عنوان مقاله:

The effects of high doses of vitamin E on histological alteration in liver of male pups from rat

محل انتشار:

چهارمین کنگره بین المللی و شانزدهمین کنگره ملی ژنتیک (سال: 1399)

تعداد صفحات اصل مقاله: 1

نویسندگان:

Sajjad Kooshki - *M.Sc. Student of Developmental Cell Biology, Faculty of Biology, Damghan University*

Mohammad taghi ghorbanian - *PhD in Anatomy of Faculty of Biology, Damghan University*

iran Goudarzi - *PhD in Physiology, Faculty of Biology, Damghan University*

hadis kooshki - *M.Sc student of pathogenic microbiology, Faculty of Biology, Azad University of Boroujerd*

خلاصه مقاله:

Background and Aim: Vitamin E is an integral element of the liver's major lipid-soluble plasma lipoproteins and antioxidants. Vitamin E is a nutrient that has both antioxidant and non-antioxidant properties. As an antioxidant, it inhibits LDL cholesterol, Vitamin E plays an important role in protecting oxidative stress and trapping free radicals in lipid membranes, degrading and enhancing ROS-related lipid peroxidation in the plasma membrane It is mutagenic and carcinogenic, it prevents both in vitro and in vivo conditions. And exerts a protective effect against oxidative-related diseases. Vitamin E can maintain liver cell morphological stability and cell membrane integrity, and improve necrosis. Research has shown that vitamin E can effectively mitochondrial morphology, network. Endoplasmic reticulum and restore the activity of antioxidant enzymes (CAT, SOD and GSH-PX). In addition, vitamin E inhibits mitochondrial ROS production by enhancing mitochondrial membrane potential and improving mitochondrial function. Among the non-antioxidant properties of vitamin E can be the administration of prostacyclin, reducing inflammation and reducing cell adhesion molecules. Vitamin E protects cells and subcellular structures from oxidative damage by reducing LPO products. When vitamin E was used alone, hepatic LPO decreased and GSH levels increased. Vitamin E as a protection against toxicity increased GSH and TAS levels and reduced MDA, TOS and XO levels compared to control groups. Vitamin E can decrease plasma corticosterone in mice exposed to stress. Vitamin E can also reduce adipose tissue lipolysis by interfering with the glucocorticoid response, because glucocorticoids stimulate lipolysis. Glucocorticoid receptor signal transduction has also been implicated in the expression of Irs۲, Pdk۴, Angptl۴ and Ppargc۱a genes. Vitamin E significantly reduced the settings of Ppargc۱a, Pdk۴, Irs۲ and Cpt۲ and tended to lower the levels of Angptl۴ and Slc۲۲a۵ mRNA genes. Vitamin E supplements prevent the increase in circulating fatty acids (FFA) and induce inflammation in the liver. Vitamin E alters the liver transcriptional response to synthesis and inflammation of fat and cholesterol. CAMP-dependent pathways of catecholamine are important activators of lipolysis as well as transcriptional regulators of Irs۲ and Ppargc۱a. The potential inhibitory effect of vitamin E on cAMP signaling begins with inhibition of the upstream regulator of CREB.Considering the above and the usefulness of vitamin E, ... consuming large amounts of vitamin E may have negative effects.Methods: Adult m

کلمات کلیدی:

Vitamin E, Liver, Antioxidant, Free Radical, Rat, Histopathology