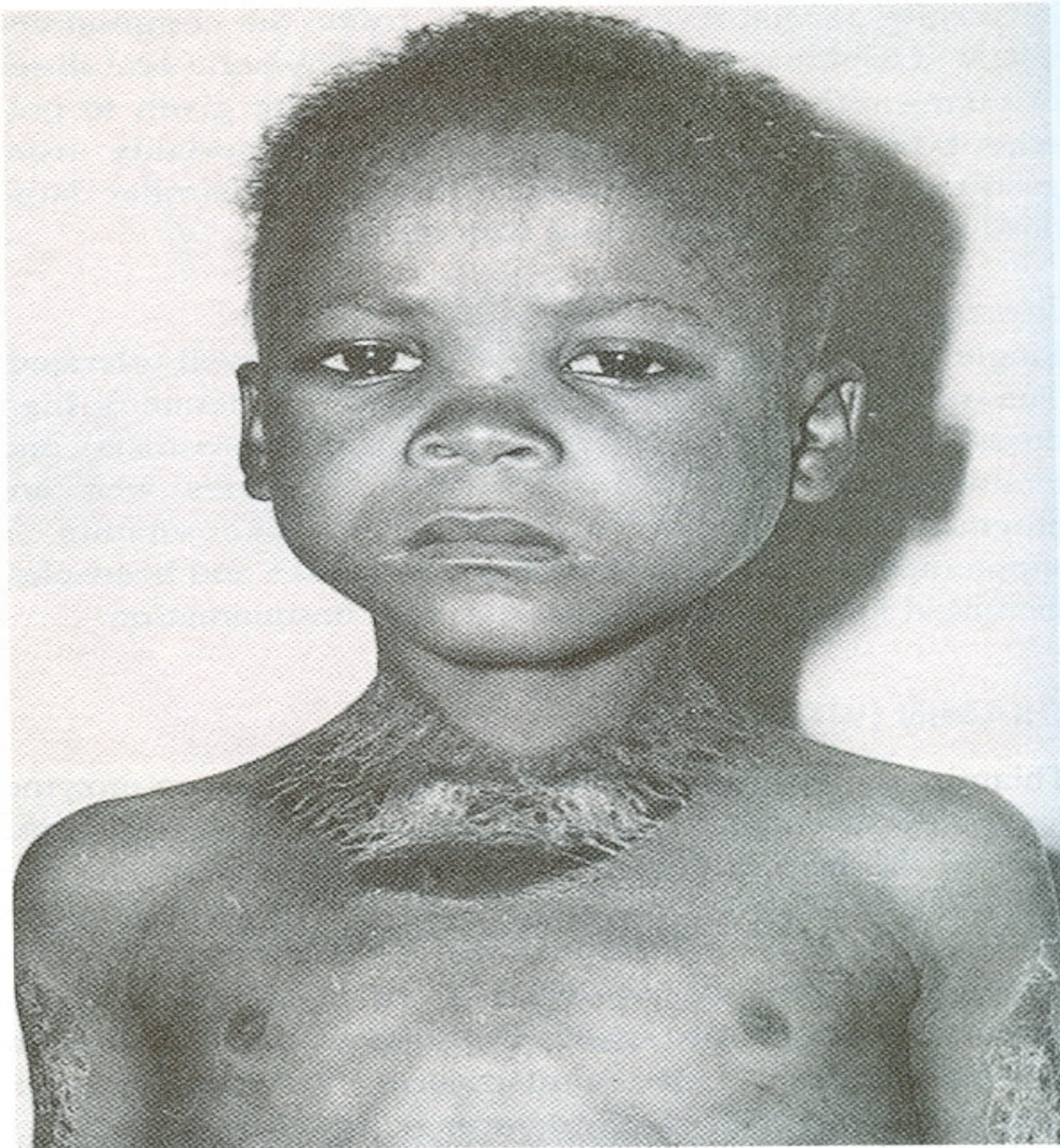
#### 6. Niacin Deficiency



# Clinical features ☒

- **Dermatitis** – bright red erythema in the feet, ankles and face.
- pigmented skin rash around the neck – **Casal's necklace**
- **Diarrhea** – with blood & mucus, inflammation of whole GIT
- **Dementia** - depression, delirium & loss of memory, if left untreated
- **Death**





**Fig. 7.27 Pellagra in a girl of 5 years.** This skin lesion on the neck (Casal's collar) is pathognomonic of niacin deficiency.



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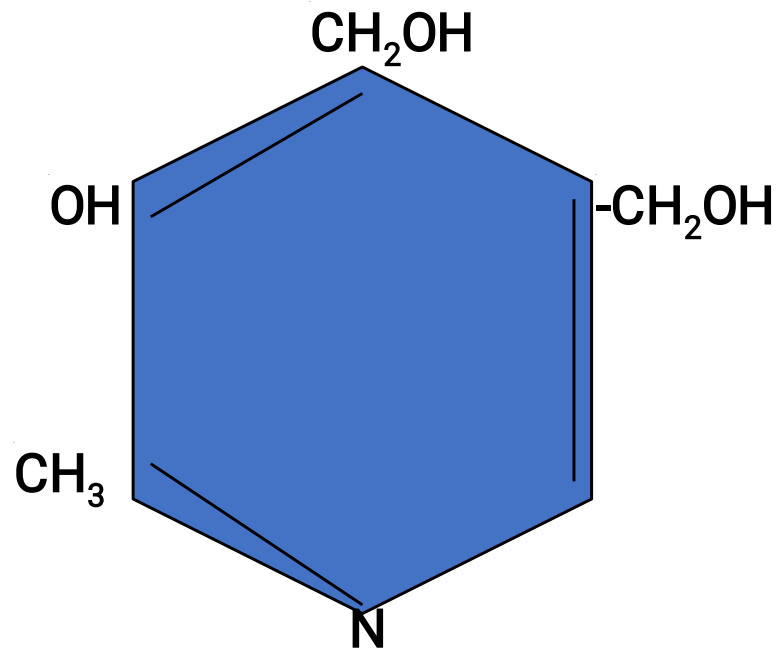
# Therapeutic use of niacin

- treatment of hyperlipidaemias
- ↓ circulating FFA inhibiting HSL
- ↓ Lp(a) levels
- ↑ HDL



# Pyridoxine (Vit-B<sub>6</sub>)

- 3 forms - pyridoxine, pyridoxal & pyridoxamine

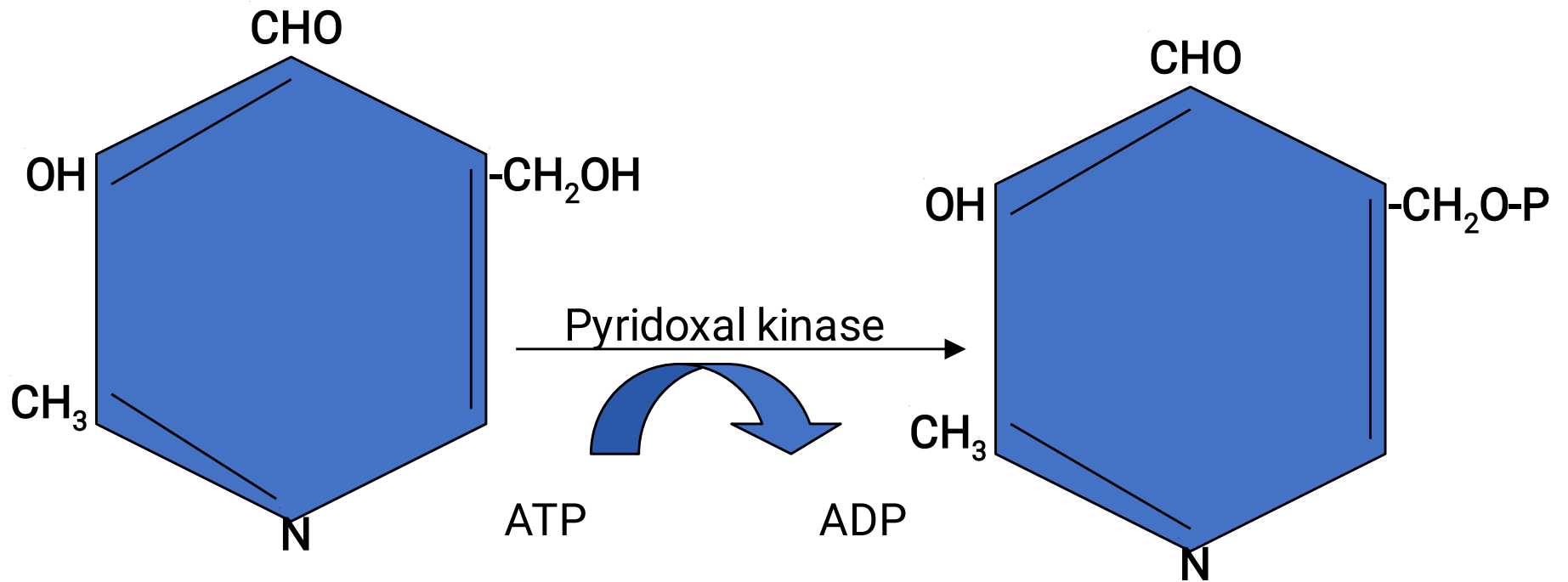


## 2. Dietary sources

- Dried yeast, rice polishing, green leafy vegetables, egg, milk, meat, banana.

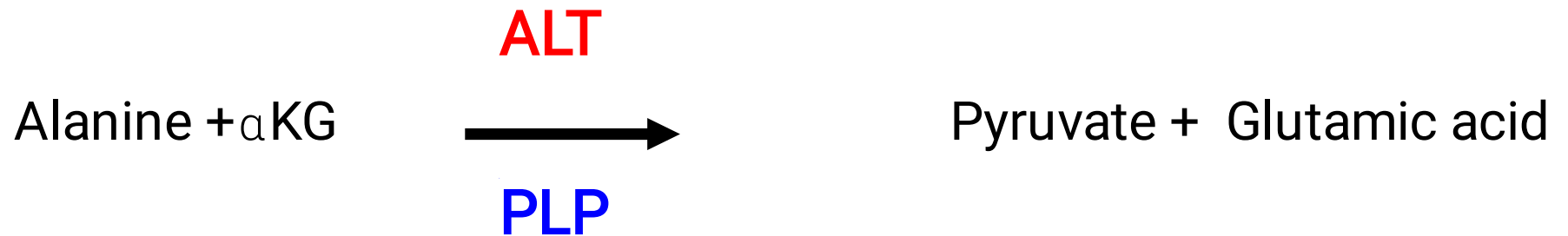


# Pyridoxal phosphate



# Metabolic role

- Transamination

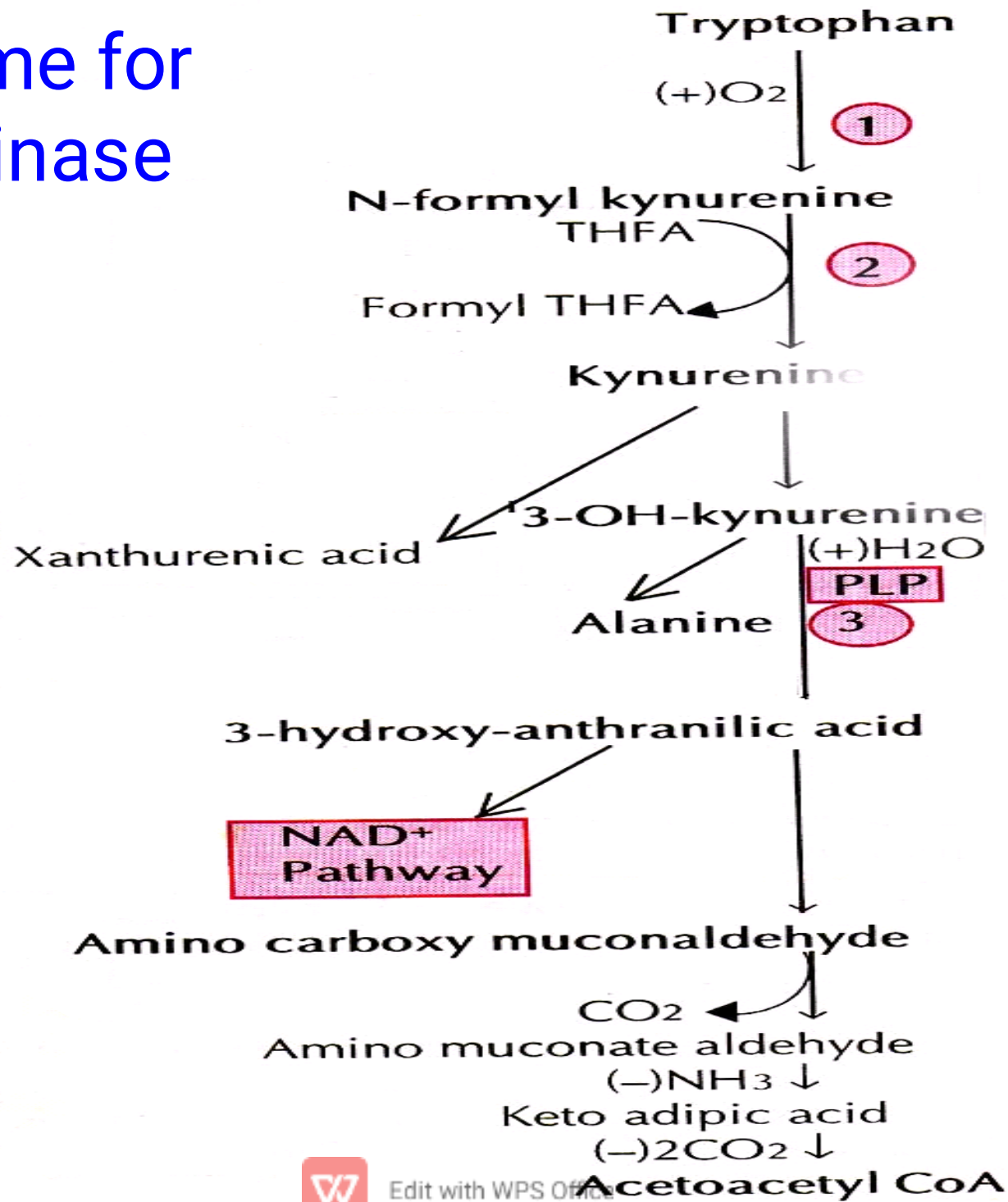


# Decarboxylation

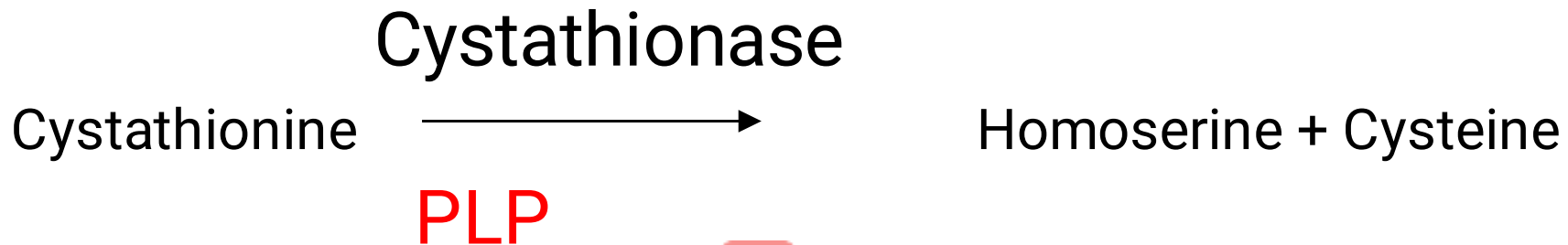
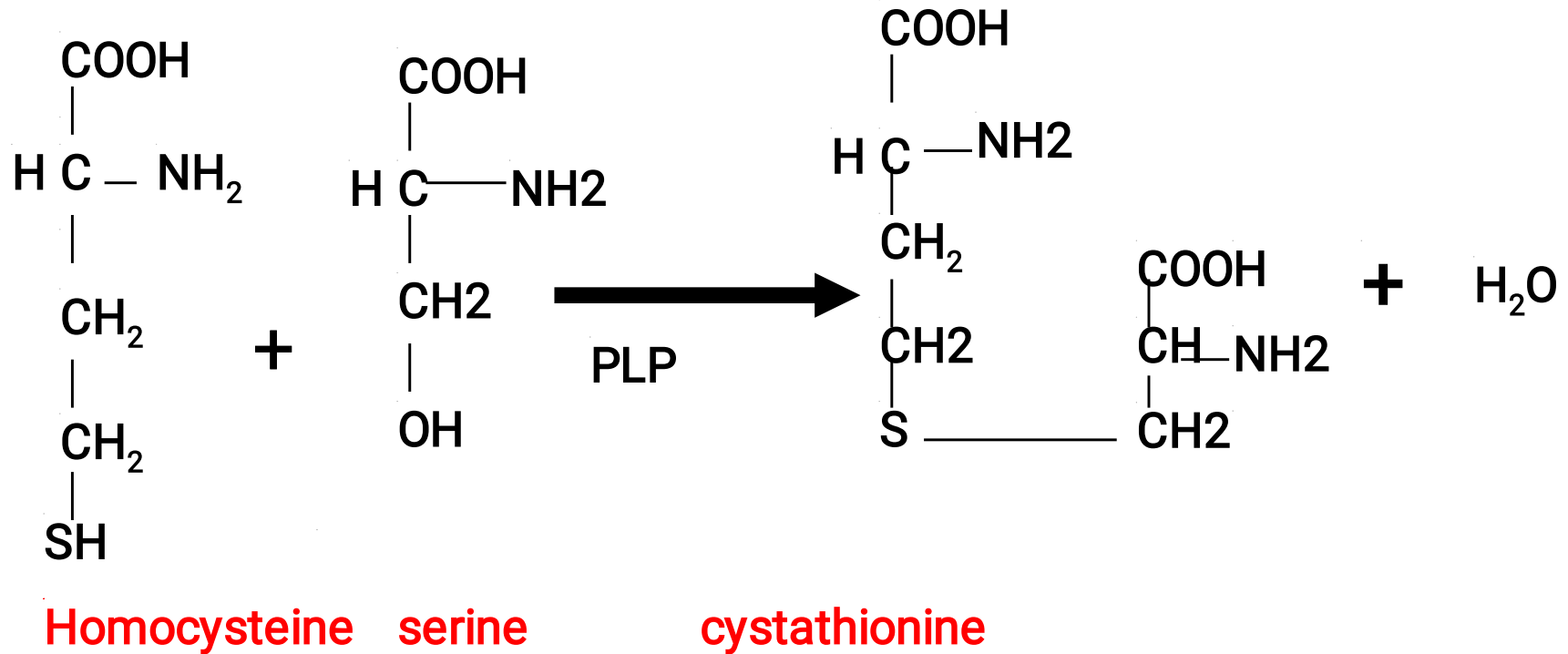
- Tyrosine  $\longrightarrow$  Tyramine
- Histidine  $\longrightarrow$  Histamine
- Glutamic acid  $\longrightarrow$  GABA
- 5-hydroxy tryptophan  $\longrightarrow$  serotonin



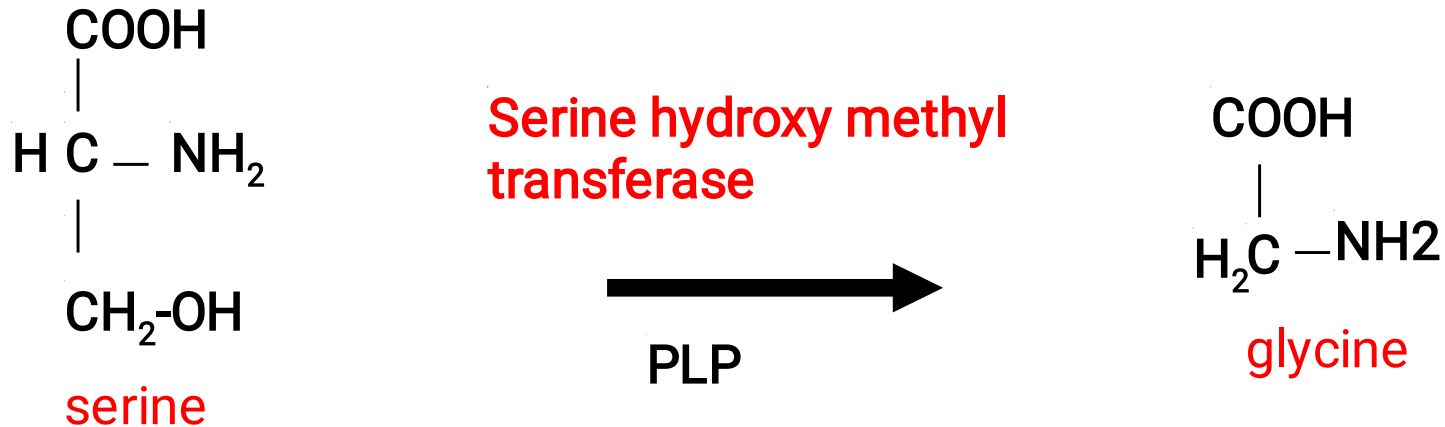
- Coenzyme for kynureninase



# Metabolism of sulfur containing amino acids



# Inter conversion of glycine and serine

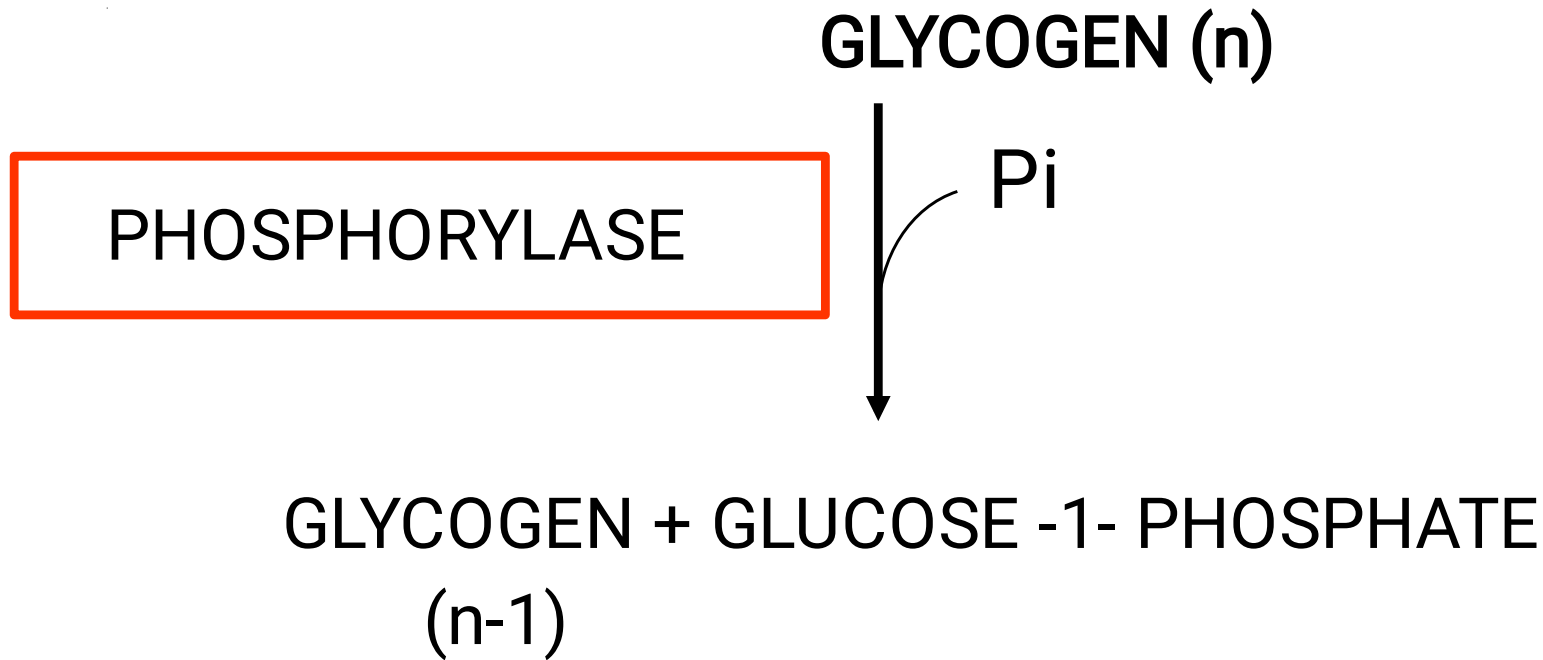


## Deamination

- Serine  $\longrightarrow$  pyruvate
- Threonine  $\longrightarrow$   $\alpha$ Keto butyric acid



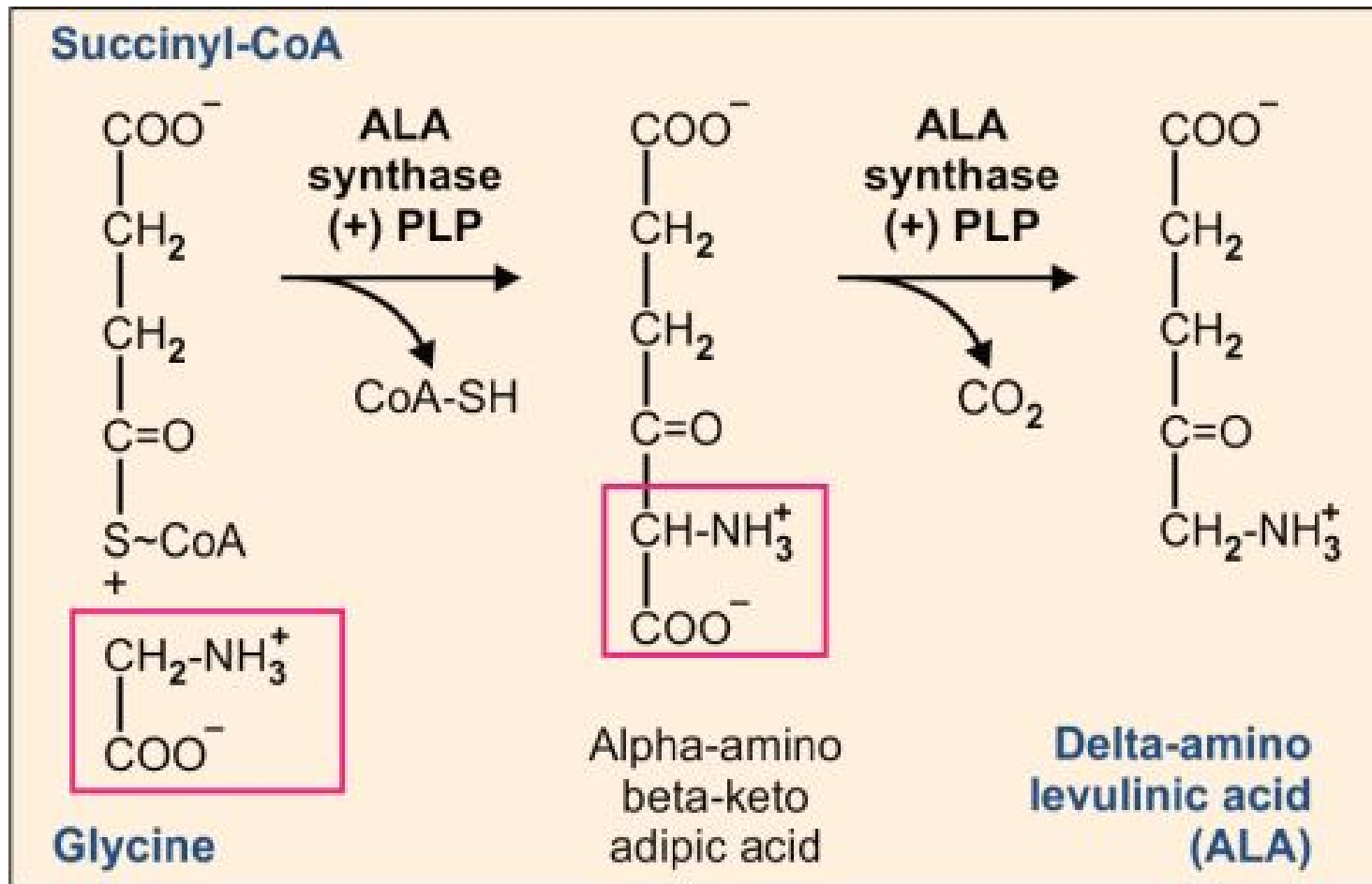
# Glycogenolysis



PHOSPHORYLASE ENZYME PRESENT IN  
- LIVER AND MUSCLE



# Heme synthesis



# Pyridoxine Function

- Transamination
- Deamination
- Decarboxylation
- Tryptophan to niacin
- Transfer of sulfur groups
- Hemoglobin formation
- Antibody production



# Deficiency

a. **Causes** : drugs like

- **INH**: antituberculosis drug, inhibits pyridoxal kinase, reduced PLP formation.
- **Oral contraceptives** : interfere with B6 metabolism
- **Cycloserine**: B6 antagonist
- **Alcohol** – acetaldehyde competes with PLP for binding to enzyme



## b. C/F & their biochemical basis

1. **Neurological manifestations:** irritability, peripheral neuropathy, confusion & depression
  - due to deficiency GABA, Serotonin, catecholamines
  - GABA deficiency – convulsions
  - Sphingolipids deficiency – demyelination of nerves & peripheral neuropathy



**2. Dermatological:** seborrheic dermatitis - due to pellagra – niacin deficiency.

**3. Hematological:** microcytic hypochromic anemia.

- due to decreased heme biosynthesis

Hyperhomocysteinemia - ↑ risk of CVD

In neonates - seizures

•



## Vitamin B<sub>6</sub> assay

- Vitamin B<sub>6</sub> status is assayed by urinary excretion of xanthurenic acid



# Pantothenic acid



# 1. Chemistry

- **Has pantoic acid &  $\beta$  -alanine**



## 2. Sources

- **Cereals, legumes, yeasts, liver, egg yolk.**
- **Synthesized by normal gut bacteria**



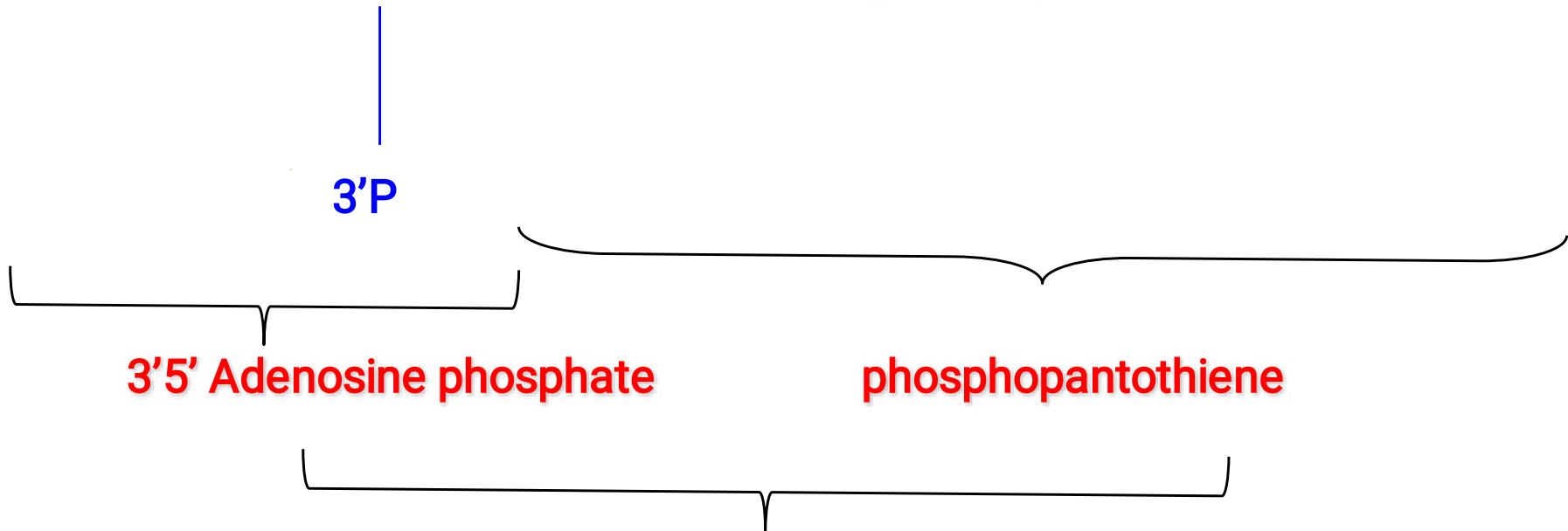
### 3. Coenzyme form

- Part of Coenzyme A
- $\beta$  mercaptoethanolamine
- SH group active site - written as CoA-SH



# Coenzyme A

Adenine-ribose-5'P-P-Pantothenic acid- $\beta$  mercaptoethanolamine



Coenzyme A

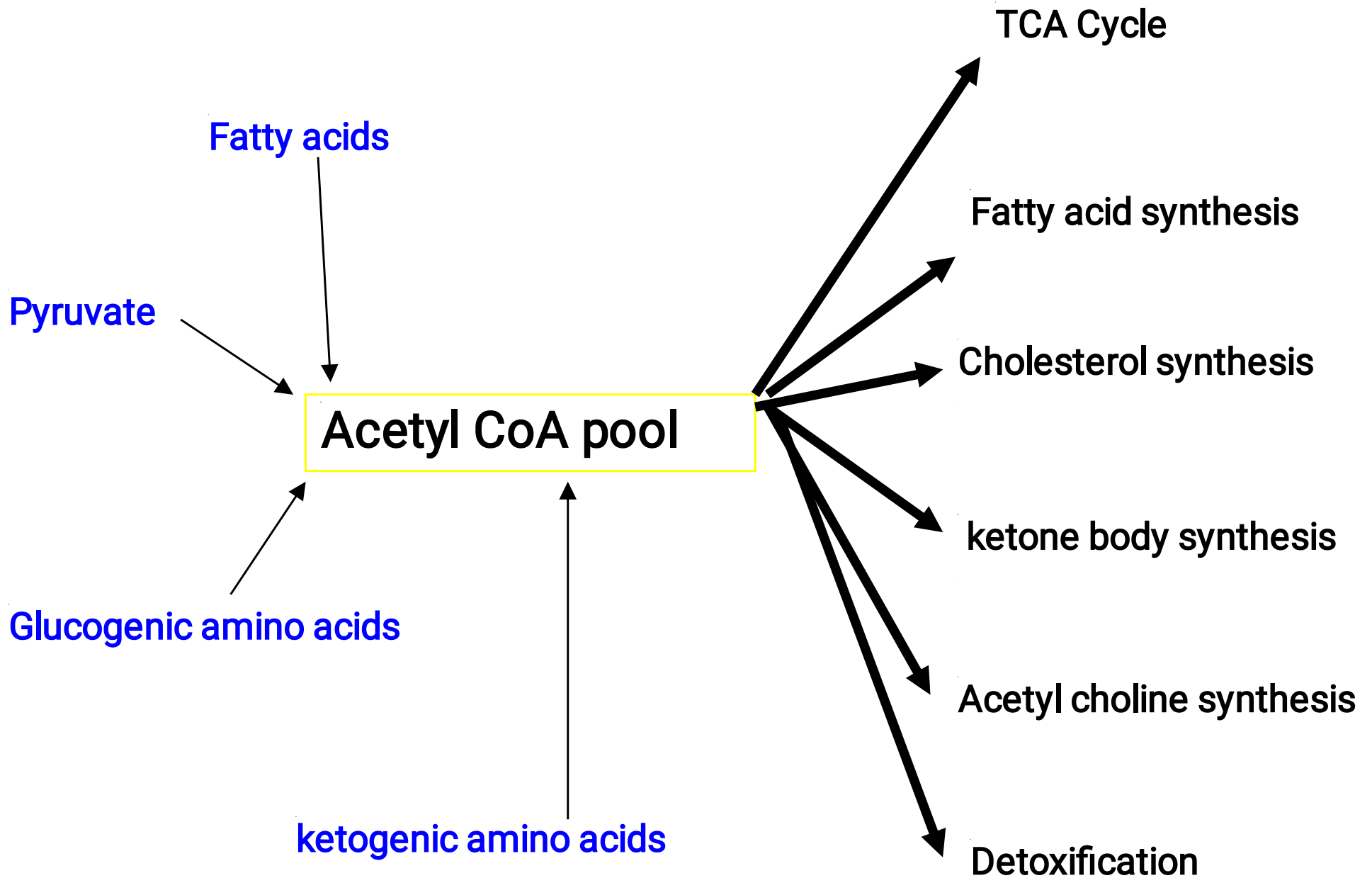
Pantothenic acid and  $\beta$  mercaptoethanol amine are parts of Co A.

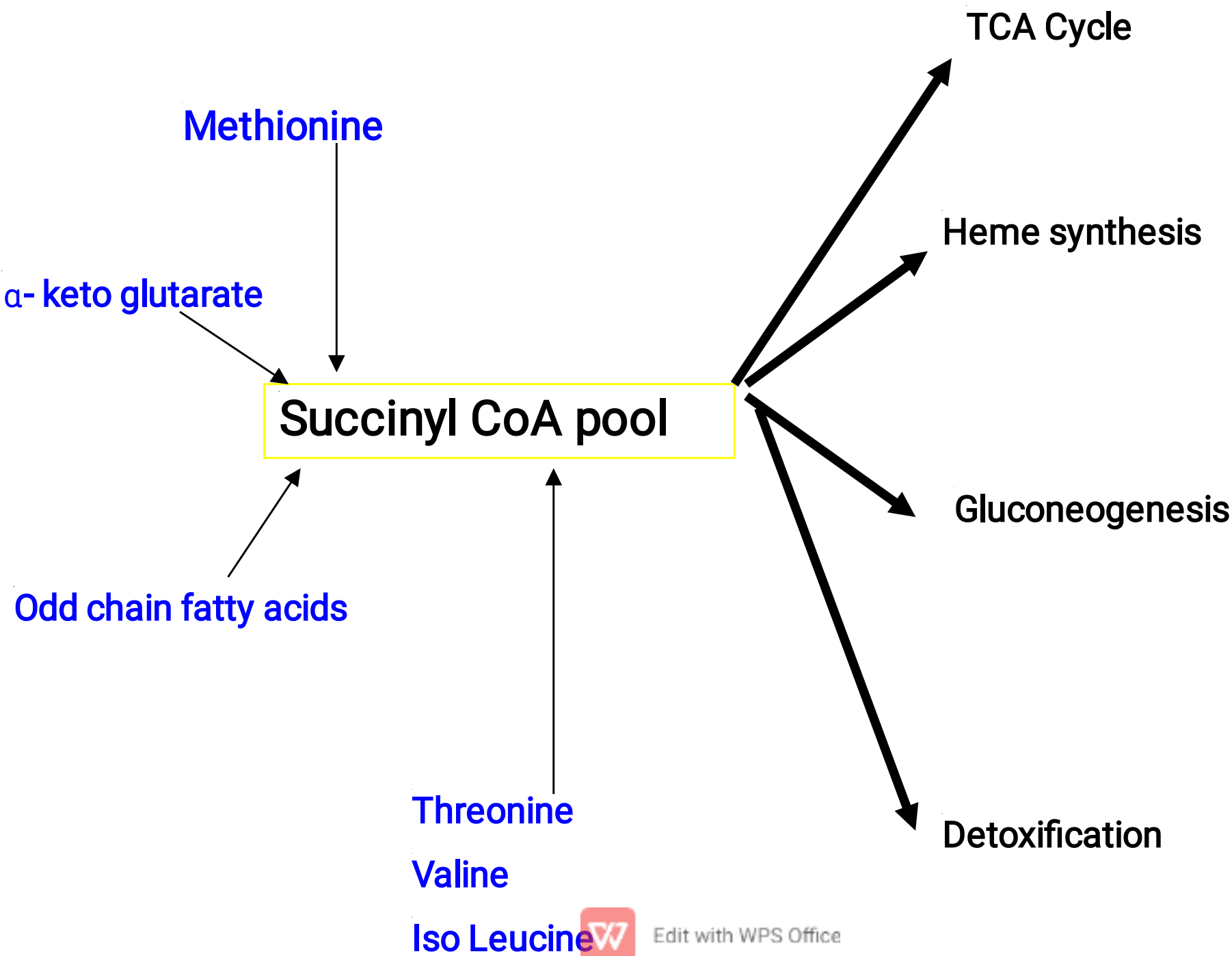


## 4. Metabolic role

- a. Fatty acids activated to acyl CoA by linking with CoA
- b. Component of acetyl CoA - starting substance / end product of all major metabolisms.
- c. Role in *denovo* synthesis of fatty acids







## 6. Deficiency

- a. **Causes : famine , in prison camps, chronic alcoholics**
- b. **C/F**

### **Gopalan's Burning foot syndrome**

- **Parasthesia in lower extremities**
- **Staggering gait**
- **Sleep disturbances**



# Biotin



# Biotin

## 1. Chemistry:

- Anti-egg white injury factor

## 2. Sources:

Milk, soybean, egg yolk, liver

Normal gut bacteria

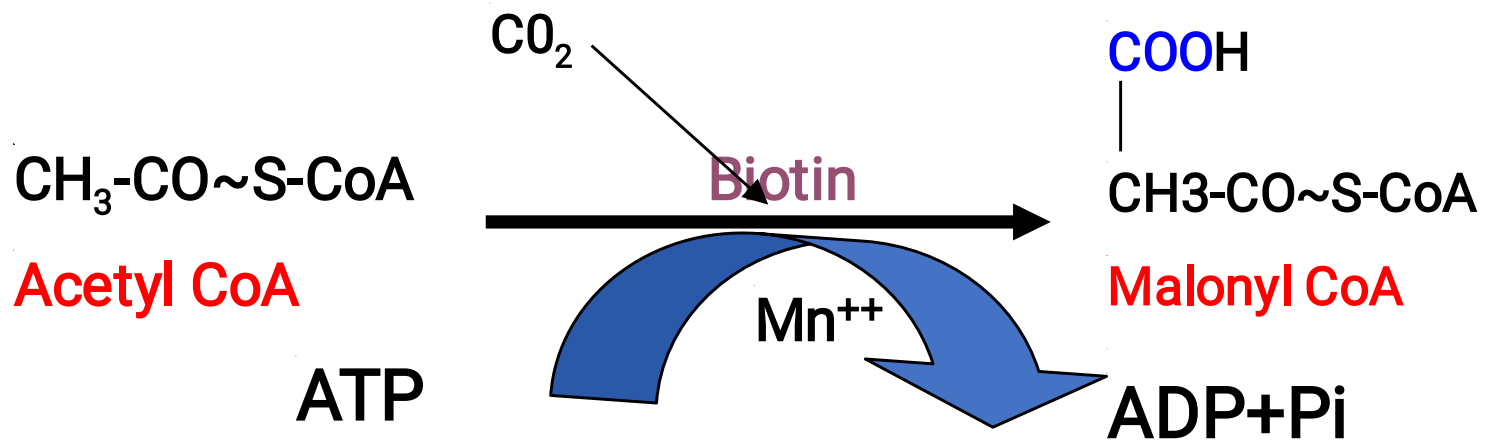


# Coenzyme activity

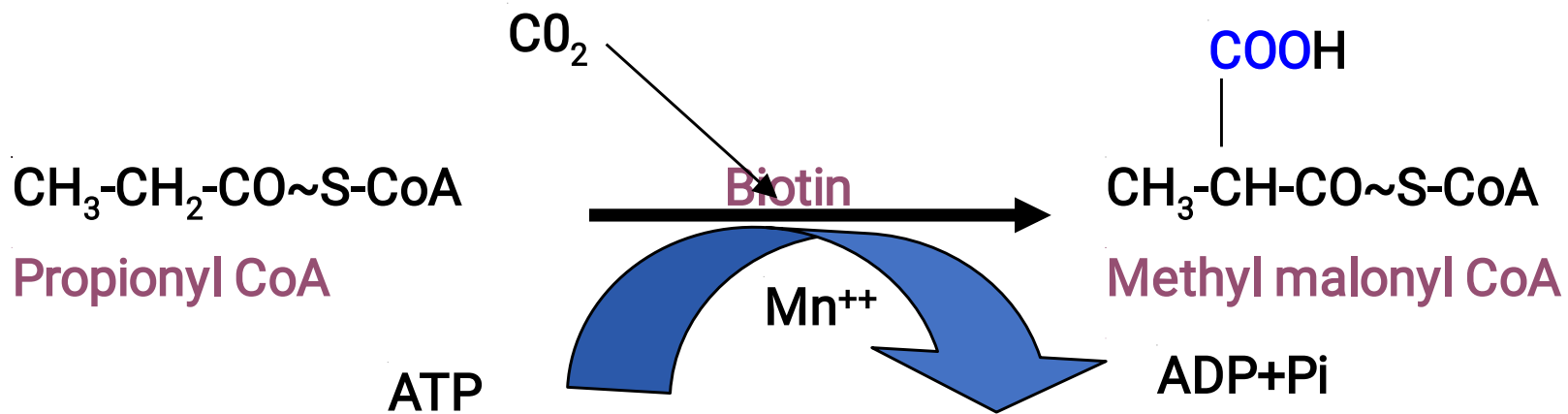
- Serves as a carrier of  $\text{CO}_2$  in carboxylation reactions.
- $\text{CO}_2$  get attached to the nitrogen of biotin.
- Energy required provided by ATP.



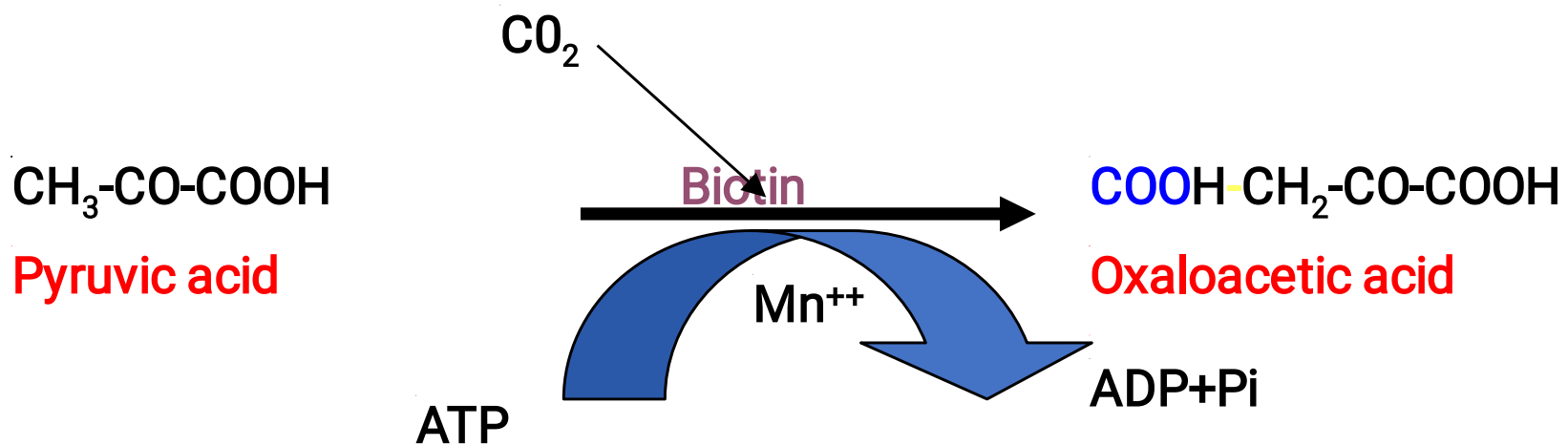
- Acetyl CoA Carboxylase



- Propionyl CoA carboxylase



- Pyruvate carboxylase



# Deficiency

- **Avidin**
  - Glycoprotein present in raw egg binds to biotin and prevents its absorption
- Prolonged use of antibacterial drugs
- Dermatitis, glossitis, muscle pain, anorexia, hallucination



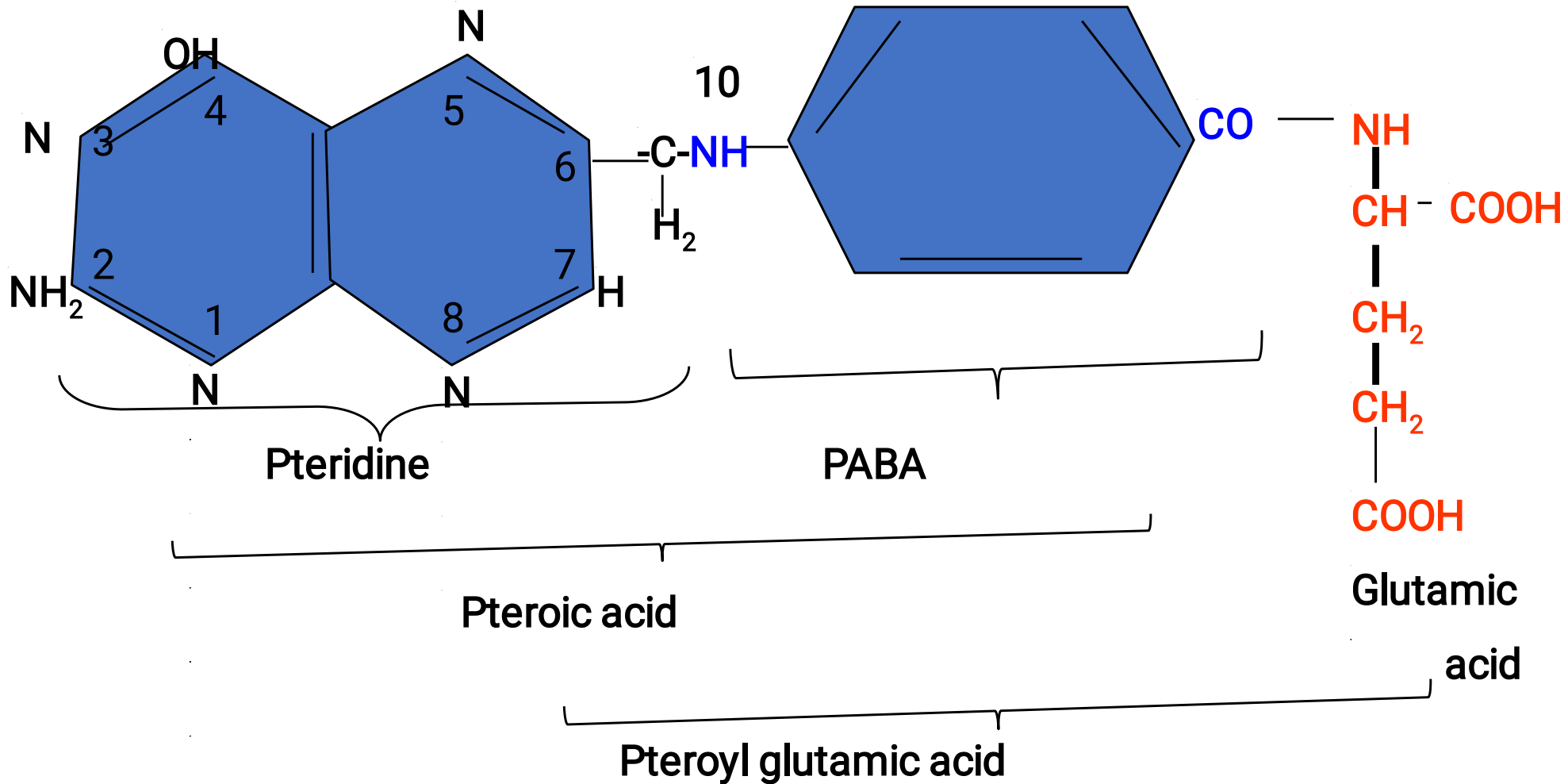
# Folic acid



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# Chemistry

3 components:



# Sources

- Green leafy vegetables
- Cereals, pulses
- Liver, meat, milk
- **RDA:** Adult-200 $\mu$ g
- Pregnant women - 400 $\mu$ g
- Lactating women - 300 $\mu$ g

## Vitamin B<sub>9</sub> Folate

Food sources of folate include beans and legumes, citrus fruits and juices, whole grains, dark green leafy vegetables, poultry, pork, shellfish and liver



ADAM.



# Absorption

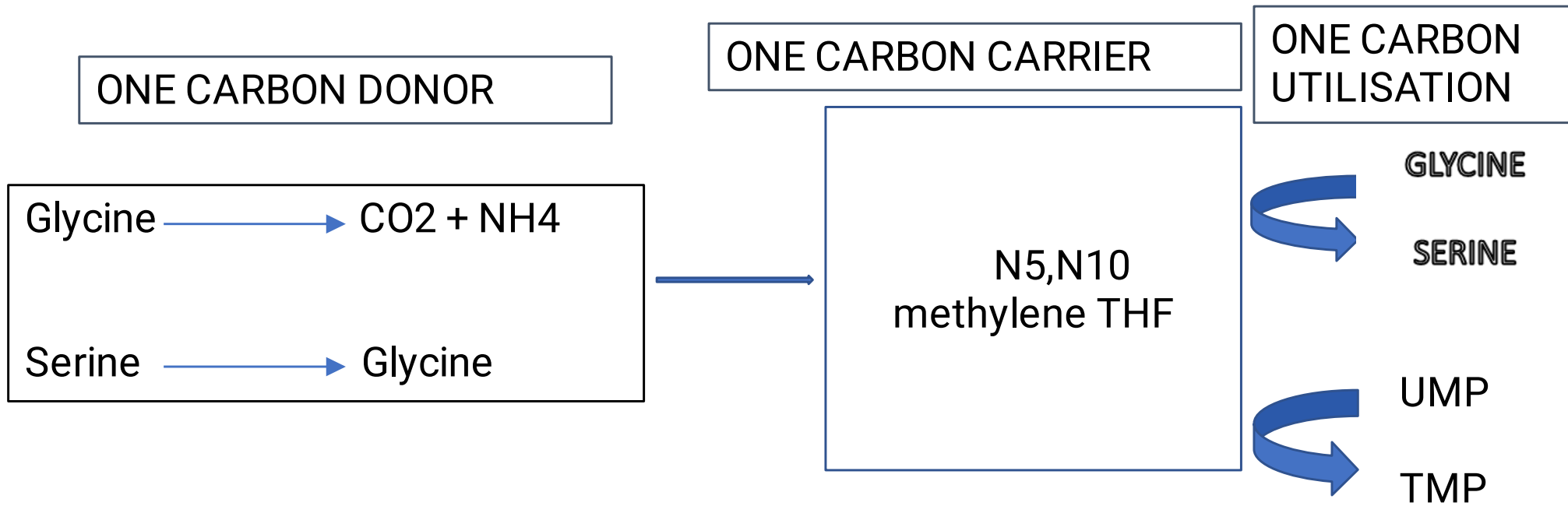
- Most dietary folic acid found as polyglutamate with 3-7 glutamate residues (Folacin).
- Folate conjugase splits glutamate residues.
- Monoglutamate of folic acid is absorbed from intestine
- Transported by  $\beta$ - globulins
- Coenzymes are produced in the liver

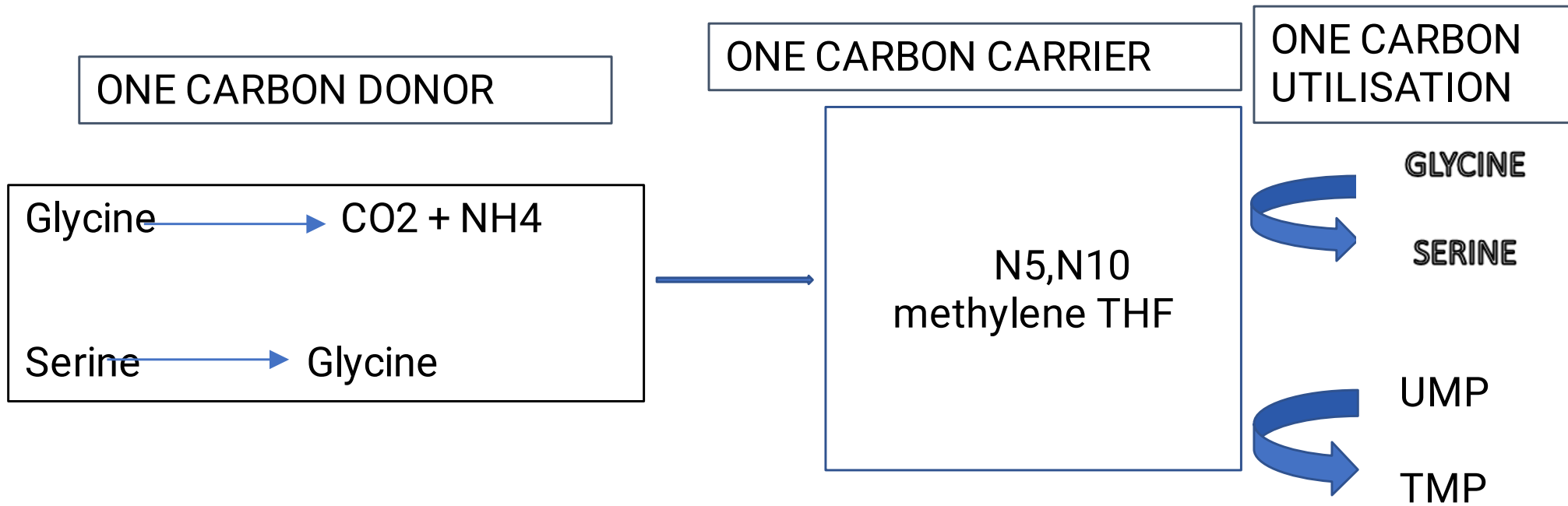


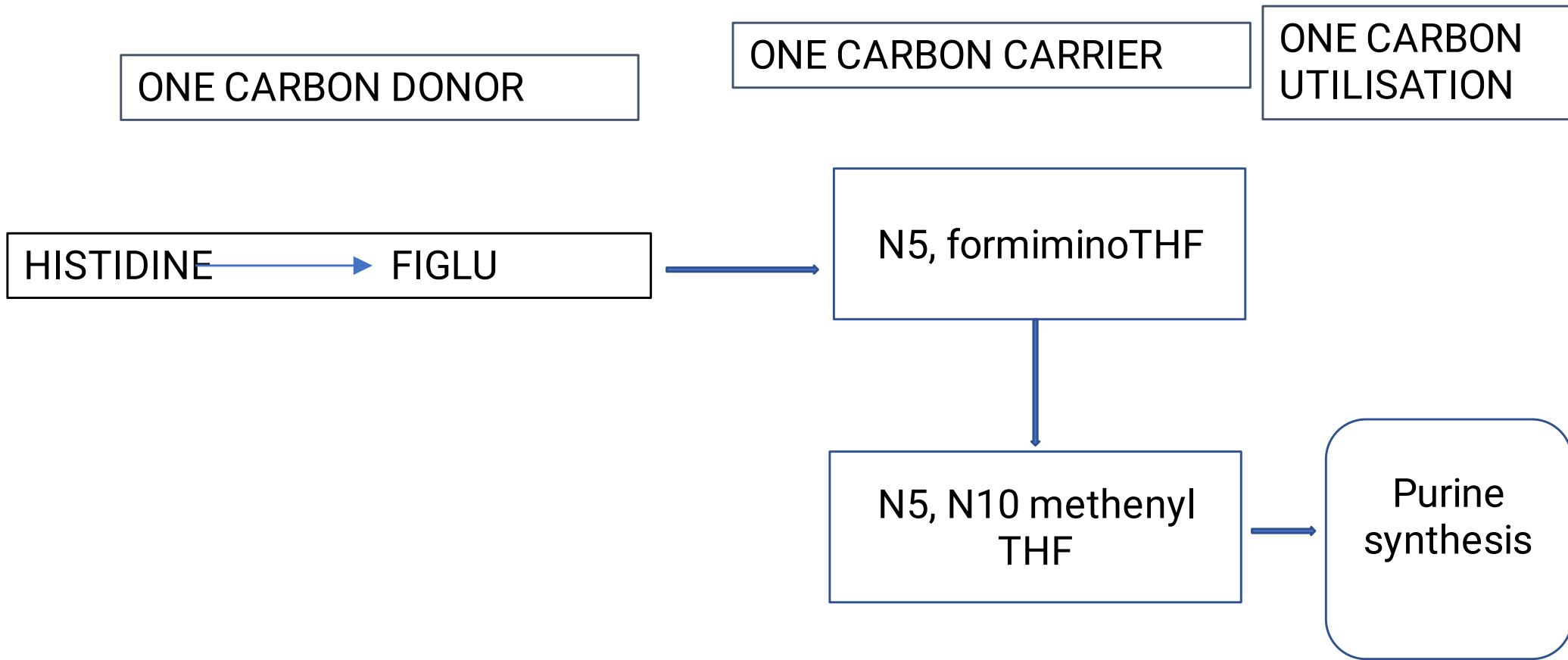
# Coenzyme function

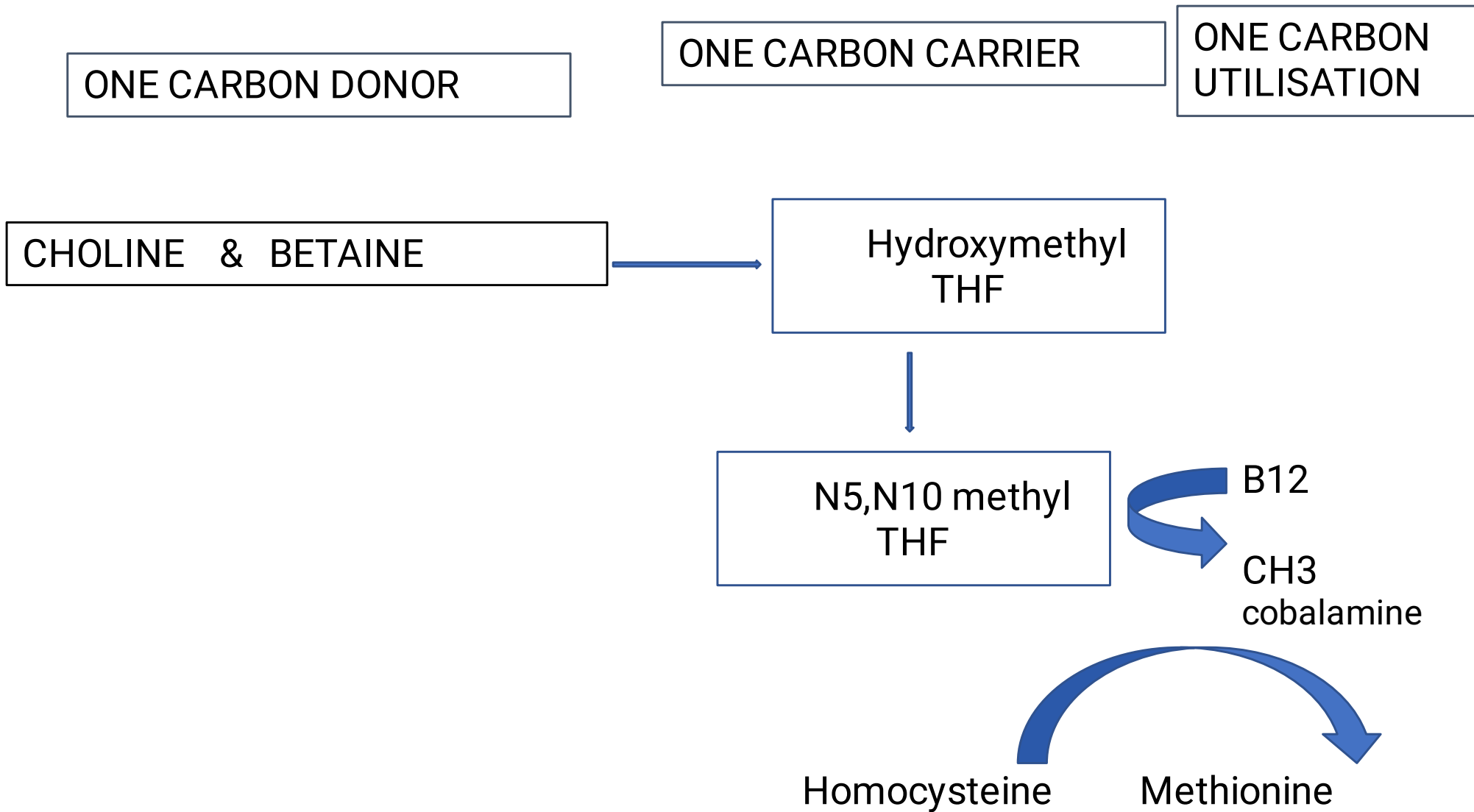
- THF is the carrier of one-carbon groups.
- One-carbon compound is an organic molecule that contains only a single carbon atom.





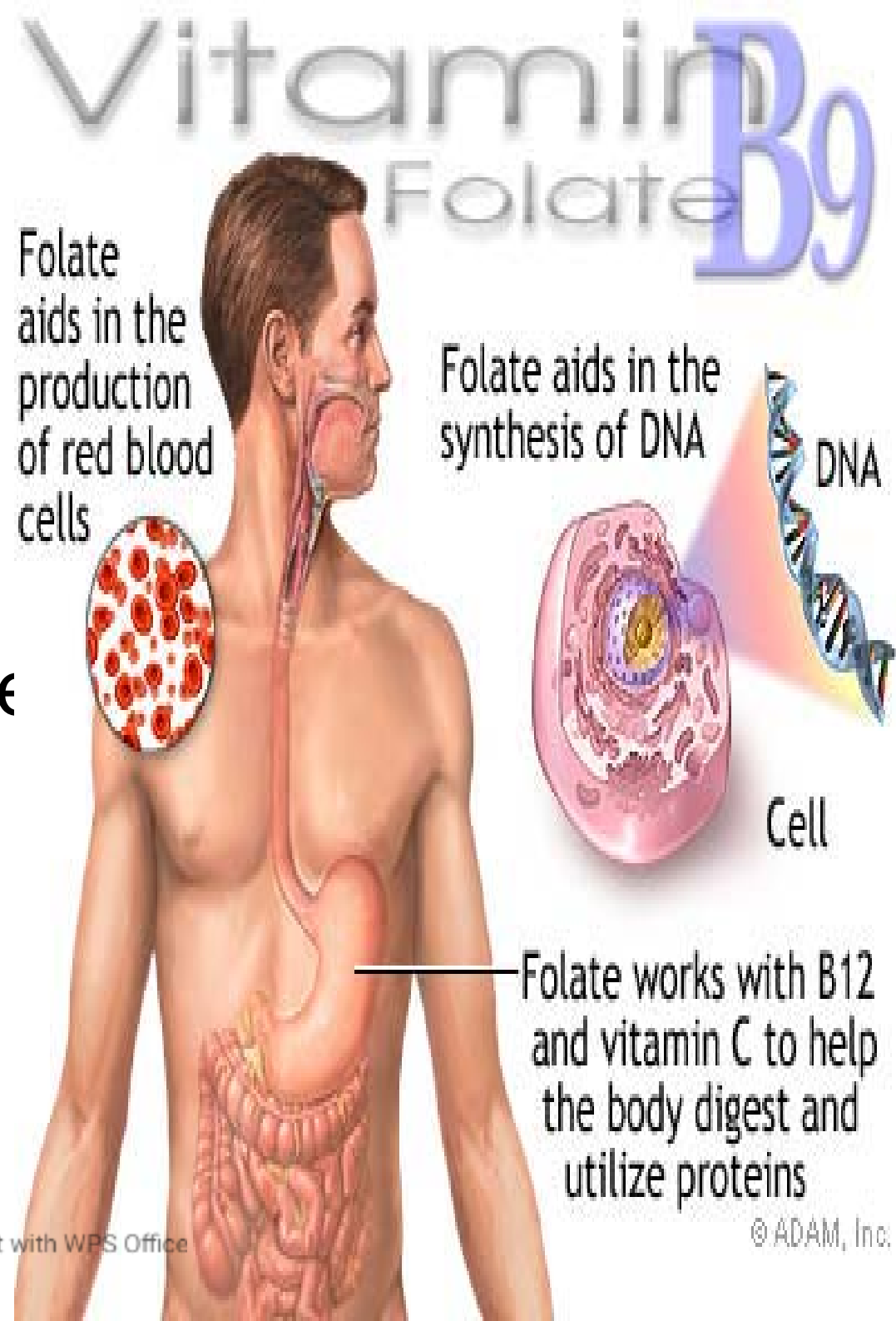






# Other functions

- Prevention of birth defects- fetal malformations such as spina bifida.
- Decrease homocysteine level in blood and therefore helps in preventing heart diseases



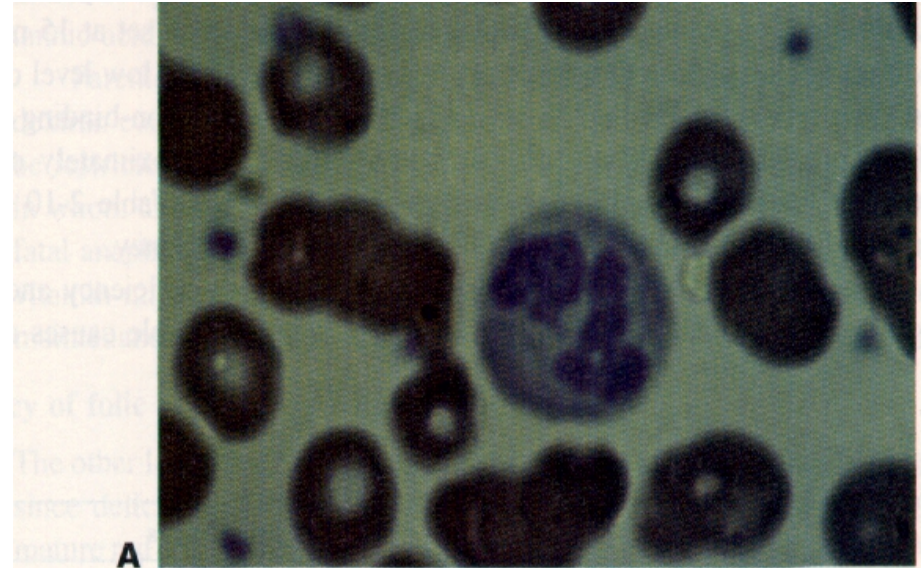
# Etiology of folate deficiency

- **Pregnancy**
- **Defective absorption (sprue, celiac disease etc)**
- **Drugs (phenobarbitone, phenytoin, hydantoin)**
- **Hemolytic anemia**
- **Dietary deficiency**
- **Folate Trap**
- **Fish tape worm**

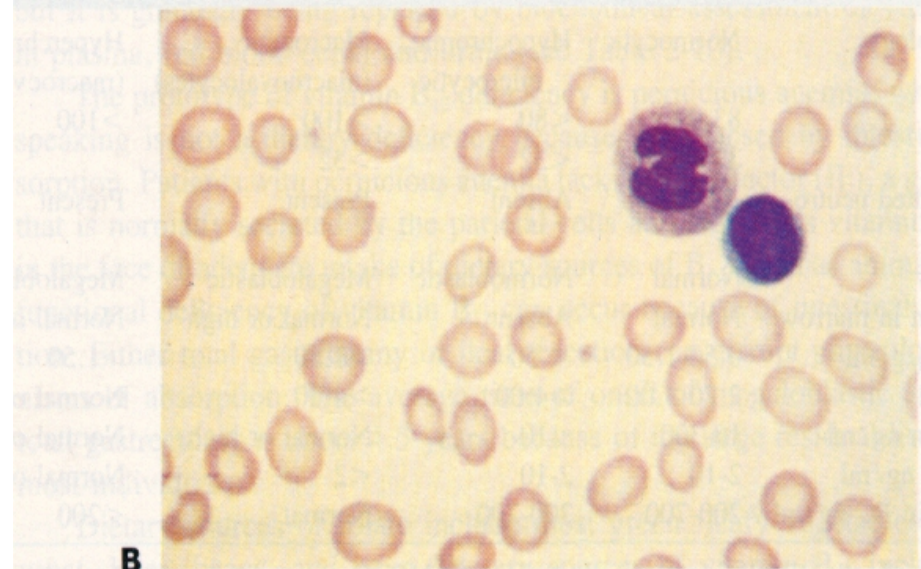


# Clinical features

- Macrocytic anemia
- Reticulocytosis
- Hypersegmented neutrophils
- Leucopenia
- Thrombocytopenia



From "Fundamentals of Clinical Nutrition" by R. L. Weinsier copyright 1993 by Mosby-Year Books Inc. N.Y.

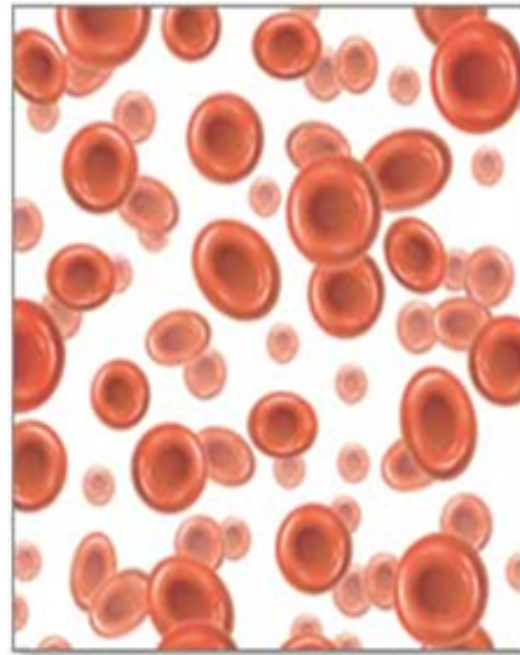


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**Fig. 2-6** A, Blood cells in macrocytic anemia; notice the hypersegmented polymorphonuclear leukocytes. B, Blood cells in microcytic anemia.

- Folic acid deficiency during pregnancy may lead to neural tube defects in the fetus
- Hyperhomocysteinemia

Folic acid is necessary for red blood cell production and neural tube formation



# Measurement of folate deficiency

- Normal serum level – 20ng/ml
- Histidine load / FIGLU excretion test
- AICAR (amino imidazole carboxamide ribonucleotide) excretion
- Peripheral smear



# Folic acid antagonists

- Aminopterine (treat cancers)
- Sulphonamides (antibacterial)
- Trimethoprim (bactericidal action)
- Pyrimethamine (antimalarial)



# Vitamin B 12



- Also known as anti-pernicious anemia vitamin.
- Only synthesized by microorganisms (anaerobic bacteria).
- Never found in Plants.



# Sources

- Only in foods of animal origin
- Liver , Eggs , Fish , Meat
- Vegetarians should take enough milk , milk products or egg
- **RDA** : Adults- 1-2 $\mu$ g

## Vitamin B<sub>12</sub>

Food sources of vitamin B<sub>12</sub>:

Eggs, meat, poultry, shellfish, milk and milk products



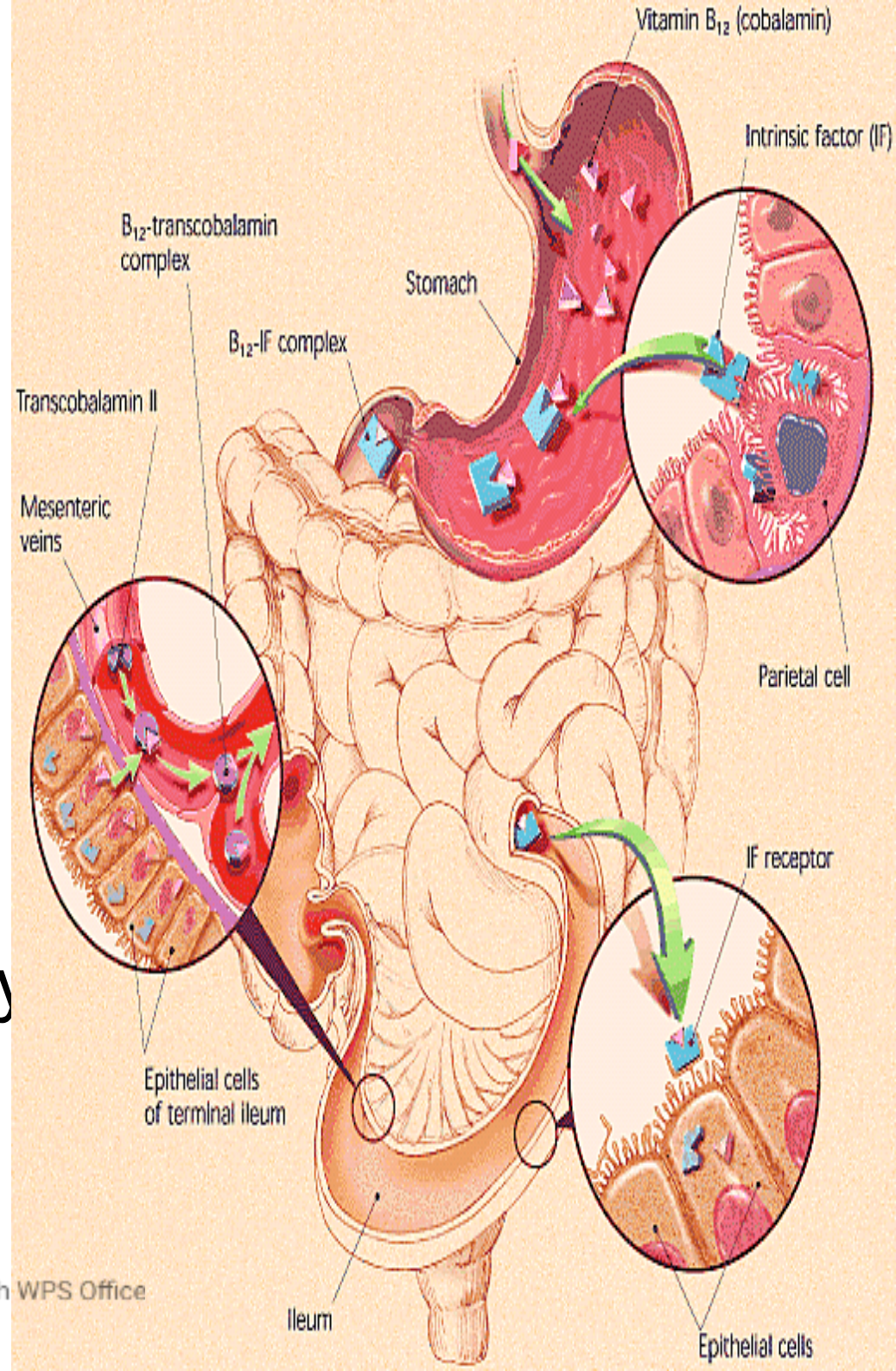
# Absorption

- Site of absorption: terminal Ileum
- Dietary B<sub>12</sub> is known as Extrinsic factor of Castle
- Haptocorrin is a glycoprotein secreted by salivary glands and parietal cells
- Intrinsic factor of Castle (constituent of normal gastric juice, glycoprotein secreted by parietal cells of the gastric mucosa).

- Gastric pepsin releases vitamin from proteins of food.
- Free vitamin B<sub>12</sub> binds to haptocorrin
- In duodenum, trypsin hydrolyses haptocorrin
- Free vitamin B12 binds to IF & forms **IF-B<sub>12</sub> complex**
- This complex attaches to specific receptors on mucosal cells & internalized



- IF-B<sub>12</sub> complex bind to specific receptors on mucosal cells.
- The whole IF-B<sub>12</sub> complex internalised.
- IF is resistant to proteolytic digestive enzymes . This binding protects vit B<sub>12</sub> against its uptake and use by bacteria.



# Transport

- The IF is digested inside the mucosal cell and  $B_{12}$  is transported to the circulation.
- In the blood Methyl  $B_{12}$  form is predominant.
- Transcobalamin II, a glycoprotein is the specific carrier.



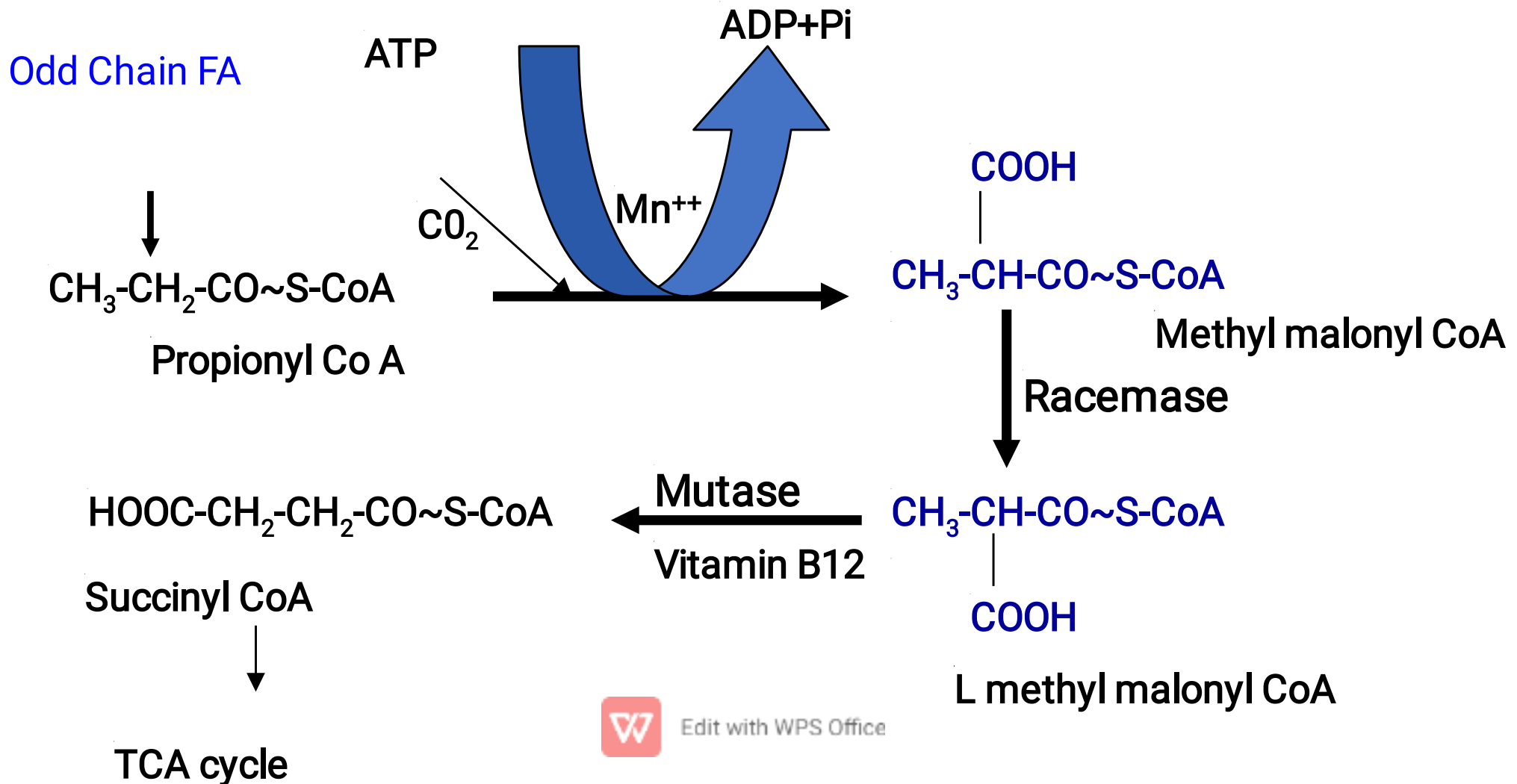
# Storage

- Methylcobalamine in excess, stored in Liver cells as Ado-B<sub>12</sub> form, in combination with transcobalamin I or transcorrin.
- Generally B complex vitamins are not stored in the body , but B<sub>12</sub> is an exception.



# Metabolic role

## Isomerisation of Methylmalonyl CoA to succinyl CoA



**N<sup>5</sup>N<sup>10</sup> Methylene THFA**

NADH<sub>2</sub>

NAD

Reductase

**N<sup>5</sup> Methyl THFA**

THFA

Cobalamin

Methyl Cobalamin

Methionine

Homocysteine



## Methyl folate trap

- Production of methyl THFA-an irreversible step.
- THFA production requires B<sub>12</sub>.
- So major pathway for conversion of N5 methyl THF to THF is blocked and body THF pool is reduced.
- Almost entire body folate becomes trapped as N5 methyl THF.
- Folate trap or methyl trap.

# Etiology of vitamin B<sub>12</sub> deficiency

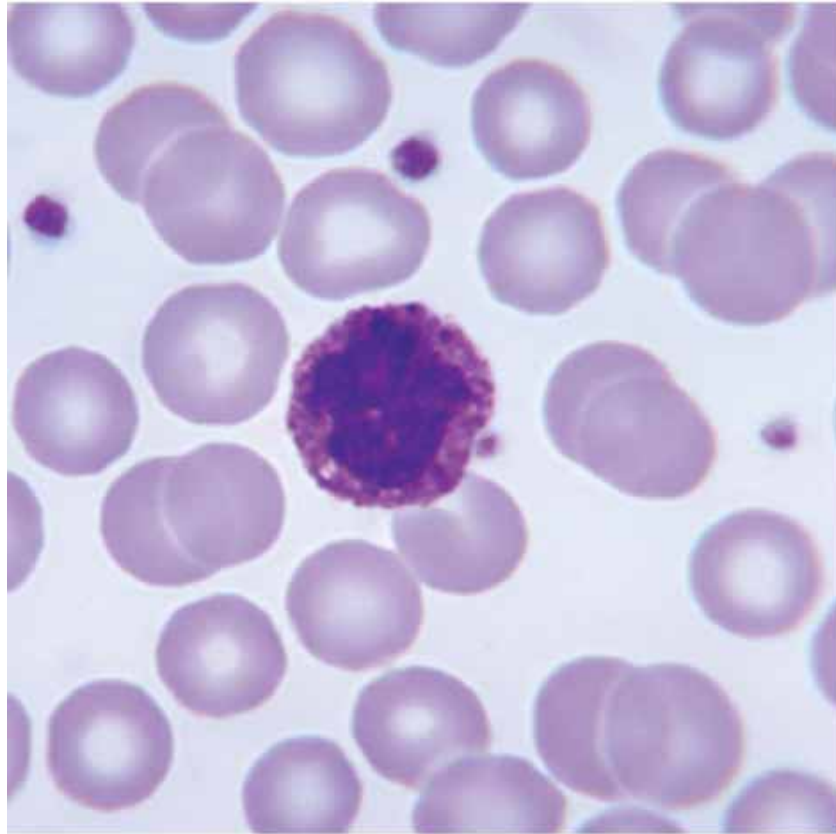
- Nutritional deficiency
- Decreased absorption- IF ↓
- Addisonian pernicious anemia
- Gastric atrophy
- Fish tape worm infestation
- Pregnancy



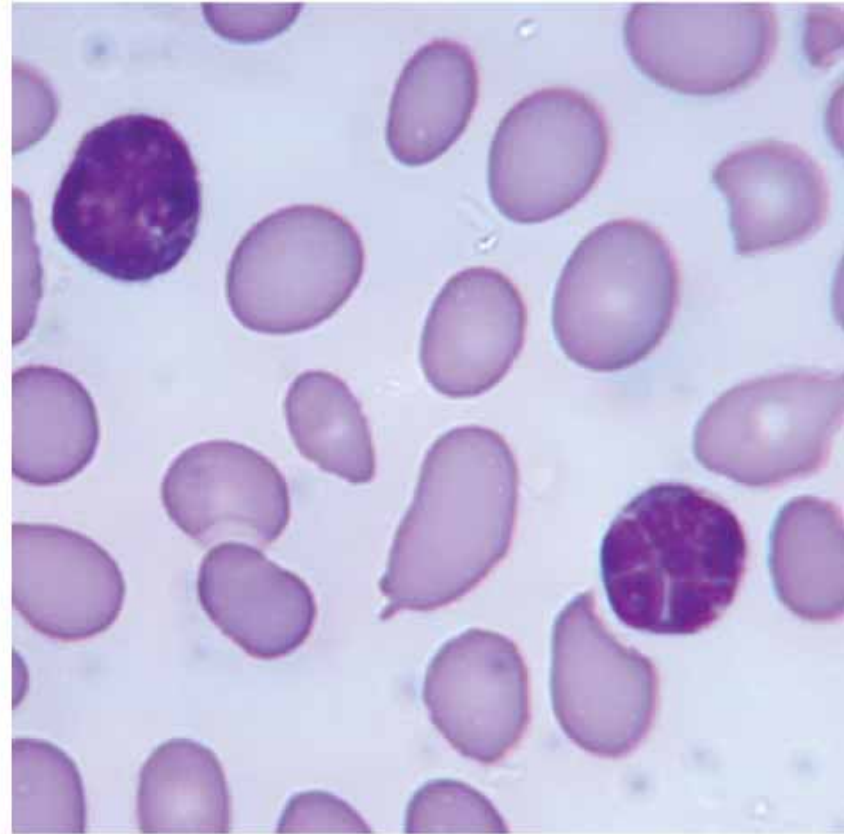
# Vitamin B<sub>12</sub>

- Deficiency of B<sub>12</sub> produces megaloblastic anemia

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Normal blood cells. The size, shape, and color of the red blood cells show that they are normal.



Blood cells in pernicious anemia (megaloblastic). Megaloblastic blood cells are slightly larger than normal red blood cells, and their shapes are irregular.

- Neurological symptoms
- Demyelination and neuronal degeneration of nervous system
- Clinical features
  - Parasthesia
  - Confusion, loss of memory
  - Psychosis
  - Ataxia
- Subacute combined degeneration



## Biochemical changes

- Serum B<sub>12</sub> level decreased
- **Assessment**
- Schilling test
- Peripheral smear
- Methyl malonic acid is excreted in urine
- **Treatment:** Vitamin B<sub>12</sub> & Folate



# Schilling test

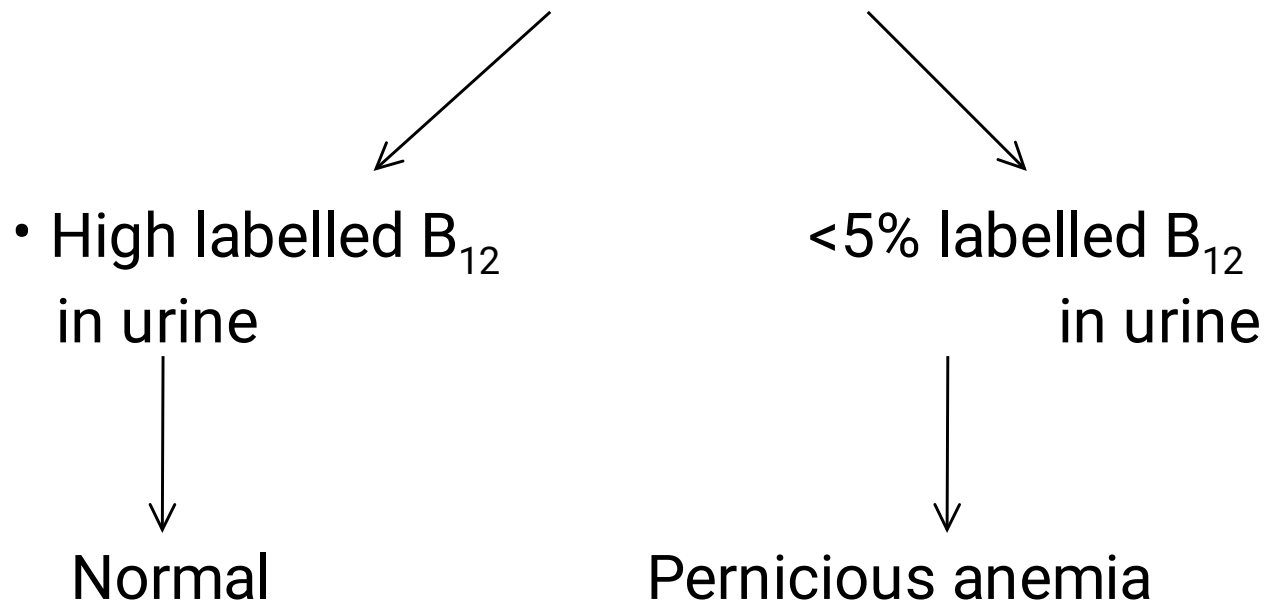
Orally :  $\text{Co}^{60}$  labelled  $\text{B}_{12}$

Intramuscular injection : unlabelled  $\text{B}_{12}$  to saturate tissues

So all  $\text{Co}^{60}$   $\text{B}_{12}$  will pass into urine as tissues are saturated



- Collect urine over next 24 hrs





- 



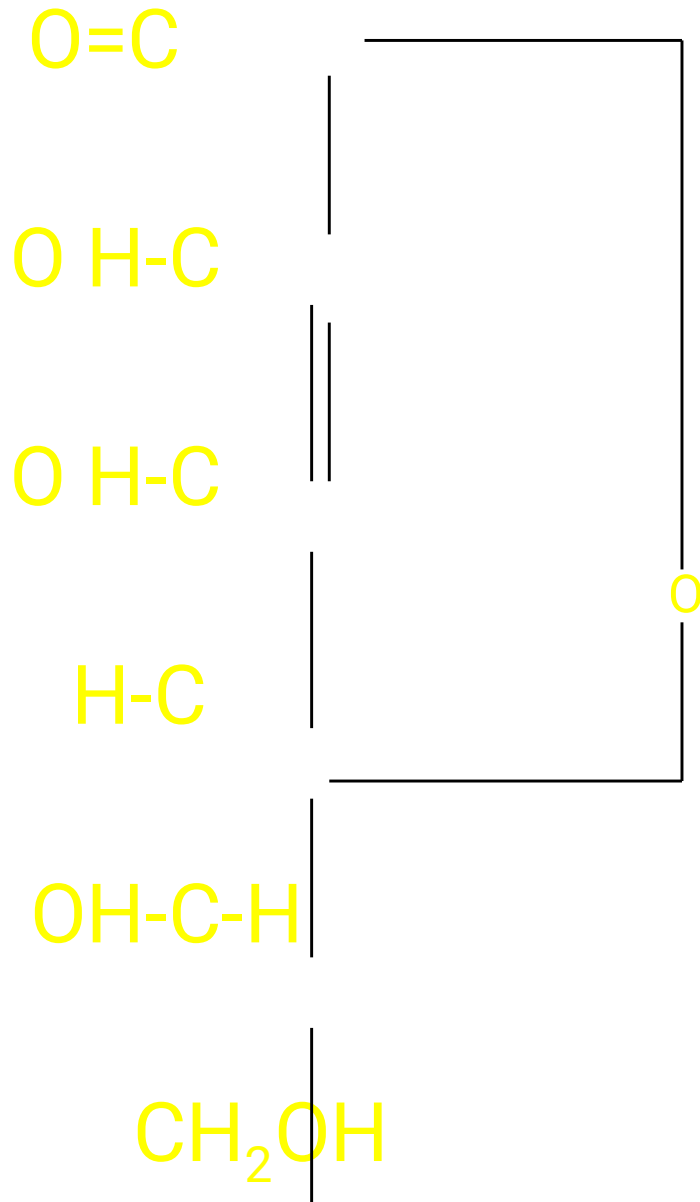
# Vitamin c



Ascorbic acid /   
Antiscorbutic factor



- 
- 



L- ascorbic acid



# 1. chemistry

- Only L-ascorbic acid & dehydroascorbic acid are active (not D-ascorbic acid )
- Has strong reducing property – gives +ve Benedict's test



## 2. Synthesis

- Most animals & plants synthesize from glucose but not humans - lack of enzyme

*L- gulono lactone oxidase*



### 3. Sources

- Amla
- Guava
- Lemon
- Green leafy vegetables





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# Vitamin C Sources

Citrus fruits  
Amla  
Cabbage-type  
vegetables, dark green  
vegetables  
strawberries  
Lettuce, tomatoes,  
potatoes  
Papayas, mangoes



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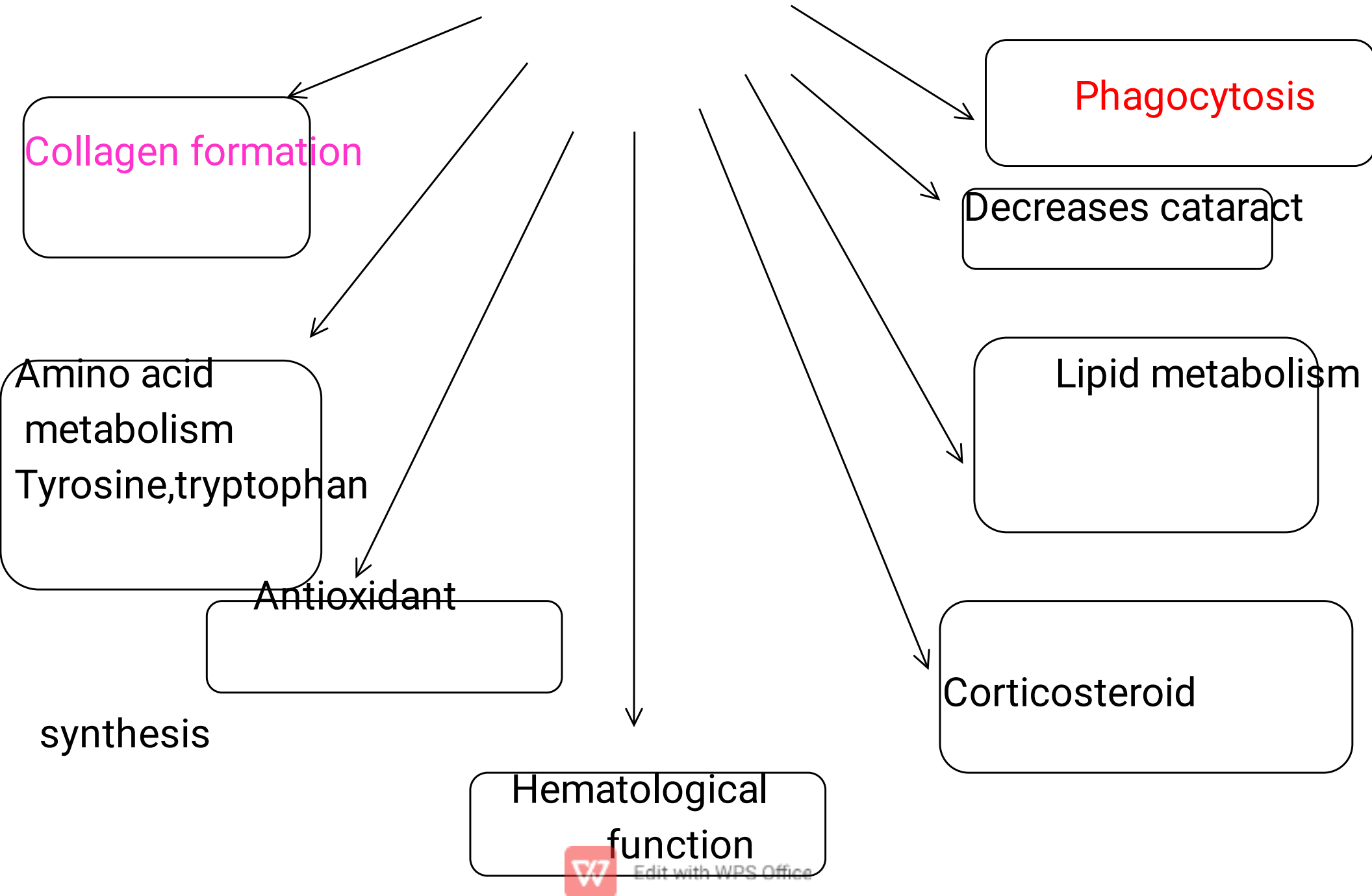


## 5. RDA

- Adult -75mg/day



## 4. Biochemical functions



Proline &  
Lysine

Hydroxyproline &  
Hydroxylysine

Proline & Lysyl  
Hydroxylases

$\text{Fe}^{2+}$

$\text{Fe}^{3+}$

Semidehydroascorbic Acid

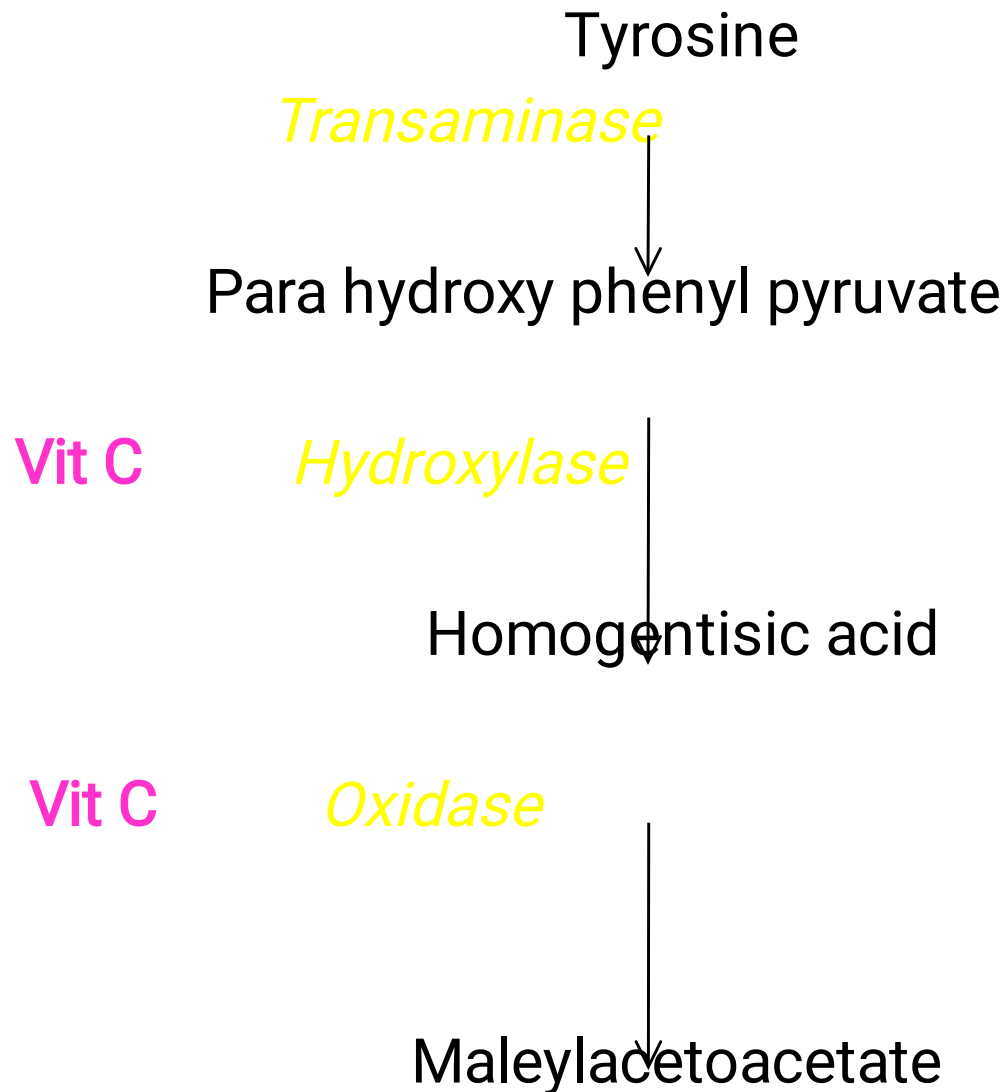
Ascorbic Acid



- This provides tensile strength to collagen by allowing cross linking due to formation of hydrogen bonds
- So vitamin C is needed for wound healing and bone formation



## 2. Aminoacid metabolism - Tyrosine



Tyrosine

*Hydroxylase*

DOPA

*Decarboxylase*

Dopamine

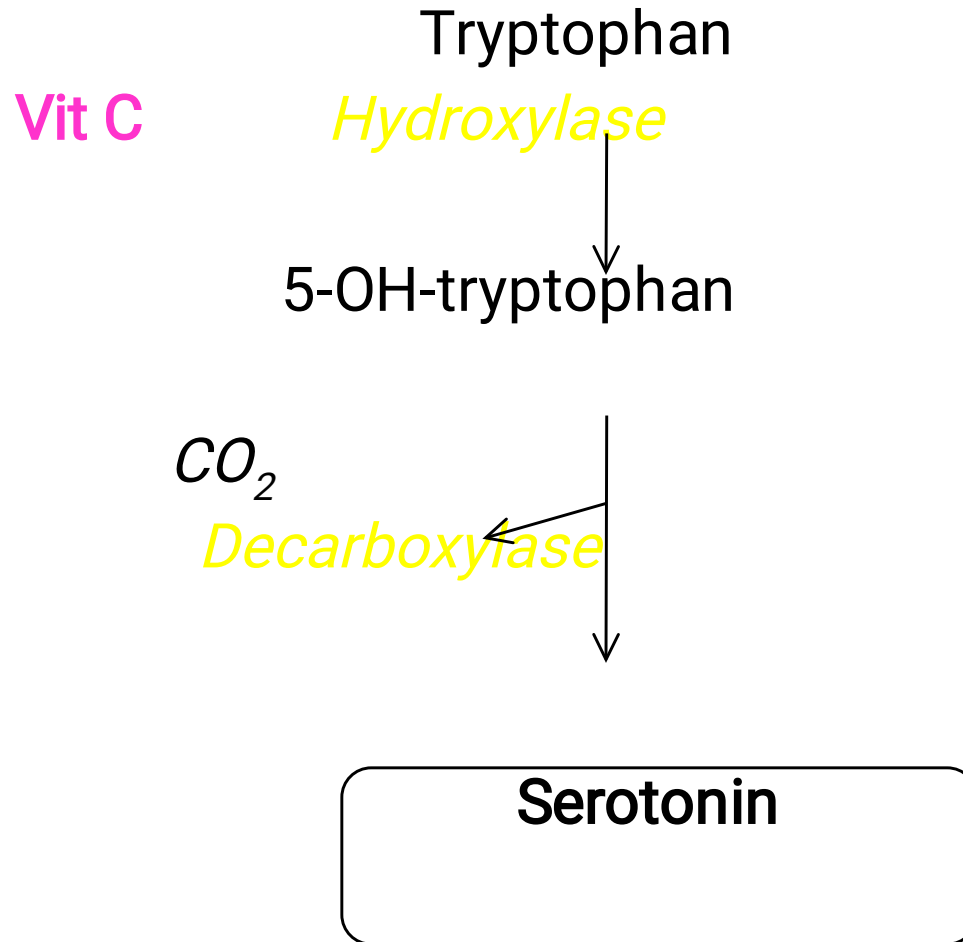
Vit C

*Hydroxylase*

Norepinephrine



# Tryptophan



### 3. Hematologic role:

#### Metabolism of

#### Iron

Reduces  $\text{Fe}^{3+}$  to  $\text{Fe}^{2+}$   
↑ iron absorption fm int.

#### Hemoglobin

reconversion of  
met-Hb to Hb

#### Folate

- RBC maturation

#### DHF reductase

Folate



DHF



THF



### 3. Hematologic role:

#### Metabolism of

**Iron**

Reduces  $Fe^{3+}$  to  $Fe^{2+}$   
↑ iron absorption fm int.

- RBC maturation

**DHF reductase**

Folate

NADPH, **Vit C**

DHF

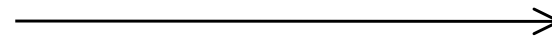
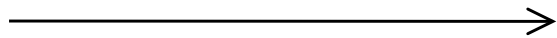
NADPH, **Vit C**

THF

**Hb**

reconversion of  
met-Hb to Hb

**Folate**



# 4. Lipid metabolism



## 5. Steroid synthesis

a. Adrenal steroidogenesis

## 6. Others

a. Antioxidant property: reduces risk of cancer

b. Phagocytosis: stimulates phagocytic action of leukocytes & helps in the formation of antibodies

c. Cataract formation: regular intake of vitamin C reduces cataract formation



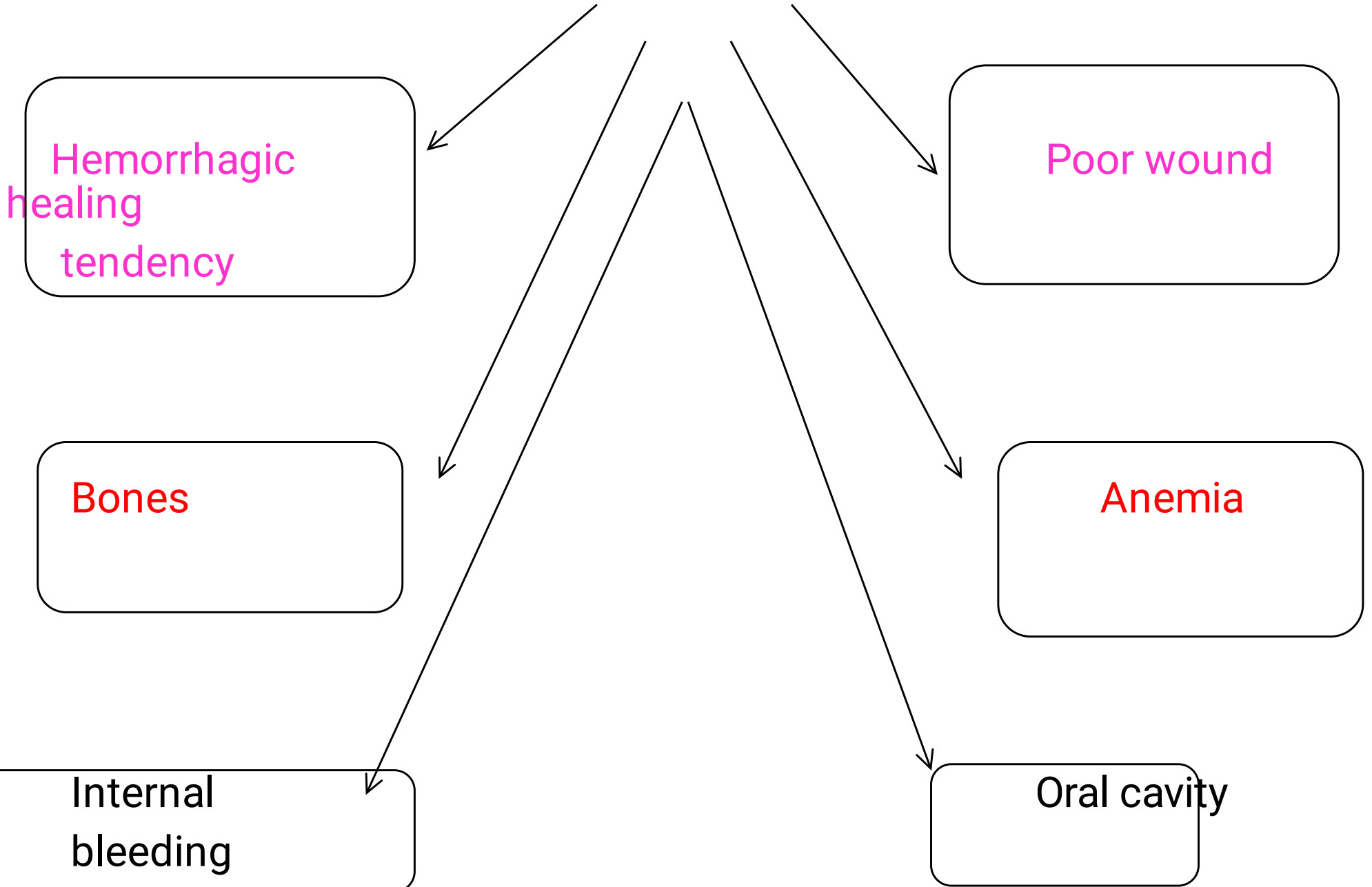


## 5. RDA

- Adult -75mg/day



## 6. Deficiency - Scurvy



## 6. deficiency

- Scurvy

- a. Hemorrhagic tendency:

- Collagen abnormal, intercellular cement substance brittle
- Capillaries fragile, tend to bleed even under minor pressure
- Subcutaneous hemorrhage may be in the form of petechiae (in mild deficiency) or ecchymoses or hematoma (in severe deficiency).



# Hemorrhagic tendency

- Collagen abnormal
- Capillaries fragile, tend to bleed under minor pressure
- Subcutaneous hemorrhage occur as
  - Petechiae
  - ecchymoses/hematoma





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ADAM



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# Internal Hemorrhage

-in conjunctiva & retina

-Epistaxis, hematuria & melena



Oral cavity

•

- Swollen, spongy & painful gums
- Pulp gets separated from dentine
- Teeth get loosened



# bones

- Osteoid formation deficient
- bone formation defective –  
Scorbutic bone
- Weak & fractures easily
- Hemorrhage into joint cavities
- Painful swelling of joints



- Scurvy

anemia:

Reasons for anemia

- Hemorrhage
- Reduced iron absorption
- Reduced THF

Microcytic, hypochromic anemia

Poikilocytosis & anisocytosis



# Antivitamins

- Def: compounds that oppose & block the action of vitamins (antagonists)
- have structural similarities with vitamins
- Administration of antivitamin cause vitamin deficiency



## Vitamin

- Thiamine
- Riboflavin
- Pyridoxine
  
- Biotin
  
- Folic acid

## Antivitamin

- Pyriothiamine, Oxythiamine
- Galactoflavin
- Isoniazid, deoxypyridoxine, methoxypyridoxine
- Desthiobiotin, biotin sulphonic acid
- Sulfonamides, Pyrimethamine, Methotrexate

