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ORIGINAL ARTICLE

Effect of L-carnitine Supplementation on Pro- and Anti-Inflammatory Responses and Blood Pressure Changes Following Resistance Exercise in Non-Athletic Overweight Young Men

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ABSTRACT

Background: Amongst different therapeutic interventions, exercise can improve and control inflammatory and non-inflammatory diseases. This study investigated the effect of L-carnitine supplementation on serum levels of interleukin-8 (IL-8), IL-4, tumour necrosis factor-alpha (TNFα), nitric oxide (NO), and blood pressure after resistance exercise in young men.

Methods: Twenty male volunteers were selected by targeted sampling, and were randomly divided into an experimental (L-carnitine supplement) and a control (placebo) groups. The subjects participated in a session of resistance exercise after taking 2 g/day of L-carnitine or a placebo for two weeks. Blood pressure was measured and blood samples after fasting were obtained in four stages of baseline, pre-exercise, immediately, and 24 hours post-exercise.

Results: Consumption of L-carnitine could significantly increase serum levels of L-carnitine, IL-4 levels and decrease IL-8 and TNFα levels in the experimental group in comparison to the control group. In the control group, a significant decrease in IL-4 level and an increase in TNFα level were observed. NO and IL-8 increased significantly in both groups immediately after exercise, and then decreased significantly in both groups 24 hours post-exercise. There were significant differences in IL-4, TNFα, and IL-8 levels between the groups at pre-exercise, immediately and 24 h post-exercise in favor of the experimental group, while no significant changes were seen in blood pressure between the groups.

Conclusion: It is likely that 2 weeks of L-carnitine supplementation may prevent the increase of inflammation caused by acute resistance exercise.

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Introduction

Inflammation plays an important role in many diseases, including cardiovascular disease (1). Amongst all types of inflammatory proteins, the cytokines play a significant role in the initiation and progression of inflammation. Cytokines are

small proteins that affect the survival, proliferation, differentiation and function of immune cells and other body systems, and they are secreted by various cells, including neutrophils, active macrophages, fibroblasts, endothelial cells, and damaged muscle cells. Moreover, cytokines have several up and down-regulation effects in degradation and repair processes (2). Amongst all types of cytokines, the pro-inflammatory cytokine interleukin-8 (IL-8) acts as an angiogenic agent, and it is a chemokine that mainly absorbs neutrophils. A high level of IL-8 has been observed in activated muscles immediately after exercises, including resistance exercises, and it appears to be related to the inflammatory response of eccentric muscle actions (3). The tumour necrosis factor-alpha (TNFα) is also a pro-inflammatory cytokine that is produced mainly by T lymphocytes and macrophages, and it causes metabolic and cellular changes in many inflammatory diseases (4). Inflammation is caused by the interaction of pro-inflammatory and anti-inflammatory factors, so that in addition to pro-inflammatory cytokine, such as interleukin-8, there are also anti-inflammatory agents, for instance (IL-4), that prevent and suppress inflammation. IL-4 is considered a multidimensional cytokine that is secreted by T lymphocytes, basophils and mast cells (5).

Inflammation was shown to contribute to high blood pressure as T cells play an important role in an increase in blood pressure. Researchers have examined a subset of T cells in rats injected with angiotensin II, and showed that production of interferon-gamma (γ-INF, secreted by donor cells 1) increased, but IL-4, which is secreted from donor cells 2, decreased in response to angiotensin (6). Therefore, interventional methods that can modulate the inflammatory effects of cytokines can play an effective role to reduce inflammation and mitigate the related diseases.

Amongst the variety of therapeutic interventions, exercise is recommended to improve and control inflammatory and non-inflammatory diseases. Regular physical exercises have been shown to increase the anti-inflammatory effects that reduce mortality from chronic inflammatory diseases, such as cardiovascular disease and type II diabetes (7). Kraemer- Aguiar *et al.* (2017) showed that resistance training might have long-term anti-inflammatory effects through the accumulation of acute inflammatory responses following resistance exercise, leading to the regulation of cytokine levels (8). Furthermore, Sardeli *et al.* (2018) also reported that resistance training reduces inflammation (9). On the other hand, performing short-term physical exercise, such as a resistance exercise, can cause damage to muscle tissues and provoke an acute inflammatory response. High-intensity and eccentric resistance exercises cause injury and higher muscle inflammation engendered by exercise, and the rate of this response is influenced by the type (eccentric and concentric muscle contraction), volume, load (weight), and intensity (degree of neuromuscular fatigue) of the resistance exercise (10). In this regard, Barquilha *et al.* (2018) reported that performing a session of resistance exercise was associated with increased muscle injury and inflammation. Increased inflammation caused by acute exercise was demonstrated to result in a higher-than-normal blood pressure, which in turn can lead to a serious damage to the vessel wall (11).

An effective treatment for inflammation is the use of nutritional supplements. Amongst the variety of supplements, L-carnitine has been recommended as a supplement to reduce obesity and increase fat metabolism. Carnitine (β-hydroxy-γ-trimethyl-aminobutyrate), with the chemical formula ($C_7H_{15}NO_3$), is secreted in the liver and kidneys with lysine and methionine amino acids. This substance, in its role as a carnitine palmitoyl transferase enzyme, facilitates the transfer of long-chain fatty acids for beta-oxidation into the mitochondria (12). Lee *et al.* (2015) reported that taking 1000 mg daily of L-carnitine supplement could reduce inflammation in people with coronary arterial disease (13). In addition, Malek-Mahdavi *et al.* (2016) reported the effect of the short-term administration of L-carnitine supplementation on reducing serum inflammatory mediators and mitigating pain in patients with knee osteoarthritis (14). Kashani *et al.* (2024) also reported the anti-inflammatory effects of L-carnitine supplementation (15).

L-carnitine supplementation, in addition to its positive effects on the metabolism, can play an important role in improving the body's antioxidant system (16), and its use before short-term exercise may be a strategy to affect the exercise-induced inflammatory cytokines. Therefore, the aim of the present study was to investigate the effect of two weeks of L-carnitine supplementation on the serum levels of L-carnitine, IL-4, IL-8, TNFα, NO and blood pressure changes in response to a session of resistance exercise in overweight men.

Materials and Methods

Twenty non-athletic young men voluntarily took part in this study which was conducted in Marivan, Kurdistan Province, Iran. The Review Board of the Islamic Azad University Ethics Committee approval the study. The statistical sample size for this study was estimated to be 24 people with the power of

0.8 using G-Power software. The sampling method was targeted and amongst 36 volunteers, 24 were selected and randomly divided into experimental and control groups (12 subjects in each group). However, four subjects were withdrawn from the study for various reasons before starting the measurements, and eventually 10 individuals in each group participated until the end of the study. The participants from the experimental group were supplemented with L-carnitine, and the control group with a placebo. The inclusion criteria for this study included no regular exercise in the past 6 months, no musculoskeletal injury, no history of chronic illness, no orthopaedic problems, as well as abstinence from alcohol, smoking and drugs (Table 1).

The exclusion criteria were gastrointestinal distress during L-carnitine supplementation, lack of regular participation in the research, and physical injuries. Prior to the start of the study, the participants were informed about the programmes, benefits, and potential risks for the subjects, and written consents were obtained from all participants. The medical records form and the international physical exercise questionnaire (IPAQ) (17) were filled in, and all of the subjects were, accordingly, healthy and non-athletic. The correct way of performing the resistance exercises was taught to the subjects, and their maximum strength was recorded for all such exercises.

The present study is an applied, randomized, double-blind and placebo-controlled clinical trial in a 4-step design. The subjects were given a supplementation or a placebo for a period of two weeks after being randomly divided into two groups of supplement (n=10) and placebo (n=10), after which they participated in a session of acute resistance exercise. Blood pressure and blood sample measurements were taken at baseline (before starting supplementation), after 2 weeks of supplementation or placebo (before starting resistance exercise), immediately after resistance exercise, and 24 hours after resistance exercise (Figure 1).

The resistance exercise program in this study was structured in a way which ensured that all large muscles of the body were used. This session of intense resistance exercise consisted of bench press, lateral pull down, leg extension, lying leg curls, bicep curls, triceps pushdown and shoulder press. Each exercise was performed in three sets to the point of exhaustion, with 85% of the one repetition maximum (1RM) of each subject, with a 2-minute rest between sets and exercises. Before the execution of the resistance exercise, all participants did a warm-up, which consisted of 3 minutes running, 5 to 10 repetitions with 50% of 1RM for each resistance exercise, as well as stretching (18). The subjects were also involved in a 10-minute cool-down at the end of the resistance exercise session.

Subjects were asked to refrain from any vigorous physical exercise 48 hours before taking the maximum strength test. The maximum strength of the subjects was determined by using the Brzycki equation (19). During 1RM, the individuals first warmed up with a light weight; then they had to choose a weight that they could lift up to 10 times.

Table 1: Descriptive and functional characteristics of the subjects at baseline.

Variable	Group (Mean±SD)		*P* value
	Supplement	Placebo	
Age (y)	28.3±3.2	27.8±2.2	0.416
Height (cm)	169.1±3.6	170.2±3.3	0.286
Weight (kg)	77.9±5.3	78.7±3.1	0.311
Body fat (%)	20.8±4.5	21.4±5.2	0.208
BMI (kg/m^2)	27.3±3.2	27.2±2.4	0.214
VO_2max (mL/kg/min)	36.7±4.6	37.5±4.1	0.387
L-carnitine serum (mg/L)	7.12±0.81	6.83±0.64	0.131

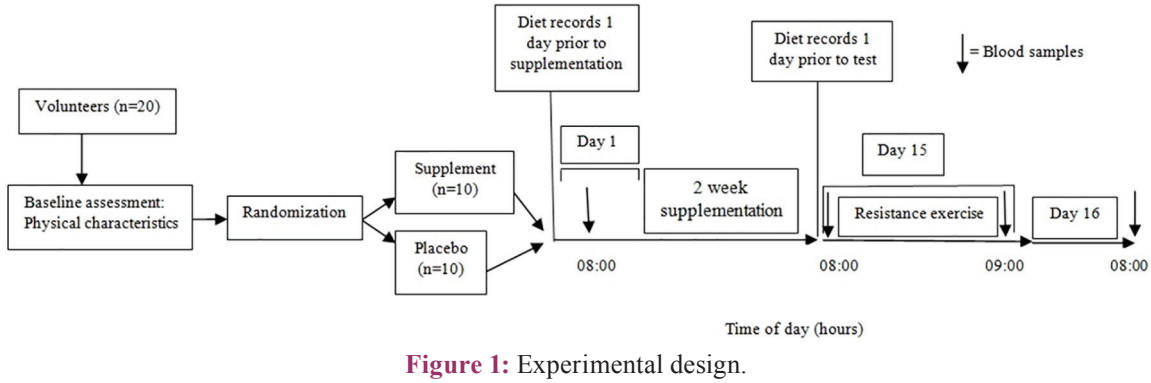

Figure 1: Experimental design.

The weight lifted and number of repetitions was recorded for each exercise and was used in the formula. Maximum strength=weight lifted (kg)/1.078-(number of repetitions to fatigue×0.0278).

The L-carnitine capsules used in the present study consisted of 1000 mg L-carnitine (Pharmaceutical Product of Shahre-Darou Co., Iran), approved by the Ministry of Health in Iran. The placebo capsules contained 1000 mg of maltodextrin, which was of the same color and size as the L-carnitine supplementation capsules. The subjects consumed 2 capsules daily (2 g of L-carnitine or maltodextrin daily) after two meals (breakfast and lunch) for a period of two weeks (20). The supplement and placebo were double blind, so the subjects and the researchers were not aware of the substance in the capsules.

Systolic and diastolic blood pressure was measured twice, at two consecutive times with a 10-minute interval in the sitting position by using the Andon digital blood pressure monitor (Andon Health Co. Ltd, model KD-5917, Nankai District, Tianjin, China), and the mean of these two measurements was recorded as the blood pressure for each subject. The validity of this blood pressure monitor was confirmed for clinical and personal use (21). The blood pressure of the subjects was measured on four occasions of at baseline (before supplementation and fasting), at the end of the supplementation phase (in fasting state), immediately after the resistance exercise, and 24 hours after the resistance exercise (in fasting state).

Prior to starting the study, all subjects were advised not to change their regular diet and to avoid any medication, supplements and food containing L-carnitine (such as red meat, fish, and dairy products) during the course of the study, and to report to the researchers in the event of consuming any of them. Information on the food consumed was collected by means of a 72-hour diary questionnaire, completed one day before the study. Food Processor software was used to calculate the energy intake, in addition to the amounts of carbohydrates, fats, proteins, as well as vitamins such as A, C, E, B6, B3, and iron (as an important antioxidant required for endogenous carnitine synthesis) (Table 2). The energy intake of the subjects was assessed again after two weeks of supplementation or placebo (one day before the resistance exercise), by using the same method.

Blood samples were taken in four stages to evaluate blood variables before the start of the 2-week L-carnitine or placebo supplementation, before the start of the resistance exercise, immediately after the exercise, and 24 hours after the resistance exercise. All blood samples were taken after blood pressure measurement. Blood samples were taken from the antecubital forearm vein (5 mL), and were centrifuged rapidly at a speed of 3000 rpm for 20 min. The separated serum was kept frozen at -80°C for later analysis. The serum levels of IL-4, NO and IL-8 were measured by the ELISA method using human kits (Hangzhou Eastbiopharm co., Hangzhou, Zhejiang, China). The IL-8 kit had a range of 5-1000 ng/L and a sensitivity of 2.51 ng/L, the IL-4 kit had a range of 0.5-100 ng/L and a sensitivity of 0.27 ng/L, and the NO kit had a range of 2-600 µmol/L and a sensitivity of 1.12 µmol/L. The intra- and inter-assay coefficients of variation of these kits were less than 10% and 12%, respectively. In addition, the serum levels of TNFα were measured using human-made kits from France (Diaclone-Besancon Cedex-France) with a range from 25 to 800 pg/mL and a sensitivity of 10 pg/mL. The intra- and inter-assay assay coefficients of variation of this kit were 5% and 9.4%, respectively. L-carnitine levels were measured using L-carnitine Enzymatic UV test (Roche Diagnostics GmbH, Mannheim, Germany) in accordance with the method of Wieland *et al.* (22).

The data was analysed by using SPSS Statistics Software (Version 22, Chicago, IL), and statistically significant differences between the average values were evaluated at $p<0.05$. The Shapiro-Wilk test was used to determine the normal distribution of

Table 2: Dietary intake of the subjects after 2 weeks of supplementation or placebo (1 day before the resistance exercises).

Variable	Group (Mean±SD)		*P* value
	Supplement	Placebo	
Energy intake (Kcal)	2535±345.6	2610±412.5	0.403
Carbohydrate (g)	342.8±85.7	357.1±109.4	0.291
Protein (g)	83.2±22.5	87.4±31.7	0.320
Fat (g)	91.4±34.7	94.6±48.8	0.411
Vitamin A (µgRE)	670.8±150.4	684.6±168.3	0.392
Vitamin C (mg)	38.2±4.6	42.5±5.3	0.216
Vitamin E (mg)	13.2±3.1	11.8±2.4	0.314
Vitamin B6 (mg)	1.6±0.3	1.2±0.2	0.514
Vitamin B3 (mg)	14.2±3.8	12.8±3.1	0.472
Iron (mg)	21.7±3.5	18.8±2.7	0.193

the data, the Levine test to determine the equality of variances in the calculated variables in the groups, and Mauchly's test to examine the hypothesis of sphericity. Descriptive statistics, an independent-sample t-test, and repeated measures ANOVA with the Bonferroni *post hoc* test (interactive effect of time and group) were used to evaluate intra-group (time-effects) and inter-group (group-effects) parameters.

Results

The distribution of the data was normal at all stages of measurements, the variances were homogeneous, and the hypothesis of sphericity has been established. There were no significant differences between the experimental (supplement) and control (placebo) groups regarding the baseline variables (p>0.05). Serum IL-4 significantly increased in the experimental group after supplementation in comparison with baseline (p=0.038), but no significant changes were observed in IL-4 concentrations immediately and 24 h after exercise in contrast to baseline (p>0.05). In the placebo group, IL-4 serum levels decreased significantly immediately after exercise in comparison with pre-exercise (p=0.027) and baseline (p=0.022). At the 24 h post-exercise, IL-4 levels were significantly lower than the pre-exercise (p=0.039) and baseline (p=0.028). In regards to the results between the groups, there were significant differences between the supplement and the placebo group at pre-exercise (p=0.046), immediately (p=0.038), and 24 h post-exercise (p=0.031, Figure 2B).

Serum level of IL-8 in the experimental group after supplementation decreased significantly in comparison to the baseline (p=0.014), followed by a significant increase immediately after the exercise in contrast to pre-exercise (p=0.001), and then IL-8 levels decreased at the 24 h post-exercise when compared to after exercise (p=0.003) and baseline (p=0.001). In the placebo group, IL-8 serum levels increased significantly after exercise in comparison to pre-exercise (p=0.001) and baseline (p=0.019). At the 24 h post-exercise, IL-8 levels were lower in contrast to after exercise (p=0.024). IL-8 levels in the experimental group were significantly lower than the placebo group at pre-exercise (p=0.042), immediately (p=0.040) and 24 h post- exercise (p=0.031, Figure 2C).

Serum level of TNFα in the supplement group decreased significantly at pre-exercise in comparison

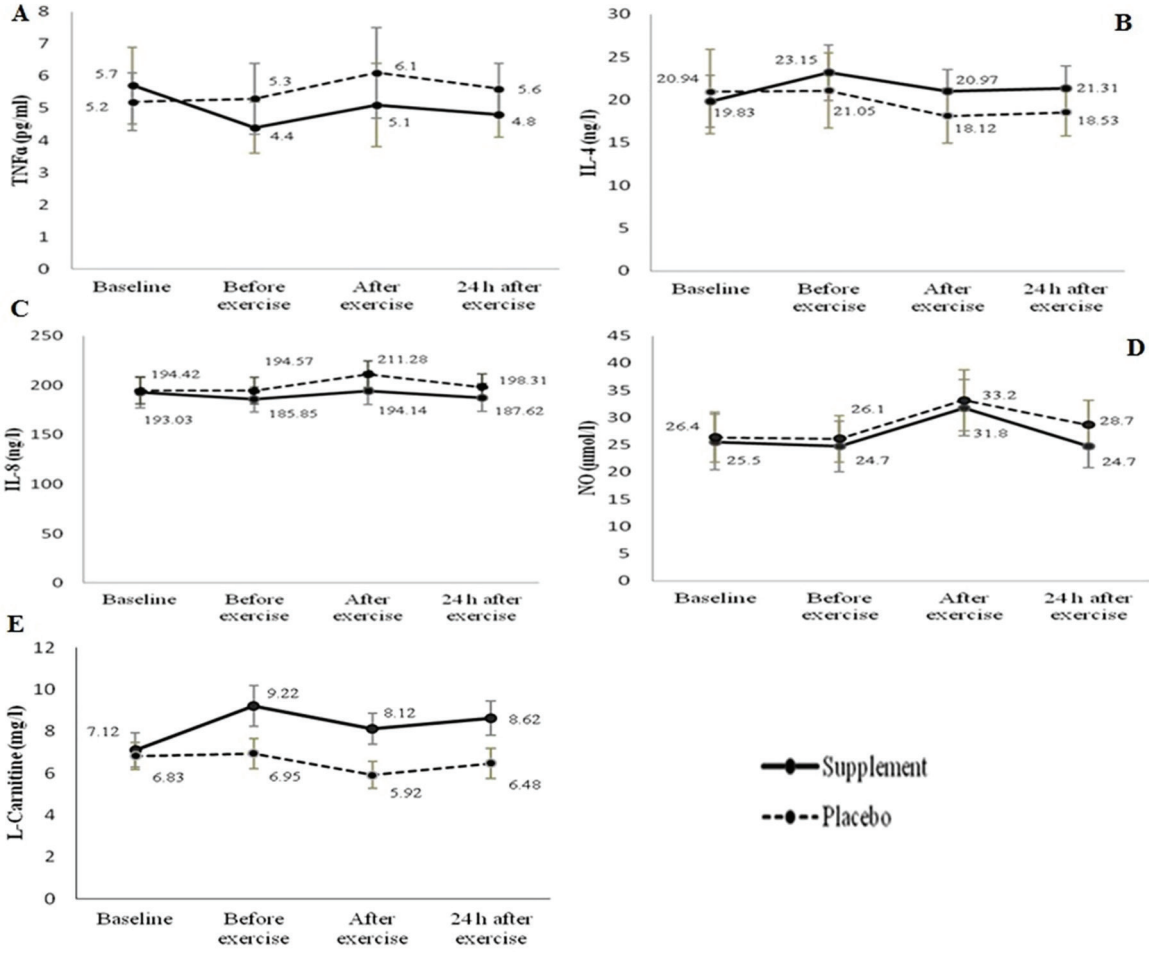

Figure 2: Changes in serum TNFα (A), IL-4 (B), IL-8 (C), NO (D), and L-carnitine (E) levels in the supplement and placebo groups at the baseline, pre-exercise, immediately after the exercise, and 24 h after the exercise.

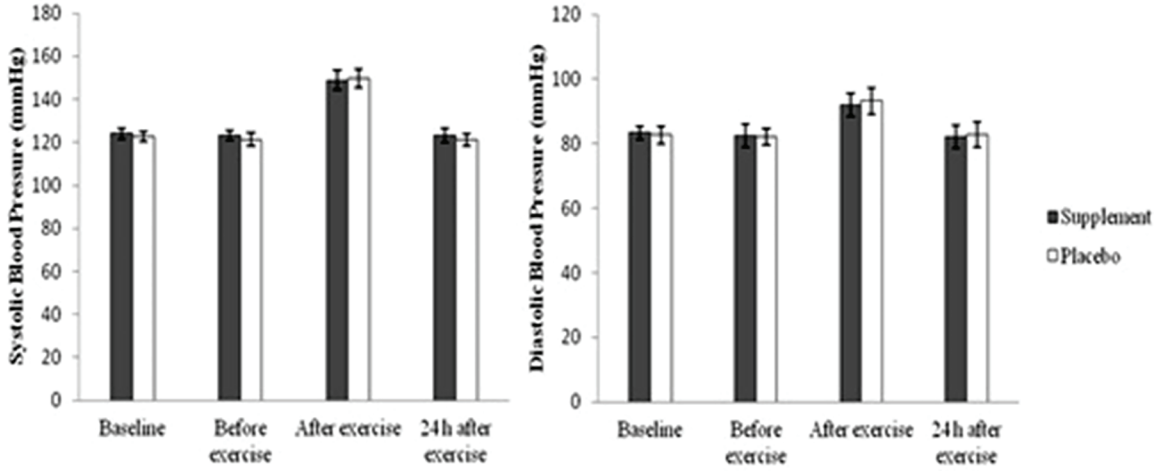

Figure 3: Changes in systolic and diastolic blood pressure in the supplement and placebo groups at the baseline, pre-exercise, immediately after exercise, and 24 h after the exercise.

to the baseline (p=0.036), but no significant changes were noticed in TNFα levels immediately and 24 h post-exercise when compared to the previous measurements (p>0.05). In the placebo group, serum TNFα level significantly increased immediately after exercise in comparison to the baseline (p=0.043) and pre-exercise (p=0.046), and there were no significant changes at 24h post-exercise (p>0.05). The comparison between groups showed that the serum levels of TNFα were significantly lower in the supplement group in contrast to the placebo group at pre-exercise (p=0.032) and immediately after exercise (p=0.041, Figure 2A).

There was no significant change in serum NO levels after two weeks of supplementation or placebo (p>0.05). Serum NO increased significantly within the groups (supplement group, p=0.039; and placebo group, p=0.024) immediately after the exercise in comparison to before exercise or the baseline. After that, serum NO levels decreased at 24 h post-exercise in supplement (p=0.001) and placebo (p=0.014) groups when compared to its level immediately after exercise (p=0.001). No differences were observed between the two groups (p>0.05, Figure 2D).

L-carnitine serum levels increased significantly in the supplement group after two weeks of supplementation (p=0.015) in comparison to the baseline. Furthermore, the levels of L- serum carnitine immediately after (p=0.041) and 24 h after exercise (p=0.035) were also significantly higher than those at the baseline (Figure 2E). No significant changes were observed in the placebo group (p>0.05). In addition, comparisons between the groups showed that the serum L-carnitine in the supplement group was significantly higher than the placebo group after two weeks of supplementation (p=0.034), immediately after (p=0.045) and 24 h after exercise (p=0.039).

There was no significant difference between the systolic (p=0.182) and diastolic (p=0.385) blood pressure of the two groups (Figure 3). The systolic blood pressure in the supplement and placebo groups significantly increased immediately after the exercise in comparison to the baseline (p=0.001, p=0.001), pre-exercise (p=0.001, p=0.001) and 24 h post-exercise (p=0.001, p=0.001), respectively. Similarly, the diastolic blood pressure of the subjects from both groups also increased significantly after the exercise in comparison to the baseline (p=0.001, p=0.001), pre-exercise (p=0.001, p=0.001), and 24 h post–exercise (p=0.001, p=0.002), respectively.

Discussion

The results of the present study showed that two weeks of L-carnitine supplementation could significantly increase the serum levels of l-carnitine and IL-4, and significantly decrease the serum levels of IL-8 and TNFα in comparison to the baseline, but no significant changes were visible in the NO serum level. There were no significant changes in the placebo group before exercise in contrast to the baseline. Inflammation is considered a controlled outcome between pro- and anti-inflammatory factors, during which cell activity is controlled (23), and according to the results of our study, the anti-inflammatory factor IL-4 increased and the pro-inflammatory factors IL-8 and TNFα decreased. In other words, inflammation appears to have decreased after two weeks of taking the L-carnitine supplement. These findingd support those of other studies that have shown the anti-inflammatory effects of L-carnitine supplementation (13, 14, 24).

However, this part of the results is not in agreement with those published by Rafraf et al. (20), while the possible causes of this discrepancy may be related to gender and type of exercise. L-carnitine

has been reported to have antioxidant properties (16, 25). Antioxidants inhibit the nuclear factor kappa-light-chain-enhancer of activated B cells (NF-KB) as an inflammatory signal cascade (26). Regulation of NF-KB can influence the inflammatory responses by affecting T cells, immune cells, and pro-inflammatory cytokine receptors, such as the TNF receptor (TNFR) (27). Although one limitation of the present study was the lack of measurement of antioxidant indices, L-carnitine may decrease NF-KB activation by reducing the production of free radicals. In this study, the serum level of free radical NO slightly decreased; although this change was not statistically significant. It has been shown that L-carnitine suppresses NO and production of nitric oxide synthase (NOS), both of which are factors in inflammatory diseases (28). On the other hand, L-carnitine supplementation has been reported to decrease NF-KB and TLR4 expression in liver tissue. And the TLR4 is another factor that can regulate inflammation (29). Therefore, it seems that one of the possible mechanisms of reduction of inflammation due to L-carnitine supplementation may be changes in the upstream factors regulating the inflammation.

In addition, the IL-4 serum levels in the experimental (supplement) group decreased immediately after the resistance exercise when compared to the pre-exercise (after supplementation), but this change was not significant and the value was actually slightly above the baseline. Moreover, there was a significant decrease in IL-4 level in the placebo group. IL-4 levels were significantly higher immediately after the exercise in the experimental group in contrast to the placebo group. It can be assumed that L-carnitine supplementation may have prevented the excessive decrease in serum IL-4 levels, since serum L-carnitine levels were significantly higher at this stage than those in the placebo group. Rahimi and Shoker-Nejad showed an increase in IL-4 concentration immediately after resistance exercise in athletic men (30), and the reason for this difference might be the training record of the subjects regarding the non-athletic men. Della-Gatta *et al.* reported that the history of training was effective in increasing the IL-4 cytokine concentrations (31).

In our study, the resistance exercise significantly increased IL-8 concentration in both groups in contrast to pre-exercise (after supplementation or placebo), but this increase was not significant in the experimental group in comparison with the baseline, and this is probably because of the changes in IL-4 concentration, due to the interaction between pro- and anti-inflammatory factors (32). In addition, the differences between the groups might be due to the use of L-carnitine supplementation. Similar to our results, Nieman *et al.* showed a significant increase in IL-8 concentration immediately post resistance exercise in resistance-trained individuals (33). On the other hand, Buford *et al.* reported that IL-8 concentration did not change significantly after resistance exercise in women, but tissue mRNA expression significantly increased (34).

Hirose et al. (2004) showed a significant decrease in IL-8 at 4 and 6 hours after eccentric resistance exercise, with 40% of 1RM. Resistance exercise may cause muscle injury due to the execution of eccentric contractions, which is ultimately associated with the production of cytokines (35). In our study, the intensity of resistance exercise was 85% of 1RM to the point of exhaustion, which may be one of the reasons for variations in results, in addition to the fact that the subjects in our study also took L-carnitine as a supplement. The serum levels of TNFα significantly increased only in the placebo group after performing the resistance exercise. Therefore, the use of L-carnitine is likely to prevent the significant increase in TNFα level. However, the NO level significantly increased in both groups, but no difference was observed between the groups. The mechanism of increased inflammation leads to the production of reactive oxygen species (ROS). Increased ROS can activate the transcription factor NF-KB, which in turn increases the synthesis of cytokines (36).

However, our study did not measure muscle injury indices. Nevertheless, serum levels of free radical NO increased immediately after acute resistance exercise in our study, which is another reason for increased inflammation. Increased levels of exercise-induced epinephrine can also increase the levels of inflammatory factors such as TNFα (37). However, another limitation of our study was the lack of measurement of epinephrine in the subjects. In addition, at 24h post-exercise, the IL-4 serum level increased, and IL-8, TNFα and NO levels decreased in both groups. This decrease in inflammation was significantly higher in the experimental group in comparison with baseline. At this stage, the rate of inflammation decline (increase in IL-4 level and decrease in IL-8 and TNFα levels) was significantly higher in the experimental group in contrast to the placebo group. A few studies investigated the response of anti- and pro-inflammatory factors to L-carnitine supplementation at different times, thus comparing results was difficult.

The reduction in inflammation in the recovery phase may be associated with supplementation. For instance, L-carnitine has been shown to increase the level of androgen receptors on muscle cells, thereby

improving protein signalling and muscle volume due to the reduction of muscle damage, in other words, improving muscle recovery (38). It seems that one of the mechanisms of increasing IL-4 concentration at this stage is due to muscle regeneration. IL-4 is involved in myogenesis and, therefore, a significant increase in IL-4 may play a role in the regeneration and enhancement of muscle myogenesis after the resistance exercise. A study showed that deletion of IL-4 or its receptor in skeletal muscle in rats impairs muscle regeneration (39). Prendak *et al.* showed that short-term consumption of L-carnitine increased the total antioxidant capacity and decreased muscle injury at 24 hours post-exercise. Another mechanism seems to be a decrease in IL-8 and TNFα induced by a reduction in muscle injury due to L-carnitine supplementation (40).

Changes in the level of inflammation can affect blood pressure. In our study, considering the use of L-carnitine supplementation, it was necessary to study blood pressure as a major biomarker of cardiovascular disease. The subjects' systolic and diastolic blood pressure was not affected by the two-week administration of L-carnitine; however, it showed a slight decrease. The subjects' initial blood pressure was normal and they maintained these values; while taking L-carnitine, but if hypertensive subjects had been studied, the results could have been different. On the other hand, this slightly decreased blood pressure is also valuable from a pathological point of view. In general, although the results of this study are applicable, and they could not be easily compared with the findings of other studies, because the research in this field is limited and, therefore, further studies are needed to obtain accurate and stable results.

Conclusion

The results showed that inflammation increased immediately after acute resistance exercise, but this change was not significant in the experimental (supplement) group in comparison with the baseline. Therefore, taking L-carnitine as a supplement for a period of two weeks can prevent inflammation caused by acute resistance exercise. Moreover, a decreased level of inflammation immediately after exercise can contribute to a quicker recovery. On this basis, consumption of L-carnitine as a supplement for 2 weeks (2 mg/day) before starting acute resistance exercise can be recommended for reducing inflammation and improving recovery.

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Authors' Contribution

MH designed and directed the project, conducted the experiments, processed the experimental data, performed the analysis, drafted the manuscript, and designed the figures. HR and SK contributed to the design and implementation of the research, to the analysis of the results and to the writing of the manuscript. All authors read and approved the final version of the manuscript.

Conflict of Interest

None declared.

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