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## Co-enzyme Q10 supplementation for the primary prevention of cardiovascular disease.

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### Author information

#### Abstract

**BACKGROUND:** Cardiovascular disease (CVD) remains the number one cause of death and disability worldwide and public health interventions focus on modifiable risk factors, such as diet. Coenzyme Q10 (CoQ10) is an antioxidant that is naturally synthesised by the body and can also be taken as a dietary supplement. Studies have shown that a CoQ10 deficiency is associated with cardiovascular disease.

**OBJECTIVES:** To determine the effects of coenzyme Q10 supplementation as a single ingredient for the primary prevention of CVD.

**SEARCH METHODS:** We searched the Cochrane Central Register of Controlled Trials (CENTRAL 2013, Issue 11); MEDLINE (Ovid, 1946 to November week 3 2013); EMBASE (Ovid, 1947 to 27 November 2013) and other relevant resources on 2 December 2013. We applied no language restrictions.

**SELECTION CRITERIA:** Randomised controlled trials (RCTs) lasting at least three months involving healthy adults or those at high risk of CVD but without a diagnosis of CVD. Trials investigated the supplementation of CoQ10 alone as a single supplement. The comparison group was no intervention or placebo. The outcomes of interest were CVD clinical events and major CVD risk factors, adverse effects and costs. We excluded any trials involving multifactorial lifestyle interventions to avoid confounding.

**DATA COLLECTION AND ANALYSIS:** Two authors independently selected trials for inclusion, abstracted data and assessed the risk of bias. We contacted authors for additional information where necessary.

**MAIN RESULTS:** We identified six RCTs with a total of 218 participants randomised, one trial awaiting classification and five ongoing trials. All trials were conducted in participants at high risk of CVD, two trials examined CoQ10 supplementation alone and four examined CoQ10 supplementation in patients on statin therapy; we analysed these separately. All six trials were small-scale, recruiting between 20 and 52 participants; one trial was at high risk of bias for incomplete outcome data and one for selective reporting; all studies were unclear in the method of allocation and therefore for selection bias. The dose of CoQ10 varied between 100 mg/day and 200 mg/day and the duration of the interventions was similar at around three months. No studies reported mortality or non-fatal cardiovascular events. None of the included studies provided data on adverse events. Two trials examined the effect of CoQ10 on blood pressure. For systolic blood pressure we did not perform a meta-analysis due to significant heterogeneity. In one trial CoQ10 supplementation had no effect on systolic blood pressure (mean difference (MD) -1.90 mmHg, 95% confidence interval (CI) -13.17 to 9.37, 51 patients randomised). In the other trial there was a statistically significant reduction in systolic blood pressure (MD -15.00 mmHg, 95% CI -19.06 to -10.94, 20 patients randomised). For diastolic blood pressure we performed a random-effects meta-analysis, which showed no evidence of effect of CoQ10 supplementation when these two small trials were pooled (MD -1.62 mmHg, 95% CI -5.2 to 1.96). One trial (51 patients randomised) looked at the effect of CoQ10 on lipid levels. The trial showed no evidence of

effect of CoQ10 supplementation on total cholesterol (MD 0.30 mmol/L, 95% CI -0.10 to 0.70), high-density lipoprotein (HDL)-cholesterol (MD 0.02 mmol/L, 95% CI -0.13 to 0.17) or triglycerides (MD 0.05 mmol/L, 95% CI -0.42 to 0.52). Of the four trials that investigated CoQ10 supplementation in patients on statin therapy, three of them showed that simultaneous administration of CoQ10 did not significantly influence lipid levels or systolic blood pressure levels between the two groups. The fourth trial showed a significant increase in the change in total and low-density lipoprotein (LDL)-cholesterol at three months across the four arms of the trial ( $\alpha$ -tocopherol, CoQ10, CoQ10 +  $\alpha$ -tocopherol and placebo), however the way in which the data were presented meant that we were unable to determine if there was any significant difference between the CoQ10 only and placebo arms. In contrast, there was no significant difference in the change in HDL-cholesterol and triglycerides after three months between the four arms of the trial.

**AUTHORS' CONCLUSIONS:** There are very few studies to date examining CoQ10 for the primary prevention of CVD. The results from the ongoing studies will add to the evidence base. Due to the small number of underpowered trials contributing to the analyses, the results presented should be treated with caution and further high quality trials with longer-term follow-up are needed to determine the effects on cardiovascular events.

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