The Influence of Supplementation With *Rhodiola rosea* L. Extract on Selected Redox Parameters in Professional Rowers

Anna Skarpanska-Stejnborn, Lucja Pilaczynska-Szczesniak, Piotr Basta, and Ewa Deskur-Smielecka

The aim of this study was to investigate the effect of *Rhodiola rosea* supplementation on the balance of oxidants and antioxidants in the serum and erythrocytes of competitive rowers. This double-blinded study included 22 members of the Polish Rowing Team who were participating in a preparatory camp. Participants were randomly assigned to the supplemented group (*n* = 11), who received 100 mg of *R. rosea* extract twice daily for 4 wk, or the placebo group (*n* = 11). At the beginning and end of the study, participants performed a 2,000-m maximum test on a rowing ergometer. Blood samples were taken from the antecubital vein before each exercise test, 1 min after completing the test, and after a 24-hr restitution period. The following redox parameters were assessed in erythrocytes: superoxide dismutase activity, glutathione peroxidase activity, and thiobarbituric-acid-reactive substances concentrations. In addition, creatine kinase activity and total antioxidant capacity were measured in plasma samples, lactate levels were determined in capillary blood samples, and uric acid concentrations were measured in serum. After supplementation, the total plasma antioxidant capacity was significantly higher (*p* = .0002) in the supplemented group than in the placebo group, and superoxide dismutase activity in erythrocytes directly after and 24 hr after the ergometry was significantly (*p* = .0461) lower in athletes receiving *R. rosea* extracts than in the placebo group. In conclusion, supplementation with *R. rosea* increased antioxidant levels in the plasma of professional rowers but had no effect on oxidative damage induced by exhaustive exercise.

**Keywords**: supplementation, antioxidants, TBARS, exhaustive exercise

*Rhodiola rosea* (roseroot, golden root) is a perennial plant in the *Crassulaceae* family that grows in the northern regions of Asia, Europe, and North America. It is used in traditional Asian medicine to improve health and quality of life. However, little research has been performed to document the therapeutic properties of *R. rosea*. It has been found that its extracts have antioxidant potential (Bat-
tistelli et al., 2005, De Sanctis et al., 2004) and cardioprotective effects (Maslova, Kondrat’ev, Maslov, & Lishmanov, 1994) and increase physical and mental performance (Abidov, Crendal, Grachev, Seifulla, & Zigenfuss, 2003; Kelly 2001). R. rosea contains a large number of active ingredients, the most important of which are phenylpropanoids (rosavin, rosin, and rosarin), phenolic compounds (salidroside and p-tyrosol), flavonoids, and organic acids (gallic acid; Ganzera, Yayla, & Khan, 2001; Kucinskaite, Sawicki, Briedis, & Szmitowska, 2007).

The antioxidant properties of R. rosea extract have been documented in studies performed on human erythrocytes exposed to hypochlorous acid (a strong oxidizing agent; Battistelli et al., 2005; De Sanctis et al., 2004). Moreover, it has been observed that high doses of R. rosea extract might induce changes in the shape of red blood cells, even in the absence of oxidative stress (Battistelli et al.). Results of some experimental studies suggest that R. rosea might be a valuable dietary supplement for athletes. Abidov et al. (2003) and Azizov and Seifulla (1998) observed improved work capacity in trained animals fed a diet enriched with R. rosea extract. Abidov, Grachev, Seifulla, and Zigenfuss (2004) found that supplementation with R. rosea significantly decreased changes in inflammatory parameters (C-reactive protein) and skeletal-muscle damage markers (creatine kinase) induced by intensive physical exercise. However, these favorable effects have not been confirmed in humans (Colson et al., 2005; Walker, Altobelli, Caprihan, & Robergs, 2007).

Benefits of supplementation with R. rosea in athletes might also depend on its central nervous system stimulant effect. Perfumi and Mattioli (2007) found that single doses of R. rosea extract induced antidepressant-like, adaptogenic, anxiolytic-like, and stimulating effects in mice. Spasov, Wikman, Mandrikov, Mironova, and Neumoin (2000) observed decreased fatigue and improved perceptual abilities in students exposed to stress associated with work. Psychostimulating effects of R. rosea are attributed to its active ingredients, rosavin and salidroside, which might influence levels and activity of opioid peptides such as beta-endorphin. Beta-endorphin decreases susceptibility to pain and thus prevents excessive reaction of an organism to stress (Maslova et al., 1994).

Because the outcomes of studies on R. rosea’s effects are incomplete and ambiguous, we investigated the influence of 4 weeks of supplementation with R. rosea extract on selected parameters of oxidant–antioxidant balance in professional rowers performing a maximal 2,000-m ergometry bout.

Materials and Methods

Study Population

The study population consisted of 22 male members of the Polish Rowing Team (13 heavyweight and 9 lightweight rowers). Basic characteristics and sport classes of the athletes are listed in Table 1. The study was performed in May and June during a 4-week training camp between the preparation and competition periods. Data concerning training profile, such as intensity, volume (in minutes), and type (specific to rowing: endurance, technical, speed, etc.; nonspecific: jogging and strength), were recorded daily. All training data were analyzed for intensities below and above the lactate threshold of 4 mmol/L, as shown in Figure 1, and
classified as extensive (below the lactate threshold) or intensive (above the lactate threshold) workload.

Over the entire study period, athletes resided in one of the Olympic Games training centers and took meals exclusively in the center. Their regular menu consisted of a mixed diet containing the recommended dietary allowance of carbohydrates, proteins, fats, and micronutrients (vitamins and minerals). Daily food and caloric intake, as well as fruit and vegetable intake, of the athletes did not change over the study period. The athletes informed the scientific staff that they had not been taking any drugs, medication, or nutritional supplements for 2 weeks before and during the study.

**Experimental Procedure**

Athletes enrolled in the study were randomly assigned to receive *R. rosea* extract (*n* = 11) or placebo (*n* = 11). The rowers in the supplemented group were given Rhodiolin (CaliVita International, USA) twice daily for 4 weeks. One gelatin capsule of Rhodiolin contained 100 mg of *R. rosea* concentrate and 5.0 mg of zinc (zinc contained in Rhodiolin constituted only 33% of the daily intake recommended by the Polish nutritional norms [Ziemlanski, 2001]; the most important source of zinc was athletes’ mixed diet). At the same time, and with the same dosing regimen, participants in the placebo group received dyed gelatin capsules containing a substance with no significant caloric value or biological activity. The antioxidant potentials (ferric-reducing ability of plasma) of water extract, ethanol extract, and a 0.1-M HCl extract of *R. rosea* were assessed with the method of Benzie and Strain (1996) with the modification described by Janaszewska and

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Supplemented group (<em>n</em> = 11)</th>
<th>Control group (<em>n</em> = 11)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Heavyweight (n)</td>
<td>5</td>
<td>8</td>
</tr>
<tr>
<td>Lightweight (n)</td>
<td>6</td>
<td>3</td>
</tr>
<tr>
<td>Age (years)</td>
<td>20.4 ± 1.2</td>
<td>21.0 ± 0.9</td>
</tr>
<tr>
<td>Body mass (kg)</td>
<td>78.6 ± 10.6</td>
<td>83.3 ± 7.9</td>
</tr>
<tr>
<td>Body height (cm)</td>
<td>189.4 ± 8.5</td>
<td>191.5 ± 7.5</td>
</tr>
<tr>
<td>Years of training</td>
<td>6.0 ± 1.6</td>
<td>5.9 ± 1.5</td>
</tr>
<tr>
<td>Fat-free mass (kg)</td>
<td>15.2 ± 3.5</td>
<td>17.7 ± 2.6</td>
</tr>
<tr>
<td>Fat-free mass (%)</td>
<td>12.0 ± 3.9</td>
<td>15.5 ± 4.9</td>
</tr>
<tr>
<td>Sport class* (n)</td>
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<td></td>
</tr>
<tr>
<td>international master class</td>
<td>3</td>
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</tr>
<tr>
<td>country master class</td>
<td>3</td>
<td>2</td>
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<tr>
<td>Class I</td>
<td>5</td>
<td>6</td>
</tr>
</tbody>
</table>

*Sport classes in Poland: Competitive rowers are classified according to the regulations of the Minister of Sport and Tourism of the Republic of Poland and the Polish Rowing Association.*
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Bartosz (2002). This method was based on ferric- to ferrous-ion reduction by nonenzymatic antioxidants contained in the assessed sample. The amount of ferrous ions was determined in the reaction with tripyridyltriazine. The antioxidant potentials of *R. rosea* extract were 56.7 μmol/L (water extract) to 131.5 μmol/L (0.1 M HCl extract, pH = 1). Total polyphenol content was assessed with the method of Singleton and Rossi (1965) and varied from 0.012 to 0.028 g/L.

All participants were informed of the nature of the investigation and gave their written informed consent to participate in the study. The Ethics Committee at the University School of Medical Sciences in Poznan approved the study protocol.

On the first day (before supplementation) and at the end of the training camp (after supplementation), the athletes performed a controlled 2,000-m rowing

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**Figure 1** — Training schedule in the week preceding blood-sample collection before and after the supplementation period (volume in minutes per day).
exercise. Each participant had to cover the distance on a rowing ergometer (Concept II, USA) in as short a time as possible. Because the results of both tests were taken into consideration during selection of athletes to the championship team, the athletes were well motivated to perform both tests with all-out effort. Before each test, participants performed a 5-min individual warm-up.

Sample Treatment

Blood samples were taken from the antecubital vein, using dipotassium ethylene diamine tetra-acetic acid as an anticoagulant, before each 2,000-m test (in the morning, after an overnight fast), 1 min after completing the test, and after a 24-hr recovery period. Samples were centrifuged immediately to separate red blood cells from plasma. Packed erythrocytes were washed three times with saline and lysed with ice-cold, redistilled water. Plasma, serum, and lysed erythrocytes were frozen immediately and stored at –28 °C until use (up to 1 week). In addition, capillary blood samples were taken by finger prick before and after each exercise test to assess the lactate levels.

Measurements

Total antioxidant capacity (TAC), used as an overall measure of plasma antioxidant capacity, was assessed with a commercially available kit (Randox-TAS, Cat. No. NX 2332, UK). This assay was based on the interaction between a chromogen (2,2ʹ-azino-di-[3-ethylbenzthiazoline sulphonate]) and ferrylmyoglobin, a free radical formed in the reaction between metmyoglobin and hydrogen peroxide.

Superoxide dismutase (SOD) activity was measured in washed erythrocytes after their lysis by means of a commercially available kit (Randox-Ransod, Cat. No. SD 125). SOD catalyzes dismutation of superoxide anion (O₂⁻), leading to the formation of oxygen and hydrogen peroxide. The determination of SOD activity was based on the production of O₂ by the xanthine and xanthine oxidase system. SOD activity was expressed in U/g Hb.

Glutathione peroxidase (GPx) activity in the hemolysate samples was measured using a commercially available kit (Randox-Ransel, Cat. No. RS 506). GPx catalyzes the oxidation of reduced glutathione in the presence of cumene hydroperoxide. The rate of glutathione oxidation was measured by monitoring the disappearance of NADPH+H⁺ in the reaction medium. GPx activity was expressed in U/g Hb.

Concentrations of thiobarbituric-acid-reactive substances (TBARS) in the hemolysate samples were assessed as a measure of oxidative damage to red blood cells. TBARS concentrations were evaluated with the method described by Buege and Aust (1991), involving the acidic breakdown of lipid peroxides into malondialdehyde molecules. Malondialdehyde subsequently reacted with thiobarbituric acid, producing substances suitable for spectrophotometric detection. The tested samples contained 0.025 ml of hemolysate, 0.5 ml of thiobarbituric acid solution (0.375 g/100 ml in 0.25 mol/L hydrochloric acid), 0.5 ml of trichloroacetic acid solution (15 g/100 ml trichloroacetic acid in 0.25 mol/L hydrochloric acid), and 0.475 ml of water. The blank samples contained 0.025 ml of hemolysate, 0.5 ml of trichloroacetic acid solution, and 0.975 ml of water. All samples were mixed
vigorously and heated for 15 min in boiling water. Next, they were cooled in ice-cold water and centrifuged at 2,500 g for 15 min. The absorbance of the supernatant was determined at 535 nm and 37 °C. The absorbance of the blank sample was subtracted from the absorbance of the tested sample, and the concentrations of TBARS were determined from a standard curve generated with known amounts of tetramethoxypropane. The concentrations of TBARS (malondialdehyde equivalents) were expressed in µmol/g Hb.

Uric acid (UA) levels were assessed with a commercially available kit (Alpha Diagnostics, Cat. No. K6681-100). The results were expressed in mg/dl.

Creatine kinase activity was determined in plasma samples with a commercially available kit (Dr Lange, Cat. No. LCN 282, Germany). The results were expressed in U/L.

Concentration of hemoglobin in hemolysate was assessed using the cyanmethemoglobin method with Drabkin’s reagent and maximal absorbance at 540 nm. The results were expressed in g/100 ml.

The lactate levels in capillary blood were determined immediately after collection of samples using a commercially available kit (Dr Lange, Cat. No. LKM 140). The lactate concentration was expressed in mmol/L.

**Statistical Analysis**

Statistical analyses were performed with the Statistica v. 7.0 software package. The data were compared using 2 (supplemented and placebo groups) × 3 (times of measurement) repeated-measures analysis of variance (ANOVA). In case significant changes in ANOVA tests were observed, data were additionally analyzed with paired and unpaired Student’s t tests and with Scheffé’s post hoc test for multiple comparisons. All values were reported as $M \pm SD$. Statistical significance was set at $p < .05$.

**Results**

The characteristics of participants in the two groups were similar with respect to mean age, height, body mass, fat-free mass, and years of training (Table 1).

Blood lactate levels, mean power output, and total run time during the 2,000-m test performed at the beginning of the training camp did not differ between the study groups; the results of the 2,000-m test did not change significantly after the training camp (Table 2).

Training volumes in minutes per day during the week preceding the first term of assessment (before supplementation) and during the week preceding the second term of assessment (after supplementation), specified for extensive rowing, intensive rowing, and extensive nonspecific training, are shown in Figure 1. In the load-training phase (before the first assessment), the training volume amounted to 950 min/week, of which about 80% was extensive rowing and the rest was nonspecific training such as power training. Total training time before the second assessment was 980 min/week and consisted of approximately 67% rowing at intensities below lactate levels of 4 mmol/L, 12% high-intensity rowing, and 21% land training.
Table 3 shows the comparative analysis of endogenous antioxidant potential parameters. The ANOVA indicates that physical exercise significantly influenced changes in these parameters directly after ergometry and during recovery. Ergometry performed at the beginning of the study yielded similar changes in the antioxidant potential parameters in both groups. After 4 weeks of supplementation with *R. rosea* extract, there were significant effects of *R. rosea* (*p* = .461) and *R. rosea* × Exercise (*p* = .0127) for SOD activity. As shown in a post hoc analysis, SOD activity was significantly lower at postexercise and recovery in the supplemented group than in the placebo group (*p* < .05). In contrast, GPx activity was not significantly affected by the supplementation and did not change significantly over the exercise period.

ANOVA showed a significant interaction effect of exercise on serum UA levels (*p* < .001). At the beginning of the study, UA concentrations immediately after ergometry and after 24-hr restitution were significantly higher in both groups than before exercise. After supplementation, UA levels 24 hr after the exercise test were significantly higher than before the test in both groups.

There were significant effects of *R. rosea* treatment (*p* = .0002) and exercise (*p* = .0046) on plasma TAC (Table 3). This effect was mainly the result of a significantly higher plasma TAC before and after exercise and after a 24-hr restitution in the supplemented group than in the placebo group.

ANOVA showed no significant interaction effect between supplementation and exercise on plasma creatine kinase activity (Table 4). No significant interaction effect of *R. rosea* on TBARS was observed (Table 4). After supplementation, the preexercise levels of TBARS were higher (by 46%) than before supplementation in the control group. Moreover, exercise caused a significant increase in oxidative-stress parameters immediately after exercise both before (by 83% in the supplemented group and 118% in the placebo group) and after supplementation (77% in the supplemented group and 75% in the placebo group). Increased levels of lipid peroxidation markers persisted during restitution.
<table>
<thead>
<tr>
<th>Parameter</th>
<th>Before Supplementation</th>
<th>After Supplementation</th>
<th>ANOVA</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Preexercise M±SD</td>
<td>Postexercise M±SD</td>
<td>Recovery M±SD</td>
</tr>
<tr>
<td>SOD (U/g Hb)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Su</td>
<td>1,758.1 ± 261.78</td>
<td>2,161.8 ± 369.24†</td>
<td>2,226.3 ± 349.43†</td>
</tr>
<tr>
<td>Pla</td>
<td>1,672.9 ± 139.83</td>
<td>2,304.8 ± 423.84†</td>
<td>2,208.1 ± 428.84†</td>
</tr>
<tr>
<td>GPx (U/g Hb)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Su</td>
<td>55.1 ± 5.36</td>
<td>57.9 ± 4.48</td>
<td>59.2 ± 5.51</td>
</tr>
<tr>
<td>Pla</td>
<td>57.3 ± 7.72</td>
<td>61.3 ± 5.31</td>
<td>64.1 ± 10.52</td>
</tr>
<tr>
<td>UA (mmol/L)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Su</td>
<td>0.28 ± 0.04</td>
<td>0.44 ± 0.09†</td>
<td>0.51 ± 0.08†</td>
</tr>
<tr>
<td>Pla</td>
<td>0.28 ± 0.06</td>
<td>0.47 ± 0.09†</td>
<td>0.51 ± 0.17†</td>
</tr>
<tr>
<td>TAC (mmol/L)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Su</td>
<td>1.17 ± 0.08</td>
<td>1.19 ± 0.20</td>
<td>1.02 ± 0.07</td>
</tr>
<tr>
<td>Pla</td>
<td>1.14 ± 0.08</td>
<td>1.25 ± 0.22</td>
<td>1.08 ± 0.12</td>
</tr>
</tbody>
</table>

*Note. SOD = superoxide dismutase; GPx = glutathione peroxidase; n.s. = nonsignificant; UA = uric acid; TAC = total antioxidant capacity. p < .05 relative to Pla group. †p < .05 relative to preexercise.
Table 4  Markers of Free Radical Production and Muscle Damage in the Supplemented (Su) and Placebo (Pla) Groups Before and After Supplementation With *Rhodiola Rosea*, Before, Immediately After, and After Recovery From Exercise

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Preexercise $M \pm SD$</th>
<th>Postexercise $M \pm SD$</th>
<th>Recovery $M \pm SD$</th>
<th>Preexercise $M \pm SD$</th>
<th>Postexercise $M \pm SD$</th>
<th>Recovery $M \pm SD$</th>
<th>ANOVA Exercise</th>
<th>ANOVA R. rosea</th>
<th>ANOVA Exercise × R. rosea</th>
</tr>
</thead>
<tbody>
<tr>
<td>CK (U/L)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>n.s.</td>
<td>n.s.</td>
<td>n.s.</td>
</tr>
<tr>
<td>Su</td>
<td>$150 \pm 71$</td>
<td>$250 \pm 133$</td>
<td>$183 \pm 101$</td>
<td>$151 \pm 96$</td>
<td>$200 \pm 93$</td>
<td>$147 \pm 83$</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pla</td>
<td>$139 \pm 73$</td>
<td>$200 \pm 85$</td>
<td>$158 \pm 46$</td>
<td>$154 \pm 63$</td>
<td>$224 \pm 116$</td>
<td>$169 \pm 117$</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>TBARS (µmol/g Hb)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Su</td>
<td>$1.2 \pm 0.49$</td>
<td>$2.2 \pm 0.55^{\dagger}$</td>
<td>$2.5 \pm 0.50^{\dagger}$</td>
<td>$1.3 \pm 0.65$</td>
<td>$2.3 \pm 0.38^{\dagger}$</td>
<td>$2.2 \pm 0.32^{\dagger}$</td>
<td>&lt;.0001</td>
<td>n.s.</td>
<td>n.s.</td>
</tr>
<tr>
<td>Pla</td>
<td>$1.1 \pm 0.59$</td>
<td>$2.4 \pm 0.48^{\dagger}$</td>
<td>$2.5 \pm 0.82^{\dagger}$</td>
<td>$1.6 \pm 0.69$</td>
<td>$2.8 \pm 0.73^{\dagger}$</td>
<td>$2.7 \pm 0.71^{\dagger}$</td>
<td></td>
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</tbody>
</table>

Note. CK = creatine kinase; TBARS = thiobarbituric-acid-reactive substances.

$^{\dagger}p < .05$ relative to preexercise.
Discussion

We examined changes in parameters describing the balance between oxidants and antioxidants in professional rowers performing a 2,000-m maximal bout of ergometry at the beginning and end of a 4-week preparatory camp. We observed a significant increase in lipid peroxidation products after exercise on both occasions. The magnitude of changes in lipid peroxidation products in athletes receiving *R. rosea* was similar to that observed in the placebo group (Table 4). There was a high correlation between TBARS concentrations and lactate levels (before supplementation $r = .77$, $p < .001$; after supplementation $r = .61$, $p < .001$). High lactate levels after the ergometry indicate an important contribution of anaerobic metabolism in the energy expenditure (Table 2). Under hypoxic conditions, degradation of ATP exceeds its synthesis, which results in the accumulation of AMP. AMP is further metabolized to inosine and hypoxanthine. After exhaustive exercise, large amounts of hypoxanthine are released from skeletal muscles into the blood. Hypoxanthine is taken up by the liver and metabolized to xanthine and UA. During ischemia, xanthine dehydrogenase converts to xanthine oxidase, an enzyme that is an important source of reactive oxygen species. Gomez-Cabrera et al. (2005) found that the xanthine oxidase inhibitor allopurinol significantly reduced oxidative damage in rats subjected to intensive physical training.

Ip, Che, and Leung (2001) suggest that *R. rosea*, because of its antioxidant properties, might prevent ischemic damage in two ways: first, by increasing the intracellular availability of oxygen and more efficiently utilizing oxygen, thus reducing the shortage of oxygen during ischemia, and second, by decreasing the activity of reactive oxygen species and enhancing the endogenous antioxidant system, thus limiting damage caused by ischemia-induced oxidative processes.

In our study, SOD activity after ergometry was significantly lower in the athletes receiving *R. rosea* extract (Table 3). This finding suggests more effective elimination of superoxide from red blood cells in the supplemented group, which might support the view of Ip et al. (2001). GPx activity did not change after exercise (Table 3). The affinity of this enzyme for hydrogen peroxide is higher than that of catalase, so GPx might play a more important role when there are low H$_2$O$_2$ concentrations (Powers, Ji, & Leewenburgh, 1999). Decreased SOD activity after the ergometry was not associated with increased damage to red blood cell lipids. Similar to our findings, Wing et al. (2003) did not observe an improvement in oxidative-stress parameters after supplementation with *R. rosea* in humans exposed to hypoxia simulated to be like that at 4,600 m above sea level.

Oxidative stress induced by ergometry was associated with changes in TAC. TAC is part of a nonenzymatic antioxidant system that protects cells and tissues against damage from excessive amounts of reactive oxygen species. In both study groups, TAC increased significantly after the exercise test performed at the end of the preparatory camp (increases in TAC after ergometry performed at the beginning of the study were not significant), which was probably a result of increased levels of UA (Table 3). Positive correlations between UA concentrations and TAC levels after supplementation ($r = .41$, $p < .013$) support this hypothesis. UA, an end product of purine metabolism, is one of the most potent antioxidants in plasma (Kaur & Halliwell, 1990). Its relative contribution to the total antioxidant activity of plasma has been estimated at 35–65% (Wayner, Burton, Ingold, Barclay, &
Halliwell and Gutteridge (1990) observed that particular antioxidants are “used up” in a specific order during oxidative stress. Ascorbate and protein thiol groups disappear first, followed by bilirubin and UA and finally by α-tocopherol. Cooperation between particular antioxidants is also an important factor. Vitamin E reacts with superoxide in the lipid phase to form a low-reacting tocopheryl radical, but it is not involved in the aqueous phase, when other antioxidants (ascorbic acid, UA, and glutathione) play a major role (Niki, 1991).

In the supplemented group, TAC values before, directly after, and 24 hr after maximal ergometry were significantly higher after supplementation with *R. rosea* extract than during the initial examination (Table 3), providing an indication of the antioxidant potential of *R. rosea* in humans. In a study on human erythrocytes exposed to oxidative stress, Battistelli et al. (2005) found that *R. rosea* had higher antioxidant potential than ascorbic acid and that it prevented both extracellular and intracellular oxidative damage. In contrast, we did not observe a decrease in lipid peroxidation markers in the supplemented group.

During oxidative stress induced by intensive exercise, UA might be taken up by skeletal muscle and used as an antioxidant (Hellsten, Tullson, Richter, & Bangsbo, 1997; Hellsten-Westling, Kaijser, Ekblom, & Sjödin, 1994). In accordance with these findings from experimental studies, we observed a correlation between TBARS levels and UA concentrations in rowers before (*r* = .60, *p* < .001) and after (*r* = .49, *p* < .001) supplementation with *R. rosea* extract.

It is well documented that intensive exercise induces hemolysis, increasing plasma free-iron levels (Córdova Martínez, Villa, Aguiló, Tur, & Pons, 2006; Vollaard et al. 2005). Free iron acts as a catalyst in reactions involving free radicals. This phenomenon is probably responsible for the enhancement of oxidative stress during restitution. Molecular oxygen additively binds to Fe^{2+} in heme groups without oxidizing this ion, which is why hemoglobin can reversibly bind oxygen. However, in particle Hb-Fe^{+2}-O_{2}, the shift of an electron from Fe^{+2} toward the oxygen molecule is possible, leading to formation of methemoglobin containing Fe^{+3} and O_{2}^{•−}. In Fenton’s reaction, Fe^{+2} reacts with H_{2}O_{2}, forming an extremely reactive hydroxyl radical. Therefore, binding of transition metal ions (including Fe^{+2}) might prevent the generation of free radicals and limit oxidative damage. Zunquin et al. (2006) observed that iron supplementation in rats performing intensive physical exercise decreased the antioxidant potential. Dietary supplementation with casein, an agent that chelates iron ions, resulted in a decrease in TBARS levels in liposomes. In our previous studies (Pilaczynska-Szczesniak, Skarpanska-Stejnborn, Deskur, & Basta, 2005; Skarpanska-Stejnborn, Pilaczynska-Szczesniak, Deskur-Smielecka, & Basta, 2005) we observed decreased TBARS levels in athletes supplemented with substances containing iron-chelating agents (flavonoids and polyphenols). Data reported in the literature indicate that *R. rosea* has no potential to chelate or remove metal ions, which was further confirmed by our analysis of the iron-reducing activity of *R. rosea* with the ferric-reducing ability of plasma assay. In contrast to the results of Abidov et al. (2004), supplementation of rowers with *R. rosea* extracts did not influence the activity of creatine kinase.

Data concerning the influence of *R. rosea* on physical capacity are ambiguous. Unlike classical stimulants of the central nervous system, *R. rosea* is regarded as an adaptogen, and its use should not be associated with a marked decrease in physical capacity after a short, initial period of improvement. Panossian, Wilkman,
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and Wagner (1999) observed a favorable effect of *R. rosea* extract on physical work capacity. In contrast, Earnest et al. (2004) found no improvement in capacity in cyclists after a 2-week supplementation with *Cordyceps sinensis* and *R. rosea* extracts. They attributed the lack of effects to the short supplementation period. De Bock, Eijnde, Remaeckes, and Hespel (2004) observed an increase in $\text{VO}_2\text{max}$ of 5% in students who received a single dose of *R. rosea* (200 mg), although muscle-strength parameters remained unchanged. Prolonged administration of *R. rosea* extract (28 days) at the same dose was not associated with further improvement. In our study, we observed no influence of a 4-week supplementation with *R. rosea* extracts on the results (time and power) of the 2,000-m ergometry (Table 2).

In conclusion, supplementation with *R. rosea* increased antioxidant levels in the plasma of professional rowers without affecting the amount of oxidative damage caused by intensive exercise or the physical performance of the athletes. Therefore, supplementation with *R. rosea* in athletes to prevent exercise-induced oxidative stress is unjustified. Antioxidants with the potential to chelate iron ions might be more effective in this application.

**References**


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