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 mechanisms. 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±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 (Cr) were measured. Results: Urinary malondialdehyde levels were increased by exercise. While pre-exercise malondialdehyde levels were 5.02±1.26 nmol/mg Cr, post-exercise levels were 6.13±1.84 nmol/mg Cr (p<0.05). Conclusion: These findings indicated that physical exercise induced lipid peroxidation.

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±1.26 nmol/mg Cr, post-exercise levels were 6.13±1.84 nmol/mg Cr (p<0.05). The difference was statistically significant (p<0.05).

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 phagositic 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.

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