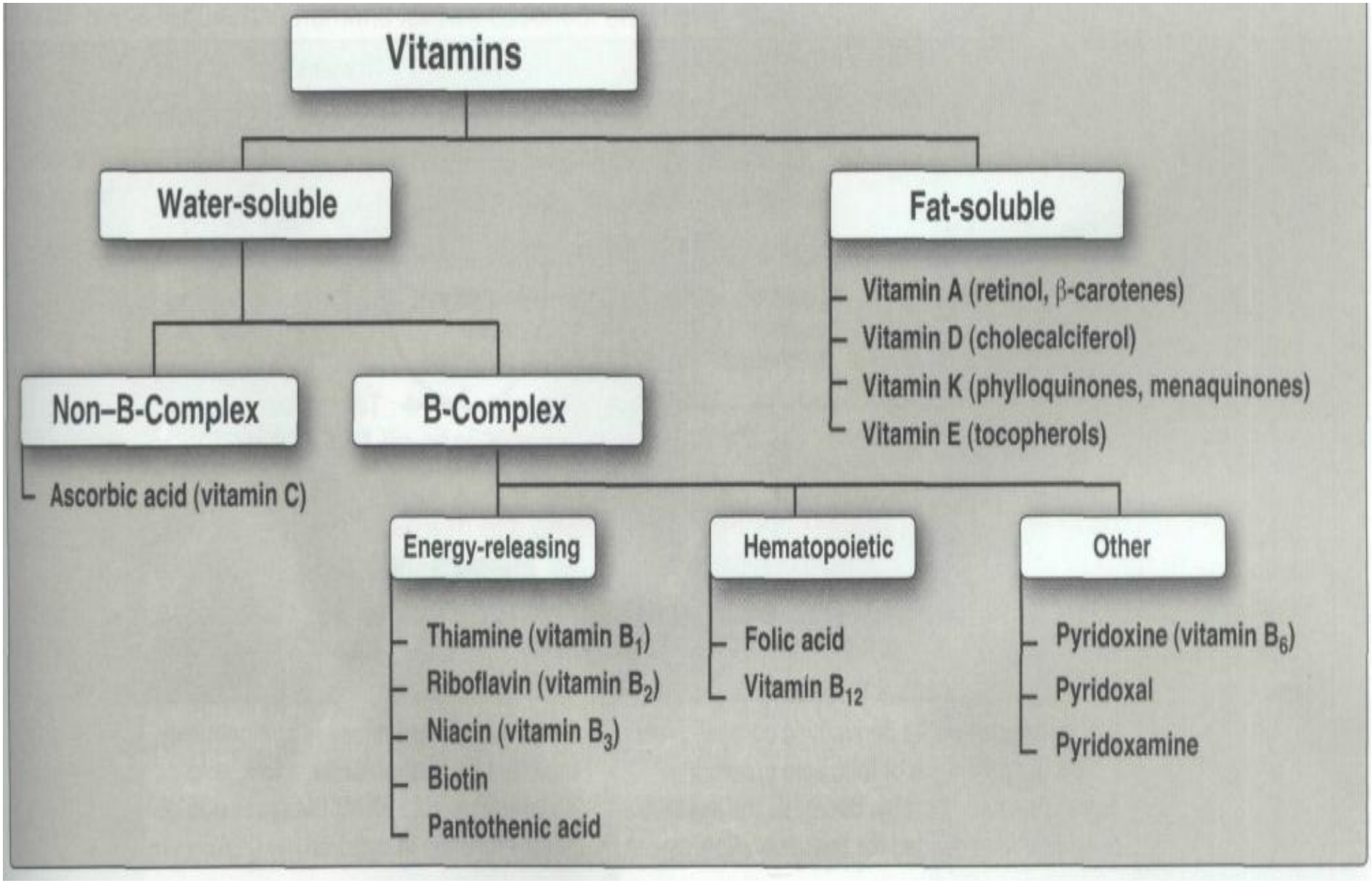

Vitamins
Water-soluble
Fat-soluble
Vitamin A (retinol, β-carotenes)
Vitamin D (cholecalciferol)
Vitamin K (phylloquinones, menaquinones)
Vitamin E (tocopherols)
Non–B-Complex
B-Complex
Ascorbic acid (vitamin C)
Energy-releasing
Hematopoietic
Other
Thiamine (vitamin B_1)
Riboflavin (vitamin B_2)
Niacin (vitamin B_3)
Biotin
Pantothenic acid
Folic acid
Vitamin B_{12}
Pyridoxine (vitamin B_6)
Pyridoxal
Pyridoxamine

Water-soluble Vitamins

Vitamin C
(Ascorbic Acid)

Antiscorbutic vitamin

Chemistry:

Enediol-lactone of an acid

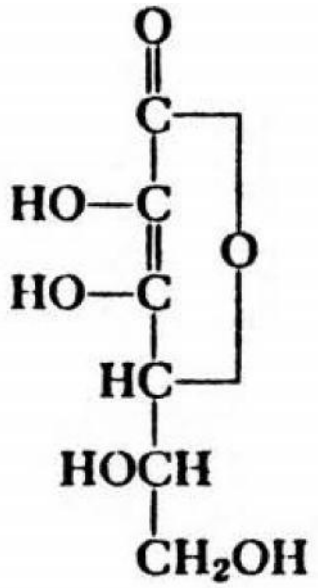
Configuration similar to L-glucose

Comparatively strong acid owing to dissociation of enolic H at C_2 & C_3

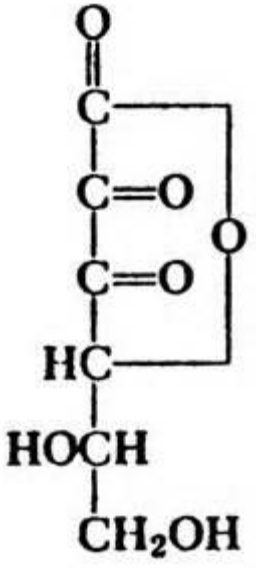
D form – inactive as Antiscorbutic agent

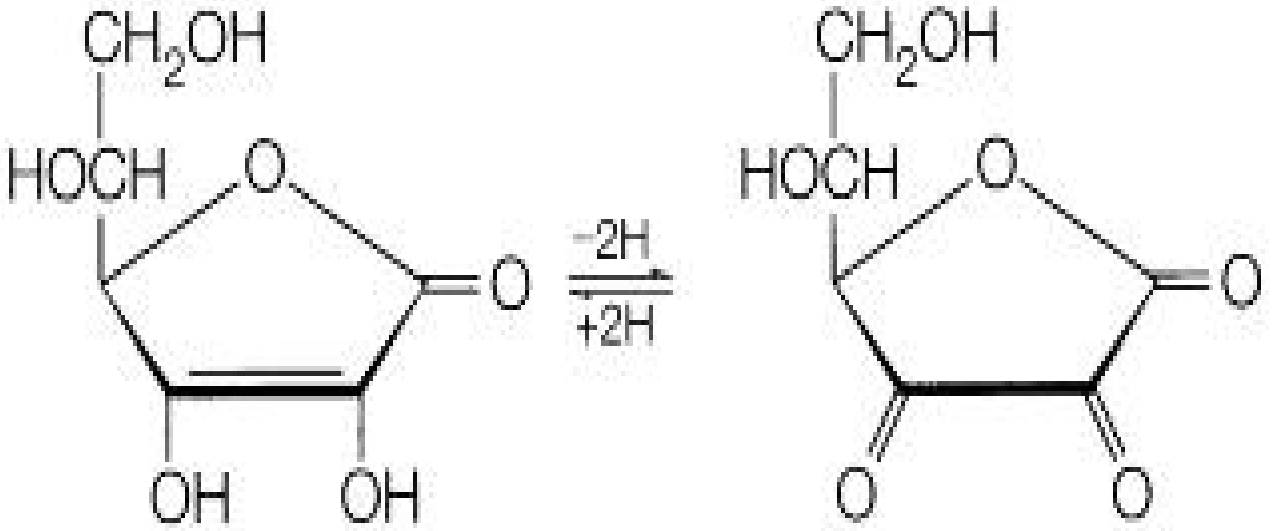
Nature – Vit.C is L-ascorbic acid

- L-ascorbic acid
  (reduced form)



L-Dehydroascorbic acid
(oxidized form)





Ascorbic acid
(reduced form)

Dehydroascorbic acid
(oxidized form)

- Strong reducing property: H atoms at C_2 & C_3
- Oxidation by air, H_2O_2, $FeCl_3$, methylene blue, ferricyanide, 2,6 DCIP
- Oxidation reduction: reversible *in vitro* by H_2O_2
- Oxidation reduction: reversible *in vivo* by
- -SH groups (e.g. Glutathione)

- Solid form and in acidic solutions – it is stable
- Rapid destruction in alkaline solutions
- Oxidative destruction increased by increasing pH (Ag^{++} & Cu^{+++})

Glutathione (GSH) - organic chemical

- Found in plants & animals
- Tripeptide, (Cys, Gly, Glu)
- An antioxidant, prevent damage to cell caused by ROS such as free radicals and peroxides

Occurrence & food sources

- Widely distributed - plant & animal tissues
- Animal: no storage, but high conc. in metabolically active organs (liver, adrenal cortex, corpus luteum)

Dietary Sources

- Vegetables
- Citrus fruits (orange, lemon, lime etc.)

- Other fruits: papaya, pineapple, banana, strawberry
- Leafy vegetable: cabbage, cauliflower
- Germinating seeds, green peas, beans, potatoes, tomatoes
- AMLA
- Large amount lost during cooking, processing & storage – due to water solubility & oxidative degradation

Biosynthesis

- Most animals, plants can synthesize from glucose by uronic acid pathway
- Humans lack enzymes (L-gluconolactone oxidase) required for biosynthesis
- So, dietary supplements required

Metabolism

Absorption, distribution, excretion

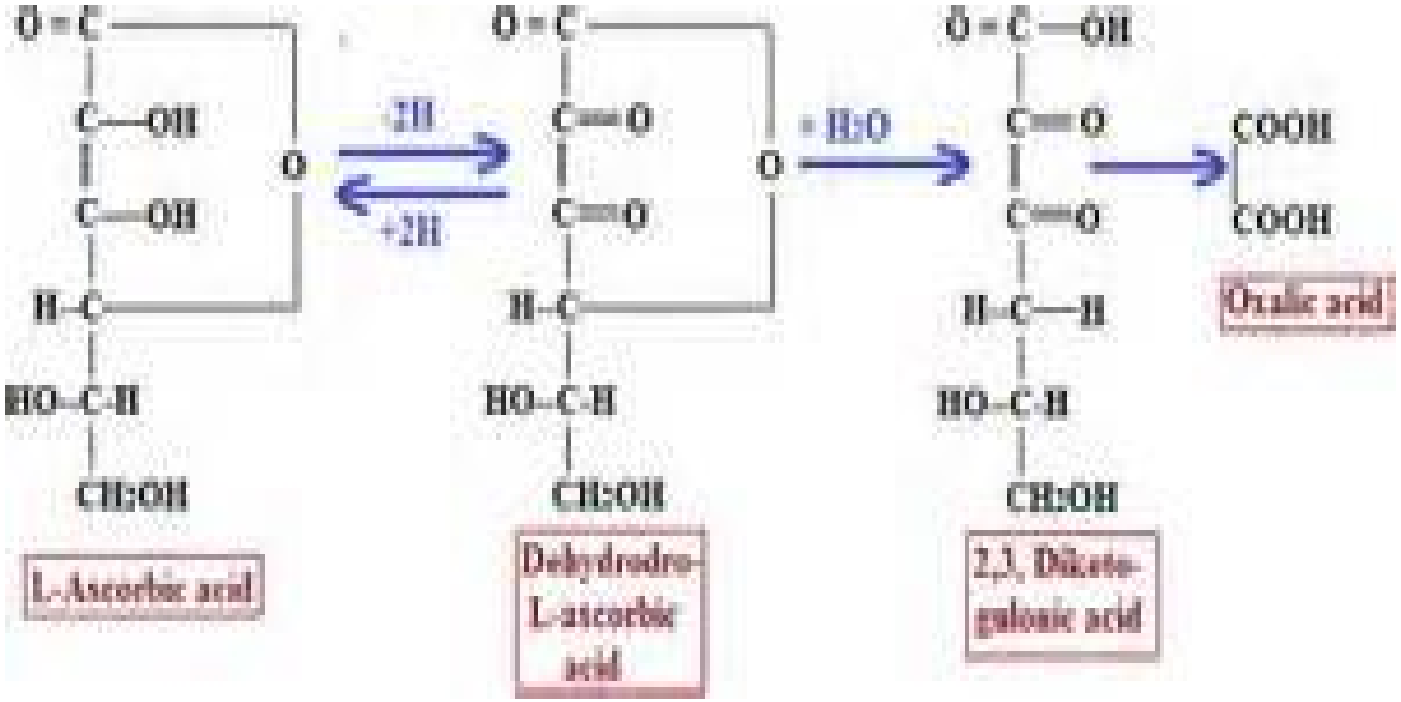
- Absorption: small intestine, peritoneum, subcutaneous tissues
- Widely distributed in body
- Conc. = metabolic activity
- > in pituitary glands, adrenal cortex
- < heart, muscles
- Normal plasma levels: 0.6-1.5 mg/dL

- Exists largely as reduced form + small amount of oxidized form
- Both are metabolically active forms

- Out of 75-100 mg (normal dietary intake)
  -50-75% converted to inactive compounds
  - 25-75% excreted in urine as such

Chief Terminal Metabolites

- Rats and other animals - CO2 & oxalic acid
- Humans – Oxalic acid & diketogluconic acid excreted in urine

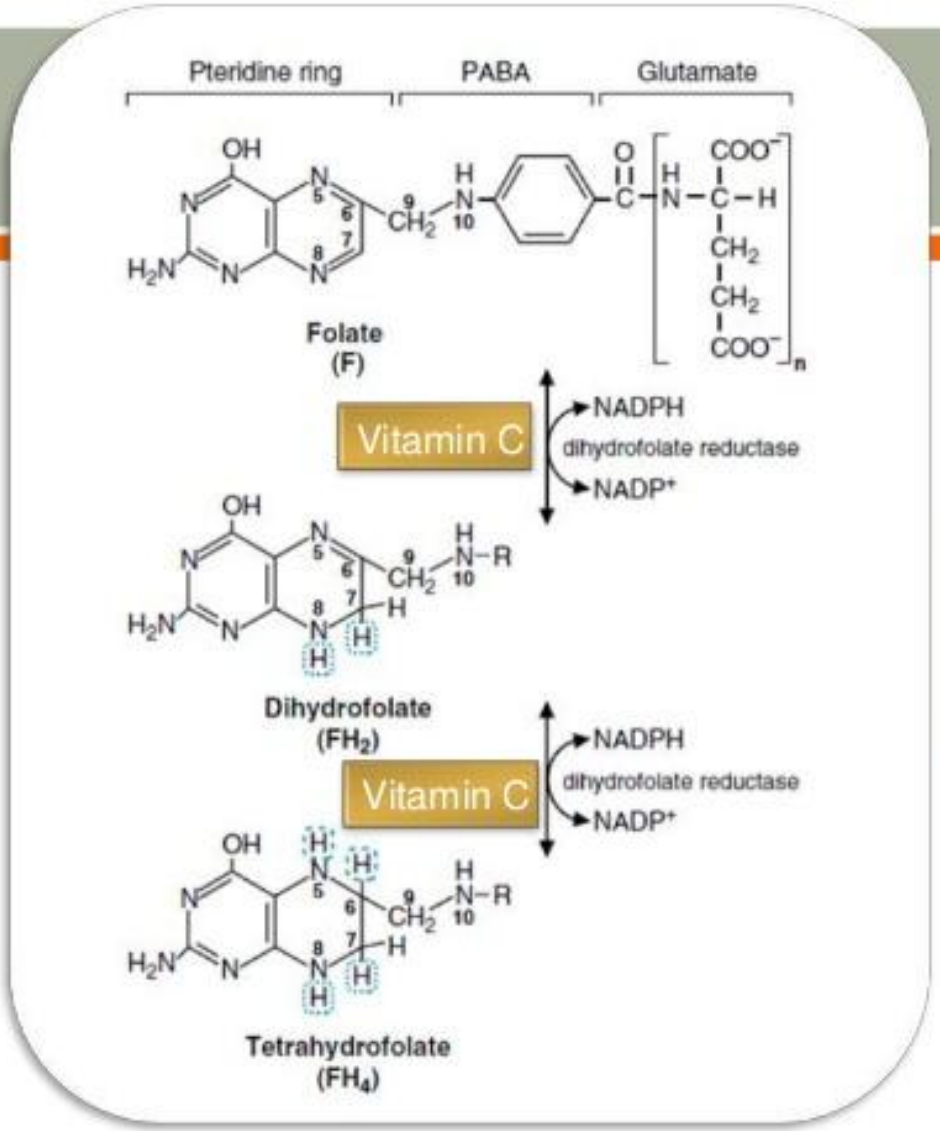


Metabolic functions

- Role in cellular oxidation reduction
- Role in collagen synthesis –scurvy
- Functional activity of fibroblasts and osteoblasts
- Role in Trp metabolism (as cofactor)
- Role in Tyr metabolism (as cofactor)

- Formation of ferritin (intracellular protein that stores Fe)
- Fe absorption (convert ferric iron to ferrous iron, mobilization of Fe from storage site)
- Folate metabolism: hypochromic microcytic anemia (small, pale RBCs)

Formation of active FH4



Folate metabolism

- Role in ETC
- Enzyme activation/inhibition

  Activation: arginase, papain

  Inhibition: urease, amylase
- Role in catecholamine synthesis

(As coenzyme – dopamine hydroxylase – dopamine to norepinephrine conversion)

- Role in carnitine synthesis

- Role in alpha oxidation of FA
- Role in STREES
- Role in hypocholesterolemia
- Role as coenzymes

Clinical Aspects

- Deficiency manifestations: SCURVY
- Defects in collagen synthesis due to Vitamin C deficiency
- Fragile capillaries & increased tendency of haemorrhage
- Delayed wound healing – as less collagen
- Poor dentine formation in children
- Swollen, spongy germs - bleeding

Proline ⟶ hydroxyproline

Lysine ⟶ hydroxyLysine

Hydroxylases, Fe^{++}, O_2, vit.C

- Hydroxyproline, hydroxyLysine – imp. part of mature collagen fibres

- Vit.C deficiency – no hydroxylation –rapid destruction of collagen intermediates

- Severe scurvy may lead to secondary infections
- Poor bone mineralization, weak bones, more fractures
- Painful swelling of joints
- Anaemia – hypochromic microcytic type

Clinical Tests

- Benedicts test

As vit.C is reducing agent, this test is positive

- Urine ascorbic acid saturation test

Test dose (5 mg/lb body weight) administered

IF 50% or > excreted in urine in next 24 hrs- NO deficiency

- Intradermal test

Intradermal injection of DCIP & determine time required for decolorization

Long persistence of blue color – indicative of sub-saturation

RDA

- 100 (75-80) mg in adults
- Infants: 30 mg
- Pregnancy/lactation: 100-150 mg

Requirement is increased in case of infections

Hypervitaminosis

- Medical names of the different conditions derived from the vitamin involved
- An excess of vitamin C is called hypervitaminosis C
- Generally, toxic levels of vitamins are achieved through high supplement intake and not from dietary sources.
- Excess in humans – on harmful effects observed
- Rats: excess leads to diabetes by beta cells destruction
- Injection of glutathione – improves the condition

Other therapeutic uses

- Control & treatment of infections
- Wound healing, ulcer, trauma, burns
- Allergy, cold
- During child birth: induce uterine contractions
- Conversion of methemoglobin to hemoglobin

Methemoglobin (**methaemoglobin**) is a form of the O2-carrying metalloprotein Hb, in which the iron in the heme is in the Fe^{3+} (ferric) state, not the Fe^{2+} (ferrous) of normal hemoglobin

- Methemoglobin cannot bind oxygen, unlike oxyhemoglobin
- Bluish chocolate-brown in color
- human blood - a trace amount of methemoglobin is normally produced spontaneously
- When it is present in excess the blood becomes abnormally dark bluish brown

- The NADH-dependent enzyme methemoglobin reductase is responsible for converting methemoglobin back to hemoglobin.
- Normally 1-2% hemoglobin is methemoglobin; a higher percentage than this can be genetic or caused by exposure to various chemicals and depending on the level can cause health problems known as methemoglobinemia

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- Vitamin C: Is the Synthetic Vitamin as Good as the Natural One?
- Are the vitamins from the two sources different?
- Can the body distinguish a vitamin's source?