



Vitamin E: Where Are We Now in Vascular Diseases?

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INTRODUCTION

Vitamin E is one of the **most popular fat-soluble** vitamins in pathological research. It has been under scrutiny since the 1980s to understand what makes it a **vital dietary component of food**. It is one of the major antioxidants of food, and plays several functions in the human body, including the regulation of the vascular system. **Cardiovascular diseases (CVD) are one of the most chronic and persistent illnesses faced by humans.**

Vitamin E is a direct deterrent of **oxidative stress and atherosclerosis** due to its antioxidation effect. Researchers continue to find supplementary protective functions of vitamin E, which leads to **various important molecular mechanisms of vitamin E that regulate various metabolic pathways and control gene expression.**

OBJECTIVES

This review **examines the role of dietary vitamin E (α -tocopherol) as an antioxidant and bioactive molecule in promoting vascular health.** While the antioxidant effect of vitamin E is well established, knowledge about its capacity as a promising regulatory molecule in the control of the vascular system is limited. **The aim of this review is to discuss some of these mechanisms and summarize their role in the prevention of cardiovascular diseases (CVD).** Here, we also briefly discuss foods rich in vitamin E, and deliberate some potential toxicological effects of excessive supplemental vitamin E in the body.

WHAT IS VITAMIN E?

'Vitamin E' broadly represents tocopherols comprising of a **hydroxylated chromanol ring** attached to a **hydrophobic phytyl side chain**. the attribute of 'vitamin' is only given to **α -tocopherol (α T)** due to its selective uptake.

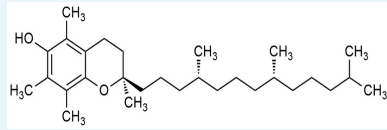


FIG 1: Chemical structure of α -Tocopherol (α T)

FOODS RICH IN α -TOCOPHEROL

RDA (for men and women) = 15 mg/day
(for lactating women) = 19 mg/day

However, no specific daily required dosage \rightarrow continuous interaction with other nutrients

Upper Level of Intake (UL) = 1000 mg/day (virtually impossible to achieve through natural intake or prescribed supplementation).

Excellent sources: wheat germ oil, almonds, sunflower oil, safflower oil, corn oil, hazelnuts, peanuts, peanut butter

Good sources: eggs, meat, fish, margarine, bread, **green leafy vegetables**, fruits and fortified cereal

New sources: quinoa, lentils, amaranth, chia seeds, cactus

Also found in **algae** such as Chlorella, *Stichococcus bacillaris*, *Dunaliella salina*

RESULTS AND DISCUSSION

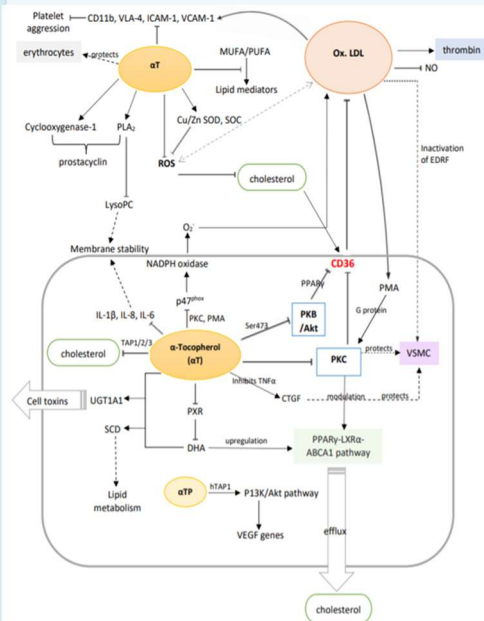


FIG 2: A summary of the potential molecular mechanisms of vitamin E (α -tocopherol) in vascular disease prevention via the suppression and upregulation of genes and metabolic pathways.

RESULTS AND DISCUSSION

First **major human trials** on relationship between Vitamin E and CVD prevention were reviewed. They contained opposing views of the antioxidant effect of vitamin E in preventing various CVDs. I then **proposed several alternate pathways and mechanisms** by which vitamin E regulates the functioning of various cellular/tissue components based on previous studies:

- ❖ Prevents haemolysis
- ❖ Regulates **phospholipid metabolism**
- ❖ Prevents apoptosis caused by cytotoxic effect of **docahexanoic acid (DHA)**
- ❖ Helps in cell detoxification
- ❖ Promotes **lipid biosynthesis**
- ❖ Prevents ROS-activated apoptosis – decreases **caspase-3 production**
- ❖ Prevents **lipid peroxidation** – activates endogenous antioxidant enzymes
- ❖ Reduces inflammation - decreases release of inflammatory cytokines
- ❖ Prevents **vascular smooth muscle cell (VSMC) proliferation**
- ❖ Prevents hypercholesterolemia and foam cell formation – **downregulation of CD36 scavenger receptor**
- ❖ Regulates **PKB/Akt pathway**
- ❖ **Inhibits superoxide production** by inhibiting NADPH oxidase activation by PKC
- ❖ Reduces risk of **venous thromboembolism (VTE)** – anticoagulation effect, inhibition of platelet adherence
- ❖ Prevents **thrombosis** and increases vasodilation

POTENTIAL CYTOTOXIC EFFECTS

- ❑ a supplemental dose of >150 IU/d or high dose (>400 IU/d) can cause a progressive increase in all-cause mortality.
- ❑ Increased risk of **prostate cancer** in men
- ❑ **risk factor for those taking vitamin K supplements** or suffering from vitamin K deficiency, since Vitamin E also shows anticlotting properties.
- ❑ inhibits the enzyme glutathione S-transferase (**GST**) in humans
- ❑ Can act as a **prooxidant**
- ❑ might increase the risk of haemorrhagic stroke in men

CONCLUSION

Through the review of old and new research on vitamin E's effect on vascular health, it can be concluded that **vitamin E is an absolute essential micronutrient for human health.**

Vitamin E acts as a major deterrent of atherosclerosis, which is the biggest cause of CVD in humans. It shows protective effects on vascular health, and the review agrees with this claim. **Newer research agrees with vitamin E's positive effects but focuses on unearthing the potential mechanisms that supplement α T in prevention of LDL oxidation.** Vitamin E not just protects arteries from atherosclerosis and platelet aggression, but has a host of other effects in the body. Studies also underlined various cellular mechanisms that allows α T to prevent the oxidation of LDL. A shortcoming of present research highlighted in this review is the lack of animal and human studies on the molecular mechanisms by which α T protects vascular health. **While in vitro studies contend that α T has a host of regulatory effects on the vascular endothelium, these claims need to be validated by in vivo studies as well.**



READ MORE HERE

You can find a detailed version of my research in my paper that has **been published** in the **Life MDPI Journal**. You can find it here: <https://www.mdpi.com/2075-1729/12/2/310>

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All diagrams are my own..