CHAPTER 20

VITAMINS AND MINERALS

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Vitamins are natural nutrients that are required for survival. Vitamins are required for proper body function. Most vitamins are not produced by us, at least not in sufficient quantities to meet our needs. As a result, they must be obtained through our diet. A mineral is a chemical element that originates in the Earth and never loses its identity. Minerals are crystalline inorganic salts. Minerals enter the body and stay there until they are eliminated. They are unable to be transformed into anything else. Heat, air, acid, or mixing have no effect on minerals. Vitamins and minerals are found in food in minute amounts compared to other nutrients like protein, carbs, and fat. Vitamins and minerals are referred to as micronutrients because we consume in less quantity.

Each vitamin and mineral has a special role in the body, making them one-of-a-kind and irreplaceable. No single diet supplies all of the vitamins and minerals that the body requires, and inadequate nutrient consumption leads to deficiencies. To meet the body's vitamin and mineral requirements, a diverse diet is essential.

There are four fat-soluble vitamins known. This indicates that the body needs consume fat or oil in order for the vitamins to be absorbed. A, D, E, and K are fat-soluble vitamins. Vitamin C and the B-complex, which includes vitamins B1, B2, B6, B12, niacin, folic acid, biotin, pantothenic acid, and choline, are water-soluble. Macrominerals and trace minerals/trace elements are the two types of minerals.

Macrominerals are required by the body in greater quantities (above 100 mg daily) than trace elements, as their name implies. We need to eat enough and a variety of foods to meet our macromineral requirements. The trace minerals get their name from the fact that they are found in minute concentrations in the body. If we combined the trace mineral requirements, we'd only get a speck of dust, barely enough to fill a teaspoon. They are, nevertheless, just as vital as macrominerals and other nutrients. Food trace mineral content is influenced by soil and water composition as well as processing methods.The body utilises more than two dozen minerals in diverse capacities. Only the minerals whose intake may become insufficient if access to a diversified food is restricted are highlighted in this book. Plants and microorganisms can manufacture the vitamins required for metabolism, but humans and animals have lost this ability as a result of evolution. Humans and animals must eat vitamins because they lack the enzymes needed to create them in their bodies (with the exception of vitamin D, which is synthesised via the action of sunlight). Choline is the newest member of the essential nutrient family.

Vitamin-rich meals have been recognised as necessary for health and well-being for over 3,500 years. The earliest reports on this issue concern the usage of specific foods, such as liver, which contains vitamin A, to avoid disorders like night blindness.

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Nonetheless, until recently, the concept of vitamins was mostly unknown. Our understanding of how vitamins and minerals work in our bodies has vastly improved since the turn of the twentieth century. Between 1928 and 1967, 20 Nobel Prizes were awarded in the field of vitamins, demonstrating this awareness. Mineral matter accounts for only 5% of a person's weight. Minerals, on the other hand, are necessary for a variety of body activities, including bone formation, hormone production, and heartbeat regulation. Minerals are required for proper growth and development.

The majority of minerals in human diets come from plants or animals. Minerals are obtained by plants from the soil. Plant mineral content will vary depending on where the plant grew and how much fertiliser it received because soil mineral content differs globally. Minerals may be present in the water we drink, and this varies depending on where we live.

Vitamins are over-marketed, over-prescribed, and over-used as a group. Vitamin myths abound, including "vitamins invigorate the body," "vitamin shortage is associated with any physical illness," "vitamin consumption in a typical diet is precariously minimal," and "vitamins are innocuous." Traditional vitamin classifications include:

(a) Fat-soluble vitamins (A, D, E, K): These vitamins (excluding vitamin K) are retained in the body for long periods of time and can induce cumulative toxicity if consumed in excessive doses on a regular basis. Some, like hormones, interact with specific cellular receptors.

(b) Water-soluble (B complex, C): Excess is excreted with low risk of harm. They serve as cofactors for specific intermediate metabolism enzymes.

FAT-SOLUBLE VITAMINS

VITAMIN A

Source and chemistry Vitamin A is found in a variety of forms in nature. Vitamin A1 (retinol) is an unsaturated alcohol with a 'ionone' ring. The liver oils of marine fish (cod, shark, halibut) are abundant. Egg yolk, milk, and butter all contain significant amounts.

Freshwater fishes contain dehydroretinol (Vit A2). Green plants (carrot, turnip, spinach) contain carotenoids, which are colours. The most important carotenoid is carotene. As such, it is inactive; one molecule divides into two retinol molecules. On a typical diet, a man gets half of his vitamin A from retinol esters and the other half from

carotenoids. 1 micro gram of retinol equals 3.3 IU of vitamin A. 1 retinol equivalent = 6 micro gram dietary carotene. (due to insufficient provitamin utilisation).

Function and actions in the body

a) (a) Visual cycle Retinal is a component of the light-sensitive pigment Rhodopsin, which is formed by rods during dark adaptation and is generated via reversible oxidation of retinol. This pigment is bleached and broken into its constituents by low light, resulting in the generation of a nerve impulse via the G-protein Transducin. Retinal that has been released is repurposed. The cones manufacture a similar pigment called iodopsin, which is important for vision in strong light, colour vision, and primary dark adaption. Rods are more impacted than cones in vitamin A deficit; long-term depletion causes irreversible structural alterations and lifelong night blindness.

b) Epithelial tissue Vitamin A. promotes epithelia development and structural integrity throughout the body. It also increases mucus secretion, prevents keratinization, and boosts infection resistance. It appears to have the ability to slow the progression of epithelial cancers. Vitamin A is also necessary for bone development.

c) Reproduction Retinol is required for spermatogenesis and foetal development to progress.

d) Immunity Vitamin A deficiency increases infection susceptibility. For correct antibody response, normal lymphocyte proliferation, and killer cell function, a physiological level of vitamin A appears to be essential.

Symptoms of deficiency

Because vitamin A is stored in the liver, deficient symptoms only show after a long period of time, although vitamin A deficiency is common, especially among newborns and children in developing nations. Symptoms include:

- Xerosis (dryness) of eye, 'Bitot's spots', keratomalacia (softening of cornea), corneal opacities, night blindness (nyctalopia) progressing to total blindness.
- Dry and rough skin with papules (phrynoderma), hyperkeratinization, atrophy of sweat glands.
- Keratinization of bronchopulmonary epithelium, increased susceptibility to infection.
- Unhealthy gastrointestinal mucosa, diarrhea

- Urinary stone production is more likely due to the shedding of the ureteric epithelial lining, which acts as a nidus.
- Sterility caused by spermatogenesis problems, miscarriages, and foetal abnormalities
- Growth delay and particular sense impairment.

Therapeutic applications

1. Vitamin A insufficiency prevention during childhood, pregnancy, breastfeeding, hepatobiliary disorders, and steatorrhoea: 3000–5000 IU/day
2. Treatment of established vitamin A deficiency: 50,000–100,000 IU i.m. or orally for 1–3 days, then intermittent additional dosages
3. Skin conditions such as acne, psoriasis, and ichthyosis. Retinoic acid and retinoids of the second or third generation are employed.

Hypervitaminosis A Nausea, vomiting, itching, erythema, dermatitis, exfoliation, hair loss, bone and joint problems, loss of appetite, irritability, bleeding, elevated intracranial tension, and chronic liver disease have all been reported after consuming large amounts of retinol (100,000 IU daily for months). Retinol excess is also teratogenic in both animals and humans. A daily consumption of 20,000 IU is not recommended.

VITAMIN E

Therapeutic applications

Source and chemistry Vit E activity is found in a variety of tocopherols, the most abundant and strong of which is tocopherol. The d-isomer is stronger than the l-isomer. The richest source is wheat germ oil, followed by cereals, nuts, spinach, and egg yolk. The -tocopherol equivalent of 1 mg of d-tocopherol is 1.49 IU of vitamin E. The daily vitamin E requirement is estimated to be 10 mg. It is exacerbated by a high polyunsaturated fat diet.

Therapeutic applications

Function and actions in the body Vit E is an antioxidant that protects unsaturated lipids in cell membranes, coenzyme Q, and other compounds from free radical oxidation damage while also reducing the production of harmful peroxidation products. Antioxidants such as cystein, methionine, selenium, and chromenols counteract some vit E insufficiency symptoms in animals when polyunsaturated fats are fed to them. Other deficient symptoms are not addressed by these unrelated

antioxidants, suggesting that vitamin E may have a more particular effect or structural involvement in biological membranes.

Symptoms of deficiency

Recurrent miscarriage, degenerative alterations in the spinal cord, skeletal muscles, and heart, and haemolytic anaemia are all symptoms of experimental vitamin E insufficiency in animals. Although there is no clear-cut vitamin E deficiency condition in humans, it has been linked to certain neuromuscular illnesses in children, neurological deficits in hepatobiliary disease, and some cases of haemolytic anaemia.

Therapeutic applications

1. There is no clinical evidence of primary vitamin E insufficiency. Patients at risk may receive additional doses (10–30 mg/day).
2. G-6-PD deficiency—long-term therapy with 100 mg/day enhances erythrocyte survival time.

Vit E has been promoted for recurrent abortion, sterility, menopausal syndrome, pregnancy toxaemia, atherosclerosis, ischaemic heart disease, cancer prevention, several skin diseases, neurodegenerative disorders prevention, postherpetic neuralgia, scleroderma, and many other conditions due to its antioxidant properties, but there is no convincing evidence of benefit.

Toxicity

Even high dosages of vitamin E for long periods of time have not caused any substantial toxicity, but creatinuria and decreased wound healing have been recorded, as have gastrointestinal cramps, loose movements, and tiredness. Iron therapy can be hampered by vitamin E.

WATER-SOLUBLE VITAMINS

THE VITAMIN B COMPLEX GROUP

Thiamine (vit B1) Source and chemistry A crystalline, colourless chemical with a pyrimidine and thiazole ring. It can be found in cereals' outer layers (rice polishing), legumes, nuts, green vegetables, yeasts, eggs, and meat. Physiological Significance- It works as a coenzyme in carbohydrate metabolism, including decarboxylation of ketoacids and hexose monophosphate shunt, after being converted to Thiamine pyrophosphate in the body.

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Carbohydrate intake—about 0.3 mg/1000 Kcal—determines the need. It appears to be involved in neuromuscular transmission as well.

Deficiency Symptoms: The dry and wet variants of thiamine deficient beriberi are as follows:

Dry Beriberi: Polyneuritis with numbness, tingling, and other neurological symptoms are common. Hyperesthesia, muscle weakness and atrophy causing 'wrist drop,' 'foot drop,' and paralysis, mental changes, sluggishness, impaired memory, loss of appetite, and constipation are all symptoms.

Wet beriberi: affects the cardiovascular system the most, causing palpitations, dyspnea, and a high output.

ECG alterations and cardiac failure Protein insufficiency is frequently linked to and contributes to CHF-induced generalised anasarka

Riboflavin (vit B2)

Source and chemistry

Milk, egg, liver, green leafy vegetables, and grains contain a yellow flavone compound.

Physiological role and actions Flavin adenine dinucleotide (FAD) and flavin mononucleotide (FAM) are two types of flavin. (FMN) are flavoprotein coenzymes involved in a variety of oxidation-reduction processes. Thiamine and riboflavin have no pharmacological effects. Symptoms of deficiency Riboflavin insufficiency is usually associated with other problems.

Deficiencies. Angular stomatitis, sore and raw tongue, lips, and throat, mouth ulcers are also common symptoms, corneal vascularization, Anemia and neuropathy develop as a result of the dry, scaly skin and hair loss.

Therapeutic applications- To prevent and cure aribo flavinosis (2–20 mg/day orally or parenterally), usually in combination with other members of the B complex. In any other situation, there is no indication of benefit.

Niacin (vit B3)

Source and chemistry

Nicotinic acid and Nicotinamide—pyridine molecules, formerly known as pellagra preventive factor, are both referred to as niacin. Liver, fish, meat, cereal husk, nuts, and legumes are also good sources. Because tryptophan (primarily from animal protein) is partially transformed to nicotinic acid in the body (60 mg tryptophan = 1 mg

nicotinic acid), it can be considered a provitamin. Corn flour is low in tryptophan and is thought to include a niacin antagonist, hence it has caused pellagra in maize eaters. As a result, the amount of tryptophan in the diet influences the daily niacin requirement.

Pysiological role and actions- In the body Nicotinic acid is easily converted to its amide, which is a component of the oxidation-reduction coenzyme Nicotinamideadenine-dinucleotide (NAD) and its phosphate (NADP). In tissue respiration, glycolysis, and fat synthesis, these pyridine nucleotides operate as hydrogen acceptors in the electron transport chain. Flavoproteins oxidise NADH and NADPH to renew them. Nicotinic acid (but not nicotinamide) is a vasodilator in high doses, especially in cutaneous vessels. It also lowers lipids in the blood.

Symptoms of deficiency: Niacin deficiency causes 'Pellagra,' with the following cardinal symptoms: Dermatitis is a sunburn-like rash on the hands, legs, and face that eventually turns black, cracks, and peels.

Diarrhea accompanied by enteritis, stomatitis, glossitis, salivation, nausea, and vomiting.

Hallucinations precede dementia, which is accompanied by headaches, sleeplessness, poor memory, and motor and sensory abnormalities.

Therapeutic applications

1. As a preventative measure (20–50 mg/day oral) in patients at risk of pellagra.
2. Pellagra treatment: 200 to 500 mg/day orally or parenterally in split dosages. Skin lesions take weeks to months to improve, although striking improvement takes 1–2 days. Nicotinamide is chosen over nicotinic acid for injections since it does not produce flushing or other negative effects.
3. Hartnup's illness: Niacin supplementation is required in which tryptophan transport is hindered, and in carcinoid tumours, which use tryptophan for the production of 5-HT.

Pyridoxine (vitamin B6)

Chemistry and source of pyridoxine (vitamin B6) Pyridoxine, Pyriodoxal, and Pyridoxamine are pyridine compounds with vitamin B6 action that exist naturally. Liver, beef, egg, soybean, vegetables, and whole grain are dietary sources.

Function and actions in the body:

Pyridoxine and pyridoxamine are quickly converted to pyridoxal, which is then phosphorylated to generate pyridoxal phosphate, the coenzyme form. Transaminases

and decarboxylases that use pyridoxal are involved in the production of non-essential amino acids, the metabolism of tryptophan and sulfur-containing amino acids, and the generation of 5-HT, dopamine, histamine, GABA, and aminolevulinic acid (first step in the synthesis of haeme). A high-protein diet raises the need for pyridoxine. Although pyridoxine has been demonstrated to interact with steroid hormone receptors, its therapeutic significance is unknown. Long-term use of high dosages of pyridoxine can lead to dependence, and megadoses (0.2–2.0 g/day) have been related to sensory neuropathy. Pyridoxine does not have any pharmacological activities or adverse effects. Lactation suppression has been observed in non-suckling postpartum women given high doses of pyridoxine, which could be related to enhanced dopamine effect on pituitary lactotropes.

Interactions between drugs

1. Isoniazid forms a hydrazone with pyridoxal, inhibiting the formation of pyridoxal phosphate. Isoniazid also binds to pyridoxal phosphate, interfering with its function as a coenzyme. The renal excretion of pyridoxine compounds is enhanced due to the production of hydrazones. As a result, isoniazid therapy causes pyridoxine deficit.
2. Pyridoxine consumption and activity are further hampered by hydralazine, cycloserine, and penicillamine.
3. Some women's pyridoxal phosphate levels are reduced by oral contraceptives.
4. Pyridoxine reduces dopamine availability in the brain by boosting dopamine production from levodopa in peripheral tissues, but not when paired with a peripheral decarboxylase inhibitor, which eliminates the therapeutic effect in Parkinsonism.

Vitamin B6 antagonist- 4-deoxypyridoxine

Deficiency symptoms Vitamin B6 deficiency is frequently associated with other B vitamin deficiencies. Seborrheic dermatitis, glossitis, growth retardation, mental disorientation, lowered seizure threshold or convulsions (owing to a drop in brain GABA levels), peripheral neuritis, and anaemia are all symptoms of pyridoxine deficiency.

Therapeutic applications

1. As a preventative measure (2–5 mg daily) in alcoholics, babies, and patients with other B vitamin deficiencies.

2. To prevent and cure neurological abnormalities caused by isoniazid, hydralazine, and cycloserine (10–50 mg/day). Massive doses (in grammes) of pyridoxine have been used to successfully treat acute isoniazid toxicity.
4. To treat mental problems in oral contraceptive users (50 mg daily).
5. Pyridoxine responsive anaemia (due to faulty haeme synthesis) and homocystinuria are rare hereditary illnesses that benefit from high pyridoxine doses (50–200 mg/day).
6. Convulsions in children and infants.

VITAMIN C (ASCORBIC ACID)

Source and chemistry Ascorbic acid is a 6 carbon organic acid with a similar structure to glucose. It is a strong reducing agent with biological activity in the l-form. The richest sources are citrus fruits (lemons and oranges) and black currants; others include tomato, potato, green chilies, cabbage, and other vegetables. Human milk contains more vitamin C (25–50 mg/L) than cow's milk.

Physiological function and actions

Many oxidative and other metabolic reactions require vitamin C, such as the hydroxylation of proline and lysine residues in protocollagen—essential for the formation and stabilisation of the collagen triple helix; hydroxylation of carnitine, conversion of folic acid to folinic acid, biosynthesis of adrenal steroids, catecholamines, oxytocin, and vasopressin, and metabolism of cyclic nucleotides and prostaglandins. It stimulates collagen synthesis directly and is critical for the maintenance of intercellular connective tissue. Ascorbic acid has been linked to a variety of undefined effects in high doses, but none have been proven.

Vitamin C deficiency symptoms- Scurvy, which was once common among sailors, is now only seen in malnourished infants, children, the elderly, alcoholics, and drug addicts. Increased capillary fragility—swollen and bleeding gums, petechial and subperiosteal haemorrhages, and deformed teeth, brittle bones, impaired wound healing, anaemia, and growth retardation are the primary symptoms.

Therapeutic uses

1. Ascorbic acid deficiency prevention in at-risk individuals (see above) and infants: 50–100 mg/day Vitamin C or orange juice can be included in an infant's diet on a regular basis.
2. Scurvy treatment—0.5–1.5 g/day

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3. Postoperatively (500 mg daily): While vitamin C does not improve normal healing, it can help to prevent suboptimal healing. It has also been shown to hasten the healing of bedsores.

Adverse effects- In normal doses, ascorbic acid is well tolerated. Megadoses given over long periods of time can result in'rebound scurvy' upon discontinuation, most likely due to an increase in its own metabolism or tissue acclimatisation. Urinary oxalate stone risk may be increased. When high doses are added to iron preparations, they may also be cytotoxic.

(Note- Vitamin B12, Vitamin D and Vitamin K discussed in previous chapter)REFERENCES

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