

Research Article

Serum Oxidant and Antioxidant Status in Adolescents Undergoing Professional Endurance Sports Training

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This study evaluated the impact of professional training on serum oxidant and antioxidant status in adolescent endurance athletes and compared it with that of untrained individuals. Firstly, serum thiobarbituric-acid-reactive substances (TBARSs), xanthine oxidase (XO), catalase (CAT), reduced glutathione (GSH), superoxide dismutase (SOD), and total antioxidant capacity (T-AOC) were measured in 67 male runners, cyclists, and untrained adolescents. Seven-day dietary intakes were also assessed. Secondly, for age- and Tanner-stage-matched comparison, 36 out of the 67 subjects (12 for each group) were then selected and investigated. In cyclists, XO, GSH, and CAT were higher as compared with runners and controls. The CAT in runners, but not GSH and XO, was also higher than in controls. TBARS, T-AOC, and SOD did not differ among the study populations. Regarding the inter-individual relationships among serum redox statuses and dietary nutrient intakes, significant correlations were noted in CAT versus carbohydrates, protein, magnesium, and manganese; GSH versus carbohydrates, protein, fat, selenium, zinc, iron, and magnesium; XO versus cholesterol; CAT versus GSH. These findings suggest that the resting blood redox balance in the professional adolescent athletes was well maintained partly by the increase of individual antioxidant in adaptation to chronic exercise.

1. Introduction

It is known that physical activity plays a critical role in enhancing growth and development in both childhood and adolescence. The healthy adaptations to repeated exercise are featured by increased muscle and bone mass, appropriate body fat composition, angio- and arteriogenesis, and increased number of mitochondria [1]. However, the induced stress and the alterations in immune and inflammatory status in response to exercise are similar to those resulting from chronic diseases such as asthma and arthritis [2]. Such physiological responses are associated with catabolism rather than anabolism. Closely linked to these catabolic responses to exercise is the exercise-mediated oxidative stress, which develops when the production of reactive oxygen species (ROS)

exceeds the antioxidant defences [3]. The excessive ROS-caused cytotoxicity, injury, and inflammation in body tissues are known to be associated with a kaleidoscope of pathologies, including cardiovascular and metabolic diseases [3].

Nevertheless, the signalling function of ROS also plays a role in mediating the physiological adaptations gathered from regular exercise, including myokine production that is essential in muscle adaptation to exercise and upregulation of antioxidant defence mechanisms [4, 5]. The regular training-induced adaptations in blood antioxidant capacity and the following attenuated exercise-induced oxidative stress have been clearly demonstrated in adult athletes [6–8]. However, the investigations of the specific training adaptations in highly trained adolescent athletes are scarce to the best of our knowledge. Year-round training in professional sports

beginning at a relatively young age is increasingly frequent in the youth, with training volume being comparable to that of adult athletes (i.e., 1.5 to 3 training hours per session, 2 sessions per day, 6 days per week). Recently, impaired oxidant-antioxidant balance was noted in adolescent endurance runners subsequent to a single-routine training session of exhaustive 21 km run [9]. Whether the resting oxidant-antioxidant levels in professional endurance-trained adolescents are comparable to those reported in their adult counterparts was not clear. As such, the aim of this study was to evaluate the impact of professional training on serum oxidant and antioxidant status in adolescent endurance runners and cyclists, and compare it with that of untrained individuals matched for sex, age, and Tanner stage. It is known that exercise mode specificities can promote different responses regarding oxidative stress [10], and that is why the redox status of adolescent runners and cyclists was examined separately. Since exogenous dietary substance may affect antioxidant defence system, the dietary intake of the athletes and the untrained individuals was also assessed.

2. Method

2.1. Subjects. In this study, totally 67 male adolescent endurance runners ($n = 27$, 15.8 ± 1.4 years), cyclists ($n = 20$, 15.7 ± 1.0 years), and untrained individuals ($n = 20$, 15.7 ± 0.4 years) had been invited to participate in the experiments. All the runners and cyclists performing at national level were trained professionally in a sports club in Liaoning province, China. The untrained adolescents were active, but did not participate in any sports training. All trained and untrained subjects had no familial history of cardiovascular disease or assumed related medication. More importantly, none of them received anti-inflammatory medication or nutritional supplements. Following an explanation of the purpose and constraints of the study, subjects and their tutors gave written informed consent for participating in this study. The local Ethical Committee for the Use of Human and Animal Subjects in Research provided ethical approval of the study.

For age- and Tanner-stage-matched comparison, 36 runners, cyclists, and untrained adolescents (12 for each group) out of the 67 subjects were selected for investigation. The physical characteristics of the selected subjects and the training background of the athletes including the training volume are shown in Table 1. The energy costs for training per day in each athlete were estimated on the bases of the metabolic equivalents (METs) corresponding to the specific training intensity expressed as average running/cycling speed that were reported in the Compendium of Physical Activities [11], the average training hours per day, and the body weight.

2.2. Procedures. Subjects of each group visited the laboratory in a single morning session. The laboratory visit of each group was arranged on separate days. Prior to the laboratory visit, the adolescents abstained from exercise training for 3 days. Upon arrival at the laboratory at 9 am after an overnight fasting, anthropometric measurement and Tanner staging assessment were carried out following a 10 min rest.

After that, blood samples were collected with subjects in a seated position. 5 mL venous blood was drawn from the ante-cubital vein using venous puncture for serum redox analyses. One week preceding the abstinence of exercise training, the diets of consecutive 7 days of the subjects were recorded according to the guidelines for monitoring dietary consumption provided. Diet records were analyzed using the dietary and nutritional analysis system designed for Chinese athletes and general population (National Research Institute of Sports Medicine, China).

2.3. Measurements. In this study, we evaluated the oxidant and antioxidant status of the subjects by quantifying serum concentrations of thiobarbituric-acid-reactive substances (TBARS), reduced glutathione (GSH), and total antioxidant capacity (T-AOC), as well as the enzymatic activity of xanthine oxidase (XO), superoxide dismutase (SOD), and catalase (CAT). After blood sampling in vacuum tubes containing no additives, the serum was separated at 2,000 g for 20 minutes, aliquoted, and stored at -20°C for later analysis.

The levels of TBARS and GSH and enzymatic activity of XO, SOD, and CAT were measured using commercial assay kits (Nanjing Jiancheng Institute, China) on a spectrophotometer (DU7400, Beckman Co, Fullerton, USA), according to the instructions of manufacturer. Briefly, lipid peroxidation was evaluated using the thiobarbituric-acid-reactive substances method and was expressed as a TBARS concentration. This method was used to obtain a spectrophotometric measurement of the colour produced during the reaction of thiobarbituric acid and malondialdehyde (an indicator of peroxidation of polyunsaturated fatty acids in cell membranes subsequent to reactions with ROS) at 535 nm. The TBARS level was expressed as $\text{nmol}\cdot\text{mL}^{-1}$. The GSH level was determined colorimetrically at 412 nm following reaction with DTNB (5,5'-dithio-bis(2-nitrobenzoic acid)) and was expressed as $\text{nmol}\cdot\text{mL}^{-1}$. XO and SOD activities were measured by the xanthine-xanthine oxidase system, which is a superoxide anion generator, following the increase or decrease of absorbance, respectively. The activity of XO and SOD was expressed as $\text{U}\cdot\text{L}^{-1}$ and $\text{U}\cdot\text{mL}^{-1}$. CAT activity, expressed as $\text{U}\cdot\text{mL}^{-1}$, and was determined by the decrease of H_2O_2 absorbance at 240 nm. T-AOC was measured by the ferric-reducing ability of plasma (FRAP) assay of Benzie and Strain [12]. The stable colour of the Fe^{2+} -o-phenanthroline complex (produced with reducing agents in plasma by reducing Fe^{3+} to Fe^{2+} , which reacts with the substrate ophenanthroline) was measured at 520 nm. T-AOC was expressed in $\text{U}\cdot\text{mL}^{-1}$, where 1 unit is defined as an increase in absorbance (A_{520}) of 0.01 per min at 37°C .

The inter- and intra-assay coefficients of variation of the above-mentioned biochemical analyses are as follows: TBARS, 5.4% and 2.2%; GSH, 4.8% and 1.8%; XO, 9.2% and 4.5%; SOD, 8.7% and 5.0%; CAT, 11.4% and 6.2%; T-AOC, 8.5% and 4.6%, respectively.

2.4. Statistical Analysis. The Kolmogorov-Smirnov normality test revealed that the data for all the variables were normally distributed. One-way ANOVA was computed to

TABLE 1: Physical characteristics of study subjects, training years, and training volume of runners and cyclists are shown.

	Runners (<i>n</i> = 12)	Cyclists (<i>n</i> = 12)	Untrained (<i>n</i> = 12)
Age (yrs)	15.5 ± 1.3	15.3 ± 0.7	15.9 ± 0.5
Tanner Stage	3.25 ± 0.87	3.08 ± 0.29	2.75 ± 0.45
Weight (kg)	57.7 ± 6.3 ^a	70.8 ± 4.3 ^{a,b}	65.0 ± 8.9
Height (cm)	170.9 ± 5.4 ^a	179.3 ± 4.1 ^{a,b}	177.7 ± 5.6
Body fat (%)	9.90 ± 2.3 ^a	11.3 ± 1.9 ^a	15.8 ± 7.0
BMI	19.7 ± 1.2	22.0 ± 1.3 ^b	20.6 ± 2.6
Years of training	2.2 ± 0.9	2.4 ± 0.6	—
Training hours/day	3	5	—
Training days/week	6.5	6	—
Energy costs for training (Kcal·d ⁻¹)	1,426.0 ± 654.1	2544.9 ± 155.8 ^b	—

^aSignificant at $P < 0.05$ when compared with untrained group.

^bSignificant at $P < 0.05$ when compared with runners.

Values are mean ± SD.

TABLE 2: Serum thiobarbituric-acid-reactive substances (TBARSs), xanthine oxidase (XO), catalase (CAT), reduced glutathione (GSH), superoxide dismutase (SOD), and total antioxidant capacity (T-AOC) in adolescent runners, cyclists, and untrained subjects are shown.

	Runners (<i>n</i> = 12)	Cyclists (<i>n</i> = 12)	Untrained (<i>n</i> = 12)
TBARS (nmol·mL ⁻¹)	4.85 ± 0.76	4.81 ± 1.04	4.46 ± 1.11
XO (U·L ⁻¹)	16.1 ± 2.1	19.1 ± 1.4 ^{a,b}	16.7 ± 1.3
GSH (mg·L ⁻¹)	15.1 ± 4.5	23.7 ± 9.6 ^{a,b}	12.1 ± 2.9
CAT (U·mL ⁻¹)	1.89 ± 0.55 ^a	2.61 ± 0.92 ^{a,b}	0.53 ± 0.36
T-AOC (U·mL ⁻¹)	15.4 ± 1.6	15.6 ± 2.5	14.3 ± 2.2
SOD (U·mL ⁻¹)	56.7 ± 3.3	58.2 ± 2.9	61.8 ± 11.1

^aSignificant at $P < 0.05$ when compared with untrained group.

^bSignificant at $P < 0.05$ when compared with runners.

Values are mean ± SD.

examine the differences in TBARS, GSH, XO, SOD, CAT, and T-AOC across groups (runners, cyclists, control). Post hoc analyses using Newman-Keuls were performed for cases in which the main effect was significant. Relationships between variables were assessed using simple regression. All tests for statistical significance were standardized at an alpha level of $P < 0.05$, and all results were expressed as mean ± SD.

3. Results

In this study, cyclists displayed higher body weight and height as compared with the control group, whereas the runners were rather short and leaner. Nevertheless, % body fat was similar between the runners and cyclists, and both were lower than in the control group (Table 1). The training hours per day and training days per week among athletes in each group were identical, since cyclists and runners were trained in same teams. The absolute energy costs (i.e., METs) for training in cyclists were higher than that in runners (Table 1) while the intensity level of both exercise trainings was classified as “rigorous” [3].

For the resting serum oxidant status (Table 2), no significant difference was observed in serum TBARS among the

runners, cyclists, and control group, while higher serum XO was found in cyclists as compared with runners and control group ($P < 0.05$). The serum XO between the runners and control group was not significantly different. For the resting serum antioxidant parameters, GSH and CAT were higher in cyclists as compared with runners and control group ($P < 0.05$). The GSH between runners and control group did not significantly differ, whereas the CAT in runners was significantly higher than in the control group ($P < 0.05$). There was no significant difference in serum T-AOC and SOD among the runners, cyclists, and control group ($P > 0.05$).

The average dietary intakes of the runners, cyclists, and control group across consecutive 7 days prior to the blood test are shown in Table 3. The absolute intakes of macronutrients (CHO, protein and fat) as well as the associated total energy intake were significantly higher in the cyclists as compared with those of the runners and control group ($P < 0.05$). The intake of CHO in runners was also higher than that in control group ($P < 0.05$). It is also noteworthy that the cholesterol intake in cyclists was higher than that of control group, while it was relatively lower in runners ($P < 0.05$). Similar to macronutrient intake, the intakes of micronutrient of zinc, iron, manganese, and magnesium

TABLE 3: Daily dietary intakes of study subjects are shown.

	Runners (<i>n</i> = 12)	Cyclists (<i>n</i> = 12)	Untrained (<i>n</i> = 12)
Total energy intake (Kcal)	2354.4 ± 234.7 ^a	3163.3 ± 259.4 ^{a,b}	2133.0 ± 289.0
Protein (g)	83.1 ± 8.95	102.4 ± 10.5 ^{a,b}	86.9 ± 12.1
Protein (%EI)	14.2 ± 1.18 ^a	13.0 ± 0.66 ^a	16.5 ± 2.37
CHO (g)	366.0 ± 49.7 ^a	508.4 ± 40.8 ^{a,b}	296.2 ± 54.7
CHO (%EI)	62.1 ± 3.95 ^a	64.3 ± 2.68 ^a	55.6 ± 7.20
Fat (g)	62.1 ± 10.5	80.4 ± 12.0 ^{a,b}	66.8 ± 22.2
Saturated fat (g)	9.37 ± 2.10	12.1 ± 4.27	10.0 ± 3.64
Monounsaturated fat (g)	18.3 ± 3.48	20.8 ± 5.03	17.8 ± 8.80
Polyunsaturated fat (g)	11.3 ± 2.34	14.6 ± 2.72	13.4 ± 8.67
Cholesterol (mg)	253.1 ± 52.7 ^a	432.5 ± 96.0 ^{a,b}	342.1 ± 119.9
Fat (%EI)	23.8 ± 3.74	21.8 ± 3.61 ^a	28.0 ± 7.52
Fibres (g)	11.6 ± 2.36	11.8 ± 1.75	10.7 ± 3.08
Vitamin A (μg RE)	712.8 ± 247.3	754.0 ± 249.1	779.6 ± 468.3
Vitamin C (mg)	86.3 ± 40.2	86.6 ± 11.1	102.3 ± 43.3
α-Tocopherol (mg)	28.8 ± 6.86	33.3 ± 6.16	31.1 ± 19.4
Selenium (μg)	81.9 ± 13.0 ^a	114.2 ± 17.9 ^b	100.8 ± 28.4
Zinc(mg)	13.5 ± 1.95	16.1 ± 1.20 ^{a,b}	13.6 ± 2.09
Copper (mg)	2.37 ± 0.38	2.92 ± 0.48	2.79 ± 1.12
Iron (mg)	27.6 ± 4.73	31.8 ± 4.05 ^{a,b}	27.3 ± 4.33
Magnesium (mg)	339.5 ± 39.2	413.7 ± 38.0 ^{a,b}	327.7 ± 46.0
Manganese (mg)	7.35 ± 1.22 ^a	7.97 ± 0.98 ^a	5.68 ± 0.88

^aSignificant at $P < 0.05$ when compared with untrained group.

^bSignificant at $P < 0.05$ when compared with runners.

EI: total energy intake, CHO: carbohydrate.

Values are mean ± SD.

were higher in cyclists ($P < 0.05$). The manganese intake in runners was higher than that of control group, while the intake of selenium was relatively lower ($P < 0.05$).

As regards the relationships between nutrient intakes and serum redox status within all the study population ($n = 36$), we observed that GSH was significantly correlated ($P < 0.05$) with CHO ($r = 0.55$), protein ($r = 0.49$), fat ($r = 0.36$), total energy intake ($r = 0.59$), selenium ($r = 0.37$), zinc ($r = 0.39$), iron ($r = 0.36$), and magnesium ($r = 0.36$). Significant correlations were also found between CAT and CHO ($r = 0.67$), protein ($r = 0.39$), total energy intake ($r = 0.64$), magnesium ($r = 0.43$), and manganese ($r = 0.54$). For serum XO, it was significantly correlated with cholesterol ($r = 0.57$). Among serum oxidant and antioxidant biomarkers, serum CAT and GSH were found to be significantly correlated ($r = 0.70$).

4. Discussion

This study evaluated the resting oxidant and antioxidant status of adolescent athletes who took part in professional training of either long-distance run or road cycling, with a training volume comparable to that of adult athletes (Table 1). In comparison to age- and Tanner-stage-matched untrained individuals, there was a trend of higher serum antioxidant status in athletes, whereas the serum oxidant status appeared to be similar. The elevated resting levels of antioxidant biomarkers in the adolescent athletes, likely

secondary to the adaptations to regular endurance training, are in agreement with those reported in adult athletes [7, 13]. These findings in adolescent athletes involved in professional endurance sports training are the first to be described in the current scientific literature to the best of our knowledge.

In the present study, the resting serum TBARS of runners and cyclists appeared not significantly different as compared with that of an untrained group, although a relative higher serum XO was observed in the cyclists (Table 2). The higher serum XO in the cyclists may be attributed to their diet habit rather than chronic exercise-induced increase in the resting enzymatic activity level. The significant correlation of serum XO and cholesterol intake (Figure 1) within our study population as well as the apparent high cholesterol intake in the cyclists (Table 3) may partly explain this difference. This is also in line with a recent notion that diet-induced hypercholesterolemia is associated with increase in XO activity [14]. In this study, the lack of apparent oxidative stress at rest in athletes is in disagreement with that previously observed in adolescent swimmers. Santos-Silva et al. [15] have reported that adolescent swimmers trained 20 hours per week exhibited a higher ratio of resting plasma oxidative stress biomarkers and antioxidant capacity in comparison to that of their untrained counterparts. Similar changes were also observed in children involved in relatively low volume (1 hr session × 4 per week) of swimming training [16]. The absence of marked oxidative stress in athletes in the present study should not be attributed to insufficient

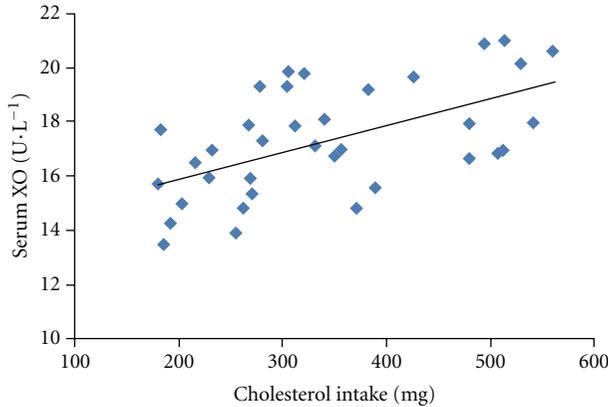


FIGURE 1: The linear relationship ($r = 0.57$, $n = 36$, $P < 0.05$) between cholesterol intake and serum xanthine oxidase (XO) is shown.

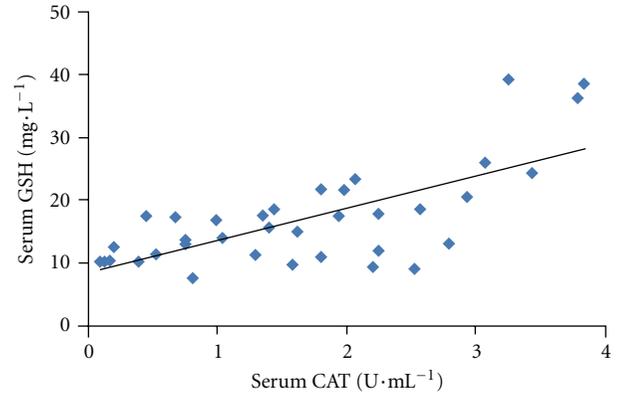


FIGURE 2: The linear relationship ($r = 0.70$, $n = 36$, $P < 0.05$) between serum catalase (CAT) and reduced glutathione (GSH) is shown.

stimulation of physical work to body as the training volumes of the runners and cyclists were much greater than those of the swimmers engaged in the previous studies. It has been reported that the performance of exhaustive one-leg stepping exercise with different contributions of concentric and eccentric contractions in a 1 : 1 versus 1 : 2 ratio of timing would result in distinct level of oxidative stress in adolescents [10]. This suggests that the nature of activity (eccentric versus concentric; land versus aquatic) may be an important factor for the generation of ROS.

Regarding the blood antioxidant status of the runners and cyclists, the relatively high serum GSH and CAT in comparison with those of untrained subjects reveal that augmented antioxidant capacity in adaptation to chronic exercise likely occurs in the athletes. Although the augmentation of antioxidant capacity with chronic exercise has been regularly reported in adult athletes, such training adaptation was equivocal in children and adolescents. Previous studies found that the antioxidant levels of trained adolescent swimmers were not different or even lower in comparison to that of age-matched untrained adolescents [15, 16]. In contrast, Carlsohn et al. [17] noted in adolescent athletes, but not in untrained controls, that antioxidant capacity increased markedly with age-associated increase in training effort. Kabasakalis et al. [18] found that children aged 10–11 years involved in 23-week intense swimming training (covered >2.5 km per session, ≥ 3 sessions per week) could improve their antioxidant capacity in the same manner as reported in adult athletes [19]. This implies that training effort/volume, rather than maturation, dominates the development of antioxidant defence system in children and adolescent athletes. This potential dose-dependent mechanism of adaptation to exercise-induced increase in ROS formation is further supported by our current findings that the serum GSH and CAT are relatively higher in cyclists (Table 2) than in runners, for whom the energy costs for training were much lower than in cyclists (Table 1).

Although resting serum CAT and GSH were greater in athletes, the serum T-AOC and SOD were similar between athletes and controls. These inconsistent changes in resting

antioxidant biomarkers in adaptation to chronic exercise have also been observed in previous studies [18, 20]. The exogenous antioxidant intakes from habitual diets and the type of exercise and its intensity and duration applied during training are all factors that would influence blood antioxidant status in athletes and might partly explain the discrepancies [21]. In the present study, the dietary intakes of vitamin A, vitamin C, and α -tocopherol, which are deeply involved in antioxidant mechanism, did not differ among runners, cyclists, and controls (Table 3). However, macronutrients and other antioxidant nutrients including CHO and manganese were in a greater amount taken by athletes in comparison to controls. It was noted in our study population that serum CAT and GSH were correlated to the intakes of macronutrient of CHO, protein, and fat. Serum CAT was also correlated with the manganese intake and tends to be correlated with the intake of iron ($r = 0.32$, $P = 0.06$), which both function as cofactor for reduction of the antioxidant enzyme in blood [22]. For the GSH, it was correlated with various antioxidant nutrient intakes including magnesium, selenium, and zinc. Magnesium is essential in GSH synthesis [23], while selenium and zinc are associated with endogenous GSH production and maintenance [24, 25]. These findings suggest that the mechanisms for up-regulation of the endogenous antioxidants in adaptation to chronic exercise in athletes, other than dose-dependent mechanism, may also be associated with their habitual intakes of exogenous antioxidant nutrients. However, the adaptive endogenous processes are not well understood. In the present study, the serum CAT and GSH within study population are significantly correlated (Figure 2). This supports the previous concept that antioxidant defences in human act as a coordinated system, with various metabolites and enzymes having synergistic and interdependent effects on one another [26]. The counterbalance effects of each antioxidant on ROS damages may depend on the proper function of other members of the system [27]. The current findings of the correlation among the antioxidant biomarkers suggest that the maintenance of the resting blood redox balance in adolescent runners and cyclists participating in

professional training might partly result from the integrative effect of augmentation of individual antioxidant.

In summary, the resting blood redox balance was well maintained in the adolescent athletes participating in professional endurance sports training, with training volume comparable to that of adult athletes. The maintenance of the redox balance might partly result from the integrative effect of augmentation of individual antioxidant in adaptation to chronic exercise. Such adaptive endogenous processes in athletes might be associated with their habitual intakes of antioxidant nutrients. Notwithstanding the limited number of subjects studied, this investigation has however several strengths. First, although the number of blood redox biomarkers may still not thoughtfully reflect the specific adaptations to chronic exercise, our findings provide for the first time a reasonable information regarding distinct resting serum oxidant and antioxidant status in professional adolescent endurance athletes. Then, the high homogeneity of the study populations allows a very reliable comparison among adolescents engaged in different sports disciplines (e.g., cycling and running), as well as with untrained controls. For future research, further assessment of additional biomarkers (e.g., plasma F₂-isoprostanes, plasma antioxidant vitamins, Trolox-equivalent antioxidant capacity, uric acid) could give us more information about oxidant and antioxidant status of adolescents professional athletes.

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References

- [1] D. M. Cooper, D. Nemet, and P. Galassetti, "Exercise, stress, and inflammation in the growing child: from the bench to the playground," *Current Opinion in Pediatrics*, vol. 16, no. 3, pp. 286–292, 2004.
- [2] B. K. Pedersen and L. Hoffman-Goetz, "Exercise and the immune system: regulation, integration, and adaptation," *Physiological Reviews*, vol. 80, no. 3, pp. 1055–1081, 2000.
- [3] A. Carlsohn, S. Rohn, F. Mayer, and F. J. Schweigert, "Physical activity, antioxidant status, and protein modification in adolescent athletes," *Medicine and Science in Sports and Exercise*, vol. 42, no. 6, pp. 1131–1139, 2010.
- [4] M. J. Jackson, "Reactive oxygen species and redox-regulation of skeletal muscle adaptations to exercise," *Philosophical Transactions of the Royal Society B*, vol. 360, no. 1464, pp. 2285–2291, 2005.
- [5] C. Scheele, S. Nielsen, and B. K. Pedersen, "ROS and myokines promote muscle adaptation to exercise," *Trends in Endocrinology and Metabolism*, vol. 20, no. 3, pp. 95–99, 2009.
- [6] H. Miyazaki, S. Oh-ishi, T. Ookawara et al., "Strenuous endurance training in humans reduces oxidative stress following exhausting exercise," *European Journal of Applied Physiology*, vol. 84, no. 1–2, pp. 1–6, 2001.
- [7] M. J. Jackson, "Free radicals generated by contracting muscle: by-products of metabolism or key regulators of muscle function?" *Free Radical Biology and Medicine*, vol. 44, no. 2, pp. 132–141, 2008.
- [8] S. Sachdev and K. J. A. Davies, "Production, detection, and adaptive responses to free radicals in exercise," *Free Radical Biology and Medicine*, vol. 44, no. 2, pp. 215–223, 2008.
- [9] Y. Tian, J. Nie, T. K. Tong, J. S. Baker, N. E. Thomas, and Q. Shi, "Serum oxidant and antioxidant status during early and late recovery periods following an all-out 21-km run in trained adolescent runners," *European Journal of Applied Physiology*, vol. 110, no. 5, pp. 971–976, 2010.
- [10] J. A. Duarte, J. F. Magalhães, L. Monteiro, A. Almeida-Dias, J. M. C. Soares, and H. J. Appell, "Exercise-induced signs of muscle overuse in children," *International Journal of Sports Medicine*, vol. 20, no. 2, pp. 103–108, 1999.
- [11] B. E. Ainsworth, W. L. Haskell, S. D. Herrmann et al., "2011 compendium of physical activities: a second update of codes and MET values," *Medicine and Science in Sports and Exercise*, vol. 43, no. 8, pp. 1575–1581, 2011.
- [12] I. F. F. Benzie and J. J. Strain, "Ferric reducing/antioxidant power assay: direct measure of total antioxidant activity of biological fluids and modified version for simultaneous measurement of total antioxidant power and ascorbic acid concentration," *Methods in Enzymology*, vol. 299, pp. 15–27, 1998.
- [13] F. Marzatico, O. Pansarasa, L. Bertorelli, L. Somenzini, and G. Della Valle, "Blood free radical antioxidant enzymes and lipid peroxides following long-distance and lactacidemic performances in highly trained aerobic and sprint athletes," *Journal of Sports Medicine and Physical Fitness*, vol. 37, no. 4, pp. 235–239, 1997.
- [14] J.-G. Park and G. T. Oh, "The role of peroxidases in the pathogenesis of atherosclerosis," *BMB Reports*, vol. 44, no. 8, pp. 497–505, 2011.
- [15] A. Santos-Silva, M. I. Rebelo, E. M. B. Castro et al., "Leukocyte activation, erythrocyte damage, lipid profile and oxidative stress imposed by high competition physical exercise in adolescents," *Clinica Chimica Acta*, vol. 306, no. 1–2, pp. 119–126, 2001.
- [16] S. Gougoura, M. G. Nikolaidis, I. A. Kostaropoulos, A. Z. Jamurtas, G. Koukoulis, and D. Kouretas, "Increased oxidative stress indices in the blood of child swimmers," *European Journal of Applied Physiology*, vol. 100, no. 2, pp. 235–239, 2007.
- [17] A. Carlsohn, S. Rohn, F. Bittmann, J. Raila, F. Mayer, and F. J. Schweigert, "Exercise increases the plasma antioxidant capacity of adolescent athletes," *Annals of Nutrition and Metabolism*, vol. 53, no. 2, pp. 96–103, 2008.
- [18] A. Kabasakalis, K. Kalitsis, M. G. Nikolaidis et al., "Redox, iron, and nutritional status of children during swimming training," *Journal of Science and Medicine in Sport*, vol. 12, no. 6, pp. 691–696, 2009.
- [19] J. Finaud, G. Lac, and E. Filaire, "Oxidative stress: relationship with exercise and training," *Sports Medicine*, vol. 36, no. 4, pp. 327–358, 2006.
- [20] T. A. Watson, L. K. MacDonald-Wicks, and M. L. Garg, "Oxidative stress and antioxidants in athletes undertaking regular exercise training," *International Journal of Sport Nutrition and Exercise Metabolism*, vol. 15, no. 2, pp. 131–146, 2005.
- [21] L. L. Ji, "Antioxidants and oxidative stress in exercise," *Proceedings of the Society for Experimental Biology and Medicine*, vol. 222, no. 3, pp. 283–292, 1999.
- [22] P. Chelikani, I. Fita, and P. C. Loewen, "Diversity of structures and properties among catalases," *Cellular and Molecular Life Sciences*, vol. 61, no. 2, pp. 192–208, 2004.
- [23] R. K. Rude and M. E. Shils, "Magnesium," in *Modern Nutrition in Health and Disease*, M. E. Shils, M. Shike, A. C. Ross, B. Caballero, and R. J. Cousins, Eds., pp. 223–247, Lippincott Williams & Wilkins, Baltimore, Md, USA, 10th edition, 2006.

- [24] B. J. Mills, R. D. Lindeman, and C. A. Lang, "Effect of zinc deficiency on blood glutathione levels," *Journal of Nutrition*, vol. 111, no. 6, pp. 1098–1102, 1981.
- [25] P. Anand, D. Rajakumar, M. Jeraud, A. John William Felix, and T. Balasubramanian, "Effects of taurine on glutathione peroxidase, glutathione reductase and reduced glutathione levels in rats," *Pakistan Journal of Biological Sciences*, vol. 14, no. 3, pp. 219–225, 2011.
- [26] C. K. Sen, "Oxidants and antioxidants in exercise," *Journal of Applied Physiology*, vol. 79, no. 3, pp. 675–686, 1995.
- [27] S. Vertuani, A. Angusti, and S. Manfredini, "The antioxidants and pro-antioxidants network: an overview," *Current Pharmaceutical Design*, vol. 10, no. 14, pp. 1677–1694, 2004.



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