ORIGINAL ARTICLE

Effect of Isotonic Exercises on Antioxidant and Free Radical Status in Healthy Adults

Sreehari P[1], Reddy LR[2], Khan MI[3]

1- Associate Professor, Dept of Physiology Rajiv Gandhi Institute of Medical Sciences (RIMS) Adilabad
2- Assistant Professor, Dept of Physiology Rajiv Gandhi Institute of Medical Sciences (RIMS) Adilabad
3- Lecturer, Dept of Physiology Rajiv Gandhi Institute of Medical Sciences (RIMS) Adilabad

Abstract

Background: *It has been postulated that exercises if done in moderate intensity for a prolonged period irrespective of age helps in promoting antioxidant state of the body.* ***Aim****: To evaluate the effects of Isotonic exercises on oxidative state and antioxidant status in healthy adult males.* ***Materials & Methods:*** *50 healthy male adults were divided into Group I and Group II (n=25). Group I was allowed to undergo training in Isotonic exercises under supervision for 6 weeks and Group II acted as controls. Blood samples were obtained prior and Post exercise training for comparison.* ***Results****: The mean value of Malondialdehyde (MDA) in Group I Prior to training was 268.96±28.97 nmol% and in Group II; it was 253.12± 21.74 nmol% while post training readings were (Group I) 428±63.95 nmol% and (Group II) 314.16±36.84 nmol%. The p value was < 0.03 which is significant. There were no significant differences in uric acid level in pre and post test in both the groups.* ***Conclusions:*** *It may be proposed that increase in oxidative stress with exercise is a necessary factor for muscle adaption to occur and long term exercises produces beneficial effects by causing better muscle adaptation. However isotonic exercises have only negligible effect in increase in antioxidant status.*

Key words: *Isotonic Exercise, Malondialdehyde [MDA], Uric Acid*

Address for correspondence: Dr Puli Sreehari, Associate Professor Department of Physiology Rajiv Gandhi Institute of Medical Sciences (RIMS) Adilabad 504001. Telangana State. Mobile: +919966604651. Email: drpulisreehari156@gmail.com

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Introduction

Regular moderate exercise is an important factor in staying healthy. As we exercise our muscles consume oxygen which in turn leads to increased production of free radicals. Free radicals are essential to many normal biological processes. Free radicals can be defined as reactive chemical species having a single unpaired electron in outer orbit. [1] Free radicals due to their unstable configuration causes release of energy and reacts with proteins, lipids, carbohydrates and nucleic acids. They are generally referred as "reactive oxygen species" (ROS) they are produced both endogenously and exogenously. The endogenous sources of ROS include mitochondria, cytochrome P450 metabolism, peroxisomes, and inflammatory cell activation. [2] Additional endogenous sources of cellular ROS are Neutrophils, Eosinophils and Macrophages. On activation, Macrophages initiate an increase in oxygen uptake giving rise to a variety of ROS, including superoxide anion, Nitric Oxide and hydrogen peroxide. [3] It has been established that ROS can be both harmful and beneficial in biological systems depending on the environment. [4] Beneficial effects of ROS involve, for example, the physiological roles in cellular responses to noxia such as defense against infectious agents, and in the function of a number of cellular signaling systems. In contrast, at high concentrations, ROS can be mediate damage to cell structures, including lipids and membranes, proteins and nucleic acids; this damage is often referred as "oxidative stress" [5] The harmful effects of ROS are balanced by the action of antioxidants, some of which are enzymes

present in the body. [6] Despite the presence of the cell's antioxidant defense system to counteract oxidative damage from ROS, oxidative damage accumulates during the life cycle and has been implicated in aging and age dependent diseases such as cardiovascular disease, cancer, neurodegenerative disorders and other chronic conditions. [7]

However it is important to recognize that free radicals are not the cause any disorder on their own, but the diseased or damaged tissue undergo radical reactions much more readily then the normal tissues exacerbating the primary lesions. [8] Antioxidants are substances that when present in low concentrations compared with those of an oxidizable substrate significantly delays or prevents oxidation of the substrate. [8] Research shows that endurance and strength training places an increased demand on blood antioxidant systems, which are body's first line of defence against free radical damage. [9] A free radical generation in cell damages it to the point that it must be removed by immune system. If free radical formation and attack are not controlled as in exercise it could lead to muscle damage. This posses a unique question, when exercise promotes health, but research suggests it may also increase cell damage by free radicals. To answer such a question we undertook the present study in which we tried to the find some of the answers.

Materials and Methods

The present study was conducted at Department of Physiology and Biochemistry, Osmania Medical College Hyderabad, and Hanuman Vyayamasala Gym centre Koti, Hyderabad. The Biochemical analysis for estimation of Serum Malandialdehyde (MDA, a marker of lipid per oxidation) and Serum Uric acid (Natural antioxidant of the body) levels by calorimetry by using spectrophotometer.

A group of 50 male age matched volunteers having good health, non-smoking, and non-alcoholic were selected and randomly assigned into two groups, Group-I (Isotonic exercise group), Group-II (Control group) consisting 25 subjects each. Both the groups are screened for general health and vital data collected. They are also screened for their food habits. Only vegetarians are selected as non-vegetarian food can interfere in the results of the experiment. The subjects selected were not previously trained for Isotonic exercises. Procedures followed in this study were in accordance with the ethical standards laid down by ICMR's Ethical guidelines for biomedical research on human subjects (2006). Informed consent was taken from all the patients participated.

Group-I (Isotonic exercise): Group-1 subjects were given programmed training in Isotonic exercise (i.e., Bench press, chest press with Dumbbells and Barbells). They exercise daily for 30 minutes under supervision for 6 weeks in the morning 6:00Am to 6:30Am.

Group-II Control group: The control group did not receive any training and they were advised not to deviate from their routine work. At the start of the training schedule blood samples were collected from all subjects of both groups for the estimation of MDA and Uric acid levels. Similarly the samples of blood were collected for the estimation of MDA and Uric acid levels at the end of 6 weeks in both the groups.

Biochemical Tests

Aldehydes, especially Malondialdehyde [MDA] a product of fatty acid peroxidation MDA, is an indicator of the extent of peroxidation and has been frequently used as marker of oxidative stress in response to exercise. The most common method used to assess changes in MDA with exercise is Thiobarbituric Acid (TBARS) Assay. This method works well when used on defined membrane systems such as microsomes in vitro. [10]

Estimation of Uric Acid

Uric acid is a final enzymatic product in the degradation of purine nucleosides and free bases in humans. Urates appear to play role beyond the end product of purine metabolism. Urate by itself serves as an antioxidant undergoing non-enzymatic conversion to Allantoin. It is now considered as a naturally occurring antioxidant. Estimation of uric acid gives the level of antioxidants in the body [11]. Normal range: Male: 2.5 to 7.0 mg%.

Results

There was no significant difference in MDA level, between test and control group before exercises. While after training of 6 weeks significant difference was observed between both the groups in their MDA level (Table- 1).

The value of serum uric acid is an indication of natural antioxidant levels in the body. Before the training phases, the mean values of serum uric acid were 4.64 in the test group and 4.43 in the control group the calculated p values were greater then 0.1 which indicate it is not significant. But after exercise training, in both the groups the mean values of test group was 5.42 and control group was 4.56 which indicates a slight increase in the Uric acid levels in test group but the p values were non-significant (Table-2).

Table- 1: MDA level before & after exercise

Groups	MDA nmol% Mean±SD	p value
Test Group [Before Exercise]	268.96±28.97	> 0.1
Control Group [Before Exercise]	253.12±21.74	
Test Group [After Exercise]	428.0±63.95	< 0.03*
Control Group [After Exercise]	314.16±36.84	

* p<0.05

Table- 1: Uric acid before & after exercise

Groups	MDA nmol% Mean±SD	p value
Test Group [Before Exercise]	4.64±0.96	> 0.1
Control Group [Before Exercise]	4.43±0.84	
Test Group [After Exercise]	5.42±1.38	>0.01
Control Group [After Exercise]	4.56±1.06	

Discussion

The main findings of the present study show that there is an increase in MDA a marker for free radical in test group post exercise to significant levels. However the uric acid which is a marker for antioxidant activity did not increased significantly. There are several studies which indicate that an increase in free radical production especially in active skeletal muscles. [12-14] Sureda et al [15] found an increased Malondialdehyde due to oxidative stress in lymphocytes after a single bout of intense exercise. Similarly Ajmani et al [16] noted increased membrane rigidity in erythrocytes after strenuous exercise because of oxidative stress. It has been proposed that exercise can produce imbalance between the oxidants and antioxicants which is commonly referred as oxidative stress. Physical activity increases generation of free radicals and 2-5% of oxygen used in mitochondria forms free radicals. As oxidative phosphorylation increases in response to exercise, there will be concomitant increase in free radicals. Exercise is known to cause muscle inflammation and also contribute to increased levels of lipid peroxidation due to macrophage reactions in the tissues. [17]

In the present study as Malondialdehyde [MDA] was found to increase after one month of regular exercise training such findings has also been reported several studies. [18,19] Similarly Knater et al [20] reported increase in plasma MDA following extreme endurance. However there are other studies that have found no change in MDA in response to exercise. [21, 22] Our study disagrees with such findings. This study also shows that there was no significant increase in uric acid concentration in both groups post exercise such findings has been reported by Alessop et al [23] who showed that total antioxidant capacity did not increase in response to 30 minute of exercise despite an increase in MDA levels. In a study by Green H et al [24] they concluded that exercise intensity is rather than total work output is a critical factor in mediating increase in uric acid concentration. Since our exercises were also low intensity Isotonic exercises this could be the reason why we found No significant increase in uric acid concentration marker of antioxidant status in our study.

Conclusion

Within the limitations of the study it is concluded that exercises may result in increased oxidative stresses. It may be proposed that increase in oxidative stress with exercise is a necessary factor for muscle adaption to occur and long term exercises produces beneficial effects by causing better muscle adaptation. However isotonic exercises have only negligible effect in increase in antioxidant status.

Conflict of Interest: *None declared*
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