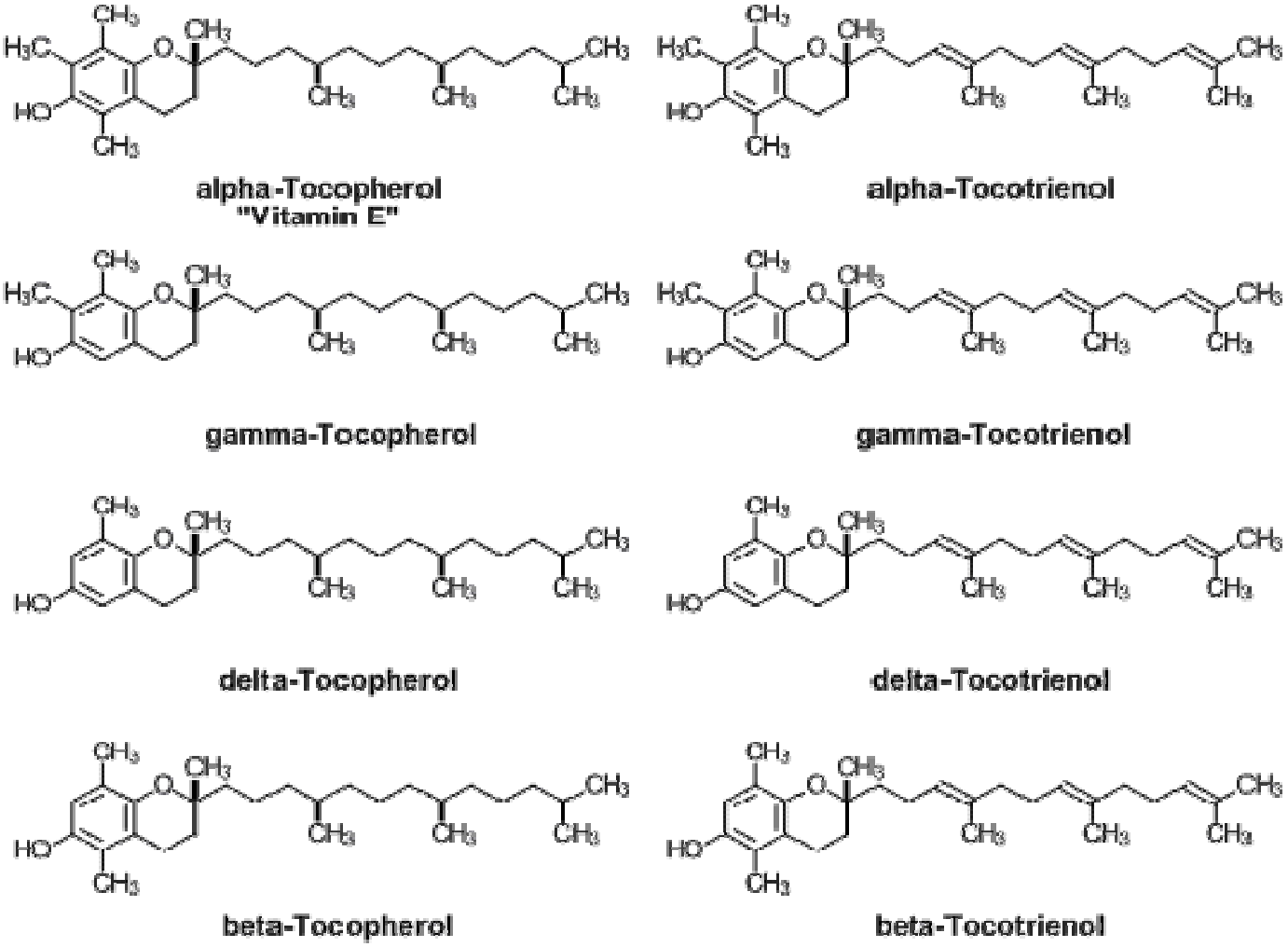
Sultan Qaboos Univeristy

College of Agricultural & Marine Sciences

Vitamins & Human Metabolism

FSHN6026 with Dr. Amanat Ali

Lecture No. (3+4): Vitamin (E): Summary



Mohsin Mohammed Taqi Mohsin Ali Mohammed Al-Lawati

Student ID: M020944

➢ Introduction:

o Vitamin E (Tocochromanols):

Vitamin E includes eight compounds designated as (alpha, beta, gamma and delta) and this is due to difference in the number and location of methyl group. In 1959 it was shown that alpha-tocopherol is essential for human nutrition and that all the forms synthesized by the plants and some animal sources. Tocopherols have saturated side chain whereas; tocotrienols unsaturated side chain. Naturally occurring d-α-tocopherol has the highest vitamin E activity in human body then followed by beta then by gamma and least activity found in delta.

Its consider as major lipid soluble anti-oxidant. While it was initially hoped that vitamin E supplementation would have a positive effect on health, research has not supported these conclusions. Vitamin E does not decrease mortality in adults. It does not improve blood sugar control in an unselected group of people with diabetes mellitus or decrease the risk of stroke. Daily supplementation of vitamin E does not decrease the risk of prostate cancer.

o Metabolism, Function, and excretion:

It is shown to have role in preventing oxidative damage (anti-oxidant) which is associated with disease like cancer, coronary heart disease, cataracts and Alzheimer's disease. It protects cell membrane by preventing lipids oxidation and enhances the immune response. Other functions include enzymatic activities, gene expression and neurological functions. It's also been suggested that the most important function of vitamin E is in cell signaling. Take a part in platelet aggregation by inhibiting platelet cyclo-oxygenase (COX) activity thus decreasing prostaglandin production and protein kinase C activation.

It become oxidized itself during protecting others from oxidation. It requires vitamin C, reduced glutathione (GSH), and NADPH for regeneration. It protects heart from oxidized LDL molecules which may cause atherosclerosis. Moreover; it's also prevent poly unsaturated fatty acid (PUFA) from oxidation. It works by terminating the effect (through scavenging activity) of hydroxide radicals (-OH) on unsaturated fatty acid.

It also affects the cholesterol metabolism by suppression rate limiting enzyme 3-hydroxy-3-methgyl glutaryl (HMG) CoA reductase. Vitamin E interacts with selenium (Se), sulphur containing amino acid, vitamin K, A, and vitamin D.

Vitamin E also has an effect on gene expression. Macrophages rich in cholesterol are found in the atherogenetic tissue. Scavenger receptor CD36 is a class B scavenger receptor found to be up-regulated by oxidized low density lipoprotein (LDL) and binds it. Treatment with alpha tocopherol was found to down regulate the expression of the CD36 scavenger receptor gene and the scavenger receptor class A (SR-A) and modulates expression of the connective tissue growth factor (CTGF). CTGF gene, when expressed, is responsible for the repair of wounds and regeneration of the extracellular tissue that is lost or damaged during atherosclerosis.

Metabolic pathway of vitamin E is largely unknown in the human body. However; in non-polar solvent it is oxidized into tocopheroxy radicals. In polar solvent it's oxidized into tocopheryl quinone. The major excretion pathway of vitamin E is through feces. Water soluble metabolites can be conjugated with glucuronic acid and excreted in urine and skin.

- **Stability, Chemical & Physical Properties:**

Natural source of vitamin E is a single isomer (d-α-tocopherol). One 1 IU of natural vitamin E is equal to 0.67 mg of alpha–tocopherol. One IU of synthetic vitamin E is equal to 0.45 mg of alpha–tocopherol. Thus natural source has twice bioavailability.

- **Absorption and Measurement of Vitamin E:**

Its absorption mechanism is closely related to fat absorption by body intestinal tract through bile salt action transport mechanism. It's readily absorbed into lymphatic system through migration and incorporation in the chylomicrons. Majority of vitamin E is in LDL cholesterol. There are positive association between the serum lipid concentrations and tocopherol levels. Alpha-tocopherol is the major tocopherol in plasma. Because of linear relationship of vitamin E and total serum lipids so it's not possible to accurately estimate its concentration in hypo- or hyperproteinemia conditions.

- **Requirements, Supplementation & Sources:**

Normal serum tocopherol concentration is between 0.5-1.6 mg/dL in adults. Recommended dietary allowance (RDA) of vitamin E is 2000 (in Adults: 15 mg/day). It is estimated that 0.4 mg of vitamin E is required per 1 g of PUFA. A small amount also recommended for pregnant. Supplementation is desirable in fat malabsorption like in premature babies receiving artificial foods in large amount (PUFA)

Intakes of 10-30 mg per day will maintain serum vitamin E in normal concentration. It can be found in both plant and animal source and especially in vegetable oils (soybean oils and wheat germ oils). Also from fortified foods and the best source comes from lightly process foods.

There are many different forms of vitamin E. The γ-Tocopherol can be found in corn oil, soybean oil, margarine and dressings. The α-Tocopherol, the most biologically active form of vitamin E. This variant of vitamin E can be found most abundantly in wheat germ oil, sunflower, and safflower oils.

mg/day	Age
Infants	
4	0 to 6 months
5	7 to 12 months
Children	
6	1 to 3 years
7	4 to 8 years
11	9 to 13 years
Adolescents and Adults	
15	14 and older

Table (1): Recommended daily intake

- **Deficiency:**

At lower level it may causes red blood cell lysis and nerve damage as well as loss of muscle coordination, impaired movement, and loss of vision and speech. In infants causes haemolytic anemia. In adult cause malabsorption of fats and values less than 0.5 mg/dL indicates deficiency.

Vitamin E deficiency is very rare and if happen may causes:

1. Myopathy
2. peripheral neuropathy
3. ataxia
4. retinopathy
5. impairment of the immune response

A few population groups are at risk of vitamin E deficiency for example; cystic fibrosis, hepatobiliary system disorders, and abetalipoproteinemia. Because the digestive tract requires fat to absorb vitamin E, people with fat-malabsorption disorders are more likely to become deficient than people without such disorders.

- **Toxicity:**

Its least toxic vitamin if compared to others and its affects is not as serious as other vitamins. At high dose it may interfere with vitamin K activity and thus may cause bleeding problems. Upper level of vitamin E for adult is 1000 mg/day. It also associated with gastrointestinal distress. Vitamin E can increase the risk of hemorrhagic stroke by 22%.

➢ References:

1. Suzanne K. Gaby, *et al.*, Vitamin Intake and Health-Scientific Review, 1st ed.270 Madison Avenue, New York: Marcel Dekker, Inc. USA; 1991.

2. Wilhelm Friedrich. Vitamins, 1st ed. Berlin. New York: Walter de Gruyter. 1988.

3. John Marks. The vitamins – their role in medical practice. 1st ed. Lancaster, England-Falcon House. MTP press limited. UK. 1985.

4. Sareen S. Gropper, *et al.*, Advance Nutrition and Human Metabolism. 4th ed. 10 Davis Drive Belmont, CA 94002-3098, Thomson-Wadsworth.USA. 2005.

5. Maurice E. Shils, et al., Modern Nutrition in Health and Disease. 10th ed. 530 Walnut Street, Philadelphia, PA 19106. Lippincott Williams & Wilkins.USA. 2006.

6. http://en.wikipedia.org/wiki/Vitamin_E