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Effects of active vs. passive recovery during Wingate-based training on the acute hormonal, metabolic and psychological response

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ABSTRACT

Objective: The exercise-induced metabolic stress can be influenced by the mode of recovery and is associated with acute hormonal responses. Therefore, it is hypothesized that active recovery between high intensity intervals reduces the metabolic stimulus and therefore the hormonal response compared to passive recovery.
Design: 12 male cyclist/triathletes performed four 30 s all-out intervals, either with active (A) or passive (P) recovery between each bout. Human growth hormone (hGH), testosterone and cortisol, vascular endothelial growth factor (VEGF), hepatocyte growth factor (HGF) and macrophage migration inhibitory factor (MIF) were determined pre, 0', 30', 60' and 180' after both interventions. Metabolic stimuli and perturbations were characterized by lactate, blood gas (pH, BE, HCO_3^-, PO_2, PCO_2), and spirometric analysis.
Results: Both interventions caused a transient increase in circulating levels of cortisol, testosterone, testosterone/cortisol-ratio, hGH, VEGF and HGF. Transient differences between A- and P-recovery were found only for testosterone and HGF directly after exercise, despite significant differences in metabolic disturbances (lactate, acid base status).
Conclusion: Based on the data of testosterone, hGH and the testosterone/cortisol-ratio, as well as on the data of VEGF and HGF it appears that this kind of exercise protocol may promote anabolic processes and may lead to pro-angiogenic conditions independent of the mode of recovery. However transient differences between A- and P-recovery were shown for testosterone and HGF. In contrast, cortisol and hGH, which are known to be sensitive for metabolic perturbations (e.g. pH) showed no differences. Therefore, it is proposed that if a certain threshold for metabolic perturbations is exceeded, a hormonal response is induced, which does not differ between A- and P-recovery.

1. Introduction

In the last years lots of studies were published about high intensity training (HIT). Most studies mainly focused on the comparison of HIT vs. high-volume low-intensity training (HVT), showing that HIT is an effective and time saving method to improve endurance performance (for review see [1,2]). Summarizing the design of HIT protocols, intensities of 90–95% of maximal heart rate (HR) up to "all-out" and durations of 30 s–5 min were mainly used. Only a few studies investigated the effects of different arrangements of HIT (on performance parameters), mainly focusing on different intensities, durations and work-to-rest ratios [1]. Influences and effects of other arrangements or modifications of HIT protocols are largely unknown. Studies about the effects of active (A) and passive (P) recovery do exist, but these investigations mainly focused on acute changes of performance and changes in lactate levels and pH [3–5]. Up to now, no study focused on the effects of A- and P-recovery on other parameters like the acute hormonal response. These previous studies showed, that A-recovery between bouts leads to a faster normalization of pH and a faster elimination of lactate compared to P-recovery [6]. This exercise-induced metabolic stress is associated with acute hormonal responses [7,8] and chronic muscular adaptations following training [8]. Therefore, acute metabolic stress and acute hormonal responses appear to be critical for adaptations, tissue growth and remodeling [8]. It was shown that the reason of an increase in circulating cortisol and hGH levels is in part a decreased blood pH and is influenced by lactate levels [7]. Furthermore, microenvironment signals, such as acidosis and lactate have been suggested to play a major role in the control of VEGF-A production, and consequently in modulation of angiogenesis [9,10]. As these anabolic and angiogenic hormones play an important role in inducing endurance adaptations (e.g. angiogenesis, erythropoiesis, lactate transport), training-induced increases of

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these hormones may be beneficial for endurance performance. However this hormonal response might be influenced by the mode of recovery. The faster elimination of lactate and the faster normalization of pH during A-recovery decrease the time of exposure to these potent metabolic stimuli, possibly leading to different hormonal responses to the same training load. Therefore, it is hypothesized that active recovery between high intensity intervals reduces the metabolic stimulus and therefore the hormonal response compared to passive recovery.

2. Materials and methods

2.1. Subjects

Twelve healthy, nonsmoking male triathletes/cyclists (mean ± SD, age: 24.7 ± 3.4 years, weight: 77.5 ± 6.3 kg, height: 183.9 ± 6.3 cm, relative VO_2max: 64.3 ± 9.7 $mL \cdot min^{-1} \cdot kg^{-1}$) volunteered and gave written informed consent to participate in this study. The weekly training amount was ~12–17 h per week. All athletes had at least 4 years of training experiences. The study protocol was performed in accordance with the declaration of Helsinki and the Ethical Committee of the university.

2.2. Exercise study protocol

Before the participation, subjects performed a step test to determine VO_2max (Zan 600, Zan Messgeräte, Oberthulba, Germany) and peak power output (PPO), in order to determine the proper warming-up and recovery intensity for each subject for the main experiments. The step test consisted of cycling at ≥80 rpm with an initial workload of 100 W for 5 min and incremental 40-W increases every 5 min until volitional exhaustion was reached. After termination of the step test and a 30 min rest period, the subjects were familiarized with the exercise study protocol, as they had to perform two 30 s all-out exercise bouts.

In order to test the hypothesis that the mode of recovery influences the acute hormonal response, the subjects participated in two experimental trials, each separated by one week in a randomized order. Both interventions consisted of four 30 s maximal effort ("all-out") exercise bouts on a cycle ergometer (Schoberer Rad Meßtechnik SRM GmbH, Jülich, Germany) adjusted to an isokinetic mode set to a cadence of 120 rpm, each separated by 7:30 min of recovery (10 min after the last bout). In order to modulate the metabolic stress, recovery was performed either active (A) at an intensity of 45% PPO between the all-out intervals, or passive (P) in a resting position on the cycle ergometer (Fig. 1). After P-recovery the subjects started pedaling 20 s before the next bout, in order to have similar prerequisites for each bout between A- and P-recovery. Before each experimental trial, the subjects warmed up for 10 min at an intensity of 50% PPO. The subjects were instructed to perform the tests in a sitting position on the ergometer. During the HIT interventions all subjects were vocally encouraged to achieve maximal power output. During each session environmental conditions (temperature and humidity) were kept constant and all tests were carried out at the same time of the day (8 a.m.) in order to prevent diurnal variations in performance and the hormonal status. Always the same two investigators attended the tests. During each session spirometric data were determined. The subjects were not allowed to perform strenuous exercise 24 h before testing.

The food intake before the tests was standardized to the extent that subjects recorded their food intake on the day before the first test, and then were advised to reproduce their diet before each test day. In addition carbohydrate-rich foods were recommended to the subjects.

The 1-d food record was used to make sure that the diet on the day before each test was similar in terms of meal and snack times, meal composition and sizes, and fluid intake. The participants were instructed how to fill out a food record, and before each test day, the participants were given a photocopy of their record and they were instructed to reproduce their diet as precisely as possible. A trained nutritionist gave recommendations on how to substitute certain foods in order to achieve a comparable nutrient intake. In addition, each participant consulted a trained nutritionist during a preliminary meeting before the study. In this meeting, the importance of carbohydrates was emphasized and instructions were given on how to achieve a high carbohydrate intake in the range of 6 g/kg [11].

A last snack was allowed 2 h before the test. 30 min after each of the three tests, the subjects received 500 mL of a low fat chocolate milk and additional energy-bars. Food intake was adjusted so that energy intake matched the calculated energy expenditure of each trial. After the ingestion, the subjects were only allowed to drink water until the last blood sample was withdrawn.

2.3. Calculations

For each of the two sessions, total energy expenditure (EE) [kJ] was calculated according to Scott et al. [12] by the following formula:

$$EE_{\text{Warm-Up/Interval}} = \text{time} \cdot 21.1 \cdot VO_{2\text{Warm-Up/Interval}} + (\Delta\text{lactate} \cdot \text{body weight} \cdot 0.003 \cdot 21.1)$$

$$EE_{\text{recovery}} = \text{time} \cdot \left(19.6 \cdot \left(VO_{2\text{Recovery}} - VO_{2\text{Warm-Up}}\right)\right) + 21.1 \cdot VO_{2\text{Warm-Up}}$$

where the caloric equivalent was 21.1/19.6 kJ per liter of oxygen, and 1 $mmol \cdot L^{-1}$ of lactate was equivalent to 0.003 L of oxygen. For warm-up, Δlactate was defined as the difference between after warm-up and rest-values, and for the intervals, Δlactate was equivalent to the difference between the value after the interval and after warm-up. The first interval was considered representative for all intervals so that the relative contribution of Δlactate was extrapolated based on the mean power during the interval. The calculated EE for warm-up, the intervals and the recovery phases were then summed up: $EE_{\text{total}} = EE_{\text{Warm-Up}} + EE_{\text{Intervals}} + EE_{\text{recovery}}$. Furthermore total work [kJ] was determined by the following formula:

$$\text{Total work} = (\text{MP [W]} \cdot \text{exercise time [s]}) \cdot 1000^{-1}, \text{ where MP is the mean power.}$$

2.4. Measurements

In order to quantify the metabolic stress, capillary samples from the earlobe were collected for blood gas (AVL Omni 6; Roche Diagnostics GmbH, Mannheim, Germany) and lactate analysis (EBIOplus; EKF Diagnostic Sales, Magdeburg, Germany) during each of the two sessions at the time points shown in Fig. 1. Under our laboratory conditions, the coefficient of variation for repeated measurements of blood lactate concentration is routinely 1.2% at 12 $mmol \cdot L^{-1}$. For blood gas parameters the corresponding coefficient of variation is 3.2%.

Venous samples were collected for the determination of cortisol, human growth hormone (hGH), testosterone, vascular endothelial growth factor (VEGF), hepatocyte growth factor (HGF) and migratory inhibiting factor (MIF). One venous blood sample was taken before exercise (pre), and four samples were taken at 0 min (0′), 30 min (30′), 60 min (60′) and 180 min (180′) post-exercise. Nine and a half milliliters of blood was collected by the Vacutainer blood withdrawal system (Becton Dickinson). After storage at 7 °C for ~30 min for deactivation of coagulation factors, the blood samples were centrifuged for 10 min at 1.861 g at 4 °C (Rotixa 50, Hettich Zentrifugen, Mühlheim, Germany). The serum was stored at −80 °C till analysis. Serum levels of cortisol ($ng \cdot mL^{-1}$), hGH ($mLU \cdot mL^{-1}$), testosterone ($ng \cdot mL^{-1}$), VEGF ($pg \cdot mL^{-1}$), HGF ($pg \cdot mL^{-1}$) and MIF ($ng \cdot mL^{-1}$) were determined by using human ELISA kits (Cortisol ELISA EIA-1887 (detection range: 2.5–800 $ng \cdot mL^{-1}$; intra-assay and inter-assay variations: 8.1%