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Effects of Taurine Supplementation during Endurance Training on Antioxidant Activity in Males

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ABSTRACT

Background and objectives: Endurance exercise causes fatigue due to mitochondrial dysfunction and oxidative stress. The present study was designed to investigate the effects of taurine supplementation on lipids peroxidation and antioxidant activity during endurance activities.

Methods: Twenty-four male volunteers aged 27 ± 1.8 years and weighting 74.9 ± 5.9 kg were randomly divided into three groups: taurine supplementation (n=8), placebo (n=8), and control (n=8). The subjects completed a 28-day endurance training protocol. Biochemical parameters such as superoxide dismutase (SOD), glutathione peroxidase (GPX) activities, as well as malondialdehyde (MDA) concentrations (8 hours before the first session and 8 hours after the last session) and maximum rate of oxygen consumption, were measured to evaluate the antioxidant, lipid peroxidation, and VO_2 max status respectively. Finally, data were analyzed by SPSS software at a significance level of <0.05.

Results: Taurine supplementation significantly increased SOD (p=0.001) and GPX (p=0.001) but significantly decreased MDA (p=0.001). However, it had no significant effect on the VO_2 max.

Conclusion: The results of the present study indicate that taurine has antioxidant effects against endurance exercise-induced oxidant stress and lipid peroxidation.

Keywords: Glutathione peroxidase, Superoxide dismutase, male, Taurine.

INTRODUCTION

Endurance exercise causes fatigue due to mitochondrial dysfunction and oxidative stress (1, 2). Reactive oxygen species (ROS) are generated during mitochondrial oxidative metabolism, known as oxidative stress (3), which refers to the imbalance between excess ROS or oxidants and antioxidant response (4). Reactive oxygen species, such as superoxide anion (O2−), hydrogen peroxide (H2O2), and hydroxyl radical (HO•), consist of radical and non-radical oxygen species formed by the partial reduction of oxygen. Cellular ROS are generated endogenously in the process of mitochondrial oxidative phosphorylation, or they may arise from interactions with exogenous sources, such as xenobiotic compounds (5). Some ROS are capable of reacting with membrane lipids, nucleic acids, proteins, enzymes, and other small molecules and lead to cell damage (6). Oxidative stress results in direct or indirect ROS-mediated damage of nucleic acids, proteins, and lipids and has been implicated in atherosclerosis, diabetes, and aging (7).

Proteins, carbohydrates, and lipids (including phospholipids) are molecules that excessive ROS can modify. Lipids are susceptible targets of oxidation because of their molecular structure, which is abundant with reactive double bonds. Oxidative degradation of lipids by ROS is known as lipid peroxidation. One of the most known lipid peroxidation markers is malondialdehyde (MDA) (8), an organic compound with the nominal formula CH2 (CHO)2. The production of this aldehyde is used as a biomarker to measure the level of oxidative stress in an organism. Thus, by measuring MDA levels in biological samples, the extent of lipid peroxidation can be determined (9).

Contrary to oxidative stress, antioxidants can control autoxidation by interrupting the propagation of free radicals or by inhibiting the formation of free radicals and subsequently reducing oxidative stress, improving immune function, and increasing healthy longevity. Glutathione peroxidase (GPX) and superoxide dismutase (SOD) are examples of endogenous antioxidant enzymes (10). Several studies have reported that supplementation with exogenous antioxidants such as taurine maintains the antioxidant defense and leads to healthy longevity. Taurine is an essential amino acid for the body's metabolic processes that are thought to have antioxidant properties. Studies show that taurine supplementation increases total thiol content and decreases muscle soreness, lactate dehydrogenase level, creatine kinase activity, and oxidative damage (7). Several studies have reported the cytoprotective actions of taurine against pathologies in the cardiac (10) and skeletal (11, 12) tissues. Accelerated aging (13) and the incidence of muscular disorders (11) have been observed in taurine-depleted skeletal muscle. In skeletal muscle, contractile activities (such as physical activity) causes loss of taurine from the muscle cell (14), an increase in plasma taurine (15), and a decrease in antioxidant capacity (16-18).

Humans and other aerobic organisms constantly produce free radicals as part of normal metabolic processes. This process is more acute during intense physical activities (4). For instance, a 10-fold increase in the rate of whole-body oxygen consumption and a 100-fold increase in the oxygen flux in active muscles occur during whole-body exercise that results in increased ROS formation (14). Acute exercises generally induce an increase in ROS. In addition, it has been demonstrated that ROS production increases under muscle contraction and physical activity (19-21).

Taurine's complementary role during sporting activities has been explored (22-24). However, less attention has been paid to its antioxidant role. Therefore, this study aimed to investigate the effects of taurine supplementation during endurance training on antioxidant activity in males.

MATERIALS AND METHODS

The study included 24 untrained male volunteers with a VO_2 max of 46.1 ± 1.34 ml. kg −1. min −1, the mean age of 27 ± 1.8 years, a body weight of 74.9 ± 5.9 kg, and a height of 178.7 ± 5 cm. The subjects were randomly divided into three groups: taurine supplementation (n=8), placebo (n=8), and control group (n=8). After explaining the study's objectives and potential risks, written consent was taken from the subjects, and personal characteristics were recorded. The study protocol was approved by the Ethics Committee of Lorestan University and retrospectively registered at the Iranian Registry of Clinical Trial (IRCT2017052128429N3). All subjects were

nonsmokers. None of the subjects were taking taurine or other antioxidant or related supplements.

Before and after the protocol, the weight (Seca scales), height (Seca stadiometer), body mass index (BMI), VO_2 max (2.4 Km run test), GSH, MDA, and GPX were determined. Fasting blood samples (10 ml) were taken from the subjects 8 hours before the first exercise session and 8 hours after the last session to determine serum levels of GSH, MDA, and GPX. Test tubes were inverted several times, left at room temperature for 30 minutes, and then centrifuged at 4 ◦C for 10 minutes at 2,000 rpm. The resulting serum samples were kept at −80 °C until analysis.

The GPx activity was measured according to the method described by Rotruck et al. (25). Briefly, the reaction mixture containing 0.1 ml of 10 mmol/l sodium azide, 0.2 ml of 0.4 M phosphate buffer (pH = 7), 0.2 ml of glutathione, 0.2 ml of serum in 0.4 M phosphate buffer (pH = 7), and 0.1 ml of 0.2 mmol/l H_2O_2 was prepared. The contents were incubated at 37 °C for 10 minutes.

To stop the reaction, 0.4 ml of 10% trichloroacetic acid was added and then centrifuged at 3,200 rpm for 20 minutes. For glutathione content, the supernatant was assayed using Ellman's reagent [19.5 g 5,5-dimethylbarbituric acid in 100 ml 0.1 % sodium citrate]. The activity was expressed as U/mg protein.

Lipid peroxidation was assessed by determining serum MDA concentration using thiobarbituric acid as described by Templar et al. (26). Briefly, the reaction product was extracted from a mixture containing serum with 20% trichloroacetic acid solution, 8.1% sodium dodecyl sulfate, and 0.8% aqueous solution of thiobarbituric acid. The mixture was boiled for 1 hour, followed by cooling and the addition of n-butanol. The organic layer was separated by centrifugation at 4,000 rpm for 10 minutes and read at 532 nm. To assess serum MDA concentration by HPLC, 20 µl of supernatant was taken and injected into the HPLC. In addition, 1,1,3,3-tetra ethoxy propane (TEP) was used as standard.

All participants were subjected to 45 minutes of running at 70% of VO_2 max (based on heart rate), three sessions per week (at 5 PM), for four weeks. During the 28-day protocol, the subjects completed 12 sessions of exercise. The exercise protocol included 10 to 15 minutes of warm-up and 10 minutes of cool-down. Before (one hour) and after (24 hours after the latest session) protocol, VO_2 max was assessed by a 2.4 Km run test as described by Burger et al. (27).

Data analysis was conducted using SPSS software (IBM SPSS Statistics version 20.0). Data are represented as mean ± standard deviation. Data were analyzed using one-way ANOVA (after the difference of the post-test from the pre-test), followed by a Tukey test. A *p*-value of less than 0.05 was considered statistically significant.

RESULTS

All subjects completed the study. The characteristics of the subjects are shown in table 1. Weight, BMI, and VO_2 max as well as training heart rate and the 2,400-meter record did not change significantly in the study groups (Figure 1).

At the end of the study, SOD and GPX enzyme activity in the taurine supplementation group was significantly higher than in the control and placebo groups (p=0.0001). Moreover, the serum MDA level in the supplementation group was significantly higher than in the other study groups (p=0. 0001).

Table 1- The mean level of anthropometric characteristics of subjects at

Variables	Control (n=8)	Placebo (n=8)	Taurine (n=8)
Age (years)	27.75 ± 1.2	27 ± 2.1	26.25 ± 1.75
Height (cm)	179 ± 6.3	179 ± 4.5	178.1 ± 4.5
Weight (kg)	76.25 ± 8	75.1 ± 4.3	73.3 ± 5.1
BMI (kg/m^2)	23.7 ± 1.3	23.4 ± .92	23.1 ± .85
VO_2 max	46.2 ± .84	46.47 ± 1.8	45.7 ± 1.2

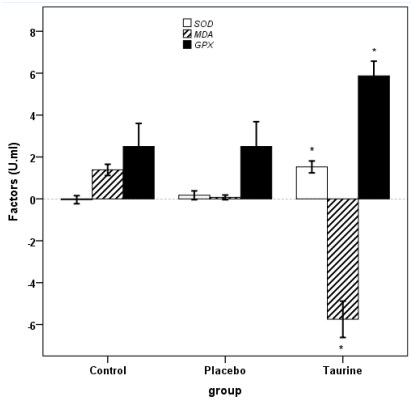

Figure 1- Changes of VO_2 max during the 28-day protocol in the study

DISCUSSION

The results of the present study indicate that taurine supplementation during an endurance activity program may increase GPX and SOD activities and decrease serum MDA concentration, a marker of lipid peroxidation. The production of ROS in cells promotes redox disturbances, thereby leading to oxidative damage to cellular components. Indeed, oxidative damage is associated with cell death through the destruction of biomolecules such as DNA, increased lipid peroxidation, protein oxidation, and production of free radicals (28). Interestingly, while regular physical activity has health benefits and increases antioxidant enzyme activity, and decreases oxidative stress, rigorous or prolonged exercise results in an acute increase in the production of ROS, as evidenced by elevated biomarkers of oxidative damage in both blood and skeletal muscles. It is thought that muscular contractions stimulate ROS production in active muscle fibers and that skeletal muscle is a primary source of ROS production during exercise. This contraction-induced ROS generation is associated with oxidant damage in several tissues (e.g., increased protein oxidation and lipid peroxidation), accelerated muscle fatigue, and activation of biochemical signaling pathways that contribute to exercise-induced adaptation in the contracting muscle fibers (28, 29).

In the present study, taurine supplementation significantly decreased serum MDA levels. In line with this finding, Abdel-Daim and coworkers showed that oral administration of taurine significantly ameliorated and normalized the harmful effects of oxidative stress on serum biomarkers of hepatorenal injury, lipid peroxidation, and tissue antioxidants. In that study, taurine provided significant hepatorenal protection against oxidative stress and apoptosis factors (30).

In another study, taurine significantly inhibited arsenic (As)-induced enhancement of MDA in the liver of mice (31).

It has been found that taurine could positively affect human organelles by enhancing mitochondrial membrane potential, increasing ATP levels, and mitigating mitochondria-mediated ROS formation (32). Mohammadi and coworkers reported that taurine supplementation could significantly enhance parameters such as mitochondrial ATP levels and mitochondrial membrane potential compared to a control group. Moreover, they reported that taurine prevented Ca2+-induced mitochondrial permeabilization, indicating that taurine might be an ideal and safe agent to protect mitochondria against toxic insults or regulate cellular function in different mitochondria-linked disorders (32). The antioxidant activity of taurine is linked to improved mitochondrial function, which diminishes mitochondrial superoxide. Taurine could be also associated with reduced lipid peroxidation either by prevention of ROS formation or by binding Fe2+ like a chelator as confirmed by Wu et al. (33). Regarding the role of taurine in the decrease of lipids peroxidation, Ogasawara et al. (34) showed that taurine can react with aldehydes, such as acetaldehyde and MDA, and has higher reactivity with these aldehydes than other amino acids. Also, the taurine-glucose reaction product has an antioxidant effect on lipids peroxidation of constituted liposomes (35). Since most cell membranes consist of lipid compounds, the observed effects of taurine may also be attributed to its ability to resist cell damage by membrane stabilization and osmoregulation in a non-specific way (35). Taurine also inhibits lipid peroxidation by inducing GPx and SOD. Taurine could protect tissues against reduced glutathione pool depletion by preventing a decrease in

glutathione reductase activity.

At the beginning of the study, to ensure the stability of the desired variables (SOD, GPX, and MDA), taurine supplementation was measured twice (two days and 1 hour before the start of the protocol). The results of the paired t-test showed no significant difference between the two measurements (Table 1). It seems that the following changes observed in the taurine supplementation group after the protocol was due to the impact of taurine (18, 36).

In this study, we implemented moderate to high endurance activities (with 75% VO2 max) as an inducer of ROS production. Lipid peroxidation caused an insignificant increase in VO2 max (less than 0.5 ml min. kg) in all three groups. However, taurine consumption did not significantly alter VO2 max. In a previous study, taurine along with a high dose of fructose was effective against fatigue (37). The cause of this contradiction may be due to the co-consumption of fructose.

A study by Ommati and coworkers showed that taurine supplementation could be a great option to increase muscle strength. Moreover, it was found that the beneficial effect of taurine on skeletal muscle mainly relies on its effect on mitochondrial function and energy status (38). Our findings indicate that taurine supplementation without a change in performance indicators caused by physical activity will increase SOD and GPX and decrease MDA. This suggests that physical activity can increase oxidation without increasing functional capacity. Although there are enzymatic and non-enzymatic systems for controlling the oxidation capacity of the body, taurine supplementation can play an essential role in reducing oxidation.

Our findings showed that following endurance activities that increase oxidative stress, taurine supplementation could exert positive effects such as reduced lipids peroxidation and increased activity of antioxidant enzymes, which is in line with the results of a similar study (39).

After endurance activities, oxidative and antioxidant changes occur in various tissues of the body, especially skeletal muscles involved in endurance activities (21); however, the results of this study were limited to measurements of the activity of the enzymes at the serum level. Therefore, we could not measure the effects of taurine supplementation and changes at the tissue level.

CONCLUSION

This study showed that taurine supplementation, even less than the recommended amount, increases the activity of oxidative enzymes and decreases the lipid peroxidation index. Moreover, taurine could be a potential preventive/therapeutic agent against oxidative stress linked to mitochondrial impairment. It is suggested that people who are somewhat involved in endurance activity include foods or supplements that contain taurine in their diet. The combination of endurance activity with taurine supplementation can modulate the lipid peroxidation index and oxidative stress caused by endurance exercise. Hence, this amino acid might be feasibly used as a supplement to improve oxidative enzymes and athletes' performance.

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DECLARATIONS

FUNDING

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Ethics approvals and consent to participate

The study protocol was approved by the Ethics Committee of Lorestan University and retrospectively registered at the Iranian Registry of Clinical Trial (IRCT2017052128429N3). Written consent was taken from all participants after explaining the study’s purpose objectives and potential risks.

CONFLICT OF INTEREST

The authors declare that there is no conflict of interest regarding the publication of this article.

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