Plasma Ghrelin Is Altered After Maximal Exercise in Elite Male Rowers

JAAK JÜRIMÄE,[1] TOIVO JÜRIMÄE, AND PRIIT PURGE

Institute of Sport Pedagogy and Coaching Sciences, Centre of Behavioral and Health Sciences, University of Tartu, Tartu, Estonia

The aim of the present investigation was to investigate plasma ghrelin response to acute maximal exercise in elite male rowers. Eight elite male rowers performed a maximal 6000-m rowing ergometer test (mean performance time: 19 mins 52 secs; 1192.1 ± 16.4 secs), and venous blood samples were obtained before, immediately after, and after 30 mins of recovery. In addition to ghrelin concentration, leptin, insulin, growth hormone, insulin-like growth factor-1 (IGF-1), testosterone, cortisol, and glucose values were measured. Ghrelin was significantly increased immediately after the exercise (+24.4%; $P < 0.05$) and was not significantly different than baseline after 30 mins of recovery. Leptin was significantly decreased immediately after the exercise (−15.8%; $P < 0.05$) and remained significantly decreased after the first 30 mins of recovery. No changes occurred in insulin concentrations. Growth hormone, IGF-1, and testosterone values were significantly increased and decreased to the pre-exercise level immediately after the exercise and after the first 30 mins of recovery, respectively. Cortisol and glucose values were significantly increased immediately after the exercise and remained significantly increased during the first 30 mins of recovery. There were no relationships between plasma ghrelin and other measured blood parameters after the exercise, nor were changes in ghrelin related to changes in other measured blood biochemical values after the exercise. In conclusion, these results suggest that acute negative energy balance induced by specific maximal short-term exercise elicits a metabolic response with opposite changes in ghrelin and leptin concentrations in elite male athletes. Exp Biol Med 232:904–909, 2007

Key words: ghrelin; leptin; acute exercise; elite athletes

Introduction

Ghrelin, a 28–amino acid peptide, is involved in growth hormone secretion and energy homeostasis (1). The principal site of ghrelin synthesis is the stomach (2). Ghrelin transfers information from the stomach to the hypothalamus and influences growth hormone release in response to changes in energy homeostasis (1). Plasma ghrelin levels are reduced in obese individuals (3) and substantially elevated in anorexic subjects (4). Ghrelin is, therefore, another hormone, in addition to leptin, that reflects peripheral nutritional status (5, 6). It has been well established that leptin is an adipocyte-derived hormone that acts directly on the hypothalamus, where it regulates a large number of molecules that are involved in the energy homeostasis (7). In addition, leptin may have a role in the control of growth hormone release (8, 9). Accordingly, peripheral signals, such as ghrelin and leptin, could be influenced by changes in energy balance to opposite directions (1, 5).

Intensive physical exercise is a well-recognized stimulator of plasma growth hormone release that is linear with exercise intensity (1, 10). Information regarding the regulatory effect of ghrelin on growth hormone release as a result of acute physical exercise is limited. To date, the published acute exercise investigations with ghrelin suggest that regardless of intensity, exercise alone does not alter ghrelin concentration in healthy adults (10–15). However, the published acute exercise studies have used exercise protocols in which only the lower extremeties were involved (i.e., running or cycling; Refs. 10–14). Our recent study demonstrated that the increase in plasma ghrelin concentration almost approached significance when the constant load rowing exercise was performed at an intensity slightly above the individual's anaerobic threshold (15). In addition, the only other study that has used high-intensity exercise protocol was that of Kraemer *et al*. (10), in which six well-trained male runners performed a progressively intense intermittent exercise trial on a treadmill for the duration of 27 mins. However, to our knowledge, no studies have yet investigated the effect of short term maximal exercise on plasma ghrelin using an exercise protocol in which all major

This study was supported by the Estonian Science Foundation grant GKKSP 6638.

[1] To whom correspondence should be addressed at Institute of Sport Pedagogy and Coaching Sciences, University of Tartu, 18 Ülikooli Street, Tartu 50090, Estonia. E-mail: jaakj@ut.ee

Received January 8, 2007.
Accepted March 3, 2007.

1535-3702/07/2327-0904$15.00

Table 1. Subject Characteristics ($n = 8$)

Variable	Mean ± SD	Minimum–maximum
Age (yrs)	21.3 ± 2.8	19.0–26.0
Height (cm)	193.7 ± 5.3	185.6–199.6
Body mass (kg)	97.4 ± 7.4	87.0–104.0
Body fat (%)	11.2 ± 2.4	7.2–14.6
FM (kg)	10.2 ± 2.5	6.7–13.8
FFM (kg)	80.7 ± 5.0	73.1–86.1
P_{max} (W)	440.0 ± 39.8	400–530
V_E (l/min)	193.5 ± 11.3	171.0–203.0
Vo_2max (l/min)	6.26 ± 0.56	5.40–7.10
Vo_2max/kg (ml/min/kg)	65.70 ± 3.80	61.60–73.20

muscle groups are involved. This may cause additional energy expenditure that may be needed for plasma ghrelin to be reduced by acute exercise. In support of this finding, a recent study by Christ *et al*. (5) with endurance athletes demonstrated that ghrelin was significantly increased as a result of a prolonged 3-hr aerobic exercise session on a cycle ergometer at the intensity of 50% of maximal aerobic power.

The present study was designed to evaluate the effects of maximal short-term ergometer rowing (6000-m all-out test) on plasma ghrelin in elite male rowers. It was hypothesized that maximal ergometer rowing with higher metabolic strain on the organism (16) would have an impact on plasma ghrelin and leptin concentrations in response to an altered concentration of growth hormone, as elite rowers present relatively large body mass values and all extremeties and trunk are involved in rowing compared with other endurance sport disciplines.

Materials and Methods

Study Design. Eight highly trained male rowers volunteered to participate in the study (Table 1). They all have been medal winners in international world championships either at the junior, under-23 or senior-A level. Measurements took place at the beginning of the preparatory period after a relative rest. The participants had trained, on average, four times per week during the 4 wks preceding the study. The main goal of training during that period was to recover from the previous competition season and to prepare for the coming training season. The mean weekly training volume during this period was about 6 hrs/wk. The rowers were fully familiarized with the procedures before providing their written consent to participate at the experiment, as approved by the Medical Ethics Committtee of the University of Tartu. Each subject completed body composition and incremental rowing ergometer exercise tests. Second measurement consisted of a maximal 6000-m rowing ergometer test to determine ghrelin responses to a relatively short-term exercise protocol in which all major muscle groups are involved (16). Rowers were asked not to particpate in any physical activity in the 24 hrs before each session.

Body Composition Assessment. The height (Martin metal anthropometer) and body mass (A&D Instruments, Abingdon, UK) of the participants were measured to the nearest 0.1 cm and 0.05 kg, respectively. Body composition was measured using dual-energy x-ray absorptiometry. Scans of the whole body were performed on each of the subjects using a Lunar DPX-IQ scanner (Lunar Corp., Madison, WI) and were analyzed for fat (FM) and fat-free (FFM) mass.

Incremental Ergometer Exercise. A progressive test to exhaustion was performed on a rowing ergometer (Concept II, Morrisville, VT) to determine maximal oxygen consumption (Vo_2max; l/min), relative Vo_2max (Vo_2max/kg; ml/min/kg), maximal ventilation (V_E; l/min), and maximal aerobic power (P_{max}; W). Power and stroke frequency were monitored and recorded continuously *via* computer display of the rowing ergometer. Vo_2 consumption and carbon dioxide production were continuously measured during the test using a portable open circuit spirometry system (MetaMax I; Cortex, Leipzig, Germany). The analyzer was calibrated before the test with the gases of known concentration according to manufacturer's guidelines. Athletes performed an initial work rate of 150 W with increments of 50 W every 3 mins until fatigue.

Rowing Performance Test. Maximal 6000-m ergometer performance was assessed in the afternoon on a wind resistance–braked rowing ergometer (Concept II). All subjects were in a postabsorptive condition, having eaten a meal about 2 hrs before the test (13, 15, 17). Our subjects were instructed by an experienced dietician previously, and their daily food intake consisted of a high-carbohydrate diet with the composition remaining stable. The pre-exercise diet and the fluid intake was not measured. During the rowing performance test, athletes were asked to cover a distance of 6000 m in the least time possible. The athletes were fully familiarized with the use of this apparatus. Power and stroke frequency were monitored and recorded continuously *via* computer display of the rowing ergometer (15, 17).

Analytical Methods. A 10-ml blood sample was obtained from an anticubital vein with the participant in the upright position. Blood samples were taken before (PRE), immediately after (POST), and 30 mins after (POST-30) the 6000-m maximal rowing ergometer test (15, 17). Similarly to other recent studies, no control trial was conducted, as diurnal changes of measured hormones were considered not to occur during this short time period (5, 15, 18–20). The plasma was separated and frozen at –20°C for later analysis. Ghrelin was assessed using a commercially available radioimmunoassay (RIA) kit (Linco Research, St. Charles, MO). The sensitivity of the ghrelin kit was 93 pg/ml, and the intra-assay and inter-assay coefficients of variation (CV) were $<10\%$ and $<14.7\%$, respectively. Leptin also was determined by the RIA kit (Mediagnost GmbH, Reutlingen, Germany). This assay has intraassay and interassay CVs of less than 5%. Insulin was determined on Immulite 2000 (DPC, Los Angeles, CA). The intraassay and interassay

CVs for insulin were 4.5% and 12.2%, respectively, at an insulin concentration of 6.6 μl/U/ml. Growth hormone, IGF-1, testosterone, and cortisol also were analyzed on Immulite 2000 (DPC), and the interassay and intraassay CVs were less than 5%. All samples were run on the same assay. Glucose was measured by means of the hexokinase/glucose 6-phosphate-dehydrogenase method using a commercial kit (Boehringer, Mannheim, Germany). Aliquots of the whole blood also were analyzed in quadruplicate for hematocrit at 12,000 rpm for 5 mins and for hemoglobin using a Lange (Berlin, Germany) microanalyzer. Postexercise changes in plasma volume PV were calculated using the formula of Dill and Costill (21).

Statistical Analysis. Means and standard deviations were determined. For continuous variables, the distribution was tested by the Shapiro-Wilk statistical method. 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-ranks test was used where post-hoc analysis was relevant. Kendall rank correlation coefficients were used to evaluate associations among different variables of interest. An alpha level of $P < 0.05$ was considered significant. All statistics were performed with SPSS (version 11.0; Chicago, IL).

Results

The 6000-m rowing ergometer mean performance time was 19 mins 52 secs (1192.1 ± 16.4 secs), with a mean power of 357.3 ± 14.3 W. The test was performed at 81% of Vo_2max, with an estimated energy expenditure of approximately 400 kcal during the test. Plasma volume was not significantly changed immediately after exercise (1.1% ± 2.6%) or after the first 30 mins of recovery (0.7% ± 1.3%). No relationships were observed between basal ghrelin concentration and the measured body composition, physical performance, or blood biochemical data ($r < 0.47$; $P > 0.05$). Similarly, no relationships were observed between basal leptin concentration and the measured body composition, physical performance, or blood biochemical data ($r < 0.49$; $P > 0.05$).

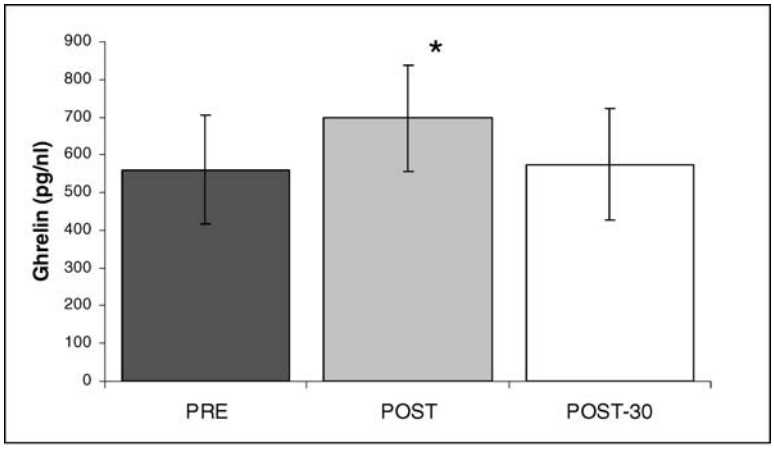
Ghrelin was significantly increased immediately after the exercise (+24.4%; $P < 0.05$) and was decreased during the first 30 mins of recovery (–17.5%; $P = 0.093$) compared with the postexercise level (Fig. 1). Ghrelin concentration was increased in six of eight rowers. There were no relationships between plasma ghrelin and other measured blood parameters after the exercise, nor were changes in ghrelin related to changes in other measured blood biochemical values after the exercise.

Plasma leptin was significantly decreased immediately after the exercise (–15.8%; $P < 0.05$) and remained significantly decreased after the first 30 mins of recovery (Fig. 1). Absolute changes in leptin concentration (PRE vs. POST) were significantly related to the absolute changes in growth hormone values ($r = –0.78$; $P = 0.042$). No changes occurred in insulin concentrations with exercise (Table 2). Growth hormone, IGF-1, and testosterone values were significantly increased and then decreased to the pre-exercise level immediately after the exercise and after the first 30 mins of recovery, respectively. Cortisol and glucose values were significantly increased immediately after the exercise and remained significantly increased during the first 30 mins of recovery.

Discussion

The present study demonstrated a significant effects of maximal short-term rowing ergometer exercise on ghrelin and leptin concentrations in elite male rowers. In contrast, previous studies that have investigated the effects of short-term exercise (up to 45 mins) on ghrelin concentration have demonstrated no changes in plasma ghrelin concentrations after acute submaximal rowing (15), running (12, 14), or cycling (11, 13), and maximal running (10) exercise in healthy subjects. In our recent study, however, plasma ghrelin concentrations almost approached significance after the first 30 mins of recovery when the rowing exercise was performed at an intensity slightly above the individual's anaerobic threshold (15). It could be suggested that the absence of any significant change in plasma ghrelin found in the previous short-term exercise studies may be due to the limited negative energy balance during these exercise bouts (22), study population (5), and/or the exercise protocol used (16). Plasma ghrelin responses may differ from previous short-term exercise studies that included more sedentary subjects and/or exercise protocols using fewer muscle groups (10–14) compared with our study design, which used elite athletes capable of both greater intensity and use of more muscle groups during ergometer rowing (17). In addition, the sustained anaerobic nature of the rowing exercise in our study resulted in a greater energy expenditure and may have contributed to the significant increases in ghrelin concentration immediately after the acute exercise (16). In support of our findings, a 3-hr aerobic exercise at 50% of maximal aerobic power caused a negative energy balance that resulted in a significant increase in ghrelin concentration (5). In addition, a 1-hr strenous exercise in rats caused a significant increase in plasma ghrelin concentration (23). Previous short-term rowing exercise studies also have demonstrated significant reductions in leptin concentration after the exercise (16, 19). Accordingly, the results of the present investigation demonstrate that peripheral markers of negative energy balance, such as ghrelin and leptin, could be regarded as signals for metabolic reaction to the energy needs of the organism during physical exercise.

The main finding of the present investigation was a mean ≈24% ($P < 0.05$) increase in plasma ghrelin concentration immediately after the maximal 6000-m rowing ergometer test (mean performance time: 19 mins



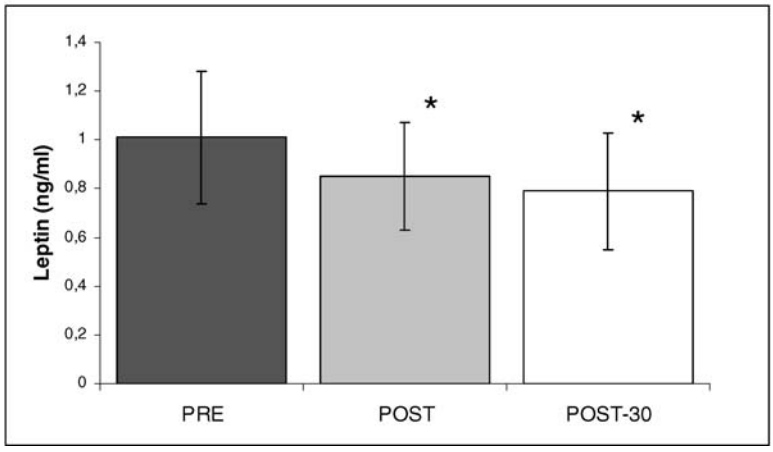


Figure 1. Mean ± SD values in ghrelin and leptin concentrations during a maximal 6000-m rowing ergometer test in elite male rowers ($n = 8$). *Significantly different from PRE; $P < 0.05$.

52 secs) in elite male rowers. In addition to ghrelin, significant increases in growth hormone values after the maximal exercise were observed (Table 2). The neurotransmitters participating in the control of exercise-induced growth hormone release remain uncertain, but the final common pathway is assumed to involve either the stimulation of hypothalamic growth hormone–releasing hormone and/or inhibition in the release of somatostatin (1, 11). In addition, ghrelin has been demonstrated to strongly stimulate growth hormone release during conditions of acute or chronic perturbations in energy balance, such as the postprandial phase, obesity, and/or anorexia nervosa, but not during acute energy expenditure (1, 11). The findings of the present investigation demonstrated that

Table 2. Mean ± SD Blood Biochemical Parameters During a Maximal 6000-m Rowing Ergometer Test in Elite Male Rowers ($n = 8$)

Variable	PRE	POST	POST-30
Insulin (μl/U/ml)	13.1 ± 10.7	10.4 ± 5.4	17.8 ± 10.5
Growth hormone (μl/U/ml)	0.9 ± 0.6	72.1 ± 9.5*	31.9 ± 13.6*,**
IGF-1 (μg/l)	237.7 ± 83.9	268.3 ± 91.7*	246.5 ± 79.4**
Testosterone (ng/ml)	20.3 ± 5.7	33.2 ± 5.9*	23.7 ± 4.0**
Cortisol (ng/ml)	356.7 ± 112.2	516.2 ± 125.3*	589.7 ± 80.5*
Glucose (m*M*)	4.2 ± 0.6	7.3 ± 2.6*	6.0 ± 1.4*

* Significantly different from PRE; $P < 0.05$; ** Significantly different from POST; $P < 0.05$.

exercise-induced increase in ghrelin concentration was not related to growth hormone values after maximal ergometer rowing in elite male rowers, which is in agreement with the results of previous studies (11, 13–15). However, ghrelin concentration was significantly increased in six of eight subjects, suggesting that circulating ghrelin may still play an important role in energy expenditure during relatively short-term acute exercise.

Growth hormone has been demonstrated to respond to different exercise modalities in subjects with different performance levels (10, 11, 13, 16, 17, 19), and it has been suggested that exercise intensity predicts growth hormone secretion in a linear dose-response manner (24). The exercise-induced increases in growth hormone as well as in IGF-1 and cortisol concentrations were higher compared with other acute exercise studies (5, 10, 11). If we assume that the energy balance drives the hormonal response, it is conceivable that the measured hormone profile is related mainly to the exercise-induced energy expenditure (5). This further demonstrates that the energy expenditure during this exercise session was higher compared with previous studies investigating ghrelin response to acute exercise session (5, 10, 11). In addition, resting plasma ghrelin concentrations in our elite male rowers (560.1 ± 144.1 pg/ml) were markedly lower compared with healthy subjects (10). This may have influenced the exercise-induced rise in ghrelin concentration.

The resting and postexercise values of leptin, growth hormone, IGF-1, insulin, and glucose concentrations were similar to those in our previously published investigations with highly trained rowers (15, 16, 18). Another finding similar to those in other recently published investigations (15, 25) was that ghrelin concentration was not related to leptin in elite male rowers. However, Shintani *et al.* (7) postulated that ghrelin and leptin share the hypothalamic NPY/Y1 receptor pathway, whereas Brüning *et al.* (26) suggested that leptin and insulin depress the activity of excitatory neurons in lateral hypothalamus and affect energy expenditure, body mass control, and sympathetic activity. In addition, leptin may have a role in the control of growth hormone release (8, 9). In accordance with this, changes in leptin concentration were significantly related to changes in growth hormone values as a result of maximal ergometer exercise in elite male rowers ($r = -0.78$; $P = 0.042$). Together, these findings suggest that ghrelin and leptin can be considered as an interface between energy intake and expenditure, the main function being to regulate metabolism together in order to respond adequately to the energy needs of the organism (5).

This study has some limitations. A nonexercise control trial was not performed. In addition, this study examined ghrelin responses to maximal rowing exercise in postprandial conditions in rowers. It is possible that a nutrition-induced decrease in ghrelin concentration may have influenced a possible exercise-induced effect. Therefore, it may be difficult to separate the effects of the exercise itself and the pre-exercise snack. Consequently, the current results have to be interpreted with caution. However, previous research has demonstrated that ghrelin concentration reaches nadir approximately 1 hr after meal, and changes in ghrelin concentration are smaller after meal than after overnight fasting in healthy subjects (27). In addition, it is possible that a fasting-induced elevation of ghrelin may mask an exercise-induced effect (10). To date, the influence of acute exercise on ghrelin concentration has been studied in fasting (10) and postprandial (5, 11, 13, 15) conditions. Since the exact effect of either caloric intake or restriction on acute exercise-induced responses of ghrelin concentrations is currently not known, we used a condition of 2 hrs postprandial in the present investigation; a condition similar to the study of Kallio *et al.* (13). Clearly, this issue warrants further investigation.

In conclusion, this study demonstrates that acute negative energy balance induced by maximal short-term exercise elicits a metabolic response with changes in ghrelin and leptin concentrations to opposite directions.

1. Popovic V, Duntas LH. Leptin TRH and ghrelin: influence on energy homeostasis at rest and during exercise. Horm Metab Res 37:533–537, 2005.
2. Kojima M, Hosoda H, Date Y, Nakazato M, Matsuo M, Kangawa K. Ghrelin is a growth-hormone-releasing acylated peptide from the stomach. Nature 402:656–660, 1999.
3. Tschop M, Wawarta R, Riepl RL, Friedrich S, Biedlingmaier M, Landgraf R, Folwaczny C. Post-prandial decrease of circulating human ghrelin levels. J Endocrinol Invest 24:RC19–RC21, 2001.
4. Haqq AM, Farooqi IS, O'Rahilly S, Stadler DD, Rosenfeld RG, Pratt KL, LaFranchi SH, Purnell JQ. Serum ghrelin levels are inversely correlated with body mass index, age, and insulin concentrations in normal children and are markedly increased in Prader-Willi Syndrome. J Clin Endocrinol Metab 88:174–178, 2003.
5. Christ ER, Zehnder M, Boesch C, Trepp R, Mullis P, Diem P, Decombaz J. The effect of increased lipid intake on hormonal responses during aerobic exercise in endurance-trained men. Eur J Endocrinol 154:397–403, 2006.
6. Whatmore AJ, Hall CM, Jones J, Westwood M, Clayton PE. Ghrelin concentrations in healthy children and adolescents. Clin Endocrinol 59: 649–654, 2003.
7. Shintani M, Ogawa Y, Ebihara K, Aizawa-Abe M, Miyanaga F, Takaya K, Hayashi T, Inoue G, Hosoda K, Kojima M, Kangawa K, Nakao K. Ghrelin, an endogeneous growth hormone secretagogue, is a novel orexigenic peptide that antagonizes leptin action through the activation of hypothalamic neuropeptide Y/Y1 receptor pathway. Diabetes 50: 227–232, 2001.
8. Zieba DA, Amstalden M, Morton S, Gallino JL, Edwards JF, Harms PG, Williams GL. Effect of leptin on basal and GHRH-stimulated GH secretion from the bovine adenohypophysis are dependent upon nutritional status. J Endocrinol 178:83–89, 2003.
9. Luque RM, Huang ZH, Shah B, Mazzone T, Kineman RD. Effects of leptin replacement on hypothalamic-pituitary growth hormone (GH) – axis function and circulating ghrelin levels in ob/ob mice. Am J Physiol Endocrinol Metab 292:E891–E899, 2007.
10. Kraemer RR, Durand RJ, Acevedo EO, Johnson LG, Kraemer GR, Hebert EP, Castracane VD. Rigorous running increases growth hormone and insulin-like growth factor-1 without altering ghrelin. Exp Biol Med 229:240–246, 2004.

11. Dall R, Kanaley J, Hansen TK, Moller N, Christiansen JS, Hosoda H, Kangawa K, Jorgensen OL. Plasma ghrelin levels during exercise in healthy subjects and in growth hormone-deficit patients. Eur J Endocrinol 147:65–70, 2002.
12. Jorgensen JO, Krag M, Kanaley J, Moller J, Hansen TK, Moller N, Christiansen JS, Orskov H. Exercise, hormones, and body temperature. Regulation and action of GH during exercise. J Endocrinol Invest 26: 838–842, 2003.
13. Kallio J, Pesonen U, Karvonen MK, Kojima M, Hosoda H, Kangawa K, Koulu M. Enhanced exercise-induced GH secretion in subjects with Pro7 substitution in the Prepro-NPY. J Clin Endocrinol Metab 86: 5348–5352, 2001.
14. Schmidt A, Maier C, Schaller G, Nowotny P, Bayerle-Eder M, Buranyi B, Luger A, Wolzt M. Acute exercise has no effect on ghrelin plasma concentrations. Horm Metab Res 36:174–177, 2004.
15. Jürimäe J, Hofmann P, Jürimäe T, Palm R, Mäestu J, Purge P, Sudi K, Rom K, von Duvillard SP. Plasma ghrelin responses to acute sculling exercises in elite male rowers. Eur J Appl Physiol 99:467–474, 2007.
16. Jürimäe J, Jürimäe T. Leptin responses to a short-term exercise in college level male rowers. Br J Sports Med 39:6–9, 2005.
17. Jürimäe J, Purge P, Jürimäe T. Adiponectin is altered after maximal exercise in highly trained male rowers. Eur J Appl Physiol 93:502–505, 2005.
18. Ferguson MA, White LJ, McCoy S, Kim HW, Petty T, Wilsey J. Plasma adiponectin response to acute exercise in healthy subjects. Eur J Appl Physiol 91:324–329, 2004.
19. Jürimäe J, Purge P, Jürimäe T. Adiponectin and stress hormone responses to maximal sculling after volume-extended training season in elite rowers. Metabolism 55:13–19, 2006.
20. Weltman A, Pritzlaff CJ, Wideman L, Considine RV, Fryburg DA, Gutgesell ME, Hartman ML, Veldhuis JD. Intensity of acute exercise does not affect serum leptin concentrations in young men. Med Sci Sports Exerc 32:1556–1561, 2000.
21. Dill DB, Costill DL. Calculation of percentage changes in volumes of blood, plasma and red cells in dehydration. J Appl Physiol 37:247–248, 1974.
22. Bobbert T, Brechte L, Mai K, Otto B, Maser-Gluth C, Pfeiffer AFH, Spranger J, Dietrich S. Adaptation of the hypothalamic-pituitary hormones during intensive endurance training. Clin Endocrinol 63: 530–536, 2005.
23. Anderrson U, Treebak JT, Nielsen JN, Smith KL, Abbott CR, Small CJ, Carling D, Richter EA. Exercise in rats does not alter hypothalamic AMP-activated protein kinase activity. Biochem Biophys Res Commun 329:719–725, 2005.
24. Pritzlaff-Roy CJ, Wideman L, Weltman JY, Abbot R, Gutgesell M, Hartman ML, Veldhuis JD, Weltman A. Gender governs the relationship between exercise intensity and growth hormone release in young adults. J Appl Physiol 92:2053–2060, 2002.
25. Oh KW, Lee WY, Rhee EJ, Baek KH, Yoon KH, Kang MI, Yun EJ, Park CY, Ihm SH, Choi MG, Yoo HJ, Park SW. The relationship between serum resistin, leptin, adiponectin, ghrelin levels and bone mineral density in middle-aged men. Clin Endocrinol 63:131–138, 2005.
26. Brüning JC, Gautam D, Burks DJ. Role of brain insulin receptor in control of body weight and reproduction. Science 289:2122–2125, 2000.
27. Ariyasu H, Takaya K, Tagami T, Ogawa Y, Hosoda K, Akamizu T, Suda M, Koh T, Natsui K, Toyooka S, Shirakami G, Usui T, Shimatsu A, Doi K, Hosoda H, Kojima M, Kangawa K, Nakao K. Stomach is a major source of circulating ghrelin, and feeding state determines plasma ghrelin-like immunoreactivity levels in humans. J Clin Endocrinol Metab 86:4753–4758, 2001.