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Hormonal responses to repeated bouts of supramaximal cycle ergometer exertions

Authors' contributions:
A Study design
B Data collection
C Statistical analysis
D Data interpretation
E Literature search
F Manuscript preparation
G Funds collection

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Summary

Study aim: To determine hormonal (growth hormone, cortisol and testosterone in blood) responses to a series of supramaximal cycle ergometer exercise bouts.
Material and methods: Seven physical education students were subjected to two series of exercises, 5 bouts each, spaced by one week. The load in the first bout in each series amounted to 100% of work output recorded previously in the Wingate test, all other bouts amounting to 50%. In Series A and B, the loads were 10 and 5% of body mass (BM), respectively. Individual bouts were separated by 2-min intervals. Growth hormone (GH), cortisol (C), testosterone (T) and blood pH were determined in fingertip blood before the exercise, following Bouts 3 and 5, and 30 min after the exercise was discontinued.
Results: Growth hormone, cortisol and testosterone concentrations increased significantly after both series of exertions.
Conclusions: Repeated bouts of sprint-like exertions may be applied to design training protocols involving the desired kind of stimulation of the hormonal control, considering the necessary external load and exercise duration.

Key words: Growth hormone – Testosterone – Cortisol – Supramaximal exercise – Anabolic-catabolic index

Introduction

Isolated or serially repeated bouts of supramaximal laboratory exercises are known to be a potent stimulus of hormonal responses [3,7,31,35]. This is associated with the duration of exertion, one of the most potent stimuli (like exercise intensity) of hormonal responses. The overall drop in power output amounts to 15 – 60%, depending on bout and intermission durations [10,14]. That decrease in power output is related to intracellular phosphagen depletion [3,36], and the recovery-induced repletion is very fast and related to intermission duration [6]. This process is influenced by the acid-base equilibrium [28] and hormonal status [24,29]. A 30-s workout induced increases in cortisol concentrations in athletes [24,25], but not in control subjects [29]. On the other hand, cortisol levels decreased immediately after five 15-s bouts and returned to initial values after a 15-min rest [16], testosterone concentrations remaining unchanged.

Bernardes and Radomski [1] reported growth hormone responses to a continuous exercise at 60% and to intermittent one at above 80% VO_2max, hormone concentrations being higher after the former. Opposite results were reported by VanHelder et al. [33], although work outputs in both kinds of exercise were the same in both papers. The exercise-induced burst of growth hormone is supposed to depend on exercise intensity [1,11,33,34] and duration [19,21,34], changes in body temperature [22], disturbances in the acid-base balance [27] and work capacity [32].

Despite a vast literature on that subject, very few reports exist on the effects of muscle contraction velocity (e.g. pedalling at various rates) on hormonal responses to supramaximal exercise [4,8,31]. Thus, the aim of the study was to determine growth hormone, cortisol and testosterone responses to serially repeated bouts of supramaximal exercise differing in the pedalling rate but identical with respect to work output.

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Material and Methods

Seven male, untrained, physical education students volunteered to participate in the study after having familiarised themselves with the study aim and protocol. They were informed they could withdraw from the study at any moment. Their age was 24.4 ± 0.8 years, body height 181.5 ± 3.8 cm, body mass 85.0 ± 9.6 kg. The study was approved by the local Commission of Ethics.

Physical capacity of all subjects was determined by applying PWC_{170} test [30] prior to the study. The test consisted of two cycle ergometer exercises lasting 5 min each, the loads amounting to 100 or 150 W, respectively. Heart rate was recorded by using Sport Tester (Polar Electro Oy, Finland). The obtained PWC_{170} index served to compute maximal oxygen uptake ($\dot{V}O_2max$) according to Karpman's formula [20].

Standard 30 s Wingate test [17] was used to determine anaerobic capacity. The test was preceded by a standard warm-up followed by 5-min rest. The loads applied were equal to 7.5% of individual body mass. Monark 824 E cycle ergometer (Sweden) was used, exercise variables being recorded on-line by using MCE 4.0 software (JBA, Z.Staniak, Poland). After having adjusted the seat and handlebar, the subjects were prompted to pedal at maximum rate, without leaving the seat, the feet being fastened to the pedals. The following variables were recorded: maximum power output (P_{max}), mean power output (P_m), total work output (W), and fatigue index (FI), defined as the ratio of power decrease to the final power output.

Participants were subjected to two sessions of exercises, spaced by one week.

Session A consisted of 5 supramaximal exercise bouts, the external load being equal to 10% of body mass. Work output in Bout 1 was equal to that predetermined in the Wingate test and amounted to 20.79 ± 2.07 kJ, and in Bouts 2 – 4 was by one-half lower (10.43 ± 1.05 kJ each).

The protocol of Session B was identical, except the external load, which amounted to 5% of body mass. In order to keep the same work outputs as in Session A, the pedalling rate had to be increased. Individual bouts of exercise were spaced by 2-min rests in both sessions.

Blood for hormonal assays was sampled from fingertips before the exercise, after Bouts 3 and 5, and 30 min following the exercise. Growth hormone (GH) was determined by using commercial ELISA kits (Eurogenetics, Germany), for cortisol (C) and testosterone (T) specific ELISA kits were used (Orion Diagnostics, Finland); a Micro-Reader photometer I (UK) was used to read light absorbance at 450 nm. Ciba-Corning 248 blood gas analyser (UK) was used in determining blood pH. The ratio of the testosterone-to-cortisol concentrations served as the so-called anabolic-catabolic index (T/C).

Data analysis included ANOVA with repeated measures, followed by a *post-hoc* LSD test, and Pearson's correlation coefficients, the level of $p \leq 0.05$ being considered significant. Statistica™ v. 5.5 (StatSoft, Inc., USA) software was employed.

Results

Mean values (±SD) of PWC_{170} and $\dot{V}O_2max$ amounted to 288 ± 76 W and 4173 ± 778 ml/min, respectively.

Table 1. Mean (±SD) blood pH and hormonal responses to repeated cycle ergometer exercises at external loads equal to 10 or 5% of individual body mass (n = 7)

Load		pH	GH [ng/ml]	T [ng/ml]	C [ng/ml]	T/C
10% BM	0	7.400 ± 0.017	0.29 ± 0.06	4.30 ± 0.95	163 ± 28	3.41 ± 0.92
	3	7.227 ± 0.034*	0.32 ± 0.06	5.41 ± 1.18*	161 ± 32	4.32 ± 0.89*
	5	7.216 ± 0.052*	0.93 ± 0.73	5.54 ± 1.89*	151 ± 32	4.57 ± 0.95*
	R30	7.400 ± 0.042#	11.8 ± 9.9*# º	5.14 ± 0.54*	196 ± 28*#	3.37 ± 0.56#
5% BM	0	7.414 ± 0.016	0.36 ± 0.16	4.71 ± 0.94	164 ± 38	3.70 ± 0.69
	3	7.228 ± 0.045*	0.41 ± 0.15	5.60 ± 1.28*	172 ± 39	4.13 ± 0.55*
	5	7.204 ± 0.043*	1.68 ± 1.86	5.80 ± 1.20*	175 ± 41	4.32 ± 1.06
	R30	7.380 ± 0.026*#	7.9 ± 7.3*#	4.90 ± 0.69	197 ± 39*	3.22 ± 0.60#

Legend: 0: Pre-exercise; 3, 5: After Bout 3 or 5; R30: 30 min post-exercise; * Significantly ($p<0.05$) different from the pre-exercise value; # Significantly ($p<0.05$) different from the value after Bout 5; º Significantly ($p<0.05$) different from the respective value at 5% load

Mean power outputs at 5 and 10% BM loads amounted to 542 ± 77 and 688 ± 95 W, respectively, and differed significantly ($p<0.05$) from one another.

In Session A, fatigue index decreased from 27 ± 6% in Bout 1 to do 20 ± 5% after Bout 5, pedalling rate decreased from 87 ± 7 to 83 ± 7 rpm, and exercise duration from 30 ± 3 to 16 ± 2 s, respectively. In Session B, the respective values were 21 ± 7 and 13 ± 5% (fatigue index), 138 ± 9 and 129 ± 17 rpm (pedalling rate), and 38 ± 5 and 20 ± 3 s (bout duration).

Significant changes in hormone concentrations and in the acid-base equilibrium were observed in both exercise sessions (Table 1). Highest concentrations of GH were noted 30-min post-exercise, those for loads equal to 10% BM being significantly greater that for the 5% load. No other between-session differences were found for pH or GH, as well as for the areas under the GH-curves in time (AUC). The latter values for Sessions A and B amounted to 196 ± 154 and 150 ± 129 ng/min/ml, respectively. The concentrations of testosterone (T) significantly increased 5 min post-exercise, but 30 min later were close to the pre-exercise levels. Cortisol (C) concentrations under a load equal to 5 or 10% of body mass gradually and significantly increased 30 min post-exercise compared with the resting value. The anabolic-catabolic index (T/C) significantly increased immediately post-exercise at both loads and the decreased 30 min post-exercise.

The values of $\dot{V}O_2max$ were significantly ($p<0.05$) correlated with relative power output ($r = –0.607$) or fatigue index in the Wingate test ($r = –0.768$). Peak GH concentration also correlated significantly ($p<0.05$) with the relative power or work outputs ($r = –0.617$ and $–0.814$, respectively). Growth hormone levels recorded after Bouts 3 or 5 in both sessions correlated with maximum power output or fatigue index in the Wingate test, the coefficients ranging from 0.642 to 0.815.

Discussion

The intensity [11,34] and duration [19,21,34] of exercise are known to affect the exercise-induced hormonal responses. Our results show that intermittent exercise brought about a marked lactate acidosis, disturbances in acid-base equilibrium and is a strong stimulator of hormonal secretion (GH, testosterone and cortisol).

Some authors [5,26] reported short-lasting exertions to increase serum testosterone levels. In contrast to Obmiński *et al*. [26], who noted increases amounting to 8.5 and 11.3% following the first or second exercise, respectively, as high increases as 29 and 23% at loads 10 or 5%, respectively, were recorded in this study. After a 30-min recovery, testosterone concentrations decreased to pre-exercise values. However, unlike the findings of Ježova *et al*. [18], changes in pedalling rhythm or intensity did not bring about significant changes in testosterone levels.

Sprint-like exertions disturb homeostasis but also enable adaptation to an increased energy demand by mobilising the hormonal system [13]. Local hypoxia in working muscles may bring about increased secretions of cortisol and growth hormone [23]; the cortisol response, apart from being individually variable, depends on the intensity and duration of exercise, i.e. on its energy cost [2,9,12]. As reported by Obmiński *et al*. [26], two supramaximal bouts of exercise 15 min apart, lasting 10 s each, induced no changes in the post-exercise cortisol secretion. In this study, a series of 5 bouts of supramaximal exercise at a load equal to 5 or 10% of body mass was followed by significant increases of cortisol concentration 30 min post-exercise but not immediately after the last exercise bout. The observed differences in the anabolic-catabolic index were due to a faster and shorter exercise-induced response of testosterone compared to cortisol.

Sutton [32] reported significant cortisol and growth hormone responses in subjects of low $\dot{V}O_2max$ (2.3 l/min) following a submaximal exercise (750 kpm/min) lasting 20 min. In subjects with higher capacity (4.55 l/min), GH remained unchanged, cortisol level decreased, and that difference between subjects of low and high capacity persisted until 50 min of recovery. The negative correlations between $\dot{V}O_2max$ and peak power output or fatigue index in the Wingate test, together with a higher GH-response observed in subjects with low anaerobic power output, are indicative of low tolerance of subjects with low aerobic and anaerobic capacity in this study. Besides, an increased GH secretion was associated with greater decreases in power output and lower work output. Therefore, GH concentration may serve as a marker of anaerobic, glycolytic endurance. On the other hand, the GH-response was higher in subjects who had greater anaerobic, lactic maximal power output. Thus, the GH-response would be higher in sprinters than in endurance athletes. That view is supported by the results of Nevill *et al*. [24], who noted greater GH-responses to 30-s sprints in short-distance (100 – 400 m) male or female runners than in the long-distance (1.5 – 10 km) ones.

High concentrations of cortisol may inhibit an excessive GH secretion in an intense exercise [15] but high post-exercise levels of cortisol were also reported to be accompanied by high GH concentrations [2]. In this study, GH concentrations 30 min post-exercise were

higher at the load equal to 10% of body mass than under a 5% load, the cortisol levels being alike. This suggests that pedalling rate and/or exercise intensity are a significant, physiological stimulus of growth hormone secretion.

Earlier results of Cherry *et al*. [4] suggested that pedalling rate in supramaximal exertions lasting 6 or 30 s had little effect on muscle metabolism. However, a 30-s exercise at a load equal to 7.5% of body mass brought about an increase in cortisol (but not in growth hormone) while at 10%-load a decrease was observed [31]. In contrast, in this study significantly higher concentrations of growth hormone were noted 30 min post-exercise at the load equal to 10% of body mass compared with the 5% one. A difference in exercise protocols between the two studies was probably responsible for various hormonal responses.

In conclusion, the protocols of sprint-like exertions presented in this study may be applied to design training protocols involving the desired kind of stimulation of the hormonal control. Of course, such training protocol would be based on the necessary external load as well as exercise duration.

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