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Metabolism Clinical and Experimental 55 (2006) 13–19

Metabolism
Clinical and Experimental

www.elsevier.com/locate/metabol

Adiponectin and stress hormone responses to maximal sculling after volume-extended training season in elite rowers

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Received 17 March 2005; accepted 20 June 2005

Abstract

The purpose of this study was to investigate the resting and short-duration exercise-induced hormone responses of male rowers as a result of 6 months of volume-extended training season. Body composition, maximal aerobic capacity, and on-water 2000-m sculling performance were assessed before and after a 24-week training in elite rowers (n = 11; 193.1 ± 5.2 cm; 91.6 ± 5.8 kg; maximum oxygen consumption [$\dot{V}O_2$max], 6.2 ± 0.5 L · min^{-1}). Six rowers were selected (SEL; 192.0 ± 6.3 cm; 93.5 ± 7.1 kg; $\dot{V}O_2$max, 6.4 ± 0.4 L · min^{-1}) and 5 were not selected (N-SEL; 194.8 ± 4.1 cm; 89.6 ± 4.0 kg; $\dot{V}O_2$max, 6.0 ± 0.5 L · min^{-1}) for the national team. Resting adiponectin did not change as a result of prolonged training. Adiponectin did not change after 2000-m rowing at baseline either. No responses were also observed 24 weeks later in SEL rowers, whereas a significant decrease ($P < .05$) was observed in N-SEL rowers. At the same time, leptin also decreased after the first 30 minutes of recovery in N-SEL rowers. After the training period, immediate postexercise increases in growth hormone and testosterone were significantly higher in the whole group of rowers. No differences in cortisol responses were observed before and after the training period in SEL and N-SEL rowers. In conclusion, it appears that resting adiponectin does not change as a result of prolonged training. Training may modify adiponectin response to an short-duration exercise depending on the performance level of athletes. Decreased postexercise adiponectin and leptin values in rowers with lower performance capacity may be indicative of the inadequate recovery of these athletes.

1. Introduction

Recent research in the biology of adipose tissue indicates that it is not simply an energy storage organ, but also a secretory organ that synthesizes multiple adipocytokine proteins, including leptin, tumor necrosis factor α, resistin, adipsin, plasminogen-activator inhibitor type I, interleukin 6, and adiponectin, thus acting as an endocrine organ [1,2]. Elevated levels of several adipocytokines, such as leptin, tumor necrosis factor α, and interleukin 6, have been found to be associated with different indices of increased adipose tissue (eg, body mass, body fat mass, fasting insulin, calculated insulin resistance) [2]. In contrast, adiponectin is negatively regulated in obesity, diabetes, and cardiovascular disease [2]. To date, much attention has been focused on the role of leptin in the regulation of body mass and energy expenditure [3,4]. Adiponectin has been implicated in the regulation of energy homeostasis in combination with leptin [5].

A number of different hormones, such as insulin, catecholamines, and cortisol [6], may affect adiponectin levels. It has been suggested that insulin, catecholamines, and cortisol suppress adiponectin gene expression [6]. These hormones are known to be immediately altered during intense exercise [7], whereas insulin, cortisol, and growth hormone have been reported to affect leptin as a result of short-duration exercise [8,9]. Accordingly, the evaluation of different adipocytokines and stress hormones during prolonged exercise training is of interest because of their implications for general adaptive mechanisms. Prolonged exercise training that has been carried out for several months represents a physical stress condition in which different hormonal responses are apparently linked to changes in physical performance. Despite the importance of insulin and growth hormone for maintaining the normal physiological function of many body tissues, there are only limited data on their responses to the prolonged exercise training of highly

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0026-0495/$ – see front matter © 2005 Elsevier Inc. All rights reserved.
doi:10.1016/j.metabol.2005.06.020

trained athletes. Furthermore, to our knowledge, there are insufficient data about the responses of specific adipocytokines to prolonged exercise training and the interrelationship of these adipocytokines and different stress hormones during such training period. Therefore, the purpose of the present study was to investigate the resting and short-duration exercise-induced hormone responses of male rowers during rest and after a period of prolonged exercise training. Specifically, this study examined whether adiponectin, leptin, insulin, growth hormone, testosterone, and cortisol concentrations during rest and their responses to maximal 2000-m sculling exercise changed in response to a 24-week preparatory period in male rowers with different performance levels.

2. Materials and methods

2.1. Subjects

Eleven elite male rowers, members, and candidates for the Estonian National Team, preparing for 2004 Olympic Games participated in this study. The subjects were not taking any drugs or medication and had no history of any endocrine disorders before or during this study. The rowers' mean age was 20.2 ± 2.9 years, and they were in good mental and physical state. None of them had any family history of diabetes or obesity. The rowers were fully familiarized with the procedures before having to provide their written informed consent to participate in the experiment as approved by the Medical Ethics Committee of the University of Tartu. At the end of the study, 6 rowers were selected (SEL) for the national team and represented Estonia at the Olympic Games in double and quadruple sculls, and 5 were not selected (N-SEL). The mean age of SEL rowers was 22.2 ± 2.1 years (range, 19-24 years), and they had been training regularly for the last 8.2 ± 3.1 years. The mean age of N-SEL rowers was 19.8 ± 4.0 years (range, 18-26 years), and they had been training regularly for the last 5.8 ± 4.0 years.

2.2. Experimental design

The subjects were tested on 2 occasions over the 6-month training season (November to April): at the beginning of the preparatory period and 24 weeks later at the beginning of the competition period [10]. In both testing sessions, body composition, maximum oxygen consumption ($\dot{V}O_2max$), and on-water rowing performance were evaluated. Body composition and $\dot{V}O_2max$ were estimated 1 week before rowing performance test. All rowers were in a postabsorptive condition, having eaten a standardized light breakfast about 2 hours before the start of the on-water sculling session [11,12]. Maximal 2000-m single sculling was performed on water on a nonwindy day at an air temperature of approximately 10°C to 12°C and humidity of 40% to 45%. Performance testing sessions were carried out at the same time (ie, between 10:00 AM and 12:00 PM), and testing time was identical for each subject across the tests [4]. Energy intake was not measured in this study [3,4]. However, all the athletes were instructed by an experienced dietician during the whole study period, and their daily food intake consisted of a high-carbohydrate diet with the composition remaining stable [3,4]. The estimation of training volume (hours per week) was obtained after the calculation from the individual training diaries kept by the rowers. The main aim of training during the preparatory period was the development of basic strength endurance [10,13]. The training regimen of rowers was typical for this period of year and consisted of high-volume, low-intensity strength training aimed to improve strength endurance and extensive endurance training aimed to improve basic endurance [10,13]. The training regimen remained the same for each week of the study [4]. During the 24-week preparatory period, on-water rowing training was mainly performed on single sculls, and the second testing session was also considered as one selection criteria for double and quadruple sculls.

2.3. Body composition and $\dot{V}O_2max$ assessment

The height (Martin metal anthropometer) and body mass (A&D Instruments, Oxfordshire, UK) of the participants were measured to the nearest 0.1 cm and 0.05 kg, respectively. Body composition was measured by the dual-energy x-ray absorptiometry using the DPX-IQ densitometer (Lunar, Madison, WI) and analyzed for fat mass (FM) and fat-free mass (FFM). A progressive test to exhaustion was performed on a rowing ergometer (Concept II, Morrisville, VT) to determine $\dot{V}O_2max$ and aerobic power (Pa_{max}) values. Oxygen consumption and carbon dioxide production were continuously measured during the test using a portable open-circuit system (MetaMax I, Cortex, Germany). The analyzer was calibrated before the test with the gases of known concentration. Athletes performed an initial work rate of 150 W with increments of 50 W every 3 minutes until fatigue.

2.4. Blood analysis

A 10-mL blood sample was obtained from an anticubital vein with the participant in the upright position. Blood samples were taken before, immediately after the sculling exercise, and 30 minutes postexercise [4,14]. Similar to other recent studies, no control trial was conducted as diurnal changes of measured hormones were considered not to occur during this short period [4,15]. The plasma was separated and frozen at −20°C for later analysis. Adiponectin was assessed by using a commercially available radioimmunoassay (RIA) kit (Cat. No. HADP-61HK, Linco Research, St. Charles, MO). The intra- and inter-assay coefficient of variation (CV) was less than 7.6%. Leptin was also determined by the radioimmunoassay kit (Mediagnost, Tübingen, Germany). This assay has intra- and inter-assay CVs of less than 5%. Insulin was determined on Immunolite 2000 (DPC, Los Angeles, CA). The intra- and inter-assay CVs for insulin

Table 1
Mean ± SD characteristics of the rowers (n = 11)

Measurements	Baseline	24 wk
Height (cm)	193.1 ± 5.2	193.3 ± 5.2
Weight (kg)	91.6 ± 5.8	92.2 ± 6.3
Body fat (%)	10.2 ± 2.4	9.3 ± 1.6*
FM (kg)	8.9 ± 2.4	8.0 ± 1.5
FFM (kg)	82.8 ± 4.8	84.2 ± 5.9*
$\dot{V}O_2$max (L · min^{-1})	6.2 ± 0.5	6.4 ± 0.6*
Pa_{max} (W)	442.8 ± 40.5	465.9 ± 26.2*
2000-m sculling (s)	439.9 ± 8.9	442.1 ± 9.7

* $P < .05$, significantly different from baseline.

were 4.5% and 12.2%, respectively, at an insulin concentration of 6.6 μL · U · mL^{-1}. Cortisol, testosterone, and growth hormone were also analyzed on Immunolite 2000 (DPC). The inter- and intra-assay CVs were less than 5%. All samples were run on the same assay. Aliquots of the whole blood were also analyzed in quadruplicate for hematocrit at 12 000 rpm for 5 minutes and for hemoglobin using a Lange (Berlin, Germany) microanalyzer. Postexercise changes in plasma volume were calculated by using the formula of Dill and Costill [16], and reported hormone values have been corrected for plasma volume changes.

2.5. Statistical analysis

Means and SDs were determined. Friedman analyses of variance by ranks were used to examine changes, as the row data and their logarithmic transformations were not normally distributed. The Wilcoxon matched-pairs signed rank test was used where post hoc analysis was relevant. The Wilcoxon matched-pairs signed-ranks test was also used to assess the differences between the measured variables in SEL and N-SEL groups. Kendall rank correlation coefficients were used to evaluate associations among different variables of interest. The level of significance was set at $P < .05$.

3. Results

3.1. Subject characteristics

Mean weekly training volume increased significantly ($P < .05$) during the 24-week training period (by 23.4%; from 12.8 ± 0.4 to 16.7 ± 0.5 h · wk^{-1}). The whole group of rowers was relatively young and tall (Table 1). During the training period, no significant changes were observed in body mass. By the end of the preparatory period, significant decrease in body fat percentage was reflected by a significant increase in FFM, whereas FM remained relatively unchanged ($P > .05$). It appeared that SEL rowers had significantly higher body mass at the beginning of the study, whereas no differences in body mass were observed 24 weeks later (Table 2). In the whole group of rowers, the 24-week preparatory period significantly increased $\dot{V}O_2$max and Pa_{max} values, whereas 2000-m performance time on single sculls remained relatively unchanged. $\dot{V}O_2$max and Pa_{max} values showed no change and a significant increase by the end of the preparatory period in SEL and N-SEL rowers, respectively. SEL rowers had a significantly higher Pa_{max} and 2000-m sculling performance values at baseline and 24 weeks later compared with N-SEL rowers.

3.2. Hormones

No differences ($P > .05$) were observed in any measured resting hormonal values between SEL and N-SEL rowers at the beginning of study. In the whole group of rowers, adiponectin did not change as a result of maximal 2000-m rowing on single sculls before and after the 24-week training period (Fig. 1). No significant responses to maximal 2000-m rowing were observed in SEL and N-SEL rowers before the training period (Figs. 2 and 3). Similarly, no responses were observed 24 weeks later in SEL rowers, whereas a significant decrease was observed in N-SEL rowers. Adiponectin was significantly higher immediately after the sculling test at the end of the preparatory period when compared with the baseline in SEL rowers. Leptin decreased significantly after the first 30 minutes of recovery compared with the immediate postexercise value at baseline only in the whole group of rowers. Leptin was significantly lower after the first 30 minutes of recovery after 24 weeks of training compared with the corresponding baseline value in N-SEL rowers. After the 24-week training period, resting insulin was significantly lower in the whole group of rowers. The pattern of insulin response was similar, demonstrating an increase and a decrease below the resting conditions immediately after the exercise and after the first

Table 2
Mean ± SD characteristics of the rowers selected (SEL; n = 6) and not selected (N-SEL; n = 5) for the national team

Measurements	Baseline SEL	Baseline N-SEL	24-wk SEL	24-wk N-SEL
Height (cm)	192.0 ± 6.3	194.8 ± 4.1	192.3 ± 6.1	194.8 ± 4.1
Weight (kg)	93.5 ± 7.1	89.6 ± 4.0*	93.5 ± 7.1	90.6 ± 5.4
Body fat (%)	10.8 ± 3.0	9.4 ± 1.3	9.4 ± 2.0**	9.1 ± 1.2
FM (kg)	9.6 ± 3.1	8.0 ± 1.2	8.2 ± 1.8**	7.7 ± 1.0
FMM (kg)	83.9 ± 5.8	81.9 ± 3.3	85.3 ± 6.8**	82.9 ± 5.0
$\dot{V}O_2$max (L · min^{-1})	6.4 ± 0.4	6.0 ± 0.5*	6.5 ± 0.5	6.3 ± 0.7**
Pa_{max} (W)	461.8 ± 33.9	420.0 ± 38.4*	478.7 ± 24.4	450.6 ± 20.9*
2000-m sculling (s)	434.5 ± 8.0	446.4 ± 4.7*	435.9 ± 9.2	449.5 ± 1.6*

* $P < .05$, significantly different from SEL.
** $P < .05$, significantly different from baseline.

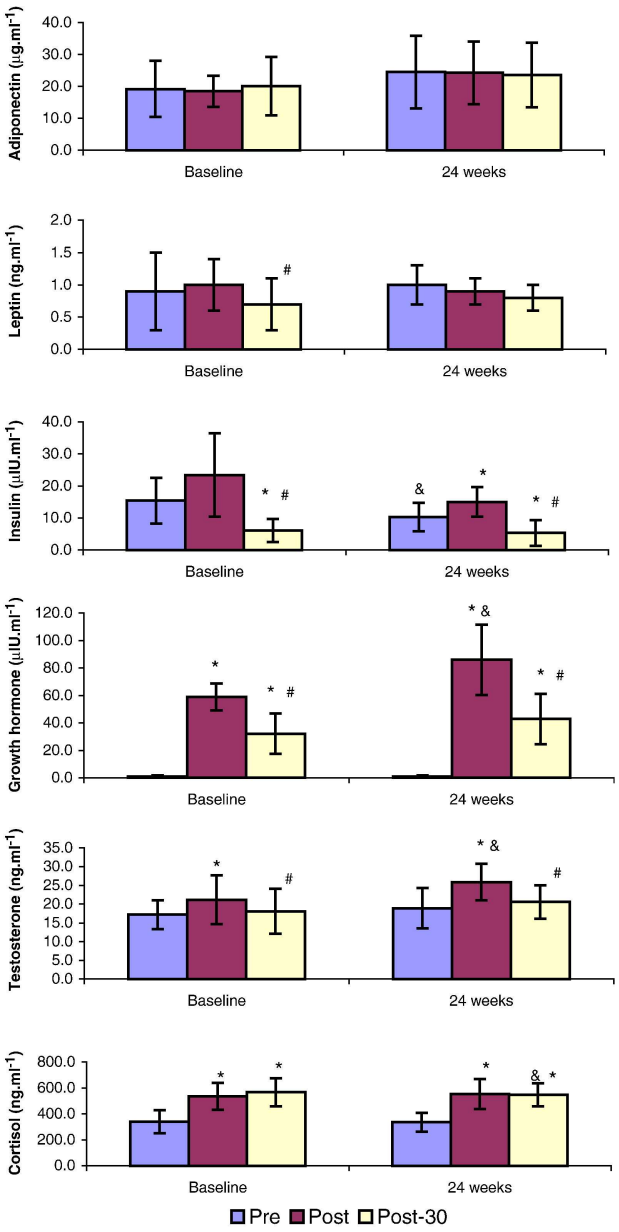

Fig. 1. Mean ± SD hormone responses to 2000-m single sculling in rowers (n = 11). *$P < .05$, significantly different from Pre; #$P < .05$, significantly different from Post; &$P < .05$, significantly different from baseline. Pre indicates before exercise; Post, immediately after exercise; Post-30, after the first 30 minutes postexercise.

30 minutes of recovery, respectively, in the whole group of rowers as well as in SEL rowers. Immediate postexercise increase in insulin was significantly lower after the 24-week training period compared with baseline in SEL rowers. Insulin response was also similar before and after the training period in N-SEL rowers, demonstrating a significant decrease below the resting value after the first 30 minutes of recovery. The pattern of growth hormone responses to maximal sculling was not different between both testing times showing a significant increase immediately after the exercise, which decreased significantly during the first 30 minutes of recovery but remained significantly elevated compared with the resting values in the whole group of rowers and in both groups separately. A similar pattern of testosterone response was observed in the whole group of rowers and in both groups separately at the end of the preparatory period and at baseline in SEL rowers, whereas N-SEL rowers demonstrated no significant changes in testosterone value at baseline measurement. Cortisol increased significantly immediately after the exercise and remained significantly elevated after the first 30 minutes of recovery in both measurement sessions in the

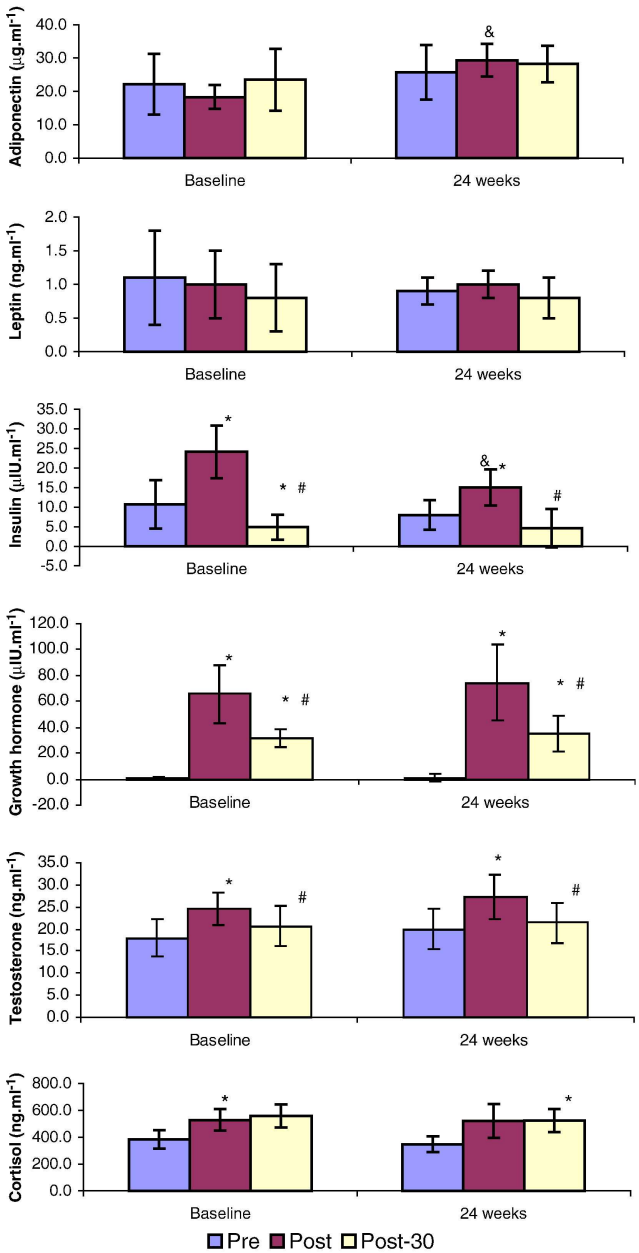

Fig. 2. Mean ± SD hormone responses to 2000-m single sculling in the rowers selected (n = 6) for the national team. *$P < .05$, significantly different from Pre; #$P < .05$, significantly different from Post; &$P < .05$, significantly different from baseline.

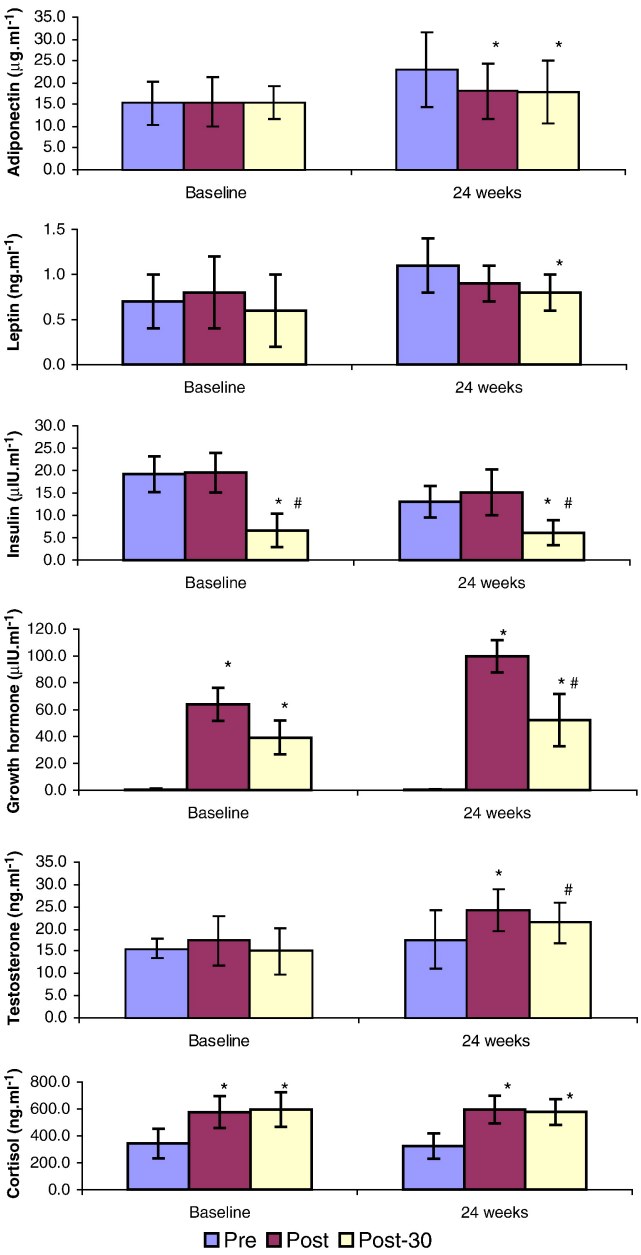

Fig. 3. Mean ± SD hormone responses to 2000-m single sculling in the rowers not selected (n = 5) for national team. *$P < .05$, significantly different from Pre; #$P < .05$, significantly different from Post; &$P < .05$, significantly different from baseline.

whole group of rowers as well as in SEL and N-SEL rowers separately.

3.3. Correlations

Resting adiponectin and insulin were significantly related ($r = -0.327$, $P < .05$). In addition, resting insulin was significantly related to testosterone ($r = -0.320$, $P < .05$). Resting testosterone ($r = 0.415$, $P < .05$) and cortisol ($r = 0.517$, $P < .05$) were significantly related to $\dot{V}O_2$max, whereas resting insulin ($r = -0.399$, $P < .05$) and growth hormone ($r = -0.335$, $P < .05$) were significantly correlated with the mean weekly training time. Adiponectin demonstrated no correlation with any measured body composition parameters ($r < 0.265$, $P > .05$). Whereas leptin was related only to FM ($r = 0.342$, $P < .05$), Pa_{max} was related ($P < .05$) to the mean weekly training time ($r = 0.350$) and maximal 2000-m single sculling performance time ($r = -0.351$). No other relationships were observed between any measured dependent variables ($r < 0.290$, $P > .05$).

4. Discussion

The focus of recent short-duration exercise [1,7,17] and exercise intervention [2,18] studies has been the potential role of adiponectin in energy homeostasis. Energy intake and energy expenditure are of the utmost importance for the competitive athlete in relation to maintaining energy stores with respect to heavy training stress [19]. Training in international rowing is mainly characterized by low-intensity high-volume training sessions during the preparatory period [4,10,13]. Therefore, the responses of resting [19] and exercise-induced [4] adiponectin concentrations to an increase in training volume were examined in the present study, and the possibility to use these adiponectin values as markers of heavy training stress in elite rowers were also studied. Similar to the training volume reports for other international level rowers [10,13], we observed an increase in training volume from the beginning to the end of the preparatory period inducing an increase in energy expenditure. To our knowledge, this is the first study to have investigated the potential role of adiponectin in response to prolonged heavy training stress in highly trained athletes selected and not selected for the national team. The main findings of the present study were the following: (1) resting adiponectin concentration is not sensitive to a prolonged low-intensity heavy training stress in highly trained male rowers; (2) an increase in training stress is accompanied by higher postexercise adiponectin values only in rowers with higher performance level; and (3) an increase in training stress is accompanied by a significant decrease in postexercise adiponectin values in rowers with lower performance level.

Our study is the first to demonstrate that increased training stress over a preparatory period for rowers [10,13] modifies adiponectin response to a bout of a short-duration 2000-m maximal sculling exercise without having an effect on the resting adiponectin levels. It has been suggested that the changes in adiponectin may be linked with the changes in energy homeostasis [2]. In addition to this, immediate effects of exercise on circulating adiponectin concentrations have been reported [7,17]. The current study was undertaken to investigate the possible adiponectin and exercise interaction during prolonged exercise training at a relatively high daily energy expenditure [4,10,13] in a specific group of highly trained athletes. The fact that no changes have been found in resting adiponectin despite an increase in training volume and lack of a relationship between these

parameters suggests that resting adiponectin concentration is not regulated in a dose-response manner (eg, References 4,19) in highly trained rowers. As blood adiponectin levels depend on adiponectin synthesis in adipose tissue and its clearance in plasma, the lack of alterations could not rule out the possibility that the expression of adiponectin gene in adipose tissue is not affected in response to short-duration exercise. However, according to the results of the present study, short-duration maximal exercise-induced adiponectin concentration may be indicative of the amount of physical stress and consequently on the condition of the athletes' organs in male rowers. It has to be considered that all rowers had trained using a similar training program with no specific difference in training volume and intensity. Significantly lowered levels of adiponectin concentration as a result of 2000-m maximal sculling at the end of the 24-week preparatory period in N-SEL rowers may be a sign of inadequate recovery and inadequate performance level of these athletes. In contrast, SEL rowers demonstrated higher exercise-induced adiponectin values compared with the corresponding adiponectin values obtained before the 24-week preparatory period. This may suggest that these athletes adequately recovered from previous trainings. These findings together suggest that the maximal exercise-induced changes in adiponectin during prolonged training depend on the previous physical condition and/or the amount of the physical stress of these athletes.

The inadequate recovery of N-SEL rowers at the end of the 24-week preparatory period was also confirmed by a significant decrease in leptin concentration after the first 30 minutes of recovery period (see Fig. 3). This is in accordance with our previous study with male rowers, in which a 3-week heavy training period caused a significant postexercise decrease in leptin levels after the maximal 2000-m rowing ergometer test [4]. We believed that at the end of the 3-week heavy training period, the athletes were in an early overtraining syndrome state, as the decreased metabolic rate during the 2-week tapering period was accompanied by no changes in leptin concentration after the 2000-m rowing ergometer test [4]. It could be speculated that similar to our previous study [4], a 24-week preparatory period in N-SEL rowers led to the disrupted metabolic homeostasis [20], such that energy expenditure during the maximal 2000-m sculling exercise was already enough to produce a significant reduction in adiponectin and leptin levels.

The results of the present study also demonstrate a training effect on resting and short-duration exercise-induced insulin concentration in rowers with different performance levels. These responses are of particular interest as it has been suggested that insulin may provide a mechanism by which adipose tissue detects changes in overall energy balance, and, in turn, up-regulates or down-regulates *ob* gene expression accordingly [21]. This regulation occurs through endocrine feedback loop, in which the pancreatic beta cell–derived hormone insulin and the adipocyte-derived hormone leptin signal the status of body energy stores to the hypothalamus [22]. Specifically, leptin and insulin depress the activity of excitatory neurons in lateral hypothalamus and affect energy expenditure, body mass control, and sympathetic activity [23]. It is interesting to note that adiponectin was significantly correlated with insulin concentration in highly trained male rowers ($r = -0.327$, $P < .05$). There is also evidence that insulin is an inhibitor of adiponectin gene expression [24]. When taking into account that insulin was related to the mean training volume ($r = -0.399$, $P < .05$), it can be suggested that the effects of training on insulin could also mediate the adiponectin response to short-duration exercise. However, the proposed mechanism linking adiponectin with insulin sensitivity is unclear, and further studies are warranted. Taken together, studying the effects of exercise on adiponectin and leptin may give the advantage of knowing the amount of stress affecting the organism.

To date, the published longitudinal exercise training studies with adiponectin suggest that exercise alone does not alter its concentration [2,18]. In one study, previously sedentary subjects trained for 6 months using different forms of aerobic exercises and did not display significant changes in adiponectin concentrations from pre- to post-training [2]. It was therefore suggested that adiponectin does not contribute to exercise improvements in insulin sensitivity [2]. However, the subjects in these longitudinal studies were sedentary and relatively overweight [2,18]. To our knowledge, this is the first study on the possible adiponectin responses to exercise training where subjects are highly trained athletes who had been training regularly with high training volume for several years and present relatively high muscle mass. Whereas resting adiponectin demonstrated only a trend ($P > .05$) to increase as a result of a 24-week training period, a significantly higher postexercise adiponectin value was observed in SEL rowers compared with the corresponding baseline value. No training effect on postexercise adiponectin value was observed in N-SEL rowers (see Figs. 2 and 3). It was therefore speculated that similar to leptin concentration [14], the total amount of physical stress (ie, years of training) can have an influence on adiponectin concentration. Secondly, the amount of muscle tissue used during a short-duration exercise bout can also affect adiponectin response. It is well known that about 70% of the whole body muscle mass is involved in rowing. In accordance with this, Berg et al [25] demonstrated that adiponectin therapy in mice resulted in decreased hepatic glyconeogenesis and muscle triglyceride count, suggesting that adiponectin carries signals between adipose and muscle tissues. It is also established that adiponectin can activate adenosine monophosphate–activated protein kinase and increase fatty acid oxidation in skeletal muscle [26].

The increase of growth hormone, testosterone, and cortisol levels after a short bout of maximal exercise in athletes is often observed and described in the literature [7,20,27]. Taking into account that immediate postexercise

increases in growth hormone and testosterone values were significantly higher after the 24-week heavy training period (see Fig. 1) and that immediate postexercise increases in these anabolic hormones were also higher after the 24-week heavy training period in N-SEL rowers compared with the corresponding value obtained before training period (see Figs. 2 and 3), it can be suggested that there should be a wide reserve to increase the stress hormone responses to exercise. Furthermore, a certain performance capacity has to be achieved to cause correspondingly heavy increase in these hormone values. This is also supported by a significant relationship between $\dot{V}O_2max$ and resting testosterone (r = 0.415) and cortisol (r = 0.517) values in rowers. However, the mechanism of these immediate hormonal changes due to maximal exercise is not very clear. It has been suggested that decreased liver perfusion, central stimulation of the hypothalamic-pituitary axis, and an increased perfusion of the hypothalamus, pituitary gland, testicles, or adrenals could be responsible for these immediate hormonal changes [27].

In conclusion, our results extend data about the adiponectin response in athletes after a heavy training period. It appears that resting adiponectin does not change as a result of the heavy training period in highly trained rowers with different performance levels. In contrast, training modifies adiponectin response to an immediate bout of maximal exercise depending on the performance level of athletes. Specifically, heavy training period caused significantly higher postexercise values in rowers with better performance capacity, whereas significant decreases in postexercise adiponectin and leptin values were observed in rowers with lower performance capacity. This was thought to reflect the inadequate recovery capacities of these rowers.

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