Effects of Sustained Training on Exercise-Induced Oxidative Stress among Top Level Rowers

S.Manoj[a], Joji M. Philip[b], Sajeev Jos [c]

[a]Associate Professor- Department of Physical Education ,NSS Hindu College Changanacherry, Kerala, India 686102,

[b]Assistant Professor- Department of Physical Education , K G College, Pampady College, Pampady, Kerala, India 686502,

[c]Assistant Professor- Department of Physical Education, St.Albert's College,Ernakulam, Keral, India- 682802

Abstract

The study was conducted to determine the effects of sustained training in high intensity, aerobic workout on the selected biochemical variables related to exercise-induced oxidative stress and the effect of training-whether any kind of acclimatization occurs due to regular training on the selected variables, and to determin how much changes are occuring in the level of oxidative stress due to training, through the selected variables.For conducting this study Sixteen boys were selected from Rowing 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 eight subjects one control group and the other experimental group. The experimental group will undergo very rigorous training (12kms rowing) for eight weeks whereas the control group was kept sedentary. This method helped the researcher to determin the immediate effect of exercise and also the effect of sustained training on the selected biochemical variables- 15 ml of venous blood was collected into heparinised test tubes, from 16 subjects, (First Test-Pre and Post & Second Test Pre & Post). After blood collection the students were asked to row for a distance of 12 km. Keeping their heart rate in 160bpm and the stroke frequency 24spm. The data of criterion variables like Malonedialdehyde (MDA), Membrane Malonedialdehyde (Membrane MDA) , and Creatine Kinase (CK) were selected as the variables for this study.The collected data was statistically analysed by using Analysis of Coariance (ANACOVA). Results of this study on CK indicate that acute exercise cause significant increase in the amount of CK in blood both for control group as well as experimental group. This was true for both the groups before training and after training. For the control group level of CK after acute exercise was almost same before and after training. But for experimental group condition of CK (due to acute exercise) after training was much lower as compared to the same state before training. From the above report it is clear that elevation of CK is the result of muscle damage. Muscle damage might have occured due to lipid peroxidation by the free radicals that might have been produced due to acute strenuous exercise. Hence it may be inferred that the oxidative stress occurs as a result of acute exercise.

Introduction

Physical fitness is a must for all sports and games. It provides the capacity for doing all kinds of activities. The greater the physical fitness, the better will be the physical endurance, precision of movement performance and capacity for recovery which are highly essential for delivering top performance in any activity. The general health and the ability to excel in sports and games depends mainly on the athletes' physical fitness levels. Fitness can be achieved only through regular physical exercise and training.

Oxidative stress occur as a result of the metabolic generation of reactive oxygen species, most of which are free radicals. Reactive oxygen species are generated continuously as by products of aerobic metabolism (John A Smith, 1995).

Free radicals are highly reactive oxygen species that are formed in the human body as a result of various mechanisms. Radiation from the sun produces free radicals from the basic components of the atmosphere which then combine to form amino acids and other molecules characteristic of living cell. Free radicals are produced in the body during disease conditions. They are also produced as a result of strenuous exercise.

The production of high levels of reactive oxygen species (ROS) in cells promotes redox disturbances leading to oxidative damage to cellular components. Indeed, it is clear that chronic oxidative damage is associated with the pathogenesis of cancer, cardiovascular disease, diabetes mellitus, hypertension, and several neurodegenerative diseases.

Interestingly, while regular physical activity promotes health benefits, rigorous and/or prolonged exercise results in an acute increase in the production of ROS as evidenced by elevated biomarkers of oxidative damage in both blood and skeletal muscles.5 The fact that muscular exercise promotes ROS production appears enigmatic because regular exercise is the only health behavior associated with a decrease in all-cause mortality in humans.6 This review addresses this exercise/oxidative stress paradox by discussing the cellular consequences of exercise-induced ROS production. We begin with a review of the primary oxidants produced in cells followed by a summary of cellular antioxidant systems. We then discuss the sources of ROS production during exercise and debate the question of whether exercise-induced ROS production is beneficial or harmful to health.

Oxidative stress occur as a result of the metabolic generation of reactive oxygen species, most of which are free radicals. Reactive oxygen species are generated continuously as by products of aerobic metabolism (John A Smith, 1995).

Several studies that exercise can trigger antioxidant depletion and cause oxidative damage to a variety of cell types, including muscle, liver and RBCs. During exercise when the oxygen flux through the circulation increases at least 10 fold and it can increase 200 fold in some muscle fibres, free radical generation may increase in proportion to the oxygen uptake induced by exercise.

Since the 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 all the structural integrity of the cells and thereby cause reduction in muscle tone and cause pathogenic condition. The damage caused to the living tissues by toxic free radicals is known as lipid peroxidation. If it is ignored it may lead to permanent damage that may lead to stagnation in performance.

The human body has its own defensive mechanisms against free radical toxicity or lipid peroxidation. There are different types of chemical substances known as antioxidants in the human body. These antioxidants absorb the free radicals or convert them to harmless non-radical species as and when they are produced.

The human body can adapt itself to fight against lipid peroxidation or exercise induced oxidative stress as a result of training. Training is the process of bringing a person or a group to an agreed standard of proficiency by practice and instruction. According to Fox, sports training is a programmed of exercise designed to improve the skills and increase the energy capacities of an athlete for a particular event (Fox, 1984).

The best training programme is the one which increases the desired quality at a higher rate without causing unwanted effects. To enhance physiological improvement effectively,

specific exercise and overload must be followed. Numerous training procedures are in practice to develop each and every motor fitness factors at various levels.

Three primary antioxidant strategies are used to protect cells against ROS-mediated damage. First, numerous low-molecular weight molecules capable of scavenging ROS exist in both the extracellular space and within cells. Second, some enzymatic antioxidants act by converting ROS into less reactive molecules; this limits oxidation and prevents the transformation of these ROS to more damaging species. A final antioxidant strategy involves the binding of pro-oxidant transition metals (e.g., iron and copper) via metal binding proteins; these chelating molecules prevent these transition metals from participating in ROS formation.

Methodology

The subjects for this study were sixteen boys who were Rowingtrainees of the Sports Authority of India (SAI) Aquatic Complex, established at Alappuzha, Kerala.This consisted of two groups randomly selected Each group consisted of eight subjects one control group and the other experimental group. The experimental group will undergo for sustained rigorous training (12kms rowing) for eight weeks whereas the control group was kept sedentary. This method helped the researcher to find out the immediate effect of exercise and also the effect of long term training on the selected biochemical variables- 15 ml of venous blood was collected into heparinised test tubes, from 16 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 24spm. The data of criterion variables likeMalonedialdehyde (MDA), Membrane Malonedialdehyde (Membrane MDA) , and Creatine Kinase (CK) were selected as the variables for this study.

Analysis of the study and the results of the study

The collected data on Malonedialdehyde (MDA), Membrane Malonedialdehyde (Membrane MDA) , and Creatine Kinase (CK) of the control group and the experimental group, before and after the training programme were statistically treated and the results are presented below.

Creatine Kinase (CK)

The mean values of Creatine Kinase of the control group and the experimental group before and after training are presented in Table 1.

TABLE -1

Acute- Exercise Induced Changes and the effect of training on Creatine Kinase (CK)

	Control Group		**Experimental group**		Sum of square	Df	Mean square	**'F'**
	Before Acute Exercise	After Acute Exercise	Before Acute Exercise	After Acute Exercise		Exercise		
Before Training	233.5714 401.7857		316.8571	221.7143		315.7143	B:401.78571	0.3113
Mean Difference	83.2857 t=6.40*		94.00 t=6.65*		W: 15487.428612		1290.6190	

After Training	235.2857	319.5714	213.2857	289.7143	B: 216.07141	216.0714
						0.2116
Mean Difference	84.2857	76.4286	W:12253.14	12	1021.0952	
	t=6.98*	t=6.33*				
Adjusted Mean			B:10112.13	1	1012.13	
Difference	88.969	71.745				26.83*
			W: 414.95	11	37.72	

*Significant at .05 level of confidence

CK is measured in IU/Litre

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

df (1,11) - 4.84

df (1,12) - 4.75

Table value of 't' ratio required for significance ar. .05 level df 6 - 2.45

Before commencement of training the level of CK control group at resting condition was 233.5714 and that immediately after acute exercise was 316.8571. The mean difference was 83.2857 which was significant at .05 level of confidence as denoted by the 't' value 6.40. This meant that due to the acute exercise done, the level of CK of the control group had increased by 83.2857. IU/ Litre.

****

After six weeks during which the control group was kept sedentary and the experimental group was undergoing training, level of CK of the control group at rest was 235.2857 and that immediately after acute exercise was 319.5714. The mean difference was 84.2857 and it was significant at .05 level of confidence (t=6.98). This indicated that there was a significant increase in the level of CK by 84.2857 I.U due to the acute exercise for the control group.

Level of CK for the experimental group at rest after training was 213.2857 and that after acute exercise was 289.7143 with a mean difference of 76.4286 which was significant at .05 level of confidence (t=6.33). Although there was a significant rise in the level of CK after acute exercise, after training, the rate of increase was lower after training, as compared to the rise before training.

In order to understand whether adaptation has occured due to training data on CK of the experimental group at resting state before and after training were analysed using paired 't' test and the results are presented in table II.

TABLE II

Table showing the result of the paired 't' test on Creatine Kinase

Experimental Group	Before Training	After Training	Mean Difference	't'
at Rest	220.2857	211.8571	8.4286	3.25

Significant at .05 level of confidence

Level of CK in increased in IU/Litre

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

Level of CK of the experimental group at resting state before training was 220. 2857 I.U and that after six weeks training was 211.857 with a mean difference of 8.4286 IU. The

obtained 't' value was 3.25 and it was significant at .05 level of confidence. This indicated that the given six weeks of training resulted in significantly lowering the amount of CK in the blood of the experimental group.

This means that after lesser amount of CK is produced due to acute exercise as compared to the state before training. This is an indication that adaptation has occured as a result of training .

Glutathione Peroxidase (GSHPx)

The mean values of Glutathione peroxidase of the control group and the experimental group before and after training are presented in Table IX.

TABLE IX

Acute-exercise induced changes and the effect of training on Glutathione Peroxidase (GSHPx)

	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	6.1286		5.8	6.0571		5.3857	B:0.4114	1
	0.4114							2.51
Mean Difference		0.3286		0.6714	W: 1.9686	12	0.1.64	
	t=**3.23***		**t=3.51**					
After Training	6.0		5.8	6.5	7.3857	B: 1.6457	1	1.6457
								15.82*
Mean Difference		0.2		0.88		W:1.2486	12	0.104
	t=4.1*		**t=5.36***					
Adjusted Mean						B:2.11	1	2.11
Difference	0.116			0.97				29.83*
					W: 0.78	11	0.07	

*Significant at 0.05 level of confidence
GPx is measured in moles of GSH consumed/min/mg. Hb
Table value of 'F' ratio requirements for significance
atdf (1,11) - 4.84
dt (1,12) - 4.75
Table value of 't' ratio required for significance at df 6 - 2.45

Before commencement of training, GPx activity of the control group at resting state was 6.1286 and that immediately after acute exercise was 5.8, with a mean difference of 0.3286 mols which was significant at 0.5 level of confidence.

*****

This meant that as a result of the acute exercise there was a significant reduction in the GPx activity of the control group by 0.3286 mols as indicated by 't' value 3.23. This reduction was significant at 0.05 level of confidence.

GPx activity of the experimental group before commencement of training at rest was 6.0571 and that immediately after acute exercise was 5.3857, with a mean difference of 0.6714 which was significant at .05 level of confidence. This meant that due to the acute

exercise, GPx activity of the experimental group had decreased by 0.9714 mols and this reduction was significant at .05 level of confidence ('t' = 3.51).

The exercise induced reduction the GPx activities of the control group and the experimental group have been analysed further using one way analysis of variance (ANOVA) to know whether there was any significant difference between the two groups before training. The resultant 'F' ratio, 2.51 was not significant at 0.05 level of confidence. This meant that there was no significant difference between the control group and the experimental group before commencement of training.

After training for six weeks, GPx activity for the control group at rest was 6.0 and that after acute exercise was 5.8 with a mean difference of 0.2 mols which was significant at .05 level of confidence. This meant that during the period of six weeks when the control group was kept sedentary, there was significant reduction in GPx activity after acute exercise as indicated by the t value 4.1.

GPx activity of the experimental group at rest, after six weeks training was 6.5 and that immediately after acute exercise was 7.3857 with a mean difference of 0.88 mols which was significant at 0.05 level of confidence (t = 5.36). This indicated that after six weeks of training acute exercise induced a significant elevation in GPx activity for the experimental group.

The exercise induced changes in the activity levels of GPx control group and the experimental group after training were further analysed using one way analysis of variance to know whether there was any significant difference between the two groups as a result of training. The resultant 'F' ratio was 15.82 and it was significant at 0.05 level of confidence. This mean that during the six weeks period when the control group was kept sedentary and the experimental group was undergoing training, there was significant difference in the activity of GPx of the experimental group as compared to the control group. The GPx activity of the experimental group was significantly higher than the control group (0.2). This indicate that the training resulted in increased GPx activity for the experimental group.

In order to understand the actual effect of training on GPx, the initial mean difference (before commencement of training) were nullified and the final values in the GPx activity after training adjusted using analysis of covariance (ANCOVA). The adjusted mean difference for the control group was 0.116 and the experimental group was 0.97. The resultant 'F' ratio was 29.83 and it was significant at .05 level of confidence.

This clearly indicated that after training the experimental group has significantly higher GPx activity in their blood as compared to the sedentary control group. Training resulted in significant elevation of GPx activity for the experimental group.

TABLE X

Table showing the results of the paired 't' test on GPx

Experimental Group	Before Training	After Training	Mean Difference	't'
at Rest	6.0571	6.5	0.4429	4.07*

*Significant at 0.05 level of confidence.

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

In order to understand the level of adaptation due to training, data on the GPx activities of the experimental group at resting condition, before and after training were analysed using paired 't' test and the results are presented in Table X.

GPx activity of experimental group at resting conditions, before training was 6.0571 mols and the GPx activity at rest after training for six weeks is 6.5 mols, with a mean difference of 0.4429 mols. The obtained 't' value 4.07 indicate that it is significant at .05 level of confidence. This meant that training resulted in significant elevation of resting state GPx activity. Hence it can be inferred that as a result of training adaptation has occured in the case of GPx.

Significantly higher activity of GPx after training may be due to the production of free radicals during workout, when free radicals are produced regularly from regular intense training, the adaptive mechanism of the body will increase the reserve of GPx level in blood-GPx being a primary anti oxidant which neutralises the toxic free radicals.

Conclusions

1. Creatine Kinase (CK) level significantly increased after acute exercise.
2. There was significant difference in the level of CK between the control group and the experimental group after training. After training production of CK was significantly lower for experimental group as compared to the control group.
3. Training significantly lowered the resting CK level of the experimental group.
4. GPx activity decreased after acute exercise.
5. There was significant difference between the control group and the experimental group after training. After training, GPx activity was significantly higher for experimental group as compared to the control group.
6. Training resulted in elevating the resting GPx activity of the experimental group.

BIBLIOGRAPHY

Ji L.L., Kang C., Zhang Y. Exercise-induced hormesis and skeletal muscle health. Free RadicBiol Med. 2016;98:113–122.

Reid M.B. Redox interventions to increase exercise performance. J Physiol. 2016;594:5125–5133.

Valko M., Rhodes C.J., Moncol J., Izakovic M., Mazur M. Free radicals, metals and antioxidants in oxidative stress-induced cancer. ChemBiol Interact. Pubmed. 2006;160:1–40.

Wei W., Liu Q., Tan Y., Liu L., Li X., Cai L. Oxidative stress, diabetes, and diabetic complications. Hemoglobin. Pubmed. 2009;33:370–377.

Powers S.K., Jackson M.J. Exercise-induced oxidative stress: cellular mechanisms and impact on muscle force production. Physiol Rev. Pubmed. 2008;88:1243–1276.

Förstermann U., Sessa W.C. Nitric oxide synthases: regulation and function. Eur Heart J. 2012;33 829–37a-d.

Culotta V.C., Yang M., O'Halloran T.V. Activation of superoxide dismutases: putting the metal to the pedal. BiochimBiophysActa. 2006;1763:747–758.

Alessio- HM., Exercise-induced Oxidative Stress., Medicine and Science in Sports and Exercise . 25 (2): 218-22, 1993.