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EFFECT OF GREEN TEA EXTRACT ON THE OXIDATION-REDUCTION BALANCE IN MEN EXPOSED TO INTENSIVE STRENGTH EXERCISE

Key words: green tea extract, oxidation-reduction balance, strength exercise.

ABSTRACT

The objective of this study was to evaluate the effect of green tea extract on the selected parameters of oxidation-reduction balance in blood of volunteers participating in intensive strength exercise. Students were exposed to four-week strength training and received green tea extract (GTE, n=17; 640 mg polyphenols/day) or placebo (n=18). Prior to (term I) and after the study (term II) all students performed strength exercise test. Blood samples were collected at rest, five minutes after completing exercise and after 24 h of restitution and were analysed for TBARS, TAS and LA concentration, also for SOD and CK activity. The exercise did not intensify lipid peroxidation and did not affect SOD activity, but caused decreases in plasma TBARS and TAS concentration at 24 h restitution. Strength training prevented exercise-induced rise in plasma CK activity and caused a drop of SOD activities. Supplementation with GTE enhanced resting plasma TAS, but did not affect blood parameters of oxidation-reduction balance, neither at rest nor at post-exercise.

INTRODUCTION

Oxidative stress is defined as a shift from the prooxidant-antioxidant balance towards oxidation reactions [33]. It results from enhanced production of reactive oxygen species (ROS), exceeding the potential of antioxidant defense of an organism [1]. In such conditions, ROS react with macromolecules, including lipids, proteins and DNA, which finally results in their damage [32].

It is common knowledge that a factor inducing the oxidative stress is long-term, strenuous aerobic exercise [22]. It is suggested that during such exercises, oxidative damages occur, most of all, as a result of increased oxygen consumption. However, a number of other factors, including acidosis, catecholamine autooxidation, ischemia-reperfusion syndrome, are known to be capable of inducing the oxidative stress in *in vitro* conditions. They are likely to serve as an important oxidative stress inducers during intensive, short-time anaerobic exercise [16].

Results of ample studies indicate that physical training, especially endurance training, increases the body's antioxidant potential [18]. On the other hand, it is suggested that endogenous antioxidant defense, even enhanced upon training, may be insufficient to prevent oxidative stress

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evoked by intensive exercise [10]. Taking this into account, in order to attenuate oxidative stress, it is recommended to increase the supply of dietary antioxidants. Today, much evidence indicates that dietary supplementation with plant biophenols may be a successful strategy to decrease the risk of pathologic conditions related to free radical overproduction and/or to prevent their complications [8].

Compounds of polyphenolic structure occurring in vegetables and fruits include flavonoids that cover: anthocyanidins, flavonols, flavones, flavononones and catechins [43]. Much emphasis has been focused on beneficial effects of green tea beverages which are the source of catechins, mainly epigalocatechin gallate (EGCG), epigalocatechin (EGC), epicatechin gallate (ECG) and epicatechin (EC) [13]. The mechanism of catechin activity includes free radical scavenging, metal chelation, inhibition of redox-sensitive transcription factors and pro-oxidant enzymes, and induction of phase II detoxifying enzymes [23]. Catechin supplementation can affect oxidative stress by a direct action and also by affecting the levels of other antioxidants present in human body [46].

Literature provides numerous reports indicating that regular consumption of green tea may reduce risk of cardiovascular disease [37, 38] or different types of human cancer [42]. So far, there has been no information on the effect of green tea upon the post-exercise oxidative stress indicators in humans. It seems interesting, therefore, to investigate the effect of green tea supplementation on parameters of oxidative stress induced by intensive strength exercise. It is common knowledge that strength exercise, especially eccentric, often leads to a considerable microinjury of skeletal muscle cells, with concomitant activation of neutrophiles and monocytes, and that the appearing inflammatory focus is an additional source of free oxygen radicals [24].

Therefore, the aim of this study was to evaluate the effect of green tea extract administration on the selected parameters of oxidation-reduction balance in blood of volunteers exposed to intensive strength exercise.

METHODS

The study was carried out on 35 male students of physical education, non-smokers, not practicing high-performance sports and consuming meals at a University canteen (three meals a day), who for at least two months prior to the study were not provided with any supplements (including vitamins). The volunteers signed their written consent for participation in the study and the protocol of investigations was approved by the Commission of Ethics at the Warsaw Academy of Physical Education. Before the tests, all volunteers were familiarized with the aim and schedule of the study. They were also informed about the possibility of resigning from participation at any stage of the experiment. In addition, the subjects were instructed not to take vitamins or other supplements, and to avoid consumption of flavonoid-rich products between meals throughout the experimental period.

Each participant filled in their food intake questionnaires covering three days (including 2 working days and 1 holiday) at the first and three days of the last week of the study. Daily intake of energy, protein, fat and carbohydrates as well as vitamins A, C and E with food rations was calculated with the use of Dietus software based on national food tables [20].

For the period of four weeks all participants were exposed to strength training focused on the development of strength endurance. Prior to the study and fours weeks after, each student was subjected to measurement of one maximum repetition (1RM) in all exercises included in the training program. It is the highest possible weight in a single lift in a given strength exercise [39]. Trainings were carried out three times a week. Each training unit covered eight strength exercises engaging the main groups of muscles (among others: bench press and back squat), performed in three series in 15 repetitions with a load of 60% 1-RM [39]. At the same time, students were randomly divided into two groups. One group was treated with a green tea extract – two capsules a day for four weeks (one capsule contained 320 mg of polyphenols, calculated as the content of epikatechin, group GTE, n=17), whereas students from the second group obtained at the same time a placebo capsule (containing maltodextrin, with identical appearance and dosage as the capsules with green tea extract, group P, n=18). Both the green tea extract and placebo were produced by Olimp Labs (Dębica, Polska). The dosage of the green tea preparation was based on producer's recommendations.

Before training and supplementation (term I) and after completing the study (term II), all students performed intensive strength exercise (i.e. test exercise), consisting of two exercises: bench press and back squat (one series each), performed to exhaustion, with a load at 60% 1-RM and a 2-min rest between exercises.

Blood samples were collected from the ulnar vein into test-tubes with heparin before test exercise (rest), five minutes after exercise and after 24-hour restitution. Blood was centrifuged at 3000 x g for 10 min at a temperature of 4ºC. Erythrocytes were rinsed three times with a cold isotonic solution of sodium chloride. Both erythrocytes and plasma were frozen at –70ºC until analyzed. Red blood cells were determined for the activity of superoxide dismutase (SOD) with the use of a diagnostic kit by Randox (UK), which was expressed in U/gHb. Blood plasma was analysed for: activity of creatine kinase (CK), at a temperature of 37ºC, with the use of an Alpha Diagnostics kit (USA), concentration of substances reacting with thiobarbituric acid (TBARS), with the spectrophotometric method according to Buege and Austa [9] and concentration of total antioxidant status (TAS), with the use of a Randox diagnostic kit (UK).

In addition, prior to exercise, five minutes after it and 24 hours following its completion, blood was collected from the finger pulp. Capillary blood was assayed for the concentration of lactic acid (LA) with a ready kit by Dr Lange (Germany) as well as for parameters of acid-base equilibrium, hematocrit and hemoglobin concentration – with the use OMNI-C analyzer (Roche).

The results were statistically evaluated using a two-way analysis of variance (ANOVA) and NIR post-hoc test (with the use of Statistica v. 6.0 software). Differences were found statistically significant at $p<0.05$.

RESULTS

Table 1 presents anthropometric characteristics of volunteers and data obtained in the test exercise. No significant differences were found between P and GTE in age, height and body mass. After 4 weeks, as a result of training, in both groups a significant increase was observed in one repetition maximum, both in bench press and back squat. In addition, in both groups improvement was also observed in the number of repetitions in a set of back squat (increase by 21.7% in GTE and by 22.7% in P group), whereas the number of repetitions in a set of bench press did not change in both groups.

The mean daily energy intake as well as mean intakes of protein, fat, carbohydrates and antioxidant vitamins in daily food rations were compiled in Table 2. No significant differences in the diet were observed between GTE and P groups.

In both terms, the applied test exercise disturbed the acid-based equilibrium of blood (a significant decrease in blood pH and an increase of

Table 1. Characteristics of volunteers

		GTE (n=17)		Placebo (n=18)	
Age (yr)		21.5±1.1		21.2±1.3	
Height (cm)		176.8±7.3		179.9±3.7	
Body mass (kg)		73.7±8.1		74.6±8.7	
		Term I	Term II	Term I	Term II
Back squat	1RM	89.3±12.5	96.8±12#	85.3±14.9	93.9±14.7#
	number of repetitions	18.9±5.0	23.0±3.7#	18.5±5.2	22.7±4.3#
Bench press	1RM	76.1±18.0	84.1±17.2#	74.3±14.0	79.8±14.4#
	number of repetitions	22.4±3.2	24.1±3.7	23.3±5.5	23.2±5.3

Values are means ± SD, # significant differences ($p<0.05$) as compared to analogous value in term I (within the same group), P – placebo group, GTE – green tea group, Term I – before training and supplementation, Term II – after training and supplementation.

Table 2. Daily intake of energy and antioxidant vitamins in GTE and placebo groups

	GTE (n=17)	P (n=18)
Energy intake (kcal)	2611.4±515.1	2522.2±704.3
Protein (g)	87.7±33.9	93.5±35.1
Carbohydrate (g)	276.1±57.8	265.1±90.7
Fat (g)	99.9±31.1	106.7±52.9
Vitamin C (mg)	60.8±13.6	65.8±18.0
Vitamin E (mg)	7.3±1.8	7.4±2.4
Vitamin A (RE)[1]	1371.9±339.0	1258.1±382.6

Values are means ± SD, P – placebo group, GTE- green tea group, [1] – retinol equivalents

Table 3. Changes (mean values ± SD) in blood pH, base deficiency (BE) and lactic acid (LA) concentration induced by test exercise before (term I) and after (term II) four weeks of training with concomitant supplementation of placebo (P, n=18) or green tea extract (GTE, n=17)

	Placebo				GTE			
	Term I		Term II		Term I		Term II	
	rest	after 5 min	rest	after 5 min	rest	after 5 min	rest	after 5 min
pH	7.40±0.05	7.29±0.06*	7.41±0.01	7.28±0.04*	7.42±0.02	7.29±0.05*	7.41±0.01	7.27±0.03$^{*\#}$
BE [mmol/L]	1.29±1.05	–9.28±2.9*	0.86±0.7	–10.5±2.5*	1.42±1.7	–9.55±2.2*	0.86±0.8	–11.4±1.5$^{*\#\dagger}$
LA [mmol/L]	1.40±0.55	10.37±2.81*	1.51±0.68	11.86±3.33*	1.59±0.77	11.33±3.28*	1.86±0.63	13.12±2.65$^{*\#}$

* significantly different ($p<0.05$) as compared to the resting value (before exercise), $^{\#}$ significantly different ($p<0.05$) as compared to the analogous value in term I (within the same group), † significantly different ($p<0.05$) as compared to the analogous value in placebo group.

base deficiency) as well as evoked a significant increase in blood lactic acid concentration (Table 3), indicating metabolic acidosis. In the placebo group, post-exercise changes in the above-mentioned parameters did not differ significantly between term I and II. In the GTE group, the changes were more intensified in term II, as compared with term I, which may indicate an increase of exercise tolerance.

The test exercise did not intensify lipid peroxidation (a lack of significant changes in plasma TBARS concentration 5 min after the exercise; Fig. 1). In contrast, both in terms I and II, after 24-h restitution, a significant decrease was observed in TBARS (term I: a decrease by 31% and 37% in P and GTE, respectively; term II: a decrease by 46% and 37% in P and GTE, respectively).

In term I, exercise test did not affect plasma TAS (Fig. 2) in the P group, whereas in the GTE group a significant increase of that parameter (by 9.6%) was observed five min after exercise. In term II, no significant changes were reported in plasma TAS at five min in any of the groups. In turn, after 24-h restitution both groups were characterized by its significant decrease (by 9.2% and 10.8% in P and GTE, respectively).

The activity of SOD in erythrocytes (Fig. 3) did not change significantly under the test exercise in both terms, but in term II significant drop of resting and post-exercise SOD activity occurred in both groups, as compared with analogous values recorded in term I (a decrease by 71% in P and by 60% in GTE on average).

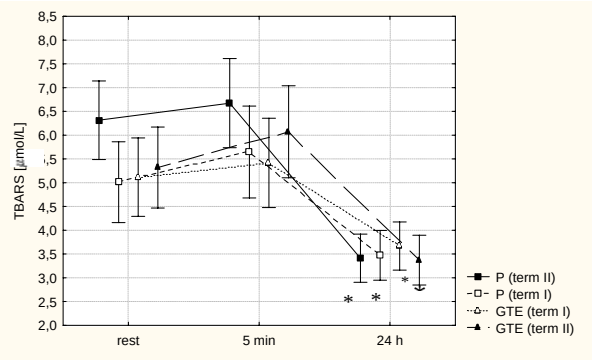

Figure 1. Mean changes (±95% of confidence interval) in the concentration of substances reacting with thiobarbituric acid (TBARS) in plasma induced by test exercise before (term I) and after (term II) four weeks of training and supplementation of placebo (P, n=18) or green tea extract (GTE, n=17). * significantly different ($p<0.05$) as compared to the resting value (before exercise) and 5 minutes after exercise completion

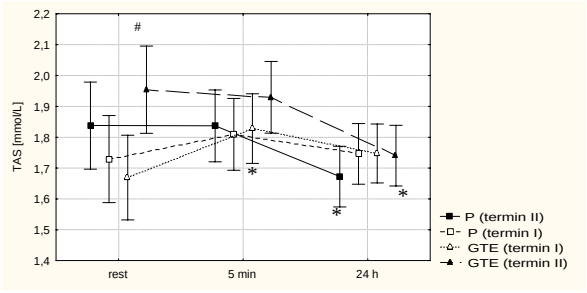

Figure 2. Mean changes (±95% of confidence interval) in the total antioxidant status (TAS) in plasma induced by test exercise before (term I) and after (term II) four weeks of training and supplementation of placebo (P, n=18) or green tea extract (GTE, n=17). * significantly different ($p<0.05$) as compared to the resting value (before exercise), # significantly different ($p<0.05$) as compared to the analogous value in term I (within the same group)

Test exercise, performed before the training and supplementation (in term I) caused a significant increase in plasma CK activity (Fig. 4), observed at 24-h restitution, as compared to the resting activity and that measured five min post-exercise (in P an increase by 88% and 66%, in GTE an increase by 76% and 72%, respectively). In contrast, any changes of plasma CK activity were noted in P and GTE groups after the exercise in term II. In addition, CK activity measured after 24-h restitution was significantly lower than the analogous value in term I.

Administration of green tea extract (GTE) does not seem to affect any changes in the above-discussed parameters, except for a significant increase of TAS resting values in plasma (by 6.5%).

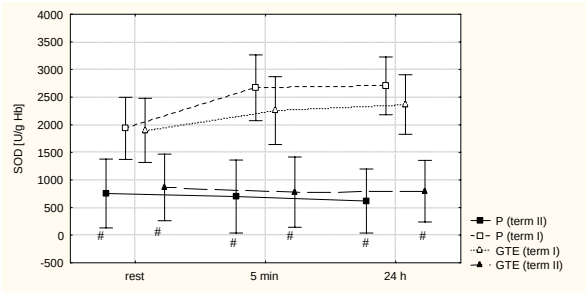

Figure 3. Mean changes (±95% of confidence interval) in the activity of superoxide dismutase (SOD) in erythrocytes induced by test exercise before (term I) and after (term II) four weeks of training and supplementation of placebo (P, n=18) or green tea extract (GTE, n=17). # significantly different ($p<0.05$) as compared to the analogous value in term I (within the same group)

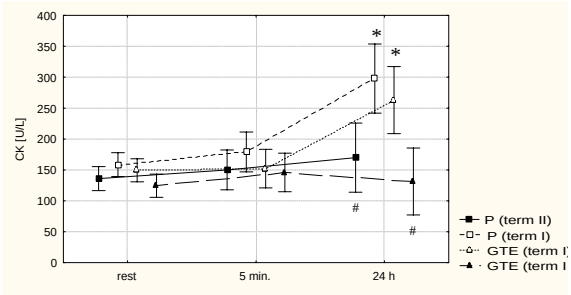

Figure 4. Mean changes (±95% of confidence interval) in the activity of creatine kinase (CK) in plasma induced by test exercise before (term I) and after (term II) four weeks of training and supplementation of placebo (P, n=18) or green tea extract (GTE, n=17). * significantly different ($p<0.05$) as compared to the resting value (before exercise) and 5 minutes after exercise completion, # significantly different ($p<0.05$) as compared to the analogous value in term I (within the same group)

DISCUSION

Results of recent investigations provide clear evidence that intensive physical exercise, not only long-term aerobic, but also short-term anaerobic may induce oxidative stress [2]. It is postulated that oxidative stress is likely to contribute to fatigue and damage of muscle cells and, as a consequence, may affect exercise performance [41].

One of the effects of oxidative stress is intensification of lipid peroxidation, which is reflected by an increase of blood concentration of its products, i.e. lipid hydroxyperoxides (LOOH), malondialdehyde (MDA), as well thiobarbituric acid-reacting substances (TBARS) [11]. In studies carried out by McBride et al. [24] repeated intensive strength exercises evoked an increase in MDA concentration in blood that maintained at a higher level than pre-exercise even at 24 h after the exercise.

The test exercise strength in character, performed in our study, despite significant disturbance of the acid-base equilibrium and a high increase in blood concentration of LA, did not induce oxidative stress, since the level of TBARS in blood plasma did not change five min after exercise, and finally decreased after 24-h restitution. Theoretically, an increase in the production and release of LA to blood may evoke a decrease in the TBARS level, since *in vitro* studies demonstrated the antioxidant activity of lactate ion itself [15]. However, according to other authors [31], under *in vivo* conditions, acidosis accompanying the accumulation of lactate is a factor enhancing peroxidation of lipids and production of MDA through, among others, intensification of superoxide radical transformation into highly reactive hydroxyl radical or more lipid soluble hydroperoxyl radical. Thus, in our study, the applied test exercise should intensify lipid peroxidation since, as indicated by other authors' findings [45], a positive correlation exists between post-exercise concentrations of LA and TBARS. In contrast, some reports have emphasized that MDA or TBARS are indirect and insensitive markers of lipid peroxidation processes and their concentration in blood not always reflects the actual degree of that process intensification [21]. It is confirmed by a decrease in TBARS (–23.7%) observed by [16] in plasma after 30-s Wingate test, paradoxically accompanying the increase in blood level of lipid radicals detected by the electron spin resonance (ESR) spectroscopy. In addition, that work [16] demonstrated a negative correlation between the post-exercise TBARS level and peak power developed during the test, which prompted the authors to conclude that exercises of that type are likely to stimulate MDA elimination from blood. This was also indicated in a study carried out by [2] a group of non-training men, who observed an increase in lipid hydroxyperoxides in blood soon after and one hour after repeated isometric contractions (at 50% maximal voluntary contraction, using a hand grip dynamometer), and a simultaneous lack of significant changes in blood concentration of MDA. No changes in the MDA level in blood were either observed in trained men, both after a single set of barbell squats (15 repetitions at 70% 1RM) [6], as well as after intermittent (13 sets of 10 repetitions at 70% 1RM) dumbbell squatting [7].

In our study, diminished plasma concentration of TBARS after 24-hour restitution points rather to a decrease in oxidative stress as a result of the exercise performed and is difficult to explain, especially in term I, when a decrease in the TBARS plasma level is accompanied by enhanced plasma CK activity. The peroxidation of membranous lipids under conditions of oxidative stress is claimed to be the cause of exercise-induced release of intramuscular enzymes to blood, and that the post-exercise increase of CK activity in plasma is a specific indicator of cellular damage of myocytes [24]. Although many authors have demonstrated a positive correlation between post-exercise activity of CK in plasma and blood concentration of TBARS or MDA [12, 24], still in other studies [17], including our own, such a relationship was not observed, which indicates the contribution of other factors, in addition to free radicals, in determining the integrity of myocyte membranes and affecting CK activity in plasma [17].

Likewise in our study, a trend for MDA decrease after 24-hour restitution was also observed by Bloomer et al. [5] in anaerobically trained men, performing repeated barbell squats (6 sets of 10 repetitions using a load equal to 70% 1RM, with three min of recovery between sets). Although the exercise applied in the above-cited paper evoked muscle soreness and an increase in CK activity in plasma, no loss was observed of cytoskeletal protein-desmin in the vastus lateralis muscle samples as a marker of cytoskeletal disruption. Taking into account a lack of detectable muscle injury, the authors [5] suggested that the factor affecting the intensification of exercise-induced

oxidative stress may be exercise volume, which means that a higher volume and inclusion of pure eccentric muscle action instead of traditional concentric/eccentric muscle actions may be required to induce oxidative stress and muscle injury. It is likely that in our research not only could the observed decrease of plasma TBARS level have been too low but also the intensity of the performed test, since resistance exercise was noted to increase MDA in the blood, but only at a high intensity [21, 24]. Indeed, in the study of McBride et al. [24] heavy resistance exercise was applied (three sets of 10RM for each of eight exercises, with 2-min rest between sets). It is also possible that the decrease of TBARS in our study could have been due to the stimulation of efficient antioxidant mechanisms.

It has been previously demonstrated, that ROS generation during intense physical exercise evokes an increase in the activity of defense systems, both the enzymatic and non-enzymatic ones. A marker of the combined capacity of antioxidants in plasma is TAS, and biological molecules contributing to plasma TAS include urate, plasma proteins, ascorbate, vitamin E and other antioxidants such as carotenoids and flavonoids [40]. Data on plasma TAS changes after exercises are conflicting. Skarpańska-Stejnborn et al. [36] using an incremental rowing exercise test, observed an exercise decrease of plasma TAS concentration accompanied by a TBARS level increase in erythro-cytes maintained in the restitution period (24 h post-exercise), both indicating intensification of the oxidative stress due to insufficient antioxidant defense. In turn, based on other investigations, it may be concluded that generally intensive physical exercise evokes an increase in the TAS level, although it does not always refer to the immediate post-exercise period, and that one of the mechanisms responsible for that increase is mobilization of tissue antioxidant stores into plasma [3]. The increased level of TAS, accompanied by elevated serum TBARS concentration, were observed by Nikolaidis et al. [27] at two min after treadmill running to exhaustion, both long-term and short-term. In turn, a similar long-term exercise protocol in study of Watson et al. [40], performed in the period of habitual high-antioxidant diet (H-AO) induced a decrease in plasma TAS immediately after the exercise, and then an increase of TAS above the pre-exercise value after 1-h recovery, and changes of plasma TAS were accompanied by the drop of plasma free F_2 – isoprostanes concentration, which pointed to reduce lipid peroxidation. It can be concluded, therefore, that the increase of TAS observed in our study five min after the test exercise in GTE group in term I, at a lack of TBARS changes in that period, as well as TAS drop after 24-h restitution (in both groups in term II) at a decrease of TBARS in that period, point to a highly efficient non-enzymatic antioxidant defense system of blood plasma. This is also indicated by s higher, as compared to findings of other authors [26, 35, 36] resting level of TAS which, when taking into account the norm (1.30-1.77 mmol/L), in term I reached the upper limit and in term II to exceed it, in spite of the fact that the analyses of nutritional questionnaires demonstrated low intake of vitamins C and E in both groups examined as compared with the recommended levels for adults [47].

In our study the activity of SOD in erythrocytes, as a representative of enzymatic antioxidant defense, was not affected by exercise, which confirms absence of oxidative stress. In contrast, completely unexpected was the decline in resting and post-exercise SOD activity following the four-week training in both groups. In contrast, other authors presented the increase in resting activity of SOD in adaptation to training. The resting SOD activity increase was demonstrated in a study by Miyazaki et al. [25] after 12 weeks of intensive endurance training in non-trained men. Zembroń-Łacny et al. [44] observed a high, positive resting correlation between SOD activity and TBARS concentration in erythrocytes in athletes, indicating the chronic state of oxidative stress induced by training. The increase in SOD activity indicates enhanced generation of ROS, and specifically superoxide anion radical [19]. According to the above, the lower post-training activity of SOD in our study may point to a lower rate of dismutation of the superoxide anion to hydrogen peroxide. In analyzing the resting activity of SOD in kayakers in the period of various-intensity training, Skarpańska-Stejnborn [35] observed an increase in its activity after a week of intensive training preparing athletes for competitions as well as a decrease of SOD activity after one-week training of lower intensity (rest after competitions). In turn, Poprzęcki et al. [30], similarly to our study, reported on the lower resting and post-exercise activities of SOD after four months of specific training in elite hurdle sprinters.

The strength training focused on the development of strength endurance, applied in our study,

apart from affecting the activity of SOD was also found to protect muscle cells against damage induced by single test exercise (a lack of increase in CK at 24 h restitution in term II), which is consistent with findings of other authors [4].

It is mentioned that dietary supplementation of antioxidants, such as antioxidant vitamins or especially flavonoids, may lower the level of oxidative stress induced by either intense training or single intense exercise, yet flavonoids seem to be more efficient than vitamin C [23] or vitamin E [28]. An increased intake of chokeberry juice (providing a daily dose of 750 mg of anthocyanins for 1 month) in rowers attenuated oxidative stress induced by incremental rowing exercise test, thus affecting lower, as compared to the control group, post-exercise level of TBARS and activity of SOD in erythrocytes, as well as post-exercise CK activity in plasma [29]. In the last experiment of those authors [34], 6-week supplementation with the black currant extract (3 capsules a day, 8.5 mg of polyphenols each) in rowers was found to decrease the TBARS level and SOD activity in erythrocytes during 24-h restitution after 2000 m exercise test and to increase plasma TAS after the exercise (at 1 min and 24 h after the exercise), without affecting the resting level of TAS. In contrast, the green tea extract administered in our study increased the level of TAS at rest, yet it did not affect levels of oxidative stress parameters nor CK activity neither in rest or post-exercise.

Likewise in our research, the increase in the resting level of TAS in plasma in handball players in competition season was reported by Bonina et al. [8] after one-month supplementation with red orange complex, still in that work the increase of TAS contributed to attenuation of stress induced by intensive training (a decrease in resting concentrations of LOOH and MDA in plasma). In our experiment, although in term II the resting level of TBARS in the placebo group tended to be higher than in the GTE group, the differences were not statistically significant. Some papers report also that polyphenols, administered both in the natural form and in the form of extracts, were observed to decrease oxidative stress intensity, even at a lack of changes in antioxidant potential of plasma [23, 26]. The increase in TAS concentration obtained in our study upon supplementation with GTE proves that a daily dose of polyphenols provided in a green tea extract was sufficient to counteract or attenuate the intensity of oxidative stress induced by the test exercise. It is likely that the effect of GTE on the level of oxidative stress parameters would also be observed under conditions of intensified oxidative stress. Therefore, the application of a test exercise with higher intensity and/or load in the next study seems to be advisable to investigate the effect of GTE supplementation on selected parameters of the redox status under conditions of exercise-induced oxidative stress. In summary, intensive strength exercise does not intensify the oxidative stress. Dietary supplement of green tea extract enhances endogenous antioxidant defense system in plasma at rest, but it does not alter selected blood indicators of redox-state induced by maximal strength exercise.

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