

Effect of Intensive Exercise and Training on Exercise-Induced Oxidative Stress among Standard Athletes

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Abstract

The study was conducted to find the effect of intensive exercise, especially aerobic workout on the selected biochemical variables related to exercise-induced oxidative stress and the effect of training-whether any kind of adaptation occurs due to regular training on the selected variables, and to study the variations in the level of oxidative stress due to training, through the selected variables. The subjects for this study were eighteen boys who were from Canoeing trainees of the Sports Authority of India (SAI) Aquatic Complex, established at Alappuzha, Kerala. This consisted of two groups randomly selected. Each group consisted of nine subjects: one control group and the other experimental group. The experimental group will undergo very rigorous training (12 kms canoeing) for eight weeks whereas the control group was kept sedentary. This design helped the investigator to find out the immediate effect of exercise and also the effect of long term training on the selected biochemical variables- 10 ml of venous blood was collected into heparinised test tubes, from 18 subjects, (First Test-Pre and Post & Second Test Pre & Post). After blood collection the students were asked to row or canoe for a distance of 12 km. Keeping their heart rate in 160 bpm and the stroke frequency 25spm. The data of criterion variables like Superoxide Dismutase (SOD) Vitamin E were selected as the variables for this study. The collected data was statistically analysed by using Analysis of Covariance (ANACOVA). Results of this study on SOD activity of the experimental group was higher after acute exercise. There was significant difference between the experimental group and the control group in SOD activity as a result of training. Training resulted in elevating the SOD activity of the experimental group. Level of Vitamin E decreased significantly after acute exercise. Training resulted in significant reduction of resting Vitamin E level. After training vitamin E level of experimental group decreased significantly when compared with the control group. Hence it may be inferred that the oxidative stress occurs as a result of acute exercise.

INTRODUCTION

Sports and games occupy a unique position in the modern competitive world. Physical fitness is a must for all sports and games. It provides the capacity for doing all kinds of activities. Greater the fitness, better will be the performance. Fitness can be achieved only through regular physical exercise and training.

Moderate physical exercise has been shown to be healthful and beneficial. However several studies indicate that intense exercise, especially in untrained subjects leads to oxidative damage to cell membranes. This is contrary to the common belief that exercise is always beneficial.

Free radicals are highly reactive oxygen species that are formed in the human body as a result of various mechanisms during radiation and pathological conditions. They are also produced as also produced as a result of strenuous physical exercise.

Exercise has a unique relation to the free radical theory. Physical exercise causes upto 20 fold increase in whole body oxygen consumption and upto 200 fold in exercising muscles. During exercise, free radicals are produced as a result of electron leak in the mitochondrial electron transport chain and also through ischemia -reperfusion mechanisms. Considering that thousands of free radicals are produced in each resting cell everyday, it is tempting to speculate on the number of free radicals that may be produced as a result of elevated metabolism. Furthermore, during exercise, damage to active tissues is likely to occur and oxidative stress reactions are known to increase in damaged tissues.

Free radicals are highly reactive. They destroy the cell walls and cause the contents of the cell to leak into the blood stream. Thus they destroy the cell structure and integrity and thereby cause reduction in muscle tone and cause pathogenic condition. This process is known as lipid peroxidation. If it is ignored, it may lead to permanent damage that might lead to stagnation in performance.

The human body has its own defensive mechanisms against free radical toxicity or lipid peroxidation. There are different chemical substances known as antioxidants which absorb the free radicals or convert them to harmless non-radical species and when they are produced. There are two types of antioxidants - enzymatic and non-enzymatic. Major enzymatic antioxidants are Superoxide Dismutase (SOD), Glutathione Peroxidase (GPx) and Catalase. Glutathione (GSH) and Vitamin E are non - enzymatic antioxidants.

To a certain extent, body can adapt itself against free radical generation through regular training. Rate of production of free radicals is drastically reduced as a result of training. Training also causes increase in the levels of activities of antioxidants in the blood, muscles, and, liver of the trained individuals, thereby minimising the toxic effects of lipid peroxidation.

Canoeing is aquatic sports that required extreme levels of fitness. They are highly strenuous, predominantly aerobic, middle and long distance events. So their training will induce strenuous exercises and rowing/ canoeing for tens of kilometers almost every day. Almost every muscle in the body are utilized while rowing or canoeing. Hence during rowing or canoeing whole body oxygen consumption will increase drastically that may induce large amounts of free radicals generation.

Methodology

The subjects for this study were eighteen boys who were from Canoeing trainees of the Sports Authority of India (SAI) Aquatic Complex, established at Alappuzha, Kerala. This consisted of two groups randomly selected. Each group consisted of nine subjects one control group and the other experimental group. The experimental group will undergo very rigorous training (12kms canoeing) for eight weeks whereas the control group was kept sedentary. This design helped the investigator to find out the immediate effect of exercise and also the effect of long

term training on the selected biochemical variables- 10 ml of venous blood was collected into heparinised test tubes, from 18 subjects, (First Test-Pre and Post & Second Test Pre & Post). After blood collection the students were asked to row or canoe for a distance of 12km. Keeping their heart rate in 160 bpm and the stroke frequency 25spm. The data of criterion variables like Superoxide Dismutase (SOD) Vitamin E, were selected as the variables for this study. The collected data was statistically analysed by using Analysis of Covariance (ANACOVA).

Analysis of the study and the results of the study

Superoxide Dismutase (SOD)

The mean value of Superoxide dismutase of the control group and the experimental group before and after training are presented in this Table

Acute-exercise induced changes and the effect of training on Superoxide Dismutase (SOD)

	Control Group		Experimental group		Sum of square	Df	Mean square	'F'
	Before Acute	After Acute	Before Acute	After Acute				
Before Training	2.5973	2.267	2.8671	3.004	B:2.7016	1	2.7016	
Mean Difference	0.3303		0.1369	W: 7.1041	12	0.5920		4.5635
	t=3.4*		t=0.96					
After Training	2.5961	2.4376	2.8727	4.2304	B: 5.0328	1	5.0328	
Mean Difference	0.1586		1.3577	W:6.4433	12	0.5369		9.37*
	t=0.9		t=3.88*					
Adjusted Mean Difference	0.075		1.441		B:4.73	1	4.73	
					W: 6.19	11	0.56	8.41*

Significant at 0.05 level of confidence

SOD is measured in units/mg of Hb

Table values of 'F' ratio required for significance at .05 level

df (1,11) - 4.84

df (1,12) - 4.75

Table value of 't' ratio required for significant at .05 level , df 6 - 2.45

Table showing the result of paired 't' test on SOD .

Experimental Group	Before Training	After Training	Mean Difference	't'
at Rest	2.8671	2.8727	0.0056	1.02

*Significant at .05 level of confidence

SOD activity is measured in units/mg of Hb

Table value of 't' required for significance at df '6' is 2.45.

SOD activity of the experimental group at resting state, before training was 2.8671 and that after nine weeks training was 2.8727 with a mean difference of 0.0056. The obtained 't' value was 1.02 and it was not significant at .05 level of

confidence. This indicated that the given nine weeks of training did not significantly alter the resting state SOD activity of the experimental group.

Vitamin E

The mean values of vitamin E of the control group and the experimental group before and after training are presented in table XV

Acute - exercise induced changes and the effect of training on vitamin E

	Control Group		Experimental group		Sum of square	Df	Mean square	'F'
	Before Acute Exercise	After Acute Exercise	Before Acute Exercise	After Acute Exercise				
Before Training	1.6853	1.134	1.4664	1.0093	B:0.031	1	0.031	0.51
Mean Difference	0.5513 t=5.49*		0.4571 W: 0.7292 t=5.36*		12	0.0608		
After Training	1.6837	1.1467	0.8523	0.8127	B: 0.86	1	0.86	19.09*
Mean Difference	0.537 t=5*		0.039 t=1.04		W:0.5443	12	0.0454	
Adjusted Mean					B:0.63	1	0.63	
Difference	0.505		0.072		W: 0.21	11	0	33.52*

*Significant at 0.05 level of confidence

Vitamin E is measured in mg/100ml

Table values of 'F' ratio required for significance at .05 level

df (1,11) - 4.84

df (1,12) - 4.75

Table value of 't' ratio required for significance at .05 level for df 6 is 2.45

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Table showing the results of the paired 't' test on Vitamin E

Experimental Group	Before Training	After Training	Mean Difference	't'
at Rest	1.4664	0.8523	0.6141	4.57

*Significant at .05 level of confidence

Vitamin E is measured in Mg/100ml

Table value of 't' required for significance at .05 level, for df 6 is 2.45

Level of Vitamin E of the experimental group at resting state before training was 1.4664 and that training was 0.8523 a with a mean difference of 0.6141. The obtained 't' value was 4.57 and it was significant at .05 level of confidence. This indicated that the given nine weeks of training resulted in significantly reducing the level of Vitamin E. Naturally training should elevate the resting level of Vitamin E as protection against free radical toxicity.

DISCUSSION

Superoxide Dismutase (SOD)

There was significant difference between the control group and the experimental group on SOD activity before commencement of training. It may be due to the uneven distribution of SOD in the groups. Moreover, when the net result of training would be calculated using analysis of covariance, the initial mean differences would be nullified.

Acute exercise resulted in reduction of SOD activity of the control group before and after training. It may be due to the continuous scavenging of free radicals by SOD as and when they were formed as result of exercise.

For experimental group before training, acute exercise resulted in slight increase in SOD activity. But after training for six weeks, SOD activity was significantly elevated as a result of acute exercise. This may be due to the body adaptive mechanism to training. When free radicals are produced continuously as a result of acute exercise and training, body's antioxidant defence mechanism will elevate SOD activity to counter the toxic free radicals, SOD being the most important and primary antioxidant enzyme.

When considering the effect of training on SOD activity, as a result of the given six weeks of training, the experimental group had significantly higher SOD activity as compared to the sedentary control group. Here also the above mentioned reasons hold good.

Vitamin E

Vitamin E is the major lipid soluble chain breaking non enzymatic free radicals scavenging antioxidant found in biological membranes. By scavenging oxygen free radicals Vitamin E interrupts cascade reaction of free radicals production. It thus maintains the structural and functional integrity of cells. Because it is present in cell membranes, it is more susceptible to free radicals attack.

Acute exercise induced significant reduction in the levels of Vit E both for control group and experimental group, before and after training. Only in the case of experimental group, after training the reduction was not significant. Insignificant reduction in Vitamin E level for experimental group may be due to the reduced resting state Vitamin E level after training. Significant reduction in Vitamin E levels due to acute exercise is indicative of excessive free radicals generation over and above the natural antioxidant defense system of the body and hence increased lipid peroxidation. Since it is not an enzyme, amount of Vit E decreases with increasing scavenger action. Hence level of Vitamin E goes down after acute exercise. This is in accordance with the following report.

As a result of eight weeks of training there was significant difference between the control group and the experimental group in the case of Vitamin E. After training, experimental group had significantly lower levels of Vitamin E as compared to control group.

Similarly after training, experimental group had significantly reduced level of Vitamin E at resting condition as compared to the resting state Vitamin E level before training. Naturally resting Vitamin E level has to be more after training. These results indicated that training resulted in depletion of Vitamin E level of the experimental

group. Hence it may be inferred that experimental group lacked Vitamin E in their diet during training.

From the results it is clear that Vitamin E should be supplemented to the trainees regularly through diet during training.

Since Vitamin E levels have been shown to decrease in mitochondria after exercise, it appears that supplementation of vitamin E may be necessary in certain muscles to break up the chain reaction of free radicals in cells membranes. Goldfarb et al, in their study on rats found that supplementation of Vitamin E was apparently successful in preventing the increase in lipid peroxidation in the muscle. It was also concluded that vitamin E which is a lipid soluble antioxidant was effective in preventing the exercise- induced increase in lipid hydroperoxides in plasma and TBARS in the muscles. This results suggest that vitamin E was able to protect muscles from exercise-induced oxidative stress (Goldfarb et al)

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