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THE COMPARISON OF ANTIOXIDANT STATUS OF ENDURANCE ATHLETES OXIDATIVE STRESS

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Abstract:
The activity levels of superoxide dismutase (SOD, EC 1.15.1.1), Catalase (EC 1.11.1.6) and status of lipid peroxidation in male athletes and deskbound population was investigated. Both the groups i.e. deskbound (D group) and athlete (A group) consist of thirty subjects each. Sedentary subjects are included in group D who are not involved in regular physical activity and Group A includes endurance athletes of 15 Km walk involved in regular physical activity. The blood serum was used for activity levels of enzymes and other parameters. The serum catalse, superoxide dismutase & malondialdehyde were significantly (P< 0.05) high in group A (athletes) as compare to group D (Deskbound). Due to continuous physical activity both oxidative stress and antioxidant capacity increased in athletes as compared with deskbound controls. Pakistani endurance athletes have increased markers for malondialdehyde and antioxidant enzyme compared to their desktop counterparts. Increased lipid peroxidation and the antioxidant status of Pakistani athletes may be due to regular physical activity and prolonged endurance.

Keywords: *Comparison, Antioxidant, Status of Endurance, Athletes, Oxidative, Stress.*

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INTRODUCTION:

Free radicals are constantly produced by cells and ROS as portion of metabolic procedures. A detailed antioxidant defense system neutralizes a number of non-enzymatic antioxidants such as superoxide dismutase, glutathione peroxidase, catalase, and vitamins C, A and E, flavonoids, glutathione and ubiquinone. May reason an inequality among ROS and antioxidants known as oxidative stress (Urso and Clarkson). Many health benefits are associated with regular exercise, but they can be seen as intense physical stress that causes increased damage to oxidative cells (Bloomer et al.). High formation of responsive oxygen types can be answerable for a number of biological and chemical fluctuations that happen during workout (Alessio,). Tiring physical exercise has been found to lower antioxidant stages and rise markers of lipid peroxidation in target tissues and lifeblood (Vasankari et al. Davies et al.). The spread of these free radicals can reduce the function of the affected cells and reduce the ability of the muscles to continue working. Hydrogen peroxide is a harmful byproduct of many normal metabolic processes; To avoid damage, quickly transform them into other less hazardous substances. To this end, cells use catalase (EC 1.11.1.6) to quickly catalyze the decomposition of hydrogen peroxide into less reactive water and oxygen molecules (Gaetani et al.). Superoxide dismutase is a class of enzymes that catalyze the decomposition of peroxide into oxygen and hydrogen peroxide. Therefore, superoxide dismutase is an significant antioxidant resistance in almost all cells showing to oxygen. Malonicdehyde is a natural product of lipid peroxidation. Lipid peroxidation is used as an pointer of oxidative stress in cells and tissues (Yagi, Armstrong and Brown). Lipid peroxides of polyunsaturated fatty acids are replaced and decomposed to form a complex series of compounds such as malodialdehyde. The purpose of this study is to understand the lipid peroxidation and antioxidant status of Pakistani endurance athletes by computer type, age and gender equivalent.

METHOD:

There are two groups of 35 male endurance athletes (Cayman TBARS Medium Tiobarbituric Acid Test Kit), providing a simple, repeatable and standard tool for assessing lipid peroxidation in serum. The MDA and TBA reaction at high temperature (90-100 ° C) and acidic conditions was measured at 530-540 nm. Control was carried out at room temperature. When ten human plasma / serum samples were analyzed on the same day, the coefficient of variation for the experiment was 5.5%. Eight series of human plasma / serum samples over seven different days under the same experimental conditions. During the analysis, the coefficient of variation between analyzes is 5.9% (Ohkawa et al., Draper et al.

RESULTS:

Table 1 shows the mean SD and t value of malonic aldehyde and antioxidants from both groups as shown in Table n. If ° 1, there is a significant difference between the table group (15.83 ± 6.37) and the group of athletes (46.86 ± 8, 96) at the level of malonic aldehyde with a significant level of 5% (15.71). It should be emphasized that the level of catalase between the table (9.30 ± 2.95) and the group of athletes (22.97 ± 5.55) shows a significant difference at the level of significance of 5% (12.10). The level of superoxide dismutase between the table (0.09 ± 0.05) and the group of athletes (0.21 ± 0.18) showed a significant difference at the level of significance of 5% (3.63). age ± SD, 23.18 ± 1.83 years) and 30 male table controls (mean age ± SD, 24.55 ± 1.8 years). Endurance athletes belong to 15 km of athletics competitions. On the other hand, control

people are tied to the table, they do not participate in regular exercise, but are healthy. People with any disease and smoking were excluded from the study. Written consent to voluntary preparation for this study was obtained from each participant. Five (5) ml venous blood was collected from each patient after 12-hour fasting overnight.

Table-1

	Group D	Group A	
BIOCHEMICAL PARAMETERS	(N=30) Mean±SD	(N=30) Mean±SD	t-value
MALONDIALDEHYDE (nmole/ml)	15.83±6.37	46.86±8.96	15.71*
CATALASE (nmole/min./ml)	9.30±2.95	22.97±5.55 12.10*	CATALASE (nmole/min./ml)
SUPEROXIDE DISMUTASE(U/ml) 0.09±0.05	0.21±0.18	3.63*	SUPEROXIDE DISMUTASE(U/ml) 0.09±0.05

Malonodialdehyde catalase and superoxide dismutase activities in serum were determined by a colorimetric method using Cayman test kits. The effect obtained using all parameters was analyzed by means of the t test to discover the difference between the two groups. The Cayman Catalase Test Kit uses the CAT oxidation function to determine enzyme activity. The process is grounded on the response of the enzyme with methanol in the existence of an optimal concentration of H2O2. Formaldehyde formed is measured colorimetrically using chromogenic amino-3-hydrazine-5-mercapto-1,2,4-triazole (Purpald). Purpald specifically forms a bicyclic heterocycle with aldehydes that change from color to purple by oxidation. The test temperature is 25 ° C and the catalase activity is measured at 540 nm. When a sequences of 45 catalase dimensions were made on the similar day, the coefficient of variation in the test was 3.8%. When a series of 45 catalase measurements were carried out for five dissimilar days under the same tentative conditions, the coefficient of variation between samples was 9.9% (Johansson and Borg, 1988). The Cayman Oxide Peroxide Removal Test Kits use a tetrazolium salt to perceive peroxide radicals produced by xanthine oxidase and hypoxanthine. One SOD unit is well-defined as the volume of enzyme required to reduce the superoxide radical by 50%. The test temperature is 25ºC. SOD activity is measured at 530-540 nm. When a sequence of 60 standard SOD measurements were made on the same day, the coefficient of variation in the test was 3.2%. When a series of 60 standard SOD dimensions were done over five diverse days under the similar investigational conditions, the coefficient of variation between samples was 3.7% (Marklund).

DISCUSSION:

In this study, we checked the effect of exercise intensity on changes in plasma oxidative stress and antioxidant capacity. Exercise intensity was based on the EC established by HSBP. We found that the plasma concentration of d-ROM did not change in C, LI or MI tests. To support this study, Goto et al.) reported that a 30-minute ergometric cycle at 50% V ・ O2max did not increase the concentration of low density lipoprotein modified with malonic aldehyde as a marker of lipid peroxidation. These data show that at these intensity exercises, oxidative blood stress does not exceed the blood's antioxidant capacity to extinguish ROS. Whereas Lovlin et al.) reported that maximum exhaustion efforts increase plasma lipid peroxides, while Goto et al.) reported that a 30-minute ergometer cycle at 75% V ・ O2max increased serum concentration. lipid peroxidation. In addition, Seifi-Skishahr et al. It has been observed that 30 minutes of exercise at 60% V with O2max causes less lipid peroxidation compared to 30 minutes of exercise at 75% V ・ O2max). Some studies suggest that exercise intensity may be more important than total energy expenditure in response to oxidative stress after exercise). These results suggest that increased ROS production during high-intensity exercise may exceed the endogenous antioxidant capacity.

In this study, the plasma d-ROM concentration immediately after and 30 minutes after exercise was significantly higher in the HI study than at the pre-exercise level. High intensity exercises over AT will increase catecholamines. This adrenal stimulation may be another mechanism that increases oxidative stress associated with catecholamine autooxidation. Previous studies also suggest that lactate metabolism and oxidative stress may be associated. Lactic acid can transform a somewhat harmful free radical (superoxide radical) into a much more harmful peroxide. In this study, lactic acid plasma levels after the HI test were higher than in other tests that may be associated with high oxidative stress.

Higher intensity aerobic exercise is likely to lead to increased oxygen absorption and, consequently, to ROS production in mitochondria. It should be remembered that the source of ROS production in high intensity exercises is not only the mitochondrial respiration chain, but also neutrophils and monocytes. MPO is a common marker of neutrophil-induced degranulation. MPO produces a large number of ROS, which cause oxidative damage to proteins, lipids and DNA. MPO performance depends on the intensity of the exercise: Suzuki et al. reported increased ROS and MPO production after high-intensity exercise. Although none of the studies found a significant change in MPO plasma levels, we observed that the percentage change in pre-exercise MPO concentration to post-exercise score was higher than in C tests immediately after the HI test. and MI. Therefore, d-ROM and MPO plasma concentrations did not change after resistance exercises below AT; This suggests that ROS may be produced from MPO secreted by higher intensity neutrophils when resistance to AT increases after exercise.

Our study showed that heartrotectin levels did not change significantly in 70-130% AT exercises. Mooren et al. reported a significant increase in plasma calcium protection immediately after exercise with acute resistance and during convalescence. In our study, the lack of a significant change in the heart can be attributed to the shorter duration of exercise. Most previous studies have looked at the effects of resistance exercises on heartrotectin for longer hours (e.g. Marathon 6.32). Therefore, our results show that the duration of exercise has a greater impact on

changes in markers of leukocyte activation during exercise than exercise intensity.

Antioxidants such as vitamin C and vitamin E in the blood help reduce the severity of oxidative stress by creating fewer active radicals or by quenching the free radical chain reaction. Fatouros et al. reported that the non-enzymatic antioxidant capacity, measured by the same method as ours (TEAC), increased until exhaustion after a gradual diagnostic test on a treadmill. Seifi-Skishahr et al. also reported that moderate intensity exercises significantly increased uric acid, which may act as an antioxidant. In the HI study, we found a significant increase in TEAC plasma levels immediately after exercise. These results show that the non-enzymatic antioxidant capacity is increased in resistance exercises above AT. To assess the endogenous antioxidant defense system, an enzymatic and nonenzymatic antioxidant defense system should be investigated simultaneously. Although some studies report changes in SOD and CAT after exercise with sharp resistance, the results are controversial. We didn't find any significant changes in SOD and CAT plasma movement in various exercise studies. Neubauer et al. recently reported that GPX in an enzymatic antioxidant defense system can be very sensitive to resistance exercises in general, and especially to high-intensity exercises. In our study, plasma GPX activity increased immediately after and 30 minutes after pre-workout HI exercises, and a significant increase in plasma GPX activity occurred immediately after exercise. In the MI process. It is also possible that the increase in GPX is the result of cell damage (i.e., cytoplasm release from erythrocytes, etc.) rather than a specific antioxidant response. The mechanisms underlying GPX change require more work.

CONCLUSION:

Pakistani endurance athletes have increased markers for malondialdehyde and antioxidant enzyme compared to their desktop counterparts. Increased lipid peroxidation and the antioxidant status of Pakistani athletes may be due to regular physical activity and prolonged endurance.

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