Original Paper

DE GRUYTER OPEN

Biomedical Human Kinetics, 10, 67–75, 2018

DOI: 10.1515/bhk-2018-0011

Effect of chronic high-intensity exercise on hunger and satiation and levels of acylated ghrelin and leptin in women

Mahmoud Hesar Koshki[1], Azam Mollanovruzi[2], Amin Rashid Lamir[3]

[1] Faculty of Physical Education and Sport Sciences, Hakim Sabzevari University, Sabzevar, Iran; [2] Faculty of Physical Education and Sport Sciences, Kosar University of Bojnord, Iran; [3] Faculty of Physical Education and Sport Sciences, Neishabour University, Neishabour, Iran

Summary

Study aim: The purpose of this study was to investigate the effect of chronic high-intensity exercise training on hunger, satiation, related hormones and weight loss among women who are obese or overweight.

Material and methods: The sample group was composed of 25 subjects, divided into two groups: a high-intensity exercise group (n = 15) intensity of 80–90% of maximum heart rate and a non-training control group (n = 10). The blood sample test was performed in two stages of rest, fasted state and before breakfast and an appetite questionnaire was completed. The training procedure was a high-intensity exercise, three sessions a week for twelve weeks and two hours after breakfast. Independent and dependent t-tests were used to analyze the data.

Results: The results showed that high-intensity exercise caused a significant loss of weight ($p = 0.01$) and fat percentage ($p = 0.001$) and plasma insulin levels ($p = 0.03$), but it had no effect on hunger and satiation, calorie intake or plasma leptin. Also, it significantly increased the plasma acylated ghrelin ($p = 0.04$) and maximum oxygen consumption ($p < 0.001$).

Conclusions: Based on the results of this study, it can be noted that high-intensity exercise is suitable for weight loss and not increasing hunger. The lack of calorie intake along with weight loss showed that negative energy balance caused no appetite compensatory responses. Also, high-intensity exercise stimulates physiological responses to increase appetite, but it did not affect the feeling of appetite. In other words, changes in mental and physiological appetite because of high-intensity exercise do not match.

Key words: High-intensity exercise training – Appetite – Obesity – Weight loss

Introduction

The Organization for Economic Co-operation and Development in 2012 reported that in many countries, one in two people are overweight or obese [46]. It is estimated that if obesity continues at the current estimated rate, 3.3 billion adults, or 57.8% of the world's population, will be obese by 2030 [24]. An increase in the pervasiveness and prevalence of obesity and related diseases worldwide is indicative of the fact that human progress has not succeeded in identifying factors and mechanisms for regulating weight, and in particular preventing, fighting and treating obesity. It has been shown that weight balance has two sides and disruption on each side leads to obesity, and the weight stability during long periods requires a balance between receiving and expending energy [10]. Appetite, which plays an important role in controlling energy balance, is one of the factors affecting body weight and hemostatic energy. The regulation of appetite control and energy balance is an area of scientific inquiry which continues to receive widespread attention across disciplines [1]. Physical activity can also affect nutritional behavior and improve appetite control [8, 20]. In order to develop evidence-based guidelines for weight management, it is important to understand how exercise affects energy balance (energy intake-energy expenditure) [16]. Numerous studies have evaluated the effectiveness of exercising programs with different structures regarding the number of sessions in a week and the intensity and duration of training [12, 41].

Physical activity is a factor that generates a negative energy balance by increasing energy expenditure [8]. Thus, in order for obese and overweight people to lose weight, physical activity should also be increased. High-intensity exercise compared to low-intensity exercise causes loss of more energy in a short time. Thus, it is possible that increasing the intensity of exercise will lead to more weight

Author's address Azam Mollanovruzi, Kosar University of Bojnord, Iran mollanovruzi@kub.ac.ir

loss. Obese women suffering from metabolic syndrome showed a significant reduction in fat by participating in physical activities, and the maximum decrease was among those who performed high-intensity exercise [21]. High-intensity exercise training may reduce abdominal fat, which is a known risk factor for metabolic syndrome [9]. Most exercise programs have focused on moderate-intensity exercise to reduce fat [47]. Evidence suggests that high-intensity exercise can also lead to weight loss among obese people [6]. More weight loss has been reported among people with an initial higher fat mass. The possible mechanism for high-intensity exercise is to reduce weight loss, increase fat oxidation and reduce appetite after exercise [4].

The physiological control of appetite regulation involves circulating hormones with orexigenic (appetite-stimulating) and anorexigenic (appetite-inhibiting) properties that induce alterations in energy intake via perceptions of hunger and satiety [17, 29]. Not performing physical exercise in the long term reduces the self-regulation of energy balance and leads to a positive energy balance and it leads to overweight. In addition, it has been shown that weight loss affects the level of ghrelin, which plays a key role in regulating energy balance [32].

Acylated ghrelin is considered as the first hunger hormone [5]. Ghrelin occurs in both an acylated ghrelin and de-acylated ghrelin form. The acylated ghrelin type is active and is considered to be the first and only hunger hormone in the bloodstream [26]. Leptin is a hormone that regulates energy balance. It seems that leptin is transmitted to the central nervous system as an energy-saving signal to reduce appetite [18]. Due to specific receptors in the hypothalamus, the hormone reduces appetite by inhibiting neuropeptide Y secretion, and on the other hand, by increasing the body's metabolism, it controls the amount of needed energy and thus the amount of body fat. Also, the reduction in fasting insulin because of exercise training has been reported to be between 23% and 33% [45].

Some studies have reported the effect of physical activities on the acylated ghrelin level as increasing it and some as not changing it [22, 31, 37, 42]. Studies have also been conducted on leptin compatibility with long-term exercise training and the reported results were conflicting [37].

A growing body of research has demonstrated a link between exercise and the physiological mechanisms controlling appetite and energy intake, with inactivity potentially contributing to a positive energy balance and subsequent weight gain (37). If it is proved that high-intensity exercise, in addition to providing a foundation for weight loss, can reduce appetite, it may be helpful in providing exercise recommendations to obese people. Considering what has been mentioned and contradictory information on the effect of different types of long-term exercise programs on levels of acylated ghrelin, leptin, and insulin as well as hunger and satiation, the purpose of this study was to examine the effect of chronic exercise (high-intensity exercise compared with a no-exercise control group) on appetite regulation, perceptions of appetite and the circulating concentrations of appetite-related hormones (particularly acylated ghrelin, leptin and insulin) in the fasted state in previously inactive overweight women. It was hypothesized that 12 week of exercise would improve appetite regulation compared with a no-exercise control group. In an effort to guide future research, it is important to consider how exercise alters the circulating concentrations of these appetite-regulating hormones.

Material and methods

To select the subjects, first the volunteers were informed about the study subject, its purpose and method and 25 were selected as a statistical sample. Subjects were divided into two groups, a control group (n = 10) and a high-intensity exercise group (n = 15), based on weight and body fat percentage. All subjects were asked to complete the written consent form to participate in the research, a medical history questionnaire, and the Physical Activity Readiness Questionnaire (PAR-Q) [39]. The Ethics Committee of Hakim Sabzevari University approved the study. The inclusion criteria comprised body mass index (BMI) more than 25 kg/m^2, being less than 50 years old, being female, not having a specific diet and having almost constant weight (±2 kg) for at least six months, no menopause, non-athletic, and having a sedentary lifestyle (not doing regular exercise). Exclusion criteria were smoking cigarettes or any drug abuse, a history of heart disease, renal disease, or type 1 or 2 diabetes [22, 42]. This information was obtained through a questionnaire.

Research method and study protocol

The research method was quasi-experimental. One week before beginning the exercise program, according to the timetable of the research design, the anthropometric and physiological parameters comprising height, weight, fat percentage, body mass index and waist-to-hip ratio (WHR) were measured.

Fat percentage

As a measure of adiposity, skinfold thickness is a valuable tool in both the clinical and research settings [44]. In this research, the body fat percentage was measured using calipers (SAEHAN, SH5020 made in South Korea) by the skinfold body fat method. The different sets of calipers had been calibrated to ensure consistent instrumentation. The Jackson-Pollock 3-point method was used to calculate the skinfold body fat and the fat was measured at three points in the triceps muscle, suprailiac and thighs by calipers. Skinfold measurements were taken on the right side of the body

while the subject was standing erect with her arms by her sides. The locations of the skinfolds at the three sites were:

1. Triceps – a vertical fold was picked up about 1 cm on the back of the arm, midway between the tip of the acromion process of the scapula and the olecranon process of the ulna. The mid-point was measured with the arm flexed at the elbow; the skinfold was lifted parallel to the long axis of the arm with the arm hanging freely at the side.
2. Suprailiac – a slanted fold was lifted in the midaxillary line at the level of the iliac crest of the ilium from the anterior part of the body.
3. Mid-thigh – a vertical fold was lifted about 1 cm from the midline of the front of the thigh halfway between the inguinal ligament and the top of the patella while the body weight shifted on to the left leg and the right leg was relaxed but not lifted off the floor.

Each site was located visually and marked so that consecutive trials of measurements were at the identical site. Each measurement was repeated until three identical readings were taken from all sites and the average of the three values was calculated for subsequent analysis. The sum of the three skinfolds was used to estimate body density. Predicted density was converted to percent fat using the Siri equation [14].

Energy expenditure

The 24-hour physical activity information recording form was provided to subjects to complete it during a one-week course prior to the implementation of the research program and the sixth week of exercise to determine the subjects' calorie expenditure during the pre-test period and during exercise. The calorie expenditure is the amount of energy used daily in calories, calculated by recording the 24-hour activity of the person [43].

Maximum oxygen consumption

The Rockport 1-mile (1609 m) walk test was used to estimate the maximum oxygen consumption. In this test, a Polar F11 Heart Rate Monitor Watch (made in Finland) and chronometer were used to record heart rate and the final time, and then the oxygen consumption of the subject was calculated in milliliters of oxygen per kg of body weight per minute ($ml \cdot (kg \cdot min)^{-1}$) [25].

Habitual calorie intake

During the 12 weeks of exercise the subjects were asked not to change their diet and three days before the first and second blood sample test, the amount of their energy intake was determined by 24-hour dietary recall [30]. N4 software was used to determine the energy received by the subjects. A guide book to domestic scales, conversion factors and edible food percentages was used to determine the food weight.

Measuring the appetite and blood sampling

All subjects were asked not to exercise for 48 hours before the first blood sampling. On the blood sampling day, the subjects completed the appetite questionnaire while the subjects were fasting for at least eight hours [13]. The questionnaire had two items:

1. How hungry are you?
2. How full are you?

The used scale is graded from zero to 150 ml and is divided into five modes that determine the severity of individual mental emotions. Then, while the subjects were in a sitting position and at rest, 5 ml of blood was taken from their antebrachial vein. After that, the members of the experimental group performed exercises for twelve weeks and the control group only performed their daily routine activities. They had no exercise training at all. Also, the appetite questionnaire was completed on the second day of blood sampling in a fasting state and before lunch, and during all twelve weeks before exercise and at a specified hour by the subjects. After twelve weeks, the subjects were under pre-exercise conditions, i.e. physical inactivity 48 hours prior to blood sampling and they completed the appetite questionnaire like the first stage, and blood samples were taken from the subjects. The resulting plasma was frozen at –80 °C.

Hormonal measurements

The plasma samples were transferred to a specialized laboratory to determine the concentration of acylated ghrelin, leptin, and insulin. The Human Acylated ghrelin(AG) ELISA Kit (EASTBIOPHARM Company) China was used to measure acylated ghrelin, with a sensitivity of 2.6 ng/ml, and a 10% intragroup variation coefficient. The Human Leptin(LEP)ELISA Kit (EASTBIOPHARM Company) China was used to measure leptin, with a sensitivity of 1.2 ng/ml, and a 10% intragroup variation coefficient and Human Insulin(INS)ELISA Kit (Demeditec Company) Germany was used to measure insulin, with a sensitivity of 1.76 ng/ml, and a 2.6% intragroup variation coefficient.

Training procedure

It was a twelve-week course of exercise three sessions per week. Exercise time in the high-intensity group was two hours after breakfast. High-intensity exercise included fast-running forward and backward for 30 seconds, with 80–90% of maximum heart rate. The interval break time was 90 seconds. The training period was 51 minutes (Table 1). The maximum heart rate was calculated from the formula (220 – age) for each subject. A Polar F11 Heart Rate Monitor Watch was used to record heart rate. The control group only performed their daily routine activities. They had no exercise training at all.

Table 1. High intensity exercise training protocols

Week / Variables	1	2	3	4	5	6	7	8	9	10	11	12
Intensity (maximum heart rate)	80–85	80–85	80–85	80–85	80–85	80–85	85–90	85–90	85–90	85–90	85–90	85–90
Rest time [min.]	1.5	1.5	1.5	1.5	1.5	1.5	1.5	1.5	1.5	1.5	1.5	1.5
Repeat of running	10	12	14	16	18	20	20	21	22	23	24	25
Warm up time [min.])	10	10	10	10	10	10	10	10	10	10	10	10
Cool down time [min.]	5	5	5	5	5	5	5	5	5	5	5	5
Total workout time [min.]	33.5	37.5	41.5	45.5	49.5	53.5	53.5	55.5	57.5	59.5	61.5	63.5

Statistical analysis

Descriptive statistics were used to calculate the central index and dispersion. Descriptive statistics were calculated for all results as means ± SD. The Shapiro-Wilk test was also used to verify the normal data distribution. The independent t-test was used to examine the significance of differences between independent groups. The dependent t-test was used to evaluate within-group differences. All statistical operations were performed in SPSS 18 (IBM Corporation, Armonk, NY, USA) for specific purposes. The significance level of the hypothesis test was considered as $\alpha = 0.05$.

Results

Before beginning the course and after 12 weeks of high-intensity exercise, using descriptive statistics and standard deviation of age, the anthropometric and physiological parameters comprising height, weight, fat percentage, maximum oxygen consumption, body mass index and waist-to-hip ratio (WHR) were measured. As can be seen, the fat percentage in the high-intensity exercise group significantly decreased after exercise training ($p = 0.004$). Also oxygen consumption in this group was significantly increased after exercise ($p < 0.001$) (Table 2). Changes in some anthropometric indices after exercise training were also calculated, and the results are shown in Table 3. It is clear that weight loss ($p = 0.01$) and decrease in fat percentage ($p = 0.001$) were significant in the high-intensity group. Also, changes in oxygen consumption in the high-intensity group were significantly increased ($p = 0.001$). Changes in waist-hip ratio and BMI were not significant (Table 3).

The results showed that there was no significant difference in plasma leptin levels before and after 12 weeks of exercise training. Therefore, high-intensity exercise did not have any significant effect on plasma leptin levels, while there was a significant decrease in within-group leptin levels between pre-test and post-test in the high-intensity exercise group ($p = 0.01$). The changes between

Table 2. Between-group changes of anthropometric and physiological characteristics of subjects

Anthropometric and physiological characteristics	HIT group (Pre)	Control group (Pre)	Between-group p value	HIT group (Post)	Control group (Post)	Between-group p value
Age [years]	33.92 ± 5.19	37.30 ± 8.25	0.28	–	–	–
Height [cm]	160.46 ± 3.41	158.40 ± 4.53	0.23	–	–	–
Weight [kg]	74.51 ± 4.65	72.57 ± 9.55	0.62	70.80 ± 5.87	73.05 ± 9.87	0.60
Body fat percentage	40.55 ± 3.59	40.09 ± 3.15	0.78	36.78 ± 2.16*	41.04 ± 3.41	0.004
Waist to hip ratio (WHR)	0.86 ± 0.05	0.82 ± 0.05	0.19	0.84 ± 0.06	0.84 ± 0.07	0.82
Body mass index (BMI) [kg/m^2]	28.62 ± 1.87	30.20 ± 3.07	0.17	27.74 ± 2.08	30.02 ± 3.01	0.06
Maximum oxygen consumption [$mL \cdot kg^{-1} \cdot min.^{-1}$]	32.92 ± 3.71	29.92 ± 5.44	0.06	42.03 ± 3.87*	30.51 ± 4.62	0.0001

HIT: high intensity exercise training; * – significantly different ($p < 0.05$) from control group.

Table 3. Changes of anthropometric and physiological characteristics pre and post training

Variables	HIT group	Control group	Between-group p value
Changes of weight [kg]	–3.71 ± 3.71 *	0.48 ± 1.25	0.01
Changes of body fat percentage	–3.77 ± 2.84 *	0.94 ± 1.48	0.001
Changes of body mass index (BMI) [kg/m^2]	–0.88 ± 0.85	–0.18 ± 0.62	0.06
Changes of waist to hip ratio (WHR)	–0.01 ± 0.06	0.0008 ± 0.02	0.46
Changes of maximum oxygen consumption [mL · kg^{-1} · min.$^{-1}$]	7.20 ± 3.23 *	0.59 ± 4.09	0.001

HIT: high intensity exercise training; * – significantly different ($p \leq 0.05$) from control group.

Table 4. Changes in the concentration of plasma leptin, acylated ghrelin and insulin of subjects

Variables	Measuring time	Pre	Post	Changes of Pre and Post	Within-group p value
Leptin [ng/ml]	Control group	512.80 ± 233.88	483.20 ± 184.72	–29.60 ± 91.55	0.33
	HIT group	892.00 ± 682.59	872.70 ± 693.74	–19.30 ± 19.94	0.01
Between-group p value		0.13	0.12	0.74	–
Acylated ghrelin [pg/ml]	Control group	709.60 ± 264.19	665.10 ± 219.97	–44.50 ± 55.88	0.03
	HIT group	1154.40 ± 835.59	1167.90 ± 832.18	13.50 ± 63.68*	0.52
Between-group p value		0.14	0.09	0.04	–
Insulin [IU/L]	Control group	24.07 ± 13.94	25.76 ± 13.28	1.69 ± 6.88	0.46
	HIT group	17.67 ± 4.40	14.58 ± 3.44*	–3.09 ± 5.20	0.09
Between-group p value		0.18	0.03	0.10	–

HIT: high intensity exercise training; * – significantly different ($p < 0.05$) from control group.

pre-test and post-test showed that there was a significant increase in the level of acylated ghrelin in the high-intensity group ($p = 0.04$). Also, high-intensity exercise reduced the insulin levels significantly ($p = 0.04$) (Table 4). Also, the results showed that there was no significant difference in hunger and satiation between the two groups before breakfast and before lunch, during the blood sampling. There was a significant increase in energy expenditure in the high-intensity group ($p = 0.001$), but there was no significant difference in daily calorie intake and appetite between the two groups.

Discussion

The main purpose of this study was to study the effect of chronic weight loss induced by high-intensity exercise on appetite and acylated ghrelin and leptin hormones among overweight or obese women. The results of this study showed that anthropometric indices decreased significantly because of sport exercises. Exercise training is used as a strategy to combat overweight, and weight loss, fat percentage, and body composition changes usually occur following exercise in subjects who are obese or overweight [22, 48]. High-intensity exercise increases the amount of fat oxidation in the muscle and reduces the amount of fat. The mechanism responsible for this change may be the effect of β-adrenergic stimulation in high-intensity sports activity on lipid metabolism and increased lipolysis [8, 48].

The results of some studies have shown that exercise intensity is an important factor in improving the maximum oxygen consumption. In addition, high-intensity exercise increases mitochondrial capacity. Sim *et al*. (2015) also found that high-intensity exercise increased aerobic fitness [37]. The results of this study showed that the maximum consumed oxygen in the high-intensity group was significantly increased after exercise.

Also, the results showed there are no significant changes in calorie intake after exercise. Given the increased energy expenditure in the high-intensity group and the lack of changes in calorie intake, it can be concluded that

performing high-intensity exercise and increasing energy expenditure does not compensate for the increased calorie intake. One reason the effect of exercise activity on weight loss is not significant is that adaptation to sports activities caused compensation responses such as increasing calorie intake or reducing the energy expenditures of other physical activities. It has been reported that exercise can positively regulate the calorie intake or the negative regulation of physical activity other than exercise and thus neutralize exercise-induced calorie expenditure [34]. The results of the current study are consistent with many studies and showed that there is no increase in calorie intake due to physical activities [7, 35, 37]. Donnelly *et al.* (2013) in a review article concluded that 59% of cross-sectional studies, 50% of short-term studies and 75% of randomized intervention studies did not show any change in calorie intake after exercise. It has been shown that none of the factors related to exercise activity such as type, intensity, duration, or characteristics of subjects such as age, sex, or weight affect calorie intake [11]. Also, there was no reduction in daily activities during the training period, and subjects did not compensate for the energy expenditure of exercises by reducing other daily activities. Therefore, it can be said that exercise in this study caused negative balance and weight loss.

It is assumed that hormones regulating appetite, such as ghrelin, play a role in regulating calorie intake [38]. Studies conducted on obese and healthy individuals showed that ghrelin levels increased with weight loss after exercise and food restriction [40]. One of the consequences of physical activity is the increase in calorie expenditure that generates signals for ghrelin producing cells in the stomach, and these signals affect appetite regulation [27]. Ueda *et al.* (2013), Moraes *et al.* (2015) and Martins *et al.* (2010) reported that acylated ghrelin fasting levels significantly increased due to aerobic exercises, and this increase of acylated ghrelin can increase the appetite and consequently the calorie intake in order to restore energy balance [31, 33, 42]. Ueda *et al.* (2013) examined the effect of 12 weeks of exercise with 65% intensity on 20 middle-aged Japanese women. The results showed that exercise induced a significant increase in fasting acylated ghrelin [42]. Moraes *et al.* (2015) also performed a study of 52 patients undergoing hemodialysis to study ghrelin changes during 6 months. The results showed that the amount of acylated ghrelin increased. Therefore, the ghrelin interferes with a negative feedback loop in regulating body weight [33]. It also seems that the leptin hormone secreted from adipose tissue plays a role in long-term regulation of energy hemostasis. A significant decrease in leptin has been reported due to exercise training [19, 32, 37]. The results of this study showed that 12 weeks of high-intensity exercise did not have a significant effect on fasting plasma leptin, but significantly increased the acylated ghrelin levels. These results were not consistent with Guelfi *et al.* (2013), Sim *et al.* (2015) and Jones (2009) and no significant change in fasting acylated ghrelin levels was observed [15, 22, 37]. Sim *et al.* (2015) examined the effect of 12 weeks of high-intensity intermittent exercise training on acylated ghrelin levels among 30 men. The results showed that high-intensity intermittent exercise training did not change the acylated ghrelin [37]. In the present study, weight loss caused by high-intensity exercise increased the amount of acylated ghrelin. The results of this study showed a decrease in within-group values of leptin in the high-intensity group, but this decrease did not reach a significant intergroup level. The leptin decrease after weight loss can also reduce its repressive response to appetite and increase appetite. In the present study, the lack of changes in leptin after exercise and the lack of calorie intake confirm the positive relationship between these two. Leptin levels did not change as a result of exercise, resulting in an unchanged caloric intake.

The insulin concentration was also measured in the current study. The results showed that 12 weeks of high-intensity exercise significantly reduced plasma fasting insulin levels. Insulin is a hormone that increases with increasing blood glucose levels after food and acts as an anti-hunger signal [28]. Insulin reduction is also shown after exercise [35, 42]. Kanaley *et al.* (2014) studied the effect of 15-day walking exercise with 70–75% VO_{2peak} intensity in 13 obese subjects with glucose and insulin and the results were not consistent with the current study. The results showed that physical exercises did not change these indices significantly [23]. Insulin has a negative correlation with ghrelin [2]. Therefore, one reason for acylated ghrelin increasing due to exercise training may be insulin reduction.

The results of the current study showed that 12 weeks of aerobic exercise did not have a significant effect on hunger and satiation. Lack of appetite following exercise with weight loss indicates that the negative energy balance created by this intensity does not result in appetite compensatory responses. Previous studies have shown that exercising and increasing energy expenditures result in a negative energy balance and therefore it increases appetite [15, 31, 36]. The results of Ueda *et al.* (2013) and Sim *et al.* (2015) were consistent with our findings and showed a lack of change in appetite due to physical exercise [37, 42]. On the other hand, the appetite associated with exercise training also has different forms, depending on the duration, intensity and frequency of exercise training, physical fitness and physiological state of people. Therefore, more studies are needed in order to better understand appetite changes, as well as their relation to exercise, to examine hormonal, metabolic and other relevant factors simultaneously. The effect of exercise on appetite varies from one person to another. Individual responses to exercise can therefore vary

widely and are very difficult to predict [3, 38, 46]. Also, it is likely that measuring appetite during a course of exercise using a mental scale will affect its outcomes. Generally, it is not assumed that the fluctuation of appetite is only influenced by known and fully controllable factors. There are other factors that can affect it as well: social effects, the availability and diversity of food, the expenditure of daily energy along with the economic or cultural status factor. Since the sample size was too small and there is a lack of similar evidence, it is necessary to conduct more longitudinal and more accurate studies.

Conclusions

The results of the study showed that loss of weight and fat percentage occurred due to high-intensity exercise. Also, lack of appetite increase and calorie intake with weight loss shows that the created negative energy balance with this intensity does not result in appetite compensatory responses. Also, high-intensity exercise leads to stimulation of physiological responses to appetite increase, but it has no effect on mental appetite. In other words, the changes in mental and physiological feelings in the high-intensity exercise group are not completely consistent. The results of this study can be applied to designing sport programs and for preventing and treating obesity.

Conflict of interest: Authors state no conflict of interest.

References

1. Alajmi N., Deighton K., King J.A., Reischak-Oliveira A., Wasse L.K., Jones J., Batterham R.L., Stensel D.J. (2016) Appetite and Energy Intake Responses to Acute Energy Deficits in Females versus Males. *Med. Sci. Sports Exerc.,* 48(3): 412-20. DOI: 10.1249/MSS.0000000000000793.
2. Barazzoni R., Zanetti M., Ferreira C., Vinci P., Pirulli A., Mucci M., Dore F., Fonda M., Ciocchi B., Cattin L., Guarnieri G. (2007) Relationships between desacylated and acylated ghrelin and insulin sensitivity in the metabolic syndrome. *J. Clin. Endocrinol. Metab.,* 92(10): 3935-40. DOI: 10.1210/jc.2006-2527.
3. Blundell J.E., Gibbons C., Caudwell P., Finlayson G., Hopkins M. (2015) Appetite control and energy balance: impact of exercise. Obesity Reviews 16(1): 67-76. DOI: 10.1111/obr.12257.
4. Boutcher S.H. (2011) High-Intensity intermittent exercise and fat loss. *J. Obes.,* 2011. DOI: 10.1155/2011/868305.
5. Broom D.R., Batterham R.L., King J.A., Stensel D.J. (2009) Influence of resistance and aerobic exercise on hunger, circulating levels of acylated ghrelin, and peptide YY in healthy males. *Am. J. Physiol*., 296(1): 29-35. DOI: 10.1152/ajpregu.90706.2008.
6. Cassidy S., Thoma C., Houghton D., Trenell M.I. (2017) High-intensity interval training: a review of its impact on glucose control and cardiometabolic health. *Diabetologia,* 60(1): 7-23. DOI: 10.1007/s00125-016-4106-1.
7. Caudwell P., Gibbons C., Hopkins M., King N., Finlayson G., Blundell J. (2013) No sex difference in body fat in response to supervised and measured exercise. *Med.Sci. Sports Exerc.,* 45(2): 351-358. DOI: 10.1249/MSS.0b013e31826ced79.
8. Chaput J.P.H., Klingenberg L., Rosenkilde M., Gilbert J.A., Tremblay A., Sjodin A. (2011) Physical activity plays an important role in body weight regulation. *J. Obes.,* 2011: 1-11. DOI: 10.1155/2011/360257.
9. Coker R.H., Williams R.H., Kortebein P.M., Sullivan D.H, Evans W.J. (2009) Influence of exercise intensity on abdominal fat and adiponectin in elderly adults. *Metab. Syndr. Relat. Disord.,* 7(4): 363-368. DOI: 10.1089/met.2008.0060.
10. Diéguez C., Vazquez M.J., Romero A., Lopez M., Nogueiras R., (2011) Hypothalamic control of lipid metabolism: focus on leptin, ghrelin and melanocortins. *Neuroendocrinology,* 94(1): 1-11. DOI: 10.1159/000328122.
11. Donnelly J.E., Honas J.J., Smith B.K., Mayo M.S., Gibson C.H.A., Sullivan D.K., Lee J., Herrmann S.D., Lambourne K., Washburna R.A. (2013) Aerobic exercise alone results in clinically significant weight loss for men and women: Midwest Exercise Trial-2. *Obesity* (*Silver Spring*), 21(3): 219-228. DOI: 10.1002/oby.20145.
12. Duncan G.E., Anton S.D., Sydeman S.J., Newton R.L. Jr., Corsica J.A., Durning P.E., Ketterson T.U., Martin A.D., Limacher M.C., Perri M.G. (2005) Prescribing exercise at varied levels of intensity and frequency: a randomized trial. *Arch. Intern. Med.,* 165(20): 2362-2369. DOI: 10.1001/archinte.165.20.2362.
13. Flint A., Raben A., Blundell J.E., Astrup A. (2000) Reproducibility, power and validity of visual analogue scales in assessment of appetite sensations in single test meal studies. *Intern. J. Obes. Relat. Metab. Disord.,* 24: 38-48. DOI: 10.1038/sj.ijo.0801083.
14. Frkovic M., Bird E., Arabas J.L., Mayhew J.L., (2016) Horizontal vs Vertical Abdominal Skinfold Measurement Differences in College Men. *Missouri J. Health Phys. Educ. Recreat. Dance,* 25: 12-19.
15. Guelfi K.J., Donges C.E., Duffield R. (2013) Beneficial effects of 12 weeks of aerobic compared with resistance exercise training on perceived appetite in previously sedentary overweight and obese men. *Metab. Clin. Exp.,* 62(2): 235-243. DOI: 10.1016/j.metabol.2012.08.002.
16. Hallworth J.R., Copeland J.L., Doan J., Hazell T.J. (2017) The Effect of Exercise Intensity on Total PYY and GLP-1 in Healthy Females: A Pilot Study. *J. Nutr. Metab.,* 2017: 1-7. DOI: 10.1155/2017/4823102.

17. Hazell T.J., Islam H., Townsend L.K., Schmale M.S., Copeland J.L. (2016) Effects of exercise intensity on plasma concentrations of appetite-regulating hormones: Potential mechanisms. *Appetite,* 98: 80-88. DOI: 10.1016/j.appet.2015.12.016.
18. Hilton L.K., Loucks A.B. (2000) Low energy availability, not exercise stress, suppresses the diurnal rhythm of leptin in healthy young women. American Journal of Physiology. *Endocrinol. Metab.,* 278(1): 43-49. DOI:10.1152/ajpendo.2000.278.1.E43.
19. Hopkins M., Gibbons C., Caudwell P., Webb D.L., Hellström P.M., Näslund E., Blundell J.E., Finlayson G. (2014) Fasting leptin is a metabolic determinant of food reward in overweight and obese individuals during chronic aerobic exercise training. *Int. J. Endocrinol.,* 2014: 1-8. DOI: 10.1155/2014/323728.
20. Howe S.M., Hand T.M., Manore M.M. (2014) Exercise-trained men and women: role of exercise and diet on appetite and energy intake. *Nutrients,* 6(11): 4935-4960. DOI: 10.3390/nu6114935
21. Irving B.A., Davis C.K., Brock D.W., Weltman J.Y., Swift D., Barrett E.J., Gaesser G.A., Weltman A. (2008) Effect of exercise training intensity on abdominal visceral fat and body composition. *Med. Sci. Sports Exerc.,* 40(11): 1863-1872. DOI: 10.1249/MSS.0b013e3181801d40.
22. Jones T.E., Basilio J.L., Brophy P.M., McCammon M.R., Hickner R.C. (2009) Long-term exercise training in overweight adolescents improves plasma peptide YY and resistin. *Obesity,* 17(6): 1189-1195. DOI: 10.1038/oby.2009.11.
23. Kanaley J.A., Heden T.D., Liu Y., Whaley-Connell A.T., Chockalingam A., Dellsperger K.C., Fairchild T.J. (2014) Short-term aerobic exercise training increases postprandial pancreatic polypeptide but not peptide YY concentrations in obese individuals. *Int. J. Obes.,* 38(2): 266-271. DOI: 10.1038/ijo.2013.84.
24. Kelly T., Yang W., Chen C.S., Reynolds K., He J. (2008) Global burden of obesity in 2005 and projections to 2030. *Int. J. Obes.,* 32(9): 1431-1437. DOI: 10.1038/ijo.2008.102.
25. Kline G.M., Porcari J.P., Hintermeister R., Freedson P.S., Ward A., McCarron R.F., Ross J., Rippe J.M. (1987) Estimation of VO_{2max} from one-mile track walk, gender, age and body weight. *Med. Sci. Sports Exerc.,* 19(3): 253-259.
26. Kojima M., Hosoda H., Matsuo H., Kangawa K. (2001) Ghrelin: discovery of the natural endogenous ligand for the growth hormone secretagogue receptor. *Trends Endocrinol. Metab.,* 12(3): 118-122. DOI: 10.1016/S1043-2760(00)00362-3.
27. Kraemer R.R., Castracane V.D. (2007) Exercise and humoral mediators of peripheral energy balance: ghrelin and adiponectin. *Exp. Biol. Med.,* 232(2): 184-194. DOI: 10.3181/00379727-207-2320184.
28. Kraemer R.R., Durand R.J., Acevedo E.O., Johnson L.G., Kraemer G.R., Hebert E.P., Castracane V.D. (2004) Rigorous running increases growth hormone and insulin-like growth factor-I without altering ghrelin. *Exp. Biol. Med.,* 229(3): 240-246. DOI: 10.1177/153537020422900304.
29. Lean M.E.J., Malkova D. (2016) Altered gut and adipose tissue hormones in overweight and obese individuals: cause or consequence? *Int. J. Obes.,* 40: 622-632. DOI: 10.1038/ijo.2015.220.
30. Mahan L.K., Escott-Stump S. (2008) Krause's food, nutrition and diet therapy. 11th ed. Translated by: Vosogh S. Tehran: Hayan publication.
31. Martins C., Kulseng B., King N.A., Holst J.J., Blundell J.E. (2010) The effects of exercise-induced weight loss on appetite – related peptides and motivation to eat. *J. Clin. Endocrinol. Metab.,* 95(4): 1609-1616. DOI: 10.1210/jc.2009-2082.
32. Martins C., Kulseng B., Rehfeld J.F., King N.A., Blundell J.E. (2013) Effect of chronic exercise on appetite control in overweight and obese individuals. *Med. Sci. Sports Exerc.,* 45(5): 805-812. DOI: 10.1249/MSS.0b013e31827d1618.
33. Moraes C., Marinho S., Lobo J.C., Stockler-Pinto M.B., Barros A.F., Jacobson L.V., da Nobrega A.C., Rosa M.L., Denise M. (2015) Effects of resistance exercise training on acyl-ghrelin and obestatin levels in hemodialysis patients. *Renal Failure,* 37(5): 851-857. DOI: 10.3109/0886022X.2015.1033634.
34. Myers C.A., Johnson W.D., Earnest C.P., Rood J.C., Tudor-Locke C., Johannsen N.M., Cocreham S., Harris M., Church T.S., Martin C.K. (2014) Examination of mechanisms (E-MECHANIC) of exercise-induced weight compensation: study protocol for a randomized controlled trial. *Trials,* 15: 212. DOI: 10.1186/1745-6215-15-212.
35. Rosenkilde M., Auerbach P., Reichkendler M.H., Ploug T., Stallknecht B.M., Sjödin A. (2012) Body fat loss and compensatory mechanisms in response to different doses of aerobic exercise--a randomized controlled trial in overweight sedentary males. *Am. J. Physiol.,* 303(6): 571-519. DOI:10.1152/ajpregu.00141.2012.
36. Rosenkilde M., Reichkendler M.H., Auerbach P., Toräng S., Gram A.S., Ploug T., Holst J.J., Sjödin A., Stallknecht B. (2013) Appetite regulation in overweight, sedentary men after different amounts of endurance exercise: a randomized controlled trial. *J. Appl. Physiol.,* 115: 1599-1609. DOI: 10.1152/japplphysiol.00680.2013.
37. Sim A.Y., Wallman K.E., Fairchild T.J., Guelfi K.J., (2015) Effects of High-Intensity Intermittent Exercise Training on Appetite Regulation. *Med. Sci. Sports Exerc.,* 47(11): 2441-2449. DOI: 10.1249/MSS.0000000000000687.
38. Thackray A.E., Deighton K., King J.A., Stensel D.J. (2016) Exercise, Appetite and Weight Control: Are There Differences between Men and Women? *Nutrients,* 21: 8(9) pii: E583. DOI: 10.3390/nu8090583.

39. Thomas S., Reading J., Shephard R.J. (1992) Revision of the Physical Activity Readiness Questionnaire (PAR-Q). *Canadian J. Sport Sci.,* 17(4): 338-345. DOI: 10.1152/japplphysiol.00680.2013.
40. Tiryaki-Sonmez G., Vatansever S., Olcucu B., Schoenfeld B. (2015) Obesity, food intake and exercise: Relationship with ghrelin. *Biomed. Hum. Kinet.,* 7: 116-124. DOI: 10.1515/bhk-2015-0018.
41. Tremblay A., Simoneau J.A., Bouchard C. (1994) Impact of exercise intensity on body fatness and skeletal muscle metabolism. *Metabolism,* 43(7): 814-818.
42. Ueda S.Y., Miyamoto T., Nakahara H., Shishido T., Usui T., Katsura Y., Yoshikawa T., Fujimoto S. (2013) Effect of exercise training on gut hormone levels after a single bout of exercise in middle-aged Japanese women. *SpringerPlus* 2(1): 83. DOI: 10.1186/2193-1801-2-83.
43. Wardlaw G.M., Kessel M., (2007). Perspectives in Nutrition. 7th ed. MC Graw Hill. Toronto.
44. Wendel D., Weber D., Leonard M.B., Magge S.N., Kelly A., Stallings V.A. (2017) Body composition estimation using skinfolds in children with and without health conditions affecting growth and body composition. *Ann. Hum. Biol.,* 44(2): 108-120. DOI:10.3109/03014460.2016.1168867.
45. Whyte L.J., Gill J.M., Cathcart A.J. (2010) Effect of 2 weeks of sprint interval training on health-related outcomes in sedentary overweight/obese men. *Metab. Clin. Exp.,* 59(10): 1421-1428. DOI: 10.1016/j.metabol.2010.01.002.
46. Williams R.L., Wood L.G., Collins C.E., Callister R., (2015) Effectiveness of weight loss interventions – is there a difference between men and women: a systematic review. *Obesity Reviews,* 16(2): 171-186. DOI: 10.1111/obr.12241.
47. Wu T., Gao X., Chen M., van Dam R.M. (2009) Long-term effectiveness of diet-plus-exercise interventions vs. diet-only interventions for weight loss: a meta-analysis: obesity Management. *Obesity Reviews* 10(3): 313-323. DOI: 10.1111/j.1467-789X.2008.00547.x.
48. You T., Wang X., Yang R., Lyles M.F., Gong D., Nicklas B.J. (2012) Effect of exercise training intensity on adipose tissue hormone sensitive lipase gene expression in obese women under weight loss. *J. Sport Health Sci.,* 1(3): 184-190. DOI: 10.1016/j.jshs.2012.10.001.

Received 28.01.2018
Accepted 04.05.2018