

Full Length Research Paper

The effect of graded maximal aerobic exercise on some metabolic hormones, muscle damage and some metabolic end products in sportsmen

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The aim of this study was to investigate the effect of graded maximal aerobic exercise on some metabolic hormones, muscle damage and some metabolic end products in professional sportsmen. Twelve young professional football players participated in the study. Subjects performed shuttle run test. Blood samples were collected before, immediately after and one hour after the single exercise. The levels of testosterone (T) and cortisol (C) as metabolic hormones; creatine kinase (CK), lactate dehydrogenase (LDH) and aspartate amino transferase (AST) as muscle damage markers; urea and uric acid levels, as metabolic end products, were determined. Testosterone levels significantly increased immediately after exercise ($p = 0.027$), but increase in the cortisol levels was not significant. However, cortisol levels demonstrated a significant difference between immediately after and one hour after exercising ($p = 0.006$). Both testosterone and cortisol levels decreased to their initial levels one hour after exercising. Although changes in the urea levels were not significant ($p = 0.667$), increase in the uric acid level one hour after the exercising was significant ($p = 0.005$). While the increase in CK activity was significant ($p = 0.023$), changes in AST ($p = 0.170$) and LDH levels were not significant ($p = 0.139$). Besides constituting an anabolic effect, graded maximal aerobic load also caused an increase in cortisol and uric acid in professional football players. Due to being accustomed to these types of exercises, subjects had inconsiderable muscle damage after loading. The exercise caused an increase in serum uric acid due to renal clearance decrease and increasing endogenous purines with contribute protein degradation.

Key words: Exercise, testosterone, creatine kinase, urea, uric acid.

INTRODUCTION

Many researchers have reported that exercise changes the concentrations of anabolic and catabolic hormones in sedentary individuals. The main reason of this is known as the stress caused by exercise. Testosterone is secreted in the testis of men; due to the stimulation of luteinizing hormone (LH) secreted from the anterior

pituitary gland. Testosterone has anabolic and anti-catabolic effects upon the appearance of masculinity characteristics (Guyton and Hall, 2003; Hedge et al., 1987; Brownlee et al., 2005). Especially it has an effect to increase the protein metabolism, the basal metabolic rate, growth of target organs in the bone and muscle tissues. However, all the other effects depend on increasing protein production rate (Guyton and Hall, 2003). Many researchers have indicated that total and free testosterone levels increase during short term exercise, and this increase is related to the intensity of the

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performed work load (McMurray and Hackney, 2000; Viru, 1992). This increase is caused by various factors (Hackney, 1996). The more the intensity of the exercise, the more testosterone concentration increases; however when the exercise intensity decreases and the duration increases, testosterone concentration can decline below pre-exercise level (McMurray and Hackney, 2000).

Cortisol which stimulates the catabolism of proteins and lipids in the extra-hepatic tissues and increases the concentration of fatty acids and amino acids in the plasma is one of the most effective glucocorticoids secreted from the adrenal glands. It also stimulates the increase in gluconeogenesis and the plasma proteins by stimulating the protein synthesis in the liver (Guyton and Hall, 2003; Galbo, 2001; Wolfe, 2001). Additionally, it has effects on blocking and reducing the inflammation and restoration in the damaged tissues. Physical stress gives rise to enhanced secretion of Adrenocorticotrophic Hormone (ACTH) and consequently cortisol within minutes (Guyton and Hall, 2003). ACTH values increase according to the intensity of the exercise (Hill et al., 2008; Minetto et al., 2007). Urea and uric acid are the main end products of protein catabolism (Guyton and Hall, 2003). The protein metabolism, depending on the type of the exercise, has an important role in increasing the protein synthesis and has a minor role as an energy source (Lemon, 2000). Furthermore, amino acids have a regulation role over the exercise metabolism (Haralambie and Berg, 1976). The increase in the oxidation of amino acids depends on the intensity of the aerobic exercise (Babij et al., 1983), the decrease in the renal clearance and the incident of muscle damage (Banister et al., 1985) especially in endurance exercises (Fry et al., 1991) increase urea and uric acid values.

Exercise causes muscle fibers damage with myofibrillar split and Z-line flux. Exercise induced muscle damage (EIMD) is explained with two mechanisms. The first one is that the muscle architecture disrupts due to the unaccustomed exercise. The second one is, although not as clearly characterized, that local muscle ischemia may contribute to tissue injury (Staron and Hikita, 2000). The type of the exercise affects the magnitude of EIMD. Eccentric contraction causes more muscle damage than the other types of exercises (Davies et al., 2008). The enzymes special to muscle cells are used for determination of muscle damage. These enzymes are: creatine kinase (CK), aspartate amino transferase (AST) and lactate dehydrogenase (LDH) (Skenderi et al., 2006; Jiang et al., 1998). The most commonly assayed component is CK. The gradient of enzyme activity and activation time scale is correlated with the magnitude of EIMD (Staron and Hikita, 2000). Many studies have reported that exercise causes changes in the concentrations of anabolic (McMurray and Hackney, 2000; Viru, 1992; Hackney, 1996) and catabolic (Guyton and Hall, 2003; Hill et al., 2008; Minetto et al., 2007) hormones, causes muscle damage (Staron and Hikita, 2000; Davies et al., 2008) and affects the metabolic end products (Babij et al.,

1983; Fry et al., 1991; Kaya et al., 2006; Friden et al., 1983), as well. However, there are very limited studies that have searched these effects during the training process.

So, it is important to search the effects of maximal aerobic exercise in which load increases step by step upto the maxVO₂ capacity on metabolic hormones, muscle damage and metabolic end products in professional sportsmen. With this objective, this study aimed to investigate the effect of maximal aerobic exercise on some metabolic hormones, muscle damage and some metabolic end products, in professional sportsmen.

MATERIALS AND METHODS

Subjects

12 young professional football players participated in the study. All subjects gave their written informed consents for the study and the use of their medical data for research purposes.

Exercise protocol

The subjects carried out a progressive shuttle run test (Rambsbottm et al., 1988). Briefly, subjects ran back and forth between two lines, spaced 20-m apart, in which each lap started with a "beep" sound. Each successful run of the 20-m distance accepted as the end of a shuttle. The "beep" sound interval progressively decreased the pace with every minute of the test. The subjects increased their running speed accordingly. The subjects who did not reach the end line in time were cautioned. The test was stopped when the subject failed to follow the set pace of the "beeps" for two successive shuttles, or stopped voluntarily. Exercise test was performed in gymnasium of 1100 m high above sea level.

Data collection

Blood samples were taken from each subject before performing the exercise, immediately after and one hour after the exercise. Blood samples were drawn from the antecubital vein. Serums were frozen at -80°C until analysis. CK, AST, LDH, urea, and uric acid values were given in the original kit by using auto analyzer (Roche Hitachi Modular DP Systems (Mannheim, Germany). Testosterone and cortisol levels were measured by immun chemiluminesance (ICL) method. For this purpose, Immulite 2000 auto analyzer (Diagnostic Products Corporation, Los Angeles, USA) was used. Specificity, sensitivity and coefficients of variation (CV) values were respectively calculated as: CK; 76.6, 97.1 and 0.7%, AST; 87.5, 98.9 and 3.4%, LDH; 78.5, 94.8 and 3.5%, Urea; 67.7, 99.3 and 1.3%, uric acid; 64.7, 95.7 and 2.8%, testosterone; 91.7, 98.5, and 8.7%, Cortisol; 89.7, 96.8 and 6.9%.

Data evaluation

All obtained data were transferred to statistical program, for analysis. All data were normally distributed (Kolmogorov-Smirnov test). For the purpose of comparison between the measurements, repeated measures ANOVA was conducted. Multiple comparison tests were conducted with the Bonferroni test. Level of significance was set at 0.05.

Table 1. Physical characteristics of participants.

Variables	Mean \pm SD
Age(years)	23.50 \pm 2.50
Height (cm)	178.10 \pm 4.60
Weight (kg)	74,30 \pm 4.98
Sports Age(years)	12.00 \pm 4.94

Table 2. Some Hormones, metabolic end products and skeletal muscle enzymes.

Variables	Before exercise	Immediately after exercise	An hour after exercise	F value	Sig.
	Mean \pm SD	Mean \pm SD	Mean \pm SD		
Testosterone (ng/dL)	669.17 \pm 82.281(a)	732.916 \pm 96.141(a)	683.416 \pm 78.422	5.331	0.027*
Cortisol (ug/dL)	18.325 \pm 3.286	20.275 \pm 2.426(b)	17.042 \pm 3.419(b)	9.015	0.006*
Urea (mg/dL)	39.00 \pm 7.459	39.50 \pm 8.174	38.00 \pm 7.410	0.423	0.667
Uric acid (mg/dL)	5.850 \pm 0.776 (c and d)	6.758 \pm 0.811 (c)	6.533 \pm 0.909 (d)	9.196	0.005*
CK (U/L)	149.00 \pm 27.571 (e)	202.416 \pm 72.503 (e)	173.750 \pm 52.421	5.648	0.023*
AST (U/L)	22.42 \pm 2.778	24.00 \pm 3.437	22.166 \pm 3.157	2.127	0.170
LDH (U/L)	283.831 \pm 75.693	306.500 \pm 66.519	246.916 \pm 42.902	2.418	0.139

* $p < 0.05$. a): Testosterone levels showed statistically significant before exercise and immediately after exercise. b): Cortisol levels immediately after exercise and an hour after exercise showed statistically significant. c): Uric acid levels were statistically significant before exercise and immediately after the exercise. d): Uric acid levels were statistically significant before exercise and an hour after exercise. e): There was statistically significant between CK levels before exercise and CK levels immediately after exercise.

RESULTS

In the study testosterone levels were significantly increased immediately after the exercise and were determined to tend to decrease one hour after the exercise. However the increase in the cortisol levels immediately after the exercise was not significant compared to before exercise level. In addition, there were significant differences between the cortisol values of immediately after the exercise and one hour after the exercise. Changes in the urea values were insignificant but values of uric acid both immediately after the exercise and one hour after the exercise increased significantly compared to before exercise values. The CK values, as a marker of muscle damage, were determined to increase significantly immediately after the exercise compared to before exercise level, but the increase one hour after the exercise was observed insignificant. Changes in AST and LDH values were not significant (Tables 1 and 2).

DISCUSSION

The primary finding of this investigation was a significant increase in the testosterone levels immediately after exercise. But the increases in cortisol levels were not significant. It was found that testosterone level decreased one hour after the exercise but it was even higher than the base level. However cortisol was found to decrease to base level one hour after the exercise (Figure 1). In

most of the studies, it has been reported that exercise increases testosterone and cortisol levels in short term and long term; their effects are related to work load and intensity (Wilkinson et al., 2006; Kokalas et al., 2004; Reeves et al., 2006). This increase is caused by combination of decreased metabolic clearance rate, increased secretion of end products and exercise induced hemoconcentration (Hackney, 1996). However testosterone response varies in prolonged exercises. Testosterone level increases with the increasing intensity of the exercise, but testosterone level decreases as the intensity decreases and exercise duration is extended (Safarinejad et al., 2008). It is suggested that this is caused by increased metabolic clearance rate and decreased testicular secretion in the long durational exercises (McMurray and Hackney, 2000). In this study, however non-significant cortisol level increased immediately after the exercise.

The secretion rate of cortisol is controlled by ACTH. Almost all kinds of stress increase the secretion of this hormone by the pituitary gland. Therefore a few minutes after the beginning of the exercise, cortisol increases in accord with the intensity of the exercise. However, the response of ACTH to exercise is still not completely clear. Short term maximal and submaximal exercises don't affect the blood glucose levels. Also, significant stress doesn't occur unless intensity of exercise is greater than 50%. Some studies suggest that this increase is caused by the activity of neural corticotrophin releasing factor (CRF) which has a relationship with sympathetic activity

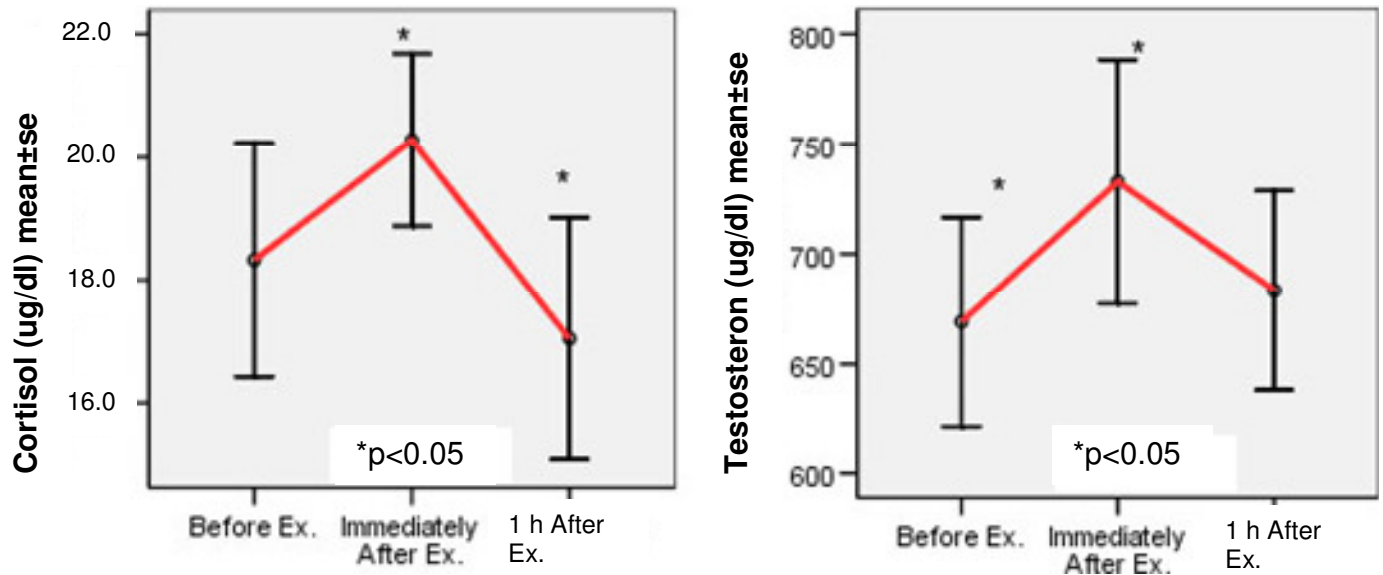


Figure 1. The changes in cortisol and testosterone level.

and stress (McMurray and Hackney, 2000). It has been reported that ACTH increase was 10 to 15% in middle intensity exercise, 100% in high intensity exercises and, 400% in exhausting exercises (Farrell et al., 1983). If the exercise period is too short and the intensity is high, cortisol secretion rate doesn't change (McMurray and Hackney, 2000). However the greater ACTH increasing occurs at exhaustive exercise, the greater cortisol occurs in long duration and high intensity exercise (Lac and Berthon, 2000). In a study, related to marathon races, has been reported that blood testosterone level decreased twofold but cortisol levels increased twofold after races (Franca et al., 2006). In a study, it has been reported that endurance exercises cause more cortisol secretion (Orwoll et al., 2006). In animal and human studies, it has been reported that cortisol loading inhibits testosterone production (Cumming et al., 1983; Bambino and Hsueh, 1981).

Brownlee et al. (2005) have found that there is a negative correlation between testosterone and cortisol levels after intensive exercise and in the recovery period. However, in short-term exercises cortisol level decreases while testosterone level increases (Reeves et al., 2006; Kraemer et al., 2007; Tsolakis et al., 2003). It has been suggested that the increase in the cortisol level at the beginning of the exercise is caused by hemoconcentration and stimulation of ACTH by increased neural CRF secretion related to sympathetic activity as a result of stress caused by the exercise. It is also suggested that the significant decrease in cortisol levels one hour after the exercise is because of the low intensity and duration of the exercise. In this study, hormonal responses of the exercise were lower compared to the literature. It can be considered that this was the result the type of the exercise which the subjects were already accustomed to.

As a matter of fact, the more a sportsman's training level increases, the less amount of secreted hormones releases at the same type of exercise load (Safarinejad et al., 2008). In the study it was found that difference in the urea levels was not significant but the increase in uric acid levels, which emerged immediately after the exercise continued one hour after the exercise and both measurements were different from the pre exercise value (Figure 2). In this study, as a result of gradually increasing exercise intensity, it was observed that there was a significant increase in the uric acid level after the exercise. But urea levels did not change. Urea is produced in the liver from ammonia which is a product of deamination of the amino acids (Guyton and Hall, 2003). However, uric acid is the end product of purine metabolism.

Protein metabolism plays an important role in the increasing muscle protein synthesis in anaerobic and resistance exercises, although protein is used as energy source in the endurance exercises (Lemon, 2000). Amino acids have an energy metabolism regulating role, as well (Haralambie and Berg, 1976). In aerobic exercises, amino acid oxidation increases in correlation with the intensity of the exercise (Babij et al., 1983). Especially urea and uric acid levels increases in the endurance type exercises (Fry et al., 1991; Kaya et al., 2006). The mechanism of this increase is explained as prolonged exercise increases proteins degradation and increasing lactic acid causes blockage in their renal clearance and urinary excretion (Kaya et al. 2006; Nichols et al., 1951). Cortisol also causes mobilization of proteins from the tissues and prevents amino acids transport to the extra-hepatic tissues; as a result, amino acids increase in the blood (Guyton and Hall, 2003). Besides, it is reported that formation of uric acid from purines is associated with

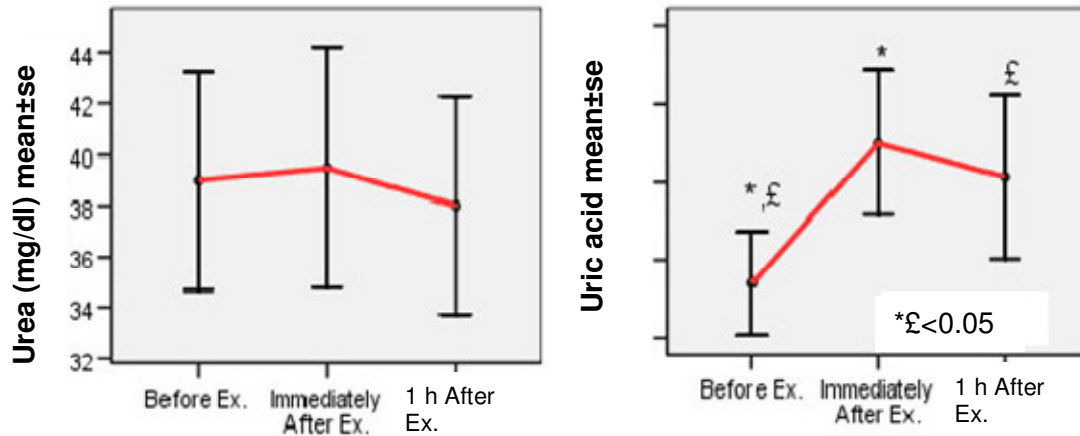


Figure 2. The changes in urea and uric acid level.

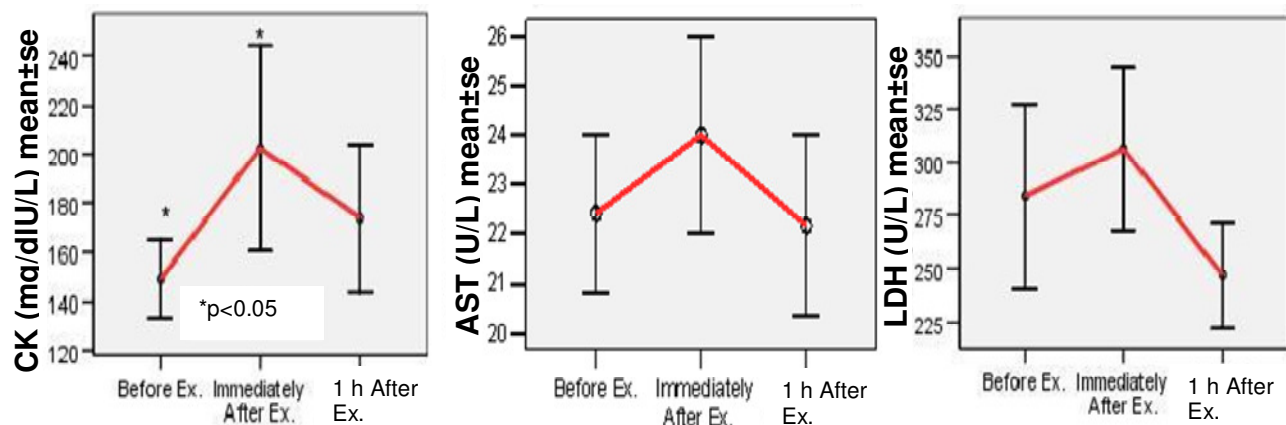


Figure 3. The changes in skeletal muscles enzyme levels.

tissue damage (Banister et al., 1985). In this study the increase in uric acid can be explained by two mechanisms: first; increased cortisol level contributes to muscle damage and releases endogenous purines from the muscle tissue and second; renal clearance rate decreases due to exercise-induced fatigue. When serum skeletal muscle enzyme levels were analyzed, it was found that the increase in the serum CK activity immediately after the exercise was significant but the increase in the CK activity one hour after the exercise was not significant.

The changes in AST and LDH enzyme levels were found to be insignificant (Figure 3). Exercise induced muscle damage occurs in the skeletal muscle following unaccustomed strenuous exercise, especially involving eccentric contractions (Staron and Hikita, 2000). Different types of exercises cause different degrees of muscle damage. In addition, eccentric contraction causes more muscle damage compared to other types of contraction (Davies et al., 2008). The damage caused by unaccustomed contraction leads to degeneration in the myofibrillar structures. Especially the disruption in Z-line emerges a

disruption in the myofibrillar structure as well (Friden et al., 1983). When muscle damage occurs, damage marker increases in the plasma after the exercise. CK, AST and LDH are usually considered as muscle damage markers (Wilson et al., 2008; Kim et al., 2007; Clarkson et al., 1986; Smith and Miles, 2000). The plasma levels of these enzymes increase when skeletal muscle damage occurs. In this study, CK increased significantly immediately after the exercise. This increase tended to decrease one hour after the exercise. The activity of CK one hour after the exercise was not statistically different from the base level. AST and LDH enzyme levels demonstrated an increase immediately after the exercise compared to before the exercise level but this increase was not statistically significant. The increase in the serum CK activity indicates that the exercise protocol used in this study causes muscle damage in micro level. The decrease in the CK enzyme activity one hour after the exercise and the insignificant change in the muscle damage markers proved that muscle damage level was at minimal degrees. The time course of damage related enzyme activity varies according to the type, intensity and duration of the

exercise. It can change between 1 to 5 days (Brancaccio et al., 2007; Howatson and Someren, 2008).

In a study which evaluated the effect of exercise intensity on the CK enzyme activity, it was reported that low and moderate intensity exercises didn't change the serum CK enzyme activity in the first 24 h, but high intensity exercises caused high levels of serum CK activity (Davies et al. 2008). In some studies, it has also been reported that muscle damage markers increase after the exercise, and they decrease to the base level after 24, 48 and 72 h depending on the intensity and duration of the exercise (Coombes and McNaughton, 2000; Maughan et al., 1989). In this study, when evaluated serum enzyme levels related to the muscle damage it can be presumed that the muscle damage was at minimal degrees after the exercise. This is because the subjects were accustomed to type and intensity of the exercise performed. As a matter of fact, training is a preventive factor for muscle damage (Howatson and Someren, 2008).

Conclusions

In conclusion, this study demonstrated that graded maximal exercise increased both testosterone and cortisol levels. It can be explained by the decreased metabolic clearance rate, increased secretion of end products and hemoconcentration caused by the exercise. It was found out that uric acid level increased significantly after exercise. This increase can be explained by the decreased excretion due to the fatigue-related decrease of renal clearance rate and increasing protein degradation as a result of the mobilization of amino acids and increased blood amino acid level with the effect of increasing cortisol level in collaboration with the muscle damage due to the exercise. It can be suggested that the muscle damage in subjects was minimal because the subjects were accustomed to the type of exercise performed in this study. The subjects' training level highly prevented muscle damage.

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