

NATIONAL OPEN UNIVERSITY OF NIGERIA

NSC 207



Medical Biochemistry Module 3

NSC 207 (Medical Biochemistry II)

Module 3

Course Developer/Writer

Dr. B. O. Emman-Okon & Dr. J.O. Areola, Obafemi Awolowo University, Ile ife

Content Editor

Dr O.O. Irinoye & Dr T.O. Oladogba, Obafemi Awolowo University, Ile ife

Course Coordinator

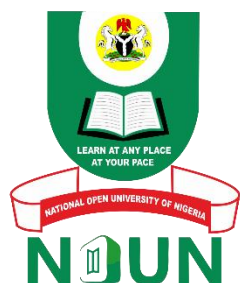
Adeolu Ejidokun, National Open University of Nigeria

Programme Leader

Jane-Frances Agbu, National Open University of Nigeria

Credits of cover-photo: Mr. Gbenga Aiyejumo, National Open University of Nigeria.

National Open University of Nigeria - 91, Cadastral Zone, Nnamdi Azikwe Express Way, Jabi, Abuja, Nigeria



www.nou.edu.ng centralinfo@nou.edu.ng
oer.nouonline.net oerunit@nou.edu.ng OER repository

Published in 2021 by the National Open University of Nigeria

© National Open University of Nigeria 2021



This publication is made available in Open Access under the [Attribution-ShareAlike4.0 \(CC-BY-SA 4.0\) license](https://creativecommons.org/licenses/by-sa/4.0/). By using the content of this publication, the users accept to be bound by the terms of use of the Open Educational Resources repository oer.nou.edu.ng of the National Open University of Nigeria.

The designations employed and the presentation of material throughout this publication do not imply the expression of any opinion whatsoever on the part of National Open University of Nigeria concerning the legal status of any country, territory, city or area or of its authorities, or concerning the delimitation of its frontiers or boundaries. The ideas and opinions expressed in this publication are those of the authors; they are not necessarily those of National Open University of Nigeria and do not commit the organization.

How to re-use and attribute this content

Under this license, any user of this textbook or the textbook contents herein must provide proper attribution as follows: “First produced by the National Open University of Nigeria” and include the NOUN Logo and the cover of the publication. The repository has a version of the course available in ODT-format for re-use.

If you use this course material as a bibliographic reference, then you should cite it as follows: “NSC 106: Medical Microbiology and Parasitology, Module 6, National Open University of Nigeria, 2021 at oer.nou.edu.ng

If you redistribute this textbook in a print format, in whole or part, then you must include the information in this section and give on every physical page the following attribution: Downloaded for free as an Open Educational Resource at oer.nou.edu.ng If you electronically redistribute part of this textbook, in whole or part, then you must retain in every digital file (including but not limited to EPUB, PDF, ODT and HTML) the following attribution:

Module 3 Vitamins and Trace Elements

Unit I The Fat Soluble Vitamins

1.0 Introduction

Vitamins are organic molecules that are required in small quantity for a variety of biochemical functions and which generally cannot be synthesized in the body but must be supplied in the diet or as supplement. These molecules serve the same roles in nearly all forms of life. Some are synthesized by intestinal microorganisms but in quantity that are not sufficient to meet our need. Human beings require at least 12 vitamins in the diet for various biochemical activities. Deficiency of vitamins can generate diseases in all organisms requiring them for important biochemical reactions. Vitamins can be grouped according to whether they are soluble in water or in non-polar solvents. Water soluble vitamins function as coenzyme, while fat soluble vitamins participate in diverse processes such as blood clotting and vision.

2.0 Objectives

At the end of this unit, you should be able to:

- define fat soluble vitamins (FSV) and list all the FSV
- describe vitamin A
- describe vitamin D
- describe vitamin E
- describe vitamin K
- give a detail description of Fat soluble vitamins.

3.0 Main Content

3.1 Vitamin A or Retinol

Two groups of compounds have vitamin A activity; the first group is called retinoid which comprises retinol, retinal and retinoic acid. They are preformed vitamin A, found only in foods of animal origin. The second group is carotenoid, found only in plants; they are composed of β -carotenes and related compounds. Carotenoids are cleaved in the intestinal mucosa by carotene dioxygenase to yield retinal which is reduced to retinol. Retinol is

stored in the liver of animals as lipid ester. Vitamin A is heat stable but sensitive to ultraviolet light (UV). This is why it is not good to put your palm oil in the sun when you want to liquefy it; application of little heat is better. This is a common practice in many African communities; it is not a good practice because the ultraviolet rays in the sun destroy the vitamin A present in the oil. Next time you see anyone doing this, please correct the person.

Sources of Vitamin A

The richest dietary sources of preformed vitamin A (retinol) are fish liver oils also known as cod liver oil. Other sources include liver of animals, milk and dairy products, dark green vegetables, yellow or red fruits, carrot and tomatoes. Palm-oil is the richest dietary source of carotenoids.

Functions of Vitamin A

Roles of vitamin a in vision- The role of vitamin A in vision was discovered by George Wald, who received the Nobel Prize in 1943 for this discovery. Vision is based on the absorption of light by photoreceptor cells in the eye. These cells are sensitive to light in a relatively narrow region of the electromagnetic spectrum, with wavelengths between 300 and 850nm. Vertebrates have two kinds of photoreceptor cells, called rods and cones because of their distinctive shapes.

Cones function in bright light and are responsible for colour vision, whereas rods function in dim light but do not perceive colour. A human retina contains about 3 million cones and 100 million rods. Rods are slender elongated structures densely packed with photoreceptors molecules. The photosensitive molecule is often called a visual pigment because it is highly coloured owing to its ability to absorb light. The photoreceptor molecule in rods is rhodopsin, which consists of the protein opsin covalently linked to 11-cis-retinal that serves as a prosthetic group. Iodopsin is the photoreceptor molecule present in the cones.

Gene expression and tissue differentiation

Another important function of vitamin A is the control of cell differentiation. All-trans-retinoic acid and 9-cis-retinoic acid regulate growth, development and tissue differentiation; they have different actions in different tissues. Retinoic acid binds to nuclear receptor and regulates the transcription of specific genes. There are two families of nuclear retinoid receptors: the retinoic acid receptors (RAR) bind All-trans-retinoic or 9-cis-retinoic acid and the retinoid X receptors (RXR) bind only 9-cis-retinoic acid.

Deficiency Vitamin A

Vitamin A deficiency is the most important preventable cause of blindness in the world. The earliest sign of deficiency is a loss of sensitivity to green light, followed by impairment to adapt to dim light followed by night blindness. More prolonged deficiency leads to xerophthalmia (dry and keratinization of the cornea and blindness). Growth retardation, dermatitis, anorexia and hypogeusia. Vitamin A also has an important role in differentiation of immune system cells. Mild deficiency of vitamin A leads to increased susceptibility to infectious diseases.

Toxicity Vitamin A

Human body has a limited capacity to metabolize vitamin A, and excessive intakes leads to accumulation beyond the capacity of binding protein (opsin), so that unbound vitamin A

causes tissue damage. Excess vitamin A is teratogenic to pregnant women (it can cause congenital deformity in the fetus).

Symptoms of toxicity affect the central nervous system and this include headache, nausea, anorexia (lack of appetite) and ataxia (defective muscular control leading to staggering). Liver and bones are also affected. Excessive dryness of skin is also a symptom of vitamin A toxicity.

3.2 Description of Vitamin D

Vitamin D is also known as cholecalciferol. Vitamin D could be thought of as a hormone rather than vitamin because it can be synthesized in the body; it is released into the blood circulation like hormones and has biochemical effects on target organs. Vitamin D is included in the list of vitamins, as it becomes an essential dietary factor when endogenous synthesis is inadequate to meet the physiological requirements. This condition is common in the temperate region where there may not be enough sunlight for greater part of a year. It is also common among women in some Arab nations where virtually all the parts of a woman's body is covered thereby preventing vitamin D synthesis in the skin.

There are two forms of vitamin D: The naturally produced vitamin D₃ or cholecalciferol is the form obtained from animal sources in the diet or made in the skin. It is produced in the skin from ultraviolet activation of 7-dehydrocholesterol. Artificially produced vitamin D₂ or ergocalciferol, is the form made in the laboratory by irradiating the plant sterol, ergosterol and it is the form most readily available for pharmaceutical use.

In the temperate regions, the plasma concentration of vitamin D is highest at the end of summer and lowest at the end of winter. In the tropical regions, vitamin D deficiency is not common due to availability of sunlight for most part of the year.

Dietary sources of vitamin D

The most reliable dietary sources of vitamin D are fortified foods. Milk for example is fortified to a level of 400 international units per quart. The recommended daily intake of vitamin D is 400 IU, irrespective of age. Other sources include the liver, cod liver oil and egg yolk.

Functions of vitamin D

Its main function is the regulation of calcium metabolism and absorption. Vitamin D is converted to calcitriol (1,25-dihydroxycholecalciferol), the active hormone by hydroxylation reactions in the liver and kidneys. It binds to receptor, structurally similar to the steroid receptors to form a complex that functions as a transcription factor thereby regulating gene expression.

Vitamin D deficiency

Vitamin D deficiency in childhood produces rickets, a disease characterized by inadequate calcification of cartilage and bone as a result of poor absorption of calcium. Similar problems occur in adult as a result of demineralization of bone, especially in women who have little exposure to sunlight. In adults, vitamin D deficiency leads to softening and weakening of bones, a condition called osteomalacia. Osteoporosis is another bone disease associated

with old age. It is described as the loss of bone density caused by excessive absorption of calcium and phosphorus from the bone.

Toxicity of vitamin D

High concentration of vitamin D in the plasma can lead to contraction of blood vessels and calcinosis, i.e. the calcification of soft tissues such as the liver and kidneys. This has serious consequences on health and can lead to death.

3.3 Description of Vitamin E

Vitamin E, also known as tocopherol is the generic name for two families of compounds. Tocopherols (α , β , γ and δ type) differ in the number and position of the methyl groups on the ring. The α -tocopherol is the most potent and it is usual to express vitamin E intake in terms of milligrams α -tocopherol equivalent. Tocotrienols are structurally related to tocopherols but they are less potent and also contain unsaturated hydrocarbon side chains.

Dietary sources of vitamin E

The major dietary sources of vitamin E are fats and oils with different tocopherol content. The richest sources are: Soya and corn oils (50-150 mg/100gm) and Palm oil (20-70 g/100gm), Coconut and olive oils are relatively low in vitamin E content (1-10mg/100gm). The major site of vitamin E storage is the adipose tissue. Synthetic vitamins E are also available as dietary supplements.

Functions of vitamin E

Vitamin E does not have a precisely defined metabolic function. It acts as lipid-soluble antioxidant in cell membranes. Its antioxidant functions include radical chain-breaking and free radical trapping in cell membrane and plasma lipoprotein by reacting with the lipid peroxide radicals formed by peroxidation of poly unsaturated fatty acids.

Vitamin E deficiency

Vitamin E deficiency in human is rare. The only known symptom of vitamin E deficiency is haemolytic anaemia due to an increased red blood cell fragility and damage

Toxicity of vitamin E

There is no record of vitamin E toxicity but because it is fat soluble, too much of it may be toxic. Unlike the other fat soluble vitamins such as vit A, K and D, vit E does not seem to have any known toxic effects.

3.4 Description of Vitamin K

Vitamin K derived its name from German word *koagulation*. The most important function of vitamin K is its role in the synthesis of blood clotting proteins, hence it is called blood clotting vitamin. There are two naturally occurring forms of vitamin K, the first is phyloquinone; the normal dietary source found in green vegetables. The second is known as menaquinones, synthesized by the intestinal bacteria of children but adult cannot synthesize

vitamin K. Menadiones and menadioldiacetate are synthetic compounds that can be metabolized to phyloquinone.

Dietary sources of vitamin K

Excellent sources of vitamin K are cabbage and green vegetables, other sources are tomatoes, cheese, meat and egg yolk.

Functions of vitamin K

Synthesis of blood clotting proteins- vitamin K has been known for many years to be essential for the synthesis of prothrombin and several other clotting factors. Vitamin K participates in the carboxylation of glutamate residues to γ -carboxyglutamate, which makes modified glutamic acid a much stronger chelator of Ca^{2+} . The results of studies of the abnormal prothrombin synthesized in the absence of vit K or in the presence of vitamin K antagonists (these are the compounds that prevent vitamin K from performing its functions) such as dicoumarol, revealed the mode of action of this vitamin.

Dicoumarol is found in spoiled sweet clover leaves and causes a fatal hemorrhagic disease in cattle fed on this hay. This coumarin derivative is used clinically as an anticoagulant to prevent thromboses in patients prone to clot formation. Dicoumarol and such related vitamin K antagonists as warfarin also serve as effective rat poisons.

Deficiency of vitamin K

The clinical manifestation of a Vitamin K deficiency is hemorrhage, when there is little cut, bleeding may continue for a very long time.

Toxicity of vitamin K

High doses of the naturally occurring fat soluble form of Vitamin K (K_1) appear to be non-toxic, but the water-soluble forms of menadione (K_3) have produced serious side effects in high doses, especially in newborn infants. Large doses of menadione given to newborns or their mothers during labour have resulted in hemolytic anemia. Premature infants have less tolerance to excess Vitamin K than full-term infants do. Adult toxicity signs are primarily circulatory and involve a variety of cardiac and pulmonary signs.

4.0 Conclusion

5.0 Summary

In this unit, you have been taken through the following:

- Vitamin A or Retinol
- Description of Vitamin D
- Description of Vitamin E
- Description of Vitamin K

6.0 Self Assessment Exercise

1. Define fat soluble vitamins (FSV) and list all the FSV
2. Describe vitamin A
3. Describe vitamin D
4. Describe vitamin E
5. Describe vitamin K
6. Give a detail description of Fat soluble vitamins

Activity: Your course facilitator would inform you about a practical assignment you expected to carry out.

Unit 2 Water Soluble Vitamins

1.0 Introduction

All water soluble vitamins are readily soluble in water and are absorbed without the involvement of fat. Excess intakes are excreted in the urine but some of them have some side effects. Some of the vitamins are stored for a short period while many are stored in the body for up to 2 months. These water soluble vitamins function as co-enzymes; they must be metabolically converted to their active forms for them to be functional but vitamins C and biotin are used directly without conversion.

2.0 Objectives

At the end of this unit, you should be able to:

- list the biochemical functions, deficiency and natural sources of vitamin B1 (Thiamine).
- list the biochemical functions, deficiency and natural sources of vitamin B2
- describe the biochemical functions, deficiency and natural sources of vitamin B3 (Niacin)
- list the biochemical functions, deficiency and natural sources of vitamin B5 (Panthotenic acid)
- enumerate the biochemical functions, deficiency and natural sources of vitamin B6 (Pyridoxine)
- list the biochemical functions, deficiency and natural sources of vitamin H (Biotin)
- itemise the biochemical functions, deficiency and natural sources of vitamin B9 (Folic acid)
- describe the biochemical functions, deficiency and natural sources of vitamin B12
- list the biochemical functions, deficiency and natural sources of vitamin C.

3.0 Main Content

3.1 Vitamin B1 (Thiamine)

Functions of vitamin B1

- Thiamine reacts with ATP to form Thiamine pyrophosphate (TPP), the active form of vitamin B1 in biochemical reactions.
- TPP is a co-enzyme in the decarboxylation of pyruvate and α -keto glutarate and also in transketolase and transaldolase reactions.
- Decarboxylation of pyruvate is a crucial reaction in nervous system; this was explained in section 2.4.

Deficiency of vitamin B1

- Thiamine deficiency is associated with elevated levels of pyruvate and lactate.
- Its deficiency also causes a clinical disorder known as Beriberi, Oedema and an array of other abnormalities.

Natural sources of vitamin B1

- Thiamine is available in a wide variety of foods making its deficiency very rare. Some of the sources include unpolished rice, whole grains such as maize, yeast, nuts and potatoes.
- It is easily destroyed by heat, so the thiamine content of foods is lowered by cooking.

Since Vitamin B1 functions in the release of energy from fuel molecules, its requirement is linked to caloric intake. The recommended daily allowance (RDA) of Thiamine is 1.0-1.5mg for most individuals. However higher quantity is required during pregnancy. Dietary deficiency is common in people with Anorexia, diarrhea, alcoholics and post-operative patients

3.2 Vitamin B2 (Riboflavin)

Functions of vitamin B2

1. Riboflavin is the electron carrier co-enzymes that exist as FAD and FMN.
2. They function in a variety of redox reactions catalysed by oxidases, reductases and dehydrogenases (fatty acid synthesis, TCA cycle and amino acid synthesis)
3. They are necessary for aerobic respiration and tissue maintenance.

Deficiency of Riboflavin

Riboflavin deficiency is widespread but not fatal. Deficiency is characterized by cheilosis, desquamation and inflammation of the tongue, when this happens the person will not be able to take food containing pepper.

Natural sources of Riboflavin

Riboflavin is found in a wide variety of foods, especially liver, kidney and green leafy vegetables. The major sources are milk and dairy products. Because of its intense yellow colour, it is used as food additives. The RDA for riboflavin is 1.2mg, but pregnant and lactating mother may require more.

3.3 Vitamin B3 (Niacin)

Functions of vitamin B3

Niacin (also known as Nicotinic acid) and its amide derivative nicotinamide are the precursor of the co-enzymes NAD⁺ and NADP⁺. They are essential coenzymes in numerous cellular reactions such as lipid biosynthesis, the pentose phosphate pathway and amino acid metabolism.

Deficiency of vitamin B3

Niacin deficiency causes the disease pellagra. The major symptoms are photosensitive dermatitis on the skin, impaired digestion, diarrhea and mental confusion.

Natural sources of Vitamin B3

Sufficient quantity can be synthesized endogenously from tryptophan. (Take note of the vitamins that are synthesized in sufficient quantity, we have mentioned vitamin D). It is present in meats, fish and nuts. The RDA for Niacin is 13mg. Excess niacin can cause liver damage.

3.4 Vitamin B5 (Pantothenic acid)

Functions of vitamin B5

1. Pantothenate is a precursor of co-enzyme A, that participates in acyl transfer reactions.
2. Coenzyme A is the carrier for acetyl groups obtained from the degradation of carbohydrates and lipids.
3. Coenzyme A also carries the acetyl and malonyl groups used in fatty acid synthesis.

Deficiency

- Vitamin B5 deficiency has not been observed in humans. No RDA has been set for Vitamin B5 yet. Most individuals ingest between 5 and 20mg of pantothenate per day.

7.4.3 Natural sources

- Beef liver, peanuts, Soybeans and wheat germ are all rich in pantothenate.

3.5 Vitamin B6 (Pyridoxine)

Functions of vitamin B6

1. The biologically active forms of pyridoxine are pyridoxamine phosphate and pyridoxal phosphate and are mostly involved in amino acid metabolism.
2. They function in various transamination and oxidation reactions. Some women use it for premenstrual pain.

Deficiency of vitamin B6

The deficiency symptoms include irritability, mental confusion, convulsions (in infants), peripheral neuropathy and inflammation of the mouth.

Natural Sources

Since the pyridoxine coenzymes are involved primarily with amino acid metabolism their daily requirement varies with the daily protein intake. The vitamin is present in meat, fish and fruits. The RDA is 2.2mg for men and 2.0mg for women.

3.6 Vitamin H (Biotin)

Functions of Biotin

1. Biotin which is used by cells without modification is the coenzyme for cellular carboxylation reactions.
2. The carboxylation of acetyl CoA to malonyl CoA in preparation for fatty acid synthesis is an example of biotin dependent carboxylation.

1. Deficiency of Biotin

- Its deficiency has not been observed in humans on a normal diet. Raw egg contains a protein called avidin that binds biotin very tightly. Biotin deficiency can occur in a person who takes raw eggs regularly.
- It is synthesized by the intestinal flora in amounts believed sufficient to meet the daily needs of man (This is the third vitamin that is synthesized in sufficient quantity by man).

Natural Sources

The intake of the average healthy adult is 100 to 300µg/day. Sources include liver, peanuts etc.

3.7 Vitamin B9 (Folic acid)

Functions of Folic acid

Folic acid, also called folicin is converted to tetrahydrofolate, which is the primary carrier of one carbon units in the cell.

Deficiency

Symptoms associated with folic acid deficiency are megaloblastic and macrocytic anemia, growth failure. All the symptoms disappear upon administration of folic acid.

Natural sources

Folic acid is synthesized by the intestinal bacteria, so the daily dietary requirement is difficult to measure. The RDA for healthy individuals has been set at 400µg with higher allowances for pregnant and lactating women. Good sources of folic acid include Beef, Chicken, liver, peanuts and brewer's yeast.

3.8 Vitamin B12

Functions of vitamin B12

- Vitamin B12 consists of cobalamin and its active derivatives, including cyanocobalamin and methyl cobalamin.
- Cobalamin is an essential cofactor in the conversion of homocysteine to methionine and in the conversion of methyl malonylcoA to succinyl-coA.

Deficiency

- Vitamin B12 deficiency impairs the metabolism of folic acid and this disturbs red blood cell synthesis.
- The clinical symptoms of vitamin B12 deficiency are collectively called pernicious anemia and include megaloblastic anemia, changes in the intestinal mucosa and in extreme cases, neuropathy.

Natural Sources

- The vitamin B12 RDA for healthy individuals has been set at 3µg. The actual daily intake for most individuals is from 5 to 15µg/day.
- Most vitamin B12 in the body is concentrated in the liver.
- In general foods derived from animals contain more of vitamin B12 than foods derived from plants.

3.9 Vitamin C (Ascorbic acid)

Functions of vitamin C

- The active forms of Vitamin C are ascorbic and dehydroascorbic acids. It is known as antiscorvy agent.
- The symptoms of scurvy are swollen gum, loss of teeth, skin lesions, pain and weakness (legs).
- Vitamin C participates in hydroxylation reactions and also promotes intestinal absorption of iron. They participate in amino acid metabolism, the synthesis of norepinephrine and the synthesis of collagen.

Deficiency of vitamin C

- Vitamin C deficiency causes scurvy. Other symptoms of scurvy include impaired wound healing and changes in bone development and growth.
- Many of these symptoms appear to be the result of the defective collagen synthesized in the absence of Vitamin C.
- Deficiency of vitamin C can also cause impaired growth in children. In severe cases, skin becomes rough, dry and develop hyperkeratotic (scaly) changes in the hair follicles.
- Drugs known to increase the urinary excretion of ascorbate include aspirin and barbiturates. Chronic diarrhea may contribute to vitamin C deficiency.

Natural Sources

Good sources of vitamin C are peppers, papaya and citrus fruits. The RDA for vitamin C is 60mg, based on the amount required to prevent or cure scurvy. More Vitamin C can be required during pregnancy and lactation.

4.0 Conclusion

5.0 Summary

In this study unit, you have been exposed to the following in details:

- Vitamin B1 (Thiamine)
- Vitamin B2 (Riboflavin)
- Vitamin B3 (Niacin)
- Vitamin B5 (Pantothenic acid)
- Vitamin B6 (Pyridoxine)
- Vitamin H (Biotin)
- Vitamin B9 (Folic acid)
- Vitamin B12
- Vitamin C (Ascorbic acid)

6.0 Self Assessment Exercise

1. List the biochemical functions, deficiency and natural sources of vitamin B1 (Thiamine).
2. List the biochemical functions, deficiency and natural sources of vitamin B2
3. Describe the biochemical functions, deficiency and natural sources of vitamin B3 (Niacin)
4. List the biochemical functions, deficiency and natural sources of vitamin B5 (Panthotenic acid)
5. Enumerate the biochemical functions, deficiency and natural sources of vitamin B6 (Pyridoxine)
6. List the biochemical functions, deficiency and natural sources of vitamin H (Biotin)
7. Itemise the biochemical functions, deficiency and natural sources of vitamin B9 (Folic acid)
8. Describe the biochemical functions, deficiency and natural sources of vitamin B12

9. List the biochemical functions, deficiency and natural sources of vitamin C

Activity: Your course facilitator would inform you about a practical assignment you expected to carry out.

7.0 References/Further Reading

Katherine, M. A. Rogers and William N. Scott (2011). Nurses! Test yourself in anatomy and physiology

Kathryn, A. Booth, Terri. D. Wyman (2008). Anatomy, physiology, and pathophysiology for allied health

Keith L.M, Persuade T.V.N (2006). The Developing Human Clinically Oriented Embryology 8th Edition Lippincott Williams & Wilkins

Kent, M. Van De Graff, R.Ward Rhees, Sidney P. (2010). Schaum's outline of human anatomy and physiology 3rd edition.

Philip, T. (2012). Seeley's principles of anatomy & physiology 2nd edition.

Sadler, T.W (2004). Langman's Medical Embryology 9th edition.

Unit 3 Trace Elements

1.0 Introduction

Dietary minerals are the [chemical elements](#) required by living [organisms](#), other than the four elements [carbon](#), [hydrogen](#), [nitrogen](#), and [oxygen](#) present in common [organic molecules](#).

For any nutrient, there is a range of intake between that which is clearly inadequate, leading to clinical deficiency disease, and that which is so much in excess of the body's metabolic capacity that there may be signs of toxicity. Between the 2 extremes, there is a level of intake that is adequate for normal health and the maintenance of metabolic integrity. Individuals do not have the same requirement for nutrients, even when calculated on the basis of body size or energy expenditure.

Therefore, in order to assess the adequacy of diets, it is necessary to set a reference level of intake high enough to ensure that no one either suffers from deficiency or is at risk of toxicity. Many of the essential minerals are widely distributed in foods, and most people eating a mixed diet are likely to receive adequate intakes, although supplements can be used when some requirements are not adequately met by the diet, or when chronic or acute deficiencies arise from pathology or injury, etc.

2.0 Objectives

At the end of this unit, you should be able to:

- distinguish between Macro and Microminerals
- list important microminerals (Trace Elements) and enumerate the functions, food sources, required daily allowance, deficiency and toxicity symptoms of each trace mineral studied.

3.0 Main Content

3.1 Macro vs Microminerals

Dietary Minerals include the macro and microminerals. Macrominerals are required in the diet in large amounts (>100mg/day). They represent about 80% of body organic matter and include [calcium](#), [phosphorus](#), [potassium](#), [sulfur](#), [sodium](#), [chlorine](#), and [magnesium](#).

Microminerals or Trace elements are needed in doses < 100mg per day. Important "trace" or minor minerals, include [iron](#), [cobalt](#), [copper](#), [zinc](#), [molybdenum](#), [iodine](#), [selenium](#) and cobalt. .

Some minerals are necessary for the body, but their exact functions are unknown. Such include Chromium, Nickel, Bromine, Lithium and Barium. Non essential minerals found as contaminants in foodstuffs include rubidium, silver, gold and bismuth. Toxic minerals include Al, Pb, Cd and Hg.

Table 3.1: Classification of Minerals

Macrominerals	Microminerals	Minerals whose functions are unknown	Food Contaminants
Calcium	Zinc	Chromium	Rubidium
Phosphorus	Iron	Nickel	Silver
Potassium	Copper	Bromine	Gold
Sulphur	Iodine	Lithium	Bismuth
Sodium	Fluorine	Barium	
Chlorine	Selenium		
Magnesium	Cobalt		

3.2 Trace Elements

Zinc

Total zinc content of the body is about 2g, out of which 60% is in skeletal muscles and 30% in bones. The highest concentration of Zinc is seen in Hippocampus area of brain and prostate fluid. More than 300 enzymes are zinc-dependent, including RNA and DNA polymerases, alkaline phosphatase and carbonic anhydrase.

It also forms what is known as zinc fingers (Zn^{2+}) coordinated to four amino acid side chains), which provide structural stability to many proteins and are important for protein-protein interactions. These are found in many signal transduction proteins. Zn is also involved in DNA and protein synthesis as well as transport of vitamin A, taste perception, wound healing, Zinc plays a vital role in fertility. In males, it protects the prostate gland from infection (prostates) and ultimately from enlargement (prostatic hypertrophy). It also helps maintain sperm count and mobility and normal levels of serum testosterone.

Zinc is important during pregnancy, for the growing foetus whose cells are rapidly dividing. Zinc also helps to avoid congenital abnormalities and pre-term delivery. Zinc is vital in ensuring proper growth and development in infants, children and teenagers.

Sources: Meat, Shellfish, Poultry, Whole grains, Vegetable, Cheese



Fig 3.1: Food Sources of Zinc

RDA: 11mg for men, 8mg for women

Deficiency: Zn deficiency in children is marked by poor growth and impairment of sexual development. In both children and adults, it results in poor wound healing and dermatitis as well as impaired immune function. Toxicity effects include loss of appetite, impaired immunity, decreased HDL, iron and copper deficiencies. Toxicity is common in welders due to inhalation of zinc oxide fumes.

Iron

Total body content is 3-5g, 75% of this is found in blood and the rest in liver, bone marrow and muscles. Iron is present in almost all cells. Blood contains 14.5g of Hb per 100ml. About 75% of total iron is in hemoglobin, 5% is in myoglobin and 15% in ferritin. Iron carries oxygen as part of haemoglobin in blood or myoglobin in muscles. It is required for cellular energy metabolism. Transferrin is the transport form while Ferritin is the storage form of iron. Transferrin is a glycoprotein, with a mol wt of 76,500 Daltons. Total iron binding capacity(TIBC) is a measure of the blood's capacity to bind iron with transferring. The ref range is about 400mg/100ml.

One third of this capacity is saturated with iron. Transferrin has a half-life of 7-19 days, and is a useful index of nutritional status. One molecule of transferrin can bind two ferric ions. In blood, ceruloplasmin is the ferroxidase, which oxidizes ferrous to ferric state. Transferrin

receptors are present on most of the body cells, especially on cells which synthesize heme. The iron-transferrin complex is taken up by the body cells by the receptor mechanism.

RDA: for men 8mg, women (19-50yrs), 18mg, women > 50yrs 8mg.

Sources: Red meat, fish, poultry, eggs, dried fruits, leafy vegetables, pulses.

Deficiency: Includes anaemia characterized by weakness, fatigue, headache and impaired mental and physical performance. It also impaired immunity and pale skin. In iron deficiency anaemia, TIBC is increased but serum iron level is reduced. Deficiency is caused by poor nutrition, hookworm infection, repeated pregnancies, chronic blood loss, and lead poisoning. Excess iron is called hemosiderosis. Hemosiderin pigments are golden brown granules, seen in spleen and liver. It occurs in persons having repeated blood transfusions. Prussian blue tests are +ve for these pigments. Toxicity leads to GI distress, infections, fatigue, joint pain, skin pigmentation and organ damage.

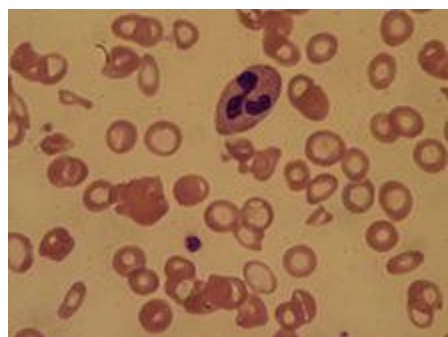


Fig 3.2a: Iron deficiency anaemia



Fig 3.3b. Hemosiderin deposit showing pale rbc's varying in shapes & sizes

Copper

Total body copper is about 100mg. It is found in muscles, liver, bone marrow, brain, kidney, heart and hair. It is a required component of many redox enzymes. Copper containing enzymes are ceruloplasmin, cytochrome oxidase, tyrosinase, superoxide dismutase and others. Required for iron absorption and incorporation of iron into hemoglobin. Only about 10% dietary copper is absorbed. Excretion is mainly through bile.

Sources: Legumes, nuts and seeds, whole grains, organ meats, drinking water.



Fig 3.3: Food sources of copper

RDA: 900µg

Deficiency: Results in microcytic normochromic anaemia, cardiovascular diseases, defective cross-linking of connective tissue and hypo pigmentation of hair. Toxicity is manifested as diarrhoea and blue-green discoloration of saliva. Copper poisoning may result in hemolysis, hemoglobinuria, proteinuria and renal failure. Toxicity could occur from eating acid foods cooked in uncoated copper cookware or exposure to excess copper in drinking water or other environmental sources. Could result from Copper poisoning, or Wilson's disease Results in vomiting, hematemesis, hypotension, coma, GI distress, jaundice.



Fig 3.3b: Kayser-Fleischer ring due to Cu deposition as a result of Wilson's disease

Fluoride

Fluoride is known to prevent caries. In the pits and fissures of premolar and molar teeth, bacterial fermentation of residual food leads to acid production. The acid removes enamel and dentine to expose the pulp, leading to inflammation and toothache. Presence of fluoride will result in a fluoroapatite layer on the enamel, which protects it from decay. The safe limit of F is about 1ppm in water (= 1mg in 1,000ml of water)

RDA: 4mg for men, 3mg for women. Toxicity leads to fluorosis (pitting and discoloration of teeth with alternate areas of osteoporosis, osteosclerosis and brittle bones), intestinal upset, loss of appetite and loss of weight.

Selenium

Selenium intake depends on the nature of soil in which food crops are grown. In mammals, glutathione peroxidase is the most important Se containing enzyme. 5'-de-iodinase, which converts thyroxine to T3 also contains Se. Se Concentration in testis is the highest in adult tissue. It is necessary for normal development of spermatozoa. Se also acts as a non specific intracellular anti-oxidant, its action being complementary to that of Vitamin E.

RDA: 50-100µg/ day.

Deficiency: Causes Liver necrosis, cirrhosis, cardio myopathy and muscular dystrophy.

Se Toxicity is called selenosis. Symptoms include hair loss, falling of nails, diarrhea, weight loss and garlicky odour in breath.

Iodine

Dietary iodine is efficiently absorbed and transported to the thyroid gland, where it is stored and used for the synthesis of triiodothyronine and thyroxine. These hormones function in regulating the basal metabolic rate of adults and the growth and development of children. Also functions as antioxidant for extrathyroidal organs such as mammary and salivary glands and for gastric juice production.

Sources: seafood, dairy products, iodized salt **RDA:** 150µg

Deficiency: Very common results in an enlargement of the thyroid gland (Goitre)

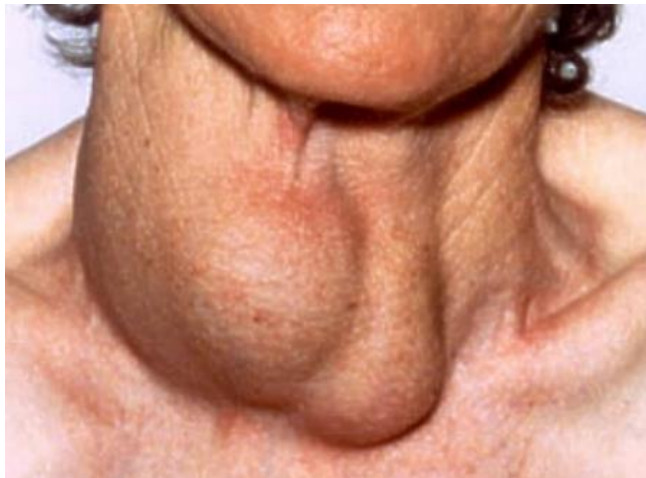


Fig 3.4: Polynodular goitre

Glossary

Tetany: A syndrome of sharp flexion of wrist and ankle joints, (carpopedal spasm), muscle twisting, cramps and convulsions due to hyperexcitability of nerves and muscles.

Microcytic, normochromic anaemia: A type of anaemia characterized by small rbcs. Here, the rbcs are not pale as opposed to hypochromic anaemia.

Osteosclerosis: Elevation in bone density, normally detected by an area of whiteness.

Cirrhosis: Scarring of the liver as a consequence of chronic liver disease.

Cardiomyopathy: Heart muscle disease. Deterioration of the functions of the myocardium.

Muscular Dystrophy: Weakening of musculoskeletal system. Characterized by progressive skeletal muscle weakness, defects in muscle protein and death of muscle cells and tissue.

4.0 Conclusion

5.0 Summary

In this unit, you have been exposed to the following concepts:

i. Trace Elements ii. Macro iii. Microminerals

6.0 Self Assessment Exercise

1. Distinguish between Macro and Microminerals
2. List important microminerals (Trace Elements) and enumerate the functions, food sources, required daily allowance, deficiency and toxicity symptoms of each trace mineral studied.

Activity: Your course facilitator would contact you on a practical assignment you are to carry out.

7.0 References/Further Reading

Katherine, M. A. Rogers and William N. Scott (2011). Nurses! Test yourself in anatomy and physiology

Kathryn, A. Booth, Terri. D. Wyman (2008). Anatomy, physiology, and pathophysiology for allied health

Keith L.M, Persuade T.V.N (2006). The Developing Human Clinically Oriented Embryology 8th Edition Lippincott Williams & Wilkins

Kent, M. Van De Graff, R.Ward Rhees, Sidney P. (2010). Schaum's outline of human anatomy and physiology 3rd edition.

Philip, T. (2012). Seeley's principles of anatomy & physiology 2nd edition.

Sadler, T.W (2004). Langman's Medical Embryology 9th edition.