

University of Al Maarif College of Medical and Health Technologies Department of Medical Laboratory Technologies



3th stage Third Lecture

Disorder of Vitamins and Trace elements metabolism

by ABDULLAH ABDULSATTAR RAEEF



- Vitamins are organic compounds required in tiny amounts for essential metabolic reactions in a living organism
- > Absence or deficiency of vitamins causes deficiency disorders
- Form Vitamin given by Casimir Funk in 1912
- Derived from plants except Vit B12

CLASSIFICATION

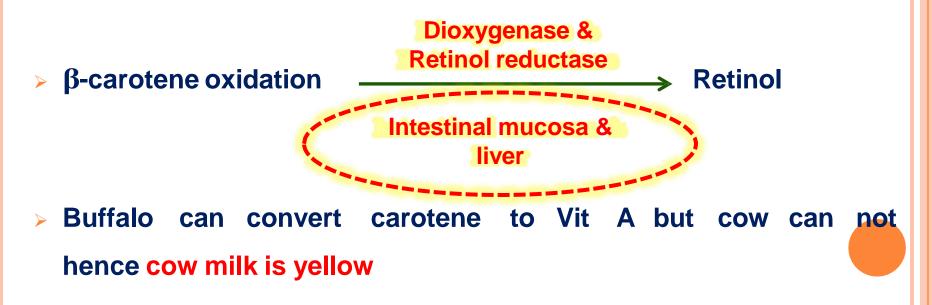
- Classified based on their solubility as fat soluble vitamins and water soluble vitamins.
- I) Fat-soluble vitamins : vitamin A, D, E and K.
- II) Water-soluble vitamins: vitamin B complex group and vitamin C.
 - **B** complex group include:
 - Vitamin B1 (thiamin)
 - Vitamin B2 (Riboflavin)
 - Vitamin B3 (Niacin/Nicotinamide/Nicotinic acid)
 - Vitamin B6 (Pyridoxine)
 - Panthothenic acid
 - Folic acid
 - Vitamin B12 (Cyano cobalamine)
 - Biotin
 - Choline

DIFFERENCES BETWEEN FAT SOLUBLE AND WATER SOLUBLE VITAMINS

Differences	Fat soluble	Water soluble vitamins
Names	A,D,E,K	Vitamin C & B complex
Solubility	Soluble in fats and organic solvents	Soluble in water
Digestion and absorption	Requires fat and bile	Easily absorbed in intestine
Excretion	Via feces	Via Urine
Storage	Stored in the body in fat depots and in liver	Not stored in body except Vitamin B12 (liver)
Toxicity	An overdosage can lead to toxicity	Usually not toxic as it is readily excreted when given in excess

VITAMIN A

- In plant exist in provitamin form i.e. carotenoids
- > Vit A exist only in animal origin feeds
- Occurs in three forms: Retinol, Retinal, Retinoic acid
- > 1molecule of carotene yields: 2 molecule of Vit A
- β-carotene is most active carotenoids



VITAMIN A: FUNCTIONS

1) Vision

- Synthesis of the visual pigment Rhodopsin
- 2) Bone growth
- Control osteoblastic & osteoclastic activity
- 3) To maintain integrity of epithelial cells
- Required for formation of mucous secreting epithelium
- In absence of Vit A, keratinization occurs which causes reduce resistance for entry of infective organism
- Play imp role in combating infection so called as Anti-infective vitamin.

VITAMIN A: DEFICIENCY

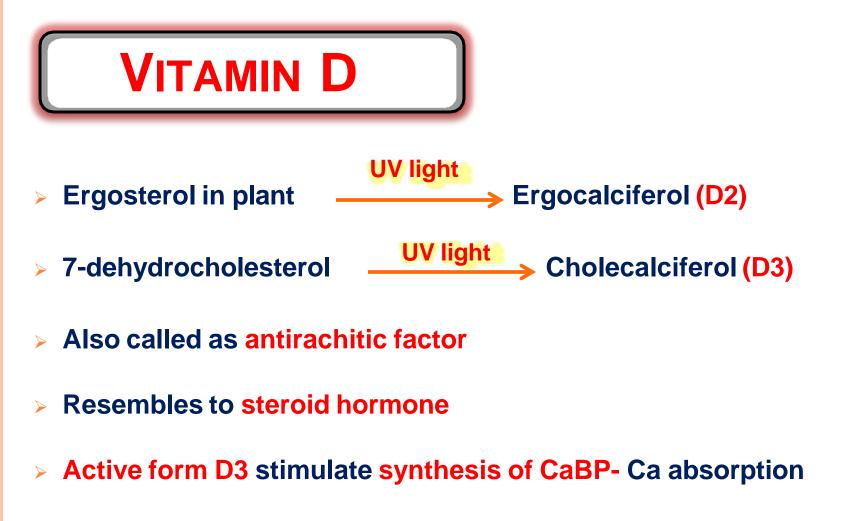
- 1) Night Blindness
- 2) Xeropthalmia
- > Advance stage of deficiency
- Dry cornea & conjunctiva, cloudiness & ulceration, keratinization of the cornea, blindness
- White patches on white portion eye: Bitot spot
- 3) Nutritional roup in poultry
- Rhinitis, nasal & ocular discharge, stuck eyelid with thick exudate rapid & difficult breathing
- 4) Keratinization of epithelium
- 5) Reduced reproductive performance

VITAMIN A: DEFICIENCY

- 6) Bone deformities.
- 7) Nervous lesion
- Skeletal growth retarded but brain grows which create pressure in nervous tissue causes increased CSF pressure
- 8) Congenital blindness
- > **Optic foramen** is not formed properly.
- Small size optic foramen leads to the constriction of optic nerve.
- Permanent damage to the nerve can lead to permanent blindness.

Sources

- > Animal source: Fish liver oils, egg yolk, milk fat
- Plant source: All green leaves are rich in provitamin A, betacarotene.



Required for calcium and phosphorus deposition in bones

VITAMIN D: DEFICIENCY

- Rickets in young ones
- > Osteomalacia in adult
- Retarded growth, decreased feed consumption
- Increased loss of Ca & P in urine
- Poultry: thin shelled eggs, reduced hatchability, enlarged parathyroid gland

Sources

- Cod liver oils (rich source)
- Egg yolk and sun dried roughage's/grains
- Provitamin D: Ergosterol plant and 7-dehydrocholestrol skin of animals.

VITAMIN E

- > Out of eight natural forms alpha tocopherol is more active
- Natural antioxidant: Firstly, radicals are scavenged by vitamin E secondly, glutathione peroxidase destroys any peroxide formed before they can damage the cell.
- Helps in absorption & utilization of Vit A & extend storage of Vit A in liver by protecting from oxidation
- > Also plays an important role in the development and function of the immune system.
- Important for reproduction

VITAMIN E: DEFICIENCY

- 1) Infertility in female, reduced sperm motility in males
- 2) Nutritional myopathy/white muscle disease
- Mulberry heart disease in pigs
- Encephalomalacia/crazy chick disease: chick is unable to walk or stand, and is accompanied by haemorrhages and necrosis of brain cells.
- 4) Exudative diathesis: Oedema of the subcutaneous fatty tissues, associated with an abnormal permeability of the capillary walls

Sources

Green fodders, cereal grains, vegetable oils, fats, and nuts, oil seeds and legumes

VITAMIN K

- K1: Phylloquinone Green plants & oil seeds
- K2: Menaquinone Intestinal bacteria
- K3: Menadione Synthetic product
- Required for synthesis of prothrombin & other clotting factors
- Vitamin K is also called as anti haemorrhagic vitamin
- Involve in electron transport & oxidative phosphorylation
- Synthesized by GI tract microorganisms

VITAMIN K: DEFICIENCY

- Low prothrombin level in blood leads to haemorrhagic conditions
- Sweet Clover disease: Mould infested sweet clover contains a compound dicoumarol, which is potent vit. K antagonist leading to haemorrhagic disease
- Deficiency more common in poultry
- Sources
- Green leafy vegetables, egg yolk, liver, fish and synthesised by bacteria in GI tract.

VITAMIN C

- > Ascorbic acid
- Synthesized in all animals except human & guinea pigs: they lack L-gluconolactone oxidase required for Vit. C synthesis from 6 carbon sugar
- Plays an important role in the formation of collagen and intercellular cement substance
- Plays an important role in the oxidative reduction reaction of living cells (Antioxidant)
- > Also used as preservative in canning & freezing industry

VITAMIN C: DEFICIENCY

Scurvy

- > Dry, rough skin covered with reddish spots
- Weakness, bleeding, ulcerated gums, loosening of teeth, swollen joints & haemorrhages

Sources

Citrus fruits, tomatoes, potatoes and green leafy vegetables are rich sources.

THIAMIN (B1)



Thiamine pyrophosphate is a coenzyme involved in oxidative decarboxylation of pyruvate to acetyl COA. and of alpha ketoglutarate to succinyl COA in TCA cycle.

Deficiency

- Beriberi (man)/ polyneuritis (chick): Accumulation of intermediates of carbohydrate metabolism (lactate, pyruvate & oxaloglutarate) causes neuritis
- Chicks sit on flexed legs & draws head backward-star gazing
- Polioencephalomalacia (ruminants): Thiamin hydrolyzed by thiaminase in rumen- Circling movements, opistotonus, convulsion & death

Sources: Yeast, bran, rice polish, egg yolk liver kidney. Pork is rich in thamine.

RIBOFLAVIN (B2)

- It is a constituent of flavin mononucleotide (FMN) and Flavin adenine dinucleotide (FAD)- used by cells to transport hydrogen in metabolic pathway
- Involved in amino acid and carbohydrate metabolism
 Deficiency
- Degeneration of myeline sheath
- Lost integrity of epithelium
- Curled toe paralysis (poultry): peripheral nerve degeneration
- Swine: stiff legs, nerve degeneration, corneal opacity, cataract

Sources: Synthesised by yeast, bacteria and fungi. Rich sources are liver, yeast, milk, egg and green leafy vegetables

NIACIN/ NICOTINIC ACID

- > Nicotinic acid: Plant; Nicotinamide: Animals
- Component of NAD & NADP: CHO, protein & lipid metabolism
- > Tryptophan is precursor of niacin (except cat & mink)

Deficiency

- Black tongue (dogs): inflammation of gums, dark patches on tongue, drooling of bloody saliva
- > Pellegra/blue tongue (man & pigs)

Sources: Rich sources of are liver, yeast, groundnuts and sunflower meals. In cereals the vitamin is present in the bound or coenzyme form.

PYRIDOXINE (B6)

- Component of pyridoxal phosphate (PP) which act as coenzyme for transaminases & decarboxylases
- Required for metabolism of tryptophan

Deficiency

- Pigs: reduced appetite, microcytic hypochromic anemia, convulsion, slow growth
- Poultry: Chicks show jerky movements, in adult birds hatchability & egg production are adversely affected

Sources: Chemically bound to protein in many sources

 Groundnut meal, rice bran, wheat bran, molasses, liver and milk

PANTOTHENIC ACID

- Constituent of coenzyme A, which is the important for acyl transfer.
- It is also a structural component of acyl carrier protein, which is involved, in the cytoplasmic synthesis of fatty acids

Deficiency

- Pigs: Goose-stepping, thin hairs, brownish secretion around eye, slow growth
- Chicks: Retarded growth, poor feather development, granular eyelid, scab around mouth

Sources: Rich sources are liver, egg yolk, groundnuts, peas, yeast and molasses. Cereal grains and potatoes are also good sources of the vitamin



- Earlier called Vitamin H
- > Transfer of carbon dioxide from one substrate to another
- > Avidin in egg white protein is antimetabolite to biotin

Deficiency

- > Pigs: foot lesions, alopecia (hair loss) and a dry scaly skin.
- Poultry: causes reduced growth, dermatitis, leg bone abnormalities, cracked feet, poor feathering and fatty liver and kidney syndrome (FLKS.(
- Sources: Biotin is widely distributed in foods; liver, milk, yeast, oilseeds and vegetable are rich sources



 Coenzyme in the mobilization and utilisation of singlecarbon groups (e.g.) formyl, methyl

Deficiency

- Chicks and turkeys: poor growth, anaemia, poor bone development and poor egg hatchability
- > Macrocytic, hyperchromic anaemia in humans

Sources: Dark green leafy materials, cereals and extracted oilseed meals are good sources



- Component of phospholipid lecithin
- Metabolic essential for building & maintaining cell structure
- It also plays an important part in lipid metabolism in the liver
- Component of acetylcholine which is responsible for the transmission of nerve impulses.
- It serves as a donor of methyl groups
- Choline can be synthesized in the liver from methionine.

Deficiency: Perosis, slow growth, fatty liver

Sources: Green leafy materials, yeast, egg yolk and cereals are rich sources of choline

VITAMIN B12

- > Cynocobalamin, Stored in liver
- > Plant do not synthesize Vit B12
- Synthesized by rumen bacteria when sufficient Co is available
- Synthesize methyl group from one carbon precursor
- Concerned in synthesis of RNA, DNA, essential for cell division
- Control protein synthesis
- Necessary for the conversion of methylmalonyl COA into succinyl COA

Deficiency: Poor growth, Poor feathering, Decreased hatchability, Dermatitis and rough coat.
 Sources: Liver kidney excellent sources, meat & fish moderate sources

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Trace Elements Metabolism Disorders

Trace elements are minerals that are required by living organisms in very small amounts (typically less than 100 mg per day), but they are essential for proper physiological and biochemical functioning. Despite their small quantities, they play crucial roles in various biological processes, including enzyme functions, hormone production, and maintaining structural integrity of tissues.

A trace elements metabolism disorder refers to a condition in which the body has difficulty properly processing or utilizing trace elements—minerals that are essential in very small amounts for various physiological functions.

Functions of Trace Elements

Enzymatic co-factors

Many trace elements act as cofactors for enzymes, helping catalyze vital biochemical reactions.

2 Antioxidant defense

Elements like selenium and zinc contribute to the body's defense against oxidative stress.

3 Hormonal functions

lodine, for example, is critical in the synthesis of thyroid hormones, while others support hormone activation and regulation.

4 Immune response

Trace elements like zinc and copper support a healthy immune system and wound healing.





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General Mechanisms Involved

Absorption

Disorders can arise when the body absorbs too much or too little of a metal due to genetic or environmental factors.

Excretion

Failure to properly eliminate excess metals (like in Wilson's disease or hemochromatosis) leads to toxic accumulation.

Transport

Defects in proteins that transport metals (like copper or zinc transporters) can lead to either accumulation or deficiency.

Enzymatic Activity

Some metals act as cofactors for enzymes, and disruptions in metal metabolism can impair enzyme function, affecting various biochemical pathways.



Common Trace Elements and Their Roles

Zinc	Involved in immune function, protein synthesis, wound healing, and DNA synthesis.
Copper	Important for red blood cell formation, iron metabolism, and antioxidant defense.
Selenium	Functions as an antioxidant and plays a role in thyroid hormone metabolism.
lodine	Essential for thyroid hormone production, which regulates metabolism.
Iron	Vital for oxygen transport in the blood, immune function, and DNA synthesis.
Manganese	Supports bone formation, wound healing, and carbohydrate metabolism.

Wilson's Disease and Hemochromatosis

Wilson's Disease

Cause: A genetic defect in copper metabolism.

Effect: Copper accumulates in the liver, brain, and other tissues.

Symptoms: Liver disease, neurological symptoms (tremors, difficulty speaking), psychiatric disturbances, and Kayser-Fleischer rings in the eyes.

Treatment: Chelation therapy to remove excess copper, medications like zinc salts to reduce copper absorption.

Hemochromatosis

Cause: A genetic condition that leads to excessive iron absorption.

Effect: Iron buildup in organs like the liver, heart, and pancreas, causing damage.

Symptoms: Fatigue, joint pain, diabetes, liver disease, heart disease.

Treatment: Regular blood removal (phlebotomy) or chelation therapy to reduce iron levels.



Menkes Disease and Zinc Disorders

Menkes Disease

Cause: A genetic disorder affecting copper transport.

Effect: Copper deficiency, affecting brain development and other vital functions. Symptoms: Developmental delays, weak muscle tone, seizures, brittle hair. Treatment: Copper injections or supplementation, though outcomes vary.

Zinc Deficiency

Cause: Poor dietary intake, malabsorption, or genetic disorders affecting zinc metabolism. Effect: Impacts immune function, growth, and wound healing.

Symptoms: Delayed growth, weakened immune response.

Treatment: Correcting diet or managing exposure, with zinc supplementation as needed.

Zinc Toxicity

Cause: Over-supplementation or environmental exposure.

Effect: Impacts immune function, growth, and wound healing.

Symptoms: Nausea, vomiting, and impaired immunity.

Treatment: Correcting diet or managing exposure, along with zinc reduction as needed.

Acrodermatitis Enteropathica and Manganese Metabolism Disorder

Acrodermatitis Enteropathica

Cause: A rare genetic disorder leading to defective zinc absorption.

Effect: Zinc deficiency.

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Symptoms: Skin lesions, diarrhea, hair loss, immune dysfunction.

Treatment: Lifelong zinc supplementation.

Manganese Metabolism Disorder

Cause: Rare mutations affecting manganese transport and metabolism.

Effect: Accumulation of manganese, potentially leading to neurological symptoms similar to Parkinson's disease.

Symptoms: Tremors, muscle stiffness, difficulty walking.

Treatment: Chelation therapy to reduce manganese levels.

Aluminum Toxicity



Cause

Usually due to excessive exposure or impaired kidney function leading to aluminum buildup.



Effect

Can affect bone health and the nervous system, particularly in patients on dialysis.

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Symptoms

Bone pain, muscle weakness, confusion, and dementia.

Treatment

Reducing aluminum exposure and using chelation therapy.

