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The Impact of Oral CoQ10 Supplementation on Peripheral Blood Lipid Profiles and Muscular Damage Indices Following Two Weeks Intense Aerobic Training in Elite Cyclists

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Abstract

The regular exercise training is so effective for improvement of health. However, there is evidence that intense aerobic training may cause damage to the tissues with different ways. Scientists of sport science are used various supplements for prevention this deleterious effects which CoQ10 (coenzymeQ10) is one of these supplements. This study was designed to investigate the effects of short-term CoQ10 supplementation on serum lipid profile and indirect muscle damage indices levels subsequence of one session intense aerobic training in cyclists. The research studied on healthy elite men cyclists to assess the efficacy of short-term CoQ10 supplementation. Cyclists were divided into 2 groups (each group 7; age range 19-25 years) to receive CoQ10 and dextrose. Blood samples were collected during 2 weeks of prepare camp, 2 d before the training camp (baseline), 18-24 hrs after supplementation and training session of the first and last training session and in order to assess serum levels of triglycerides high-density lipoprotein has been used autoanalyzer. Also, aforementioned method was used to calculate Creatine kinase and lactate dehydrogenase activity. Fourteen cyclists completed the course of the study. Serum lipid levels in the consumer CoQ10 compare placebo group decreased following one session aerobic training ($P<.05$). However, baseline lipid levels were unchanged ($P>.05$) except triglyceride which had descending procedure (from 72.7±17.9 to 51.9±11.9) in CoQ10 group ($P<.001$). Ascending response of indirect muscle damage indices (Creatine kinase and lactate dehydrogenase) and capillary lactate decreased in the CoQ10 group compare to placebo group (301±45.11 versus 340.42±36.41, 362.71±32.58 versus 407.14±60.9 and 1.04±0.24 versus 1.24±0.19 for every index, respectively) in subsequent last session of aerobic training ($P<.05$). The present study showed that short-term supplementation with CoQ10 could insignificantly reduce serum levels of some lipid profiles and significantly reduce muscle damage in healthy elite cyclists.

1. Introduction

CoQ10 is a vitamin-like fat-soluble substance benzoquinone compound and synthesized in the human body as a part of the cholesterol pathway naturally (1, 2). It is intimately involved in several important roles in the body including the transferring of electrons within the mitochondria oxidative respiratory chain, ATP production, in reduced form (ubiquinol) acting as an antioxidant, influencing the stability, fluidity and permeability of membranes and stimulating cell growth and inhibiting cell death (2-4). Aerobic exercise through positive changes in lipid and lipoprotein profiles is one of the most useful effective agent in the prevention of coronary artery risk factors (5). Also, exercise decreases the harmful lipids levels such as triglycerides and lipoprotein and by increasing the high-density lipoprotein induces improvement in living standards and life expectancy (6). However, intense aerobic exercise such as cycling with place various tissues under a metabolic and mechanical stress and increase oxygen consumption and mitochondrial respiration for increase ATP resynthesize that generated the greater disturbance of metabolic homeostasis in the muscle fiber during more intense exercise (7). Failure of ATP production subsequence to increase in the exercise lead to ATP production from anaerobic sources, which causes accumulation of metabolites (e.g. lactate, hydrogen ions, ammonia, free radical species) and decrease in blood lipids concentration (8, 9). Metabolic stress subsequently causes reduction in available energy and degradation of performance, increase in permeability of cell membranes and releases of indirect cellular damage indices into the peripheral blood circulation (10). In addition, some study has shown, in athletes CoQ10 (ubiquinone) deficiency may lead to experienced metabolic stress and increased production of free radicals during intense training (3). In recent years, CoQ10 (2,3-dimethoxy-5-methyl-6-decaprenyl-1,4-benzoquinone) as an efficacy dietary supplementation in cellular bioenergy and inhibitor in some of damages induced by energy depletion and free radicals during intense aerobic exercise has been taken into consideration by athletes and coaches (3, 11-13). According to previous investigations that have reported endurance athletes may during periods of intense training consequent to high levels of oxidative metabolic stress that may incur low plasma CoQ10 levels in athletes and subsequent increased blood metabolites levels (14). Also, the lack of cohesive sport studies on the effect CoQ10 on improvement of blood lipid level, muscle damage and lactate, and majority of studies have been on disease populations and animals (15-20) were the reasons of present study. So, the aim of present study was to investigate the impact of 14 days CoQ10 supplementation on lipid profiles and muscle damage subsequent two weeks intense aerobic training.

2. Materials & Method

The participants in this study included fourteen professional male cyclists with age

20.1±2 years old. In order to ensure the homogeneity of the group, two weeks prior to preparation camp the subjects were subjected to anthropometric (percent fat= 5.7±0.6), Astrand and Rhyming Cycle Ergometer Tests (VO2max= 66.7±3.7 ml.kg-1.min) (21). All subjects were informed about the purpose, potential risks and benefits associated with the study and before this stage; it was obtained written informed consent. They also were provided health questionnaire and 24-hours dietary records for participation. Then, the subjects were paired according to their VO2max and age and randomly allocated to dextrose placebo or CoQ10 supplementation groups. Approval of the experimental procedure was obtained from the Ethics Committee on Human Experimentation of Tabriz University of Medical Sciences.

In addition to daily diet control of subjects during the study (using 24 hrs dietary record, table1), also, the first (first day) and last meal (last day) of both groups were the same. This was monitored by having each subject document dietary intake for 2 weeks before each training session. In addition, each subject was instructed not to perform any physical activity for 20 hours preceding each training session. One day prior to starting the training (camp workouts), subjects donated approximately 6 ml of fasting blood. Then, the subjects consumed either the placebo or CoQ10 supplement. One hour following ingestion of the supplement, the subjects performed training session for 2 to 2.5 hours. approximately 18-24 hours in the first day and last day of training camp, the additional blood samples were taken during the training camp in order to essaying the acute and chronic supplementation effects.

Table1. Daily calories intake during 2 weeks.

Carbohydrate	Fat	Protein
65%	25%	10%
3918 Kcal	1359 Kcal	471 Kcal

2.1 Supplementation protocol

All subjects were randomly assigned to either a CoQ10 supplementation group (n =7) or a placebo supplementation group (n=7) in double-blind manner. Subjects in the CoQ10 group took 5 mg CoQ10 (license number of health 302011061035 Flop) (22). Supplementation included 5 mg per day for 14 days in the morning and after having the breakfast and during the training camp. Subjects in the placebo group consumed the same rate capsules per day for the same duration. Also, all subjects ate the same diet during the training camp.

2.2 Training program

Table 2 shows the exercise protocol used in this study. Training sessions were initiated at the morning after supplementation and having breakfast. All subjects participated in the preparation program for 14 days [including 11 training session of cycling approximately 1-3 hrs with 65-95% maximal aerobic capacity (heart rate reserve; manner: Karvonen) or the rating of perceived exertion (23) 15-19 according to 11-14

year]. Indeed, it should be regarded that the first and last training session in order to assaying cycling training-induced response was held competitive up to exhaustion. While, the rest training sessions mostly was held in the form normal training program in the prepare camps of elite time trial cycling. In addition, 48 hours before the first and last day of supplementation, athletes were asked to completely relax. Subjects were allowed to consume water during the exercise as needed. Also, intensity of training increased with closing to the end of the camp and rating of perceived exertion (23) was subjectively obtained by Borg scale in the end of the each training session (24, 25). It is worthy to note that the start time of every training session was in the morning.

2.3 Blood sampling

In this stage, 6 ml venous blood sampling was obtained from each athlete's forearm in a resting condition between 7.30 and 8.30 hours of morning: 2 days before the training camp (baseline), 18 to 24 hrs after first and last training sessions (first day and 14th day of training camp). Immediately after collection, 1 mL of blood was sent for hemoglobin and hematocrit analysis and 5 mL were dispensed into tubes and Serum was separated from blood cells by centrifugation (3000rpm for 10 min) and stored at -30℃ until analysis. Serum volume was adjusted according to Dill and Costill's equation (26).

2.4 Clinical chemistry analysis

The blood samples were assessed for serum levels of triglycerides, high-density lipoprotein by using autoanalyzer BT 3000 (enzymatic-photometric method) and total cholesterol (enzymatic-colorimetric CHOP-PAP method). The low-density lipoprotein- cholesterol was calculated that is described by Friedewald et al (27). Creatine kinase and lactate dehydrogenase activity in the serum was measured by using autoanalyzer BT 3000 (enzymatic-photometric DGKS method) and blood samples of the subject's finger were analyzing due to an automated lactate analyzer (Accutrend, Boehringer Mannheim, Germany). All these biochemical assays were performed by using diagnostic kits that is made by Pars Azmun Company (Tehran, Iran). Hemoglobin and hematocrit levels were determined by automatic blood analyzer (Technicon H1, Technicon, Tarrytown, NY, USA).

Table 2. The exercise protocol during 2 weeks.

First week				
Day	Total time of training session	Warm-up	Main set	Cool-down
Saturday	80 min	10 min: 80-100 rpm, RPE; 13-15	Exhaustive competition test for one hour	10 min: 65-75 rpm, RPE 10-12
Monday	180 min	10 min: 80-100 rpm, RPE; 13-15	Easy road riding	10 min: 65-75 rpm, RPE 10-12
Tuesday	90 min	10 min: 80-100 rpm, RPE; 13-15	Interval Leg pressure training	10 min: 65-75 rpm, RPE 10-12
Wednesday	60 min	10 min: 80-100 rpm, RPE; 13-15	1- Alternative training 2- continues training	10 min: 65-75 rpm, RPE 10-12
Thursday	Rest	Rest	Rest	Rest
Friday	180 min	10 min: 80-100 rpm, RPE; 13-15	Road riding training	10 min: 65-75 rpm, RPE 10-12
Sunday	90 min	10 min: 80-100 rpm, RPE; 13-15	Interval Leg pressure training	10 min: 65-75 rpm, RPE 10-12

Second week				
Day	Total time of training session	Warm-up	Main set	Cool-down
Sunday	180 min	10 min: 80-100 rpm, RPE 13-15	Continues road training	10 min: 65-75 rpm, RPE 10-12
Monday	Rest	Rest	Rest	Rest
Tuesday	90 min	10 min: 80-100 rpm, RPE 13-15	1- Interval training 2- Continues riding training 3- Interval training	10 min: 65-75 rpm, RPE 10-12
Wednesday	180 min	10 min: 80-100 rpm, RPE 13-15	Continues road riding training	10 min: 65-75 rpm, RPE 10-12
Thursday	60 min	10 min: 80-100 rpm, RPE 13-15	1- Interval training 2- continues road riding training	10 min: 65-75 rpm, RPE 10-12
Friday	Rest	Rest	Rest	Rest
Saturday	80 min	10 min: 80-100 rpm, RPE 13-15	Exhaustive competition test with duration one hour	10 min: 65-75 rpm, RPE 10-12

2.5 Statistical analysis

Data are expressed as mean ± SD. Before performing the statistical analysis, the Kolmogorov-Smirnov and t- independent test was used to assess normal distribution and homogeneity of independent and dependent variables. Then, changes in variables during the three stages were analyzed by two-way ANOVA (group by time) with repeated measurement (2×3) and Bonferroni post hoc analysis. Also, differences between means of groups were identified by using the t- independent test. Statistical significances were set at P <.05. All data were analyzed using the Stat Tools software's for Web and SPSS/PASW version 18.0 (SPSS, Chicago, USA).

3. Results

First of all, it must be mention that Blood volume changes were calculated from changes in (3) and Hct. The hematocrit and hemoglobin concentration remained stable. Decreasing and increasing of 4.56 and 2.45 percent of blood volume in the groups of CoQ10 and placebo supplement after performing of exercise protocol were not significant (P >.05). Age and physiological characteristics of the subjects have showed in the table 3. Results of present research indicated that there are no significant differences in biochemical indices and morphologic characteristics of two groups in baseline and after initiation of exercise protocol (acute effect) (P >.05).

There were no significant differences in physical activity and dietary intake of the studied subjects during the study (data are not shown). However, response of lipid profiles after training camp were so different among them, such that CoQ10 supplementation induced decreases of baseline triglyceride and increases in descending trend of serum triglyceride subsequent to last session of training (14^{th} day) (P <.05). While, significant effect on baseline total cholesterol (TC), high-density lipoprotein cholesterol (HDL-C), total cholesterol/HDL-C and low-density lipoprotein cholesterol (LDL-C) did not observed (P >.05). However, decreased serum levels of lipids subsequence of chronic aerobic training session (last session training) was shown (no significant) after supplementation and between the studied groups (P >.05). In both the CoQ10 and placebo groups, serum Creatine kinase and lactate dehydrogenase (CK and LDH) activity

significantly increased at 18-24 hours compare with baseline (P<.01). The percent increment changes in serum CK and LDH activity in the CoQ10 group were significantly lower than placebo group at 14th day of training camp. This result suggested that CoQ10 supplementation reduced exercise-induced releasing of mechanical-metabolical markers in athletes. However, in the two groups capillary lactate significantly increased (exercise-induced response) and (at last day) (P<.05). In contrast, incremental changes of lactate concentration in the CoQ10 group was lowered (data has not shown) (P<.05).

4. Discussion

In this study, we examined the influence of short-term CoQ10 supplementation on aerobic training-induced response of some peripheral blood biochemical and lipid profiles in male cyclists. The study revealed that short-term of coenzymeQ10 is effective supplement reducing the lipid profiles especially in reducing the triglycerides concentrations. This finding is important because it indicates usefulness of CoQ10 supplementation for reducing the cardiovascular risk factors. Triglyceride, TC, HDL, TC/HDL and LDL has been the most commonly used as markers of cardiovascular risk factor. In the present study, serum lipid profiles of placebo group compare to CoQ10 group was unchanged during the training camp (see Fig1).

Also, serum baseline triglyceride concentration and subsequent aerobic training significantly decreased during training camp in the CoQ10 group. However, the present study indicated that cycling training camp causes no significant changes in these risk factors. Some previous literatures suggest a possible therapeutic role for coenzymeQ10 in human disease such as mtDNA induced deafness and Parkinson', however, the potential effect of coenzymeQ10 on lipid profiles and cardiovascular risk factors in trained and untrained individuals is less clear. In a sense, the reduction in lipid levels and increase in HDL levels may be due to inhibition of LDL oxidation and reduced oxidative stress (28). However, these findings are in line with a number of previous studies. Similar to this finding, Kaikkonen and et al (1998) reported that coenzyme Q10 (90 mg.kg) supplementation in combine with α-tocopheryl acetate (13.5 mg.kg)

caused a decrease in the serum TG and LDL in marathon runners (23). Also, Cooke and et al (2008) investigated the effect of 14 days of CoQ10 supplementation twice per day and exercise on lipid profiles of aerobically and untrained subjects. Results showed no significant changes in lipid profiles in response to supplementation (3). In addition, Shojaei and et al (2011) in a clinical study on hemodialysis patients showed that three months of treatment with Co Q10 induced no significant decrease in serum triglycerides and other cardiovascular risk factors in these patients (29). Modi and et al and Cicero and et al (2006-2005), in contrast to our findings, investigated the effect of four and six weeks of CoQ10 supplementation on diabetic mouse and high baseline triglycerides patients, respectively. Results of these researches were showed significant decrease in serum and plasma cholesterol, respectively (30, 31). Exertion or inexertion of training protocol, type or health status of studied subjects and different in the type of supplementation protocol (rate of daily consumption, time of supplementation period and type of used supplement) may explain this discrepancy. However, serum CK and LDH activity have been commonly known mechanical-metabolic markers and indirect of skeletal muscle damage. CK and LDH increased after training camp in the two groups significantly in the present study. The increases of serum CK and LDH indicated that cycling training camp imposes mechanic-metabolic on active tissues. In this regard, rate of increases of these markers in the supplementation group compare to the placebo group has been lowered significantly (see figures 2). Other studies have investigated the effect of CoQ10 (ubiquinone) supplementation on exercise-induced indirect muscle damage in both human subjects (athletes or disease) and rats. In the previous study by Kon et al (2007-8) have showed that intake of CoQ10 after kendo training camp for 2 weeks and 4 weeks in the rats caused decreased muscle damage marker (12, 32). Also, Shimomura et al (1991) have reported that intravenous CoQ10 supplementation diminished increased muscle damage markers (Creatine kinase: CK and lactate dehydrogenase: LDH) in rats following downhill running (33).

Table3. Anthropometric and physiological characteristics of subjects (TN = 14).

Variable		Age (year)	Stature(height)	Weiht (kg)	Body fat (%)	BMI $(Kg/m)^2$	VO_2max (ml/kg/min)	Panaerbic cap (W)	AVanaerbic cap (W)
CoQ10	Mean	19.9	171.5	63.5	5.6	21.6	66.6	932.1	768.6
	SD	1.57	2.31	3.58	0.5	1.51	3.18	52.31	31.54
Placebo	Mean	20.4	175.5	63.7	5.8	20.7	67.8	906.2	775.5
	SD	2.51	3.92	4.77	0.69	1.16	4.25	57.1	54.24
	P value	0.3	0.9	0.7	0.9	0.7	0.8	0.7	0.9

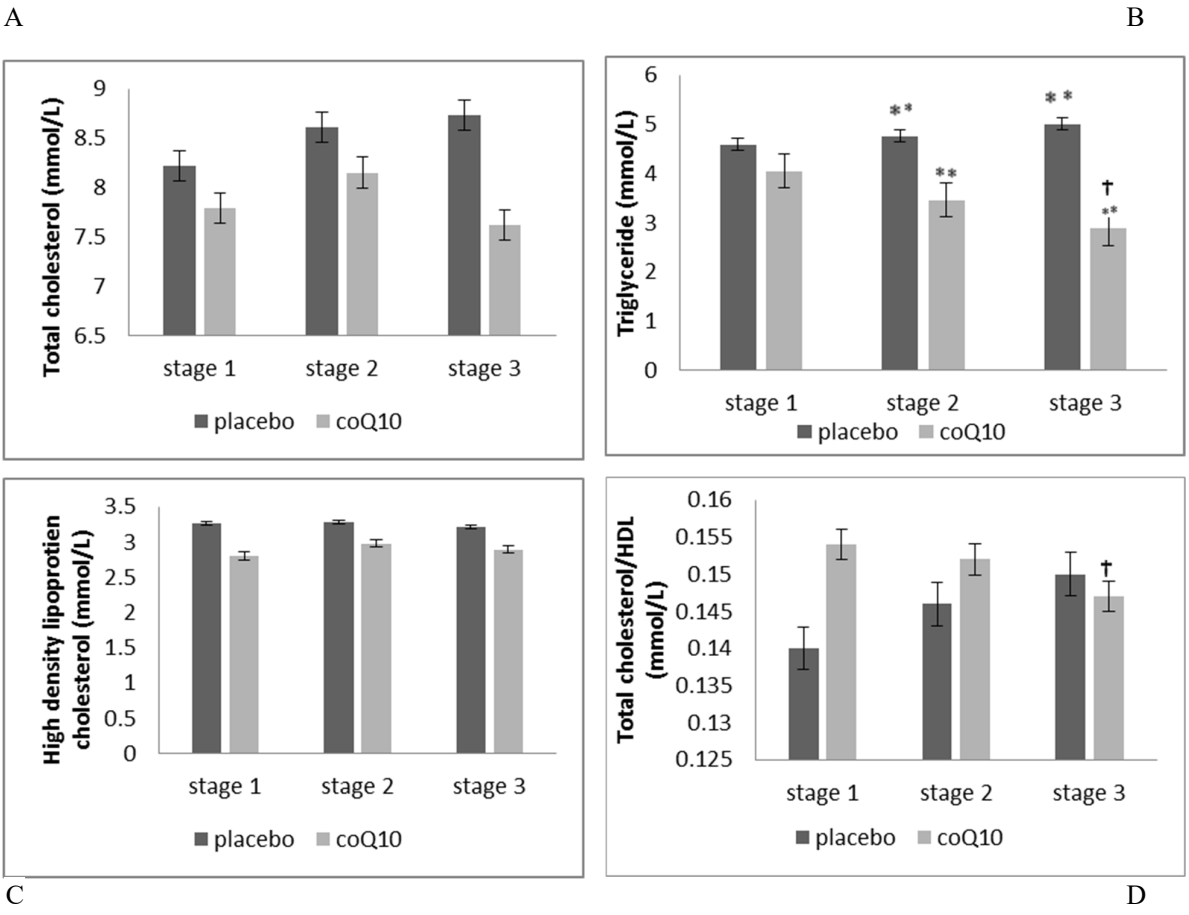

Figure1. Serum lipid profiles 2 d before the training camp (baseline), 18-24 hrs after supplementation and training session of the first and last training session. At the fig A *Mean values were not significantly different from baseline. Also, mean values were not significantly different Between CoQ10 and placebo groups. Fig B shows that Mean values were significantly different from baseline: **P<0.01. Mean values were significantly different between CoQ10 and placebo groups*: †P<0.05. However, fig C suggests not significant differences between the groups in the Serum high-density lipoprotein cholesterol. Finally, *Mean values were significantly different between CoQ10 and placebo groups: †P<0.05 (fig D)*

In addition, Okamoto et al (1995) have showed that CoQ10 protected cultured skeletal muscle cells from electrical stimulation-induced LDH release (34). In based on previous studies, it has been reported that CoQ10 had a structural stabilizing effect on cell membrane phospholipids (12, 32). Oral supplementation with CoQ10 significantly increases the CoQ10 count of cell membranes and then may reduce exercise-induced indirect muscle damage markers by raising CoQ10 concentration in muscle cell membranes and stabilizing the cell membrane. Conversely, Skough et al (2008) by intake simultaneously of CoQ10 and resistance training for 12 weeks reported no significant effect on the CK in Post-Polio syndrome patients (22). In this regard, it must be mentioned that mechanism and template of total serum CK and LDH variations different subsequent of aerobic and resistance exercises. So that, increases of total serum CK subsequence of the resistant exercises can be defined as the disruption of plasma membrane accompanied by the loss of muscle proteins (i.e. Creatine kinase (CK), lactate dehydrogenase (LDH)), the influx of serum proteins, increased population of inflammatory infiltrates in the muscle fibers such as macrophages and neutrophils, DOMS, functional impairment, and possible structural disorders such as sarcomere Z lines disarrangement; while increases of concentration this enzyme in the serum subsequent aerobic and endurance exercises often result in changes in ion concentration, accumulation of metabolic wastes and deficiency of adenosine triphosphate (ATP). In addition, it cannot be ignored the heterogenic and studied small value samples on data analysis and conclusion in Skough research group. The difference in results between the previous human study and the present study may be attributable to the intake of CoQ10. In the previous study by Skough et al (2008), the intake of CoQ10 was 200 mg/day (100 mg, twice daily; breakfast and night). On the other hand, the intake of CoQ10 in the current study was 5 mg/kg/day for 14 days.

5. Conclusion

Briefly, in addition to contradictive studies, this study showed that short-term supplementation with coQ10 significantly reduces the triglycerides level and attenuated the muscle damage indicators (creatinine kinase and lactate dehydrogenase) in healthy elite cyclists. However, could provide

appropriate practical recommendations in order to scope intense training-induced mechano-metabolic stresses with emphasis on CoQ10 supplementation with conducting this study and examination of its probably results. In addition, our study had several limitations that were include: antioxidant enzymes hadn't been investigated, inflammatory and damage indices wasn't assayed, serum CoQ10 concentrations was not examined, and finally our study hadn't any control group (sedentary) who didn't consume CoQ10 during the 2 weeks.

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