Future Journal of Pharmaceutical Sciences

RESEARCH

Open Access

Comparative study of the bioavailability of two coenzyme Q10 products following single-dose and 3-week oral administration in healthy adults

Zala Jenko Pražnikar[1], Nina Mohorko[1], Vasilij Valenčič[2], Vasja Valetič[2], Dejan Gmajner[2], Marco Morosetti[3], Patrick Orlando[3*†] and Ana Petelin[1*†]

Abstract

Background Coenzyme Q10 (CoQ10) is a widely used food supplement due to its potential therapeutic benefits, which largely depend on its bioavailability. This study compared the bioavailability of two CoQ10 formulations—unencapsulated ubiquinone (standard) and microencapsulated CoQ10 (BMT®)—in healthy adults following both acute and chronic oral administration, and examined their effects on selected metabolic parameters.

Methods This randomized, two-way crossover trial included 25 healthy adult participants. Each received a single 100 mg dose of both formulations, as well as a 3-week daily regimen of 100 mg of one of the formulations. Bioavailability was assessed by calculating the area under the curve for plasma CoQ10 concentrations over 48 h after a single dose administration and over the 3-week supplementation period.

Results The microencapsulated CoQ10 demonstrated significantly superior bioavailability at 6, 12, and 24 h, with an average fold increase of 1.8, 2.4, and 1.9, compared to standard. Following chronic supplementation, plasma CoQ10 concentrations increased by approximately 80% with standard and 95% with the microencapsulated formulation relative to baseline, while differences between formulations were no longer significant. A significant effect on vitamin D levels was also observed: microencapsulated CoQ10 led to higher vitamin D concentrations after 3 weeks ($P = 0.001$).

Conclusion These results confirm the safety and superior acute bioavailability of microencapsulated CoQ10 and suggest possible broader metabolic benefits.

Trial registration ClinicalTrials.gov (NCT06736366). Registered 11 December 2025, https://clinicaltrials.gov/study/NCT06736366#study-record-dates

Keywords Coenzyme Q10, Bioavailability, Food supplement, Microencapsulation

†Patrick Orlando and Ana Petelin have contributed equally to this work.

*Correspondence:
Patrick Orlando
p.orlando@staff.univpm.it
Ana Petelin
ana.petelin@upr.si
Full list of author information is available at the end of the article

Introduction

Coenzyme Q10 (CoQ10), also known as ubiquinone, is a highly lipophilic compound found in nearly every cell membrane in the human body [1]. Its primary and well-established biochemical function is serving as a cofactor in the mitochondrial electron transport chain, where it facilitates a series of redox reactions involved in the synthesis of adenosine triphosphate (ATP). As a result, tissues with high metabolic demands—such as the heart, kidneys, and liver—exhibit the highest concentrations of CoQ10. Because most cellular processes depend on adequate ATP production, CoQ10 plays a critical role in supporting the function and health of all human tissues and organs [2, 3]. In addition to its role in energy metabolism, CoQ10 is a potent lipid-soluble antioxidant. It protects against oxidative damage by preventing the formation of free radicals and limiting oxidative modifications to proteins, lipids, and DNA. In the bloodstream, CoQ10 is primarily transported in lipoproteins, and its plasma concentration has been shown to correlate closely with total and low-density lipoprotein (LDL cholesterol) levels [4]. Within LDL particles, CoQ10 acts as the first line of antioxidant defense during oxidative stress [5]. CoQ10 levels have been found to decline in various pathological conditions characterized by elevated oxidative stress, including chronic diseases and disorders associated with increased reactive oxygen species (ROS) production [6]. Reduced CoQ10 concentrations have also been reported in association with genetic mutations, cancer, and the use of statin medications [2].

CoQ10 is synthesized endogenously from the amino acid tyrosine (or phenylalanine) and the tail subunit IPP derived from the mevalonate pathway [7]. However, evidence indicates that the efficiency of endogenous biosynthesis declines with age [1, 2]. Exogenous CoQ10 can be obtained from various dietary sources, including fatty fish (e.g., salmon and tuna), organ meats (e.g., liver), and whole grains. Nevertheless, typical daily intake in Western diets is estimated to be only 3–5 mg [8], which is insufficient to meaningfully increase plasma CoQ10 levels.

CoQ10 has limited gastrointestinal absorption due to its high molecular weight, poor water solubility, and relatively low lipid solubility [9]. Its absorption follows a pathway similar to that of vitamin E, beginning with emulsification and micelle formation facilitated by pancreatic and bile secretions in the small intestine. In addition, the crystalline structure of CoQ10 further limits its dissolution and contributes to variability in absorption among conventional formulations, as efficient dispersion is required prior to uptake [9, 10]. Given both low dietary intake and poor absorption, CoQ10 supplementation—typically around 100 mg/day—is often recommended in cases of deficiency to restore antioxidant capacity and prevent LDL oxidation [2]. While there is limited data on the effects of CoQ10 supplementation in healthy individuals, some studies have reported improvements in metabolic status in populations with metabolic syndrome [11, 12], type 2 diabetes mellitus [13], and coronary artery disease [14]. Conversely, other studies have shown no significant effects; for example, supplementation with 200 mg/day of CoQ10 for 12 weeks did not alter lipid profiles, inflammatory markers, or oxidative stress biomarkers in obese individuals [15].

Assessing the efficacy of CoQ10 supplementation requires careful consideration of the formulation's absorption and bioavailability. Numerous promising efforts have been made to enhance CoQ10's bioavailability, including reducing particle size, improving crystal dispersion, and enhancing water dispersibility through techniques such as solubilization, complexation, lipid-based delivery systems, and chemical reduction [8–10, 16, 17]. Conventional, non-encapsulated CoQ10 formulations have been widely studied, with evidence indicating variable and often limited bioavailability depending on formulation characteristics [9, 10, 16, 17]. More recently, advanced delivery strategies such as microencapsulation have been developed, aiming to improve stability, dispersion, and gastrointestinal uptake of CoQ10 [18]. While these approaches are promising, data on specific proprietary technologies remain limited, and further studies are needed to evaluate their performance under controlled conditions.

The objective of this study was to investigate the bioavailability of CoQ10 after a single dose administration and following 3 weeks of daily supplementation in healthy adults, comparing a standard unencapsulated formulation with a microencapsulated product. This randomized, two-way crossover trial involved administration of a single 100 mg oral dose of CoQ10, followed by a 3-week supplementation phase (100 mg/day), using two different formulations: (1) capsules containing standard ubiquinone and (2) capsules containing microencapsulated CoQ10. The primary aim of the single-dose phase was to provide comparative data under controlled conditions of the relative bioavailability of the two formulations based on plasma CoQ10 concentrations. The secondary objective was to evaluate the safety and bioavailability of CoQ10 after chronic supplementation, along with its effects on selected metabolic parameters.

Materials and methods

Study population

Healthy adult volunteers were recruited from the Primorska region of Slovenia through internet forums, email lists, and local newspaper advertisements.

Eligible participants were male and female adults aged 50–65 years, with a body weight of 70 ± 5 kg for women and 85 ± 5 kg for men. All participants were required to be in good general health and to abstain from taking any food supplements for at least two weeks prior to and throughout the study period. Exclusion criteria included the use of prescription medications or food supplements within two weeks before study initiation; a clinically significant history of gastrointestinal, hepatic, renal, cardiovascular, or metabolic disease (including diabetes); any other serious acute or chronic illness; or inadequate venous access as assessed by the investigator.

Sample size calculation was based on previously published data [19–21], assuming a statistical power of 0.9, alpha level of 0.05, and an effect size of 0.5. It was determined that 24 participants would be sufficient to detect a two-fold difference between the standard and the test product. To account for potential dropouts, the sample size was increased to 30 participants.

The study protocol was approved by the National Medical Ethics Committee of Slovenia (No. 0120-557/2017/4; Ministry of Health, Republic of Slovenia) and registered on ClinicalTrials.gov (NCT06736366). The study was conducted in accordance with local regulatory requirements and the Declaration of Helsinki. All participants provided written informed consent prior to enrolment.

Tested CoQ10 formulations

For the purposes of this study, two coenzyme Q10-based products were used, both containing coenzyme Q10 compliant with the requirements of the United States Pharmacopeia (USP), European Pharmacopoeia (Ph. Eur. 8.0), Japanese Pharmacopoeia (JP), and British Pharmacopoeia (BP), sourced from the same supplier and the same production batch. Coenzyme Q10 (ubiquinone) was supplied by Pharmavit (batch number: 51-2401146). One product contained coenzyme Q10 in its conventional, non-encapsulated form, while the second product was formulated using Biostile Microencapsulated Technology®. BMT® coenzyme Q10 was produced by the company Biostile using its proprietary microencapsulation know-how, registered as Biostile Microencapsulated Technology® (EUIPO trademark filing number: 018312914). The production process was carried out under controlled technological conditions, including defined mixing regimes, temperature control, and material flow management. All raw materials—coenzyme Q10 (ubiquinone), gum arabic, corn starch, olive oil, and silicon dioxide—were introduced into a dedicated process reactor vessel in an aqueous environment. The system was intensively mixed to promote uniform dispersion of the lipophilic active compound and the formation of a stable microencapsulated suspension. The microencapsulation approach was intended to improve the dispersibility, handling properties, and technological stability of coenzyme Q10. Acetic and citric acid were subsequently added to adjust the pH of the system and to support physicochemical stability during further processing steps. The resulting slurry was then allowed to undergo decantation and was conditioned for controlled water removal. Freeze-drying (lyophilization) was employed as a gentle dehydration technique to limit oxidative stress and minimize thermal degradation, thereby preserving the structural integrity of microencapsulated BMT® coenzyme Q10. The obtained dry intermediate was subsequently blended with a defined amount of maltodextrin in order to achieve a standardized final composition containing 30% (w/w) coenzyme Q10. Final mixing was performed in a suitable industrial mixer to ensure batch homogeneity and reproducibility. The final product was subjected to routine quality control procedures, including verification of coenzyme Q10 content and homogeneity. All raw materials used complied with applicable food-grade quality standards and were suitable for use in food supplement applications.

Therefore, the study was conducted with two different formulations (Table 1):

(A) The standard product Coenzyme Q10 (ubiquinone);

Table 1 Tested CoQ10 formulations

Formulation	Company product name	Ingredients	CoQ10 intake (mg)
A	CoQ10 USP (Ubiquinone; non-encapsulated)	Maltodextrin, capsule (HPMC—hydroxypropyl methyl cellulose), coenzyme Q10 (ubiquinone)	100
B	BMT® CoQ10 (CoQ10 microencapsulated)	Maltodextrin, BMT® coenzyme Q10 [maltodextrin, coenzyme Q10 (ubiquinone), stabiliser (gum arabic), corn starch, olive oil, anti-caking agent (silicon dioxide), acidity regulators (acetic acid, citric acid)], capsule (HPMC—hydroxypropyl methyl cellulose)	100

A, the standard product Coenzyme Q10 (ubiquinone); B, the investigational product 1 (BMT® Coenzyme Q10 microencapsulated)

(B) The investigational product 1: BMT® Coenzyme Q10 microencapsulated (Biostile, Ltd., Slovenia).

The researchers were blinded to characteristics of the formulations until the end of the study; samples were codified in origin and received by the researchers without any indication of their composition, including the already commercialized formula. The nature of the formulations was only revealed after final analysis of the data.

Study protocol

This was a randomized, double-blind, crossover bioavailability study conducted at a single centre. The study investigated both the single-dose bioavailability and 3-week bioavailability of two CoQ10 formulations across two treatment periods. The first treatment period explored the single-dose bioavailability over 48 h. The participants were randomly assigned one of the formulations (A or B). The second treatment period followed after a 2-week washout phase (Fig. 1). The protocol for the single dose bioavailability study over 48 h was the same as in the first treatment period, except that the participants were assigned the other formulation (B or A). After completion of the second 48-h post-acute phase, participants continued daily supplementation with the formulation assigned for the second treatment period (B or A) for additional three weeks. Randomization with stratification was performed using MinimPy, a free open-source desktop application (https://sourceforge.net/projects/minimpy) [22]. Stratification variables included sex and body mass to ensure balanced group assignment.

Since CoQ10 is a lipophilic compound, its absorption is known to be enhanced in the presence of dietary fat [10, 16, 20]. Therefore, standardized dietary conditions were applied in single-dose bioavailability studies to minimize variability in absorption. Following an overnight fast, participants received two capsules (2×50 mg) of the assigned test formulation at time zero, administered with a 2092 kJ (500 kcal) standardized breakfast,

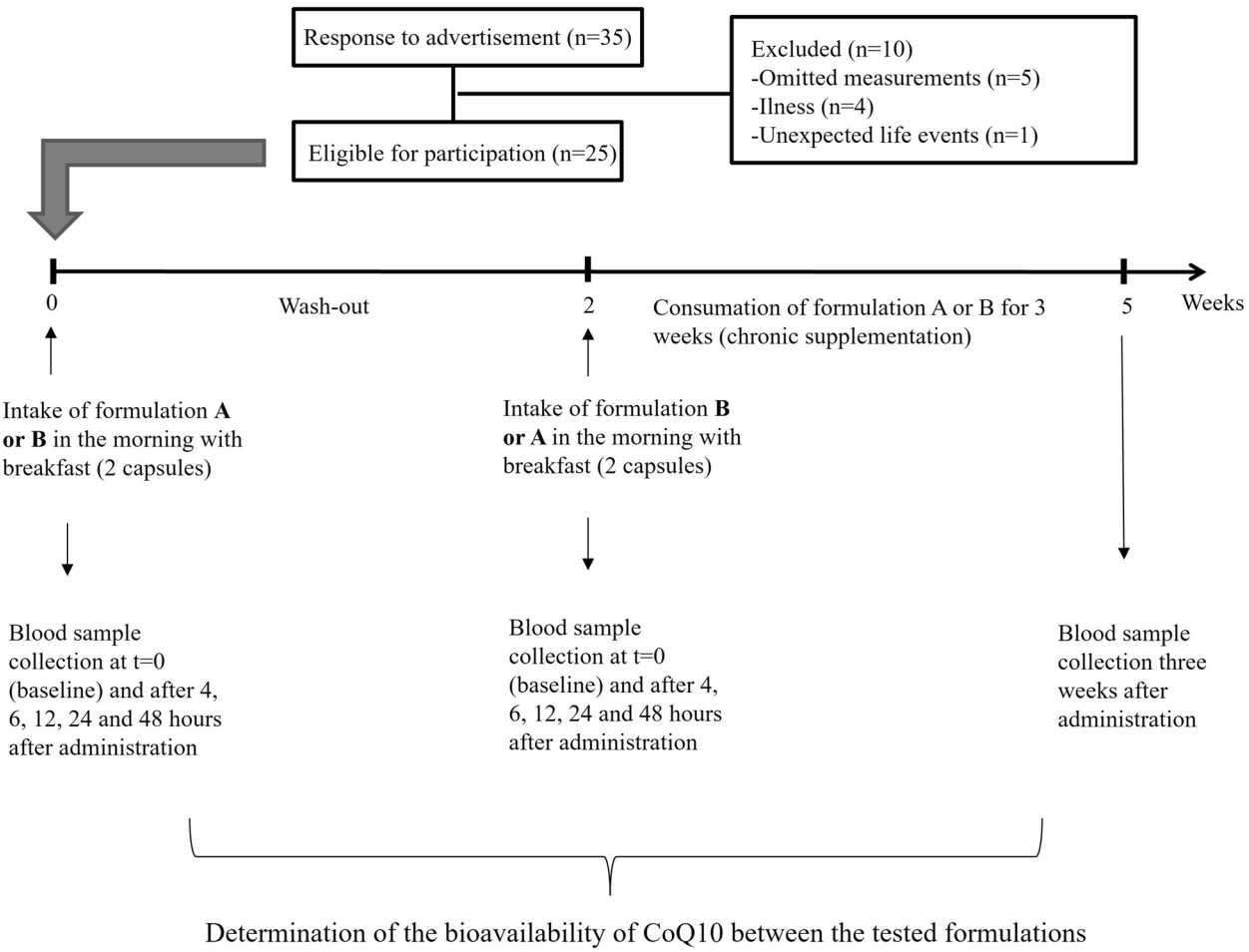

Fig. 1 Study design

composed of 150 mL of Greek-type yogurt (10% fat), 50 g of white bread, 5 g of honey and an apple. To ensure compliance, the single-dose administration was supervised on-site by the study coordinator during each treatment period. Blood samples were collected at baseline in fasting conditions (t = − 1) and at 4, 6, 12, 24, and 48 h after capsules ingestion during the first 48-h period. In the second treatment period, the participants ingested the other test formulation with the standardized breakfast described above and blood samples were collected at baseline in fasting conditions (t = − 1), and at 4, 6, 12, 24, and 48 h after capsules ingestion, with an additional sample taken in fasting conditions after three weeks of daily supplementation. The participants were asked to take the supplements (two 50 mg Q10 capsules) daily alongside breakfast, containing a source of fat. The participants were instructed to maintain their usual lifestyle and avoid major changes in physical activity throughout the study period. Adverse events were monitored throughout the study. At each visit, participants were interviewed to determine whether they had experienced any side effects since their last visit. Any changes in medication use or health status were also documented. All reported adverse events were reviewed and evaluated by the study investigators.

Blood analysis

Blood samples were collected according to standard procedures which are accessible via the website https://www.szkklm.si/assets/images/upload/PP_1_II__07_WEB.pdf (accessed on 10th January 2024). Blood samples (2 mL) were drawn into blood collection tubes containing heparin as anticoagulant and centrifuged at 1500 g for 15 min at 4 °C. The plasma was separated and transferred in aliquots into three separate vials (with a minimum of 200 µL of plasma in each). The vials were closed immediately after plasma transfer, kept frozen at − 80 °C until analysis. Plasma concentrations of fasting glucose, triacylglycerols, total cholesterol, LDL cholesterol, high density lipoprotein (HDL cholesterol), and C-reactive protein (CRP) were measured with a cobas® c 111 analyser (Roche), while vitamin D was analysed with the 25-OH-Vitamin-D ELISA EA300/96 kit as recommended by the manufacturer (DLD Diagnostica GmbH, Hamburg, Germany).

Analytical procedures

Coenzyme Q10 plasma levels were quantified chromatographically, by using Shiseido Nanospace HPLC (Shiseido Co. Ltd, Tokyo, Japan) equipped with an electrochemical detector (ECD) model 3005, pumps one and two model 3201, a flow-channel degasser model 3009, a switch valve model 3012, a column oven model 3014 and a refrigerated automatic autosampler model 3023 [23].

The system used 3 chromatographic columns: a Capcell Pack C18 MG concentrating column (2 mm I.D. × 35 mm, 5 µm), a Capcell Pack C18 AQ analytical column (2 mm I.D. × 150 mm, 3 µm), an OSAKA SODA CQ-R reducing column (2.0 mm I.D × 20 mm). Moreover, two mobile phases were used: the first one, composed of 50 mM sodium perchlorate in methanol/distilled water (95/5 v/v) to flux the sample through the concentrating column at a flow rate of 200 µL/min; the second phase, composed of 50 mM sodium perchlorate in methanol/isopropanol (95/5 v/v) to elute CoQ10 from the concentrating column to analytical one at a flow rate of 320 µL/min. The column oven was set at 40 °C and the oxidation potential for ECD was 650 mV. 50 µL of plasma were extracted with 250 µL 1-propanol and vortexed for 30 s. After centrifugation at 20,900×*g* for 2 min at 4 °C, 40 µL of supernatant was loaded in HPLC system. Ultrapure ubiquinone, kindly donated by Kaneka (Kobe, Japan), previously prepared in ethanol and stored at − 80 °C was used as external standard showing a $R^2 > 0.99$ in a range between 0.07 and 2.25 µg/mL.

The Limit of Detection (LOD) and Limit of Quantification (LOQ) were calculated following the ICH Q2(R2) guidelines, based on the standard deviation of the intercept (σ) and the slope (S) of the calibration curve. The values are as follows: LOD (3.3 σ/S) = 0.057 µg/mL and LOQ (10 σ/S) = 0.172 µg/mL. All experimental samples (minimum measured value = 0.31) were well above the LOQ, ensuring high analytical reliability and precision throughout the study.

Statistical analyses

Data are presented pooled for both sexes. The results are presented as the mean value with standard deviation (SD), as median (Q1, Q3) or frequencies (%). Since CoQ10 is transported in lipoproteins, particularly LDL, and its levels in plasma are often correlated with cholesterol levels, normalizing the values minimizes the variability caused by different cholesterol levels in individuals, providing a more accurate assessment of the body's CoQ10 status. However, in the present study CoQ10 quantification data are presented as µg/mL and normalized for cholesterol levels (expressed as nmol/mmol). Statistical analysis was conducted by using GraphPad Prism (Prism 8, GraphPad Software, San Diego, CA). In particular, we performed both a paired Student's t-test to compare AUC values and unpaired to compare formulation after chronic supplementation, while a Linear Mixed Model analysis was adopted in order to compare the time points of both formulations in acute treatment and to evaluate the time × treatment interaction with participants as a random intercept and

with adjustment for BMI. The linear mixed model analysis was performed using SPSS version 29.0 (IBM Corp., Armonk, NY, USA).

Effects of acute and chronic administration of CoQ10 on biochemical parameters and vitamin D plasma levels were evaluated with a linear mixed model, testing for the effect of time and the effect of intervention with participants as a random intercept and with adjustment for BMI. A p value ≤ 0.05 are considered statistically significant and P value ≤ 0.01 highly significant.

Results

Subjects' characteristics

A total of 25 participants (15 females and 10 males) completed both the single-dose and 3-week supplementation phases of the study. Demographic, anthropometric, and biochemical characteristics for the overall sample, as well as stratified by sex, are presented in Table 2. The average age of participants was 56 ± 6 years, with a mean body mass of 75.2 ± 9.2 kg and a mean BMI of 25.3 ± 2.8 kg/m^2. All participants were non-smokers. They were normoglycemic and exhibited normal to mildly elevated cholesterol levels. The mean ± SD plasma 25-hydroxyvitamin D [25(OH)D] concentration was 15.75 ± 2.91 ng/mL. The mean baseline CoQ10 plasma concentration was 0.59 ± 0.15 µg/mL and 130.06 ± 31.15 nmol CoQ10/mmol cholesterol, as already demonstrated in previous studies [24, 25].

All participants (both female and male) met the inclusion criteria for body mass. As expected, statistically significant sex-based differences were observed in several anthropometric parameters, including body mass, body fat percentage, fat-free mass, muscle mass, and visceral fat rating. Significant differences were also found in total and HDL cholesterol levels between females and males (Table 2).

Side effects

No adverse events were reported concerning the supplementation or study protocol. Moreover, no reports of medical problems or side effects were indicated.

Bioavailability of CoQ10 after single-dose or 3-week administration

The mean plasma concentration–time curves for CoQ10 and CoQ10 levels normalized to total cholesterol following single oral doses of formulations A and B are presented in Fig. 2A, B, respectively. As shown in Fig. 2C, D, paired Student's t-tests indicated that formulation B produced a significantly higher area under the concentration–time curve over 48 h (AUC_{48}) compared to formulation A. Specifically, the AUC_{48} for formulation B was 8729 ± 1493 (cholesterol-normalized), whereas

Table 2 Study population demographic data, anthropometric and biochemical parameters

Characteristcs	All participants N (%) or Mean ± SD	Females N (%) or Mean ± SD	Males N (%) or Mean ± SD	*p* value
Gender (F/M)	15/10 (60/40)	15 (60)	10 (40)	0.317
Age (years)	56.1 ± 6.3	55.7 ± 6.9	56.7 ± 5.4	0.699
Anthropometric parameters				
Body mass (kg)	75.2 ± 9.2	70.5 ± 6.9	82.9 ± 6.9	< 0.001
BMI (kg/m^2)	25.3 ± 2.8	24.7 ± 3.0	26.3 ± 2.4	0.175
Fat body mass (%)	28.1 ± 6.1	31.7 ± 4.1	22.0 ± 3.6	< 0.001
Fat free mass (kg)	54.2 ± 9.2	47.9 ± 3.6	64.6 ± 4.9	< 0.001
Muscle mass (kg)	51.5 ± 8.8	45.5 ± 3.5	61.4 ± 4.7	< 0.001
Visceral fat rating	8.2 ± 2.9	6.5 ± 1.6	11.0 ± 2.4	< 0.001
Biochemical parameters				
Glucose (mmol/L)	5.46 ± 0.52	5.49 ± 0.60	5.42 ± 0.41	0.756
Total cholesterol (mmol/L)	5.25 ± 0.71	5.46 ± 0.79	4.94 ± 0.45	0.047
LDL cholesterol (mmol/L)	3.66 ± 0.86	3.65 ± 1.05	3.67 ± 0.53	0.958
HDL cholesterol (mmol/L)	1.99 ± 0.57	2.28 ± 0.50	1.60 ± 0.39	0.001
CRP (mg/L)	1.13 ± 0.99	1.24 ± 1.01	0.95 ± 0.89	0.518
Vitamin D (ng/mL)	15.75 ± 2.91	14.48 ± 2.37	17.41 ± 6.11	0.663
Plasma CoQ10 (µg/mL)	0.59 ± 0.15	0.57 ± 0.15	0.62 ± 0.15	0.450
Plasma CoQ10/cholesterol (nmol/mmol)	130.06 ± 31.15	119.9 ± 21.3	145.3 ± 38.1	0.077

BMI, body mass index; CRP, C-reactive protein; F, female; M, male; N, number; SD, standard deviation

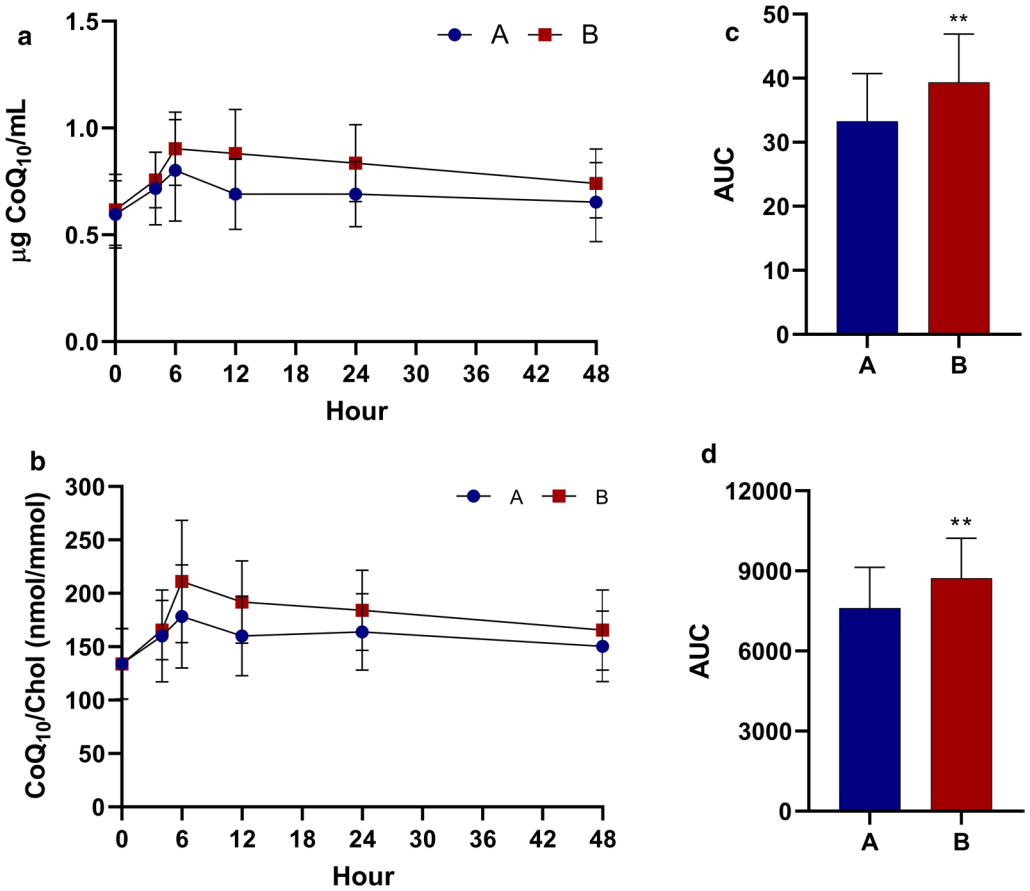

Fig. 2 Absorption curves of the kinetics of plasma CoQ10. Concentration expressed as mean plasma CoQ10 levels (**A**) and mean plasma CoQ10 levels normalized for cholesterol levels (**B**) and corresponding AUC_{48} values (**C**, **D**). CoQ10, coenzyme Q10. Blue line and blue bar—formulation A (CoQ10 USP (Ubiquinone; non-encapsulated)); Red line and red bar—formulation B (microencapsulated CoQ10). $^{**}p \leq 0.01$. Error bars indicate standard deviation

formulation A showed an AUC_{48} of 7606 ± 1525 ($p < 0.01$ for both comparisons).

The bioavailability of both CoQ10 formulations across all time points are presented in Fig. 3. Formulation B achieved significantly higher plasma CoQ10 concentrations—both absolute and cholesterol-normalized—relative to baseline as early as 6 h post-dose, with elevated levels sustained up to 24 h. In contrast, formulation A maintained significantly elevated levels only up to 6 h post-dose. At week 3, both formulations maintained significantly elevated CoQ10 levels compared to the 6-h post-dose measurement, but only when concentrations were normalized to total cholesterol (Fig. 3B). Without normalization, significantly higher levels at week 3 were observed only for formulation B (Fig. 3A).

Compared to formulation A, formulation B demonstrated significantly greater bioavailability at 6 h ($p < 0.05$), 12 h ($p < 0.01$), and 24 h ($p < 0.01$). When normalized for cholesterol, significantly higher levels were also observed at 48 h ($p < 0.01$). After 3 weeks of chronic supplementation, steady-state plasma CoQ10 concentrations achieved were higher in participants receiving formulation B compared to those receiving formulation A; however, this difference did not reach statistical significance whether the values were normalized for cholesterol or not, likely due to interindividual biological variability and the limited sample size (Fig. 3A, B).

Importantly, the model identified a significant time × treatment interaction ($p = 0.01$), indicating that the pharmacokinetic profile of formulation B was not only characterized by greater overall exposure (AUC48) and higher peak plasma concentration (c_{max}), but also by a longer duration of elevated plasma levels, as evident after 3 weeks of daily consumption. These findings suggest that formulation B offers superior pharmacokinetic performance compared to formulation A.

To better highlight inter-formulation differences while controlling for intra-individual variability, both the proportion of change and the percent changes in plasma CoQ10 levels from baseline were calculated

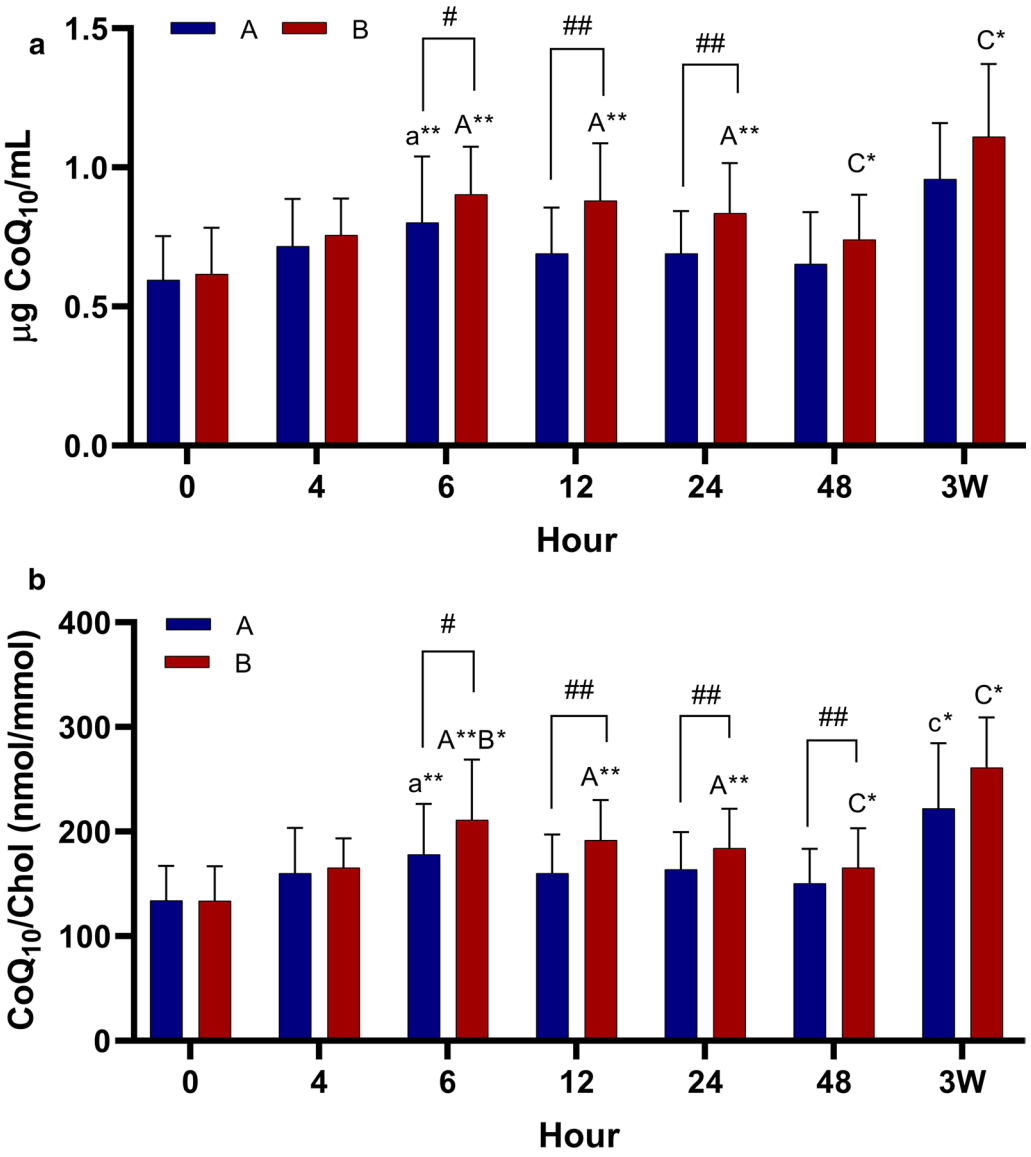

Fig. 3 Plasma CoQ10 levels (**A**) and plasma levels normalized for cholesterol levels (**B**) at individual time points and after 3 weeks of chronic supplementation. Blue bar—CoQ10 USP (Ubiquinone; non-encapsulated); Red bar—microencapsulated CoQ10. *$p \leq 0.05$; **$p \leq 0.01$: significance within the same formulation; #$p \leq 0.05$; ##$p \leq 0.01$: significance between formulations. Lowercase letters (*a–c*): significant differences in formulation A; a versus T0, c versus T6. Uppercase letters (*A–C*): significant differences in formulation B; A versus T0, B versus T4, C versus T6. Error bars indicate standard deviation

Table 3 Changes in plasma CoQ10 levels from baseline (in percent) for both formulations comparison

Time after supplementation	4 h	6 h	12 h	24 h	48 h	3 weeks
Formulation A	20%	36%	20%	24%	15%	83%
Formulation B	27%	63%	48%	46%	31%	94%
Formulation B/Formulation A	1.4	1.8*	2.4**	1.9*	2.1	1.1

* $p \leq 0.05$; ** $p \leq 0.01$

and analysed using paired t-tests. The results are presented in Tables 3 and 4. Formulation B resulted in significantly greater percent increases in plasma CoQ10 at 6-, 12-, and 24-h post-dose compared to formulation A ($p < 0.05$ at all-time points). Although the increase at 48 h did not reach statistical significance ($p = 0.061$), it remained notably higher for formulation B.

When normalized to cholesterol levels, the maximum percent increase was observed 6 h post-dose—37% for formulation A and 60% for formulation B. Overall, formulation B demonstrated significantly superior bioavailability at 12, 24, and 48 h, with an average fold increase of 2.3, 2.0, and 2.4, respectively, compared to formulation A. Following chronic supplementation, plasma CoQ10 concentrations increased by approximately 87% with formulation A and 96% with formulation B relative to baseline, while differences between formulations were not significant, and the ratio was 1.1.

Effects of single-dose and 3-week CoQ10 supplementation on biochemical parameters and vitamin D levels

No significant main effects of time or formulation were observed for key biochemical parameters—including fasting glucose, total cholesterol, LDL cholesterol, HDL cholesterol, and CRP—following either acute or chronic CoQ10 administration (Table 5). All biochemical parameters remained stable over time.

In contrast, a significant main effect of formulation was observed for plasma vitamin D levels. CoQ10 supplementation led to significantly higher vitamin D concentrations following both single-dose administration ($p = 0.004$) and 3-week chronic supplementation ($p = 0.042$) of formulation B.

Discussion

In this study, the bioavailability of two different commercially available formulations of CoQ10 was determined in a 48-h analysis after ingestion of 100 mg CoQ10 and

Table 4 Changes in plasma levels normalized for cholesterol from baseline (in percent) for both formulations comparison

Time after supplementation	4 h	6 h	12 h	24 h	48 h	3 weeks
Formulation A	20%	37%	21%	23%	12%	87%
Formulation B	27%	60%	47%	46%	29%	96%
Formulation B/Formulation A	1.4	1.6	2.3	2.0	2.4	1.1

$^{*}p \leq 0.05$; $^{**}p \leq 0.01$

Table 5 Effects of acute and chronic administration of CoQ10 on biochemical parameters and vitamin D plasma levels

Variables	Time	Formulation A	Formulation B	Significance	*p*
Biochemical parameters					
Fasting glucose (mmol/L)	Day 0	5.23 (4.87, 5.59)	5.20 (4.86, 5.55)	Formulation	0.500
	Day 21	5.33 (4.97, 5.69)	5.30 (4.96, 5.65)	Time	0.490
				Formulation × time	0.519
Total cholesterol (mmol/L)	Day 0	5.31 (4.91, 5.70)	5.20 (4.80, 5.60)	Formulation	0.715
	Day 21	5.49 (5.09, 5.89)	5.30 (4.90, 5.70)	Time	0.108
				Formulation × time	0.257
LDL cholesterol (mmol/L)	Day 0	3.43 (2.88, 3.98)	3.36 (2.84, 3.89)	Formulation	0.782
	Day 21	3.54 (2.99, 4.09)	3.51 (2.98, 4.03)	Time	0.548
				Formulation × time	0.735
HDL cholesterol (mmol/L)	Day 0	1.74 (1.41, 2.08)	1.70 (1.37, 2.02)	Formulation	0.840
	Day 21	1.76 (1.42, 2.10)	1.79 (1.47, 2.11)	Time	0.502
				Formulation × time	0.096
CRP (mg/L)	Day 0	1.36 (0.57, 2.14)	0.99 (0.21, 1.77)	Formulation	0.596
	Day 21	1.11 (0.33, 1.89)	1.21 (0.43, 1.99)	Time	0.768
				Formulation × time	0.109
Vitamin D (ng/mL)	Day 0	20.9 (14.2, 25.9)	11.8 (6.2, 17.4)	Formulation	0.042
	Day 21	17.6 (13.7, 23.5)	15.6 (9.7, 21.4)	Time	0.637
				Formulation × time	0.801

CRP, C-reactive protein; F, female; M, male; N, number; SD, standard deviation

after chronic supplementation of a daily dose of 100 mg CoQ10 for 3 weeks. Since there is significant inter-individual variability in absorption of ubiquinone from supplements [26], both formulations were evaluated by the same group of participants. Notably, the CoQ10 used in both formulations originated from the same source and manufacturer, allowing for a more controlled comparison of formulation-dependent differences.

Overall, our study demonstrated that microencapsulation significantly enhanced the bioavailability of CoQ10 compared to the standard unencapsulated formulation, as evidenced by significantly higher plasma CoQ10 concentrations following single-dose administration. These findings are consistent with previous reports showing improved absorption of CoQ10 from formulations designed to enhance solubility and dispersion [27–31]. Therefore, the present results should be interpreted as confirmatory within a controlled setting rather than as evidence of a fundamentally novel mechanism.

On the other hand, no significant differences in plasma CoQ10 levels across formulations were found after 3 weeks of continuous supplementation, probably due to the body's limited capacity to absorb CoQ10. Additionally, no significant safety concerns were revealed in the study, and no serious adverse events were observed. CoQ10 thus exhibited an acceptable safety profile as a dietary supplement up to daily dose of 100 mg for up to 3 weeks. However, given the limited sample size and short duration, no definitive conclusions regarding long-term safety can be drawn. In addition to enhanced bioavailability after acute administration, our study demonstrated that 3 weeks of microencapsulated CoQ10 supplementation significantly increased plasma vitamin D levels, suggesting a potential interaction between CoQ10 and vitamin D metabolism.

The low bioavailability of CoQ10 has been linked to its large molecular mass, high lipophilicity, and poor water solubility [9, 32]. Numerous endeavours have been undertaken to improve the poor bioavailability of crystalline CoQ10 in humans. Indeed, our results show that the bioavailability of microencapsulated CoQ10 is, on average, approximately 1.8–2.4 times higher than of the control in 48-h analyses after ingestion of 100 mg CoQ10, confirming microencapsulation as a suitable technology to optimize the bioavailability and absorption profile of CoQ10. Indeed, 75–92% of participants displayed significantly higher elevation in CoQ10 levels at 6, 12, 24 and even 48 h after ingestion of 100 mg microencapsulated CoQ10 compared to 100 mg control CoQ10. After 12 h, the mean increases in CoQ10 concentration were approximately 2.4-fold. Notably, substantial interindividual variability was observed: in most participants, levels increased 1- to threefold, while in roughly one-third of subjects, increases of up to 13-fold were recorded. In contrast, approximately 10% of participants exhibited a smaller-than-average rise. The present results are consistent with previous studies showing that CoQ10 is better absorbed from aqueous or emulsified vehicles than from powdered formulations [27, 28]. The higher CoQ10 levels in the bloodstream might be attributed to improved digestion and absorption due to the smaller emulsion size, as well as effective protection of encapsulated CoQ10 from adverse gastrointestinal conditions like pH variations or the presence of prooxidant substances. Indeed, an in vitro model showed that the rate of lipid digestion increased with decreasing droplet size, and an in vivo digestion model suggested that encapsulated CoQ10 in smaller, digestible droplets exhibited the highest bioavailability in small intestinal tissue [29]. In addition, one study showed that CoQ10-loaded emulsions had 1.7-fold higher bioavailability than crystalline CoQ10 [30]. In addition, microemulsions have been reported to enhance drug delivery and bioavailability because they are more easily transported across cell membranes and into plasma [31], which may explain the significantly higher increase in blood CoQ10 with microencapsulated CoQ10 compared with non-microencapsulated CoQ10. Moreover, microencapsulated CoQ10 was protected from the negative effects of other food components that could decrease CoQ10 availability by forming a complex with CoQ10, as has been reported for fatty acids [33].

We wanted to compare our results with previous studies on acute bioavailability of CoQ10. However, comparing our results with previous studies on CoQ10's acute bioavailability poses challenges due to variations in time periods and doses employed in the literature. Myoqinone displayed the most substantial elevation in plasma CoQ10 (around 1 mg/L) after a single dose of 100 mg [16, 34]. A comparatively smaller increase in plasma (around 0.40 mg/L for product B) in our study is consistent with other studies using a dose of 100 mg [35, 36], where the Δc_{max} value ranged from 0.025 to 0.50 mg/L, which is consistent with our results. A comparatively smaller increase in plasma might be explained by a higher baseline CoQ10 concentration as a negative correlation between baseline CoQ10 concentration and the change in plasma CoQ10 has been reported [37]. However, the average plasma CoQ10 levels in our population were around 0.6 mg/L, which is within a normal range from 0.4 to 1.8 mg/L [2, 38].

Regarding chronic supplementation, our study demonstrated a significant increase in plasma CoQ10 concentrations—reaching up to 1.0 µg/mL—with no significant differences observed between the two formulations tested. Similar findings were reported on the bioavailability of three CoQ10 formulations (ubiquinone

150 mg, ubiquinol 150 mg, and a liposomal ubiquinone) over a six-week period in 11 healthy volunteers [39]. Their results also showed no significant differences in plasma CoQ10 levels across formulations. The authors concluded that the intestinal absorption of CoQ10 is highly variable among individuals and largely independent of the specific form administered [39]. This is likely due to the body's limited capacity to absorb CoQ10 at any given time [40].

In addition, our study demonstrated that three weeks of microencapsulated CoQ10 supplementation significantly increased plasma vitamin D levels. This novel finding suggests a potential interaction between CoQ10 and vitamin D metabolism. One plausible explanation is the role of CoQ10 in enhancing mitochondrial function [2, 3], which may facilitate the energy-dependent hydroxylation steps required for vitamin D activation in the liver and kidneys. Additionally, CoQ10's potent antioxidant properties [6], may reduce oxidative stress in these tissues, potentially preserving vitamin D metabolites or enhancing their synthesis. This interpretation aligns with preclinical and clinical evidence suggesting synergistic effects of CoQ10 on vitamin D metabolism. For example, in an animal model of hypertensive rats, CoQ10 supplementation (10 mg/kg) alongside vitamin D improved renal antioxidant capacity and organ function [41]. Similarly, a randomized clinical trial in women with polycystic ovary syndrome found that CoQ10 alone or combined with vitamin E enhanced mitochondrial function, antioxidant status, and metabolic profiles—pathways closely linked to vitamin D hydroxylation and preservation [42]. Although both formulations increased plasma CoQ10 levels after three weeks of supplementation, no significant differences were observed between them at steady state. This raises the possibility that microencapsulated CoQ10 may have superior tissue uptake or distribution, even when plasma levels appear comparable. Enhanced solubility and stability of the microencapsulated formulation could facilitate more efficient cellular transport and mitochondrial incorporation, leading to greater biological effects—such as the observed rise in vitamin D—without necessarily producing higher circulating concentrations. However, the present finding should be interpreted with caution. Vitamin D status is influenced by multiple external factors, including dietary intake and sunlight exposure, which were not controlled in this study. In addition, the study was not designed to investigate interactions between CoQ10 and vitamin D metabolism. Therefore, no causal relationship can be established based on the present data. This observation should be considered exploratory and warrants further investigation in controlled studies specifically designed to assess this potential association.

We did not observe significant changes in metabolic parameters, including cholesterol or glucose levels following chronic CoQ10 supplementation; this result is consistent with prior studies in healthy populations where baseline metabolic parameters are typically within normal ranges [43]. The lack of effect may reflect a ceiling phenomenon, where improvements are unlikely without pre-existing dysregulation, or it could be attributed to an insufficient dosage. Previous research has shown that supplementing with 100 mg of CoQ10 twice daily can raise plasma CoQ10 levels from 0.90 to 3.25 μg/mL—levels considered necessary for therapeutic efficacy in cardiovascular disease [17, 34]. Indeed, numerous studies have demonstrated beneficial effects of CoQ10 on lipid profiles and glycaemic control in individuals with metabolic disorders, such as type 2 diabetes [13], coronary artery disease [14], and metabolic syndrome [11, 12]. In these populations, CoQ10 supplementation has been associated with reductions in LDL cholesterol, total cholesterol, and fasting glucose, along with improved insulin sensitivity. Therefore, while CoQ10 appears metabolically neutral in healthy individuals, it may have therapeutic potential in populations with underlying metabolic dysfunction.

Conclusion

Overall, our study demonstrated that microencapsulation significantly enhanced the bioavailability of CoQ10 compared to the standard unencapsulated formulation, as evidenced by higher plasma CoQ10 concentrations following single-dose administration. These findings are consistent with previous reports highlighting the poor solubility and absorption of native ubiquinone, and the utility of advanced delivery systems such as microencapsulation to overcome these limitations. The improved pharmacokinetic profile observed in our study supports the potential of microencapsulation to optimize CoQ10 delivery in healthy individuals. Importantly, both formulations were well tolerated, with no adverse effects reported throughout the intervention period, supporting their short-term safety. In addition to enhanced bioavailability, we also observed a significant increase in plasma vitamin D levels after three weeks of supplementation. This finding, although secondary, suggests a possible metabolic interaction between CoQ10 and vitamin D pathways, potentially mediated through improved mitochondrial function and reduced oxidative stress. Overall, our results confirm the safety and superior bioavailability of microencapsulated CoQ10 and provide early evidence for its potential role in modulating metabolic health beyond CoQ10 status alone. Further studies are needed to investigate long-term effects, clinical relevance, and underlying mechanisms.

Limitations of the study

Our study sample was relatively small and the findings should be confirmed in a larger and more diverse population. In addition, the intervention period was limited to three weeks, which may not fully reflect long-term pharmacokinetic behavior or sustained metabolic effects of CoQ10 supplementation. Another important limitation is that participants were healthy and had baseline plasma CoQ10 concentrations within the normal physiological range. Therefore, the potential benefits of supplementation may have been attenuated due to the absence of an initial deficiency. Future studies should evaluate bioavailability and clinical outcomes in individuals with lower baseline CoQ10 levels or documented deficiency, as this population may demonstrate a more pronounced response. Finally, mechanistic aspects of CoQ10 absorption and distribution were not directly investigated. These limitations should be considered when interpreting the findings.

Abbreviations

ATP	Adenosine triphosphate
BMI	Body mass index
CoQ10	Coenzyme Q10
CRP	C-reactive protein
HDL	High density lipoprotein
LDL	Low density lipopeotein
ROS	Reactive oxygen species

Acknowledgements

The authors thank the study participants for their participation, students of the Faculty of Health Sciences for their help in performing the studies and Biostile Ltd. for providing the food supplements. During the preparation of this manuscript, the authors used InstaText (https://instatext.io/) for editing purposes.

Author contributions

Ana Petelin (corresponding author) contributed to conceptualization, methodology, formal analysis, investigation, data curation, writing—original draft. Patrick Orlando (corresponding author) contributed to conceptualization, methodology, formal analysis, investigation, data curation, writing—original draft. Zala Jenko Pražnikar contributed to conceptualization, methodology, formal analysis, investigation, data curation, writing—original draft. Nina Mohorko contributed to methodology, investigation, data curation, writing—original draft. Marco Morosetti contributed to methodology, formal analysis, data curation, writing—original draft. Dejan Gmajner, Vasilij Valenčič and Vasja Valetič contributed to writing—original draft.

Funding

The study was supported by the Biostile, Ltd, Slovenian Research and Innovation Agency Slovenian Research Agency (Programme P1-0386 and Infrastructure project IO-0035).

Data availability

The data of this study are available from the corresponding author upon request.

Declarations

Ethics approval and consent to participate

The study was conducted in accordance with the Declaration of Helsinki, and approved by the by the National Medical Ethics Committee of Slovenia (No. 0120-557/2017/4; Ministry of Health, Republic of Slovenia) and registered on ClinicalTrials.gov (NCT06736366). Informed consent was obtained from all subjects involved in the study. Written informed consent has been obtained from the patient(s) to publish this paper.

Consent for publication

Not applicable.

Competing interests

The study was supported by Biostile Ltd, which provided the food supplements. Authors Dejan Gmajner, Vasilij Valenčič, and Vasja Valetič are employees of Biostile Ltd and contributed to the paper as researchers in the company, which produces food supplements. However, the company had no role in the design, execution, interpretation, or writing of the study. The remaining authors declare that the research was conducted without any commercial or financial relationships that could be construed as a potential conflict of interest. Therefore, there is no conflict of interest in relation to Biostile Ltd. The authors declare that this study received funding from Biostile Ltd. The funder was involved only in providing food supplements and was not involved in the study design, data collection, analysis, interpretation, writing of the article, or the decision to submit it for publication.

Author details

[1]Faculty of Health Sciences, University of Primorska, 6310 Izola, Slovenia. [2]Biostile Ltd, 6223 Komen, Slovenia. [3]Department of Life and Environmental Sciences, Polytechnic University of Marche, Marche, Italy.

Received: 20 February 2026 Accepted: 19 May 2026
Published online: 28 May 2026

References

1. Kalén A, Norling B, Appelkvist EL, Dallner G (1987) Ubiquinone biosynthesis by the microsomal fraction from rat liver. Biochim Biophys Acta 926:70–78. https://doi.org/10.1016/0304-4165(87)90183-8
2. Crane FL (2001) Biochemical functions of coenzyme Q10. J Am Coll Nutr 20:591–598. https://doi.org/10.1080/07315724.2001.10719063
3. Saini R (2011) Coenzyme Q10: the essential nutrient. J Pharm Bioallied Sci 3:466–467. https://doi.org/10.4103/0975-7406.84471
4. Mohr D, Bowry VW, Stocker R (1992) Dietary supplementation with coenzyme Q10 results in increased levels of ubiquinol-10 within circulating lipoproteins and increased resistance of human low-density lipoprotein to the initiation of lipid peroxidation. Biochim Biophys Acta 1126:247–254. https://doi.org/10.1016/0005-2760(92)90237-p
5. Stocker R, Bowry VW, Frei B (1991) Ubiquinol-10 protects human low density lipoprotein more efficiently against lipid peroxidation than does alpha-tocopherol. Proc Natl Acad Sci U S A 88:1646–1650. https://doi.org/10.1073/pnas.88.5.1646
6. Cirilli I, Damiani E, Dludla PV et al (2021) Role of coenzyme Q10 in health and disease: an update on the last 10 years (2010–2020). Antioxidants 10:1325. https://doi.org/10.3390/antiox10081325
7. Turunen M, Olsson J, Dallner G (2004) Metabolism and function of coenzyme Q. Biochim Biophys Acta 1660:171–199. https://doi.org/10.1016/j.bbamem.2003.11.012
8. Pravst I, Zmitek K, Zmitek J (2010) Coenzyme Q10 contents in foods and fortification strategies. Crit Rev Food Sci Nutr 50:269–280. https://doi.org/10.1080/10408390902773037
9. Bhagavan HN, Chopra RK (2006) Coenzyme Q10: absorption, tissue uptake, metabolism and pharmacokinetics. Free Radic Res 40:445–453. https://doi.org/10.1080/10715760600617843
10. Bhagavan HN, Chopra RK (2007) Plasma coenzyme Q10 response to oral ingestion of coenzyme Q10 formulations. Mitochondrion 7(Suppl):S78-88. https://doi.org/10.1016/j.mito.2007.03.003
11. Raygan F, Rezavandi Z, Dadkhah Tehrani S et al (2016) The effects of coenzyme Q10 administration on glucose homeostasis parameters, lipid profiles, biomarkers of inflammation and oxidative stress in patients with metabolic syndrome. Eur J Nutr 55:2357–2364. https://doi.org/10.1007/s00394-015-1042-7
12. Casagrande D, Waib PH, Jordão AA Júnior (2018) Mechanisms of action and effects of the administration of Coenzyme Q10 on metabolic syndrome. J Nutr Intermed Metab 13:26–32. https://doi.org/10.1016/j.jnim.2018.08.002

13. Liang Y, Zhao D, Ji Q et al (2022) Effects of coenzyme Q10 supplementation on glycemic control: a GRADE-assessed systematic review and dose-response meta-analysis of randomized controlled trials. EClinicalMedicine 52:101602. https://doi.org/10.1016/j.eclinm.2022.101602
14. Lee B-J, Huang Y-C, Chen S-J, Lin P-T (2012) Effects of coenzyme Q10 supplementation on inflammatory markers (high-sensitivity C-reactive protein, interleukin-6, and homocysteine) in patients with coronary artery disease. Nutr Burbank Los Angeles County Calif 28:767–772. https://doi.org/10.1016/j.nut.2011.11.008
15. Lee Y-J, Cho W-J, Kim J-K, Lee D-C (2011) Effects of coenzyme Q_{10} on arterial stiffness, metabolic parameters, and fatigue in obese subjects: a double-blind randomized controlled study. J Med Food 14:386–390. https://doi.org/10.1089/jmf.2010.1202
16. López-Lluch G, Del Pozo-Cruz J, Sánchez-Cuesta A et al (2019) Bioavailability of coenzyme Q10 supplements depends on carrier lipids and solubilization. Nutrition 57:133–140. https://doi.org/10.1016/j.nut.2018.05.020
17. Mantle D, Dybring A (2020) Bioavailability of coenzyme Q10: an overview of the absorption process and subsequent metabolism. Antioxid Basel 9:386. https://doi.org/10.3390/antiox9050386
18. Jones M, Walker D, Ionescu CM et al (2020) Microencapsulation of Coenzyme Q10 and bile acids using ionic gelation vibrational jet flow technology for oral delivery. Ther Deliv 11:791–805. https://doi.org/10.4155/tde-2020-0082
19. Constantinescu R, McDermott MP, Dicenzo R et al (2007) A randomized study of the bioavailability of different formulations of coenzyme Q(10) (ubiquinone). J Clin Pharmacol 47:1580–1586. https://doi.org/10.1177/0091270007307571
20. Pravst I, Rodríguez Aguilera JC, Cortes Rodriguez AB et al (2020) Comparative bioavailability of different coenzyme Q10 formulations in healthy elderly individuals. Nutrients 12:784–784. https://doi.org/10.3390/nu12030784
21. Gopi S, Balakrishnan P (2021) Evaluation and clinical comparison studies on liposomal and non-liposomal ascorbic acid (vitamin C) and their enhanced bioavailability. J Liposome Res 31:356–364. https://doi.org/10.1080/08982104.2020.1820521
22. Saghaei M, Saghaei S (2011) Implementation of an open-source customizable minimization program for allocation of patients to parallel groups in clinical trials. J Biomed Sci Eng 04:734–739. https://doi.org/10.4236/jbise.2011.411090
23. Orlando P, Silvestri S, Galeazzi R et al (2018) Effect of ubiquinol supplementation on biochemical and oxidative stress indexes after intense exercise in young athletes. Redox Rep 23:136–145. https://doi.org/10.1080/13510002.2018.1472924
24. Orlando P, Sabbatinelli J, Silvestri S et al (2020) Ubiquinol supplementation in elderly patients undergoing aortic valve replacement: biochemical and clinical aspects. Aging 12:15514–15531. https://doi.org/10.18632/aging.103742
25. Sabbatinelli J, Orlando P, Galeazzi R et al (2020) Ubiquinol ameliorates endothelial dysfunction in subjects with mild-to-moderate dyslipidemia: a randomized clinical trial. Nutrients 12:1098. https://doi.org/10.3390/nu12041098
26. Kurowska EM, Dresser G, Deutsch L et al (2003) Relative bioavailability and antioxidant potential of two coenzyme Q10 preparations. Ann Nutr Metab 47:16–21. https://doi.org/10.1159/000068910
27. Miles MV, Horn P, Miles L et al (2002) Bioequivalence of coenzyme Q10 from over-the-counter supplements. Nutr Res 22:919–929. https://doi.org/10.1016/S0271-5317(02)00402-5
28. Schulz C, Obermüller-Jevic UC, Hasselwander O et al (2006) Comparison of the relative bioavailability of different coenzyme Q10 formulations with a novel solubilizate (Solu™ Q10). Int J Food Sci Nutr 57:546–555. https://doi.org/10.1080/09637480601058320
29. Cho HT, Salvia-Trujillo L, Kim J et al (2014) Droplet size and composition of nutraceutical nanoemulsions influences bioavailability of long chain fatty acids and coenzyme Q10. Food Chem 156:117–122. https://doi.org/10.1016/j.foodchem.2014.01.084
30. Hatanaka J, Kimura Y, Lai-Fu Z et al (2008) Physicochemical and pharmacokinetic characterization of water-soluble coenzyme Q(10) formulations. Int J Pharm 363:112–117. https://doi.org/10.1016/j.ijpharm.2008.07.019
31. Huang Q, Yu H, Ru Q (2010) Bioavailability and delivery of nutraceuticals using nanotechnology. J Food Sci 75:R50-57. https://doi.org/10.1111/j.1750-3841.2009.01457.x
32. Zaki NM (2016) Strategies for oral delivery and mitochondrial targeting of CoQ10. Drug Deliv 23:1868–1881. https://doi.org/10.3109/10717544.2014.993747
33. Bremmell KE, Briskey D, Meola TR et al (2020) A self-emulsifying Omega-3 ethyl ester formulation (AquaCelle) significantly improves eicosapentaenoic and docosahexaenoic acid bioavailability in healthy adults. Eur J Nutr 59:2729–2737. https://doi.org/10.1007/s00394-019-02118-x
34. Weis M, Mortensen SA, Rassing MR et al (1994) Bioavailability of four oral coenzyme Q10 formulations in healthy volunteers. Mol Aspects Med 15(Suppl):s273-280. https://doi.org/10.1016/0098-2997(94)90038-8
35. Wajda R, Zirkel J, Schaffer T (2007) Increase of bioavailability of coenzyme Q(10) and vitamin E. J Med Food 10:731–734. https://doi.org/10.1089/jmf.2006.254
36. Young JM, Molyneux SL, Florkowski CM et al (2012) Pharmacokinetic comparison of a generic coenzyme Q10 solubilizate and a formulation with soybean phytosterols. Phytother Res 26:1092–1096. https://doi.org/10.1002/ptr.3667
37. Nylander M, Weiner J, Ruokonen I, Laakso J (1995) Plasma levels of coenzyme Q10 before and after oral supplementation: a bioavailability study. CoQ Res Biol Med 3:25–32
38. Redalieu E, Nilsson IM, Farley TM et al (1968) Determination and levels of coenzyme Q10 in human blood. Anal Biochem 23:132–140. https://doi.org/10.1016/0003-2697(68)90018-3
39. Vitetta L, Leong A, Zhou J et al (2018) The plasma bioavailability of coenzyme Q10 absorbed from the gut and the oral mucosa. J Funct Biomater 9:73. https://doi.org/10.3390/jfb9040073
40. Singh RB, Niaz MA, Kumar A et al (2005) Effect on absorption and oxidative stress of different oral coenzyme Q10 dosages and intake strategy in healthy men. BioFactors 25:219–224. https://doi.org/10.1002/biof.5520250127
41. Shamardl HA, El-Ashmony SM, Kamel HF, Fatani SH (2017) Potential cardiovascular and renal protective effects of Vitamin D and coenzyme Q10 in l-NAME-induced hypertensive rats. Am J Med Sci 354:190–198. https://doi.org/10.1016/j.amjms.2017.04.007
42. Izadi A, Ebrahimi S, Shirazi S et al (2019) Hormonal and metabolic effects of Coenzyme Q10 and/or Vitamin E in patients with polycystic ovary syndrome. J Clin Endocrinol Metab 104:319–327. https://doi.org/10.1210/jc.2018-01221
43. Hosoe K, Kitano M, Kishida H et al (2007) Study on safety and bioavailability of ubiquinol (Kaneka QH) after single and 4-week multiple oral administration to healthy volunteers. Regul Toxicol Pharmacol 47:19–28. https://doi.org/10.1016/j.yrtph.2006.07.001

Publisher's Note