NovaQ10

Coenzyme Q10

Linus Pauling Institute | Oregon State University

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).

Cardiovascular diseases

Congestive heart failure

Impairment of the heart's ability to pump enough blood for all of the body's needs is known as congestive heart failure. In coronary artery disease, accumulation of atherosclerotic plaque in the coronary arteries may prevent parts of the heart muscle from getting adequate blood supply, ultimately resulting in cardiac damage and impaired pumping ability. Myocardial infarction (MI) may also damage the heart muscle, leading to heart failure. Because physical exercise increases the demand on the weakened heart, measures of exercise tolerance are frequently used to monitor the severity of heart failure. Echocardiography is also used to determine the left ventricular ejection fraction, an objective measure of the heart's pumping ability (25). The finding that myocardial coenzyme Q10 levels were lower in patients with more severe versus milder heart failure led to several clinical trials of coenzyme Q10 supplementation in heart failure patients (26). A number of small intervention trials that administered supplemental coenzyme Q10 (100-300 mg/day of coenzyme Q10 for one to three months) to congestive heart failure patients, in conjunction with conventional medical therapy, have demonstrated improvements in some cardiac function measures (27-29). However, other researchers have found that supplementing the diet with 100-200 mg/day of coenzyme Q10, along with conventional medical therapy, did not significantly improve left ventricular ejection fraction or exercise performance in heart failure patients (30, 31). A 2006 meta-analysis of ten randomized controlled trials found that coenzyme Q10 supplementation (99-200 mg/day for one to six months) in heart failure patients resulted in a significant, 3.7% improvement in left ventricular ejection fraction; the effect was stronger in patients not taking angiotensin-converting enzyme inhibitors (32). A slight increase in cardiac output (0.28 L/min) was also found with coenzyme Q10 supplementation, but this analysis only included two trials (60 mg/day for one month or 200 mg/day for three months) (32). A recent study in 236 heart failure patients found that lower plasma coenzyme Q10 levels were associated with a heightened risk of mortality (33); however, a larger study of 1,191 heart failure patients found that plasma coenzyme Q10 level was a biomarker of advanced heart disease and not an independent predictor of clinical outcomes in heart failure patients (34). Although there is some evidence that coenzyme Q10 supplementation may be of benefit, large well-designed intervention trials are needed to determine whether coenzyme Q10 supplementation has value as an adjunct to conventional medical therapy in the treatment of congestive heart failure. One such large trial is presently being conducted.

Myocardial infarction and cardiac surgery

The heart muscle may become oxygen-deprived (ischemic) as the result of myocardial infarction (MI) or during cardiac surgery. Increased generation of ROS when the heart muscle's oxygen supply is restored (reperfusion) is thought to be an important contributor to myocardial damage occurring during ischemia-reperfusion. Pretreatment of animals with coenzyme Q10 has been found to decrease myocardial damage due to ischemia-reperfusion (35). Another potential source of ischemia-reperfusion injury is aortic clamping during some types of cardiac surgery, such as coronary artery bypass graft (CABG) surgery. Three out of four placebo-controlled trials found that coenzyme Q10 pretreatment (100-300 mg/day for 7-14 days prior to surgery) provided some benefit in short-term outcome measures after CABG surgery (36, 37). In the placebo-controlled trial that did not find preoperative coenzyme Q10 supplementation to be of benefit, patients were treated with 600 mg of coenzyme Q10 12 hours prior to surgery (38), suggesting that preoperative coenzyme Q10 treatment may need to commence at least one week prior to CABG surgery in order to realize any benefit. Although the results are promising, these trials have included relatively few people and have only examined outcomes shortly after CABG surgery.

Angina pectoris

Myocardial ischemia may also lead to chest pain known as angina pectoris. People with angina pectoris often experience symptoms when the demand for oxygen exceeds the capacity of the coronary circulation to deliver it to the heart muscle, e.g., during exercise. Five small placebo-controlled studies have examined the effects of oral coenzyme Q10 supplementation (60-600 mg/day) in addition to conventional medical therapy in patients with chronic stable angina (28). In most of the studies, coenzyme Q10 supplementation improved exercise tolerance and reduced or delayed electrocardiographic changes associated with myocardial ischemia compared to placebo. However, only two of the studies found significant decreases in symptom frequency and nitroglycerin consumption with coenzyme Q10 supplementation. Presently, there is only limited evidence suggesting that coenzyme Q10 supplementation would be a useful adjunct to conventional angina therapy.

Linus Carl Pauling PROTOCOL

Cardiologist have been kept in the dark about the vitamin C connection. Few cardiovascular drugs benefit heart patients. Several exacerbate heart conditions and should be eliminated in favor of the following othomolecular protocols:

Take Vitamin C as ascorbic acid (or sodium ascorbate, but this form may be less effective) up to bowel tolerance (6 to 18 g per day in divided doses.)
The half-life of vitamin C in the blood stream is 30 minutes. NIH findings indicate that a minimum of 500 mg every 4 hours leads to highest sustained blood levels. Take more before bed, trips, etc.
Trouble with bloating/gas/diarrhea after your vitamin C? Try Liposomal Vitamin C
Take Lysine 3000 to 6000 mg (3 to 6 g) daily for the therapeutic value. Take 2000 to 3000 mg (2 to 3 g) daily for prevention.
Supplement Coenzyme Q10 (100 - 300 mg)
Note: Vitamin C and several vitamins will help stimulate your own synthesis of CoQ10. CoQ10 is a vital substance for energy and proper heart function. Popular drugs interfere with your body's own production of CoQ10, and they may lead to heart failure.
NEW: Eliminate man-made/processed fats, such as trans fats and hydrogenated oils. Supplement Omega-3 rich oils, e.g. evening primrose, flaxaseed, and certain fish oils.
"Research has shown that an Omega-3 Index of 8 percent to 10 percent reduces a person's relative risk of death from coronary heart disease by 40 percent, and from sudden cardiac death by 90 percent." This benefit probably results from restored insulin-mediated glucose/vitamin C uptake into cells. [See: Protocol for Reversing Diabetes Type II by Eliminating Hydrogenated and Trans Fats and adding Omega-3 oils...]
Note: Following an Atkins-style diet will eliminate most trans fats because these "poisons" appear mostly in processed carbohydrate foods such as cookies, crackers, snacks, etc. Butter is vastly supperior to margarine. Natural saturated fats are superior to any fats or oils processed for longer shelf life.
Take the amino acid proline from 250 mg to 2000 mg daily.
This factor, added to Pauling's original protocol, and recommended by Mathias Rath, may lower elevated Lp(a) within 6 to 14 months. It is difficult to suggest an optimum dose for everyone because the healthy body can manufacture its own proline. A few alternative doctors recommend 2 g (2000 mg), but the Tower HeartTechnology formula has produced consistent good results. It can apparently lower Lp(a) with smaller dosages of proline.