

North Asian International Research Journal of Sciences, Engineering & I.T.

ISSN: 2454-7514 Vol. 4, Issue-7 July -2018

Index Copernicus Value: 52.88

VITAMIN E: FUNCTIONS AND METABOLISM

SHUBAM GOYAL

Introduction

Vitamin E is one of 4 fat-soluble vitamins. The vitamin is formed by plants, and has eight unlike isoforms separated into two classes of four vitamins each. The compounds include of a 6-chromanol ring and an isoprenoid side chain. The group bond to the R1, R2 and R3 position on the 6-chromanol ring verify whether the vitamin is acknowledged as alpha, beta, gamma, or delta.(www.exrx.nt, 2011) A large body of the study now focuses on the alpha tocopherol form of vitamin E, which is the most biologically active (Tiidus, P. M., and M. E. Houston, 1995, Traber, M.G., 1999). Recently gamma tocopherol has been a subject of importance by countless researchers. Vitamins are a vital source of life. Vitamins are any organic that is essential for many living things to obtain a normal healthy living. (Dorling Kindersley, 1998)

Dietary Function

Vitamin E is an essential part of cellular membranes whose main role is to protect the cell alongside to oxidation, Within cells and organelles vitamin E is the first line of defence against lipid per oxidation. The vitamin also acts as a very important function in providing elasticity to red blood cells as they travel through the arterial system.

Artificial vitamin E nominates dl-alpha-tocopherol, is less expensive from the naturally occurring form, d-alpha tocopherol. The expected/natural form of the vitamin is synthesised only by vegetation and is found mostly in plant oils. Vitamin E is also present in large numbers in the chloroplast and as a result the leaf of most plants. The fat-soluble property of vitamin E allows it to be stored within the fatty tissues of humans and animals. Therefore a diet that includes meat supplies additional vitamin E. However, the amount of vitamin E obtained in a high protein meat complete diet is less than the quantity supplied by a plant source.

Metabolism

Absorption and Bioavaliability

Absorption of vitamin E is highly reliant on the same process that develop during fatty acid digestion and metabolism. A lack of any component of these transporters will reduce carrier structure and in turn vitamin E absorption. vitriol acids are a measured essential for vitamin E absorption and micelle formation. Once formed, the micelle is then able to cross the undiluted water layer and discharge its contents into the enterocyte. A contemplation of the flow pressure of vitamin E through the enterocyte has been elusive to researchers (Traber, M.G., 1999).

The ingested Vitamin E is quickly vacant from the bloodstream, into tissues as blood concentrations of vitamin E in humans and animals have minute relation to the quantity within the body. Vitamin E is fat soluble; food intake could potentially persuade its bioavailability, it is claimed that the alpha-tocopherol has the highest bioavailability. The lower bioavailability of Vitamin E is form a typical vitamin E supplement, and the changeability which is experimented when consumed, this suggest that vitamin e is poorly absorbed through the body with low-fat meals. Vitamin E can be improved by food reinforcement makes Vitamin E more bio-available .Bioavailability of vitamin E, is influenced by the increase of risk in coronary heart disease.

Vitamin E is an antioxidant that is vital for our bodies, to preserve optimum health and in people diets. Vitamin E is extensive and in all kinds of foods. Most vitamin E comes from vegetable oil and products made from them like margarine and salad dressings. It is important for youth,in order to maintain their health. It aids in protecting red blood cells, defends the body against oxidative damage and helps avoid damage of both vitamins A & C.

Having vitamin E as part of a well balanced diet can reduce the risk of heart disease and cancer, also slows down the effect of Alzheimer's disease when consumed in high dosage

Once in the blood 15 to 45% of the total vitamin E intake can be absorbed by the cells.

Transport

Upon reaching the bicollateral exterior of the enterocyte vitamin E is packed into chylomicrons and then elated throughout the body via the movement. Within five minutes of arrangement chylomicrons are broken down by lipoprotein lipase and the contents are isolated towards a range of paths. The vitamin E in the chylomicron equilibrates both with (HDL'S) and (LDL'S) (Groff, J.L., Gropper S.S., and Hunt S.M 1995). HDL'S all circulate lipoproteins which eventually receive vitamin E, as HDL fervently relocates the compound to the lipoproteins at a rate equivalent to 10% of the plasma vitamin E per hour (Traber, M.G., 1999). The vitamin E remaining in the chylomicron becomes a chylomicron remnant and travels back to the liver for re-uptake in a process that has garnered much research, but so far is poorly understood. Once in the liver the vitamin E is packaged into Very Low Density Lipoproteins (VLDL) and excreted back into the circulation. Being the most biologically active of the eight vitamers, (Groff, J.L., Gropper S.S., and Hunt S.M 1995, Kanter, M.M. 1998, National Academy of Sciences. 2000, Traber, M.G., 1999, www.exrx.nt), alpha tocopherol is sequestered by the liver and constitutes over 80% of the total vitamin E packaged into the VLDL and secreted by the liver (Traber, M.G., 1999, www.exrx.nt). The predominant transfer of the alpha vitamer is performed by alpha tocopherol transfer protein (ATTP). As the VLDL are broken down by lipoprotein lipase, Low Density Lipoproteins (LDL) are formed and from these lipoproteins the vitamin E is transferred to HDL and eventually incorporated into either circulating lipoproteins or peripheral tissue. Any of the previously mentioned lipoproteins have the ability to transfer vitamin E to the tissue as needed (Groff, J.L., Gropper S.S., and Hunt S.M 1995, Traber, M.G., 1999, www.exrx.nt). A final mechanism for vitamin E is uptake by the peripheral tissue from the chylomicron via lipoprotein lipase activity. Unlike re-uptake of vitamin E by the chylomicron remnant, uptake of the vitamer by peripheral tissue is better understood. After vitamin E has been transferred to the LDL from the chylomicron two receptors (LDL dependent receptor and LDL independent receptor) within the tissue play a key role in the uptake of vitamin E into the cell (Traber, M.G., 1999, www.exrx.nt).

Storage

Vitamin E is a lipid soluble vitamin and therefore over 90% of total body vitamin E is found in the adipose tissue (National Academy of Sciences, 2000, Traber, M.G., 1999, www.exrx.nt). Over 90% of these pools are found as a part of an adipocyte fat droplet whereas the remaining amount is found mainly in adipocyte cellular membrane. The storage ratios of vitamin E are also very difficult to alter. It takes over two years to alter the ratio of alpha to gamma isoforms. Previous studies have shown that the ratio is altered as the alpha vitamin replaces the gamma vitamin, which is reduced by 70% (Tiidus, P. M., and M. E. Houston, 1995, www.exrx.nt). Concentrations of vitamin E cover a wide range in body tissues. In the plasma the concentration of vitamin E is approximately 27 umol/l. Within skeletal muscle protein the vitamin E concentration varies considerably depending upon the type of muscle (National Academy of Sciences, 2000). Although a large majority of vitamin E is found in adipose tissue (230 nmol/g wet weight) (National Academy of Sciences, 2000) there isn't an organ that function to store and discharge vitamin E. The actual means regarding vitamin E release from the tissue is unknown at this time. While it seems likely that vitamin E is released during lipolysis associated with exercise this may not be true. Research has shown that even during times of weight reduction vitamin E is not released from the adipose cells (Traber, M.G., 1999). Therefore, the factors that regulate bioavliability of vitamin E from adipose tissue are not known.

Requirements

As it is unknown how much vitamin E should be consumed by adults, it is aid that according to the (RDA) that men whould have a Vitamin E intake of 15 mg's and 10 mg's for females.(S.Frances,2006) Only natural alphatocopherol can typically provide supplements of Vitamin E, and only existing one stereo-isometric form. The need for vitamin E increases as people diet consist of more polyunsaturated fats and oils as the oils in Vitamin E needs a source of antioxidants to help absorption/and protection. Vitamin E is largely contained in raw oils. In 1968 the RDA for vitamin E was established at 300 IU (300 mg) for a 65 kg adult male (National Academy of Sciences. 2001). The detailed RDA is listed in table 1 and table 2. This daily level is difficult to reach unless a diet high in polyunsaturated fatty acids was consumed (Tiidus, P. M., and M. E. Houston1995) From 1 mg of vitamin E approximately .3 (Traber, M.G., 1999) to .5 is in the alpha vitamin form and therefore readily absorbed. The other vitamers are not stored as efficiently and usually excreted (Tiidus, P. M., and M. E. Houston1995, Traber, M.G., 1999). Therefore a new RDA was set based on the alpha-tocopherol form of the vitamin. In 1989 the RDA for Vitamin E was set at 10 mg alpha tocopherol for men and 8 mg of alpha-tocopherol for women (Traber, M.G., 1999, www.exrx.nt). In the year 2000 all RDA values were in the process of being replaced by Dietary Reference Intakes (DRI). The DRI has been established at 15 IU of alpha-tocopherol. The revised DRI levels are the same for both men and women (Packer, L.1997, www.exrx.nt).

Recommended Dietary Allowance (RDA)

In 1968, the recommended dietary allowance for vitamin E was established at 300 IU (300 mg) for a 65 kg adult male (National Academy of Sciences. 2001. The other vitamins are not stored as efficiently and usually excreted (Tiidus, P. M., and M. E. Houston1995, Traber, M.G., 1999). In accordance to information resourced from Traber, M.G. in 1999, there was a new recommended dietary allowance which was set based on the alpha-tocopherol form of the vitamin. In 1989 the RDA for Vitamin E was set at 10 mg alpha tocopherol for men and 8 mg of alpha-tocopherol for women.

Deficiency

Anaemia, muscle necrosis, and foetal death have been observed in over fifteen different vitamin-E-deficient animal species.(www.exrx.nt) .Humans who have fat mal-absorption suffer from the same symptoms shown in rats, but to a lesser degree. These manifestations are exhibited early in childhood. Some of the symptoms include decreased sensory perception, muscle weakness, scoliosis, and muscle structural abnormalities. These symptoms can usually be reversed using vitamin E supplementation (Tiidus, P. M., and M. E. Houston1995, www.exrx.nt)) Vitamin E deficient diets fed to adult humans have resulted in the formation of very few deficiency symptoms. (Bunnell R.H., E. De Ritter, S.H. Rubin 1975, www.exrx.nt) has shown that prisoners performing strenuous physical labour while fed a vitamin-E deficient diet for 13 months exhibited no deficiency symptoms. A diet full of vitamin E may lower the risk of cancer and the likely hood of death, in part through oxidation and inflammation. Lacking vitamin E concentrations in the blood; people die more often and than people with a high blood concentration (s.Frances, 2006).Through research and development of vitamin E has an effect on helping slowing down the loss of function that occurs in Alzheimer's disease when taken in excessive dosages.

Toxicity

Vitamin E toxicity has not often been acknowledged in humans. Doses up to 1600 I.U. have been usually administered in study with no clear unpleasant side effects. Toxicity may be complex since there is a wide variant in daily blood vitamin E levels. Increasing vitamin E levels in muscle tissue is especially difficult to attain and therefore toxic levels are difficult to achieve. Meydani et al. (Karlsson J.,1997, www.exrx.nt) given 800 I.U. of vitamin E to subjects for a period of 48 days and only saw a 37% increase in plasma alpha tocopherol levels (www.exrx.nt). The tocopherol binding protein is most commonly said to manage the quantity of vitamin E that can be physiologically stored. High levels of vitamin E are likely excreted by the body.

Conclusion

In conclusion vitamin E may help reduce oxidative stress and lipid per oxidation of cellular membranes. The current Dietary Recommended Intake for vitamin E meets the needs of most individuals can be achieved through a healthy diet, and eating plan. In April 2000 The National Academy of Sciences according to Packer, L. In 1997 established an intake maximum of 1100 I.U for synthetic and 1500 I.U. for natural vitamin E. These maximum levels are approximately one hundred times the Dietary Recommended Intake. Vitamin E supplementation by both active and non active individuals is widely employed. The choice to supplement or not supplement a diet with vitamin E is an option.

Reference

- 1. Bunnell R.H., E. De Ritter, S.H. Rubin. Effect of feeidng polyunsaturated fatty acids with a low vitamin E diet on blood levels of tocopherol in men peroforming hard physical labour. Am. J. Clin. Nutr. 28:706-711, 1975.
- 2. Clarkson P. M. Antioxidants and physical performance. Crit. Rev. Food Sci. Nutr 35: 131-141, 1995.
- 3. Class notes on vitamins and minerals. Dr. Mariam Farhad, 2011
- 4. Groff, J.L., Gropper S.S., and Hunt S.M. The Fat Soluble Vitamins. In: Advanced Nutrition and Human Metabolism. Minneapolis: West Publishing Company, 1995, p. 284-324.
- 5. Hartman, A., A.M. Neiss, M. Grunert-Fuchs, B. Poch, and G. Speit. Vitamin E prevents exercise-induced DNA damage. Mutat. Res. 346: 195-202, 1995.

North Asian International research Journal consortiums www.nairjc.com

- 6. http://consumerlab.com/RDAS viewed 15.05.2011
- 7. http://ods.od.nih.gov/factsheets/VitaminE.asp viewed 13.05.2011
- 8. http://www.exrx.nt/nutrition/antixidants/vitaminE.html viewed 15.05.2011
- 9. Karlsson J. Exercise, muscle metabolism and the antioxidant defense. World Rev. Nutr. Diet. 82:81-100, 1997.
- 10. Packer L. Protective role of vitamin E in biological systems. Am. J. Clin. Nutr. 53:1050S-1055S, 1991.
- 11. Rokitski, L., E. Logemann, A.N. Sagredos, M. Murphy, W. Wetzel-Roth, and J. Keul. Lipid peroxidation and antioxidative vitamins under extreme stress. Acta. Physiol. Scand. 151: 149-158, 1994
- 12. Traber, M.G., 1999. Vitamin E. In: Modern Nutrition in Health and Disease. Ninth Edition. Edited by Maurice Shils, James Olson, Moshe Shike, and A. Catharine Ross. Baltimore: Williams & Wilkins, 1999 p. 347-362.
- 13. Watt, T., T.T. Romet, I. McFalane, D. McGuey, C. Allen, and R. C. Goode. Vitamin E and oxygen consumption. Lancet. 2: 354-358, 1974.
- 14. T., T.T. Romet, I. McFalane, D. McGuey, C. Allen, and R. C. Goode. Vitamin E and oxygen consumption. Lancet. 2: 354-358, 1974.