Faculty of Pharmacy

Biochemistry-2

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Lecturer of Biochemestry

Vitamins

General characteristics of vitamins

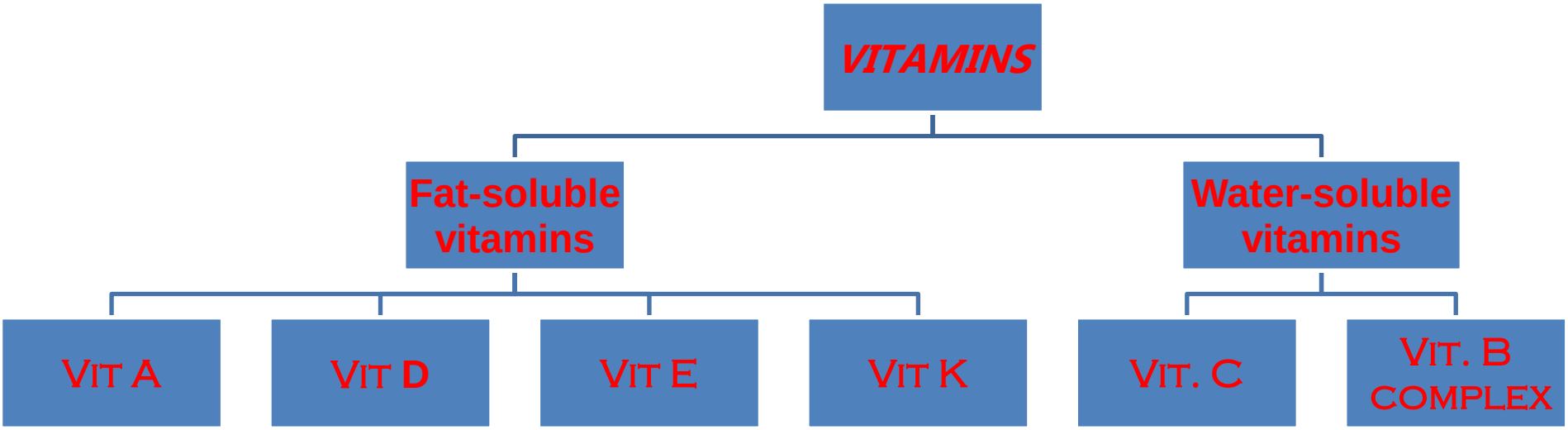
1. **Micronutrients**
2. **Essential**
3. **Non-energy producing**

What are the characteristics of vitamins?

- Vitamins are organic compounds
- They are required in trace amounts
- They perform specific cellular function
- They are essential for normal health and growth
- They cannot be synthesized by humans
- They must be supplied by the diet
- They don't enter in tissue structure
- They are not used in the production of energy

Classification of vitamins

	Fat soluble vitamins A, D, E, K	Water soluble Vitamins C, B complex (8)
Solubility	Soluble in fats	Soluble in water
Storage	Stored in body fat	Not stored but rapidly excreted in urine
Toxicity	Cause toxicity if taken in excess amount	Rare toxicity due to its rapid excretion



Fat-Soluble Vitamins

- *Excess amounts are stored in liver & adipose tissue*
- *Large doses may be Toxic*
- *Digested & absorbed with fat*
- *Not excreted in urine*

Water-Soluble Vitamins

- *Non–Toxic, Not stored*
- *Excess amounts excreted in urine*
- *Must be continually supplied in the diet*

Water-Soluble Vitamins

A. Vitamin C (L–Ascorbic acid)

B. Vitamins B Complex

1- Thiamine (B_1)

2- Riboflavin (B_2)

3- Niacin (B_3)

4- Pyridoxine (B_6)

5- Biotin (Vit. H, B_7)

6- Pantothenic acid

7- Folic acid (B_{10})

8- Lipoic Acid

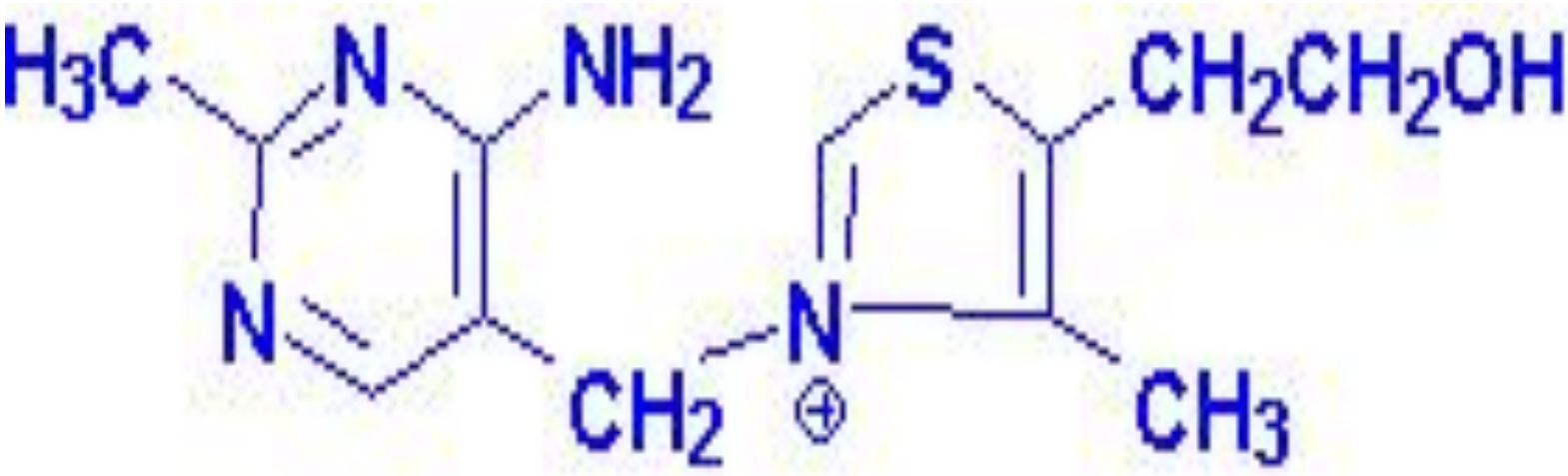
9- Inositol

10- Choline

11- Cyanocobalamine (B_{12})

VITAMINS B COMPLEX

Thiamine (B_1)



Active Form
Thiamine Pyrophosphate (TPP)

Thiamine + ATP → Thiamine Pyrophosphate + AMP

(TPP)

Metabolic Functions of TPP

- **TPP acts as a cofactor for decarboxylation reactions:**

1. **Simple decarboxylation in bacteria & yeast:**

Pyruvate —(Pyruvate Decarboxylase)→ **acetaldehyde**

2. **Oxidative decarboxylation:**

Pyruvate —(Pyruvate Dehydrogenase)→ **acetyl CoA**

3. **Transketolase reaction in Pentose shunt (HMP Shunt)**

Thiamine Deficiency

1. Thiamine is essential for carbohydrate metabolism so deficiency of thiamine leads to:
   A. Accumulation of pyruvate in blood
   B. Accumulation of lactate in blood
   C. Increases of pentoses in RBCs
2. Sever deficiency of thiamine leads to:
   A. Beri Beri
   B. Wernicke-Korsakoff Syndrome

Thiamine Deficiency

- **There are two types of Beri Beri:**
  - ❖ **Infantile (wet) Beri Beri**
  - ❖ **Adult (Dry) Beri**

Thiamine Deficiency

1. Symptoms of Infantile (wet) Beri Beri:

- **Tachycardia (↑ Heart Rate)**
- **Vomiting**
- **Convulsion**
- **Death**

Thiamine Deficiency

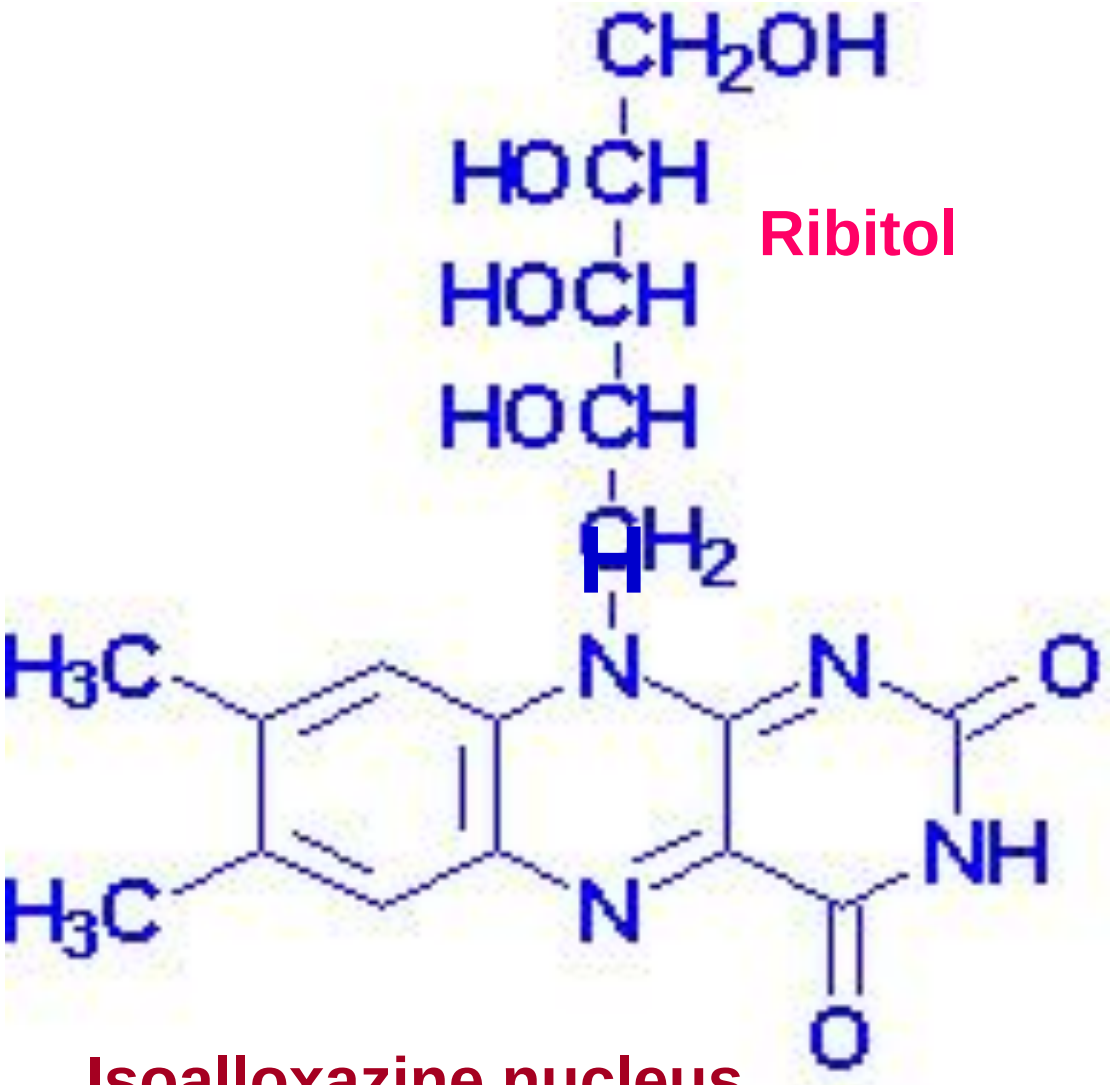
2. Symptoms of Adult (Dry) Beri Beri:

- **Dry Skin**
- **Irritability**
- **Disordered thinking**
- **Paralysis**

3. Wernicke-Korsakoff Syndrome is seen primarily in association with chronic alcoholism

- **Wernicke-Korsakoff Syndrome is characterized by:**
  - **Apathy (Lacking of feeling, lacking of desire, or ability to act)**
  - **Loss of memory**
  - **Rhythmic motion of eyeballs**
  - **Coma**
  - **Irreversible brain damage**
  - **Death**

Riboflavin (Lactoflavin, B_2)



Ribitol

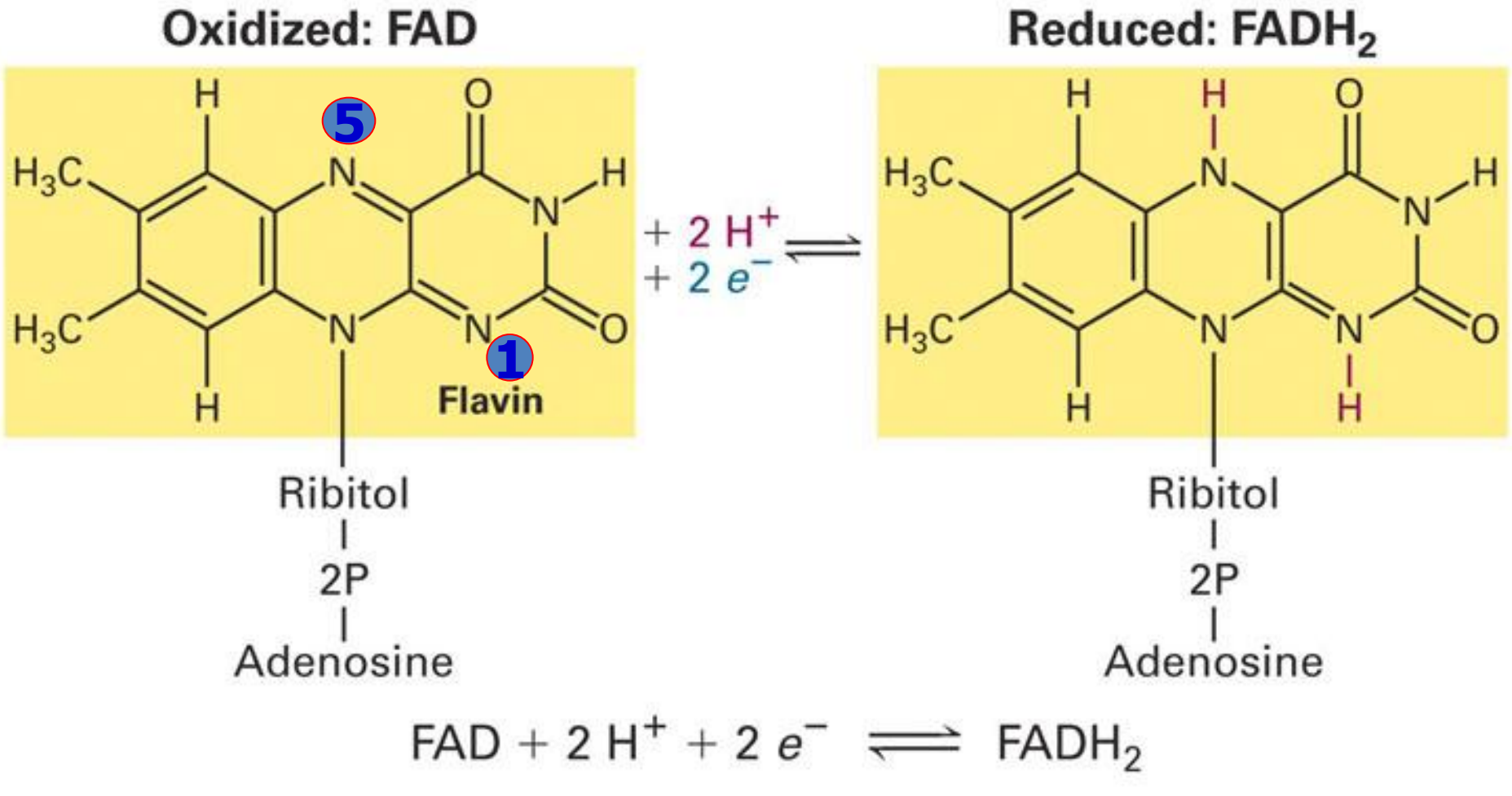
Isoalloxazine nucleus

Properties

- It is readily destroyed by ultraviolet components of sunlight.
- Riboflavin is present in tissues as biologically active forms which are:
- Flavin mononucleotide (FMN).
- Flavin adenino dinucleotide (FAD).

Function

Riboflavin gives FAD which acts as a coenzyme for several enzymes catalyzing the removal of hydrogen. So FAD acts as a hydrogen carrier for certain enzymes.

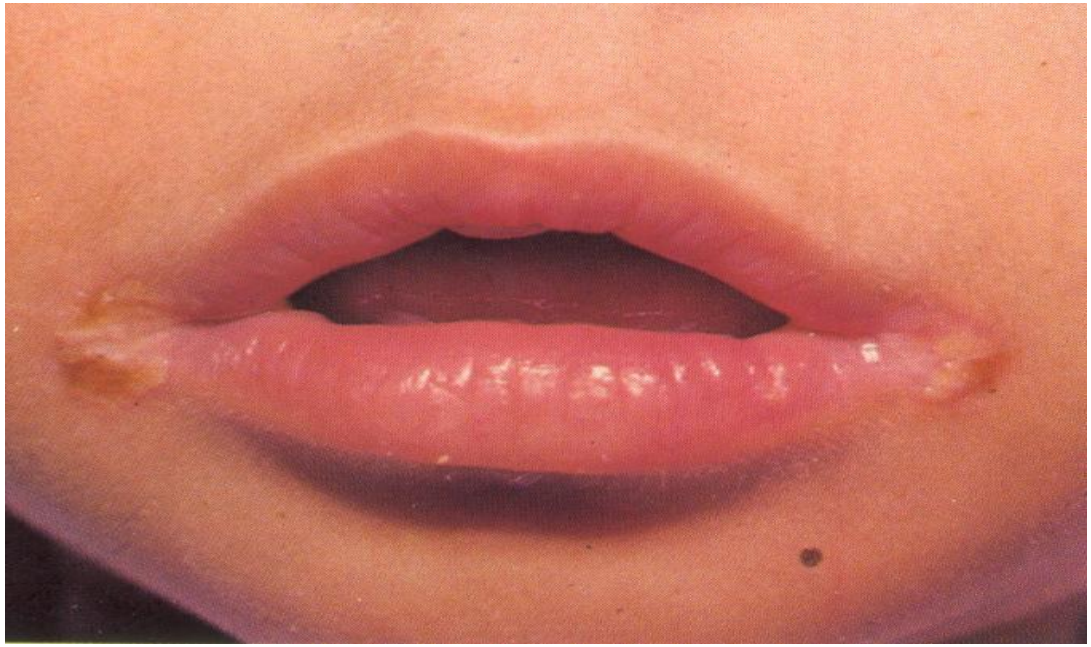


$$FAD + 2\,H^+ + 2\,e^- \rightleftharpoons FADH_2$$

Deficiency

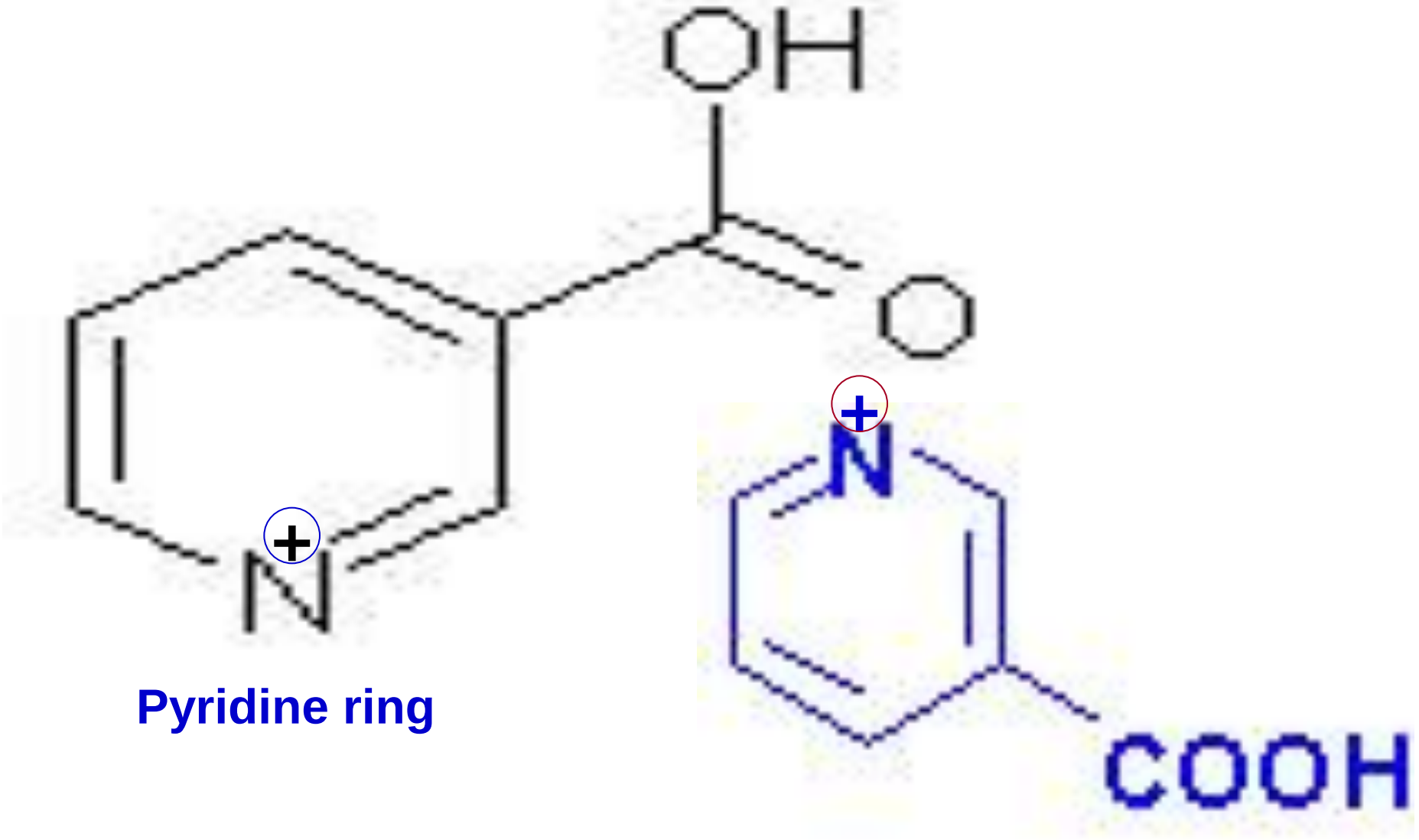
- Riboflavin deficiency is not associated with a major disease, although it is frequently accompanied with other vitamin deficiencies. Severe deficiency symptoms include:
- In skin: Dermatitis.
- Cornea: Vascularization.
- In mouth:
  - Cheilosis (fissuring at the corners of the mouth).
  - the tongue appearing smooth and purplish).
  - Lips are red and shiny.
  - Synthesis of protein is impaired

Symptoms of B_2 Deficiency

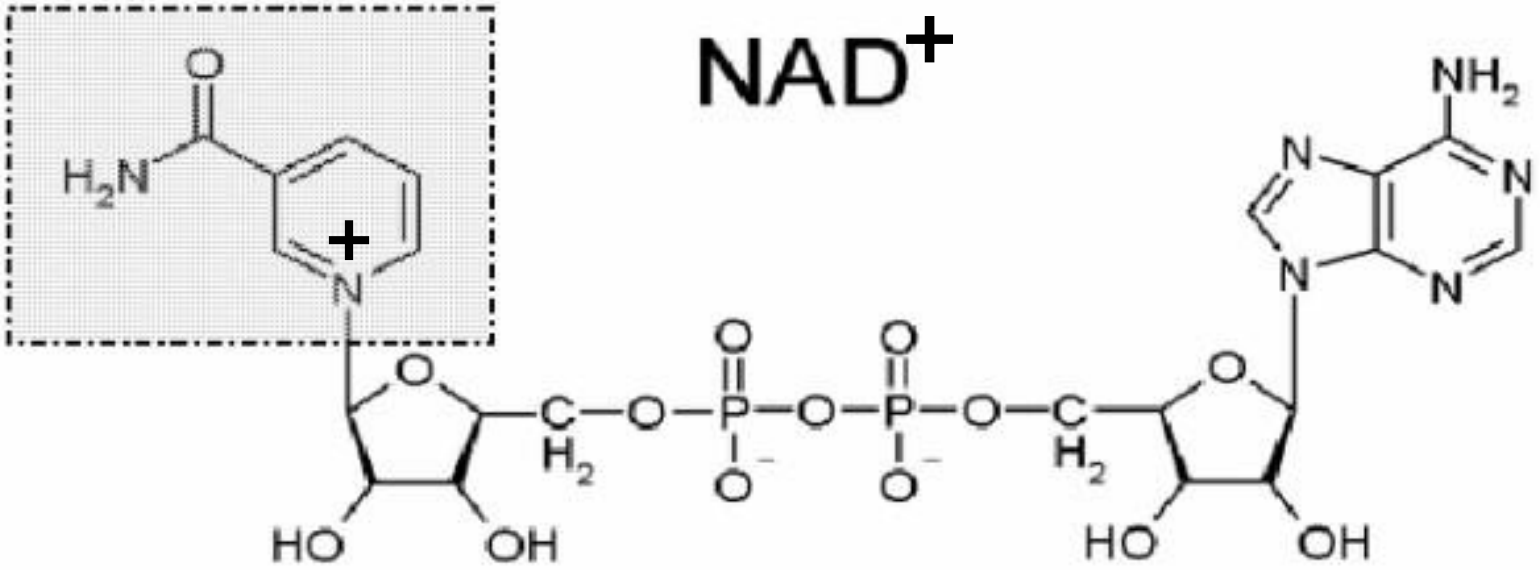


Vit B_2 deficiency Child with angular stomatitis

Niacin (Nicotinic Acid) (B_3)
Pellagra Preventing Factor (PPF)



Pyridine ring


NAD+
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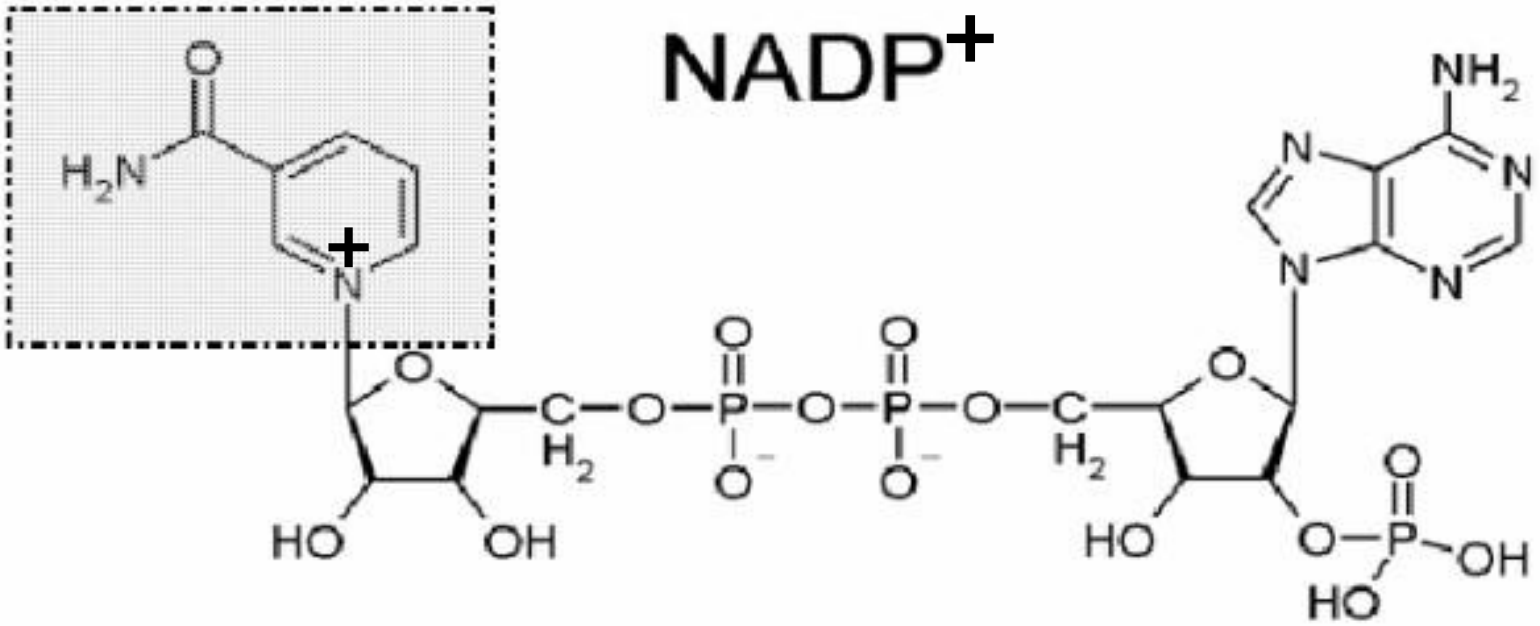
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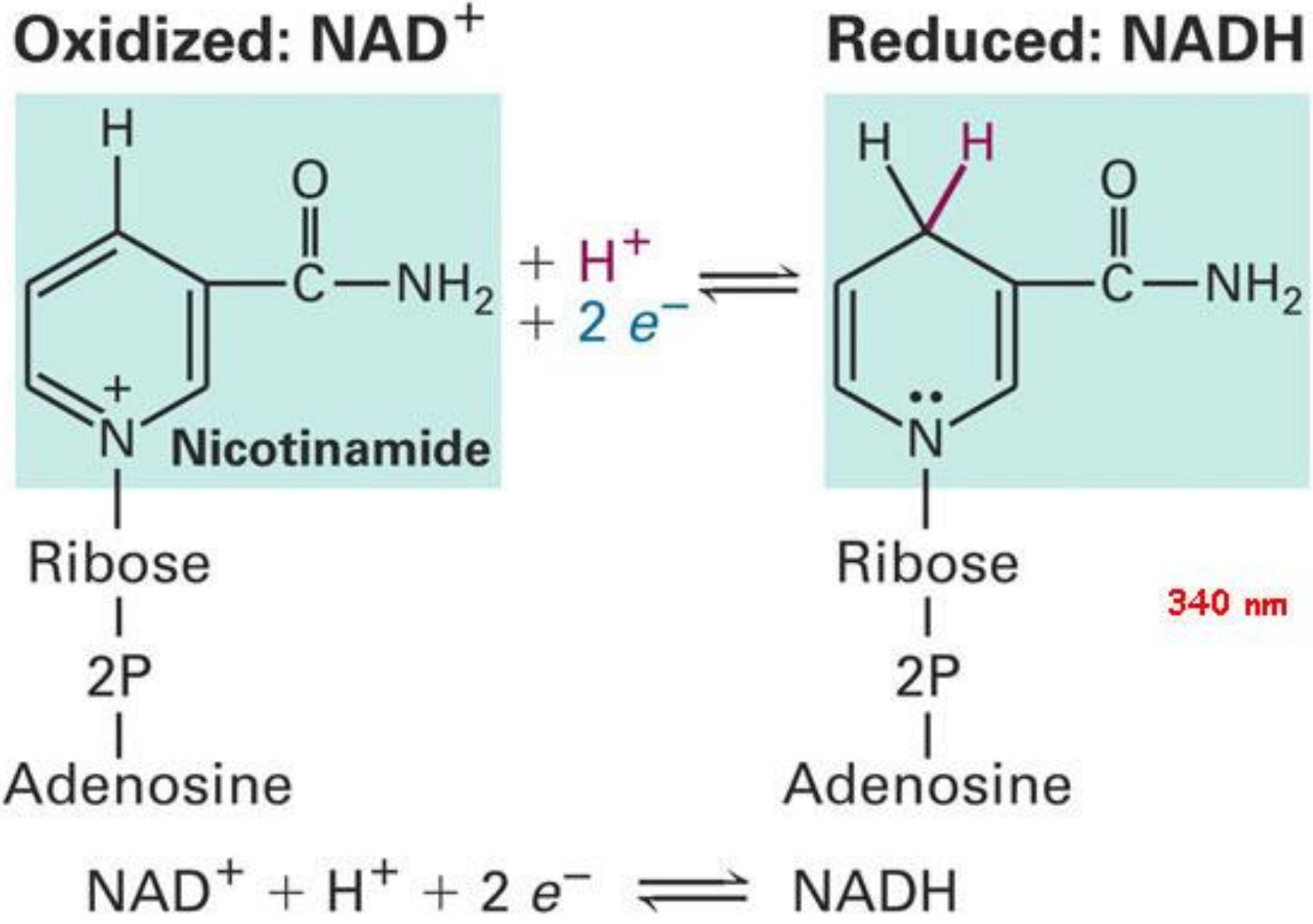
Biosynthesis

- Nicotinic acid is synthesized in the body from tryptophan (about 1 mg of nicotinic acid is formed from 60 mg of tryptophan). So diet deficient in tryptophan will result in deficiency of nicotinic acid

Function

1. Nicotinic acid is a precursor of nicotinamide from which certain coenzymes are produced; coenzymes I, II and III.
2. Coenzyme I and II help a large member of dehydrogenases. So these coenzymes act as hydrogen carriers. Nicotinic acid and its amide are stimulant for C.N.S.
3. Nicotinic acid is a vasodilator.
4. Nicotinic acid is used in treatment of hyperlipidemia.


Oxidized: NAD+
Reduced: NADH
Nicotinamide
+ H+
+ 2 e−
Ribose
2P
Adenosine
Ribose
2P
Adenosine
340 nm
NAD+ + H+ + 2 e− NADH

Deficiency

- Deficiency of niacin causes Pellagra, a disease involving the skin, gastrointestinal tract and central nervous system
- The symptoms of pellagra progress through the three **Ds**:
- **D**ermatitis (inflammation of skin).
- **D**iarrhea (GIT symptoms)
- **D**ementia (loss of mental power) and, if untreated, death.

Pyridoxine

(Anti Rat -Dermatitis Factor, B_6)

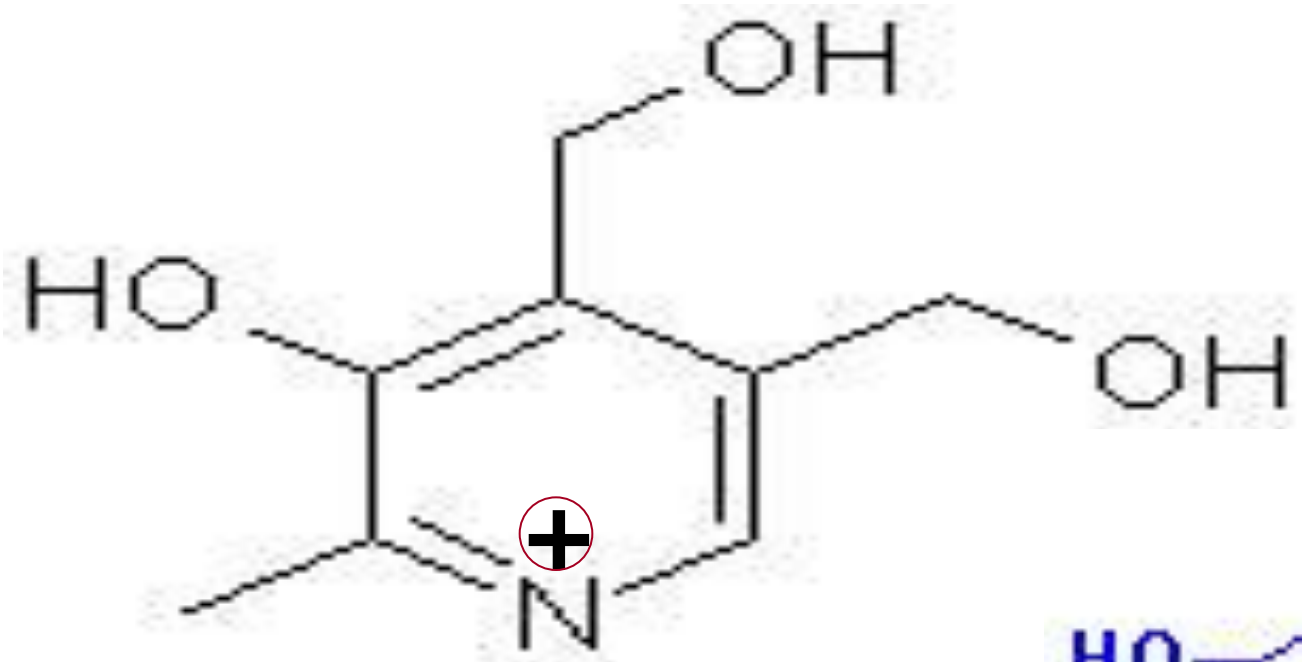
1- Pyridoxol

2- Pyridoxal

3-Pyridoxamine

Pyridoxine (Anti-Dermatitis Factor, B_6)

1- Pyridoxol



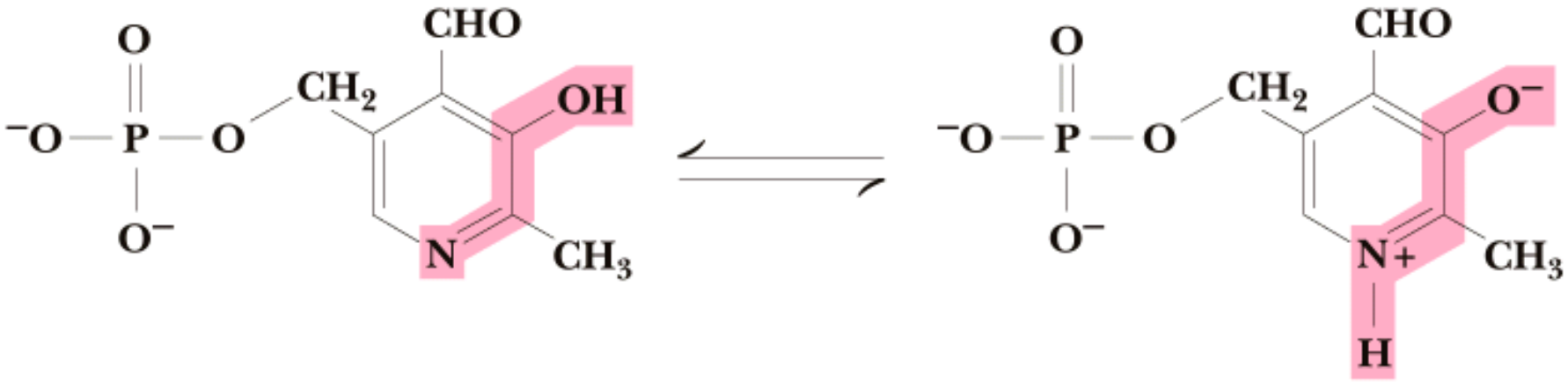
Plant sources only

2- Pyridoxal

Animal sources only

3- Pyridoxamine

Animal sources only

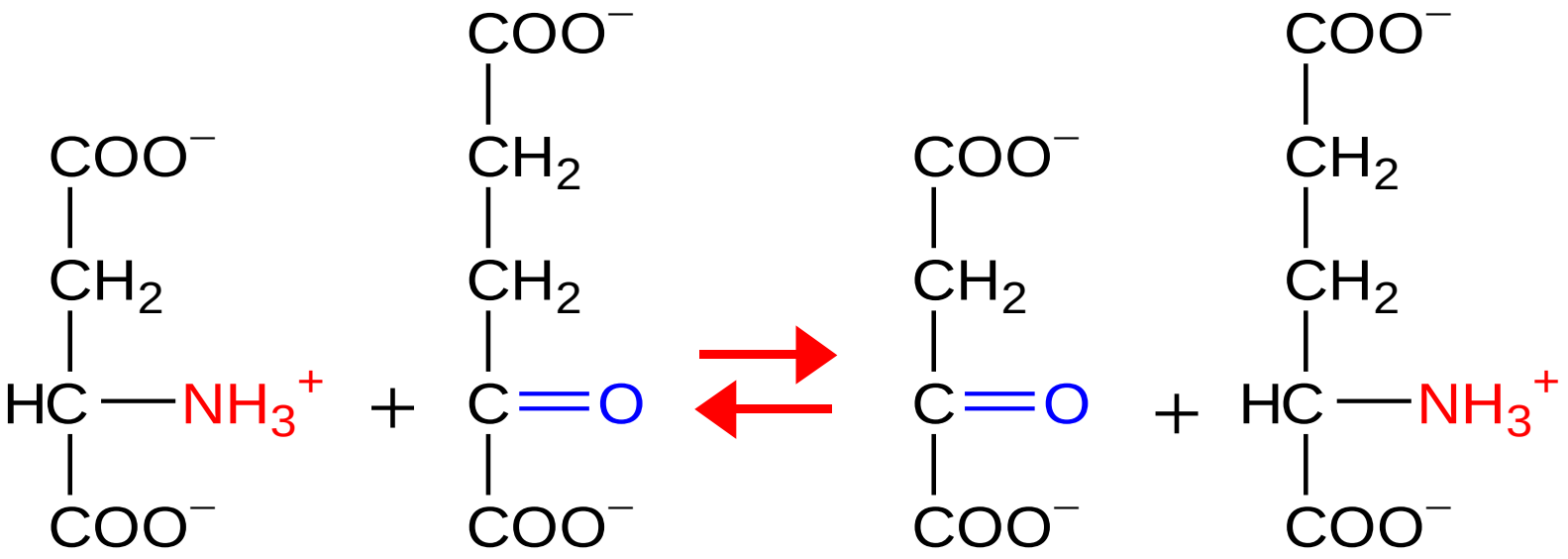


Pyridoxal Phosphate

Pyridoxal Phosphate (Pyr - P)

It is used mainly in amino acid metabolism:

1.Transamination:



Aminotransferase (Transaminase)

2.Deamination:

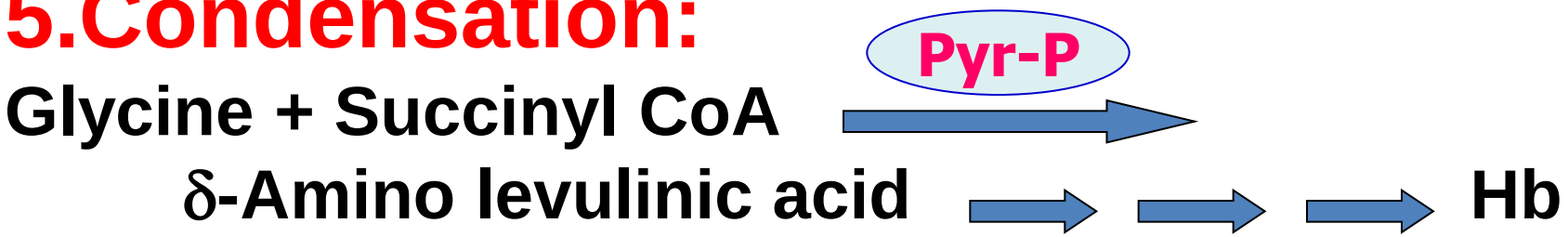
Serine —(Pyr-P; Serine deaminase)→ Pyruvate + NH_3

3.Decarboxylation:

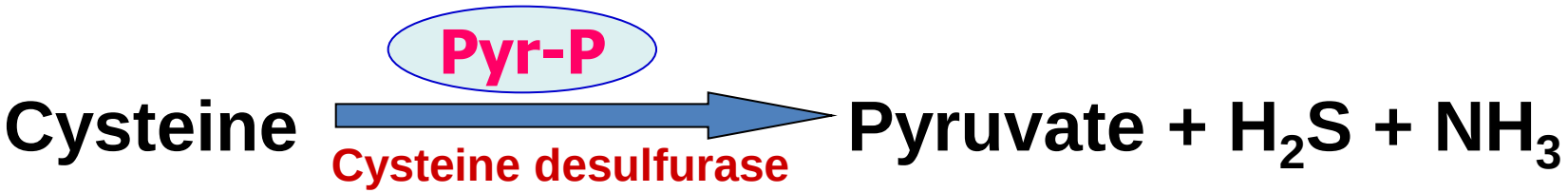
- Dopa —(Pyr-P; Dopa decarbolyase)→ Dopamine + CO_2
- Histidine —(Pyr-P; Histidine decarbolyase)→ Histamine + CO_2
- Glutamic acid —(Pyr-P; γ-Glutamate decarbolyase)→ γ-Aminobutyrate (GABA) + CO_2

4.Niacin synthesis from Tryptophan:

5.Condensation:



6.Desulfuration:



7.Trans-Sulfuration:

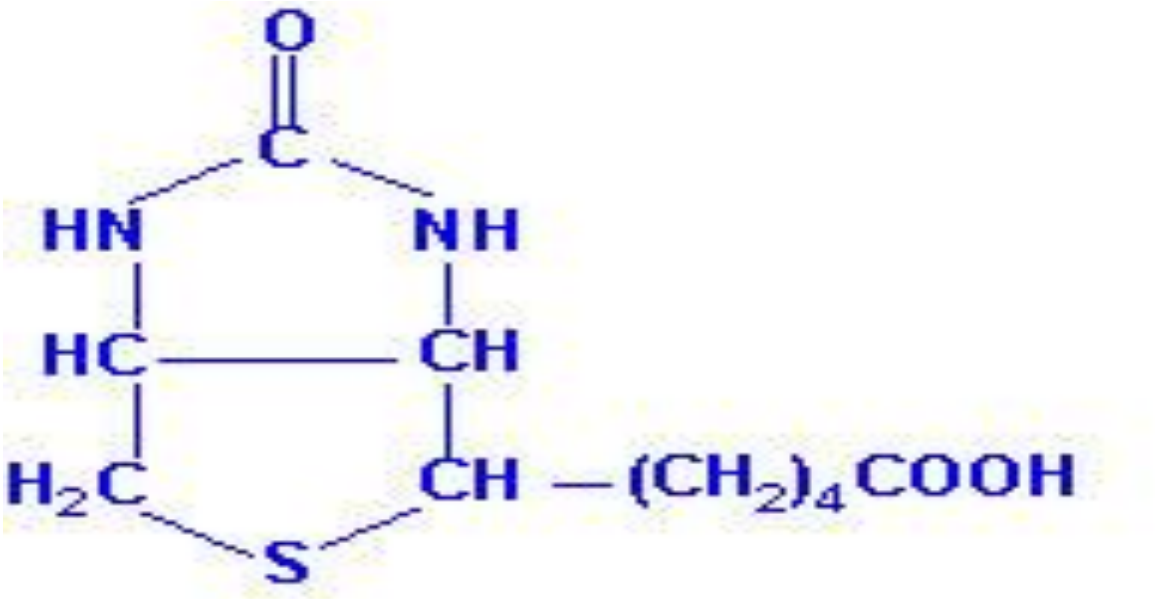
Causes of Deficiency of Pyridoxine

1. Isoniazide an antibiotic drug
2. Taking oral contraceptives
3. In Alcoholism
4. During Pregnancy
5. Low vitamin–intake

Deficiency of Pyridoxine

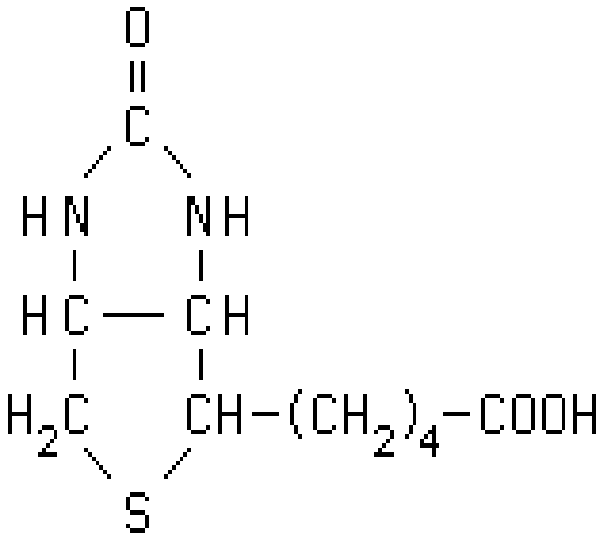
1. **Impaired growth, due to disturbed amino acid metabolism**
2. **Anemia, due to decreased synthesis of Hb**
3. **Convulsions (in infants) due to decreased GABA (γ–Amino butyrate) in brain**
4. **Pellagra, since it is required for niacin synthesis**
5. **Nausea & vomiting in early pregnancy**
6. **In Rats, lead to Dermatitis (Anti-Dermatitis Factor)**

Biotin (Vit H, B_7)

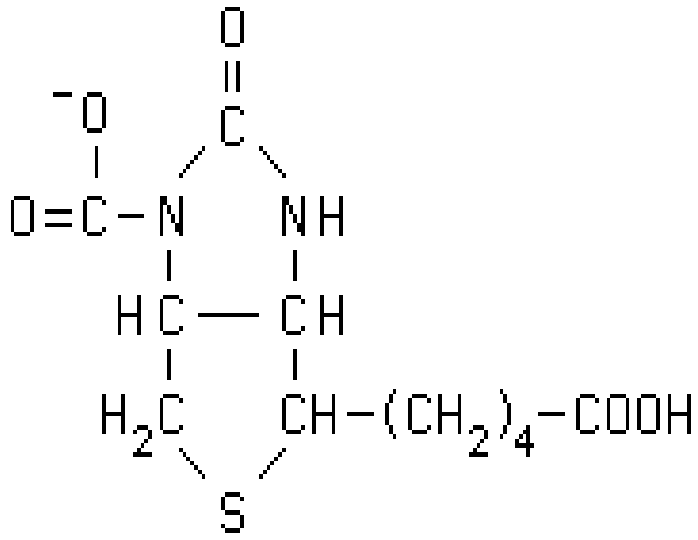


- **is used as cofactor in carboxylation reactions**

Biotin Carboxy Carrier Protein (BCCP)



Biotin



CO_2-biotin

Biotin functions as a prosthetic group. It binds to enzymes by forming an amide link to an amino group of a lysyl residue.

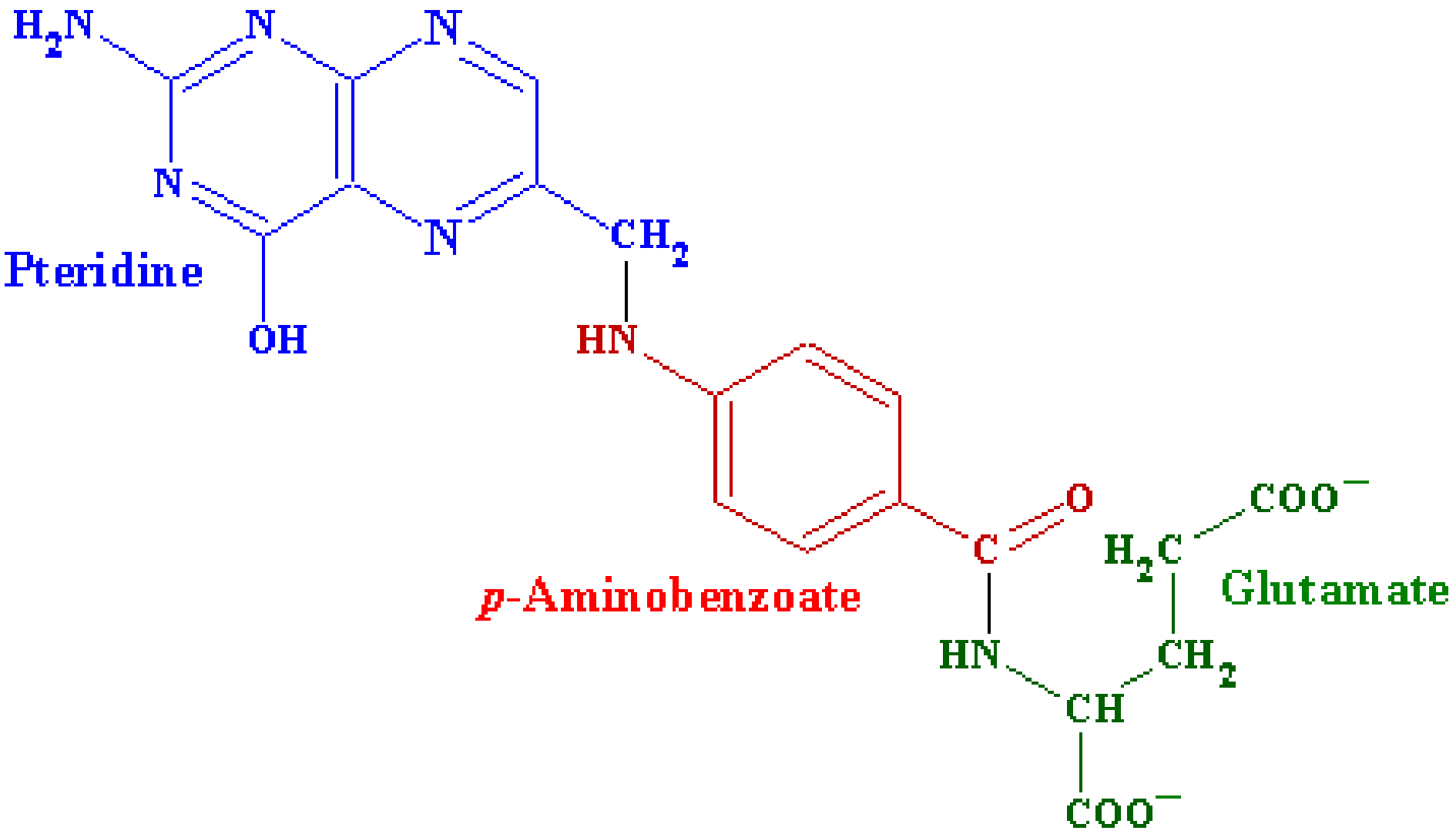
- **Biocytin (BCCP) is the coenzyme in Carboxylation Reactions**

- **Pyruvic acid** → **Oxaloacetate**
- **Acetyl CoA** → **Malonyl CoA**
- **Propionyl CoA** → **D–Methylmalonyl CoA**

1. **Biotin is widely distributed in food**

2. **Biotin is supplied by intestinal bacteria**

Folic Acid (B_{10})



Active Form of Folic Acid (Folinic Acid)

- **The biologically active form is Tetra Hydro Folic Acid (THFA) produced by 2-step reduction by Dihydrofolate reductase**

Function of Folinic Acid (THFA)

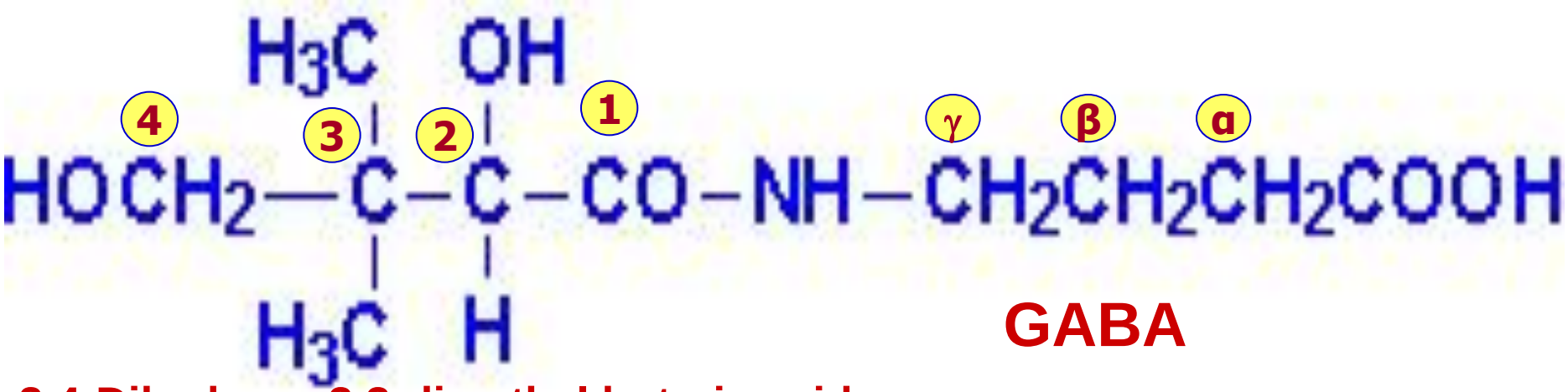
- THFA receives one-carbon fragments from donors such as serine, glycine, histidine & choline and transfer them to a methyl acceptors

- **Methyl acceptors such as:**
  - Homocysteine → Methionine
  - Norepinephrine → Epinephrine
  - Ethanolamine → Choline
  - Guanidoacetic acid → Creatine
  - UTP → TTP

Deficiency of Folic Acid

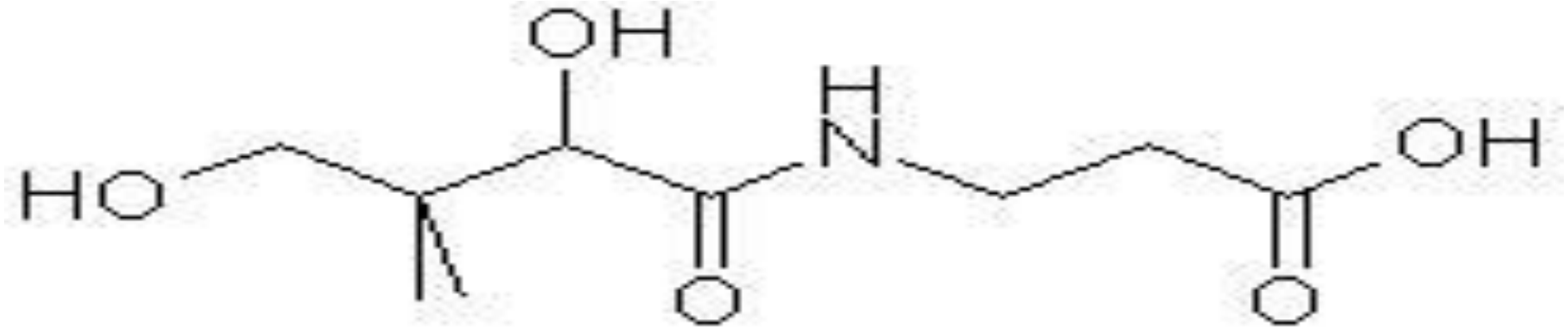
- **Growth failure**
- **Megaloblastic anemia as for vitamin B_{12} deficiency**
- **The inability to synthesize DNA during erythrocyte maturation leads to abnormally large erythrocytes termed macrocytic anemia**
- **Macrocytic anemia (Decreased No of RBCs with increased size)**
- **Leukocytic anemia (decreased No of WBCs)**

Pantothenic Acid (vit. B5)



2,4-Dihydroxy-3,3-dimethyl butyric acid

(Pantoic acid)



Function of Pantothenic Acid

1. Formation of Co A – SH
2. Component of fatty acid synthase enzyme

Function of Coenzyme A – SH

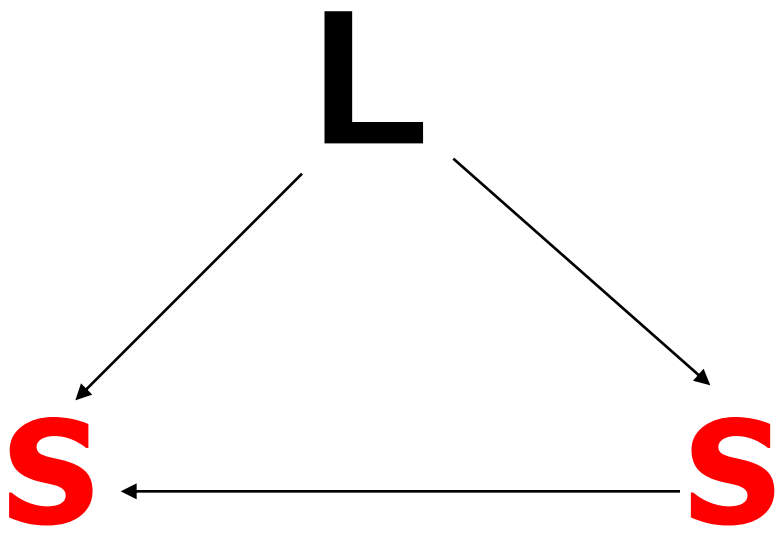
1. Formation of Active Acetate (Acetyl CoA) used in:
   - A. Fatty acid synthesis
   - B. Ketone bodies synthesis
   - C. Cholesterol synthesis
   - D. Adrenal cortical hormones synthesis
   - E. Acetylcholine synthesis
   - F. Oxidation of carbohydrates & lipids (through Kreb's Cycle)
2. Formation of Succinyl CoA

Deficiency

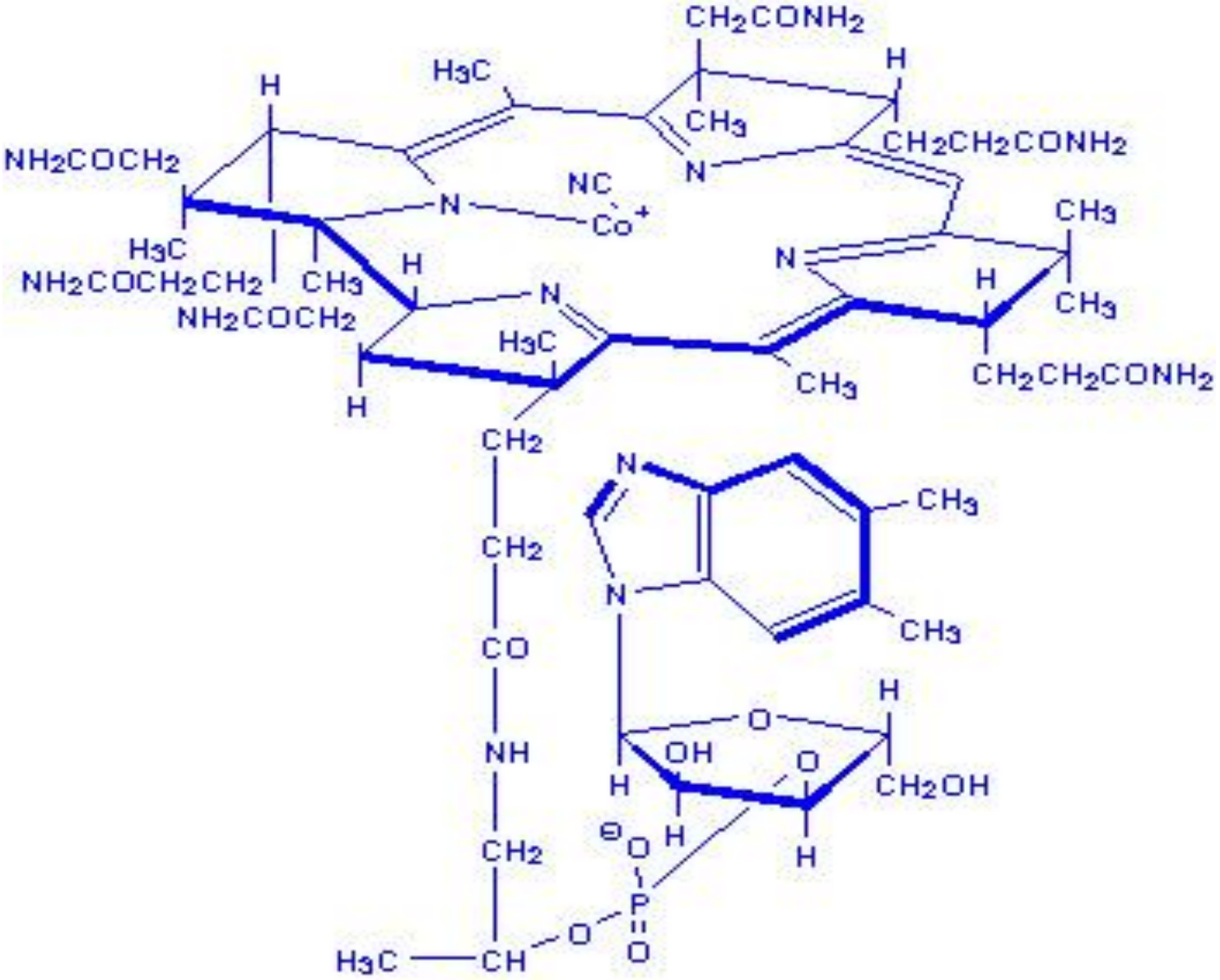
- No deficiency in human.

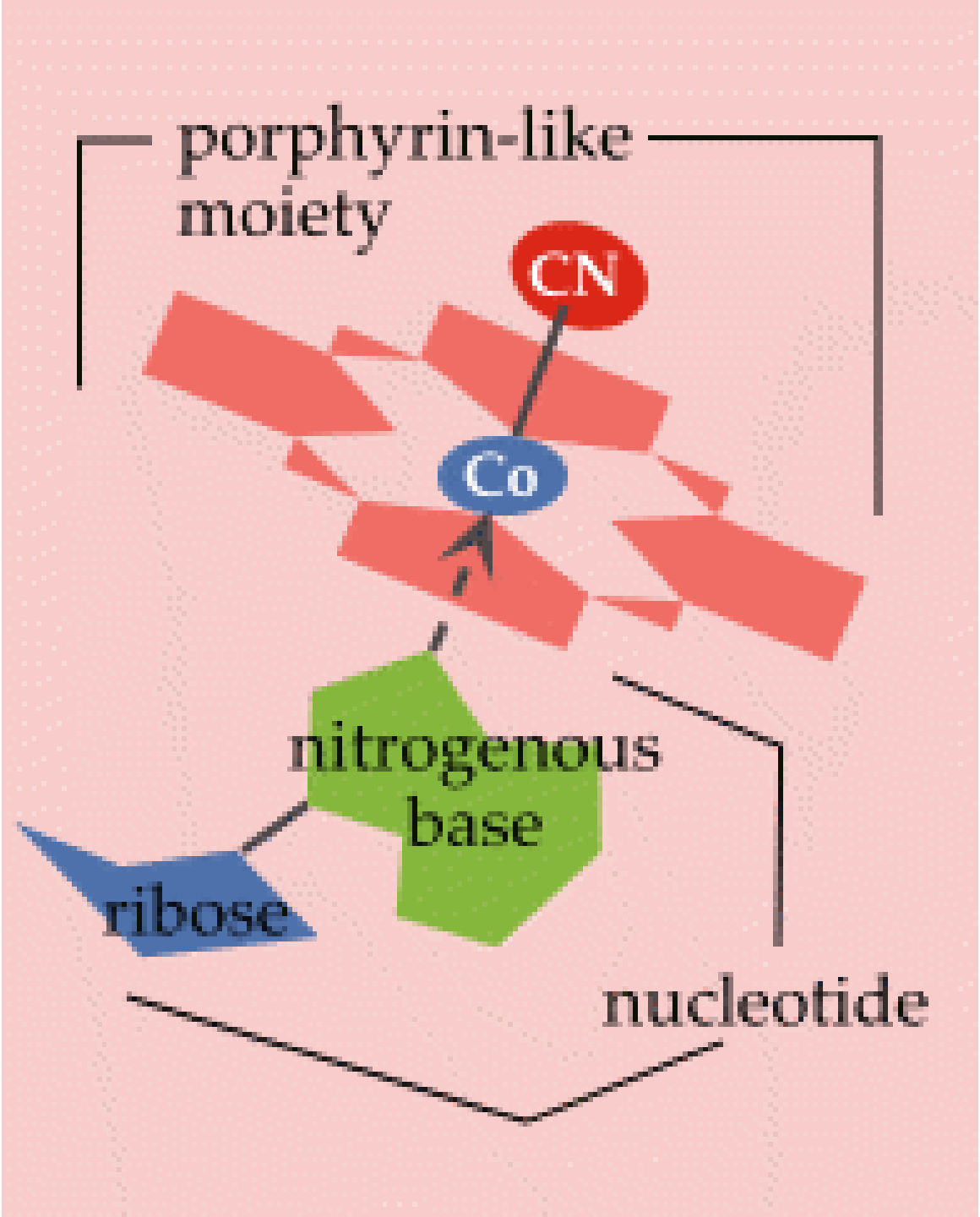
Lipoic Acid

1. Used as **hydrogen carrier** in oxidation-reduction reactions

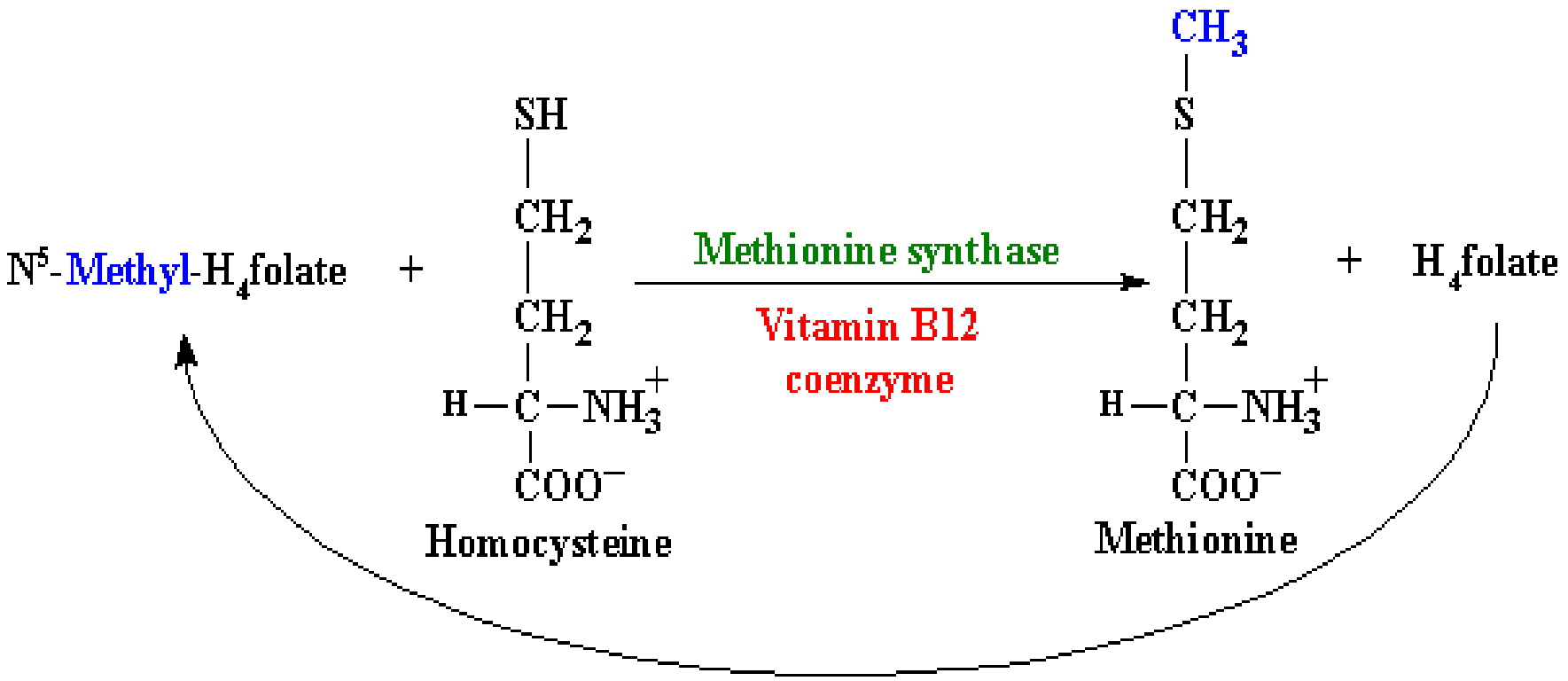


Cyanocobalamin (B_{12})



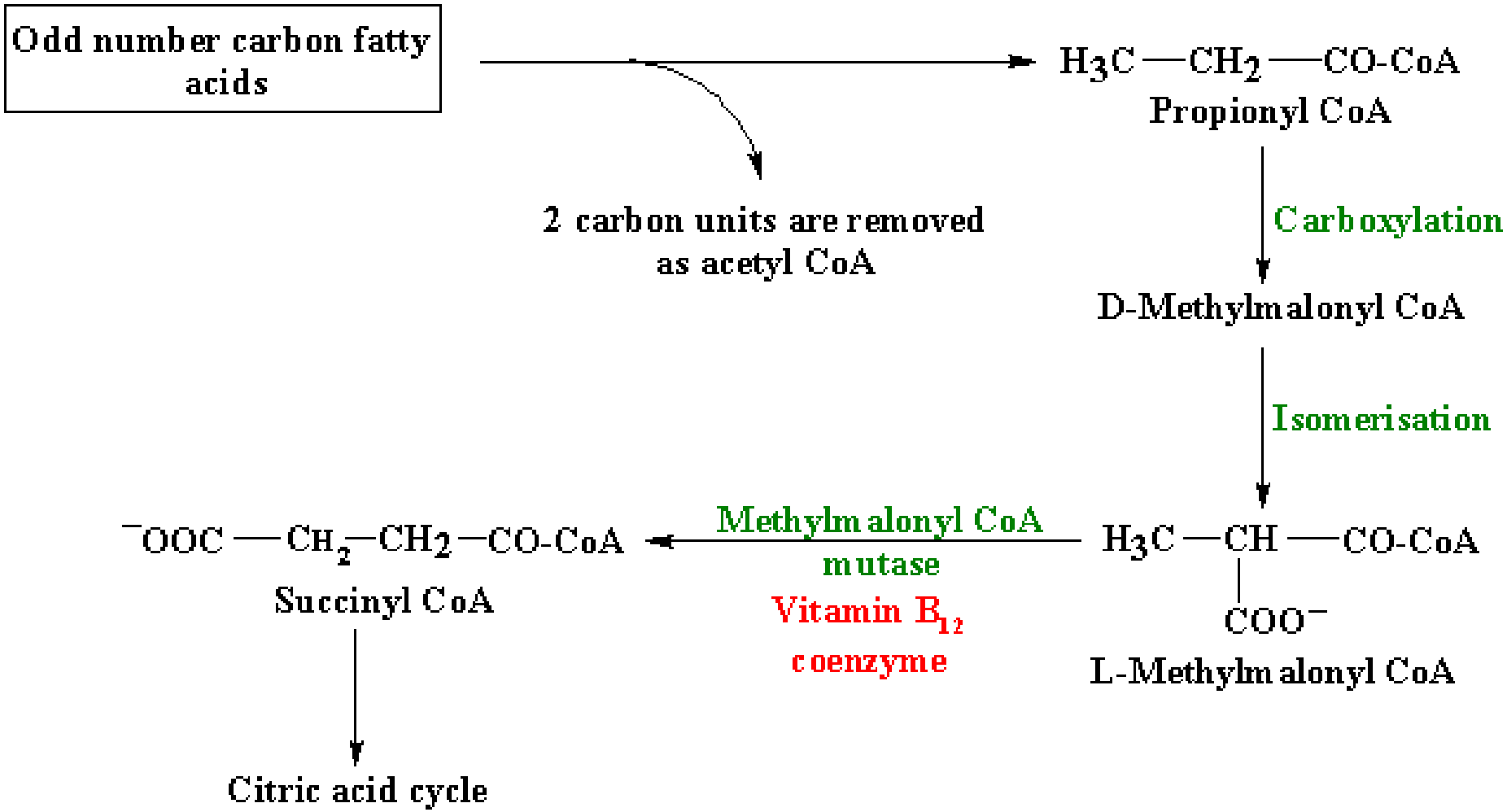

porphyrin-like
moiety
CN
Co
nitrogenous
base
ribose
nucleotide

Synthesis of Methionine from Homocysteine requires Methylcobalamin coenzyme

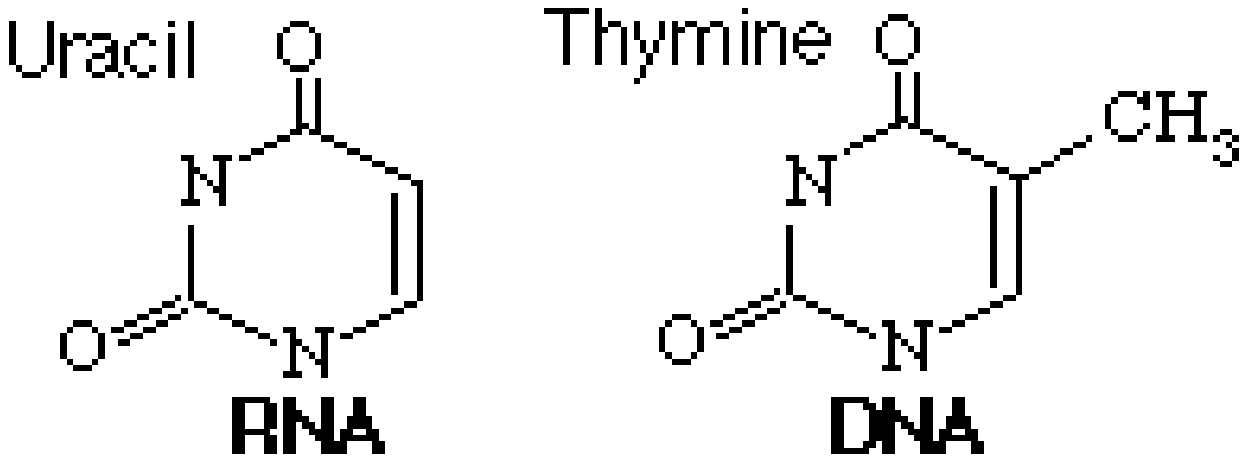


H_4folate accepts methyl groups in a number of different reactions an is converted back to N^5-Methyl-H_4folate

Rearrangement of L–Methyl malonyl CoA into Succinyl CoA requires 5′-Deoxyadenosylcobalamin coenzyme

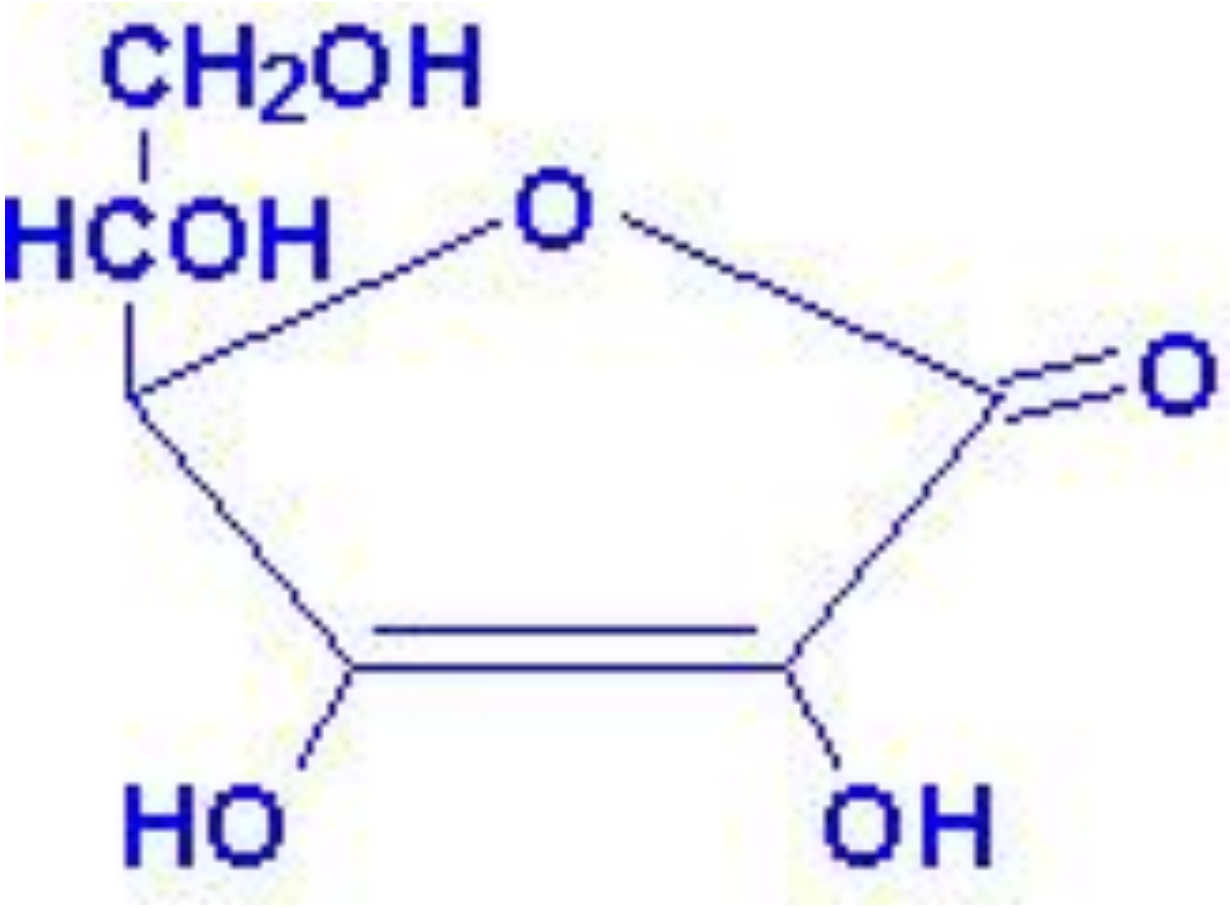


- Distribution of Vitamin B_{12}:
- it is not present in plants
- It is synthesized by microorganisms
- Deficiency of Vitamin B_{12}:
- The RDA is 3 μg/day
- 6 μg/day is required in pregnancy & lactation
- It can be stored in significant amounts (4 – 5 mg)
- It may take several years for the clinical symptoms of Vit. B_{12} deficiency
- Vit. B_{12} deficiency is rarely due to a lack of the vitamin in the diet
- Vit. B_{12} deficiency is more common due to failure of absorption of the vitamin



- **Vitamin B_{12} Deficiency:**
  - **Megaloblastic anemia (Due to deficiency of purine & pyrimidine synthesis)**
  - **Degeneration of nerve fibers in the spinal cord & peripheral nerves**

VITAMIN C (L-ASCORBIC ACID)



It is a reducing agent (requires Oxygen & metal)

SOURCE:

1)Animals:

E.G. LIVER, ADRENALS AND MILK

2)Plants:

E.G. LEAFY VEGETABLES, GREEN PEAS AND BEANS, GERMINATED SEEDS, GREEN AND RED PEPPERS, TOMATOES AND CITREOUS FRUITS

- **METABOLISM OF VITAMIN C:**

≥ No synthesis in man

≥ Biosynthesis of ascorbic acid occurs in certain animals e.g. rat

≥ Synthesis occurs from D–glucose

≥ Catabolism to oxalic and L-threonic acids

- **EXCRETION OF VITAMIN C:**
- is excreted in urine
- Also excreted in milk in active form

FUNCTIONS OF VITAMIN C

- Vitamin C is required for tyrosine & tryptophan metabolism when usually large quantities of tyrosine is being ingested
- Vitamin C is needed for Hydroxylation of proline & lysine required for normal formation of fibroblasts (Collagen) and osteoblasts
- So, vitamin C accelerates the healing of wounds and fractures of bones

FUNCTIONS OF VITAMIN C

- Vitamin C influences the biosynthesis of **adrenal cortical hormones** as it is present in high concentration in the adrenal cortex
- Vitamin C is necessary for the activation of **Folic acid** into folinic acid
- Vitamin C is used for **mobilization of iron** from its stored form (ferritin) as it helps the reduction of ferric ion to ferrous ion

DEFICIENCY OF VITAMIN C

- Scurvy resulting from failure of normal deposition of collagen, osteoid and dentin as a result of failure of normal formation of fibroblasts , osteoblasts and odontoblasts
- Scurvy is manifested by:
  - Haemorrhage from gums
  - loose teeth and fractures
  - loss of appetite and
  - lastly loss of weight

≥ EFFECTS OF EXCESS VITAMIN C:

- Chronic massive doses of Vit. C causes Kidney stones due to oxalate formation

Fat-Soluble Vitamins

1. **Vitamins A (Retinol)**
   - **Beta Carotene**
2. **Vitamins D (Calciferol)**
3. **Vitamins E (Tocopherol)**
4. **Vitamins K (Phylloquinone)**

Vitamins A (Retinol)

Carotenes

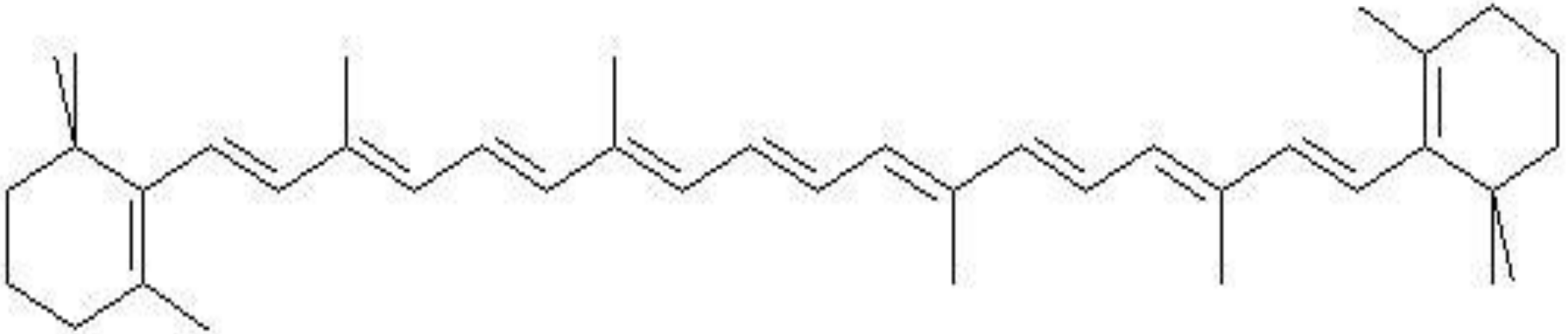
Vitamins A

WHAT IS THE CHEMISTRY OF VITAMIN A?

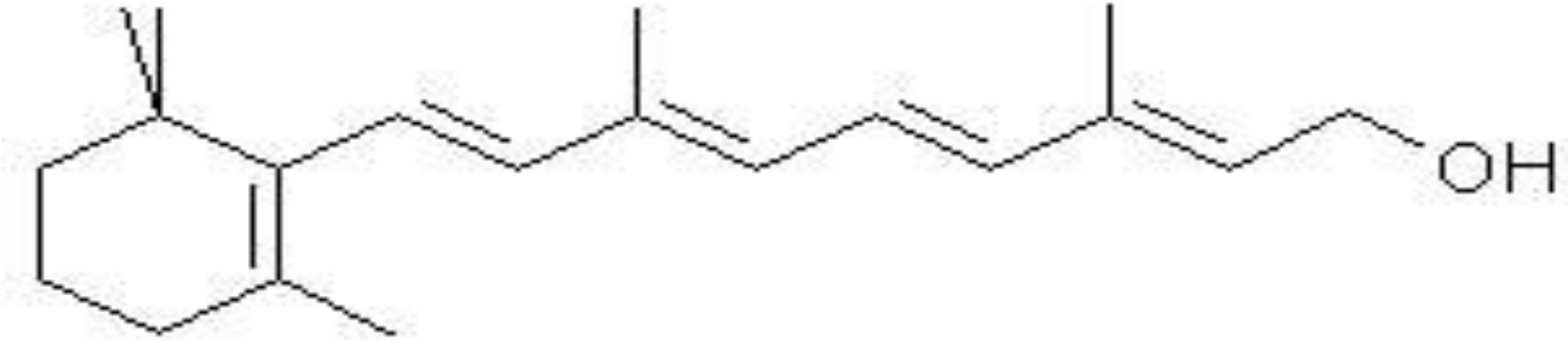
- Vitamin A is a derivative of certain carotenoids which are hydrocarbon pigments (yellow or red) widely distributed in nature
- Carotene (Provitamin A) —**Carotene Oxygenase**→ Vit. A
- All carotenes are formed from 2 rings A and B connected together by 18 carbon atoms
- Vitamin A occurs in nature in two forms

  * Vitamin A_1, Vitamin A_2 and Vitamin A_3

β-Carotenes



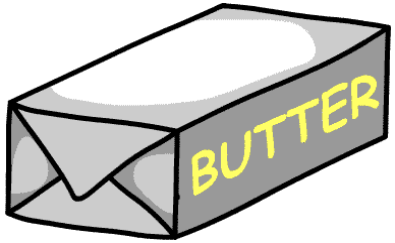
Vitamin A (Retinol)



Properties:

- Vitamin A is insoluble in water and soluble in fat solvents e.g. ether, acetone
- The biological activity of vitamin A is lost by:
  - Exposure to ordinary light
  - Ultraviolet rays
  - Oxidation

SOURCES



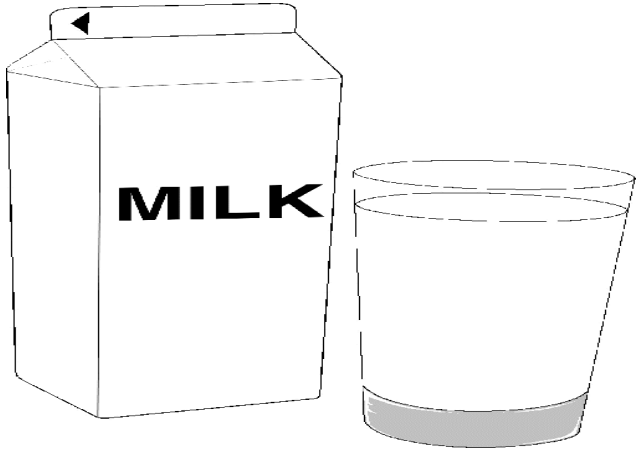
- *Vit A is present only in animals .*

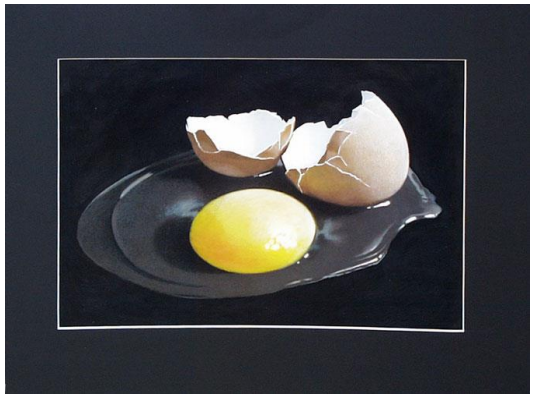
❄ *Milk*

❄ *Butter*

❄ *Egg yolk*

❄ *Livers*





❄ ***Livers of certain fishes contain higher concentration of vitamin A (Cod Liver Oil)***

The best sources of Vitamin A

- Storage of vit A:

95% of vitamin A stored in the liver as

ESTER FORM

The remaining 5% is present in:

Adrenals

Lactating breast

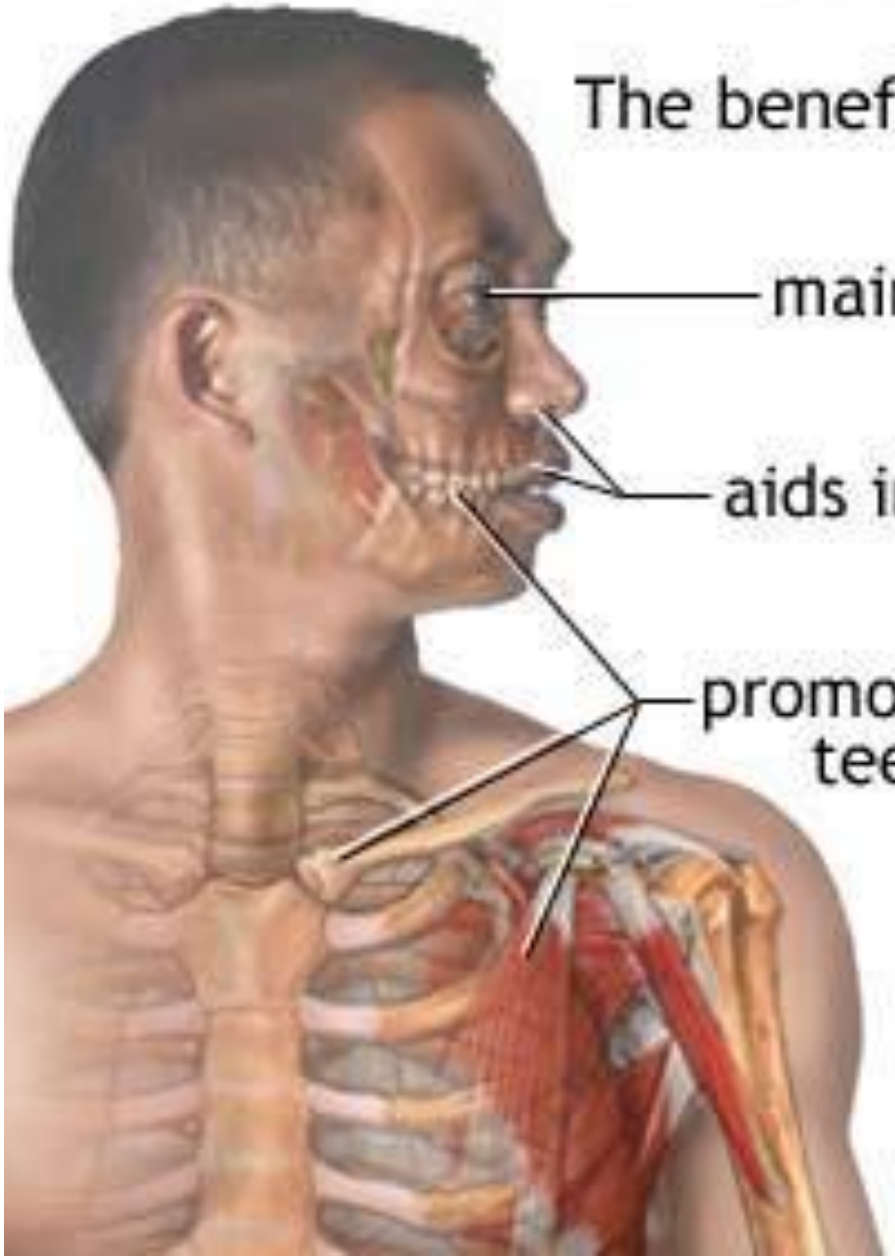
Lung and intestine

- **EXCRETION of Vitamin A:**
- *Vitamin A is excreted by:*
  - Urine
  - Stool
  - Colostrum
  - Sebacious glands of skin

FUNCTIONS:

1. Responsible for visual process
2. Necessary for normal function of the adrenal cortex
3. Maintenance of healthy epithelial tissue
4. Responsible for normal construction of bone & teeth
5. Responsible for reproduction especially in animals

Vitamin A



The benefits of vitamin A:

- maintains health of specialized tissues such as the retina
- aids in growth and health of skin and mucous membranes
- promotes normal development of teeth, soft and skeletal tissue

Adult RDA: 1000 µg RE

DEFICIENCY OF VITAMIN A

- In eyes:

a) Night blindness

b) Dry cornea

DEFICIENCY OF VITAMIN A

- In eyes:

  a) Night blindness

  b) Dry cornea

- Adrenal cortex: gluconeogenesis is decreased
- Reproduction: mainly in rats cause decrease reproductive activity
- Inflammation of the skin: The skin becomes rough, scaly and follicular
- Respiratory tract infections: Repeated cough from lowered resistance of its mucosa
- Urinary tract: Infection and stone formation

Toxic Effects of Vit. A

- **Over dose of Vit. A (50,000 IU/day) for long period may lead to hyper–vitaminosis especially in children and cause:**

1. **Loss of appetite**
2. **Loss of weight**
3. **Irritability**
4. **Fissuring of the corners of mouth**
5. **Bleeding of lips**
6. **Hemorrhagic, due to hypo–prothrombinemia**

Toxic Effects of Vit. A

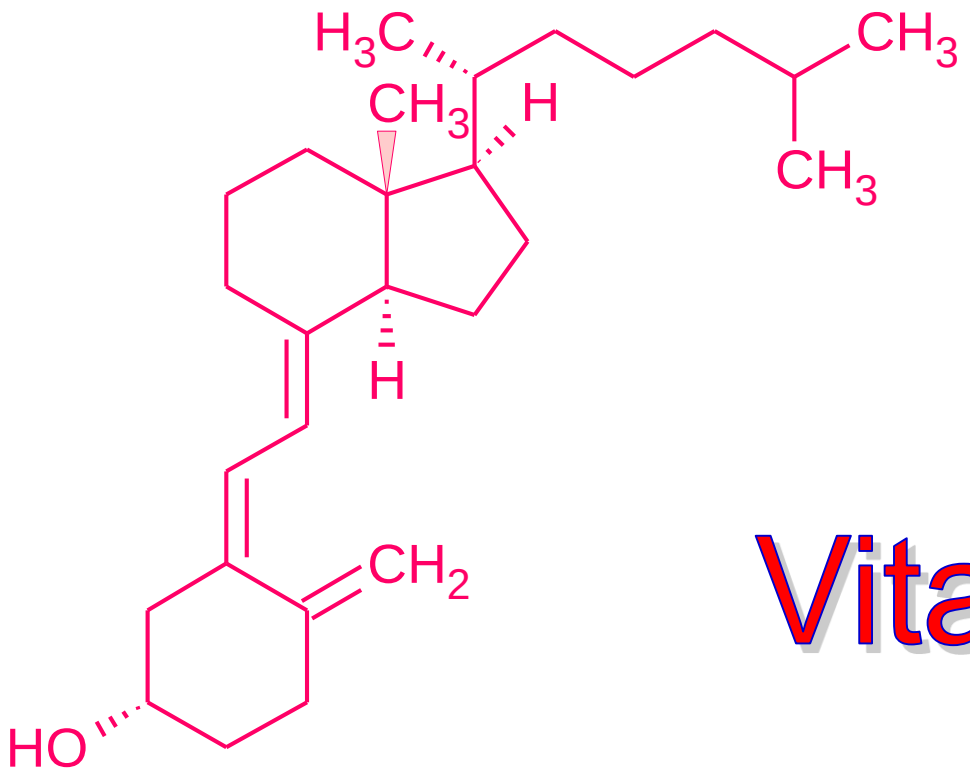
- **Excessive intake of carotene may not be as harmful but may result in yellowish discoloration of skin (carotenemia) that disappears when carotene intake is stopped**

Vitamin A Quick Guide

Alternate name	Retinol (precursor: beta-carotene)
Dietary Reference Intake (DRI)	Adult males: 900 mcg/day Adult females: 700 mcg/day
Recommended intake for athletes	700 to 900 mcg/day
Functions	Maintaining healthy epithelial (surface) cells, eye health, immune system health
Good food sources	Retinol: liver, butter, cheese, egg yolks, fish liver oils Beta-carotene: dark green and brightly pigmented fruits and vegetables
Deficiency	Dry skin, headache, irritability, vomiting, bone pain, night blindness, increased risk of infection, blindness
Toxicity (high toxicity potential)	Tolerable upper limits: 600 to 900 mcg/day for young children (age 1-8) 1.7 to 3.0 mg/day for children and adults (age 9-70+) *Symptoms:* liver damage, bone malformations, death

Vitamins D

- Vitamins D are group of compounds all of them are steroid in nature (sterols) and occur mainly in animals



Vitamin D3

PROPERTIES

- Vitamins D are soluble in fat solvent
- They are resistant to oxidation and
  heat

OCCURANCE AND FOOD SOURCES:

- Fishes mainly in their livers
- Vitamin D itself is widely distributed in animals e.g.
- Cod–liver oil, and tuna –liver oil contain high concentrations of vitamin D_3
- Egg yolk is very rich in vitamin D_3
- Milk is poor source of vitamin D_3

Vitamin D

The body itself makes vitamin D when it is exposed to the sun

Cheese, butter, margarine, fortified milk, fish and fortified cereals are food sources of vitamin D

ABSORPTION

- Irradiation increases its absorption
- Bile salts help its absorption

STORAGE

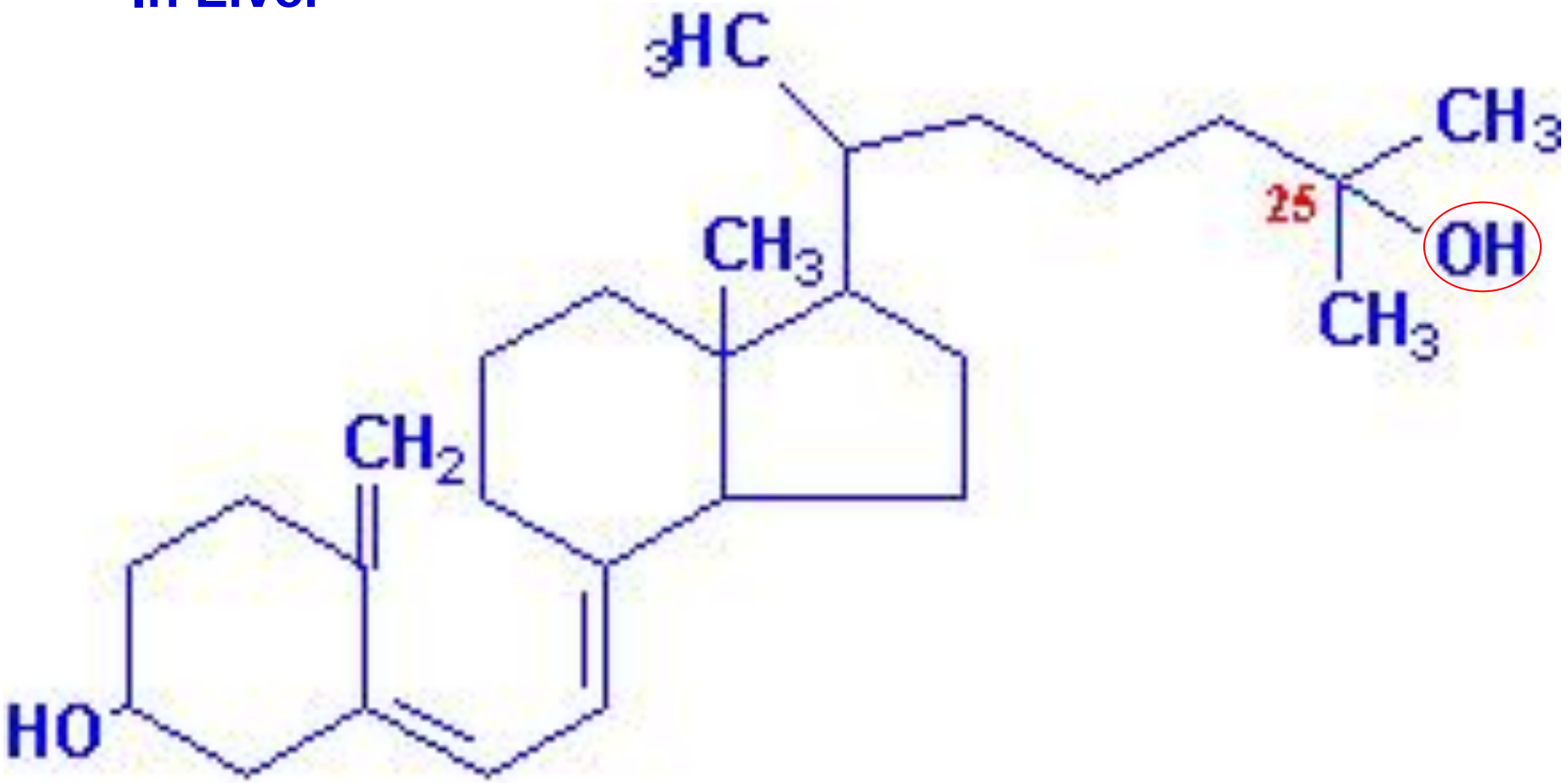
- Skin and brain (significant amounts)
- Lung, spleen and bones (small amounts)

EXCRETION

- By bile to be reabsorbed by small intestine
- By milk
- No excretion in urine

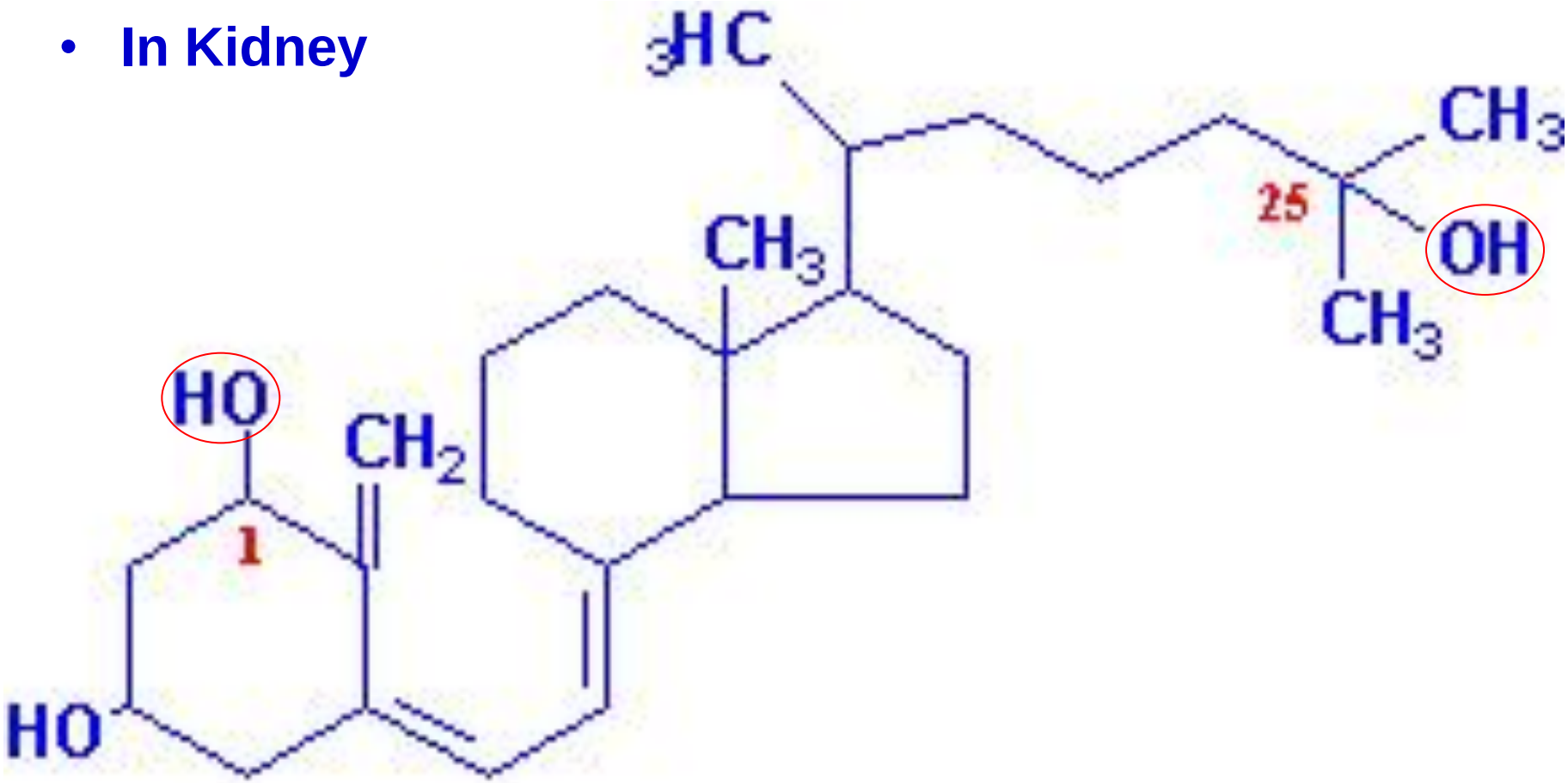
25-Hydroxycholecalciferol

- **In Liver**

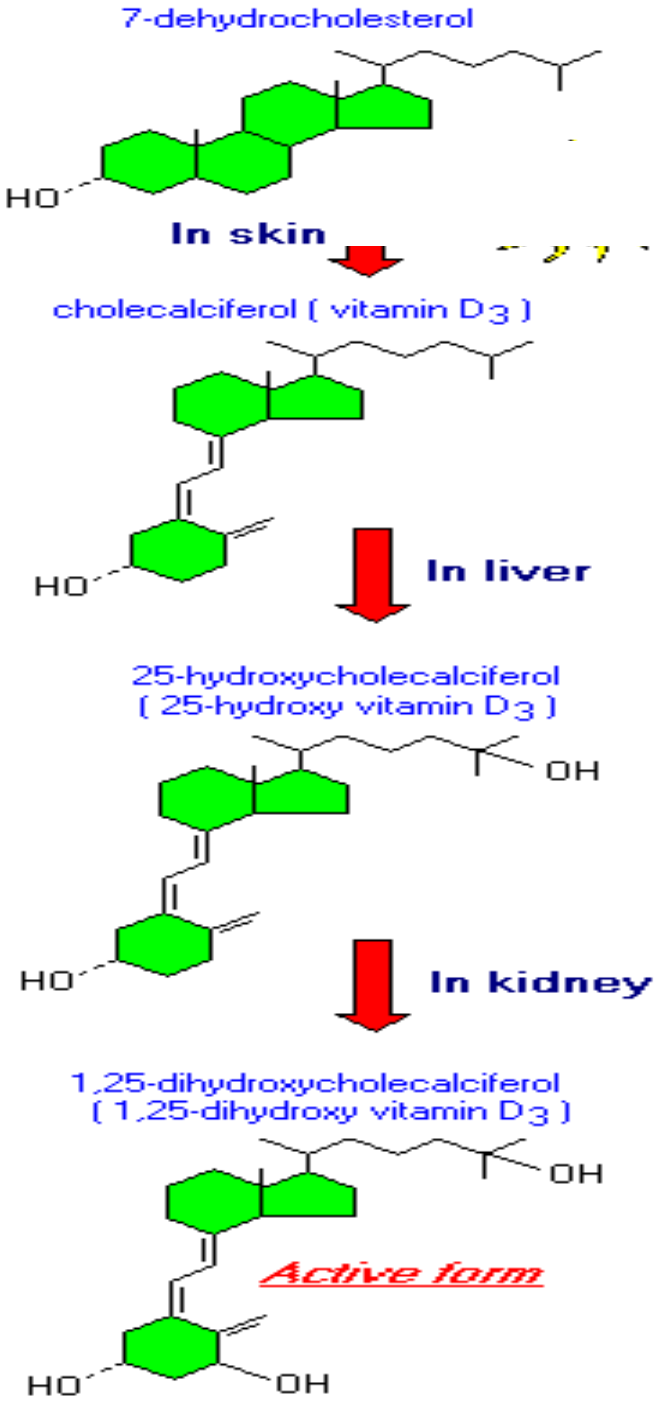


1,25-Diydroxycholecalciferol [1,25–$(OH)_2$]

- **In Kidney**

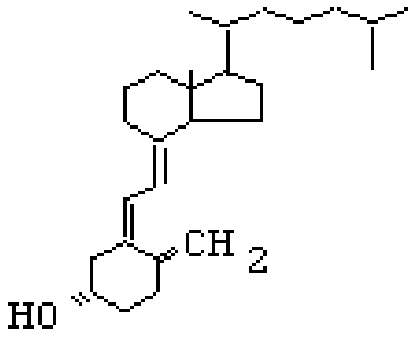


Synthesis of active form of cholecalceferol



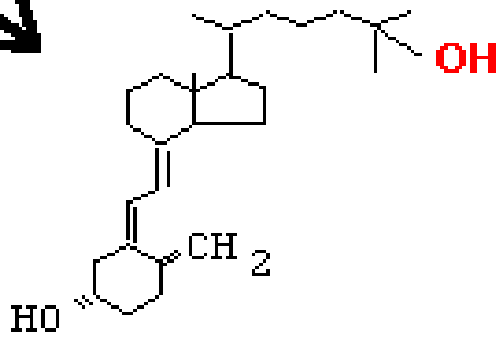
SKIN PRODUCTION

DIET



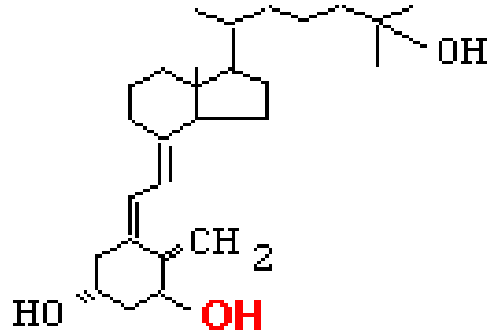
Vitamin D

LIVER

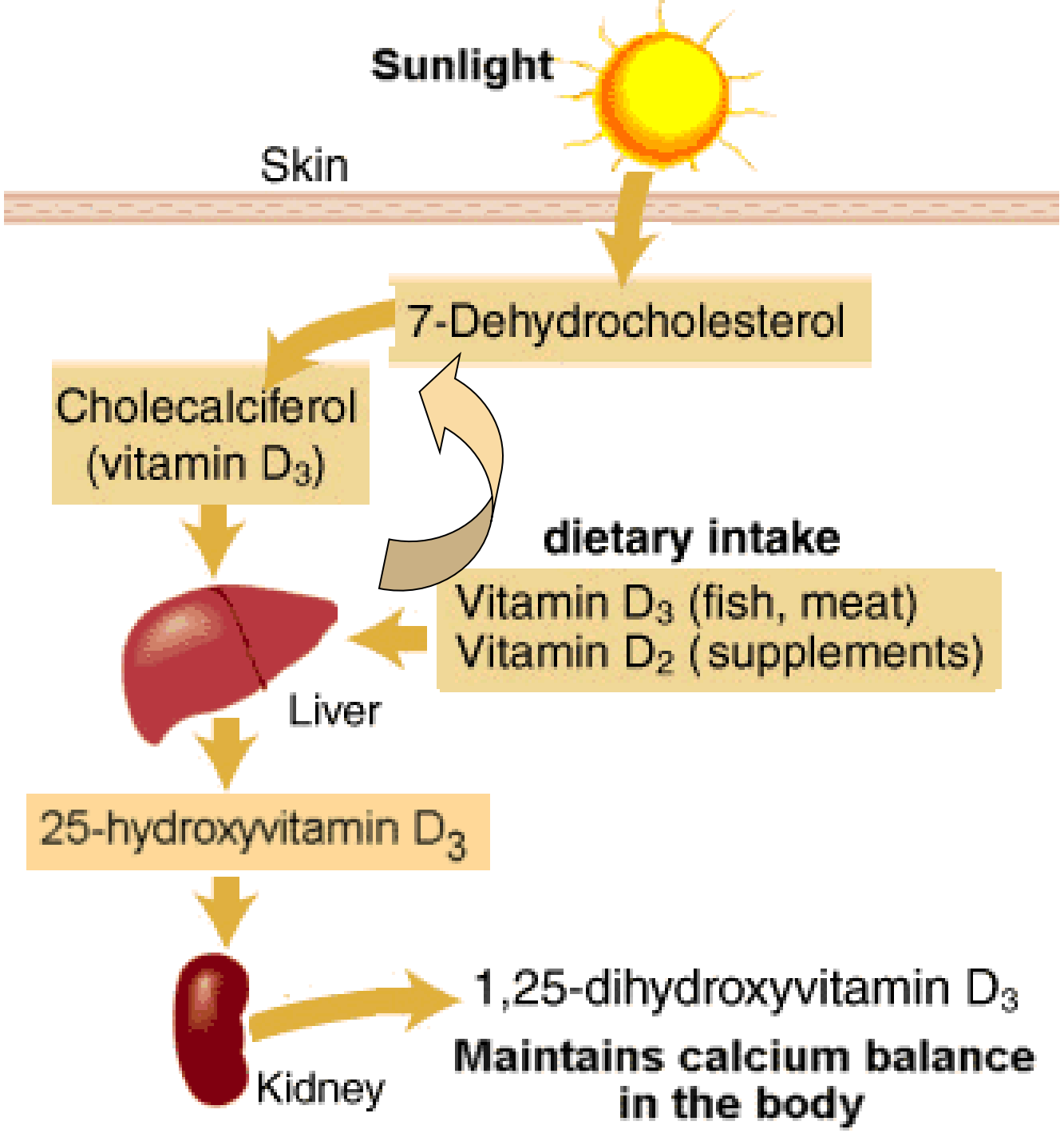


25 (OH)-Vitamin D

KIDNEY



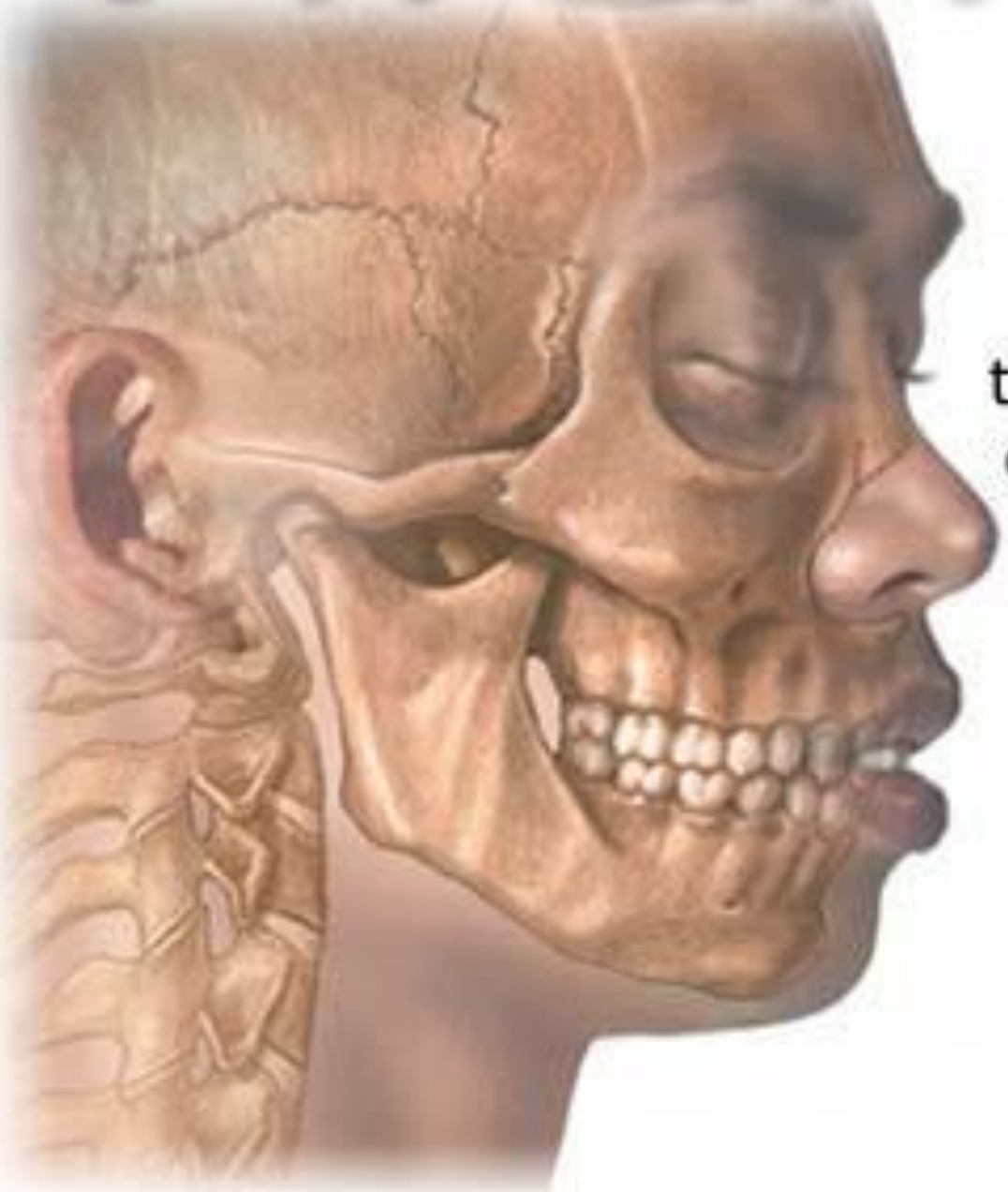
1,25 $(OH)_2$ Vitamin D


Sunlight
Skin
7-Dehydrocholesterol
Cholecalciferol
(vitamin D_3)
dietary intake
Vitamin D_3 (fish, meat)
Vitamin D_2 (supplements)
Liver
25-hydroxyvitamin D_3
Kidney
1,25-dihydroxyvitamin D_3
Maintains calcium balance
in the body

Functions of 1,25-Dihydroxy Cholecalciferol

1. Increases the absorption of calcium
2. Increases the absorption of phosphate
3. Regulates calcium and phosphorus levels in blood (i.e., Minimizes loss of calcium by the kidney)
4. Increases the citrate content of bones, blood, and other tissues as well as its urinary excretion

Vitamin D

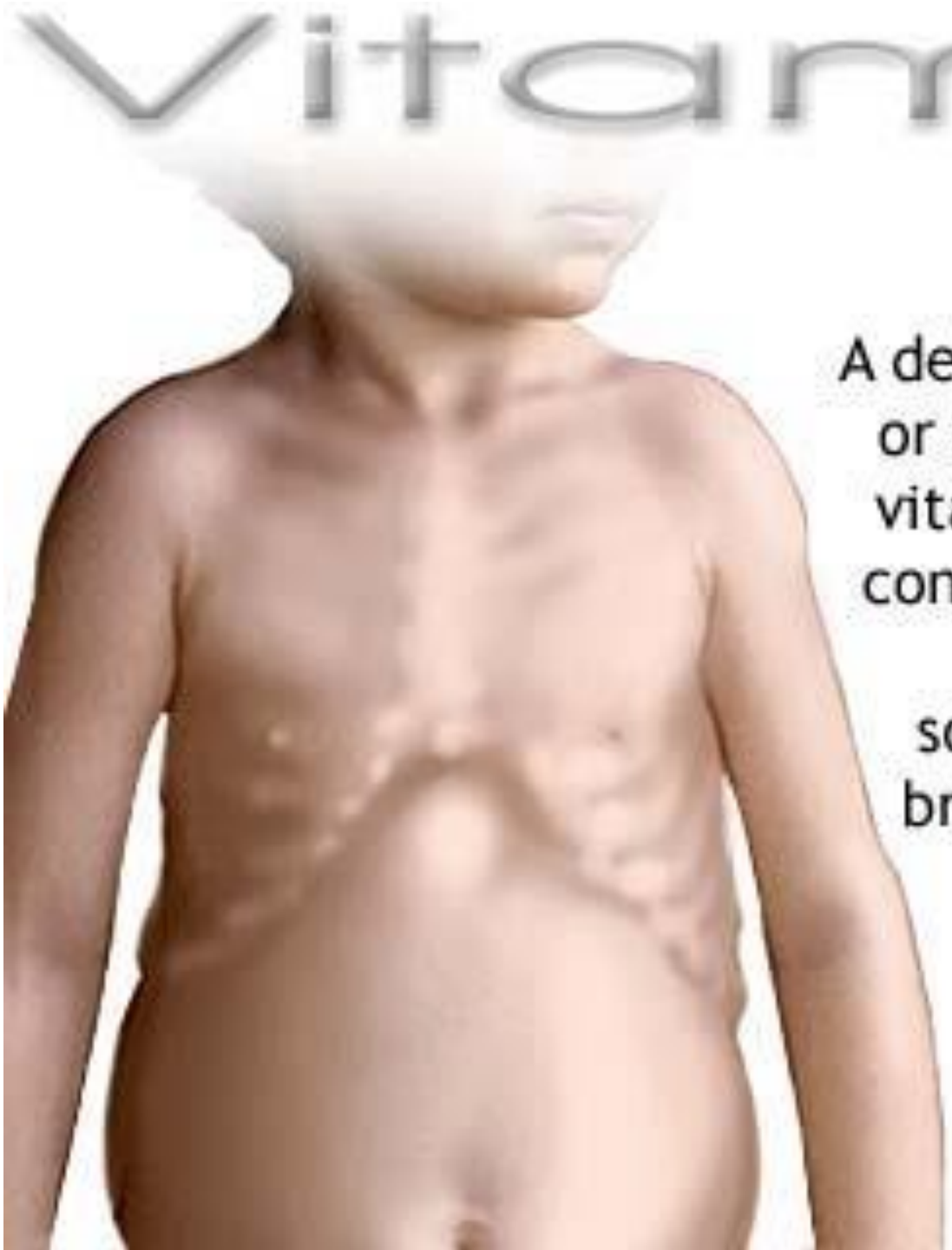


Vitamin D promotes the body's absorption of calcium, essential to development of healthy bones and teeth

DRI: 5 µg

Fat-soluble

Vitamin D



A deficiency of vitamin D or an inability to utilize vitamin D may lead to a condition called rickets, a weakening and softening of the bones brought on by extreme calcium loss

DEFICIENCY:

1. Bony manifestations:

- Rickets in children
- Osteomalacia in adults
- Vitamin D deficiency decreases the absorption of Calcium, which can lead to low levels of Calcium in the blood, thus resulting in brittled bones

Rickets in children

Rickets & Osteomalacia

DEFICIENCY :

2) Renal rickets (renal osteodystrophy)

- This disorder results from chronic renal failure and thus the decreased ability to form 1,25–di-OH D_3
- Calcitriol administration is effective replacement therapy
- Decreased mineralization leads to osteomalacia

DEFICIENCY :

3) Metabolic manifestations:

- Decrease in calcium absorption, decrease calcium level in blood and calcium excretion in urine

DEFICIENCY:

4. Early Symptoms:

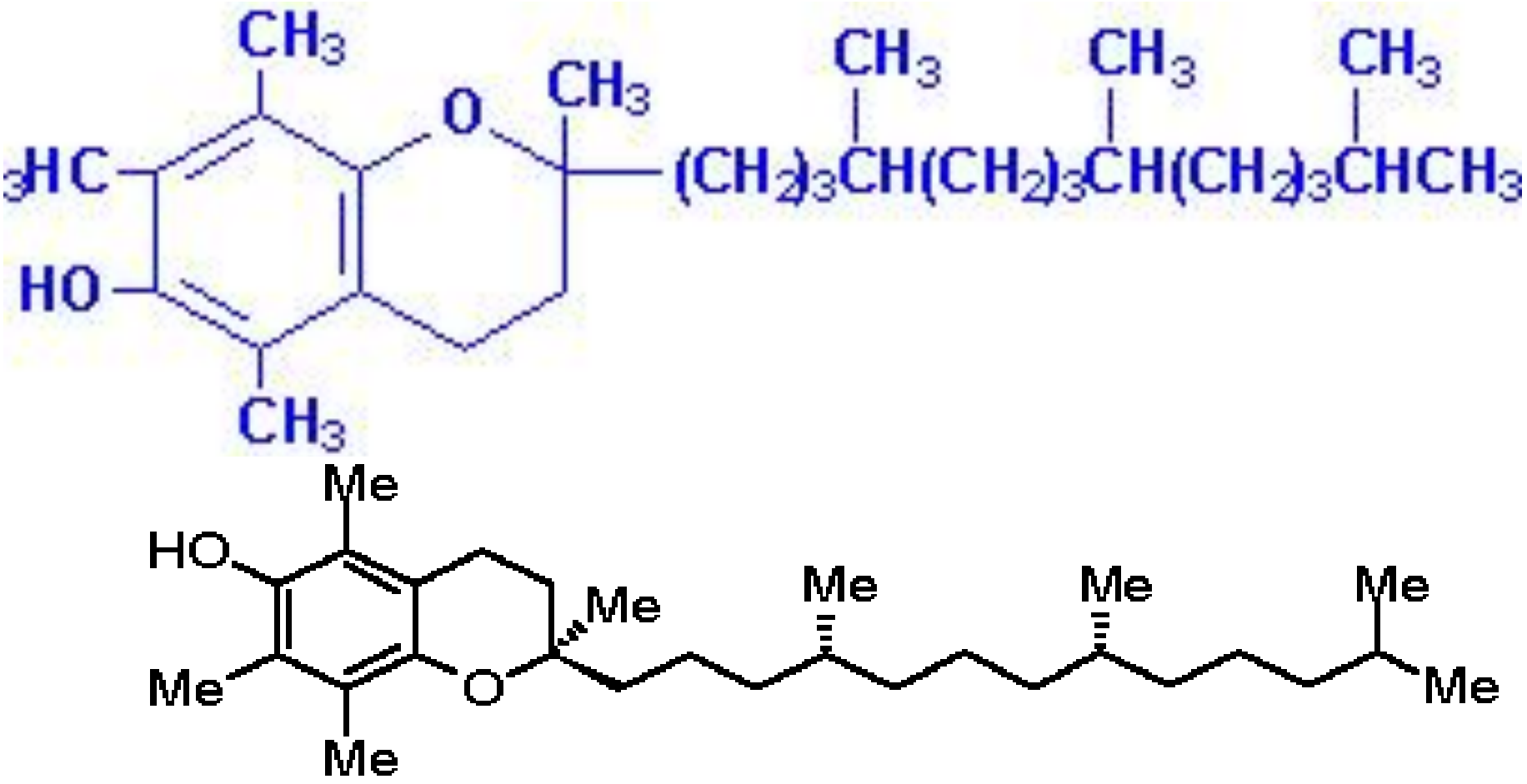
- Result from induced hypocalcaemia
  - Loss of apetite
  - Thirst
  - Constipation
  - Polyurea


Without vitamin D...
Ca
With vitamin D...
Ca

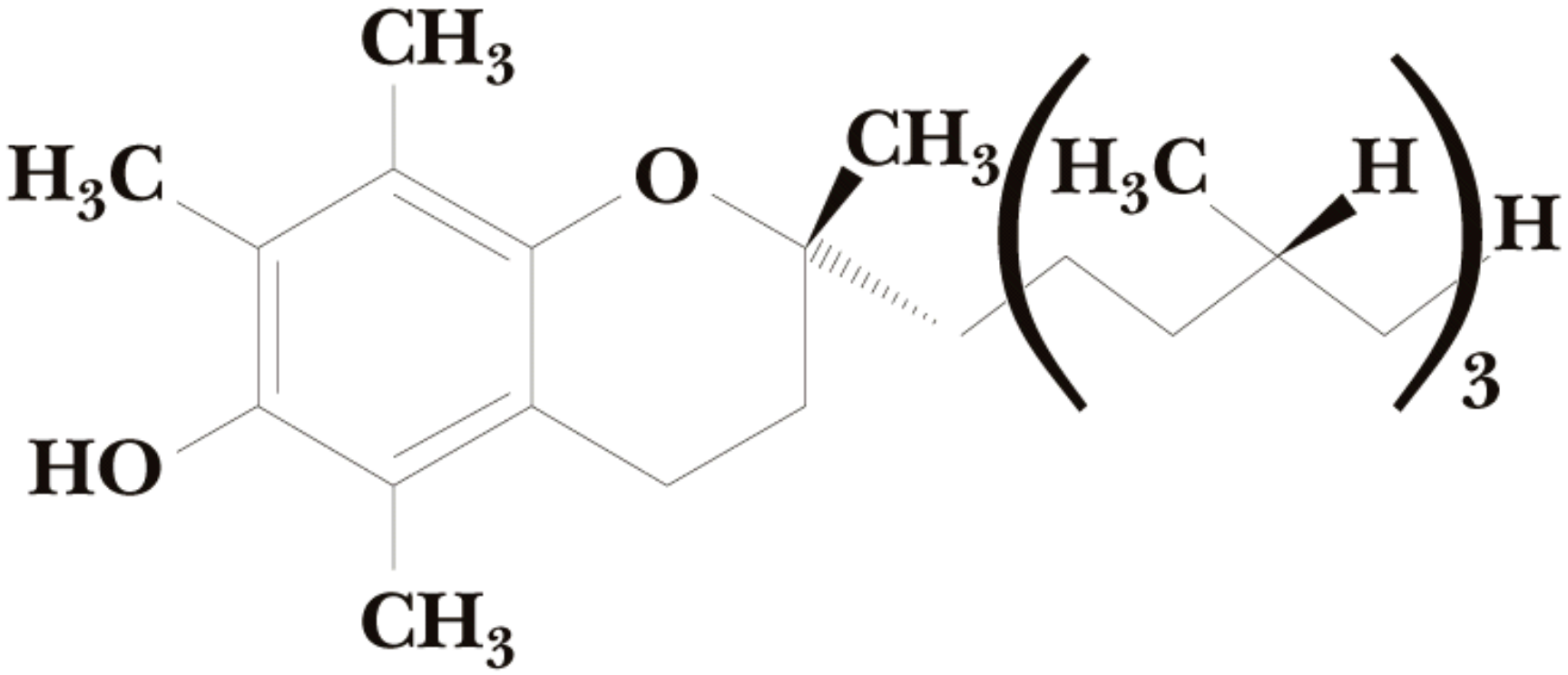
- **Requirements:**
  - 400-800 IU/day for infants
  - 400 IU/day for adults
  - 800 IU/day for pregnancy

- Toxicity of High doses (100,000 IU for weeks or months) of Vit D can cause:
  - Loss of appetite
  - Thirst
  - Nausea
  - Constipation
  - Polyuria
- Enhancement of Ca++ absorption and bone resorption leads to hypercalcaemia which can lead to deposition of calcium in many organs such as kidney and arteries

Vitamins E [α-Tocopherol]



Vitamin E



Vitamin E (α-tocopherol)

PROPERTIES:

- There are 8 naturally occurring tocopherols
- α-Form is the most active
- They are oxidized very easy due to the presence of hydroxyl group
- Vitamins E lose their biological activity by:

✓ Oxidation

✓ Ultraviolet rays

OCURRENCE AND FOOD SOURCES

I. ***Mainly in plants:*** *Green plants as lettu*

II. ***Animals:*** *contain small amounts as liv*
egg yolk, milk and colostrums

- ***ABSORPTION:***
- **From small intestine**
- **Bile salts are necessary for absorpt**

- ***STORAGE:***
- *They are stored in the liver*

Vitamin E

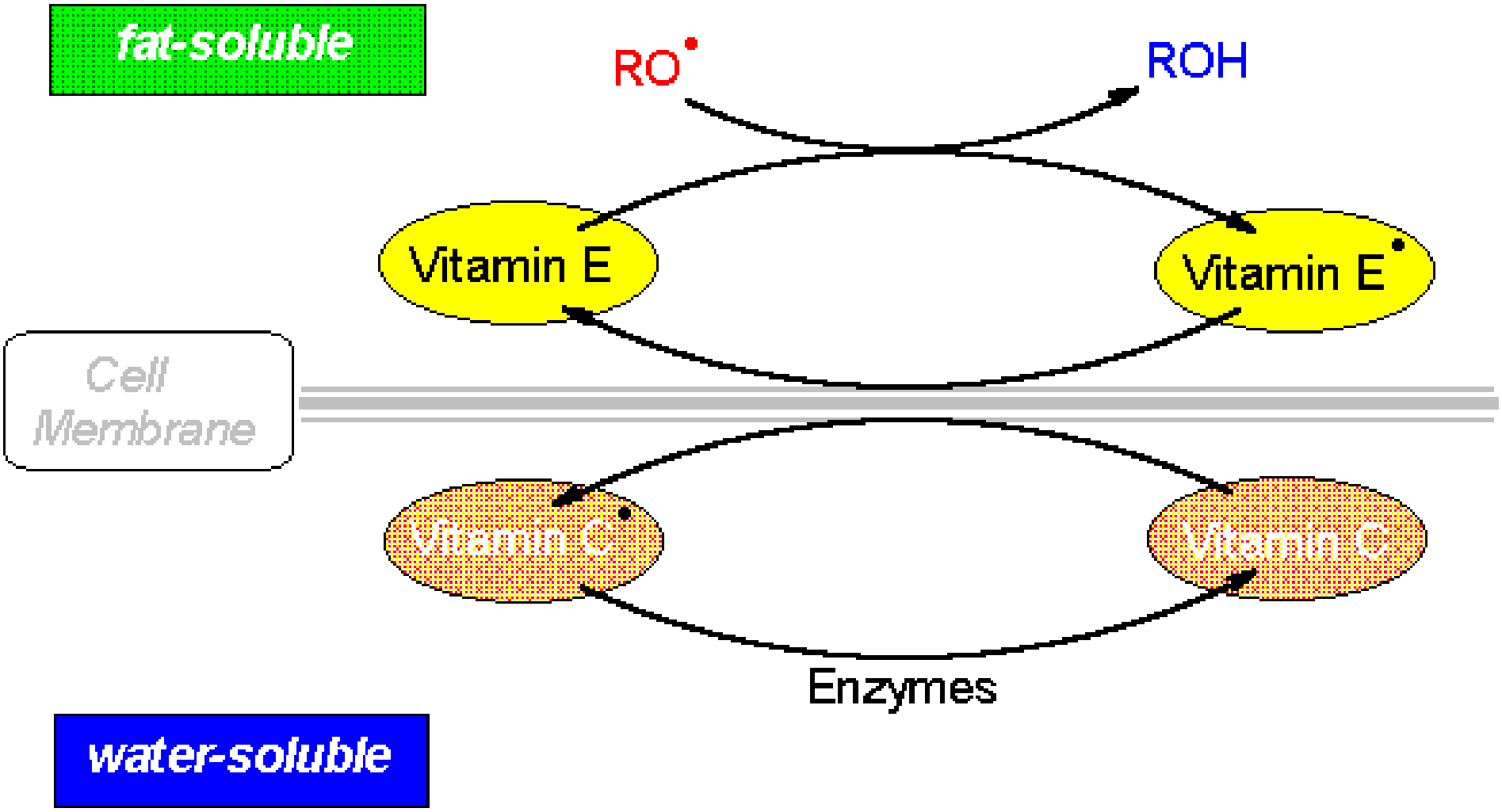
Tocopherol

Vitamin E is found in corn, nuts, olives, green, leafy vegetables, vegetable oils and wheat germ

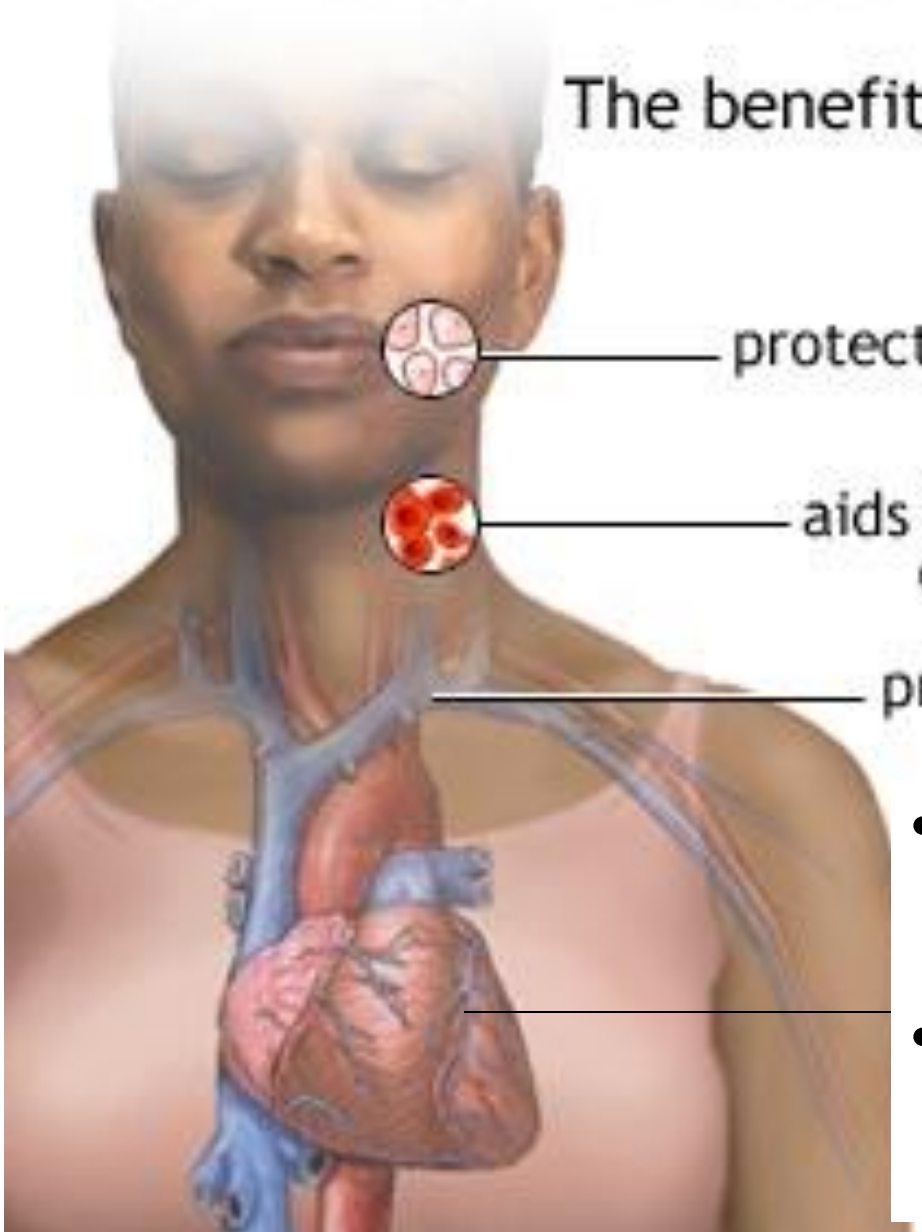
Functions of Vitamins E

- It is used as ***antioxidant***, preventing non-enzymatic oxidation of poly-unsaturated fatty acids
- it prevents a type of anaemia known as **maturation arrest** in which the maturation of red cells is arrested or stopped

Vitamins C & E as Antioxidants



Vitamin E



- **Vit E appears to protect against the development of heart disease**
- **Vit E may prevent oxidation of LDL (oxidized LDL is thought to promot heart disease)**

Deficiency of Vit E

- The signs of human vit E deficiency include:

1. Sensitivity of erythrocytes to peroxide
2. The appearance of abnormal cellular membrane which lead to anemia (maturation arrest)

VITAMINS K

(NAPHTHOQUINONES)

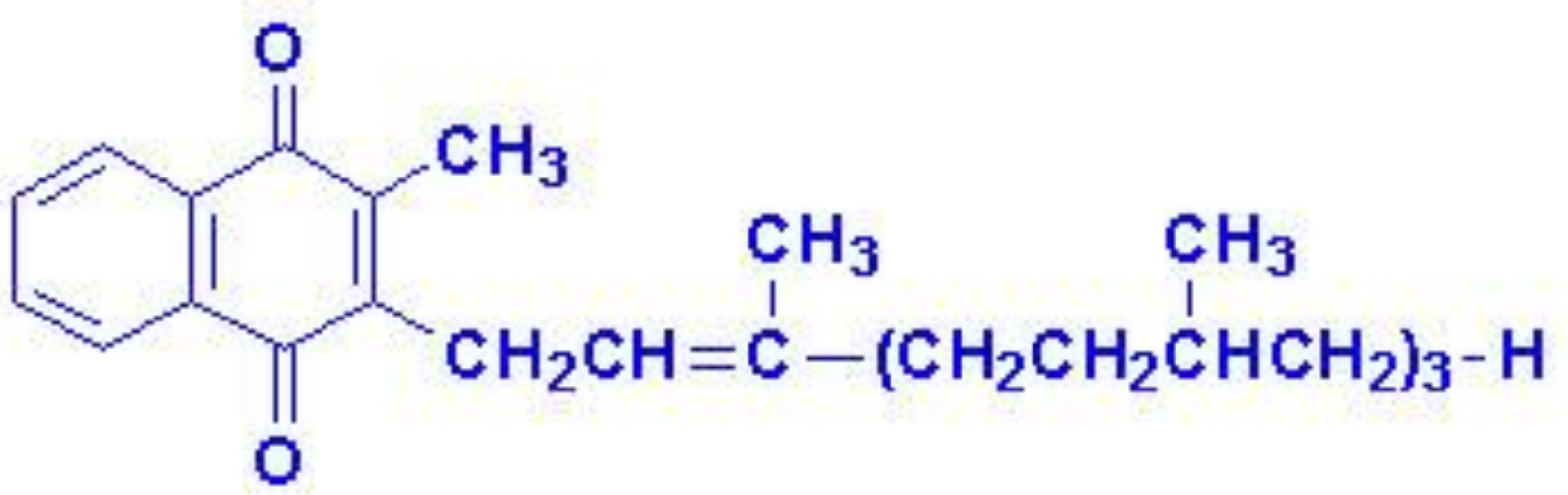
- ***TWO TYPES OF VITAMINS K:***

≥ ***Naturally occurring: K_1 & K_2***

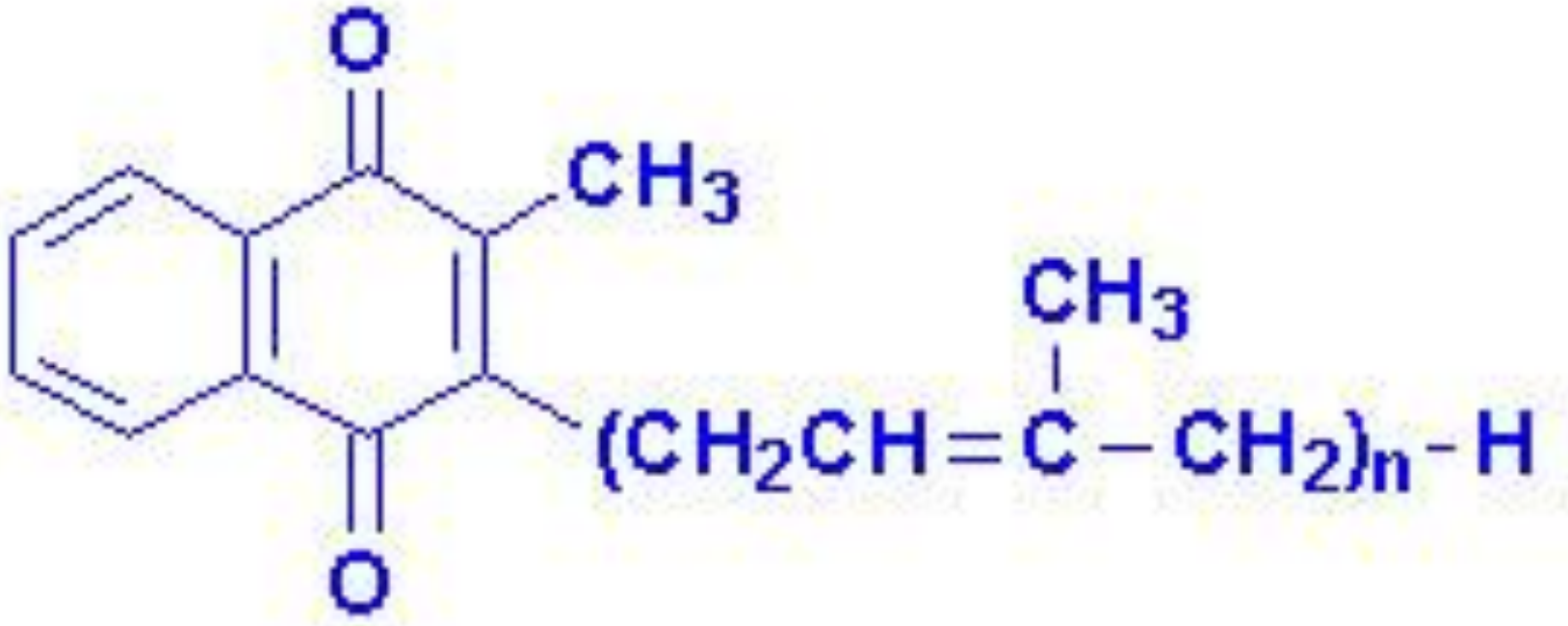
≥ ***Synthetic:***

- ***Menadione (K_3)***

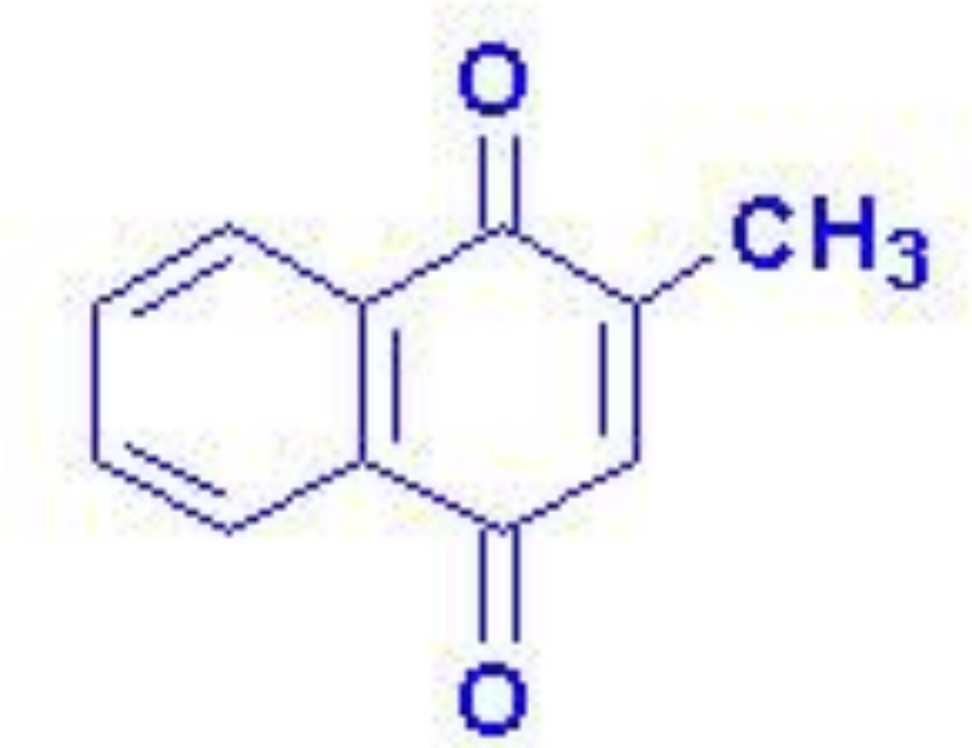
Vitamin K_1 (Phylloquinone)

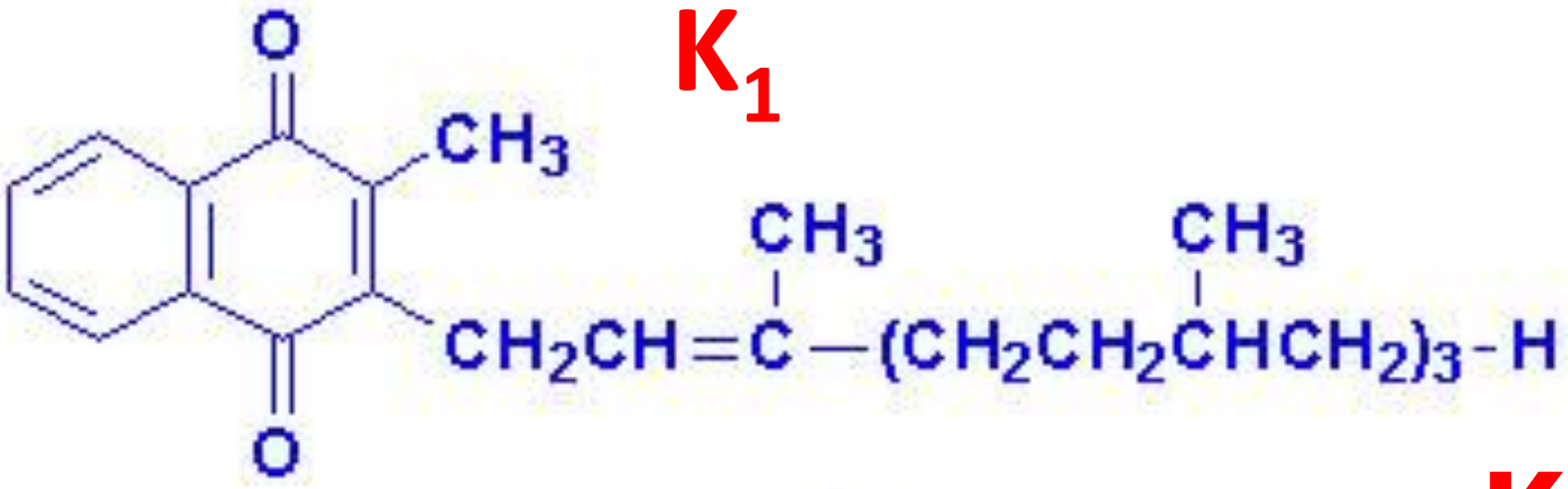


Vitamin K_2 (*Farnoquinone*)

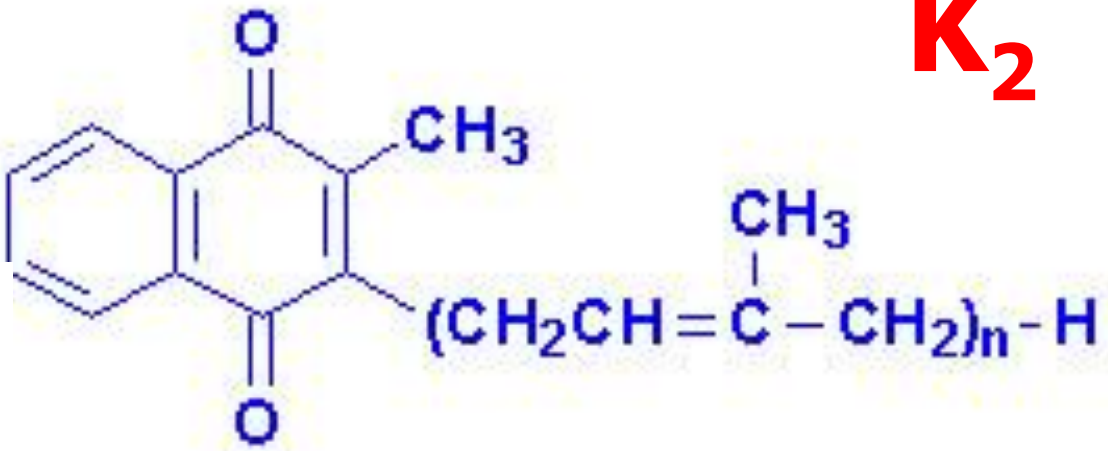


Synthetic Vitamin K (K_3) (Menadione)

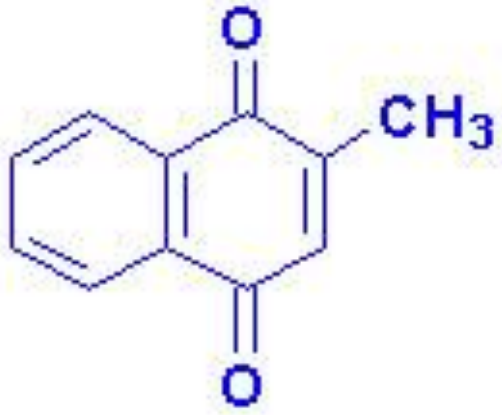



O
CH3
O
CH3
CH3
CH2CH=C—(CH2CH2CHCH2)3-H

K_1


O
CH3
O
CH3
(CH2CH=C—CH2)n-H

K_2


O
CH3
O

K_3

VITAMINS K

- All types of vitamin K contain a methyl group at carbon number 2
- Activity is due to this methyl group
- Substitution of this methyl group by other alkyl radicals or by hydrogen results in marked reduction in Vit K activity

PROPERTIES:

- *Menadione (K_3) is the most active*
- *Vitamins K are stable to heat*
- *They are destroyed by light, alkali and alcohol*
- *Naturally occurring vitamins K are present in plants*

Occurrence & Food Sources

- **VITAMIN K_1**: is present in green leafy plants e.g. spinach. Also found in cauliflower, cabbage, tomatoes and rice
- **VITAMIN K_2**: <u>is produced by normal intestinal bacteria</u>

Functions of Vit K

1. **Production of Coenzyme Q**

2. **Help blood clotting by:**

- **Formation of γ-carboxyglutamate, vit K is required in hepatic synthesis of prothrombin and the blood clotting factors VII, IX and X**

Functions of Vit K

3. Interaction of prothrombin with platelets

- **The γ-carboxyglutamate residues of prothrombin are good chelators of positively charged calcium ions because of the 2 adjacent negatively charged carboxylate groups**
- **The prothrombin–Ca^{++} complex is then able to bind to phospholipids essential for blood clotting of the surface of platelets**

Deficiency of Vit K

- A deficiency in adult is unlikely, due to the synthesis by the bacteria of large intestine
- Haemorrhagic manifestations in skin and mucous membranes
- A prolonged clotting time, characteristic of vitamin K deficiency, is sometimes observed in newborn infants, there intestine is sterile in the first several days after birth. Further, breast and cow's milk are low in vitamin K

The End