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).

Safety

Toxicity

There have been no reports of significant adverse side effects of oral coenzyme Q10 supplementation at doses as high as 1,200 mg/day for up to 16 months (61) and 600 mg/day for up to 30 months (73). In fact, 1,200 mg/day has recently been proposed as the observed safe level (OSL) for coenzyme Q10 (102). Some people have experienced gastrointestinal symptoms, such as nausea, diarrhea, appetite suppression, heartburn, and abdominal discomfort. These adverse effects may be minimized if daily doses higher than 100 mg are divided into two or three daily doses. Because controlled safety studies in pregnant and lactating women are not available, the use of coenzyme Q10 supplements by pregnant or breast-feeding women should be avoided (96, 103).

Drug Interactions

Warfarin

Concomitant use of warfarin (Coumadin) and coenzyme Q10 supplements has been reported to decrease the anticoagulant effect of warfarin in at least four cases (104). An individual on warfarin should not begin taking coenzyme Q10 supplements without consulting the health care provider who is managing his or her anticoagulant therapy. If warfarin and coenzyme Q10 are to be used concomitantly, blood tests to assess clotting time (prothrombin time; PT/INR) should be monitored frequently, especially in the first two weeks.

HMG-CoA reductase inhibitors (statins)

HMG-CoA reductase is an enzyme that plays a critical role in the regulation of cholesterol synthesis as well as coenzyme Q10 synthesis, although it is now recognized that there are additional rate-limiting steps in the biosynthesis of cholesterol and coenzyme Q10. HMG-CoA reductase inhibitors, also known as statins, are widely used cholesterol-lowering medications that may also decrease the endogenous synthesis of coenzyme Q10. Therapeutic use of statins, including simvastatin (Zocor), pravastatin (Pravachol), lovastatin (Mevacor, Altocor, Altoprev), rosuvastatin (Crestor), and atorvastatin (Lipitor), has been shown to decrease blood plasma or serum levels of coenzyme Q10 (105-114). However, it has been suggested that blood coenzyme Q10 concentrations should be reported only after normalizing to total lipid or cholesterol levels because coenzyme Q10 circulates with lipoproteins and levels of coenzyme Q10 are highly dependent upon levels of circulating lipids (115, 116). Given the lipid-lowering effects of statins, it is therefore unclear whether these drugs actually decrease coenzyme Q10 levels independent of a reduction in circulating lipids. Also, very few studies have examined coenzyme Q10 content in target organs; thus, it is not clear whether statin therapy affects coenzyme Q10 concentrations in the body's tissues (111, 113, 117). At present, more research is needed to determine whether coenzyme Q10 supplementation might be beneficial for those taking HMG-CoA reductase inhibitors.