Changes in blood antioxidant status in American football players and soccer players over a training macrocycle

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1. Introduction

Soccer (association football) and American football are speed-and-endurance team sports involving physical contact between the players and requiring them to perform acyclical movements in response to the ever-changing game circumstances. The two sports are very similar in many ways, for instance, in the number of players, the field dimensions, and the structure of the training macrocycle. In soccer, it starts with a general conditioning (preparatory) period divided into an adaptation phase and a phase aimed to strengthen players' motor skills and aerobic capacity. The training macrocycle in American footballers also starts with the adaptation phase, but the focus of the next phase is on building players' maximum strength and muscle mass. The specialist training phases preceding the competition period are similar. Both emphasize technical exercises, with American footballers additionally undergoing aerobic training. The competition periods are also much the same. In both sports, the volumes of general conditioning exercises are then reduced in favor of targeted and specialist exercises and high-intensity aerobic and speed/speed-

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endurance exercises. In the transition periods concluding the training macrocycle, training volume and intensity are decreased so that more time is available for specialist exercises addressing individual players’ technical weaknesses.4

As has been observed, the response of the antioxidant defense system to physical effort varies depending on exercise type, intensity, volume, and duration.7–10 A single session of prolonged or high-intensity exercise can lead to oxidative damage and skeletal muscle injury,11 but regular sessions involving moderate-intensity exercises promote the production of reactive oxygen and nitrogen species (RONS) that improve endurance and the ability to generate muscle strength.12–14 Exercise-induced RONS can also make the endogenous protective mechanisms more effective in lessening of oxidative stress and muscle damage.

Oxidative stress is an adverse physiological reaction that activates blood antioxidant defense responsible for maintaining and restoring prooxidant-antioxidant balance. The defense consists of two complementary mechanisms, one of which uses enzymes such as superoxide dismutase (SOD), catalase (CAT), glutathione peroxidase (GPx), and glutathione reductase (GR) to prevent the initiation and propagation of a chain reaction leading to the generation of free radicals. The other mechanism makes use of reduced glutathione (GSH) and uric acid (UA). All the compounds prevent the peroxidation of lipids and proteins by reacting with free radicals or free-radical oxidation products. It has been found that dietary antioxidants such as vitamins A, E, and C, selenium, and polyphenols also help prevent damage due to oxidative stress.11

The monitoring of the markers of prooxidant-antioxidant balance in the athletes gained in popularity following the discovery that its disturbance may lead to the overtraining syndrome or activate a signal pathway leading to post-exercise adaptive changes and super-compensation.15–19

Because of many similarities between American football and soccer and a limited number of studies investigating the biochemical aspects of American footballers’ training, this study was undertaken to assess and compare changes in the blood antioxidant defense of American footballers and soccer players over the training macrocycle based on the activity of the main antioxidant enzymes and the concentrations of selected non-enzymatic antioxidants and oxidative stress markers.

2. Methods

2.1. Participants

American footballers (AF, n = 11) and soccer players (SP, n = 11) participating in the study were recruited from the same teams competing in the first (the second highest) leagues. The exclusion criteria included the use of tobacco, alcohol, any medicines, or antioxidant supplements in the four preceding weeks. The main criteria of the study participants are presented in Table 1.

All participants were informed about the protocol and aim of the study and consented in writing to participate in it. The study protocol conformed to the ethical guidelines of the World Medical Association’s Declaration of Helsinki and was approved by the Institutional Ethics Committee at the Jerzy Kukuczka Academy of Physical Education, Poland (certificate No. 4/2013).

In order to assess changes in the blood oxidant status of the participants over the training macrocycle, biochemical measurements were performed at the start of each of the three periods making up the macrocycle, i.e., preparatory (PP), competition (CP), and transition (TP) (Table 2).

2.2. Training programs

The training programs of the AF and SP groups, which followed the general training principles, were aimed to help the players achieve the target level of performance (Table 2). In the preparatory period, the SP had 8–10 training sessions of 90 min in a week and played one test game while the AF trained 4–5 times in a week for 120–180 min in the gym and on the field. In the competition periods (running from the mid of the second week in March to the end of the second week in soccer and from the third week in April to the end of the second week in June in American football), the SP trained 6–8 times per week for 90 min and played 15 league games, and the AF had 3–4 training sessions per week of between 60 and 150 min and played 8 games. In the transition periods (2 weeks and 4 weeks, respectively), the number of specific exercises and training volumes were lower compared with the two previous periods.

2.3. Biochemical analysis

At the start of each of the macrocycle periods, resting venous blood samples were taken from the participants and immediately assayed for reduced glutathione (GSH) by a calorimetric method20 with 5,5′-dithiobis-2-nitrobenzoic acid. The remaining blood was centrifuged at 1,000 g for 10 min at 4 °C to separate plasma and erythrocytes, which were then washed three times with cold (4 °C) saline and kept frozen at –80 °C until analyzed for the activity of antioxidant enzymes, i.e. superoxide dismutase (SOD, EC 1.15.1.1), the commercially available RANSOD SD125 kit by Randox, UK; glutathione peroxidase (GPx, EC 1.11.1.9, the commercial RANSEL RS505 kit by Randox, UK), and catalase (CAT, EC 1.11.1.6, Aebi’s method11). Fresh plasma samples were assayed for the activity of creatine kinase (CK, EC 2.7.3.2) and lactate dehydrogenase (LDH, EC 1.1.1.27) and for the concentration of uric acid (UA) using diagnostic kits by Randox Laboratories (CKS22, LD3818, and UA230, respectively). The plasma level of malondialdehyde (MDA), a lipid peroxidation and oxidative stress biomarker, was estimated based on the thiobarbituric acid reaction according to Bucke and Aust.21

Biochemical assays were performed by a laboratory certified as meeting the requirements of PN-EN ISO 9001:2015, in line with the recommendations of the testing kits manufacturers.

2.4. Statistical analysis

All data stated below represent means and standard deviation (M±SD). The normality of data distribution was tested using the Shapiro-Wilk test, the homogeneity of variance by the Levene test, and sphericity by the Mauchly’s test. The significance of between-group differences and within-group differences was determined using a two-way mixed-design ANOVA with two groups (AF and SP) and three point-times (PP, CP, and TP) as the main factors, followed, when appropriate, by the HSD Tukey post-hoc test. The ANOVA effect size was assessed by calculating eta squared (h²), whose values can range from 0 to 1 (0.01 – small effect; 0.06 – medium effect; 0.14 – large effect).22 The level of significance was set at α < 0.05. All statistical analysis procedures were performed in Statistica 10.0 (StatSoft, Tulsa, OK, USA).

Table 1
The basic characteristics of participants.

| Variable         | AF (n = 11) | SP (n = 11) |
|------------------|------------|------------|
| Age, years       | 24.0 ± 3.7 | 26.5 ± 3.8 |
| Height, cm       | 183.7 ± 10.3 | 182.7 ± 5.4 |
| Body mass, kg    | 98.2 ± 15.5 | 81.1 ± 5.4 |
| VO₂max, ml/kg/min | 52.4 ± 3.7 | 56.7 ± 4.3 |

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Table 2
Characteristics of training programs for American football players and soccer players.

| Sport                  | Preparation | Competition | Transition |
|------------------------|-------------|-------------|------------|
|                        | General     | Special     |            |
| American football      | - maximum strength building, - muscle mass building, - fitness training, - technical training -tactical training | - endurance training (gym and playing field) - test matches No. of sessions per week: 2 × 1 h (the gym) + 2 × 2.5 h (the field) | - strength training with reduced volume - tactical training with speed, speed and endurance elements - plyometric exercises No. of sessions per week: 1 × 1 h (strength training) + 2 × 2.5 h (the field) Period length: 8 weeks | - lower intensity exercises - special exercises addressing players' technical shortcomings No. of sessions per week: 1 × 1 h (the gym) + 1 × 2 h (the field) Period length: 4 weeks |
| Soccer                 | - motor skills - endurance training No. of sessions per week: 3 × 1 h (the gym) + 5 × 1.5 h (the field) Period length: 6 weeks | - tactical and technical training - test matches No. of sessions per week: 2 × 1.5 h (the gym) + 4 × 1.5 h (the field) Period length: 14 weeks | - special exercises and high-intensity aerobic exercises, speed exercises, and speed-endurance exercises No. of sessions per week: 1 × 1.5 h (the gym) + 2 × 1.5 h (the field) Period length: 2 weeks | - special exercises addressing players' technical shortcomings No. of sessions per week: 1 × 1.5 h (the gym) + 2 × 1.5 h (the field) Period length: 2 weeks |

3. Results

3.1. Antioxidant enzymes

Table 3 contains values characterizing participants’ antioxidant status by period of the training macrocycle and study group. According to the two-way mixed-design ANOVA, the period effect and the group × period interaction effect were significant for SOD, CAT, and GPx. Between-group differences for SOD and CAT were statistically significant at the beginning of the TP (p < 0.05), and for GPx in the early CP. The highest activity of SOD and CAT occurred in both groups at the start of the CP. In the SP group, GPx was statistically significantly more active (p < 0.01) at the beginning of the TP than in the early PP and CP; in the AF group, its changes during the macrocycle were not significant.

3.2. Non-enzymatic antioxidants

The period effect and the group × period interaction effect proved significant for the concentrations of GSH and UA (Table 3). GSH concentrations changed in both groups, but only in the SP its values at the beginning of the CP and TP were significantly higher than in the early PP (p < 0.01). The lowest concentrations of this antioxidant were recorded in both groups at the start of preparatory period. Changes in the UA concentration in the AF were not significant, unlike the increase in the UA concentration (p < 0.01) between the beginning of the CPO and the TP in the SP.

3.3. Lipid peroxidation and muscle-damage markers

The group effect and the period effect were significant for MDA.

Table 3
The indices of participants’ blood antioxidant status by period of the training macrocycle.

| Variable          | Period | AF (n = 11)                      | SP (n = 11)                      | A mixed ANOVA          | Eta squared |
|-------------------|--------|---------------------------------|---------------------------------|------------------------|-------------|
| SOD, U/gHb        | PP     | 1270.61 ± 174.42                | 1161.10 ± 264.50               | GE: F₁,2₀ = 3.39, p = 0.08 | η² = 0.15   |
|                   | CP     | 1426.30 ± 282.49                | 1545.00 ± 413.53*              | PE: F₂,4₀ = 9.01, p < 0.001 | η² = 0.31   |
|                   | TP     | 1346.86 ± 185.49                | 988.15 ± 182.51**               | INT: F₂,4₀ = 4.37, p < 0.05 | η² = 0.18   |
| CAT, U/gHb        | PP     | 195.45 ± 26.42                  | 188.44 ± 44.21                 | GE: F₁,2₀ = 1.03, p = 0.32 | η² = 0.05   |
|                   | CP     | 199.49 ± 19.42                  | 204.55 ± 35.43                 | PE: F₂,4₀ = 6.30, p < 0.05 | η² = 0.24   |
|                   | TP     | 195.11 ± 13.37                  | 163.55 ± 36.60***              | INT: F₂,4₀ = 4.24, p < 0.05 | η² = 0.18   |
| GPx, U/gHb        | PP     | 56.60 ± 6.56                    | 44.69 ± 14.29                  | GE: F₁,2₀ = 3.98, p = 0.06 | η² = 0.17   |
|                   | CP     | 54.89 ± 8.72                    | 42.22 ± 12.07                  | PE: F₂,4₀ = 8.73, p < 0.001 | η² = 0.30   |
|                   | TP     | 59.08 ± 8.99                    | 60.35 ± 17.49***               | INT: F₂,4₀ = 3.81, p < 0.05 | η² = 0.16   |
| GSH, μg/mlHb      | PP     | 2.33 ± 0.35                     | 2.06 ± 0.12                    | GE: F₁,2₀ = 0.90, p = 0.36 | η² = 0.04   |
|                   | CP     | 2.70 ± 0.27                     | 2.94 ± 0.36**                  | PE: F₂,4₀ = 29.82, p < 0.0001 | η² = 0.60   |
|                   | TP     | 2.56 ± 0.27                     | 2.89 ± 0.35**                  | INT: F₂,4₀ = 8.36, p < 0.001 | η² = 0.29   |
| UA, mg/dl         | PP     | 5.62 ± 0.78                     | 4.28 ± 1.67                    | GE: F₁,2₀ = 0.63, p = 0.44 | η² = 0.03   |
|                   | CP     | 5.91 ± 0.62                     | 5.83 ± 1.65                    | PE: F₂,4₀ = 18.73, p < 0.0001 | η² = 0.48   |
|                   | TP     | 6.29 ± 1.21                     | 6.85 ± 1.91**                  | INT: F₂,4₀ = 6.42, p < 0.01 | η² = 0.24   |

SOD — superoxide dismutase, CAT — catalase, GPx — glutathione peroxidase, GSH — glutathione, UA — uric acid, PP — preparatory period, CP — competition period, TP — transition period, GE — group effect, PE — period effect, INT — group × period interaction effect.

Values are presented as mean [SD]. *p < 0.05, **p < 0.01, ***p < 0.0001 — significantly different vs. PP; **p < 0.01 — significantly different vs. CP; *p < 0.05, **p < 0.01 — significantly different between AF and SP.

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and LDH (Table 4). As regards CK, the two-way mixed-design ANOVA did not show the group effect, the period effect, and the group × period interaction effect to be significant. The concentration of MDA and the activity of LDH (p < 0.05) significantly differentiated the AF from the SP at the start of PP and CP. In the AF group, neither the concentration of MDA nor the activity of CK and LDH changed significantly during the training macrocycle; CK activity changes in the SP group were not significant, but the activity of LDH measured at the start of the TP was significantly higher (p < 0.05) than in the early PP and CP.

4. Discussion

This study provides an assessment of the changes in blood antioxidant defense in American footballers and soccer players recorded between the three periods making up the training macrocycle.

4.1. Changes in antioxidant enzymes over the training macrocycle

The effectiveness of antioxidant defense relies on antioxidant enzymes (SOD, CAT and GPx) and endogenous non-enzymatic antioxidants, mainly UA and GSH. The key antioxidant enzyme is SOD catalyzing the dismutation of superoxide radical anion into hydrogen oxide (H₂O₂). It is supported by synergistic enzymes CAT and GPx that decompose H₂O₂ to water and oxygen. During high-intensity physical exercise that stimulates the production of RONS and promotes the regeneration of other antioxidants as well as helping maintain the correct cellular redox potential involved in the regulation of intracellular metabolism. In our study, its concentration was lower in both groups in the early preparatory period than at the beginning of both competition and transition periods, which is consistent with variations in the redox status of the elite French league soccer players observed by Le Moal et al.15 over the training macrocycle.

Another marker we analyzed was UA, a product of the metabolic breakdown of purine nucleotides catalyzed by xanthine oxidoreductase. UA accounts for 50% of blood antioxidant capacity when in the normal range but higher concentrations turn it into a prooxidant. In our study, the plasma concentration of UA was rising over the training macrocycle in both groups, reaching its highest level in the early transition period. Accordingly, a mixed-design ANOVA confirmed by a large period effect (η² = 0.48). This finding is consistent with the results reported by other authors, according to whom the plasma concentration of UA increases after high-intensity exercise with a delay, probably due to a lower UA renal clearance.31 While UA concentrations varied over the training macrocycle, they never fell outside the reference range (3–7 mg/dl), meaning that in both groups UA retained its function of a non-enzymatic antioxidant.

4.2. Changes in non-enzymatic antioxidants over the training macrocycle

High-intensity physical exercise increasing RONS production results in the peroxidation of membrane polyunsaturated fatty acids. One of the final products of this process is malondialdehyde (MDA). In our study, the highest, although statistically non-significant, increase in the concentration of MDA occurred in both groups at the beginning of the transition, probably because of the increased lipid peroxidation and greater leakage of cellular enzymes into the bloodstream brought about by the competition period.32–34 The highest concentration of MDA was observed in both groups in the early transition period.

Biochemical monitoring for training effectiveness frequently involves the analysis of plasma CK and LDH. The post-exercise increase in the activity of these two enzymes depends on exercise intensity, duration, and the type of muscle contractions and tends to be higher in individuals who exercise regularly. In this study, the group, period, and interaction effects for CK activity proved not significant as opposed to the group effect (p < 0.0001,

![Table 4](image)

| Variable | Period | AF (n = 11) | SP (n = 11) | A mixed ANOVA | Eta squared |
|----------|--------|------------|------------|------------|------------|
| MDA, µmol/l | PP | 6.28 ± 1.02 | 4.80 ± 0.70¹ | GE: F₁,₂₀ = 16.57, p < 0.001 | η² = 0.45 |
| | CP | 6.26 ± 1.28 | 4.70 ± 0.78¹ | PE: F₁,₂₀ = 4.81, p < 0.01 | η² = 0.20 |
| | TP | 6.89 ± 1.85 | 5.91 ± 1.09 | INT: F₁,₂₀ = 0.58, p = 0.56 | η² = 0.03 |
| CK, U/l | PP | 306.92 ± 95.37 | 263.94 ± 122.76 | GE: F₁,₂₀ = 0.67, p = 0.42 | η² = 0.03 |
| | CP | 271.13 ± 102.17 | 293.89 ± 78.73 | PE: F₁,₂₀ = 1.95, p = 0.16 | η² = 0.09 |
| | TP | 293.11 ± 118.21 | 224.11 ± 126.37 | INT: F₁,₂₀ = 0.58, p = 0.57 | η² = 0.03 |
| LDH, U/l | PP | 401.78 ± 67.89 | 289.99 ± 67.54¹ | GE: F₁,₂₀ = 21.20, p < 0.0001 | η² = 0.51 |
| | CP | 382.60 ± 63.65 | 276.14 ± 77.22¹ | PE: F₁,₂₀ = 10.42, p < 0.001 | η² = 0.34 |
| | TP | 430.52 ± 48.29 | 355.20 ± 5.53* | INT: F₁,₂₀ = 0.93, p = 0.40 | η² = 0.04 |

MDA — malondialdehyde, CK — creatine kinase, LDH — lactate dehydrogenase, PP — preparatory period, CP — competition period, TP — transition period, GE — group effect, PE — period effect, INT — group × period interaction effect.

The values in the table represent means (SD). *p < 0.05 — significantly different vs. PP; †p < 0.05 — significantly different vs. CP; ‡p < 0.05 — significantly different between AF and SP.
η² = 0.51) and the period effect (p < 0.001, η² = 0.34) for the activity of LDH, which was probably associated with the athletes’ training status.

5. Limitations

The main limitation of our study is that antioxidant defense changes in American footballers and soccer players in Poland during the training macrocycle are only considered with respect to the endogenous mechanisms, without taking account of the role of dietary antioxidants.

6. Conclusion

The study has shown an association between training loads in the preparatory period and moderate improvements in American footballers’ and soccer players’ blood antioxidant status, and their prooxidant-antioxidant balance continuing into the early competitive period. It can be hypothesized that the use of dietary antioxidants or antioxidant supplements as an enhancement in the prooxidant-antioxidant balance would additionally improve the effectiveness of the athletes’ antioxidant defense mechanisms and mitigate the oxidative stress impact.

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Declaration of competing interest

The authors declare no conflict of interest.

Author statement

E.S.K. and S.B. wrote the manuscript. E.S.K. and S.B. designed the research. S.B. and A.K. performed the experiment. E.S.K., S.B. and J.I. analyzed data. All authors have read the manuscript and agreed to its publication.

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