

# Effects of oral coenzyme Q<sub>10</sub> on preventing the accumulation of lactic acid developing during the exercise performances of endurance skiing athletes

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**Abstract:** Aim: This study aims to examine the effects of oral coenzyme Q<sub>10</sub> (CoQ<sub>10</sub>) on preventing the accumulation of lactic acid developing during the exercise performances of endurance skiing athletes. Materials and Methods: The study population was composed of 15 volunteering male athletes. The blood samples thus collected were utilized for the measurement of athletes' CoQ<sub>10</sub>, lactate, lactate dehydrogenase (LDH), creatine phosphokinase (CPK), heart rate and blood pressure. Results: Significant decreases were identified in the systolic and diastolic blood pressures, pulses and plasma lactate levels of the athletes in the trial group when their pre- and post-exercise results were compared ( $p < 0,01$ ;  $p < 0,001$ ) while an increase of  $p < 0,01$  was observed in their plasma CoQ<sub>10</sub> levels. However, plasma LDH and CPK levels of the trial group athletes using 100 and 200 mg CoQ<sub>10</sub> were observed to increase less when compared to the results of the control group athletes in seven days ( $p < 0,001$ ;  $p < 0,01$ ). Conclusion: regular and gradual exercise is considered to enhance physical performance depending on the usage of CoQ<sub>10</sub> while it decreases the accumulation of La and thus delays fatigue.

**Keywords:** Endurance skiing, CoQ<sub>10</sub>, Lactic Acid, Exercise Performance

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## 1. Introduction

The basic goal of regular exercise is to prevent the organic deficiencies caused by a sedentary lifestyle, to increase the physiological capacity and tolerances, both of which are the basis of a dynamic and balanced life as much as possible, and to maintain this healthy structure as long as possible [1]. The point at which cardiac output addresses mostly is skeletal muscle. However, energy used by the skeletal muscles as fuel changes depending on the intensity and duration of the exercise as well as the performance level and nutritional status of the individual. Organism cannot supply enough oxygen when short-time exercise with high intensity is in question, so Adenosine Triphosphate (ATP) is synthesized through anaerobic means. On the other hand, fat is used as fuel in long-term exercises and thus the need for energy is met at a level of 60-70% [2]. While these exercises mentioned above do not cause an excessive burden on skeleton muscle system, too much or unusual exercises cause muscle fatigue the next day after the exercise.

Fatigue is accepted to be a natural result of exercise and mental effort; however, muscle fatigue is defined as the deficiency in producing or maintaining certain strength by means of muscular contraction [3]. When intense exercise (maximal or supra-maximal) is in question, exceeding the limits of aerobic metabolism increases the speed of glucose and lactic acid (La) is formed inevitably. When the intensity of the exercise is higher than the capacity of the body to supply oxygen, La system is used as fuel, the accumulation of La in the blood causes a decrease in the pH level and fatigue comes out as a result of enzymatic activity [2]. In this case, it is necessary to have a rest in order to ensure that lactate is eliminated from the body and damage at cellular level arises at the muscles because of exercise. This is observed as an increase in serum CPK and LDH levels, which is the indicator of specific muscle damage in humans [4].

During the exercise, coenzyme Q<sub>10</sub> (CoQ<sub>10</sub>) is a basic catalyst necessary for the energy production at cellular level. CoQ<sub>10</sub>, which is produced and synthesized naturally by human body and which is a benzoquinone compound, is

found in skeleton and cardiac muscular tissues that have a high level of physical activity as well as organs such as liver, kidney and brain [5]. Physical exercise increases the metabolic activity and oxygen consumption, and accordingly the production of reactive oxygen types, and thus it causes muscular fatigue and correspondingly oxidative damage. However, it is claimed that CoQ<sub>10</sub> enhances the energy production in the mitochondria and so increases the performance and decreases the fatigue [6]. The primary duty of CoQ<sub>10</sub> is its function as a part of mitochondrial respiratory chain and an important compound of cellular energy production [7]. This study aims to examine the effects of oral administration of CoQ<sub>10</sub> at different dosages on LDH, CPK and lactate levels which are accepted to be as fatigue parameters of post-training, heart pulse and blood pressures to determine the effectiveness of CoQ<sub>10</sub> during exercise performances.

## 2. Materials and Methods

This study is reproduced from thesis study carried out during September 2007 and June 2011 at Kafkas University, Health Sciences Institute, Department of Physiology. Thesis Protocol was accepted by Kafkas University Faculty of Medicine Ethics Committee with approval dated 09.06.2009 and numbered B.30.2.KAÜ.0.20.71.00. Conducted in line with the relevant directive specified in Helsinki Declaration, the study obtained approval from the Local Ethics Committee and ensured voluntary participation by providing study subjects with information on the objective of the study before the measurements.

The athletes were divided into 3 groups in the study. Group 1 (n=5): Control Group, Group 2 (n=5): the group taking 100 mg CoQ<sub>10</sub>, Group 3 (n=5): the group taking 200 mg CoQ<sub>10</sub>. The age, weight and height averages of the respondents are stated in Table 1.

**Table 1.** Comparison of age, weight and height of the respondent athletes. The data were expressed as mean  $\pm$  standard deviation.

	Control Group	1. Group (100 mg)	2. Group (200 mg)
Age:	21.60 $\pm$ 0.51	21.80 $\pm$ 0.73	21.60 $\pm$ 0.51
Weight:	64.20 $\pm$ 3.14	66.80 $\pm$ 2.97	63.80 $\pm$ 2.31
Height:	1.76 $\pm$ 0.02	1.76 $\pm$ 0.01	1.75 $\pm$ 0.01

### 2.1. The Implemented Training Program

The study subjects were divided into three groups, namely the control group and two subject groups taking 100 mg and 200 mg CoQ<sub>10</sub>. For a period of 1 week, each one of the 3 groups was made subject to a training load of 70-80% and repetitions making up 2 hours in total once every day. The exercise program started at 10.<sup>00</sup> and was systematically adjusted specifically for each group to obtain the most suitable effects in physiological terms. Before the athletes started the training program, their heart beat rates and blood

pressures were measured, blood samples were collected. Preparations of 100 and 200 mg of CoQ<sub>10</sub> were given to the athletes to be taken after dinner. The exercises were repeated in the same manner as the first day for a period of a week.

### 2.2. Blood Sampling

The blood samples of the athletes were collected once before exercise, and immediately after the training for a period of 1 week. HPLC [8], lactic acid levels of CoQ<sub>10</sub> levels were determined through lactate assay kit II. Blood analysis of LDH and CPK blood samples were carried out at Kafkas University Faculty of Medicine Central Laboratory.

### 2.3. Determination of Plasma CoQ<sub>10</sub> Levels

For the determination of CoQ<sub>10</sub> levels, a 50  $\mu$ l solution of 1,4-benzoquinone was added to plasma of 200  $\mu$ l (2 mg/ml) and the end product was vortexed for 10 seconds. 1 ml n-propanol was added to the product after 10 minutes. Following the vortexing of the tube for 10 seconds, 2 minutes of centrifugation was applied at 10.000 rpm to precipitate protein. The content was then injected into supernatant HPLC of two hundred micro-litres. It was transferred into a supernatant capped test tube that is capable of maintaining activity for 3 days at ambient temperature (22°C). The flow rate was 1 ml/minute following the composition of the mobile phase with ethanol-methanol (65-35%). The UV measurement was performed at 275 nm [8].

### 2.4. Measuring Blood Pressure

Systolic and diastolic blood pressures (tension) of the study subjects were measured from a. brachialis of the arm by means of using mercurial sifigmo manometer device in terms of mmHg immediately before and after the training [9].

### 2.5. Measuring Heart Rate

Heart rates (pulse/min.) of the study subjects during rest time were determined before the training and after the athletes were ensured to have a rest, by means of putting carotid and radial arteries on forefinger and middle finger for 1 minute [9].

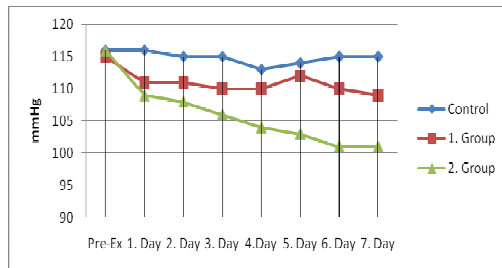
### 2.6. Statistical Analysis

The mean ( $\bar{X}$ ) and standard deviation (sd) of the data obtained in the study were measured by means of using MINITAB statistics packet program (Minitab Inc. Pennsylvania, Version 16.0 ABD). General Linear Model of the same program was used to determine the analysis in the study. Paired t-test was used to find out if there is a change between pre-substance and post-substance data. SAS 9.2 Proc GLM procedure was used to determine if there was any change among the Lactate and CoQ<sub>10</sub> levels of all 3 groups in those 7 days and the measurements were compared in line with LSD procedures [10]. The changes of all 3 groups in 7 days and their regression curves were calculated using Excel 2007. P values lower than 0.05 were regarded as significant.

### 3. Results

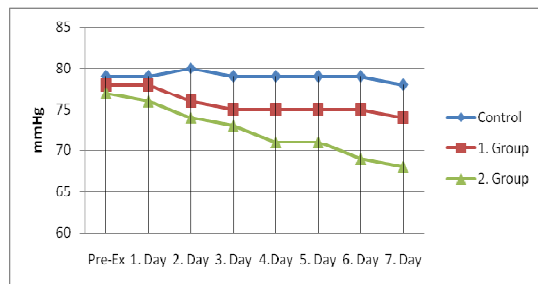
Ages, weights and heights of the athletes who participated in the study and who were all male were compared among the groups which are control and CoQ10. The results are summarized in Table 1. At the end of the comparison, the groups did not demonstrate a significant difference in terms of their ages, weights and heights ( $p>0,05$ ).

When systolic and diastolic blood pressures as well as heart rates of the trial groups during the training period of one week were compared separately with the control group, the related levels of trial groups which were high at the beginning were observed to decrease significantly in the following days and stay at a level lower than controls ( $p<0,001$ ) (Figures 1,2,3).



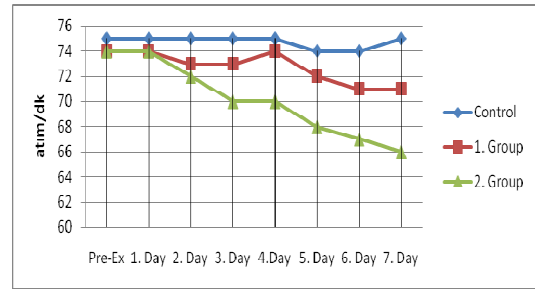
\*\*\*:  $p<0,001$ , Pre-Exercise (Pre- Ex.)

**Figure 1.** Changes in Systolic Blood Pressure Values of CoQ10 Trial Groups During One-Week-Training Period



\*\*\*:  $p<0,001$ , Pre-Exercise (Pre- Ex.)

**Figure 2.** Changes in Diastolic Blood Pressure Values of CoQ10 Trial Groups during One-Week-Training Period



\*\*\*:  $p<0,001$ , Pre-Exercise (Pre- Ex.)

**Figure 3.** Changes Heart Rates of CoQ10 Trial Groups During One-Week-Training Period

There is no significant difference between pre-training systolic and diastolic blood pressures, and heart rates of skiing athletes in the trial groups ( $p>0,05$ ); however, there are significant differences between post-training systolic and diastolic blood pressures ( $p<0,01$ ), and heart rates ( $p<0,001$ ) of the same groups. Table 2 summarizes the results of these measurements and their comparisons according to the groups.

The post-training levels of plasma LDH and CPK in all the groups were determined to increase continuously when compared to pre-training levels all throughout 7 days. However, the increase in the trial groups taking 100 and 200 mg CoQ10 was observed to be less than the increase in the control group. This increase was significant statistically and found in both of the trial groups at the level of  $p<0,001$  (Table 2).

Post-training increase in the control group was compared separately to the pre-training lactate levels of 100 and 200 mg CoQ10 trial groups, and significant decreases ( $p<0,01$ ) were observed. However, the decrease in lactate levels of the trial group taking 200 mg CoQ10 was observed to be more relevant than the decrease in the lactate levels of the trial group taking 100 mg CoQ10. When the pre- and post-training plasma CoQ10 levels of all the groups were compared, no change was observed in the control group whereas an increase of  $p<0,01$  was observed in the trial groups (Table 2).

**Table 2.** Mean and Standard Deviation Values ( $\pm$ SD) of All Groups' Pre-Training and Post-Training Systole, Diastole, Pulse, LDH, CPK, Lactate and CoQ10 Levels

Groups	Control Group		100 mg CoQ10 Group		200 mg CoQ10 Group	
	Pre-T	Post - T	Pre-T	Post - T	Pre-T	Post - T
Systole (mmHg)	116,2 $\pm$ 1,92	115,0 $\pm$ 1,00	111,6 $\pm$ 1,14	109,2 $\pm$ 0,83**	109,0 $\pm$ 1,00	101,0 $\pm$ 1,00**
Diastole (mmHg)	79,80 $\pm$ 1,64	78,80 $\pm$ 0,83	78,80 $\pm$ 0,83	74,0 $\pm$ 0,70**	76,60 $\pm$ 1,14	68,40 $\pm$ 1,14**
Pulse (atm/dak)	75,80 $\pm$ 1,09	74,60 $\pm$ 0,54	74,60 $\pm$ 0,54	71,60 $\pm$ 1,14***	74,20 $\pm$ 0,83	65,80 $\pm$ 0,83***
LDH (IU/L)	151,0 $\pm$ 12,9	442,6 $\pm$ 32,4***	146,2 $\pm$ 13,7	271,4 $\pm$ 11,2***	118,0 $\pm$ 12,9	240,4 $\pm$ 9,24***
CPK (IU/L)	135,0 $\pm$ 15,6	648,4 $\pm$ 19,5***	131,4 $\pm$ 8,01	583,6 $\pm$ 9,86***	129,0 $\pm$ 2,01	499,6 $\pm$ 13,2***
Lactate (nmol)	2,15 $\pm$ 0,12	4,40 $\pm$ 0,66**	2,59 $\pm$ 0,26	1,21 $\pm$ 0,33**	2,37 $\pm$ 0,37	0,55 $\pm$ 0,16**
CoQ10 ( $\mu$ mol/L)	1,57 $\pm$ 0,56	1,50 $\pm$ 0,27	1,57 $\pm$ 0,22	2,24 $\pm$ 0,73**	2,12 $\pm$ 0,32	3,80 $\pm$ 0,54**

\*\*\*  $p<0,001$ , \*\*  $p<0,01$ , \*  $p<0,05$ , Pre-Training (Pre.T.), Post-Training (Post.T.) coenzyme Q10 (CoQ10), Lactate Dehydrogenase (LDH), Creatine phosphokinase (CPK)

Plasma lactate levels of the groups considering the time period were compared on the basis of days (for 1 whole week of training); plasma lactate levels of the control group on the specified days were observed to increase positively whereas a negative decrease was observed in other groups. This decrease was not found to be significant between the

control group and trial group taking 100 mg CoQ<sub>10</sub> on the first day while it was found to be significant in the trial group taking 200 mg CoQ<sub>10</sub> at a level of  $p < 0.05$ . During the time period starting from the second day to the seventh day, the decreases and increases in the plasma lactate levels were observed to be significant ( $p < 0.001$ ), (Table 3).

**Table 3.** Changes in the Plasma Lactate Levels (nmol) on the Basis of Days During the Training Program of Seven Days When Control Group and Trial Groups Taking 100 and 200 mg CoQ<sub>10</sub> Were Compared

Groups	1. Day	2. Day	3. Day	4. Day	5. Day	6. Day	7. Day
	$\bar{X} \pm Sd$	$\bar{X} \pm Sd$	$\bar{X} \pm Sd$	$\bar{X} \pm Sd$	$\bar{X} \pm Sd$	$\bar{X} \pm Sd$	$\bar{X} \pm Sd$
Control	2.1±0.11 <sup>a</sup>	2.65±0.29 <sup>a</sup>	2.6±0.34 <sup>a</sup>	2.94±0.34 <sup>a</sup>	3.38±0.61 <sup>a</sup>	3.72±0.62 <sup>a</sup>	4.41±0.67 <sup>a</sup>
1. Group	2.19±0.49 <sup>a</sup>	1.65±0.12 <sup>b</sup>	1.66±0.36 <sup>b</sup>	1.39±0.22 <sup>b</sup>	1.31±0.23 <sup>b</sup>	1.3 ± 0.3 <sup>b</sup>	1.18±0.19 <sup>b</sup>
2. Group	1.31±0.22 <sup>b</sup>	1.22±0.14 <sup>c</sup>	1.09±0.25 <sup>c</sup>	0.91±0.12 <sup>c</sup>	0.77±0.31 <sup>c</sup>	0.67±0.12 <sup>c</sup>	0.53 ± 0.1 <sup>c</sup>

\* The difference between the groups expressed with different letters is significant statistically ( $p < 0.05$ ).

When plasma CoQ<sub>10</sub> levels of the trial groups were compared to the first day of the control group, no change was observed in the plasma CoQ<sub>10</sub> levels of the trial group taking 100 mg CoQ<sub>10</sub> until the third day; however, significant increases were observed starting from the 4<sup>th</sup> day.

On the other hand, plasma CoQ<sub>10</sub> levels of the trial group taking 200 mg CoQ<sub>10</sub> were observed to increase significantly in a positive way starting from the 1<sup>st</sup> day (Table 4).

**Table 4.** Changes in the Plasma CoQ<sub>10</sub> Levels (μmol/L) on the Basis of Days During the Training Program of Seven Days When Control Group and Trial Groups Taking 100 and 200 mg CoQ<sub>10</sub> Were Compared

Groups	1. Day	2. Day	3. Day	4. Day	5. Day	6. Day	7. Day
	$\bar{X} \pm Sd$	$\bar{X} \pm Sd$	$\bar{X} \pm Sd$	$\bar{X} \pm Sd$	$\bar{X} \pm Sd$	$\bar{X} \pm Sd$	$\bar{X} \pm Sd$
Control	1.43±0.18 <sup>a</sup>	1.69±0.52 <sup>a</sup>	1.65±0.38 <sup>a</sup>	1.59±0.25 <sup>a</sup>	1.86±0.58 <sup>a</sup>	1.62±0.43 <sup>a</sup>	1.5 ± 0.28 <sup>a</sup>
1. Group	1.83±0.34 <sup>a</sup>	1.75±0.54 <sup>a</sup>	2.03±0.33 <sup>a</sup>	2.11±0.44 <sup>b</sup>	2.02 ± 0.4 <sup>b</sup>	2.22±0.36 <sup>b</sup>	2.4 ± 0.45 <sup>b</sup>
2. Group	2.75±0.46 <sup>b</sup>	3 ± 0.72 <sup>b</sup>	3.4 ± 0.32 <sup>b</sup>	3.31 ± 0.5 <sup>c</sup>	3.7 ± 0.28 <sup>c</sup>	3.97±0.53 <sup>c</sup>	4.2 ± 0.21 <sup>c</sup>

\* The difference between the groups expressed with different letters is significant statistically ( $p < 0.05$ ).

## 4. Discussion

The limits of aerobic metabolism are exceeded during intense exercises and this increases the speed of glycolysis, which inevitably causes lactate formation. Because of this reason, energy consumption and metabolic activity increase to a great extent during physical exercise, in other words, intense muscular activity [11]. Accordingly, individual's need for energy increases depending on the exercise and it gets more and more difficult to continue trainings as muscular glycogen storage decreases; hence, a state of fatigue comes out quickly. However, it is also a well-known fact that CoQ<sub>10</sub> is an important catalyst that increases the energy production at cellular level during exercise. CoQ<sub>10</sub> can be effective in such a way because it increases the energy production in the mitochondria, decreases the plasma lactate level and thus lowers the feeling of fatigue [12]. In a study which was carried before [13], it was stated that taking CoQ<sub>10</sub> for 2 to 5 months caused a decrease of 16 mmHg in the systolic blood pressure while it caused a decrease of 10 mmHg in the diastolic blood pressure. It was observed that taking different dosages of CoQ<sub>10</sub> every day for 12 weeks caused a decrease in the systolic and diastolic blood

pressures to a great extent [14]. And in this study, we observed significant decreases at the level of  $p < 0.01$  in post-training systolic and diastolic blood pressures of skiing athletes taking 100 and 200 mg CoQ<sub>10</sub>. However, when systolic and diastolic blood pressures of the trial groups were compared separately to the ones in the control group during one-week training program, it was observed that these levels which were high in the trial groups at the beginning demonstrated a significant decrease of  $p < 0.001$  in the following days, staying at a level lower than the controls. Although all the cells in human body contain CoQ<sub>10</sub>, its level is higher in heart and liver, where energy is produced intensely. Because of this reason, the high level of energy necessary for systole and diastole of heart during exercise ensures that the organism adapts to this intense physiological activity by means of joining krebs cycle and being supplied by CoQ<sub>10</sub> which plays an important role in producing energy [15].

One of the most important factors that affect athletes' performances is heart beat rate. In the studies carried out by Wilmore et al. [16] and Digiesi et al. [17], taking CoQ<sub>10</sub> was observed to decrease heart beat rates to a great extent. And in this study, we observed significant decreases in post-training

heart beat rates for 1 week of skiing athletes which were administered 100 and 200 mg CoQ<sub>10</sub>. The decrease of ATP in hypoxia, which affects heart beating in a negative way, damages heart functions and ATP is not synthesized again in mitochondria. CoQ<sub>10</sub> given from outside starts ATP synthesis and normalizes heart functions by means of arriving at inner membranes of mitochondria and organizing enzymatic events playing a role in electron carrying chain in the mitochondria [18]. With regard to these results, it is possible to say that CoQ<sub>10</sub> which has a crucial role in energy production can improve diastolic functions, that there can be significant improvements in blood pressure and pulse functions of people who are hypertensive patients and who have mitral valve prolapses, left ventricular hypertrophy, hypertrophic cardiomyopathy and dysfunctional diastolic depending upon taking CoQ<sub>10</sub>.

Muscular fatigue usually comes out after exercise and muscular enzymes such as CPK and LDH are released because of the fatigue in muscular fibers during exercise. The increase in the level of serum CPK continues as muscular fatigue after exercise. Weakening energy storages and accumulation of metabolic waste products, which are listed among the reasons of fatigue, cause neural conduction and contractility to weaken [19]. In a study carried out with 17 volunteering healthy men and women in order to examine the effect of bicycle ergometric on physical fatigue, orally administered 100 mg and 300 mg CoQ<sub>10</sub> every day for 2 weeks were observed to cause a significant decrease in post-training serum CPK and LDH levels [5]. In this study, we observed that plasma CPK and LDH levels of all the trial groups increased gradually starting from the 2<sup>nd</sup> day of the one-week training, but the increase in the trial groups taking CoQ<sub>10</sub> was less than the increase in the control group. In line with these results, it has been concluded that continuous and gradually-increasing training not only generates a physiological dilatation and hypertrophy in the athlete's heart but also helps circulation to be more harmonious, and regular training can ensure that CPK and LDH levels which get higher right after the training decrease to normal levels after a while.

During intense exercise, lactate that accumulates in the muscle and blood causes fatigue. In this case, it becomes necessary to have a rest to ensure that lactate is eliminated from the body [11]. In a study carried out with 25 elite skiing athletes [19], it was determined that adding CoQ<sub>10</sub> decreases the level of muscle lactic acid, but it increases the level of plasma CoQ<sub>10</sub> after the exercise. In this study, we determined that post-training plasma lactate levels of trial groups taking 100 and 200 mg CoQ<sub>10</sub> demonstrated significant decreases. Porter et al. [20] carried out a study in which they made untrained men do bicycle ergometric as well as giving them 150 mg CoQ<sub>10</sub> every day for 2 months, and at the end of this period, they observed that lactate levels demonstrated significant decreases. Similar results were also observed by Fujimoto et al. [21]. In this study, we determined that post-training plasma lactate levels of trial groups taking 100 and 200 mg CoQ<sub>10</sub> demonstrated

significant decreases ( $p < 0,01$ ). However, a negative relation was observed between the increases in CoQ<sub>10</sub> and decreases in plasma lactate values. On the other hand, a positive relation was observed between plasma lactate levels and days in the control group whereas this relation was observed to be negative in trial groups. We can say that the increase in plasma La levels of the control group depends on the balance between catabolism and production ratio. Because during exercise, glucose is turned into lactic acid through anaerobic respiration when the level of oxygen decreases or all oxygen is consumed, and this generates a state of fatigue. However, it is possible to say that CoQ<sub>10</sub> taken at that moment carries electron to mitochondria, increases energy production in mitochondria, decreases the accumulation of lactate in the muscles, and thus increases physical performance. It was observed that CoQ<sub>10</sub> that was administered to 25 elite skiing athletes before training decreases the level of lactic acid in the muscles, increases the physical performance, and does not change the amount of VO<sub>2</sub>max, but increases plasma CoQ<sub>10</sub> level [19].

Similarly, in a study carried out by Weston et al. [22] in order to examine the effects of CoQ<sub>10</sub> on aerobic capacity, endurance and plasma CoQ<sub>10</sub>, 18 volunteering athletes were administered to take 1 mg/kg CoQ<sub>10</sub> every day for 28 days; and at the end of 28 days, plasma CoQ<sub>10</sub> levels of these athletes demonstrated significant increases while their VO<sub>2</sub>max levels did not demonstrate any change. Likewise, in a study carried out by Bonetti et al. [23], middle-aged respondents, all of whom ride a bicycle at least 1000 km a month, were administered to take CoQ<sub>10</sub> for 8 weeks and at the end of this period, plasma CoQ<sub>10</sub> levels demonstrated significant increases. Similar results were also obtained by Cooke et al. [24]. In this study, we observed significant increases in the post-training plasma CoQ<sub>10</sub> levels of trial groups. Athletes that are dealing with endurance sports have low concentrations of CoQ<sub>10</sub> in their blood; and this proves that taking CoQ<sub>10</sub> is necessary for increasing physical performance in such sports. However, although plasma CoQ<sub>10</sub> levels of trial groups demonstrated significant increases from the 1<sup>st</sup> to the 7<sup>th</sup> day, these increases were observed to be more distinct in 200 mg CoQ<sub>10</sub> groups, which is quite interesting. So, it is possible to say that energy consumption and metabolic activity increases to a great extent during physical exercise, namely, intense muscular activity, and that muscular fatigue comes out after maximal exercise activities. Because the fact that CoQ<sub>10</sub> concentration of athletes is low during exercise plays a crucial role as a main factor in the process of energy production that receives electron and gives proton in mitochondria wall.

## 5. Conclusions

It has been concluded that CoQ<sub>10</sub> caused significant decreases in post-training systolic and diastolic blood pressures and heart beat rates of trial groups whereas it caused a relatively limited decreases in LDH and CPK levels when compared to the control group; these decreases and

increases were mostly clear in skiing athletes taking 200 mg CoQ<sub>10</sub>; lactate levels demonstrated a significant decrease in trial groups to the contrary of plasma CoQ<sub>10</sub> levels; however, regular and gradual exercise increased physical performance depending on taking CoQ<sub>10</sub> while it decreased La accumulation and delayed fatigue.

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