SHORT REPORT

Influence of exercise on oxidant stress products in elite Indian cyclists

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Br J Sports Med 2007;**41**:691–693. doi: 10.1136/bjsm.2007.037663

The influence of exercise on free-radical chemistry is not well understood. It is yet to be confirmed whether an adequate biochemical defence system exists in the human body to provide protection from oxy-centred radicals generated by exercise. Fifty trained elite cyclists undertaking exhaustive endurance training were compared with a control group of 50 sedentary workers. Serum malondialdehyde (MDA), uric acid, superoxide dismutase (SOD), catalase, vitamin E, vitamin C and susceptibility to oxidative stress were assessed. Exhaustive exercise resulted in significantly ($p < 0.05$) higher concentrations of serum MDA, vitamin E and vitamin C, significantly ($p < 0.001$) higher SOD activity, but less significantly ($p < 0.01$) higher concentrations of uric acid and significantly ($p < 0.05$) lower catalase activity in elite cyclists than in the controls. Alterations in the activities of erythrocyte scavenger enzymes (SOD) and higher level of non-enzymatic defences in trained subjects may not be sufficient to counteract the increase in reactive oxygen species produced by endurance training.

As oxygen consumption increases during exercise, 95–98% of the oxygen consumed is reduced to water during aerobic metabolism, but the remaining fraction may be converted into oxidative byproducts, reactive oxygen species (ROS). During exercise, when oxygen uptake is increased to 10–15-fold above that at rest, it is very likely that free radicals are produced to a greater extent than at rest. ROS may damage body tissues if their production is not controlled precisely and adequately. Irreversible oxidative damage to certain vulnerable molecules is thought to contribute to the degenerative process associated with cell breakdown and aging. It is now widely accepted that free-radical generation is enhanced during strenuous exercise.¹ To combat the deleterious effects of free radicals and ROS, the human body has some complex internal protective mechanisms such as enzymatic defences, which include primary antioxidative enzymes, such as superoxide dismutase (SOD), catalase and glutathione peroxidase, and non-enzymatic defences, such as vitamin C, vitamin E, ubiquinol coenzyme Q-10 and reduced glutathione. During exercise, the pro-oxidant/antioxidant balance shifts in favour of the former, with the rate of radical and ROS production exceeding their rate of removal by the antioxidant defence mechanisms.

METHODS

Fifty elite cyclists (32 male, 18 female) and 50 sedentary volunteers (25 male, 25 female) participated in this investigation. They were homogeneous populations with regard to age and lifestyle. The cyclists had several years' experience of long distance cycling and trained a mean weekly cycling distance of 90 km. In the selection of the cyclists, a treadmill test (spiroergometry) was performed to determine performance

capacity and maximum oxygen uptake ($\text{VO}_{2\text{MAX}}$). Performance on the treadmill started with a speed of 8 km/h and increased every 3 min by 2 km/h to exhaustion. The nature and purpose of the study and the risks involved were explained verbally and in writing to the subjects, before written consent to participate was obtained. Body weight was measured in kilograms with calibrated beam scales. Height was measured in centimetres with a scale-mounted anthropometer. Table 1 presents descriptive data for age and anthropometric measurements of the cyclists (test group) and sedentary subjects (control group).

Before testing and at the same time of day (06:30–07:30), blood samples were drawn from the medial cubital vein, after the subjects had been sitting for 15 min. Serum was separated from whole blood. Each sample was assayed for the lipid peroxidation marker, malondialdehyde (MDA), SOD, catalase, uric acid, vitamin E and vitamin C. The Mann–Whitney U test was used to establish the statistical significance of mean differences between different variables, where $p < 0.05$ was taken as significant.

RESULTS

Table 2 gives the descriptive data showing lipid peroxidation, catalase activity, SOD activity and serum concentrations of vitamin E, vitamin C and uric acid in the sedentary control subjects and trained cyclists. A significant ($p < 0.05$) 1.4-fold increase in MDA was observed in the cyclists. A highly significant ($p < 0.001$) increase in SOD activity, a significant ($p < 0.05$) 0.75-fold reduction in catalase activity, a 1.3-fold increase in uric acid ($p < 0.01$), and a significant ($p < 0.05$) increase in vitamin E and vitamin C were observed in cyclists compared with the control group.

DISCUSSION

Our results for trained subjects (elite cyclists) are in agreement with the finding of Kanter *et al*² of an increase in MDA in the blood for 30 min after a 80 km race. High concentrations of MDA have been reported by other workers in a treadmill test at 60% and 90% of $\text{VO}_{2\text{MAX}}$.³ In a study by Jenkins *et al*,⁴ MDA concentrations did not differ between trained and untrained

Table 1 Descriptive data for age and anthropometric measurements of elite cyclists and control group

Variable	Cyclists (n = 50)	Controls (n = 50)
Age (years)	21.2 (0.43)	20.2 (0.59)
Height (cm)	168.36 (1.02)	158.71 (1.17)
Weight (kg)	63.14 (1.51)	59.28 (0.99)

Values are mean (SEM).

Abbreviations: MDA, malondialdehyde; ROS, reactive oxygen species; SOD, superoxide dismutase; $\text{VO}_{2\text{MAX}}$, maximum oxygen uptake

Table 2 Descriptive data showing lipid peroxidation, catalase activity, SOD activity and concentrations of vitamin E, vitamin C and uric acid in control sedentary subjects and trained cyclists

Variable	Controls (n = 50)	Cyclists (n = 50)
MDA formed ($\mu\text{mol}/\text{mg protein}/15 \text{ min}$)	1.59 (0.02)	2.18 (0.09)*
Catalase activity ($\mu\text{mol H}_2\text{O}_2$ consumed/ $\text{mg protein}/\text{min}$)	76.0 (5.2)	57.0 (6.10)*
SOD activity (U/mg protein)	13.4 (0.23)	16.6 (0.87)†
Vitamin E concentration ($\mu\text{mol}/\text{l}$)	23.8 (2.1)	25.5 (1.55)*
Vitamin C concentration (mmol/l)	49.5 (1.11)	51.44 (2.10)*
Uric acid (mg/dl)	5.12 (0.45)	6.80 (0.23)‡

Values are mean (SEM).

* $p < 0.05$, † $p < 0.001$, ‡ $p < 0.01$ compared with controls.

rats before exercise, but after a run to exhaustion (at 70% of VO_2MAX for 2 h), MDA concentrations had increased by 60% in the trained rats. Our findings on SOD activity agree with earlier studies,⁵⁻⁷ which also found higher SOD activity in exercised subjects than sedentary controls. The higher SOD activity in cyclists carrying out regular physical training suggests that regular training strengthens the antioxidant defence system by increasing SOD activity, thus reducing the oxidative stress of exercise. The reduction in catalase activity observed in our study is contradictory to earlier reports. A significant increase in catalase activity was observed after exhaustive exercise, but the activity remained unchanged during chronic training,⁶ whereas no change in catalase activity caused by either exhaustive exercise or training was reported.⁸ The significantly lower catalase activity in the trained group in our study indicates decreased formation of hydrogen peroxide—that is, an adaptive change during regular physical training. The decrease in activity of this enzyme may also be due to inactivation by excessive production of free radicals.

Degoutte *et al*⁹ reported that uric acid concentration was higher in the blood samples of judokas collected 1 h after a judo match and had not returned to baseline 24 h after the match. The high concentrations of uric acid noted 24 h after the match suggests that the process of recovery is not complete. This has also been observed previously.¹⁰ However, Robertson *et al*¹¹ reported that there was a significantly higher uric acid concentration in untrained subjects than in highly trained runners.

What is already known on this topic

- It is now well accepted that oxygen consumption increases up to 10–20-fold during exercise, which leads to free-radical generation.
- These exercise-induced free radicals cause oxidative stress.
- There are many worldwide studies related to increase in oxidative stress markers during endurance training especially in long-distance runners.
- However, there are some contradictory studies that indicate that oxidative stress markers return to baseline values with regular training.

What this study adds

- This study adds to current knowledge on the effect of exercise on oxidative stress markers in elite cyclists, and should be particularly useful for elite Indian cyclists.
- Alterations in the activities of erythrocyte scavenger enzymes (eg, superoxide dismutase) and a higher level of non-enzymatic defences in trained subjects may not be sufficient to counteract the increase in reactive oxygen species produced during endurance training.
- Antioxidant supplementation is thus required to combat the deleterious effects of exercise-induced oxidative stress.

In our study, the high uric acid concentration in the cyclists compared with the untrained group may partly be attributed to the relatively high-protein dietary intake of the cyclists. It has been hypothesised that uric acid has a role in the antioxidant defence of the muscle during exercise.¹² Thus the rise in uric acid concentration may represent loss of useful nucleotide precursors and may be important in protecting tissues against ROS. Our findings agree with those of Pincemail *et al*¹³ and Camus *et al*,¹⁴ who reported high plasma tocopherol concentration in trained players carrying out dynamic exercise compared with sedentary workers of the same age.

The increased lipid peroxidation observed in the trained cyclists suggests that regular physical training may produce “oxidative stress”, which may lead to generation of free radicals and lipid peroxidation, which, in turn, could lead to erythrocyte membrane damage. It is further suggested that alterations in the activities of erythrocyte scavenger enzymes (SOD and catalase), in spite of higher vitamin C and vitamin E concentrations due to exercise-induced oxidative stress in the trained subjects, may not be sufficient to counteract the increase in ROS, and this may result in a concomitant increase in free-radical production and lipid peroxidation.

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Funding: We are grateful to Sports Authority of India for their financial support.

Competing interests: None.

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Accepted 5 May 2007

Published Online First 11 May 2007

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