Effect of a Single Dose of Green Tea Polyphenols on the Blood Markers of Exercise-Induced Oxidative Stress in Soccer Players

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Purpose: To evaluate the effect of acute ingestion of green tea polyphenols (GTP) on blood markers of oxidative stress and muscle damage in soccer players exposed to intense exercise. Methods: This randomized, double-blinded study was conducted on 16 players during a general preparation period, when all athletes participated in a strength-training program focused on the development of strength endurance. After ingestion of a single dose of GTP (640 mg) or placebo, all athletes performed an intense muscle-endurance test consisting of 3 sets of 2 strength exercises (bench press, back squat) performed to exhaustion, with a load at 60% 1-repetition maximum and 1-min rests between sets. Blood samples were collected preexercise, 5 min after the muscle-endurance test, and after 24 hr of recovery. Blood plasma was analyzed for the concentrations of thiobarbituric acid–reacting substances (TBARS), uric acid (UA), total catechins, total antioxidant status (TAS), and activity of creatine kinase (CK); at the same time, erythrocytes were assayed for the activity of superoxide dismutase (SOD). Results: In both groups, plasma TBARS, UA, and TAS increased significantly postexercise and remained elevated after a 24-hr recovery period. SOD activity in erythrocytes did not change significantly in response to the muscle-endurance test, whereas in both groups plasma CK activity increased significantly after 24 hr of recovery. Acute intake of GTP caused a slight but significant increase in total plasma catechins. However, GTP was found not to exert a significant effect on measured parameters. Conclusions: Acute ingestion of GTP (640 mg) does not attenuate exercise-induced oxidative stress and muscle damage.

Keywords: catechins, antioxidants, oxidation-reduction balance

The generation of reactive oxygen species (ROS) is an inevitable consequence of normal cellular metabolism. The body defends itself against their detrimental activity by means of the antioxidant defense system, the objective of which is to render ROS harmless. The system is composed of antioxidant enzymes including, among others, superoxide dismutase (SOD) and glutathione peroxidase, as well as nonenzymatic antioxidants including glutathione, coenzyme Q₁₀, uric acid (UA), vitamins, and polyphenols (Lamprecht, Greilberger, Schwabeger, Hofmann, & Oettl, 2008). Under conditions of intensified ROS production, the antioxidant defense system may prove inefficient, which in turn leads to oxidative stress, a state induced by the imbalance between antioxidative and pro-oxidative processes (Fisher-Wellman & Bloomer, 2009).

Strenuous physical exercise is one of the factors alleged to induce oxidative stress. Under conditions of exercise-induced oxidative stress, increased oxidation of cell constituents—DNA, lipids, and proteins—has been observed (Banerjee, Mandal, Chanda, & Chakraborti, 2003). The oxidative stress induced by acute physical exercise may lead to aerobic damage of muscle tissue, consequently intensifying muscle soreness and diminishing exercise performance (Lamprecht et al., 2008).

It is suggested that lipids are more sensitive to oxidative damage than proteins are (Morillas-Ruiz, Villegas Garcia, Lopez, Vidal-Guevara, & Zafrilla, 2006). ROS release causes lipid peroxidation of polyunsaturated fatty acids in biological membranes and blood. Malondialdehyde, a by-product of lipid peroxide, and thiobarbituric acid reactive substances (TBARS; indirect assay used to measure aldehyde products, primarily malondialdehyde, formed via decomposition of lipid hydroperoxides) have been the most frequently used markers of oxidative tissue damage during exercise (Fisher-Wellman & Bloomer, 2009). In fact, increased oxidative-stress biomarkers have been observed not only after aerobic exercise (exhaustive long-distance cycling and running) but also after anaerobic exercise (supramaximal sprints or strength-type exercises; Bloomer & Goldfarb, 2004; García-López et al., 2007). It is common knowledge that intense anaerobic exercise induces oxidative stress,
mainly owing to an increased—under conditions of ischemia or reperfusion—activity of xanthine oxidase, an enzyme that is a significant source of ROS (Goldfarb, Bloomer, & McKenzie, 2005). In addition, during weight-lifting exercises cells of the skeletal muscles are subjected to microinjuries. In these areas an accumulation of infiltrated phagocytic cells, neutrophils, and macrophages occurs, while NADPH oxidase localized in the plasma membrane of the activated phagocytes constitutes an additional source of free oxygen radicals (Kon et al., 2007).

Athletes are especially exposed to oxidative stress (Alessio, 1993). While regular physical training, especially of the endurance type, may enhance the body’s antioxidant defense by various factors—for example, by increasing resting activity of antioxidant enzymes in erythrocytes (Metin et al., 2003)—the pursuit of optimal results in competitive sports requires that athletes often undertake strenuous training under conditions of insufficient postexercise restitution. This may, however, lead to overtraining. What is more, the unfavorable changes occurring in metabolism under conditions of postexercise oxidative stress may negatively affect the health of an athlete. Taking this into account, some authors suggest that supporting athletes with antioxidants could alleviate the negative effects of oxidative stress (Ho, Li, Chen, & Hsu, 2007). However, it should be mentioned that prevention of ROS formation by antioxidant supplementation may delay the recovery of muscle function (Teixeira, Valente, Casal, Marques, & Moreira, 2009) and even hamper training-induced adaptations (Gomez-Cabrera et al., 2008).

Although some surveys report no effects of antioxidant administration on the postexercise level of plasma markers of oxidative stress (Gaiani, Rahnama, & Hamedinia, 2006; McAnulty et al., 2008), and in some cases even demonstrate their pro-oxidative activity (Silva et al., 2008), numerous other studies have indicated a decrease in the postexercise oxidative stress in athletes as a result of long-term (≥14 days) supplementation with antioxidants (Bonina et al., 2005; Piłaczynska-Szczęsniaik, Skarpan ska-Steinborn, Deskre, Basta, & Horoszkiewicz-Hassan, 2005). In some studies, postexercise oxidative stress was observed to diminish after a single administration of antioxidants. One of those surveys demonstrated that the intake of a polyphenols-rich cocoa drink 2 hr before intense physical exercise (increasing-intensity test on a cycle ergometer) affected a significant reduction in the postexercise concentration of lipid-peroxidation indices in blood plasma (Wiswedel et al., 2004). Furthermore, in research conducted by Zembron-Lacny, Szyszka, Sobanska, and Pakula (2006), a single dose of vitamin E was observed to suppress the oxidative stress induced by a 2000-m laboratory rowing test. In another study with cyclists, Morillas-Ruiz et al. (2006) demonstrated a reduction in the extent of aerobic damage to protein after a strenuous exercise test on a cycle ergometer as a result of taking a drink enriched with polyphenols (black grape, raspberry, and red currant) during the exercise.

One of the most popular drinks rich in plant-originated polyphenols is green tea (Camellia sinensis). It is well documented that green tea polyphenols (catechins), apart from having strong antioxidant properties, may also act as nutritional chemopreventive agents for the treatment of cancer, atherosclerosis, and neurodegenerative diseases (Nicholson, Tucker, & Bameld, 2008). Few studies are reported in the literature concerning the effects of green tea polyphenols on blood markers of exercise-induced oxidative stress. In rats, green tea consumed for 6.5 weeks lowered renal liperoxidation after aerobic exercise (Alessio et al., 2002). In weight-trained men, Panza et al. (2008) observed protective effects of 7-day intake of green tea on blood markers of oxidative stress after resistance exercise. However, there are no reports regarding the effects of acute ingestion of green tea polyphenols on exercise-induced oxidative-stress biomarkers.

Therefore, the aim of the current study was to evaluate, in professional soccer players exposed to intense exercise, the effects of a single dose of green tea polyphenols on some noninvasive blood markers of oxidative stress: plasma total antioxidant status, plasma UA, plasma TBARS, erythrocyte SOD activity, and plasma creatine kinase (CK) activity (as a marker of muscle-cell damage).

Materials and Methods

Subjects

Sixteen healthy soccer players from the local club “Podlasie” were enrolled in the study. All subjects were nonsmokers and disease-free and had not used drugs, ergogenic aids, or antioxidant supplements for at least 2 months before the study. Before the experiment, all volunteers were informed about the aim of the studies and the experimental procedure. The study was approved by the ethical committee of the Academy of Physical Education in Warsaw, and informed written consent was obtained from all subjects before the beginning of the study.

The experiment was conducted during a general preparation period when all soccer players were pursuing strength training to develop strength endurance. Thus, as an exercise protocol, a muscle-endurance test was selected. It included three sets of bench press and back squat to exhaustion.

Three-day dietary records (2 working days and 1 weekend day) were used to estimate each subject’s average daily intake of energy, protein, fat, and carbohydrates, as well as vitamins A, C, and E, at the beginning of the study. Records were analyzed using the Dietus program based on the Table of Composition and Nutritional Value of Foodstuffs (Kunachowicz, Nadolna, Przygoda, & Iwanow, 2005).

To minimize the effect of other dietary products with high polyphenol content on the collected data, the subjects were asked to limit fruits, juice, wine, tea, chocolate, and cocoa for 2 days before the muscle-endurance test and to refrain from consuming foods other than meat, egg,
milk products, and water in the evening the day before the test was conducted.

Seven days before the muscle-endurance test, each subject was tested for the measurement of a one-repetition maximum (1-RM) in bench press and back squat, according to a previously described procedure (Cramer & Coburn, 2004). The subjects were asked to cease (discontinue) their training for 2 days before the muscle-endurance test and 24 hr after it.

**Experimental Procedure**

The experimental procedure is displayed in Figure 1. Athletes consumed a standardized breakfast of two rolls with butter, one slice of ham, and 0.75 L of mineral water at the university canteen at 6.00 a.m., 3 hr before they underwent an exercise test. Then, they were randomly assigned, in a double-blind fashion, to receive two dark gelatin capsules containing either green tea polyphenols (GTP group, \( n = 8 \)) or placebo (PL group, \( n = 8 \)). Both the GTP and PL capsules were identical in appearance (i.e., size, shape, and color), and they both were manufactured by Olimp Laboratories (Debica, Poland). The capsules with GTP were commercially available as Olimp green tea (Olimp Laboratories, Debica, Poland). Each GTP capsule contained standardized green tea extract (total of 320 mg polyphenols, including about 250 mg catechins, of which about 176 mg was epigallocatechin-3-galate—EGCG) and additional substances (maltodextrin, microcrystalline cellulose, magnesium stearate). Thus, two capsules delivered 640 mg of polyphenols (including 500 mg of catechins). PL capsules contained microcrystalline cellulose, magnesium stearate, and maltodextrin instead of GTP. The capsules were administered 2 hr after the breakfast (at 8.00 a.m.).

One and a half hours after taking the capsules (at 9:30 a.m.), athletes performed the muscle-endurance test (according to the protocol shown in Figure 2), using a previously described procedure (Schmitz, Hofheins, & Lemieux, 2010) that was modified for the purposes of this experiment. The test consisted of three sets of two strength exercises (bench press, back squat) performed to exhaustion, with a load of 60% 1-RM and 1-min rests between sets. The exercise protocol was performed using a bar and free weights. Before the test, athletes carried out a warm-up consisting of bench press and back squat, both performed in one set of five repetitions with loads of 40% 1-RM.

The 90-min interval between capsule consumption and the muscle-endurance test was adapted from the studies of Lee et al. (2002), where the highest plasma catechin content was observed 1.3–1.6 hr after the administration of a single dose of green tea (394 mg polyphenols).

**Blood Sampling and Biochemical Analysis**

Venous blood samples were drawn into heparinized test tubes from an ulnar vein before the muscle-endurance test (preexercise), 1.5 hr after oral administration of GTP and PL capsules; 5 min after completing the muscle-endurance test (postexercise); and after the 24-hr recovery period (recovery). Blood samples were centrifuged (for 10 min at 3,000 \( g \) at 4 °C) to separate red blood cells from plasma. Erythrocytes were washed three times with a cold isotonic saline solution. Both erythrocytes and plasma were frozen at –80 °C for later analysis.

In addition, capillary blood samples were taken from a finger prick before the muscle-endurance test and 3 min after completing the test. Capillary blood was assayed for the concentration of lactate, as well as for parameters of acid-base equilibrium.

Red blood cells were analyzed for the activity of SOD by using a commercially available kit (RanSOD, 2002).
and areas of chromatographic peaks, taking into account their retention times, as well as the ratio of the peak area for the dominating electrode to that of neighboring electrodes. Total plasma catechin concentration was expressed as ng/ml.

Lactate levels in the capillary blood were determined using a diagnostic cuvette kit (Dr. Lange, Cat. No. LKM 140, Germany) and Miniphotometer Plus LP 20 (Hach Lange, Germany). Parameters of acid-base equilibrium in capillary blood, as pH, base deficiency, and anion gap, and hematocrit were evaluated with the use an automated analyzer (OMNI-C, Roche Diagnostics, Austria). All samples were corrected for plasma volume shift according to the method of Dill and Costill (1974).

**Statistical Analysis**

Statistical analysis was performed with the Statistica v. 6.0 software package. Comparisons between groups regarding dietary intakes, subject characteristics, and results of the exercise test were made by applying an unpaired Student’s t test. Biochemical-parameter data were analyzed using two-way mixed ANOVA with two independent variables: group (GTP vs. placebo) and time (pre- vs. postexercise vs. 24-hr recovery). If significant differences in ANOVA tests were observed, data were analyzed with a near-infrared post hoc test for multiple comparisons. All values were reported as M ± SD. The level of statistical significance was set at p < .05.

**Results**

The physical characteristics of the athletes and results obtained in the muscle-endurance test are shown in Table 1. Subjects in both groups were similar in regard to mean age, height, body mass, body-mass index, and years of training. In addition, results obtained in the muscle-endurance test—total number of repetitions in three sets of bench press or back squat—did not vary significantly between the two groups (Table 2). Similarly, exercise volume (i.e., total kilograms lifted in three sets of bench press or back squat) did not differ significantly.

Table 3 presents the daily energy intake and dietary composition (mean intakes of protein, fat, carbohydrates, and antioxidant vitamins in daily food rations of participants). No significant difference was found between GTP and PL groups for any of the variables studied. The diet in both groups was adequate in terms of the dietary recommendations for physically active men (Ziemlanski, 2001).

As shown in Table 4, the muscle-endurance test performed by soccer players induced significant changes in measured parameters of blood acid-base equilibrium (a drop in pH and increases in base deficiency and anion gap). These disturbances clearly pointed to metabolic acidosis as a result of increased lactic acid production during the exercise (postexercise lactate concentration amounted to 14.70 ± 2.59 and 15.80 ± 1.44 mmol/L in GTP and PL, respectively). However, a single dose of green tea polyphenols ingested before the test did not affect these changes.
As shown in Figure 3, acute GTP intake increased preexercise plasma total catechin level (by 33% compared with the corresponding value in the PL group; group main effect, \( p < .05 \), and Group \( \times \) Time interaction, \( p < .05 \)). In the PL group, no significant changes in plasma catechins were observed after the muscle-endurance test. However, a significant decrease in plasma catechins was observed in the GTP group after 24 hr of recovery (decrease of about 22% compared with the preexercise value, \( p < .001 \), and 10%, compared with the postexercise value, \( p < .05 \); time main effect, \( p < .05 \)).

Changes in the markers of oxidative stress are presented in Table 5. The muscle-endurance test elevated lipid peroxidation as indicated by significant increases in comparison with preexercise values in postexercise plasma TBARS, which remained elevated 24 hr later (time main effect, \( p < .001 \)).

Similarly, plasma TAS and UA alterations (Table 5) were observed in response to the muscle-endurance test (time main effect, \( p < .001 \)). Both plasma TAS and UA increased postexercise and remained elevated compared with preexercise level until 24 hr after the test.

No significant changes in SOD activity in erythrocytes (Table 5) were observed in either group as a result of the muscle-endurance test (time main effect, \( p > .05 \)). However, the PL group exhibited a tendency for a significant increase in SOD activity at 5 min postexercise (\( p = .0503 \)) compared with resting activity.

Plasma CK activity did not change significantly immediately postexercise (Figure 4), but it increased after 24 hr of recovery (time main effect, \( p < .001 \)). In the PL group, after the 24-hr recovery period CK activity was higher than preexercise and postexercise values, whereas in the GTP group, CK activity was higher than the preexercise value only.

Apart from increased preexercise plasma total catechin level as a result of acute GTP ingestion, no significant effect of GTP intake on plasma TBARS, TAS, and UA concentrations was found. Similarly, a single dose of GTP did not affect SOD activity in erythrocytes or plasma CK activity (\( p > .05 \) for group main effect and Group \( \times \) Time interaction in all mentioned parameters).

### Table 1 Characteristics of Soccer Players, \( M \pm SD \)

<table>
<thead>
<tr>
<th>Parameter</th>
<th>PL ((n = 8))</th>
<th>GTP ((n = 8))</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>22.9 ± 5.5</td>
<td>22.4 ± 3.4</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>180.9 ± 4.4</td>
<td>183.3 ± 5.6</td>
</tr>
<tr>
<td>Body mass (kg)</td>
<td>77.6 ± 3.6</td>
<td>78.7 ± 7.4</td>
</tr>
<tr>
<td>Body-mass index (kg/m²)</td>
<td>23.8 ± 1.7</td>
<td>23.4 ± 1.4</td>
</tr>
<tr>
<td>Years of training</td>
<td>9.3 ± 3.7</td>
<td>9.3 ± 1.9</td>
</tr>
</tbody>
</table>

Note. PL = placebo group; GTP = green tea polyphenol group.

### Table 2 Results Obtained in the Muscle-Endurance Test by Soccer Players, \( M \pm SD \)

<table>
<thead>
<tr>
<th>Parameter</th>
<th>PL ((n = 8))</th>
<th>GTP ((n = 8))</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total repetitions in 3 series</td>
<td></td>
<td></td>
</tr>
<tr>
<td>bench press</td>
<td>43.0 ± 9.9</td>
<td>50.7 ± 11.2</td>
</tr>
<tr>
<td>back squat</td>
<td>46.9 ± 6.1</td>
<td>53.7 ± 12.3</td>
</tr>
<tr>
<td>Total kilograms lifted in 3 series</td>
<td></td>
<td></td>
</tr>
<tr>
<td>bench press</td>
<td>2,053.6 ± 410.9</td>
<td>2,427.1 ± 364.5</td>
</tr>
<tr>
<td>back squat</td>
<td>2,686.4 ± 476.7</td>
<td>3,047.1 ± 607.2</td>
</tr>
</tbody>
</table>

Note. PL = placebo group; GTP = green tea polyphenol group.

### Table 3 Daily Nutritional Intake in PL and GTP Groups, \( M \pm SD \)

<table>
<thead>
<tr>
<th>Parameter</th>
<th>PL ((n = 8))</th>
<th>GTP ((n = 8))</th>
</tr>
</thead>
<tbody>
<tr>
<td>Energy (kcal)</td>
<td>3,611.4 ± 315.1</td>
<td>3,822.2 ± 304.3</td>
</tr>
<tr>
<td>Protein (g)</td>
<td>119.7 ± 13.9</td>
<td>122.9 ± 15.1</td>
</tr>
<tr>
<td>Carbohydrate (g)</td>
<td>492.1 ± 57.8</td>
<td>520.4 ± 36.7</td>
</tr>
<tr>
<td>Fat (g)</td>
<td>109.2 ± 31.1</td>
<td>116.7 ± 52.9</td>
</tr>
<tr>
<td>Vitamin C (mg)</td>
<td>71.6 ± 14.6</td>
<td>75.2 ± 17.0</td>
</tr>
<tr>
<td>Vitamin E (mg)</td>
<td>9.9 ± 1.9</td>
<td>11.3 ± 2.5</td>
</tr>
<tr>
<td>Vitamin A (RE)</td>
<td>1,264.9 ± 309.1</td>
<td>1,358.4 ± 372.6</td>
</tr>
</tbody>
</table>

Note. PL = placebo group; GTP = green tea polyphenol group; RE = retinol equivalents.

### Table 4 Changes in Blood pH, Base Deficiency, Anion Gap, and Lactate Concentration Induced by a Muscle-Endurance Test Performed After Ingestion of Placebo or Green Tea Polyphenols in Soccer Players, \( M \pm SD \)

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Placebo ((n = 8))</th>
<th>Green Tea Polyphenols ((n = 8))</th>
</tr>
</thead>
<tbody>
<tr>
<td>Blood pH</td>
<td>7.40 ± 0.02</td>
<td>7.39 ± 0.02</td>
</tr>
<tr>
<td>Base deficiency (mmol/L)</td>
<td>0.56 ± 0.95</td>
<td>0.93 ± 1.63</td>
</tr>
<tr>
<td>Anion gap (mmol/L)</td>
<td>15.04 ± 1.25</td>
<td>14.57 ± 2.34</td>
</tr>
<tr>
<td>Lactate concentration (mmol/L)</td>
<td>1.51 ± 0.42</td>
<td>1.42 ± 0.69</td>
</tr>
</tbody>
</table>

***Significantly different \((p < .001)\) from the preexercise value.
**Discussion**

The objective of this study was to determine the effect of acute ingestion of green tea polyphenols on blood markers of oxidative stress in soccer players subjected to an intense muscle-endurance test.

Described disturbances of acid-base balance (Table 4) suggest that when the soccer players performed the high-intensity muscle-endurance test it increased peroxidation of membrane lipids, as indicated by elevated TBARS concentration in the plasma. This rise was observed in both groups 5 min postexercise and maintained during the 24-hr recovery period. The TBARS response observed in this study is in agreement with previous investigations involving resistance exercise (three sets of eight exercises at 10-RM) in a group of recreationally weight-trained men (McBride, Kraemer, Triplett-McBride, & Sebastianelli, 1998). In contrast, while using an exercise protocol similar to that applied in the study by McBride et al., Dixon et al. (2006) did not observe any significant changes in plasma level of TBARS, both in resistance-trained and in untrained men. Likewise, Bloomer et al. (2006) and Panza et al. (2008) reported no increase in lipid-peroxidation markers after resistance-type exercise in trained men. The discrepancy in lipid peroxidation between our results and those reported earlier by McBride et al., as well as other findings (Bloomer et al., 2006; Dixon et al., 2006;
Panza et al., 2008), might be due to methodological differences—the type of contraction performed, duration of the protocol (Dixon et al., 2006), intensity and volume of exercises (Bloomer et al., 2006), limited number of muscle groups involved in the exercise (Bloomer et al., 2006), and methodological limitations associated with TBARS analysis (Dixon et al., 2006). However, we used a modified TBARS assay for lipid peroxidation (Jentzsch et al., 1996) recommended to minimize artefactual oxidative degeneration of lipids during the assay and used recently by other workers (Bloomer, Cole, & Fisher-Wellman, 2009).

The enhanced generation of ROS with acute physical exercise evokes changes in the body’s antioxidant defense. There seems to be a general consensus in the literature, supported by the findings in the current study, that plasma antioxidant levels increase after exercise, although opposite results have been reported, as well (Margonis et al., 2007; Skarpanska-Stejnborn, Pilaczynska-Szczesniak, Basta, Deskur-Smielecka, & Horoszkiewicz-Hassan, 2008). Mobilization of the tissue nonenzymatic antioxidant stores into the plasma is a widely accepted phenomenon that helps maintain the antioxidant status of plasma when needed (Margonis et al., 2007). In our study, TAS plasma level increased after the muscle-endurance test and was maintained during the recovery period. Likewise, the concentration of TBARS in plasma after 24 hr of recovery was still higher than the preexercise values. This suggests an enhanced generation of free oxygen radicals as a result of the test performed by the athletes, overwhelming their capacity to detoxify ROS and thus producing oxidative stress. A similar tendency of changes in blood levels of TAS and malondialdehyde was demonstrated by Shing et al. (2007) after high-intensity interval cycling in highly trained cyclists.

UA, a primary end product of purine metabolism, is the principal water-soluble antioxidant present in blood (Rabovsky, Cuomo, & Eich, 2006). It is well known that elevated plasma UA concentration is responsible for one third of the plasma TAS increase after strenuous exercise (Child, Wilkinson, Fallowfield, & Donnelly, 1998). This was confirmed by our study, as the muscle-endurance test affected plasma UA concentrations in the same pattern as plasma TAS levels.

It has been previously demonstrated that ROS generation could induce changes in the activity of antioxidant enzymes in the red blood cells (Fisher-Wellman & Bloomer, 2009). Reports of alterations in erythrocyte SOD activity after exercise are equivocal. Decreases in erythrocyte SOD activity after extreme running competition in well-trained athletes were observed by Machefer et al. (2004). In turn, other works report increased SOD activity in the red blood cells after a half-marathon (Marzatico, Pansarasa, Bertorelli, Somenzini, & Della Halle, 1997) and rowing incremental test (Skarpanska-Stejnborn et al., 2008) or a lack of changes after incremental exercise (Vider et al., 2001) or sprint-running exercise (Marzatico et al., 1997). In the current study, SOD activity in erythrocytes did not change significantly after the muscle-endurance test. However, in the PL group, a tendency for increased SOD activity was seen at 5 min postexercise \( (p = .0503) \), indicating an enhanced generation
of superoxide anion associated with the intense muscle-endurance test.

There is evidence that under conditions of exercise-induced oxidative stress, enhanced peroxidation of membrane lipids is one of the factors leading to changes in the integrity of the cellular membrane (Marzatico et al., 1997). This results in an increase in the postexercise activity of CK in plasma (Branaciaccio, Maffulli, & Limongelli, 2007). Free radicals’ substantial contribution to muscle-tissue damage linked with acute exercise is indicated by a positive correlation between postexercise activity of CK and concentration of TBARS in blood plasma that has been reported by numerous investigations (Urso & Clarkson, 2003). In the current study, the significant increase in plasma CK activity noted 24 hr after the muscle-endurance test in both groups indicates that the exercise-induced oxidative stress disrupted the integrity of muscle-cell membranes. These results are consistent with those of other authors (Dixon et al., 2006; McBride et al., 1998).

Dietary antioxidants are claimed to play a major role in the protection of cells from oxidative damage by improving the body’s defense systems’ handling of free-radical attack and by helping to reduce postexercise elevations in plasma CK activity (Bloomer, Goldfarb, McKenzie, You, & Nguyen, 2004). Green tea is a rich source of catechins—compounds that belong to the polyphenol group. The antioxidative properties of green tea polyphenols were observed in both in vitro (Raza & John, 2007) and in vivo studies (Alexio et al., 2002). In our study, acute ingestion of GTP resulted in a slight but significant increase in total catechin concentration in plasma. Taking into account the results of other studies (Lee et al., 2002; Renouf et al., 2010), the time span used in the current study between the administration of GTP and the preexercise blood-sample collection (1.5 hr) should be sufficient for blood catechins to reach peak concentration. Chow et al. (2005) showed that taking polyphenon E with a meal reduced oral bioavailability of green tea catechins and considerably extended the time required to reach the peak concentration of plasma catechins in comparison with taking polyphenon E on an empty stomach as in an overnight fast. In our study, GTP capsules were administered 2 hr after a light breakfast, so the meal was presumed not to affect catechin absorption in the small intestine. In our work, however, total plasma catechin concentration after ingestion of GTP was consistent with Chow et al.’s observations in a fed condition, but not in a fasting condition. It can therefore not be excluded that, in our study, the meal could have affected the oral bioavailability of GTP and that a period longer than 1.5 hr would be needed to reach the peak concentration of plasma catechins.

In the current study, despite an increase in total plasma catechins, no changes in TAS plasma level after acute ingestion of GTP were observed. This may explain why GTP ingestion did not affect any other parameters of oxidative stress. It is well known that, due their low bioavailability, the contribution of polyphenolic compounds to the antioxidative capacity of blood plasma is restricted (Morillas-Ruiz et al., 2006). For this reason, the dose of green tea polyphenols administered in our study might have been insufficient to modify the whole antioxidant plasmatic state. In the study of Henning et al. (2005), a single intake of polyphenon E containing twice the dose of polyphenols as in our study was insufficient to achieve an increase in plasma antioxidant capacity determined using Trolox equivalents. On the other hand, in another study (Rabovsky et al., 2006), a single administration (to fasted subjects) of a green tea extract in a dose of 600 mg was demonstrated to increase plasma antioxidant capacity, measured as FRAP after enzymatic removal of UA. In our study, acute ingestion of GTP was observed to have no effect on plasma TAS, even when plasma TAS levels were corrected for plasma UA (data not shown). Therefore, it is likely that there is no acute effect of GPT ingestion, and a longer period of GTP supplementation (i.e., 1–7 weeks) is necessary to induce protection against exercise-induced oxidative stress, as revealed by previous studies (Alexio et al., 2002; Panza et al., 2008) and our latest study (Jówko et al., 2011).

It must be emphasized that in the current study the results obtained in the muscle-endurance test (i.e., the number of repetitions and total weight lifted in both exercises) were higher (although this rise was not significant) in the GTP group than in the PL group. Thus, the actual differences between groups, although not statistically significant, may reach physiological significance, which could mask potential effects of GTP ingestion on the parameters of oxidative stress. However, the results of another study (Viitala, Newhouse, LaViole, & Gottard, 2004), in which the effect of vitamin E supplementation on resistance-exercise-induced lipid peroxidation was evaluated in trained and untrained subjects, do not confirm the mentioned hypothesis. In Viitala et al.’s study, a similar magnitude of oxidative stress (plasma malondialdehyde concentration) was observed in all investigated groups (i.e., trained supplemented, trained placebo, untrained supplemented, and untrained placebo), despite significantly higher plasma vitamin E concentration in supplemented than the unsupplemented groups, as well as significantly greater work output in trained than untrained participants (Viitala et al., 2004).

On the other hand, it cannot be excluded that, in our study, the differences in the number of repetitions and total weight lifted between groups resulted solely from acute GTP ingestion. Consequently, the higher volume of work done in the GTP group (though it was not significant), accompanied by postexercise changes in the parameters of acid-base equilibrium and indices of oxidative stress similar to those of the PL group, may have resulted from acute GTP ingestion.

In conclusion, the intense muscle-endurance test employed in the current study was observed to induce oxidative stress and muscle damage in soccer players. A single dose of green tea polyphenols administered before the test failed to suppress the exercise-induced oxidative stress. Presumably, a higher dose of polyphenols may
be required to exert a positive effect on oxidative-stress parameters; this needs to be confirmed by further studies with higher numbers of athletes per group.

References


Ziemlanski, S. (2001). *Norm of man’s nutrition*. Warsaw, Poland: PZWL.