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## EARLY EFFECTS OF SHORT-TERM ENDURANCE TRAINING ON HORMONAL RESPONSES TO GRADED EXERCISE

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Twelve male, sedentary volunteers ( $22.0 \pm 0.7$  yrs) were submitted to three weeks of a bicycle ergometer training, consisting of 45 min exercise (at 70%  $\text{VO}_2\text{max}$ ), 4 times in the first week and 3 times in the next 2 weeks. They performed four incremental exercise tests with the power output increased by 50 W every 3 min until volitional exhaustion: two before training (C1 and C2), and after one (T1) and three (T3) weeks of training. Before and after each load the plasma noradrenaline (NA), adrenaline (A) and blood lactate (LA) concentrations were determined in venous blood samples as well as plasma growth hormone (HGH) and cortisol concentrations before and at the end of exercise. A decrease in NA concentration was found already after 1 week of training at power output of 100 W ( $p < 0.01$ ) and 200 W ( $p < 0.05$ ). Similar decline was maintained after 3 weeks of training. No significant training-induced differences in plasma A concentration were found, however, the thresholds for both catecholamines were significantly shifted towards higher values after 3 weeks of training. One week of training caused a decrease in the pre-exercise ( $p < 0.01$ ), as well as post-exercise ( $p < 0.05$ ) plasma cortisol and HGH concentrations. It was concluded that endurance training induced a decrease in HGH, cortisol and NA concentration already after one week of training. A decline of pre-exercise plasma HGH and cortisol levels with time of experiment may, in part, indicate familiarization to exercise protocol.

**Key words:** *Short-term training - catecholamines -growth hormone - cortisol - catecholamine thresholds*

## INTRODUCTION

Endurance training, as a complex process, involves not only cardiovascular and muscular adaptations to exercise, including diminished heart rate (HR), at rest and during submaximal exercise, increased maximal oxygen uptake ( $VO_{2max}$ ), decreased blood lactate (LA) accumulation with a subsequent shift of the anaerobic threshold towards higher work loads, but also changes endocrine responses to exercise. It has been known for many years that after endurance training the exercise-induced rise in plasma catecholamine, glucagon, human growth hormone (HGH) and cortisol concentrations are markedly diminished (1-3). However, some recent findings on the hormonal responses to physical training have brought confusing results. Most authors confirmed the training induced resting or post-exercise decrement in the plasma catecholamine (CA) (4-5), HGH (6) and cortisol concentrations (7-11) but there are reports neglecting such effect of training (11-14).

It has been emphasized that some adaptive cardiovascular and metabolic responses to endurance training may develop quite early. Only few training sessions have been shown to decrease heart rate (HR) both at rest and during submaximal exercise (15-16). Some authors reported a decrease in blood and muscle LA concentration and a decline in muscle glycogen and phosphocreatine utilization already after 5-7 training sessions (17-19). Moreover, some findings provided an evidence that the adaptive enhancement of the mitochondrial potential in response to endurance training may occur fairly rapidly, i. e. already after 6-10 days (20-22). It should be noted that the mechanism underlying the early endurance training-induced changes still remains unclear. Thus, the investigations on the accompanying hormonal changes may help to explain some conflicting results. Furthermore, little or only controversial informations are available on the time course of potential hormonal adaptations to endurance training and on their physiological consequences. For example, the diminished response of plasma CA concentration to exercise, accompanying the training induced decline in HR or blood lactate level, was found as early as after 3 consecutive days of endurance training performed at 60% of  $VO_{2max}$  (5), while other authors reported such effect not earlier than after 3 weeks of training (4,23). It seems worth to mention, that the results of the previous study from our Laboratory (24) revealed contribution of familiarization of sedentary subjects with the exercise testing protocol in development of the early endurance training-induced adaptations.

Thus, the present study was designed to determine how quickly the moderate exercise training program evokes the hormonal changes in previously sedentary men and if so whether these hormonal responses to exercise correlate with other training-induced early circulatory and metabolic changes.

## MATERIALS AND METHODS

*Subjects*

Twelve healthy, sedentary subjects, defined as individuals not participating in any regular physical activity, gave their written consent to participate in this study, which was approved by the Ethics Committee at the Medical Research Centre, Polish Academy of Sciences in Warsaw. The physical characteristics of the subjects are given in *Table 1*.

*Table 1.* Characteristics of subjects (the values are means  $\pm$  SE, n = 12).

|  |                 |
|--|-----------------|
| Age (years)  | 22.0 $\pm$ 0.7  |
| Body mass (kg)                                     | 79.7 $\pm$ 2.0  |
| Height (cm)  | 181.0 $\pm$ 1.0 |
| VO <sub>2</sub> max (l $\cdot$ min <sup>-1</sup> ) | 2.96 $\pm$ 0.11 |
| HRmax (beats $\cdot$ min <sup>-1</sup> )           | 191.0 $\pm$ 1.9 |
| Maximal Power Output (Watts)                       | 250.0 $\pm$ 9.0 |

VO<sub>2</sub>max = maximal oxygen uptake; HRmax = maximal heart rate

*Experimental design*

The subjects were submitted to a 3-week, controlled endurance training program consisting of 45 min of continuous exercise on a bicycle ergometer (Ital Bike-Atala, Italy) with a mean intensity of approx. 70% of their pre-determined maximal O<sub>2</sub> uptake (VO<sub>2</sub>max). The VO<sub>2</sub>max was measured during the first control incremental exercise test (C1) performed according to the procedure described below. They practiced four times a week during the first week of training and three times a week during the remaining two weeks. The subjects were submitted to 4 graded, incremental exercise tests until volitional exhaustion: twice before starting the training in one week interval (C1 and C2), and then after one (T1) and three (T3) weeks of training. In all four tests the power output was increased by 50 W every three minutes until exhaustion starting from 50 W. The stages were separated by 60-s rest intervals for blood sampling. Two control exercise tests (C1 and C2) were performed in the present investigation to find out whether familiarization with the laboratory testing procedure (for example an insertion of the intravenous catheter) of the subjects participating in such trials for the first time has any influence on hormonal responses to the applied exercise. All exercise tests were conducted at the same time of a day at ambient temperature of 22°C and 60% relative humidity. T1 and T3 tests were performed 48 hours after the last training session.

*Heart rate (HR)*

At rest and during exercise tests HR was measured using Sport Tester (PE 3000, Oulu, Finland).

*Respiratory gas exchange*

Oxygen uptake (VO<sub>2</sub>) was analysed and computed every 30 sec during the control exercise tests (C1 and C2) and then after one (T1) and three (T3) weeks of training with the Cardiopulmonary Exercise System CPX (Med Graphics TM, USA). The VO<sub>2</sub> "leveling off" criterion was used to establish the VO<sub>2</sub>max, necessary to predict an individual mean endurance training intensity.

Before exercise and immediately after each work load blood samples were taken from an antecubital vein via the catheter inserted 30 min before the test for the plasma catecholamine (CA) and blood lactate (LA) determinations. Before exercise and immediately after the last load plasma growth hormone (HGH) and cortisol concentrations were determined.

### *Analytical methods*

Plasma adrenaline (A) and noradrenaline (NA) concentrations were determined by the radio-enzymatic method of Da Prada and Zurcher (25) using the Catechola tests produced by the Institute for Research, Production and Application of Radio-isotopes (Prague, Czech Republic). Plasma HGH and cortisol concentrations were measured by radio-immunoassay using RIA-MJ-99 set or Kortyzol <sup>[125]</sup> Spectria set, (Institute of Atomic Energy, Swierk, Poland) respectively. Blood LA concentration was determined using commercial kits (Boehringer, Mannheim, Germany).

### *Calculations*

Blood lactate threshold (LA-T) was detected for each subject using a log-log transformation of Beaver et al. (26). The threshold work intensity, expressed in watts (WL), was assessed from the intersection of the two linear segments (log LA plotted vs. log WL). Similarly, using the respective two-segmental linear regression, the mean threshold power output was detected also for both catecholamines (plasma NA and A thresholds).

### *Statistics*

The data are presented as means with standard errors (SE) unless otherwise stated. Statistical evaluation of mean differences was made using a two way analysis of variance for repeated measures. Paired Student's t-test was used for the post hoc analyses in the event of a significant F ratio. The level of significance was set at  $p < 0.05$ .

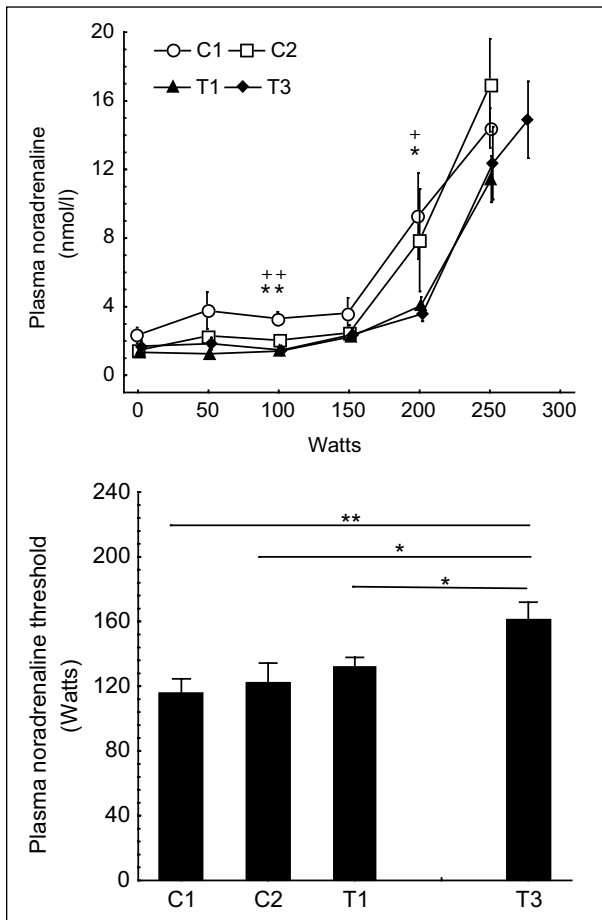
## RESULTS

### *Comparison of two control trials (C1 vs. C2)*

No significant differences in plasma NA, A, HGH and cortisol levels, as well as in HR,  $VO_2$  and blood LA concentration, were found between the two control trials (C1 and C2).

### *Plasma noradrenaline concentration*

Plasma noradrenaline concentration started to rise in response to all four incremental tests just after the beginning of exercise, however, only slight increases were noted during exercise of low and moderate exercise intensities and then the plasma NA increased gradually with increasing work intensity until exhaustion (*Fig.1*). Already after one week of training plasma NA concentration decreased at the power output of 100 W ( $p < 0.01$ ) and 200 W ( $p < 0.05$ ) in comparison with control values (C1). Similar decline in NA concentration was noticed also after 3 weeks of training (*Fig.1*).



*Fig. 1.* Plasma noradrenaline concentrations during incremental exercise tests before training (C1, C2) and after one (T1) and three weeks (T3) of training (upper part) with noradrenaline threshold changes (lower part). Values represent means $\pm$ SE. Significant differences between pretrening (C1) and after one week of training (T1) are denoted as asterisks (\* $p$ <0.05; \*\* $p$ <0.01). Crosses denote differences between values obtained in C1 and after three weeks of training (T3): + $p$ <0.05; ++ $p$ <0.01.

### *Plasma adrenaline concentrations*

Plasma adrenaline concentrations revealed the exponential increase during all the incremental tests (*Fig.2*) similar to that in the plasma NA changes. No significant training-induced differences in the course of plasma A concentration were found either after 1 or 3 weeks of training, as compared with control trials (C1 and C2).

### *Plasma HGH concentration*

The pre-exercise values of plasma HGH were continuously decreasing from C1 to T3 test, with a strong tendency to decline noted already in C2 in contrast to C1 trial ( $p=0.07$ ), and a significant reduction occurring after one and three weeks of endurance training ( $p<0.01$ ; *Fig.3*). A significant training-induced decrease in the post-exercise HGH levels was shown only in T1 as compared with the control trial ( $p<0.05$ ).

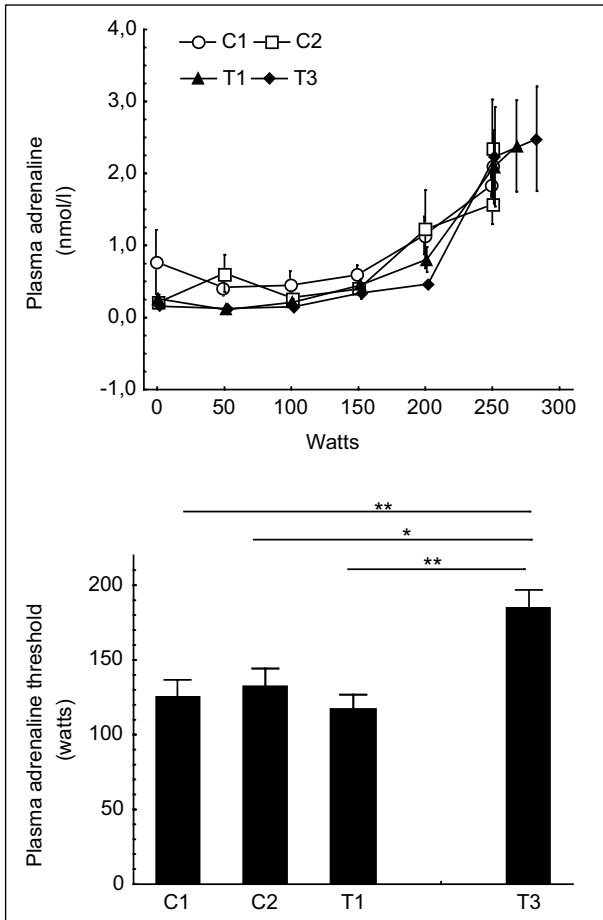


Fig. 2. Plasma adrenaline concentrations during incremental exercise tests before training (C1, C2) and after one (T1) or three (T3) weeks of training (upper part) and with adrenaline threshold changes (lower part). Values represent means and SE. Asterisks denote significant differences between pre- and post three weeks of training: \*  $p < 0.05$ ; \*\*  $p < 0.01$ .

*Plasma cortisol concentration*

The pre-exercise changes in plasma cortisol level were similar to those in HGH concentrations (Fig.3) revealing a decrease in cortisol in T1 and T3 tests in comparison with control trials (C1 and C2;  $p < 0.01$ ). Similarly as for HGH, the training-induced decline in the post-exercise cortisol concentration was found in T1, and T3 test, as compared with the control test ( $p < 0.05$ ).

*Heart rate*

A significant decrease in resting ( $p < 0.05$ ) and submaximal HR (at 150 and 200 W,  $p < 0.01$ ) response to training were noted already after one week of training in comparison with the values obtained in C1 and C2 tests. With continuation of training for further two weeks a significant decline in HR ( $p < 0.01$ ) was found at rest, as well as at each power output, but not at the maximal exercise intensity (Fig. 4).

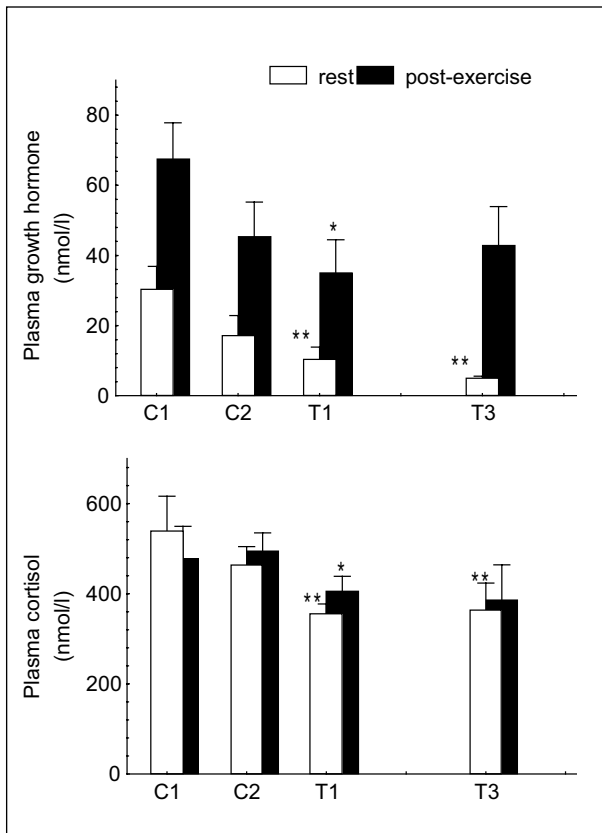


Fig. 3. Pre- and post-exercise changes in plasma growth hormone and cortisol concentrations during incremental exercise tests before training (C1, C2) and after one (T1) or three weeks (T3) of training. Values represent means and SE. Asterisks denote significant differences between pretraining (T1) and after one or three weeks of training: \* $p < 0.05$ ; \*\* $p < 0.01$ .

### Oxygen uptake and maximal power output

The resting and submaximal  $O_2$  uptake values measured during the test performed after one and three weeks of training were similar to those obtained before the training. However, the  $VO_2$  max in T3 ( $3.36 \pm 0.13 \text{ l} \cdot \text{min}^{-1}$ ) differed significantly from that in C1 ( $2.96 \pm 0.11 \text{ l} \cdot \text{min}^{-1}$ ,  $p < 0.03$ ) and in C2 ( $2.98 \pm 0.09 \text{ l} \cdot \text{min}^{-1}$ ,  $p < 0.01$ ) trials. Similarly, after 3 weeks of training maximal power output increased to  $277 \pm 10.4 \text{ W}$  and it was significantly different from both pretraining ( $p < 0.05$ ) tests ( $250 \pm 9.5 \text{ W}$  in C1 and  $250 \pm 9.0 \text{ W}$  in C2), but not with that in T1 test ( $268 \pm 13.9 \text{ W}$ ).

### Blood lactate

Changes in blood LA showed an exponential course of changes during all four exercise tests (Fig 4). After both 1 and 3 weeks of training blood LA concentrations did not significantly differ from the values obtained in C2 test. The lactate threshold (LA-T) showed no change after 1 week of training but it was elevated ( $p < 0.05$ ) at the end of the 3-week training period (Table 2).

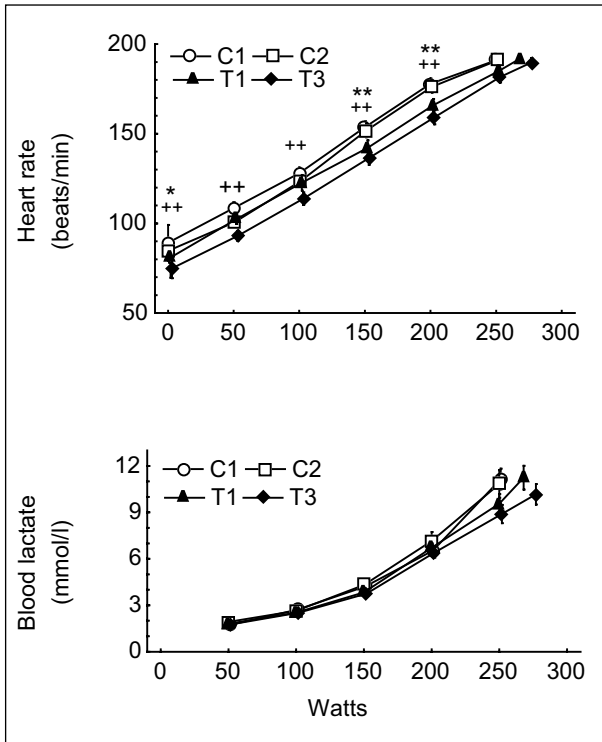


Fig. 4. Heart rate and blood lactate concentrations during incremental exercise tests before training (C1, C2) and after one (T1) or three (T3) weeks of training. Values represent means±SE. Significant differences between pretraining (C1) and after one week of training (T1) are denoted as asterisks (\*p<0.05; \*\*p<0.01). Crosses denote differences between values obtained in C1 and after three weeks of training (T3): +p<0.05; ++p<0.01.

*Catecholamine thresholds*

The sudden, non-proportional rise in plasma NA and A levels during progressive exercise test, indicated here as NA-T and A-T, did not change after 1 week of training but they were shifted towards higher exercise loads at the end of the 3-week training period (Fig.1 and Fig.2). A significant increase in both NA- and A-thresholds was also found between T1 and T3 trials (p<0.02).

A significant relationship between exercise blood LA and both plasma NA and A concentrations (n=180) was ascertained (r=0.751; p< 0.001 and r=0.655; p< 0.001, respectively). Moreover, the mean threshold exercise intensities for NA and A during both control trials (C1 and C2) and the training-induced changes of

Table 2. Threshold exercise intensities for catecholamines (NA-T, A-T) and blood lactate (LA-T). The values are means ± SE.

|          | C1           | C2           | T1           | T3           |
|----------|--------------|--------------|--------------|--------------|
| NA-T (W) | 115.5 ± 9.1  | 122.0 ± 10.5 | 131.7 ± 6.2  | 161.0 ± 10.9 |
| A-T (W)  | 125.1 ± 15.6 | 132.2 ± 12.1 | 116.6 ± 10.2 | 185.2 ± 11.6 |
| LA-T (W) | 120.9 ± 6.1  | 116.2 ± 8.1  | 127.7 ± 5.2  | 140.8 ± 7.0  |



these values after 1 week of training (T1) were comparable to those noticed for LA threshold but after 3 weeks of training they were significantly higher ( $p < 0.05$  and  $p < 0.01$ , respectively) (Table 2).

## DISCUSSION

The present study showed that in previously sedentary young men only four 45 min-exercise sessions performed within 1 week (with the intensity of approx. 70%  $VO_{2max}$ ) caused a significant decrease in the plasma noradrenaline concentration measured at moderate work intensities. The next two weeks of training led to a similar decline in NA concentration. In contrast, the time-course of plasma A level did not differ from that determined during control tests either after 1 or 3 weeks of training. The early decrement in exercise NA concentration induced by endurance training was accompanied by a significant decrease in HR which was even more distinct after further 2 weeks of training. This finding confirms the report of Helyar *et al.* (5), who applied similar training regime, although other authors described such effect not earlier than after 3 weeks of training (15,23). In spite of these differences it can be assumed, that reduced sympathoadrenal drive to the heart might be responsible for the well-known training induced decrement in HR. It is worthy to notice, that a significant decline in HR was found in our study also at rest already after 1 week of training and with continuation of training for further two weeks, whereas the training-induced attenuated noradrenaline level was observed only during exercise. It may be suggested that the reduced sympathetic response is not solely responsible for the reduced heart rate, since Winder *et al.* (15) have found that heart rate during exercise continued to decrease as an effect of training even after the catecholamine response had plateaued. It should be mentioned that the results of the previous study from our Laboratory, in which the same exercise protocol was used (24) showed no effects of 3-week endurance training on other circulatory response, like resting and post-exercise blood pressure and cardiac output.

The results of the present study confirmed an exponential pattern of increase in plasma catecholamine concentration during progressive exercise, parallel to that in blood lactate concentration, shown in our earlier findings (27-28). The similarity of the time-courses of changes in plasma CA to that of LA during graded exercise is emphasized by close correlations found in this study between plasma catecholamine and blood lactate concentrations. Moreover, the calculated thresholds of plasma CA (NA-T and A-T) occurred at almost the same exercise intensity as lactate threshold during both the control tests and those during T1, but not T3. The similar effect of training on all those thresholds, (as concerns its magnitude and time of appearance), shifted them towards higher exercise intensities already after few training sessions with the most distinct rise after 3 weeks of training. It is worthy to notice that the significant increase in both

VO<sub>2</sub>max and maximal power output was also observed in the present study not earlier than after 3 weeks of training. The apparent coincidence of catecholamine and lactate thresholds does confirm earlier data (27-30). An important role of central command for the sympathetic nervous system response to exercise has been shown (31). Thus, the attenuated stimulating effect of the efferent impulses derived from the cortical motor centres on the control systems of catecholamine release during exercise, diminished due to training or familiarization process to the exercise protocol, cannot be excluded. Furthermore, it can be assumed that the neural afferent signals from the muscle metabolic receptors via a fatigue-feedback mechanism from and to the working muscles might participate in the activation of catecholamine release (31-32). It seems probable, that a delayed lactate and other metabolite accumulation as an early effect of aerobic training might be responsible for the training-induced inhibition of CA release observed in this study (33). In most papers it has been shown that during the first days of training LA accumulation was significantly decreased both in muscles (17-18,21) and in blood (33-35).

The present results demonstrated the early pre-exercise, as well as post-exercise, decrement in the plasma HGH levels. The further decrease in resting HGH values was observed after next 2 weeks of training, what is in accordance with the findings of Horber (6). Other authors did not confirm these observations (8,10-12,14). It should be noted, however, that the exercise-induced changes in HGH concentrations exhibit rather irregular, polyphasic pattern probably due to its delayed and pulsatile secretion and, therefore, there are difficult to evaluate (2).

Although the role of the increased amount of HGH released during exercise is not fully understood, seems that it contributes in part to metabolic fuel adaptations during exercise and post-exercise tissue repair. Both nervous and humoral factors have been considered as signals for human growth hormone release (2). Significant correlations between plasma HGH and catecholamine and blood LA concentrations during progressive exercise found in our previous study (27) and also in the present study would allow us to suggest that lactate accumulation and the increment in CA levels may be considered as humoral signals for HGH release. In fact, greater activation of anaerobic glycolysis and lactate formation for both endurance and resistance efforts increases the amount of HGH released (36). It seems also likely that neural afferent signals from muscle metabolic receptors might participate not only in the activation of exercise catecholamine release (32,37) but also in stimulation of HGH secretion. Thus, the diminished CA levels found in this study may partly contribute to attenuated HGH response to graded exercise, the more that close correlations have been established between plasma HGH and CA concentrations during progressive exercise. Moreover, the early training-induced decrement in HGH response to exercise observed in our study after only one week of training might be explained by lower contribution of LA accumulation in evoking the GH release, since the

increased muscle potential has been evidenced already after few days of endurance training (20-22) accompanied by a decrease in muscle and blood LA concentration (17-19). Nevertheless, the results of other studies (38, 39) demonstrated that the close relationship between plasma HGH and blood LA concentrations during a prolonged continuous or incremental exercise was not always present, indicating that the HGH response to physical exercise might not be directly related to blood lactate accumulation.

We have also found, that endurance training does modify the pre-exercise and post-exercise cortisol levels in a similar way as plasma HGH concentrations. The training-evoked decrease in plasma cortisol concentrations was observed also in other reports (7,9), although Marc *et al.* (13) found this effect not earlier than after 20 weeks of training.

As it has been hypothesized the familiarization with exercise protocol might reduce intraindividual variability of some cardiovascular and hormonal responses because of physiological stress limitation.

The early training-induced decrease in resting and submaximal HR and exercise plasma noradrenaline concentration may be partly due to familiarization of subjects to the exercise testing protocol. This assumption has been supported by our previous study of similar protocol (24), which revealed more pronounced decrease of EMG activity of the *soleus and triceps femoris* muscles in the second control test (C2) than in the C1 test. Moreover, a decline of pre-exercise plasma HGH and cortisol levels found in the present study already after one week of training, may also, in part, indicate familiarization of sedentary subjects to exercise protocol.

In summary, this study demonstrated that moderate endurance training induced a decrease in HGH, cortisol and NA concentration already after one week of training, which is the early hormonal effect of increased physical activity. A decline of pre-exercise plasma HGH and cortisol levels with time of experiment may, in part, indicate familiarization to exercise protocol.

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