Mol Biotechnol. 2007 Sep;37(1):31-7.

Bioenergetic and antioxidant properties of coenzyme Q10: recent developments.

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Abstract

For a number of years, coenzyme Q (CoQ10 in humans) was known for its key role in mitochondrial bioenergetics; later studies demonstrated its presence in other subcellular fractions and in plasma, and extensively investigated its antioxidant role. These two functions constitute the basis on which research supporting the clinical use of CoQ10 is founded. Also at the inner mitochondrial membrane level, coenzyme Q is recognized as an obligatory co-factor for the function of uncoupling proteins and a modulator of the transition pore. Furthermore, recent data reveal that CoQ10 affects expression of genes involved in human cell signalling, metabolism, and transport and some of the effects of exogenously administered CoQ10 may be due to this property. Coenzyme Q is the only lipid soluble antioxidant synthesized endogenously. In its reduced form, CoQH2, ubiquinol, inhibits protein and DNA oxidation but it is the effect on lipid peroxidation that has been most deeply studied. Ubiquinol inhibits the peroxidation of cell membrane lipids and also that of lipoprotein lipids present in the circulation. Dietary supplementation with CoQ10 results in increased levels of ubiquinol-10 within circulating lipoproteins and increased resistance of human low-density lipoproteins to the initiation of lipid peroxidation. Moreover, CoQ10 has a direct anti-atherogenic effect, which has been demonstrated in apolipoprotein E-deficient mice fed with a high-fat diet. In this model, supplementation with CoQ10 at pharmacological doses was capable of decreasing the absolute concentration of lipid hydroperoxides in atherosclerotic lesions and of minimizing the size of atherosclerotic lesions in the whole aorta. Whether these protective effects are only due to the antioxidant properties of coenzyme Q remains to be established; recent data point out that CoQ10 could have a direct effect on endothelial function. In patients with stable moderate CHF, oral CoQ10 supplementation was shown to ameliorate cardiac contractility and endothelial dysfunction. Recent data from our laboratory showed a strong correlation between endothelium bound extra cellular SOD (ecSOD) and flow-dependent endothelial-mediated dilation, a functional parameter commonly used as a biomarker of vascular function. The study also highlighted that supplementation with CoQ10 that significantly affects endothelium-bound ecSOD activity. Furthermore, we showed a significant correlation between increase in endothelial bound ecSOD activity and improvement in FMD after CoQ10 supplementation. The effect was more pronounced in patients with low basal values of ecSOD. Finally, we summarize the findings, also from our laboratory, on the implications of CoQ10 in seminal fluid integrity and sperm cell motility.

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Oxidative stress, endothelial function and coenzyme Q10.

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Abstract

Reactive oxygen species seem to play an important role in vascular homeostasis. In conditions of high oxidative stress, such as chronic heart failure and multiple coronary risk factors, the rate of inactivation of nitric oxide to peroxynitrite by superoxide anions may be reduced by CoQ10, which can also protect against nitrosative damage. CoQ10 may also influence vascular function indirectly via inhibition of oxidative damage to LDL. Patients with lower levels of extracellular superoxide dismutase (ecSOD) demonstrate greater improvements than patients with normal ec-SOD levels, suggesting that the higher the oxidative stress the greater the improvement in the endothelium-dependent relaxation after the administration of a compound with antioxidant properties like CoQ10. Future studies are needed to inquire whether these effects may translate into benefits in clinical practice.

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Reversal of mitochondrial dysfunction by coenzyme Q10 supplement improves endothelial function in patients with ischaemic left ventricular systolic dysfunction: a randomized controlled trial.

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Abstract

AIMS:
Coronary artery disease (CAD) is associated with endothelial dysfunction and mitochondrial dysfunction (MD). The aim of this study was to investigate whether co-enzyme Q10 (CoQ) supplementation, which is an obligatory coenzyme in the mitochondrial respiratory transport chain, can reverse MD and improve endothelial function in patients with ischaemic left ventricular systolic dysfunction (LVSD).
METHODS AND RESULTS:
We performed a randomized, double-blind, placebo-controlled trial to determine the effects of CoQ supplement (300 mg/day, n=28) vs. placebo (controls, n=28) for 8 weeks on brachial flow-mediated dilation (FMD) in patients with ischaemic LVSD(left ventricular ejection fraction <45%). Mitochondrial function was determined by plasma lactate/pyruvate ratio (LP ratio). After 8 weeks, CoQ-treated patients had significant increases in plasma CoQ concentration (treatment effect 2.20 μg/mL, P<0.001) and FMD (treatment effect 1.51%, P=0.03); and decrease in LP ratio (treatment effect -2.46, P=0.03) compared with controls. However, CoQ treatment did not alter nitroglycerin-mediated dilation, blood pressure, blood levels of fasting glucose, haemoglobin A1c, lipid profile, high-sensitivity C-reactive protein and oxidative stress as determined by serum superoxide dismutase and 8-isoprostane (all P>0.05). Furthermore, the reduction in LP ratio significantly correlated with improvement in FMD (r=-0.29, P=0.047).
CONCLUSION:
In patients with ischaemic LVSD, 8 weeks supplement of CoQ improved mitochondrial function and FMD; and the improvement of FMD correlated with the change in mitochondrial function, suggesting that CoQ improved endothelial function via reversal of mitochondrial dysfunction in patients with ischaemic LVSD.

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Role of coenzyme Q10 (CoQ10) in cardiac disease, hypertension and Meniere-like syndrome.

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Abstract

Coenzyme Q10 (ubiquinone) is a mitochondrial coenzyme which is essential for the production of ATP. Being at the core of cellular energy processes it assumes importance in cells with high energy requirements like the cardiac cells which are extremely sensitive to CoQ10 deficiency produced by cardiac diseases. CoQ10 has thus a potential role for prevention and treatment of heart ailments by improving cellular bioenergetics. In addition it has an antioxidant, a free radical scavenging and a vasodilator effect which may be helpful in these conditions. It inhibits LDL oxidation and thus the progression of atherosclerosis. It decreases proinflammatory cytokines and decreases blood viscosity which is helpful in patients of heart failure and coronary artery disease. It also improves ischemia and reperfusion injury of coronary revascularisation. Significant improvement has been observed in clinical and hemodynamic parameters and in exercise tolerance in patients given adjunctive CoQ10 in doses from 60 to 200 mg daily in the various trials conducted in patients of heart failure, hypertension, ischemic heart disease and other cardiac illnesses. Recently it has been found to be an independent predictor of mortality in congestive heart failure. It has also been found to be helpful in vertigo and Meniere-like syndrome by improving the immune system. Further research is going on to establish firmly its role in the therapy of cardiovascular diseases.

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Coenzyme Q10 terclatrate and creatine in chronic heart failure: a randomized, placebo-controlled, double-blind study.

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Abstract

BACKGROUND: Studies have suggested that micronutrient deficiency has some role in the progression of chronic heart failure (CHF).
HYPOTHESIS: Oral supplementation with coenzyme Q(10) (CoQ(10)) and creatine may reduce mitochondrial dysfunction that contributes to impaired physical performance in CHF.
METHODS: We conducted a randomized, double-blind, placebo-controlled trial to determine the effect of a mixture of water-soluble CoQ(10) (CoQ(10) terclatrate; Q-ter) and creatine on exercise tolerance and health-related quality of life. Exercise tolerance was measured as total work capacity (kg·m) and peak oxygen consumption (VO(2), mL/min/kg), both from a cardiopulmonary exercise test. Health-related quality of life was measured by the Sickness Impact Profile (SIP) in CHF secondary to left ventricular systolic dysfunction (left ventricular ejection fraction $\leq$ 35%). After baseline assessment, 67 patients with stable CHF were randomized to receive Q-ter 320 mg + creatine 340 mg (n = 35) or placebo (n = 32) once daily for 8 weeks.
RESULTS: At multivariate analysis, 8-week peak VO(2) was significantly higher in the active treatment group than in the placebo group (+1.8 $\pm$ 0.9 mL/min/kg, 95% CI: 0.1-3.6, $P < 0.05$). No untoward effects occurred in either group.
CONCLUSIONS: This study suggests that oral Q-ter and creatine, added to conventional drug therapy, exert some beneficial effect on physical performance in stable systolic CHF. Results may support the design of larger studies aimed at assessing the long-term effects of this treatment on functional status and harder outcomes.