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# **OXIDATIVE STRESS AND TRAINING**

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A. Study design/planning

- B. Data collection/entry
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- G. Funds collection

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Key words: reactive oxygen species, oxidative stress, exercise, training

#### Abstract.

**Introduction.** Reactive oxygen species (ROS) are produced in the body under physiological conditions. ROS are involved, among others, in cell signaling. Imbalance between ROS creation and their neutralization is called oxidative stress and is characterized by the uncontrolled oxidation of lipids, proteins or DNA. Research indicates that increased production of ROS and prooxidative-antioxidant imbalance may occur during intense exercise.

**Aim.** The aim of the study was to determine the effect of different sports on the prooxidative-antioxidant blood status among highly trained athletes on the basis of a review of scientific literature.

**Material and methods.** A review of scientific literature was done from 2011 to June 2015 using the databases: Medline, PubMed and Google Scholar web browser. The review included original works which contained the following expressions in the text of their abstracts: "oxidative stress and sport", "oxidative stress and athletes", "oxidative stress and endurance," "free radicals and sport", "free radicals and endurance". 570 scientific papers were selected in total.

The analysis excluded the impact of work on the nutrition strategy, vitamin and mineral supplementation, sport nutrition supplements that could affect the level of oxidative stress, and in which a group of test persons were untrained and casual exercisers. Criteria fulfilled 20 scientific papers (original), which present the results of studies involving 458 players individual sports and team, among others: footballers, volleyball, handball, players: martial arts, tennis, triathlon, swimming.

**Results.** Numerous studies have shown that an increase in ROS production as a result of physical exercise is conditioned by among others: degree of fitness competitor, gender and intensity, duration and type of exercise.

**Conclusions.** Physical training models the efficiency of the antioxidant defense mechanisms of the body by increasing the activity of antioxidant enzymes such as catalase and superoxide dismutase, resulting in increased levels of total antioxidant status in plasma and increase the share of glutathione antioxidant capacity in trained athletes.

**Summary.** ROS synthesis depends on the type of muscle work, intensity and duration of exercise. The improvement in exercise capacity is an effective way to improve antioxidant defense system, and thus to stay healthy.

#### Introduction

Oxygen is an essential element for the proper conduct of oxidative phosphorylation. About 2-5% of the oxygen absorbed by the body is converted into reactive oxygen species (ROS), which may damage the biologically active compounds, in particular proteins [1]. ROS include, inter alia, superoxide anion  $(O_2^{-})$ , converted to the representative body of free radicals and hydrogen peroxide -  $H_2O_2$ , the most important precursor of free radicals [2, 3]. Oxygen free radicals belonging to ROS include at least one oxygen atom and have at least one or more unpaired electrons. ROS has a high reactivity and easily reacts chemically with cellular components. ROS interact with cells or signal molecule relay, which indicates that they play an important role in the proper functioning of the whole cell [3]. They are involved in many processes, including muscle work, secretion of hormones, the functioning of the immune system, or in the regulation of vascular tone [4]. On the other hand, ROS excess produces negative effects in the body as oxidation of macromolecules: proteins, fats and nucleic acids. Excess of ROS is observed in the course of some chronic non-contagious diseases (diabetes, cancer, atherosclerosis) [4]. The phenomenon of imbalance between the production of ROS and their neutralization is called oxidative stress.

The level of reactive oxygen metabolites can be measured by the d-ROM test (diacrons Reactive Oxygen Metabolic test). Quantitation of d-ROM is modified depending on the plasma concentrations of hydrogen peroxide  $(H_2O_2)$ , peroxynitrite (ONOO<sup>-</sup>) and hydroperoxides (ROOH) [5].

The ability of reactive oxygen species synthesis belongs to neutrophils, which play a fundamental role in immune response. They are considered the first line during the defense against invading pathogens, which are destroyed as a result of the production of cytotoxic reactive oxygen species [6]. Oxidative burst - OBA (Oxidative Burst Activity) is an indicator showing the ability of ROS production by neutrophils [6].

Free radicals are neutralized by antioxidant defense of the body including enzymatic and non-enzymatic mechanisms. The enzymatic antioxidant defense system creates enzymes, among which we can distinguish superoxide dismutase (SOD) - an enzyme that catalyzes the reaction dismutation of the superoxide radical to hydrogen peroxide, catalase (CAT) - decomposing hydrogen peroxide into water and oxygen, peroxidase, glutathione peroxidase (GPx) - convering reduced glutathione (GSH) into oxidized glutathione (GSSG), and reducing hydrogen peroxide to oxygen [7] or glutathione reductase (GR), which catalyzes the reduction of glutathione (GSSG to GSH ) in the presence of NADPH which is oxidised to NADP<sup>+</sup> [8].

Reduced glutathione GSH is a naturally occurring endogenous antioxidant. By reacting with reactive oxygen species to protect the thiol groups of proteins against oxidation by ROS. Evaluation of the GSH/GSSG redox potential is one of method of determining the level of oxidative stress [9]. Bilirubin, in addition to glutathione, is a natural antioxidant in plasma and in cell membranes. The level of total bilirubin (TBIL) significantly increases after intense exercise [10]. Not all endogenous antioxidants are characterized only by positive action. An example can be uric acid (UA) and nitric oxide (NO). UA is a scavenger of peroxyl, peroxynitrite and hydroxyl radicals. At the same time, the high level correlates with the occurrence of non-infectious chronic diseases (obesity, hypertension, cardiovascular disease or cardiac disease) [10,11]. In contrast, NO secreted by the vascular endothelium against increase of vascular tone plays an important role in physiological processes of the nervous system, both as a signaling molecule and the sodium pump cell modulator. On the other hand, the excess can contribute to the formation of toxic compounds reacting with oxygen, iron or copper [4].

Together, antioxidants determine total antioxidant status (TAS - Total Antioxidant Status), which is the body's ability to counteract the damage induced by free radicals [12]. Another indicator of the level of oxidative stress is total oxidation potential (TOS - Total Oxidative Status) [13]. In contrast, the index of oxidative stress OSI (Oxidative Stress Index) is estimated on the basis of TOS/ TAS, and reflects the balance of oxidative and antioxidant processes in the body [12]. Many studies have used estimated concentrations of malondialdehyde (MDA) as a marker of oxidative stress and the main product of lipid peroxidation [14]. Lipid peroxidation is a process of oxidation, especially polyunsaturated fatty acids included in phospholipids that form the basic building block of biological membranes (mainly phosphatidylethanolamine and phosphatidylcholine) [15]. This leads to the formation of peroxides of such compounds and damage associated with the plasma and mitochondrial membranes. Evaluation of products reacting with thiobarbituric acid (TBARS) is a well-known marker of lipid peroxidation [16]. This process is free radical in nature and occurs rapidly. An additional marker may be fatty acid peroxide (LOOH), which emerges considerably earlier than MDA in one of stages of lipid peroxidation [15]. A relatively sensitive gauge of the size of oxidative stress is the assessment of changes in the level of protein oxidation products. Among them can be distinguished such indicators as AOPP (Advanced Oxidation Protein Products) [17], RCD (Reactive Carbonyl Derivatives) [18].

Exercise contributes to increased production of ROS, which, as it has been shown in numerous studies, has adverse effects, particularly in the case of unprepared tissue [12, 13, 19, 20, 21, 22]. Whether the oxidative stress induced by exercise reveals only harmful effects remains a matter of debate.

Although increased production of ROS induced by exercise is potentially damaging to physiological function, the repeated exposure of the body's increased production of ROS during regular physical exercise leads to positive adaptive changes in the body. This is manifested in the regulation of expression of gene encoding proteins responsible for the antioxidant defense of the body [7,19]. This was found to increase the activity of antioxidant enzymes such as superoxide dismutase (SOD) and catalase (CAT) in the muscles, liver and heart after intense physical exercise [20].

This adaptation provides protection against ROS during subsequent sessions. A series of high-intensity

exercises by increased production of ROS activates the NF-κB transcription factor by increasing transcription of genes for antioxidant enzymes such as mitochondrial superoxide dismutase (MnSOD) and inducible nitric oxide synthase (iNOS) [23, 24].

Intense aerobic exercise exacerbates oxidative stress and the increase of endogenous antioxidants in untrained persons, whereas in those training, significantly lower levels of oxidative stress are achieved as the result of regular exercise programs [20]. In addition, [25,26], athletes have a higher antioxidant capacity than untrained people, but the capacity is different in different phases of meso and macro-training process [27].

The increase in free radical production as a result of exercise depends on many factors, such as age, sex, the athlete's degree of fitness and the intensity and duration of exercise, the type of exercise and eating habits [28].

The aim of the study was to determine the effects of different sports on the prooxidative - antioxidant status in the blood among highly trained athletes on the basis of a review of the scientific literature on the subject.

### **Material and methods**

A review of scientific research from 2011 to June 2015 was conducted using the databases: Medline, PubMed and Google Scholar web browser. The review includes original papers, containing the following expressions in their abstracts: "oxidative stress and sport", "oxidative stress and athletes", "oxidative stress and endurance," "free radicals and sport", "free radicals and athletes," "free radicals and endurance".

A total of 570 scientific papers were selected. The analysis excluded the impact of works on nutrition strategy, vitamin and mineral supplementation, sport nutrition supplements and in which the study group consisted of untrained persons and those exercising regularly. The criteria were fulfilled by 20 scientific papers (original), which present the results of studies involving 458 players of individual and team sports, among others: footballers, volleyball, tennis and handball players, athletes doing martial arts, the triathlon and swimming. The average age of the youngest group of athletes included in the analysis was  $15.2 \pm 0.9$  years, and the oldest  $37 \pm 6.7$  years.

## The level of oxidative stress and individual sports

Many external stimuli, including physical exercise, increase the number of neutrophils and increase their activity, together with their ability to synthesize reactive oxygen species. The results of the research by Kudoh et al. [6] confirm earlier reports [29] on the positive impact of exercise in increasing the number of neutrophils. The study by Kudoh et al. included 39 men, members of the Nippon Sport Science University judo club. Before and after training (duration: 2 hours, HR 129  $\pm$  12 beats/min), venous blood was collected for biochemical assays. There was a statistically significant increase in OBA after the completion of training, while a statistically significant reduction of phagocytic activity also occurred (Table 1) [6].

Lipid peroxidation is the most common consequence of oxidative stress leadings to an increased level of oxidation products of fatty acids including LOOH and MDA. In their study, Eroglu et al. [21] indicate the level of MDA and activity of sodium in judo competitors and healthy untrained persons. The subjects were exposed to submaximal (75% VO<sub>2</sub>max), aerobic exercise on a mechanical treadmill.

MDA concentration after exercise significantly increased in both groups of judo competitors by 32.7%, and 15.5% in untrained, but the activity of SOD significantly increased in judo competitors by 13.2% (Table 1). The degree of lipid peroxidation was determined by de Lucas et al. [30] using TBARS as a marker. The authors studied a group of 11 highly trained athletes of different sports who participated in the Multisport Brasil Race, consisting of three disciplines: running - 28.5 km, mountain biking - 42.5 km and kayaking - 17.5 km.

Before and after the race, blood was collected and analyzed, and among others, CAT and TBARS were determined. They found that the CAT activity after exercise did not change, and the level of TBARS increased significantly to 145% (Table 1). In contrast, Rowlands et al. [31] examined long-distance runners after they ran the distance of 894 km in 47 stages, extending over 95 hours (Bruce Trail). TAS was indicated in the athletes blood but the exercise-induced increase was not significant. The statistically significant higher rate of TAS, TOS and OSI, measured during rest, was found in children who trained swimming, as compared to the untrained group of children (Table 1) [13]. Other studies related to the assessment of indicators of oxidative stress among tennis players, and athletes doing karate and wrestling. Research by Knez et al. [5] was conducted among 10 tennis players, who played two friendly matches in two different controlled climatic conditions: indoor court, the temperature of approx. 22°C and humidity of approx. 73%; at a temperature of about 37°C and humidity of approx. 12%, on an outdoor court, with an interval of 72h or 144h. Blood was collected before the warm-up, in the middle of and immediately after each match and 24h and 48h after the game. The d-ROMs test was performed (H<sub>2</sub>O<sub>2</sub> ONOO<sup>-</sup>, ROOH). The results showed that the concentration of d-ROMs did not show statistically significant changes depending on the environmental conditions, as well as the length of rest after the match

Authors, year	Trained: discipline, number (n), gender, age, training experience (TE), frequency of training	Control group: un- trained (UT), number (n), gender (M, F), age	Applied test (type, duration of exercise, blood collec- tion - BC)	Results – statistically significant
Bulduk,et al., 2011 [20]	Volleyball, n=10, F, 20±1.2 years	UT, n=10, F, 19±1.8 years	20-m transfer run, BC before and after exercise	TR after exer- cise: ↓GSH, ↑MDA, ↓CAT UT after exer- cise: ↓GSH, ↑MDA, ↓CAT
Conti et al., 2012 [37]	Triathlon (T), n=10, M, 29.8 $\pm$ 8.7 years; Football (FB), n=15, M, 26 $\pm$ 0.6 years; Sprinters (S), n=10, M 31.3 $\pm$ 6.4 years, TE $\geq$ 5 years	none	Treadmill stress test according to Bruce protocol, spirometric test, BC in the morning, fasting state, 12h after mean	CAT: FB>S>T NO: T>FB>S TBARS: S>FB>T
da Costa et al., 2011 [34]	Football, n=10, M, 18.3±0.7 years	none	Loughborough Intermit- tent Shuttle Test (LIST): march, sprint, jogging (55%VO <sub>2</sub> max and 95%VO- 2max alternately for 90 min: 5x15 min and 3 min interval), BC before, during and after the test	After exercise: ↓Urea, ↑MDA
Djordjevic et al., 2012 [19]	Handball, n=58, M, 17.3±0.2 years, Training sessions 5 times a week, 1.5 h each	UT, n=37,M, 17.5±0.3 years	Progressive test (until refusal) 2W/kg, load increase every co 3 min by 50W, 60 rev/min), BC before and after exercise	none
Eroglu et al., 2013 [21]	Judo, n=16, M, 20±1.9 years	UT, n=16, M, 20±1.2 years	Treadmill test,75%VO2max accord- ing to Bruce protocol, BC before and after exercise	TR after exer- cise: ↑MDA,↑SOD NT after exer- cise: ↑MDA
Gökhan, 2013 [12]	Volleyball, n=20, M, 15±1.64 years, TE 2 years, Training sessions 3 x week, lasting 2 h	UT, n=12, M, 15.3±1.2 years	none	TR vs. UT: ↑0SI, ↓TAC, ↑TOS
Gökhan et al., 2013 [13]	Swimming, n=18, M, 15.2±0.9 years, TE=2 years, Training sessions 3 x week, lasting 2 h	UT, n=18, M, 15.3±1.1 years	none	TR vs. UT: ↑TAC, ↑TOS, ↑OSI
Hadžović-Džu- vo et al., 2014 [17]	Wrestling (W), n=12, Football (FB), n=14, Basketball (B), n=13, 22.1 $\pm$ 4.4 years, TE>10 years, training sessions min. 6 x week	none	none	MDA: K>Z>P ADPP: K>Z>P ImAnOx: K>P>Z
Hammouda et al., 2011 [10]	Football, n=12, M, 17.4±0.4 years, Training sessions 4 x week, lasting 2 h	none	RSA Test– 5 x 6s Maximal sprint cycloergometer, BC 7:00 a.m. and 5:00 p.m. before and after exercise	Before exercise in the evening vs. morning: ↓TBIL, ↓UA, ↓TAS After exercise in the evening vs. morning:↓TBIL, ↓UA, ↓TAS
Hammouda et al., 2012 [11]	Football, n=18, M, 17.5±0.4 years, Training sessions 4 x week, lasting 2 h	none	30-s Wingate test, BC 10 h before and 3 min after test	After exercise:↑TBIL, ↑UA, ↑TAS

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Knez et al., 2014 [5]	Tennis, n=10, M, 22.6±4,6 years	none	2 matches (indoor and outdoor) on hard surfaces at interval of 72 or 144 h. BC before, in the middle, immediately after, 24 h and 48 h after the match	↑↓ d-R0Ms
Kudoh et al., 2014 [6]	Judo, n=39, M	none	2-hour UEL training sessions - uni- fied exercise loading, BC before and after UEL.	After exercise: ↑0BA, ↓PA
de Lucas et al., 2014 [30]	Various disciplines, $n=11$ , M, $34.3\pm3.1$ years	none	Multisport Brasil race: 28.5 km - run, 42,5 km – mountains cycling, 17.5 km - kayaking	After exercise: ↑LDH, ↑TBARS, ↓↑CAT
Marin et al., 2011 [36]	Handball, n=14, M, 25±4.5 years, TE=11.4±3.1 years	none	Handball match, BC before warm- up, immediately after and 24 h after match	After exercise: ↑SOD, ↓CAT, ↓GPX, ↓GR After 24h: ↑SOD, ↓CAT, ↑GPx, ↓GR
Mila – Korze- niowska et al., 2013 [35]	Volleyball, n=18, M, 28.3±4 years, TE=11.8±3.2 years	The same study group + exercise	Stage 1: systemic cryostimulation (WBC) and 40 min exercise on ergometer 85% HRmax, 165 W and 50 rev/min Stage 2: exercise on ergometer, 160 W and approx. 50 rev/min BC before and after exercise, + after WBC	Stage 1: after WBC: $\uparrow$ SOD, $\uparrow$ GPx, $\uparrow$ CAT, $\downarrow$ TOS After exercise and WBC: $\downarrow$ SOD, $\uparrow$ GPx, $\downarrow$ CAT, $\uparrow$ TOS Stage 2: after exercise: $\uparrow$ SOD, $\uparrow$ GPx, $\uparrow$ CAT, $\uparrow$ TOS
Neto et al., 2013 [22]	Volleyball, n=9, M, 17.9±1.1 years	UT, n=9, M, 18.7±1.8 years	3 training phases 3 months before phase 1 and after phases 2 and 3, the following tests were conducted: vertical jump using a free counter movement and bench press, BC in the final week of each stage, 48 h after final exercise training session	$\begin{array}{c} Phase 1: \downarrow CAT, \\ \uparrow GR, \uparrow TSG, \\ \uparrow RCD, \uparrow TBARS \\ Phase 2: \downarrow CAT, \\ \uparrow GR, \uparrow TSG, \\ \uparrow RCD, \uparrow TBARS \\ Phase 3: \uparrow CAT, \\ \uparrow GR, \uparrow TSG, \\ \downarrow RCD, \uparrow TBARS \\ \end{array}$
Olubajo et al., 2015 [33]	Football, n=12, F, 18.8±1.2 years, TE≥2 years	UT, n=10, F, 20.6±0.5 years	Treadmill run, 20 min: Speer increase every 2 min by 0.5km/h up to 1.5km/h, BC before and after exercise	TR after exer- cise: ↓SOD, UT after exer- cise: ↑SOD, ↑MDA
Pesic et al., 2012 [2]	Karate, n=30, M, 20.9±4.1 years, TE=8.7±3.6 years	none	2 x graded maximal test (on cycle ergometer), BC before and after exercise	After training session: $\downarrow$ CAT, $\downarrow$ O <sub>2</sub> <sup>-</sup> , $\uparrow$ H <sub>2</sub> O <sub>2</sub> , $\uparrow$ CAT
Rowlands et al., 2011 [31]	Long-distance runs, n=12 (M), n=3 (F), 37 $\pm$ 6.7 years, long TE	none	894 km run, BC before and after run	none
Shadab et al., 2014 [16]	Hockey, Football, Long-distance runs, $n=60, 22.5\pm4,6$ years, TE>1 year, training sessions 4 x week	none	Run 10km/h (90min), BC before and 6h after exercise	After exercise: ↑MDA
Trivić et al., 2011, [32]	Wrestling, n=14, M, 21.9±3.5 years, TE=11.7±4 years	none	Aerobic training (75-85% HRmax) 4 weeks, 3 times a week, lasting 60 min each, fasting state BC, day before and after training session	After training session: ↑SOD, ↑CAT, ↓TAC, ↓↑GPx, ↑↓GR

was played (Table. 1). Pesic et al. [2] studied a group of 30 karatekas who performed twice a graded test to exhaustion on a cycle ergometer: 1 - in the first week of the preparatory period (lasting 3 months), 2 - in the first week of the competition. Additionally, a single controlled karate training session was conducted 36 h after the second maximal test. Before and after exercise, and before and after the endurance test, the following indicators of oxidative stress were determined: SOD, CAT, H<sub>2</sub>O<sub>2</sub>,  $O_{\circ}$ . After the end of the preparatory period, a statistically significant decrease of CAT activity by 16.4% compared baseline was observed, while the single karate practice session influenced the statistically significant increase in CAT activity by 28.8%, statistically significant increase in H<sub>2</sub>O<sub>2</sub> concentration of 20.3% and a statistically significant decrease of 0, by 25.1% (Table 1). However, in the study by Trivić et al. [32], 14 wrestlers who had completed a 4-week training program took part (3 x a week., 60 min aerobic training at an intensity of 75-85% of maximal heart rate). Blood samples were taken twice in the morning: the day before and the day after the end of the training program. The activity of SOD, CAT, TAS, GPx and GR was marked. After completing the training, the TAS level statistically significantly reduced by approx. 1.5%. It showed an increase in the activity of SOD and CAT, and there were no other changes in the activity of antioxidant enzymes (Table 1).

## The level of oxidative stress in team sports

Olubajo et al. [33] conducted a study on two groups: study group comprised of female footballers, a control group of untrained women. The study used a 20-minute graded test on a treadmill consisting of a three-minute warm-up at a speed of 0.5 km/h. Then, the speed of the belt of the treadmill was increased every two minutes from 0.5 km/h to 1.5 km/h. After the exercise, there was a statistically higher level of SOD activity while there was a reduction in CAT activity among the footballers. Additionally, the level of SOD and MDA determined after the exercise test significantly increased in the untrained group (respectively by 33.3% and 39.7%) compared to the value before exercise, and the level of SOD and MDA decreased (by 19.8% and 7.5%) compared to pre-exercise values (Table 1). In other studies on football players, the Wingate test was used [11]. Before and after the test, the levels of TAS, UA and TBIL were determined, the levels of which increased significantly by 4%, 6.5% and 8.3% relative to the resting values (Table 1). In earlier studies, Hammoud et al. [10] also examined a group of football players using the RSA test (Repeated Sprint Ability) on a cycle ergometer (5x 6s maximal sprint + 24 sec rest). The levels of TAS, UA and TBIL were determined at various intervals (morning and evening before and after

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the exercise test). The morning before the exercise, the level of TAS, UA and TBIL were higher than in the evening of the same day, and in the morning after the exercise, the levels of these indicators increased by 10.7%, 12.2% and 15%. In the evening, the level of TAS, UA and TBIL decreased to the level from before the morning exercise (Table 1) [10]. Shadab et al. [16] examined the level of MDA in football players, hockey players and mediumdistance runners before and after 1.5 hours running on a treadmill. MDA levels in these athletes after exercise increased significantly, by more than 6 times (approx. 635.3%) (Table. 1). Furthermore de Costa et al. [34] examined a group of 10 footballers who participated in the LIST test (Loughborough Intermittent Shuttle Test), which consisted of walking, sprinting and jogging at an intensity of - 55% VO, max and the intensity of 95% VO<sub>2</sub>max. Before, during and after the test, the concentration of urea and MDA was determined. It turned out that the level of MDA significantly increased by 24.8% (Table 1) while the resting values of AOPP, MDA, TAS were marked by Hadžović - Džuvo et al. [17] in the study group consisting of football players, basketball players and wrestlers. The results were compared between the groups of athletes. It was shown that a significant statistical difference was only in the level of MDA of basketball players - considerably higher (80.4%), in comparison to the footballers (Table 1). Gokhan [12], in his study, compared the results of indicators of oxidative stress marked during resting of the volleyball and untrained young men. The level of TAS, TOS and OSI was significantly higher in volleyball players than in the control group, which may be the result of the conducted training (Table 1). However, Bulduk et al. [20] conducted a 20-m zig-zag run test in groups comprising of volleyball players and untrained women, and determined MDA, GSH and CAT. They found that the concentration of GSH and CAT assay in both groups significantly decreased by 26% and 13%, while the level of MDA significantly increased by 31.5% in the volleyball group, and 14.5% in the control group (Table 1). Neto et al. [22] in turn, studied volleyballers, introducing a 3-phase training program lasting three months, which corresponded to the periods of training of elite volleyball players (phase 1 - exercises of low intensity for 5 weeks; phase 2 - exercise of increased intensity and at the beginning of the championship for 5 weeks; phase 3 - exercise with reduced load and during the finals of the championship for 3 weeks). They compared the concentration of CAT, RCD, TBARS, GR levels of the study and control groups, at various stages of preparation for the championship. The concentrations of GR in the study during phases 1 and 2 were significantly higher than in the control group, and in phase 3, they were significantly higher relative to the control group and phase 2. Statistically significant changes in the level of CAT can be seen in the third phase of exercise when compared to the control group. The concentration of RCD in the study group was reduced in phase 2, both with respect to phase 1 and the control group. In phase 3, in both study and control groups, comparatively higher levels of RCD were reported. There were no differences in the levels of TBARS between groups and in different phases. On the basis of these results, it can be stated that the volleyball players presented the lowest level of oxidative stress in combination with the best indicators of the possibility of exercise in the fundamental phase - phase 3 (Table 1). Other studies included comparison of the effect of a single cryostimulation session and exercise on the ergometer on the activity of antioxidant enzymes and TOS in volleyball players. The test results showed no statistical difference in the activities of GPx, SOD and TOS, while the CAT activity after the same exercise increased significantly by 100%, and for cryostimulation and exercises, they were approx. 2 times lower than in the case of the same exercise on the ergometer (Table 1) [35]. The level of reactive oxygen and nitrogen species resulting from the exercise leading to a significant level of muscle damage may persist for up to several days, in contrast to the low-level exercise causing muscle damage resulting in normalized ROS, occurring for only a few hours. Marin et al. [36] examined 14 handball players before, immediately after and 24 hours after the match was played. Determined were: TBARS, TAS, GSH, GSSG, GSH/GSSG, SOD, GPx, GR and CAT. It was found that a single game of handball leads to oxidative stress. Found were: a statistically significant decrease in GSH concentration of approx. 18% immediately after the match, a statistically significant increase SOD activity after the match by 166% compared to the resting value and 248% after 24 hours of rest, and a statistically significant decrease in the activity of CAT and GR immediately after the match by 36 % and 46%, and 24 hours after the match by 57% and 53%. In addition, handball players's plasma showed a statistically significant increase in TBARS concentration immediately after the end of the match by 62%, and this upward trend persisted for 24 h after the end of the match (up to about 153%) (Table 1). Other researchers, in turn, researched handball players and people leading a sedentary lifestyle. Blood was collected before and after the exercise test on a cycle ergometer until exhaustive.

The results showed [19] a statistically significant reduction of CAT activity in the group of people training handball (Table 1). In contrast, a study involving athletes of various disciplines was conducted by Conti et al. [37]. Their aim was to compare the response of the antioxidant system as indices of oxidative stress in the blood serum of athletes. The study consisted of athletes from disciplines of a different nature: aerobic-anaerobic - 10 triathletes, aerobic - 15 footballers and anaerobic - 10 sprinters. Venous blood was collected from the subjects once, during fasting, and the following were determined: CAT activity, TBARS and NO in blood serum. TBARS concentration differed significantly between the groups. The level of lipid peroxidation was significantly higher among football players compared with triathlon athletes and sprinters. CAT activity was significantly higher in the footballs players compared to other players and statistically higher in sprinters than triathletes (Table 1) [37].

### Summary

Free radicals are highly reactive. They are synthesized in the body under physiological conditions. Depending on the concentration, they can have positive or negative effects on the body. In accordance with the principle of hormesis, increases in ROS production are a positive incentive for the regulation of the formation of endogenous antioxidant defense of the organism [38]. As follows from this study, regular physical activity is associated with the adaptation of the body in response to oxidative stress. Because regular exercise increases the antioxidant defense mechanisms in the skeletal muscle by activating both the gene expression of SOD and glutathione peroxidase, thereby increasing the effect of the body's defense against the harmful effects of ROS [7]. The results of studies confirm that intense exercise can cause intensification of ROS production, and thus lead to oxidative stress. Depending on the type of exercise, a variety of oxidation products can be produced in different amounts. Thus, increasing physical fitness proves to be an effective way to improve the antioxidant defense system, and therefore, to stay healthy. However, it should be noted that the degree of redox homeostasis disorders caused by exercise depends on many factors, including the type, intensity and duration of the exercise, the exerciser's state, gender and age.

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