NovaQ10

Coenzyme Q10

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Summary

Coenzyme Q10 is a fat-soluble compound primarily synthesized by the body and also consumed in the diet.
Coenzyme Q10 is required for mitochondrial ATP synthesis and functions as an antioxidant in cell membranes and lipoproteins.
Endogenous synthesis and dietary intake appear to provide sufficient coenzyme Q10 to prevent deficiency in healthy people, although tissue levels of coenzyme Q10 decline with age.
Oral supplementation of coenzyme Q10 increases plasma, lipoprotein, and blood vessel levels, but it is unclear whether tissue coenzyme Q10 levels are increased, especially in healthy individuals. Coenzyme Q10 supplementation has resulted in clinical and metabolic improvement in some patients with hereditary mitochondrial disorders.
Although coenzyme Q10 supplementation may be a useful adjunct to conventional medical therapy for congestive heart failure, additional research is needed.
Roles for coenzyme Q10 supplementation in cardiovascular diseases, neurodegenerative diseases, cancer, and diabetes require further research.
Coenzyme Q10 supplementation does not appear to improve athletic performance.
Although coenzyme Q10 supplements are relatively safe, they may decrease the anticoagulant efficacy of warfarin.
Although the use of cholesterol-lowering medications known as HMG-CoA reductase inhibitors (statins) decreases circulating levels of coenzyme Q10, it is unclear whether coenzyme Q10 supplementation provides any health benefit to patients taking these drugs.

Introduction

Coenzyme Q10 is a member of the ubiquinone family of compounds. All animals, including humans, can synthesize ubiquinones, hence, coenzyme Q10 cannot be considered a vitamin (1). The name ubiquinone refers to the ubiquitous presence of these compounds in living organisms and their chemical structure, which contains a functional group known as a benzoquinone. Ubiquinones are fat-soluble molecules with anywhere from one to 12 isoprene (5-carbon) units. The ubiquinone found in humans, ubidecaquinone or coenzyme Q10, has a "tail" of ten isoprene units (a total of 50 carbon atoms) attached to its benzoquinone "head" (diagram) (2).

Disease Prevention

Aging

According to the free radical and mitochondrial theories of aging, oxidative damage of cell structures by reactive oxygen species (ROS) plays an important role in the functional declines that accompany aging (10). ROS are generated by mitochondria as a byproduct of ATP production. If not neutralized by antioxidants, ROS may damage mitochondria over time, causing them to function less efficiently and to generate more damaging ROS in a self-perpetuating cycle. Coenzyme Q10 plays an important role in mitochondrial ATP synthesis and functions as an antioxidant in mitochondrial membranes. Moreover, tissue levels of coenzyme Q10 have been reported to decline with age (9). One of the hallmarks of aging is a decline in energy metabolism in many tissues, especially liver, heart, and skeletal muscle. It has been proposed that age-associated declines in tissue coenzyme Q10 levels may play a role in this decline (11). In recent studies, lifelong dietary supplementation with coenzyme Q10 increased tissue concentrations of coenzyme Q10 but did not increase the lifespans of rats or mice (12, 13); however, one study showed that coenzyme Q10 supplementation attenuates the age-related increase in DNA damage (14). Presently, there is no scientific evidence that coenzyme Q10 supplementation prolongs life or prevents age-related functional declines in humans.

Cardiovascular disease

Oxidative modification of low-density lipoproteins (LDL) in arterial walls is thought to represent an early event leading to the development of atherosclerosis. Reduced coenzyme Q10 (CoQ10H2) inhibits the oxidation of LDL in the test tube (in vitro) and works together with α-TOH to inhibit LDL oxidation by reducing the alpha-TO· back to alpha-TOH. In the absence of a co-antioxidant, such as CoQ10H2 (or vitamin C), alpha-TOH can, under certain conditions, promote the oxidation of LDL in vitro (4). Supplementation with coenzyme Q10 increases the concentration of CoQ10H2 in human LDL (15). Studies in apolipoprotein E-deficient mice, an animal model of atherosclerosis, found that coenzyme Q10 supplementation with supra-pharmacological amounts of coenzyme Q10 significantly inhibited the formation of atherosclerotic lesions (16). Interestingly, co-supplementation of these mice with alpha-TOH and coenzyme Q10 was more effective in inhibiting atherosclerosis than supplementation with either alpha-TOH or coenzyme Q10 alone (17). Another important step in the development of atherosclerosis is the recruitment of immune cells known as monocytes into the blood vessel walls. This recruitment is dependent in part on monocyte expression of cell adhesion molecules (integrins). Supplementation of ten healthy men and women with 200 mg/day of coenzyme Q10 for ten weeks resulted in significant decreases in monocyte expression of integrins, suggesting another potential mechanism for the inhibition of atherosclerosis by coenzyme Q10 (18). Although coenzyme Q10 supplementation shows promise as an inhibitor of LDL oxidation and atherosclerosis, more research is needed to determine whether coenzyme Q10 supplementation can inhibit the development or progression of atherosclerosis in humans.

Disease Treatment

Mitochondrial encephalomyopathies

Mitochondrial encephalomyopathies represent a diverse group of genetic disorders resulting from numerous inherited abnormalities in the function of the mitochondrial electron transport chain. Coenzyme Q10 supplementation has resulted in clinical and metabolic improvement in some patients with various types of mitochondrial encephalomyopathies (19). Neuromuscular and widespread tissue coenzyme Q10 deficiencies have been found in a very small subpopulation of individuals with mitochondrial encephalomyopathies (20, 21). In those rare individuals with genetic defects in coenzyme Q10 biosynthesis, coenzyme Q10 supplementation has resulted in substantial improvement (22, 23). It is not clear whether coenzyme Q10 supplementation might have therapeutic benefit in patients with other mitochondrial disorders; a phase III clinical trial investigating that question is currently under way (23).