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Tocotrienols (vitamin E) are bioactive

Tocotrienols (found in palm oil, rice bran and cereal grains) are more potent antioxidants than the tocopherols. However, previous work had suggested that they were poorly digested and distributed to the tissues via the blood, then rapidly metabolized and eliminated from the body. Despite this, Pramod Khosla and colleagues from Wayne State University in the US found that this form of vitamin E could be digested, absorbed and give rise to bioactive concentrations in the blood.

Vitamin E comes in four different molecular forms, four tocopherols (α-, β- γ- and δ-isomers) and four tocotrienols (also in α-, β- γ- and δ-isomers). According to a summary in NutraIngredients.com (17/07/06), α-tocopherol is the main form in supplements and in the European diet. Gamma-tocopherol is the most common form in the American diet. Research on vitamin E has mostly focussed on the tocopherols, with less than 1% of studies examining the effects of tocotrienols. The problem has been that tocotrienols are not so readily delivered to the blood by the tocopherol transfer protein (TTP). However, Khosla has now managed to show that although TTP does have a lower affinity for the tocotrienols than the tocopherols, it still delivers concentrations of tocotrienols to the blood that are sufficient to protect against the neurological damage that follows a stroke. To do this he asked a group of women with normal cholesterol levels to consume a fat-rich strawberry smoothie containing 400 mg of vitamin E (containing both tocotrienols and tocopherols). In the post-prandial period, results showed that maximal α-tocotrienol concentrations reached 3 micromoles in blood plasma, 1.7 micromoles in low density lipoproteins (LDL) and 0.5 micromoles in high density lipoproteins (HDL). (More in Antioxidants and Redox Signalling, 2006, 8 (5-6): 1059-1068).

Further work on the subject is also described by Chandan Sen and his colleagues at Ohio State University. They discovered that tocotrienol (TCT) was effective at two concentrations. At a higher concentration it functioned as an antioxidant, while at lower concentrations its action was independent of its antioxidant properties. To try to discriminate between these two functions, the Ohio team used two triggers of neurotoxicity: homocysteic acid (HCA) and linoleic acid. Both HCA and linoleic acid cause neurotoxicity with comparable features, such as an increased ratio of oxidized to reduced glutathione GSSG/GSH, raised intracellular calcium concentrations and compromised mitochondrial membrane potential. HCA causes vascular and neuronal lesions associated with cardiovascular disease. The fatty acid, linoleic acid, can directly stimulate damaging free radical activity. In relation to stroke, fatty acids rapidly accumulate when a clot in a blood vessel forms, stopping the blood flow to the brain and damaging it irreversibly.

To observe the activity of tocotrienol, rat neurons were treated with nanomolar concentrations of the vitamin. The results indicated that the neurons were protected from the cell death caused by the HCA by these very low concentrations of tocotrienol. At micromolar concentrations, tocotrienol exhibited antioxidant properties against the oxidative stress caused by linoleic acid. (From Journal of Neurochemistry online 26/06/06. More information in a press release from Ohio State University. See also previous work by Chandan Sen's group in Stroke. 2005;36:e144.-152).