

## Original Article

# Treatment of coenzyme Q10 for 24 weeks improves lipid and glycemic profile in dyslipidemic individuals

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**KEYWORDS:**

CoQ10;  
Dyslipidemia;  
Insulin resistance;  
Clinical trial

**BACKGROUND:** The use of coenzyme Q10 (CoQ10) as an adjuvant treatment with routine clinical therapy against metabolic diseases has shown benefit. However, the effect of CoQ10 as a primary preventive agent against cardiovascular diseases (CVDs) has not been well studied.

**OBJECTIVE:** The objective of this study was to investigate the effect of CoQ10 on CVD risk factors in dyslipidemic patients.

**METHODS:** In this randomized, double-blinded, placebo-controlled trial, 101 dyslipidemic subjects without taking any hypoglycemic or hypolipidemic drugs were administered to 120 mg CoQ10 or placebo daily for 24 weeks. Anthropometric parameters, lipid and glycemic profile, biomarkers of inflammation, and antioxidant capacity were evaluated before and after 12 and 24 weeks of intervention.

**RESULTS:** All 101 subjects were included in the analysis. On the 12th week, compared to placebo, CoQ10 supplementation decreased systolic ( $P = .010$ ) and diastolic pressure ( $P = .001$ ) and increased serum total antioxidant capacity (TAC;  $P = .003$ ). On the 24th week, compared to placebo, CoQ10 supplementation further lowered blood pressure and TAC, reduced triglyceride ( $P = .020$ ) and low-density lipoprotein cholesterol ( $P = .016$ ), and increased ApoA-I ( $P < .001$ ) while decreasing homeostasis model assessment of insulin resistance index ( $P = .009$ ). Adjustment for change of physical activity and energy intake did not alter the effect of CoQ10 on the aforementioned parameters but led to significant decrease of non-high-density lipoprotein cholesterol in CoQ10 group compared to placebo ( $P = .031$ ).

P.Z., C.Y., and H.G. contributed equally to this work.

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Clinical Trial Registry: This trial was registered at [clinicaltrials.gov](http://clinicaltrials.gov) as NCT02407548.

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**CONCLUSIONS:** Twenty-four-week treatment of CoQ10 ameliorates multiple CVD risk factors. The versatility and safety of CoQ10 makes it a potential candidate for the primary prevention of CVD.  
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## Introduction

Dyslipidemia is a well-documented and important risk factor of cardiovascular diseases (CVDs) along with the other components of the metabolic syndrome (MetS), including insulin resistance (IR), glucose intolerance, and hypertension. The regulations of these conditions are thought to be essential for the primary prevention of CVD.<sup>1,2</sup>

Increasing attention has been directed toward finding effective strategies to detect and treat the risk factors of CVD. Coenzyme Q10 (CoQ10) is one such candidate. As a lipophilic benzoquinone, CoQ10 is rich in mammalian organs, such as heart, liver, and kidneys. It is present in the membrane of almost all mammalian cell types and can reversibly accept or lose 2 electrons to form hydroquinone or benzoquinone, respectively, which makes it a crucial component in the mitochondrial electron transport chain and important constituent of membrane oxidoreductase systems.<sup>3</sup> It has been reported that CoQ10 exerts anti-lipogenesis,<sup>4</sup> anti-diabetes,<sup>5</sup> anti-atherosclerosis,<sup>6,7</sup> and broad gene regulatory properties<sup>8</sup> in studies in animals and cells. Previous clinical trials have shown that adding CoQ10 to existing antihypertensive treatments further lowered systolic blood pressure (SBP) and diastolic blood pressure (DBP) compared to treatment with routine antihypertensive agents alone.<sup>9</sup> In diabetic patients, CoQ10 supplementation promoted the decrease in fasting blood glucose (FBG) and glycosylated hemoglobin with routine hypoglycemic therapy.<sup>10,11</sup> However, CoQ10 could decrease low-density lipoprotein cholesterol (LDL-c) and total cholesterol (TC) in non-statin-treated patients but not patients treated with statins.<sup>12,13</sup> These findings piqued our interest to explore whether CoQ10 exerts antihypertensive, hypoglycemic, and lipid-lowering effects by itself as an initial intervention.

Therefore, the present study was designed to investigate the 24-week effect of CoQ10 on glycemic or lipid profile and other MetS components in subjects with dyslipidemia. Furthermore, we investigated if improvements of inflammation and oxidative stress were involved in the metabolic improvement of CoQ10 supplementation.

## Methods

### Study design and subjects recruitment

All subjects were recruited from 2 community health service centers in Guangzhou and Foshan, Guangdong Province, from July 2015 to September 2016 by flyers and posters. Free and rapid lipid tests with CardioChek PA

Analyzer (PTS Diagnostics) were issued in these 2 centers for primary screening. Subjects aged 18 to 70 years were diagnosed of dyslipidemia if they satisfied 2 or more of the following 4 conditions<sup>14</sup>: serum fasting TC  $\geq$  5.20 mmol/L (200 mg/dL), fasting total triglycerides (TGs)  $\geq$  1.70 mmol/L (150 mg/dL), fasting LDL-c  $\geq$  3.12 mmol/L (120 mg/dL), and fasting HDL-cholesterol (HDL-c)  $\leq$  0.91 mmol/L (35 mg/dL). Also, the subjects had no intention to change their diets and physical activity during the trial. The exclusion criteria were as follows: serum fasting TC  $\geq$  8.0 mmol/L (309 mg/dL); fasting TG  $\geq$  4.5 mmol/L (395 mg/dL); history of CVD or atherosclerosis including angina, myocardial infarction, stroke, coronary artery bypass grafting, coronary angioplasty, or angiographically defined coronary heart disease; hyperthyroidism or hypothyroidism; cancer; liver or renal dysfunction; or the consumption of any medicine or dietary supplement that influences lipid and glucose metabolism, inflammation, and oxidative stress. A total of 127 people met the screening criteria and were subjected to detailed baseline examination. Baseline examination included blood sample collection, anthropometric measurement, and assessment of basic information and lifestyle conditions. Basic information regarding birth, sex, occupation, marriage, education, smoking, alcohol consumption, and history of metabolic disease was collected. Twenty-one subjects were ineligible, and 5 refused to participate. A researcher who did not participate in data collection, analysis, or reporting was in charge of randomized assignment and managed the packaged supplements from the external pharmaceuticals company. Computer-generated random numbers were allocated to each patient at the time of recruitment. After matching sex and age in 4 blocks, 101 subjects meeting the inclusion criteria at baseline examination were randomly administrated to 120 mg CoQ10 or placebo daily.

Two types of softgel with identical appearance were obtained from an external pharmaceuticals company (BY-Health Co Ltd, China) for intervention. Softgel in CoQ10 group contained 30 mg CoQ10 dissolved in soybean oil and identical quantity of soybean oil in placebo group. The different groups were identified by codes printed on the packaging bottles. Subjects, investigators, and data analysts were blinded from the group information. Subjects in each group consumed 2 corresponding softgels twice a day after meals (4 softgels daily) for a total daily intake with or without 120 mg CoQ10. The intervention continued for 24 weeks, and the subjects were requested to maintain their normal lifestyle and visit the study center every 4 weeks. During each visit, the remaining softgels were counted as an assessment of the adherence to the protocol. Adverse

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