

EFFECT OF EXERCISE ON LIPID PEROXIDATION

Süleyman Demir¹, Günfer Turgut², Özlem Yurtseven¹, Diler Aslan¹, Osman Genç²Pamukkale University, Medical Faculty, Denizli, Turkey: Department of Biochemistry¹, Department of Physiology²

Summary: Purpose: The effect of physical exercise on lipid peroxidation was investigated. Method: 27 healthy young adult male subjects were included in this study. Urine samples were collected before and after exercises. Urinary malondialdehyde and creatinine levels (Cr) were measured. Results: Urinary malondialdehyde levels were increased by exercise. While pre-exercise malondialdehyde levels were $5,02 \pm 1,26$ nmol/mg Cr, post-exercise levels were $6,13 \pm 1,84$ nmol/mg Cr ($p < 0,05$). Conclusion: These findings indicated that physical exercise induced lipid peroxidation.

Key words: Lipid peroxidation; Exercise; Oxidative stress

Introduction

Oxygen derived free radical damage is widely considered as etiological factors in many disorders (7). Free oxygen radicals rapidly react with polyunsaturated fatty acids in the cell membranes, proteins, and other cellular components. Since it is impossible to measure free radicals directly in vivo, it is necessary to rely on the quantitation of their reaction products such as protein carbonyls, modified DNA and lipid peroxidation products. Malondialdehyde (MDA) is the most widely used index of lipid peroxidation (8).

Physical exercise can induce oxidative stress and free radical production by different mechanism. These mechanisms related to the time course and exercise intensity (2,10,11,12).

The measurement of excretion of lipid peroxidation products in vivo most probably indicates the global oxidative status of the whole body and the samples can easily obtained from human volunteers without the need of access to the internal medium (5,13,16).

The purpose of this study was to investigate the effect of exercise on lipid peroxidation in healthy young men.

Material and method

Twenty-seven healthy young adult male subjects were included in this study (aged $21,04 \pm 2,44$, ranged 17-26 years). The subjects trained to prepare playing football, including warming exercises, sprints etc. for two hours. After the exercises, participants were relaxed for one hour. Urine samples were collected before and after exercises. Urinary malondialdehyde and creatinine levels were immediately measured.

Malondialdehyde levels were determined the method in which MDA reacts with thiobarbituric acid (14). Urinary creatinine levels were measured by automated system

(ILAB 900). The results of MDA were expressed as nmol/mg creatinine.

Results

We observed that urinary malondialdehyde levels were increased by exercise. The difference between with pre and post exercise urinary creatinine levels was not statistically significant. Urinary MDA concentration is shown in Fig. 1. While pre-exercise malondialdehyde levels were $5,02 \pm 1,26$ nmol/mg Cr, post-exercise levels were $6,13 \pm 1,84$ nmol/mg Cr ($p < 0,05$). The difference was statistically significant ($p < 0,05$).

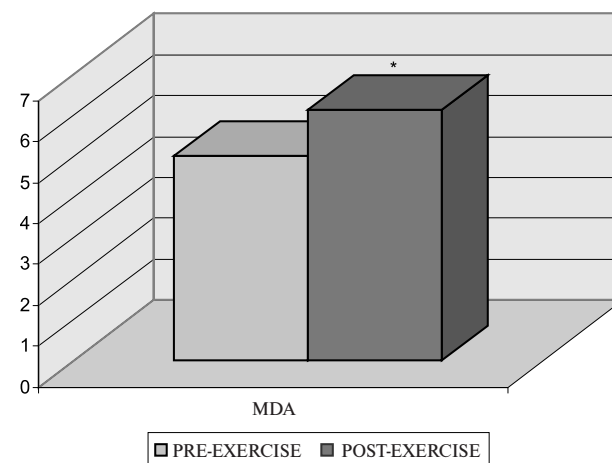


Fig. 1: Pre- and post exercise urinary MDA levels

Discussion

Physical exercise can induce oxidative stress and free radical production. However, studies in humans on the in-

fluence of exercise on the levels of lipid peroxidation markers are limited and the findings are contradictory (10,11, 12). Initial suggestions that free radical processes, such as lipid peroxidation, were elevated during exercise came from studies of whole body exercise in man and rats (1,3). It is now recognised that there are a number of potential intracellular sites for the production of free radicals within muscle such as the mitochondrial electron transport systems, membrane bound oxidases and infiltrating phagocytic cells (4,6). In addition, xanthine oxidase within endothelial tissue closely associated with muscle is a potential site for the free radical production. Muscle is unique in its ability to undertake very rapid and co-ordinate changes in energy supply for repeated contractions. These changes requiring major variations in oxygen flux through the tissue and the electron flux through the mitochondrial respiratory chain might predispose to the formation of oxygen-centred free radical species (3,13).

In addition, exercise increases the number of circulating neutrophils and may produce some features of an acute-phase response. It is certain that can induce muscle damage. Damaged tissues are more rapidly oxidised than normal (3,9,15).

Oxidative stress can be measured with various markers in blood and several tissues that typically reflect tissue peroxidation (8). The measurement of urinary excretion of products of lipid peroxidation in vivo most probably indicates the global oxidative status of the whole body. The samples can be easily obtained from human volunteers (5,13,16).

In this study, we observed that urinary MDA levels were increased by exercise. These findings indicate that physical exercise may induce lipid peroxidation.

References

1. Brady PS, Brady LJ, Uhey DE. Selenium, vitamin E and the response to swimming stress in the rat. *J Nutr* 1979;109:1103-9.
2. Cadenas S, Rojas C, Mendes J, Herrero A, Brja G. Vitamin E decreases urine lipid peroxidation products in healthy human volunteers under normal conditions. *Pharm Toxic* 1996;79:247-53.
3. Dabrit CT, Joy RB, Sawof WM, Tappel AE. Effects of exercise, vitamin E and ozone and pulmonary function and lipid peroxidation. *J Appl Physiol* 1978;45:927-32.
4. Davies KJA, Quintanilla AI, Brooks GA, Packer E. Free radicals and tissue damage produced by exercise. *Biochem Biophys Res Commun* 1982;107:1198-1205.
5. Drury JA, Nycyk JA, Cooke RW. Comparison of urinary and plasma malondialdehyde in preterm infants. *Clin Chim Acta* 1997;263:177-85.
6. Halliwell B, Gutteridge JMC. Exercise-induced oxidant damage. In: Halliwell B, Gutteridge JMC. eds. *Free Radical in Biology and Medicine*. Oxford: Clarendon Press, 1995:449-50.
7. Halliwell B. Antioxidants in human health and disease. *Annu Rev Nutr* 1996;16:33-50.
8. Holley AE, Cheeseman KH. Measuring free radical reactions in vivo. Cheeseman KH, Slater TF. eds., *Free Radicals in Medicine*. The British Council, 1993:494-505.
9. Jackson MJ, O'Farrell S. Free radicals and muscle damage. In: Cheeseman KH, Slater TF. Eds. *Free Radicals in Medicine*. The British Council, 1993:630-41.
10. Leaf DA, Kleinman MT, Hamilton M, Barstow TJ. The effect of exercise intensity on lipid peroxidation. *Med Sci Sports Exerc* 1997;29:1036-9.
11. Marzatico F, Pansarasa O, Bertorelli L, Somenzini L, Della Valle G. Blood free radical antioxidant enzymes and lipid peroxides following long-distance and lactacidemic performances in highly trained aerobic and sprint athletes. *J Sports Med Phys Fitness* 1997;37:235-9.
12. McBride JM, Kraemer WJ, Triplett McBride T, Sebastianelli W. Effect of resistance exercise on free radical production. *Med Sci Sports Exerc* 1998;30:67-72.
13. Nacitarhan S, Özben T, Tuncer N. Serum and urine malondialdehyde levels in NIDDM patients with and without hyperlipidemia. *Free Radic Biol Med* 1995;19:893-6.
14. Ohkawa H, Ohishi N, Yagi K. Assay for lipid peroxides in animal tissues by thiobarbituric acid reaction. *Anal. Biochem* 1979;95:351-8.
15. Ortenblad N, Madsen K, Djurhuus MS. Antioxidant status and lipid peroxidation after short-term maximal exercise in trained and untrained humans. *Am J Physiol* 1997;272:1258-63.
16. Ozben T, Nacitarhan S, Tuncer N. Plasma and urine malondialdehyde levels in non-insulin-dependent diabetic patients with and without microalbuminuria. *Int J Clin Lab Res* 1995;25:162-4.

Submitted September 2000.

Accepted January 2001.

Dr. Süleyman Demir,
Yeni Mah. Tokat C. No: 41,
Özkan Apt. D:5,
Denizli-TURKEY.
e-mail: suleyman@pamukkale.edu.tr