ACUTE EFFECTS OF CONCURRENT TRAINING ON SERUM LEPTIN AND CORTISOL IN OVERWEIGHED YOUNG ADULTS

EXERCISE AND SPORTS MEDICINE CLINIC



ORIGINAL ARTICLE

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ABSTRACT

Objective: This study aimed to analyze the immediate effects of concurrent training on leptin and cortisol levels in overweighed young adults. Methods: This study used a quasi-experimental methodology. We included 20 volunteers of both sexes, randomly divided into a training competitor group (TCG n = 10) and a control group (C n = 10). Blood collection was performed in individuals at rest after fasting for 12 hours. The leptin and cortisol levels were analyzed by radioimmunoassay and chemiluminescence immunoassay before and immediately after training. Two-way ANOVA was used for statistical analysis with a significance level of p < 0.05. Results: In the analysis of leptin levels, there was significant difference between groups (TCG x C) in the pre intervention (p = 0.02) and post intervention (p = 0.01). In the intra groups, no significant changes were found, and in the analysis of cortisol levels between groups (TGC x C), a significant difference in the pre intervention (p = 0.01) and post intervention (p = 0.01) was observed; however, in the intra groups there were no significant changes. Conclusion: A single concurrent training session is not sufficient to promote acute changes in the leptin and cortisol levels of the volunteer overweighed young adults in this study.

Keywords: obesity, cycling, resistance training.

INTRODUCTION

Over the 1990 decade, the percentage of individuals with overweight and obesity has significantly increased¹, and, nowadays is considered a global epidemics². Although the scientific community does not know if overweight and obesity are multifactorial phenomena, it acknowledges the need to explore many ways of increasing the daily energetic cost in order to control and reduce the prevalence of these diseases².

The recent advances in the endocrinology and metabolism field show that, contrary to what was believed some years ago, the adipocyte is not a cell of energy storage, but it also synthetizes and releases many substances, including leptin³, which derives from the Greek *leptis*, which means thin⁴, and was discovered in 1994. Leptin is a peptide hormone composed of 167 amino acids and is mainly produced in the white adipose tissue, being greater in the subcutaneous fat when compared with the visceral adipose tissue⁵, but is different between the adipocytes. Its release occurs during the peak of the night and the first hours of the morning, and its plasma half-life is of 30 minutes⁶.

Leptin concentration is partially related to the size of the mass of the adipose tissue in the body^{7,8}. It is known as the "satiety hormone" for having influence on the appetite reduction process³. Its secretion may be influenced by many physiological mechanisms, such as fasting, the glucocorticoids, the sympathetic activity, physical exercise and alterations in the body weight and energetic balance, which can drastically alter the quantity of leptin intrinsically associated with fat mass⁹a fail in its production, and/or its activity over the receptors of the hypothalamus could distort the positive energetic balance, creating the obesity scenario¹⁰.

Many studies show that the leptin plasma levels are not altered according to the acute aerobic exercise in athletes or nn athletes.

Ribeiroet al.¹¹report that it has been common to communication that exercise, either acute or chronic, causes decrease of leptin concentration. Kraemer et al.¹², in a revision study, suggest that there are many studies on the effect of acute exercise on leptin, and that the majority did not presented any alteration or decrease in the leptin concentration; however, the studies are very contradictory due to the acute or chronic plasma leptin aerobic physical activity.

The study by Weltmanet al. 13 reported that 30 minutes of exercise above or below the lactate threshold, are able to lead to acceleration of metabolism rate, but the exercise intensity may have not been sufficient to alter the leptin concentrations in young men during exercise or recovery periods (3.5 hours post--years), compared with control values. In this study, the exercise intensity was not a factor which affected the leptin responses in young men. Tuominen et al.14 found 34% of reduction in the leptin concentrations in the plasma 44 hours after two hours of exercise at 75% of $\dot{V}O_{2max}$. In the study by Olive and Miller¹⁵, the leptin plasma concentrations were analyzed in the 24 and 48 hours after one hour of moderate exercise periods (~ 900kcal spent) and after intense exercise of short duration (~ 200kcal spent), where decrease of 18% and 40%, respectively after the long duration and moderate intensity activity was observed. None alteration has occurred after the leptin activity concentration of short duration and high intensity.

Cortisol is the main glucocorticoid produced by the adrenal and its secretion is related to many factors, such as: rhythm of the circadian cycle^{16,17}, stress, hypoglycemia and some amino acids¹⁸. The cortisol circulating concentration is at maximal values at the first morning hours, immediately before waking up, and progressively decreases completely during the day, presenting very low levels at night^{19,20}. Exercise and training process are classified as stress

to the human body²¹ and powerful stimulators of fast secretion²².

During acute exercise, in a test of maximum oxygen consumption ($\dot{V}O_{2max}$), the level of blood cortisol increases according to the exercise intensity, once the work load is above the critical threshold (50-60% $\dot{V}O_{2max}$)^{21,22}. Therefore, in the same absolute intensity of exercise, cortisol may present low response from the practice of an exercise training program.In the submaximal exercise, the cortisol response is more variable and influenced by many external factors¹⁹.

If the intensity is below the critical threshold, then the cortisol levels may not increase above the rest levels, or may become in fact reduced²³. However, if it is above the critical threshold intensity, then the cortisol levels will initially increase and subsequently enter a plateau (*steady-state exercise*)²⁴, since while cortisol increases during exercise, at the end of it many alterations and possible effects of this hormone occur during the early recovery²⁵.

The correlation between cortisol and the weight loss process is explained by the effects of the intermediary metabolism²⁶, which among the main functions we can mentionthe maintenance of glycemia and consequent lipolysis stimulation.

Considering hence that one of the stimulating factors of leptin secretion is the increased cortisol level, he aim of this study was to analyze the acute effect of concurrent training on the leptin and cortisol serum levels in overweighed young adults.

METHODOLOGY

Study outlining

This study is quasi-experimental for trying to establish cause/ effect correlations²⁷.

Sample

The non-probabilistic sample was limited to 20 individuals sorted in two groups: concurrent training – TCG (10), and control group – C (10), age between 20 and 35 years, of both sexes, who exercised in the Westfit health club, located in the Realengo neighborhood, in Rio de Janeiro city. The subjects were randomly selected and volunteers, following these inclusion criteria: non-athletes, practitioners of indoor cycling and bodybuilding, with a minimum of six months of activity and weekly frequency of three times, who were with body mass index (BMI) between 25.0 and 29.9, characterizing overweight.

The selected subjects were told about the eating routine and training protocol, signed the Free and Clarified Consent Form and the PAR-Q – *Physical Activity Readiness Questionnaire* application.

This research is according to the Ethical Guidelines for Biomedical Research Involving Humans according to the principles established in the Declaration of *Helsink* (1996). The research Project was approved in Rio de Janeiro, Brazil, by the Ethics in Research Committee of the Castelo Branco University under the protocol number 0157/2008.

DATA COLLECTION

Measurement protocol

During the weeks which preceded the day of the test, the anthropometric measures, one repetition maximum (1RM) test and submaximal oxygen consumption ($\dot{\mathbf{V}}\mathbf{O}_{2\text{submaximum}}$) were performed with the subjects of the sample groups.

The collection of the anthropometric measures of total body mass, fat mass, lean mass, height and BMI was performed according to the protocol by the *International Society for the Advancement of Kinanthropometry* (ISAK)²⁸, in a room specific to functional evaluation in the Westfithealth club premises with the instruments: a Filizola® digital scale with anthropometer previously calibrated, a Sanny® metal measuring tape and a calculator.

The 1RM test²⁹was performed only in the C, to measure the intensity of each exercise which was part of the strength training protocol. The exercises were performed in Biotech® machines and the total used weight (long bar, short bar and free weights) was previously checked on a Filizola® digital scale.

Two weeks after the 1RM test, the measure of the $\dot{V}O_{2submaximal}$ for cycle ergometer expressed in kg.min (ml)⁻¹was done only in the C group, to determine the individual intensity to be used in the indoor cycling class. The test followed the Balke/ACSM protocol (1980), which allows suitable physiological adaptation, as well as guarantees the cardiac safety of the individuals. The test was performed in an Inbrasport*cycle ergometer previously calibrated.

One week after the $\dot{V}O_{2submaximal}$ test, the subjects of the TCG and C group were referred to the blood collection. Both groups received an eating routine of 1,200kcal which was prescribed by nutritionist Fernanda Albuquerque de Andrade, CRN 2003100304, to be followed on the day prior to the test in order to avoid possible increase in fat intake.

Protocol for concurrent training

The exercise sequence for the TCG consisted of two exercise modalities, one aerobic (indoor cycling) andonestrength (bodybuilding). The concurrent training occurred in the following order: indoor cycling practice followed by bodybuilding practice, with no intervals between them, in a single session with approximate duration of two hours.

Protocol for indoor cycling

The cycling classes were performed in a separate room, specific to this practice, with temperature of 17°C in Tomawank bicycles, model XL. Duration of the indoor cycling classes was of 50 minutes with no interruption, with cadence set at 80rpm, according to the bpmof the music corresponding to the necessary rpm, since, according to Mello³0, the rpm has straight relation with the music bpm. The class protocol followed the specifications of the sports continuous training³¹with intensity at 65% of $\dot{\mathbf{V}}\mathrm{O}_{2\text{submaximal}}$ for all subjects in the TCG. The OMNI RES scale (subjective perception scale for resistance exercise) for cycling was used for intensity maintenance for the whole time of the exercise³².

Protocol of strength training

The strength training session was performed immediately after the indoor cycling session in bodybuilding machines rand name Biotech®. The program consisted of three bouts, 65% of 1RM load for each of the proposed exercises, with 20 repetitions and interval of one to two minutes between sets for recovery of the worked muscle group³¹, since, according to Kawakami *et al.*³³, the isometric muscular activities repeated at high intensity may produce decrease in strength with time. The strength training protocol (ST) was composed of seven exercises for up-

per and lower limbs, in the following order: supported row, leg press 45°, bench press (long bar), knee extension(extensor chair), straight bar curl (short bar), knee flexion (flexor chair) and triceps extension (high pulley). The session had approximate duration of one hour and 10 minutes.

Protocol for blood collection

The volunteers were asked to arrive at the collection place on the set day at eight o'clock in the morning, on 12-hour absolute fast to better evaluate the basal serum leptin and cortisol levels of the subjects of TCG and C groups at absolute fast. The 20 blood samples of leptin and cortisol of both groups were collected by the Sérgio Franco Medicina Diagnóstica Laboratory, Rio de Janeiro, in an itinerant laboratory in the Westfit health club. Immediately after the end of the training session, the collection of more than 20 blood samples of leptin and cortisol was performed in both groups at the same conditions and places described above. The method used by the laboratory for the analysis of the leptin samples was the radioimmunoassay, where the reference values are: up to 18.5ng/mL in women and up to 9.2ng/mL in men. The method used by the laboratory for the cortisol samples analysis was the immunoenzimatic assay by chemiluminescence, which has reference values of 5.0 to 25.0mcg/dL, in the seven to nine o'clock in the morning period.

STATISTICAL ANALYSIS

All statistical procedures were processed in the *Statistical Package for the Social Sciences* program (SPSS 14.0, Chicago, USA). The sample characterization of the population was investigated for applying the described echniques. In order to obtain a description of the collected data, mean measure was used, which is a central tendency measure, and standard deviation for the dispersion measures. Inferential analysis of the study used two-way ANOVA in order to determine differences between the groups at different moments. The sample homogeneity and the results were analyzed by the Shapiro Wilk test, and for sample normality the Tukey post hoc test was used. The significan celeveladopted was of p < 0.05.

RESULTS

Table 1 presents the descriptive results of the anthropometric variables of the TCG and C groups, as well as the normality analysis of the sample through the Shapiro-Wilktest (SW). Eachgroup was composed of 10 subjects.

It can be analyzed that all groups presented normal distribution or all the analyzed variables. Kurtosis was also performed to guarantee homogeneity of the sample. BMI between 26.84 and 27.5 was observed, which lets classify them as with overweight.

Table 2 presents the descriptive results of the leptin serum level of the training competitor group (TCG) and control group (C) at the pre and post-intervention moments. Normality was also verified through the SW test.

When the mean values of the first and second leptin collection, it was observed that the TCG group presented values of 14.53ng/mL \pm 6.79 and 13.09ng/mL \pm 8.46, and was within the reference mean thresholds expected for plasma leptin (until 13.85ng/mL \pm 6.57 for women and men), while the C group which obtained values

of 27.09ng/mL \pm 10.70 and 21.49ng/mL \pm 9.44, these were much higher than the mean reference thresholds.

Table 3 presents the descriptive results of the cortisol serum level in two moments (pre and post-intervention).

The descriptive analysis of the plasma cortisol samples of the TCG and C groups demonstrated that the values obtained in the first and second collections of both groups were within the mean values of 15.0 ± 14.14 mcg/dL for the seven to nine o'clock time.

Table 1. Descriptive results of the anthropometric variables of the two groups.

	Age (years)	Weight (kg)	Height (m)	BMI (kg.m ⁻²)	WHI (cm)
TCG (10)	27.9 ± 4.97	76.9 ± 17.9	1.67 ± 0.14	26.84 ± 1.51	74.5 ± 4.30
C (10)	27.5 ± 5.48	80.1 ± 14.6	1.70 ± 0.12	27.5 ± 5.48	76 ± 4.34
Kurtosis	-1.302	-0.768	-0.882	-0.641	1.631
SW (20)	0.594	0.579	0.464	0.631	

 \overline{TCG} = training competitior group; C = control overweight group; SW = Shapiro-Wilk; BMI = body mass index; WHI = waist/hip index.

Table 2. Descriptive results of the serum level of leptin of the two groups at the pre and post-intervention moments.

LEPTIN (ng/mL)				
	TCG		С	
	Pre	Post	Pre	Post
Mean	14.53	13.09	27.09	21.49
Standard devia- tion	6.79	8.46	10.70	9.44
Standard error	2.142	2.677	3.39	2.99
VC (%)	43.15	60.39	39.50	43.93
Kurtosis	-1.565	3.274	-1.092	-0.860
Minimum value	5.80	4.20	12.90	8.50
Maximum value	24.30	33.30	42.30	35.90

TCG = training competitor group; C = control overweight group; SD = standard deviation; SE = standard error; VC = variation coefficient.

Table 3. Descriptive results of the serum level of cortisol of the two groups in the pre and post-intervention moments.

Cortisol (mcg/ dL)				
	TCG		С	
	Pre	Post	Pre	Post
Mean	18.1	11.86	13.77	7.06
SD	5.9	6.74	2.23	2.58
SE	1.868	2.133	0.71	0.82
VC (%)	32.60	56.83	16.19	36.54
Kurtosis	-0.093	2.345	-0.477	4.015
Minimum value	11.1	5.9	10.3	4.5
Maximum value	29.5	27.4	17.1	13.4

TCG = training competitor group, C = control overweight group; SD = standard deviation; SE = standard error; VC = variation coefficient.

Table 4 presents the leptin statistical values for the TCG and C groups. The data presented significant difference when the groups were compared to the preand post-intervention moments.

Intergroup significant difference has been observed (TCG x C) in the pre-intervention (p = 0.02) and post-intervention moments (p = 0.01). In the intragroup analysis significant alterations have not been observed.

Table 5 presents the cortisol statistical values for the TCG and C groups. In the intergroup analysis (TCG x C), significant difference was observed at the pre-intervention (p = 0.01) and post-intervention moments (p = 0.01). In the intragroup analysis significant alterations have not been observed.

Table 4. Inferential analysis of the leptin levels of the TCG and C groups in the pre and post-training periods.

Leptin				
	TCG – Pre	TCG – Post	C – Pre	C – Post
TCG – Pre	=	0.98	0.02*	0.32
TCG – Post	0.98	-	0.01*	0.17
C – Pre	0.02*	0.01*	-	0.51
C – Post	0.32	0.17	0.51	-

^{*}significance (p < 0.05).

Table 5. Statistical analysis of the levels of cortisol of the TCG and C groups in the pre and post-training periods.

Cortisol				
	TCG – Pre	TCG – Post	C – Pre	C – Post
TCG – Pre	-	0.01*	0.186	0.00
TCG – Post	0.01*	=	0.620	0.246
C – Pre	0.19	0.62	-	0.02*
C – Post	0.00	0.25	0.02*	-

^{*}significance (p < 0.05).

DISCUSSION

According to the revision study by Kraemer *et al.*¹², many studies which have investigated the effects of the acute exercise on leptin showed reduction or no alteration in the concentrations. Steinberg *et al.*³⁴ and Jen *et al.*³⁵ stated that the articles published have demonstrated that the leptin plasma levels are reduced after physical training in animals.

In the study by Estadella*et al.*³⁶, the found results verified that endurance training in rats would avoid increase of leptin plasma levels induced by a hyperlipidic diet, and that decrease in the leptin levels was also followed by lower body weight and adipose mass gain and lower levels of serum triglycerides and insulin.

Kraemer *et al.*¹² state that many studies say that the leptin plasma levels are not altered due to acute aerobic exercise in athletes or non-athletes. However, the decrease of these levels may be connected to the time of leptin collection after the exercise session, while leptin is directly or indirectly related to the control of the energy balance in the long run. Besides being an important lipostate (measurer of the lipid buildupof the body), leptin increases lipolysis and lipidic oxidation³⁷.

The research by Rosa et al.³⁸ investigated the effects of the con-

current training on the leptin concentrations in young adults in overweight state. Their results pointed that there was reduction of the variable levels. Such decrease in the leptin concentrations do not seem to depend on the performance order of the concurrent training, since in the studies by Rosa *et al.*^{39,40}, the used protocols caused reduction in the concentrations of this hormone regardless of the performance order.

In the present study, leptin presented significant difference (p = 0.02) when the pre-moments of the groups were compared, and in mean values, the C group was higher (27.09 \pm 10.70). The two-way ANOVA results pointed the existence of statistically significant difference (p = 0.01) in the comparison of the pre moment of the C group with the post moment of the TCG group.

In the study by Bouassida *et al.*⁴¹, the leptin behavior was evaluated in trained and untrained individuals after exercise in cycle ergometer. The protocol consisted of two exercise periods: 45 min and 85 min. The leptin samples were collected before, during, at the end, after two hours and 24 hours of recovery from each protocol. It was verified that leptin did not significantly alter during both protocols for both groups, but was lower (p < 0.05) in all samples in the trained group when compared with the untrained group.

In our research, the leptin mean values demonstrated slight decrease in both groups (TCG: 14.53 ± 6.79 to 13.09 ± 8.46 ; C: 27.09 ± 10.70 to 21.49 ± 9.44) after the used training protocol. Such findings corroborate Essig *et al.*⁴², who state that the exercise effects with period equal or longer than 60 minutes usually do not reveal any decrease or slight decrease in the leptin levels post-exercise, and Ribeiro *et al.*¹¹, mentioning that the report that acute or chronic physical exercise has been commonly causing decrease in the leptin concentrations.

However, according to Fisher *et al.*⁴³,the studies suggest that exercise practiced in an acute manner (< 60 min) does not present acute effect on the leptin production, regardless of the exercise intensity in healthy men and women and that the reduction found may be attributed to the circadian rhythm or hemo concentration. Hulverand Houward⁴⁴ also questioned whether exercise per se causes this reduction or if body fat decrease, the typical chronic response to physical training or the energetic imbalance created would not be responsible for this decrease.

Essig *et al.*⁴², in their study, also found low leptin concentration in trained men after 48 hours of exercise, but not immediately after exercise and 24 hours after it. It was two separated years of treadmill running – 800kcal and 1,500kcal. The authors speculate that the alterations in the leptin stimulus, as well as the alterations in the inhibitors, may have affected the leptin concentrations.

Longer exercise duration has also been widely discussed 12 . The study performed by Keller *et al.* 45 tried to observe the leptin behavior after three hours of exercise in cycle ergometer. It was observed that the leptin concentration decreased after this period. IN the study by Landt *et al.* 46 , reduction of 8% in the fasting leptin was observed followed by two hours of exercise in cycle ergometer at 75% $\dot{\mathbf{V}}\mathbf{O}_{2max}$ and ended with five chunks of oneminute sprints. The same authors observed in another study that the leptin concentration before and after exercise of a group of ultramarathon runners presented decrease of 32% after mean of 35 hours of continuous exercise, which, according to the authors,

suggests that the most remarkable decrease occurred due to the energy imbalance and that leptin may serve as an important sign of this imbalance, but only in extreme situations.

Despite the found results, some studies did not observe any alteration in the leptina concentration, such as the study by Racette *et al.*⁴⁷, who measured the difference of arteriovenousleptin concentration after 60 minutes of exercise on ergometric bicycle and a study by Nindl*et al.*⁴⁸, where exercise with weights was applied in physically active individuals and the decrease of the leptin levels occurred after only nine hours of post-exercise. In the present study there was no significant difference in the comparison of the leptin concentration in the pre and post in the two groups (TCG – p = 0.98; C – p = 0.51). According to the obtained results, it can be observed that there was no significant difference in the analyzed variable after the intervention protocol.

The study by Cruz *et al.*⁴⁹ did not demonstrate significant alterations in the leptin concentrations after a protocolof concurrent training with characteristics of modality, intensity and duration similar to the ones used in the present study, in which significant difference was not observed in the leptin levels of the participants either.

Due to its nature of fast response to many kinds of stress, which usually occurs within some minutes, cortisol points strength exercises as powerful promoters to reach fast increase of its concentrations in the blood stream²⁰, also due to its characteristic to promote adaptative microtrauma. The influence of external factors may also modify the cortisol response when in submaximal exerce, which may be extremely remarkable¹⁹.

However, if exercise is below the critical threshold intensity, the cortisol levels may not increase above the rest levels, or may really become reduced²³, corroborating Viru *apud* Hackney¹⁹, who also state that in the same exercise absolute intensity cortisol may present low response from the practice of an exercise training program. Never the less, if submaximal exercise is above the critical threshold intensity, then the cortisol levels will initially increase and subsequently enter in plateau (steady state exercise)²⁴.

Many investigations ^{17,23,25,50-53} which propsoed to analyze the exercise effect on the plasma cortisol levels pointed significant increase during and after exercise. According to Viruand Viru⁵⁴, cortisol increase observed during exercise is crucial to the control and regulation of the energetic metabolism and hence, to exercise performance.

Some investigations⁵⁵⁻⁵⁷reported reduction in the plasma cortisol levels, but these results seem to be associated with the chronic effect of training⁵⁸, seen that the strength training routine applied in these studies had duration between 12 and 24 weeks. The investigation by Rosa *et al.*⁵⁹ had as aim to analyze the serum cortisol behavior after an acute session of the concurrent training protocol (indoor cycling and bodybuilding, 5-7 on the OMNI RES scale and 85% 1RM, respectively). The obtained result was significant decrease of serum cortisol, in the experimental group and in the control group. But the authors describe that this reduction may be influenced by the cortisol circadian cycle.

Nevertheless, in the present study, the acute response of the concurrent training resulted in significant decrease of the cortisol descriptive mean values (table 2) when the pre and post-training

moments of the TCG and C groups were compared. The mean of the groups was close to the mean reference value for adults (15.0 \pm 14.14mcg/dL), in the period between seven and nine o'clock in the morning. The inferential analysis (table 3) showed that there was only significant difference in the intergroup comparison of the pre and post moments in the TCG group (p = 0.01) and in the pre and post moments of the C group (p = 0.01). Based on the citation by Hill et al.²³, who suppose that decrease in the cortisol values of the present study may be associated with the intensity used in the exercise, in the current study the intensity applied to the volunteers was of 65% \dot{V} O_{2submaximal}, which does not seem to have been sufficient to cause the expected increase. Such findings corroborate Rudolph and McAuley⁶⁰, who state that the cortisol levels are linearly related t the exercise intensity and duration. In the comparison between groups, there was not statistically significant difference in any moment.

The interval between bouts, according to Hackney⁶¹, also seems to play an important role in the significant increase of plasma cortisol after an acute strength training session. The study by Kraemer *et al.*⁶² tried to analyze the cortisol acute response after eight bouts of 10RM in leg press with interval times of one minute and three minutes between bouts, and recorded that, with one- minute interval, there was significant increase compared with the three minutes for the same exercise protocol. In the present study, the bodybuilding protocol followed an interval time of one to two maximal minutes for three bouts of 20 repetitions, according to the ACSM guidelines³³, differently from the study by Kraemer *et al.*⁶², which could have been another factor which led to cortisol increase besides the number of bouts and repetitions performed.

Foschini and Prestes⁵³ mentioned studies which used number of bouts and repetitions similar to the ones used by Kraemer *et al.*⁶² and which presented increase in the plasma cortisol concentration.

However, some studies $^{35-65-67}$ did not find differences in plasma cortisol levels after exercise. Grandys $et~al.^{56}$, for example, analyzed the plasma cortisol between other hormones before and after five weeks of endurance training in cycle ergometer – in the continuous method (2x/week at 90% $\dot{V}O_2$) and intermittent method (2x/week at 50% $\dot{V}O_2$) – on Mondays and Thursdays, and at the end no alterations in the plasma cortisol were found.

The results presented in the current research demonstrate that a single session of the concurrent training protocol used in the present study was not able to generate the necessary stress to increase the plasma cortisol secretion in sufficient quantity to promote increase of the serum leptin levels in overweighed young adults.

CONCLUSIONS

The results presented in the present research demonstrate that a single session of the concurrent training program used in this study was not able to generate the necessary stress to increase the plasma cortisol secretion in sufficient quantity to promote increase in the serum leptin levels in the evaluated individuals.

All authors have declared there is not any potential conflict of interest concerning this article.

REFERENCES

- Jarosz M, Rychlik E. Overweight and obesity among adults in Poland, 1983-2005. Adv Med Sci 2008;53:158-66.
- Barreto y Glória, ACL. Efeito do exercício resistido executado em diferentes velocidades nas aulas de ginástica localizada sobre o coeficiente respiratório (R) e o excesso de consumo de oxigênio consumido (EPOC) pós-exercício. Dissertacão de Mestrado. Rio de Janeiro: Universidade Castelo Branco; 2006.
- 3. Mota G, Zanesco A. Leptin, ghrelin, and physical exercise. Arq Bras Endocrinol Metab 2007;51:25-33.
- Zhang Y, Proenca R, Maffei M, Barone M, Leopold L, Friedman J. Positional cloning of the mouse obese gene and its human homologue. Nature 1994;372:425-32.
- Mistry A, Swick A, Romsos D. Leptin rapidly lowers food intake and elevates metabolic rates in lean and ob/ob mice. J. Nutr 1997;127:2065-72.
- Maugeri D, Bonanno M, Speciale S, Santangelo A, Lentini A, Russo M, et al. The leptin, a new hormone
 of adipose tissue: clinical findings and perspectives in geriatrics. Arch Gerontol Geriatr 2002;34:47-54.
- Fors H, Matsuoka H, Bosaeus I, Rosberg S, Wikland K, Bjarnason R. Serum Leptin Levels Correlate with Growth Hormone Secretion and Body Fat in Children. J Clin Endocrinol Metab 1999:84:3586-90
- Lee JH, Reed DR, Price RA. Leptin resistance is associated with extreme obesity and aggregates in families. Int J Obes Relat Metab Disord 2001;25:1471-3.
- Negrão A, Licinio J. Leptina: o diálogo entre adipócitos e neurônios. Arq Bras Endocrinol Metab 2000:44:205-14.
- 10. Bray G. Progress in understanding the genetics of obesity. J Nutr 1997;127:940S-2S.
- Ribeiro S, Santos Z, Silva R, Louzada E, Donato Junior J, Tirapegui J. Leptin: aspects on energetic balance, physical exercise and athletic amenorhea. Arg Bras Endocrinol Metab 2007;51:11-24.
- 12. Kraemer R, Chu H, Castracane V. Leptin and exercise. Exp Biol Med (Maywood) 2002;227:701-8.
- 13. Weltman A, Pritzlaff C, Wideman L, Considine R, Fryburg D, Gutgesell M, et al. Intensity of acute exercise does not affect serum leptin concentrations in young men. Med Sci Sports Exerc 2000;32:1556-61.
- Tuominen J, Ebeling P, Laquier F, Heiman M, Stephens T, Koivisto V. Serum leptin concentration and fuel homeostasis in healthy man. Eur J Clin Invest 1997;27:206-11.
- Olive J, Miller G. Differential effects of maximal-and moderate-intensity runs on plasma leptin in healthy trained subjects. Nutrition 2001;17:365-9.
- Veldhuis J, Iranmanesh A, Lizarralde G, Johnson M. Amplitude modulation of a burstlike mode of cortisol secretion subserves the circadian glucocorticoid rhythm. Am J Physiol 1989;257:E6-14.
- Kanaley J, Weltman J, Pieper K, Weltman A, Hartman M. Cortisol and growth hormone responses to exercise at different times of day. J Clin Endocrinol Metab 2001;86:2881-9.
- Duclos M, Guinot M, Le Bouc Y. Cortisol and GH: odd and controversial ideas. Appl Physiol Nutr Metab 2007;32:895-903
- 19. Hackney A. Exercise as a stressor to the human neuroendocrine system. Medicina (Kaunas) 2006;42:788-97.
- Smith LL. Cytokine hypothesis of overtraining: a physiological adaptation to excessive stress? Med Sci Sports Exerc 2000;32:317-31.
- 21. Viru A. Plasma hormones and physical exercise. Int J Sports Med 1992;13:201-9.
- Hackney A, Viru A. Research methodology: endocrinologic measurements in exercise science and sports medicine. J Athl Train 2008;43:631-9.
- 23. Hill E, Zack E, Battaglini C, Viru M, Viru A, Hackney A. Exercise and circulating cortisol levels: the intensity threshold effect. J Endocrinol Invest 2008;31:587-91.
- 24. Viru A, Viru M. Metabolic adaptation in training. Biochemical monitoring of sport training Champain: Human Kinetics. 2001:11-25.
- Daly W, Seegers C, Timmerman S, Hackney A. Peak cortisol response to exhausting exercise: effect of blood sampling schedule. Med Sport 2004;8:17-20.
- 26. Fleck SJ, Kraemer WJ (editor). Fundamentals of Muscular Strength Training.: Editora Artes Médicas; 2006.
- 27. Thomas JR, Nelson JK, Silverman SJ (editor). Métodos de Pesquisa em Atividade Física. 5ª ed.; 2007.
- 28. Marfell-Jones M, Olds T, Stewart A, Carter L (editor). International standards for anthropometric assessment (2006) ISAK. 1ª ed. Potchefstroom, South Africa: Int. Soc. for the Adv. Kinanthropometry; 2006.
- 29. Baechle T, Earle R (editor). Essentials of strength training and conditioning: Human Kinetics; 2008.
- 30. Mello DB, editor. Ciclismo Indoor. 1ª ed. Rio de Janeiro: Editora Sprint; 2004.
- Kraemer WJ, Adams K, Cafarelli E, Dudley GA, Dooly C, Feigenbaum MS, et al. American College
 of Sports Medicine position stand. Progression Models in Resistance Training for Healthy Adults.
 Med Sci Sports Exerc 2002;34:364-80.
- Robertson R, Goss F, Dubé J, Rutkowski J, Dupain M, Brennan C, et al. Validation of the Adult OMNI Scale of Perceived Exertion for Cycle Ergometer Exercise. Med Sci Sports Exerc 2004;36:102-8.
- Kawakami Y, Amemiya K, Kanehisa H, Ikegawa S, Fukunaga T. Fatigue responses of human triceps surae muscles during repetitive maximal isometric contractions. J App Physiol 2000;88:1969-75.
- Steinberg G, Smith A, Wormald S, Malenfant P, Collier C, Dyck D. Endurance training partially reverses dietary-induced leptin resistance in rodent skeletal muscle. Am J Physiol Endocrinol Metab 2004;286:57-63.
- Jen K, Buison A, Pellizzon M, Ordiz F, Santa Ana L, Brown J. Differential effects of fatty acids and exercise on body weight regulation and metabolism in female Wistar rats. Exp Biol Med (Maywood) 2003;228:843-9.

- Estadella D, Oyama L, Dâmaso A, Ribeiro E, Oller Do Nascimento C. Effect of palatable hyperlipidic diet on lipid metabolism of sedentary and exercised rats. Nutrition 2004;20:218-24.
- Fonseca-Alaniz M, Takada J, Alonso-Vale M, Lima F. O tecido adiposo como centro regulador do metabolismo. Ara Bras Endocrinol Metab 2006:50:216-29.
- Rosa G, Braga de Mello D, Daoud R, Cruz I, Dantas HME. Concentración de Leptina en Adultos com Sobrepeso Sujetos a un Entrenamiento Concurrente. Mot Hum 2010;10:95-102.
- Rosa G, Braga de Mello D, Dantas HME. Níveis séricos de leptina em adultos submetidos a distintas ordens de execução de treinamento concorrente. RBAFS 2011; 16:304-8.
- Rosa G, Mello D, Daoud R, Abdalla A, Antônio J, Rubia L, Dantas HME. Concentraciones Séricas de Leptina y Zinc: Efectos Agudos de las Distintas Órdenes de Ejecución del Entrenamiento Concurrente. AMD 2011:28:309-16.
- Bouassida A, Chatard JC, Chamari K, Zaouali M, Feki Y, Gharbi N, et al. Effect of energy expenditure and training status on leptin response to sub-maximal cycling. JSSM 2009;8:190-6.
- 42. Essig D, Alderson N, Ferguson M, Bartoli W, Durstine J. Delayed effects of exercise on the plasma leptin concentration. Metabolism 2000;49:395-9.
- 43. Fisher J, Van Pelt R, Zinder O, Landt M, Kohrt W. Acute exercise effect on postabsorptive serum leptin. J App Physiol 2001;91:680-6.
- 44. Hulver MW, Houward JA, Plasma Leptin and Exercise: Recent Findings, Sports Medicine 2003;33:473.
- Keller P, Keller C, Steensberg A, Robinson L, Pedersen B. Leptin gene expression and systemic levels in healthy men: effect of exercise, carbohydrate, interleukin-6, and epinephrine. J App Physiol 2005;98:1805-12
- Landt M, Lawson G, Helgeson J, Davila-Roman V, Ladenson J, Jaffe A, et al. Prolonged exercise decreases serum leptin concentrations. Metabolism 1997;46:1109-12.
- 47. Racette S, Coppack S, Landt M, Klein S. Leptin Production during Moderate-Intensity Aerobic Exercise.

 J Clin Endocrinol Metab 1997;82:2275-7.
- 48. Nindl BC, Kraemer WJ, Arciero PJ, Samatallee N, Leone CD, Mayo MF, et al. Leptin concentrations experience a delayed reduction after resistance exercise in men. Med Sci Sports Exerc 2002;34:608.
- Cruz I, Rosa G, Fortes MSR, Mello DB, Dantas EHM. Effect of concurrent training on serum leptin concentrations in overweigth young adults. ISMJ 2012 [in press].
- Cadore E, Brentano M, Lhullier F, Silva E, Spinelli R, Kruel L. Hormonal concentrations at rest and induced by a superset strength training session in long-term strength-trained and untrained middle-aged men.. In: Science BoACoECoS (ed.). Annual Congress of Euro. Col. Sports Science. Vol. 10. 2005;104-05.
- Dagogo-Jack S, Tykodi G, Umamaheswaran I. Inhibition of Cortisol Biosynthesis Decreases Circulating Leptin Levels in Obese Humans. J Clin Endocrinol Metab 2005;90:5333-35.
- Daly W, Seegers C, Rubin D, Dobridge J, Hackney A. Relationship between stress hormones and testosterone with prolonged endurance exercise. Eur J Appl Physiol 2005;93:375-80.
- Foschini D, Prestes, J. Acute hormonal and immune responses after a bi-set strength training. Fit Perf J 2007;6:38-43.
- 54. Viru A, Viru M. Cortisol Essential adaptation hormone in exercise. Int J Sports Med 2004;25:461-4.
- França S, Neto T, Agresta M, Lotufo R, Kater C. Resposta divergente da testosterona e do cortisol séricos em atletas masculinos após uma corrida de maratona. Arq. Bras endocrinol. metab;50:1082-87.
- Grandys M, Majerczak J, Duda K, Zapart-Bukowska J, Sztefko K, Zoladz J. The effect of endurance training on muscle strength in young, healthy men in relation to hormonal to hormonal status. J Physiol Pharmacol 2008;59:89-103.
- 57. Häkkinen K, Pakarinen A, Alén M, Komi P. Serum hormones during prolonged training of neuromuscular performance. Eur J Appl Physiol Occup Physiol 1985;53:287-93.
- Marx JO, Ratamess NA, Nindl BC, Gotshalk LA, Volek JS, Dohl K, et al. Low-volume circuit versus highvolume periodized resistance training in women. Med Sci Sports Exerc 2001;33:635-43.
- Rosa G, Braga de Mello D, Biehl C, Dantas HME. Níveis de Cortisol em Adultos com Sobrepeso Submetidos a Treinamento Concorrente. BJSER 2010;1:11-15
- 60. Rudolph D, McAuley E. Cortisol and affective responses to exercise. J Sports Sci 1998;16:121-8.
- 61. Hackney A. Exercise as a stressor to the human neuroendocrine system. Medicina (Kaunas) 2006;42:788-97.
- Kraemer WJ, Koziris LP, Ratamess NA, Hakkinen K, Triplett-McBride NT, Fry AC, et al. Detraining produces minimal changes in physical performance and hormonal variables in recreationally strengthtrained men. J Strength Cond Res 2002;16:373-82.
- Mccall G, Byrnes W, Fleck S, Dickinson A, Kraemer W. Acute and chronic hormonal responses to resistance training designed to promote muscle hypertrophy. Can J Appl Physiol 1999;24:96-107.
- Fleck SJ, Kraemer WJ (editors). Fundamentos de Treinamento de Força Muscular. 3ª ed.: Editora Artes Médicas; 2006.
- Fry AC, Kraemer WJ, Stone MH, Warren BJ, Fleck SJ, Kearney JT, et al. Endocrine responses to overreaching before and after 1 year of weightlifting. Can J Appl Physiol 1994;19:400-10.
- Häkkinen K, Pakarinen A, Kallinen M. Neuromuscular adaptations and serum hormones in women during short-term intensive strength training. Eur J Appl Physiol Occup Physiol 1992;64:106-11.
- Kraemer W, Clemson A, Triplett N, Bush J, Newton R, Lynch J. The effects of plasma cortisol elevation on total and differential leukocyte counts in response to heavy-resistance exercis. Eur J Appl Physiol Occup Physiol 1996;73:93-7.