Many individuals and even researchers are unaware that natural Vitamin E is a family of four tocopherols and four tocotrienols, which occur in various ratios in different foods. Since humans and animals do not synthesize their own vitamin E, they primarily acquire tocopherols from plants, which are the only living things capable of making vitamin E. Gamma-Tocopherol is often the most prevalent form of vitamin E in plant foods and seeds. Vegetable oils such as corn, soybean, and sesame, and nuts such as walnuts, pecans, and peanuts are rich sources of Gamma-tocopherol. Because of the widespread use of these plant products, Gamma-tocopherol represents 70% of the Vit E consumed in the typical US diet.

In general, the estimated amount of vitamin E (as d-alpha-tocoopherol) supplied by the "normal" diet ranges from 6-13 mg per day in various countries. About three-quarters of adults do not even meet the RDA for vitamin E, which is 15 mg/day alpha-tocopherol, beginning at age 14 (19 mg/day for nursing women). In two recent surveys in the United States, average dietary intakes of vitamin E were 7.8 mg (CSFII) and 9.5 mg (NHANES III), quite below the RDA of 15 mg. There is no RDA yet established for the other tocopherols (beta, gamma, delta) or any of the tocotrienols because of our new understanding of these compounds. It is important to note that the synthetic Vit E mixture contains 7 isomers that are a byproduct of chemical synthesis of vit E and are not naturally occurring. A lot of studies using synthetic Vit E have produced disappointing results.

It is unclear what an optimal Vit E intake would be, especially in the face of increased oxidative stress caused by environmental (pollution, metabolic, exercise) or dietary factors (oxidized fats, excessive PUFA intake). The 400 IU level was observed to be the threshold for which oxidation of lipoproteins in vitro was compensated to a significant extent.

All forms of Vitamin E are equally absorbed from the intestine into the chylomicrons, thus they all have a chance to be delivered to the liver in the first passage through lymph and blood until they get to the liver. Here, a special transfer protein incorporates them into the VLDL and gives them a second chance to go into the blood stream for tissue uptake. The following chart depicts the relative affinities of the liver transfer protein for each form of Vit E.

This transfer protein is called ATTP, or alpha tocopherol transfer protein because it preferentially binds alpha tocopherol. This explains why when a high dose alpha-tocopherol supplement is taken at the same meal as a gamma-tocopherol rich food, the alpha levels of the blood increase way before gamma does.

This also explains why alpha-tocopherol is the predominant form of vitamin E in most human and animal tissues, including blood plasma. However, we learn from researchers such as Burton et al that gamma-tocopherol constitutes as much as 30-50% of the total vitamin E in human skin, muscle, vein, and adipose tissue. Furthermore, gamma-tocopherol concentrations are substantially higher in human than in rodent tissues. This might explain the different results from interventions with alpha-tocopherol in animal studies.

Studies indicate that high dose alpha-tocopherol supplementation considerably decreases the tissue accumulation of gamma-tocopherol. After supplementing with 1,200 IU of synthetic alpha-tocopherol daily for 8 weeks, plasma gamma-tocopherol decreased in all subjects to 30-50% of initial values. A Swedish study found that patients with coronary heart disease had lower levels of gamma tocopherol and a higher alpha-to-gamma ratio than healthy age-matched subjects. Maybe foods don’t contain high amounts of alpha tocopherol for a good reason.
One recent group found that gamma-tocopherol is significantly more effective than alpha-tocopherol in inhibiting the oxidizing agent peroxynitrite.\textsuperscript{18} While alpha-tocopherol can to some extent inhibit free radical generation, gamma-tocopherol is able to trap and remove existing free radicals.\textsuperscript{19} Gamma tocopherol is thought, therefore to protect cells against the mutagenic and carcinogenic effects of the reactive nitrogen species.

Recently, it was found that gamma-tocopherol possesses anti-inflammatory activity by inhibiting prostaglandin E\textsubscript{2} synthesis in lipopolysaccharide-stimulated macrophages and in interleukin 1\textbeta (IL-1\textbeta)-activated epithelial cells, by directly inhibiting cyclooxygenase-2 (COX-2) activity. In contrast, alpha-tocopherol has no similar effect.

Other studies found that gamma-tocopherol supplementation led to a more potent decrease in platelet aggregation and delay of thrombin-induced fibrinogen deposition in platelets.\textsuperscript{20} Gamma tocopherol supplementation also resulted in stronger ex vivo inhibition of superoxide generation, lipid peroxidation, and LDL oxidation. In a follow-up study, this same group reported that gamma-tocopherol was significantly more potent than was alpha-tocopherol in enhancing SOD activity in both canine and rat arterial tissue and in increasing the arterial protein expression of both manganese SOD and Cu/Zn SOD.\textsuperscript{15} Furthermore, although both tocopherols increased nitric oxide generation and endothelial nitric oxide synthase activity, only gamma-tocopherol supplementation resulted in increased protein expression of this enzyme. Because endothelium-derived nitric oxide is a key regulator of vascular homeostasis, up-regulation of endothelial nitric oxide synthase and nitric oxide formation by gamma-tocopherol could be important in preventing vascular endothelial dysfunction.\textsuperscript{15} Gamma-Tocopherol is metabolized in the human body to a compound called gamma-CEHC which was shown to have natriuretic effect and so it may be of importance in regulating the blood pressure.\textsuperscript{15}

Recent research is pointing towards the tocotrienol side of the Vitamin E family to be very active in the area of antioxidant activity even against hydroperoxides, cholesterol management including apolipoprotein a and b, and cancer protection.\textsuperscript{30}

References