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Acute leptin response after high intensity interval and moderate intensity continuous runs

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Abstract: The possible direct role of exercise intensity and duration on leptin concentrations is conflicting. The aim of this study was to evaluate the acute effects of high intensity interval (HIIE) and moderate intensity continuous (MICE) exercise on plasma leptin response. Seven young volunteers underwent three tests: 1) a treadmill graded exercise test to identify running peak velocity (PV); 2) HIIE: 5 × 2 min work bouts at 90% of PV, interspersed by 2 min of passive recovery and; 3) MICE: 30 min at 70 % of PV. Blood samples were drawn for the assays of leptin before and 30 minutes after HIIE and MICE. A 2-way repeated measures ANOVA showed a significant main effect of time [F(1,6) =17,52; p=0,006], no significant effect of condition (type of exercise) (F(1,6) = 0,16; p = 0,68) and no significant interaction (condition × time) (F(1,6)= 0,48, p=0,51). Leptin decreased 30 min after HIIE (t= 2,95, p=0,025) and MICE (t=4,18; p=0,005). There was no difference between the HIIE and MICE conditions immediately after exercise (t=0,90; p=0,40). After HIIE and MICE, leptin decreased in the same magnitude. It appears that both exercise modalities result in physical stress which is sufficient to improve short-term leptin sensibility.

Keywords: Leptin, Exercise metabolism, Endurance Training, High Intensity Interval Training, Running.

1. Introduction

Several hormonal responses may contribute to control of energy balance, weight management, body fat distribution and inflammatory status Fisher et al. (2001). Leptin, a 167-amino-acid polypeptide hormone that is produced by adipocytes in proportion to their triglyceride content, influences food intake and control of body weight by binding to and activating the long form of its receptor in the brain Park; Ahima (2015). Evaluating the time course of leptin changes in distinct situations provides an indirect estimative of its influence on human metabolism. Serum leptin falls dramatically with short-term fasting (in the absence of weight loss) Weigle et al. (1997) and is elevated as a result of 1 day of massive overfeeding Kolaczynski et al. (1996). Leptin action is required for energy stores to be sensed in the central nervous system (CNS), is essential for normal energy homeostasis and its major physiological role is to respond to and defend against reductions of body fat, that might impair survival and reproductive fitness Myers et al. (2010). Thus, the coexistence of elevated leptin levels with obesity is considered a strong evidence of "leptin resistance" Myers et al. (2010 e Park; Ahima (2015).

The effects of exercise, independently of loss of fat mass, have not been clearly established and reports on leptin response to exercise in

humans have been conflicting Guerra et al. (2011 e Weltman et al. (2000). Perusse et al., (1997) showed considerable interindividual differences in the leptin response to acute and chronic effects of aerobic exercise, some individuals showing either increase or reduction in leptin, others showing almost no change. Weltman et al., (2000) demonstrated that continuous exercise for 30 min, at intensities ranging from well below to well above the lactate threshold with exercise caloric expenditure ranging from 150 to 529 kcal, does not alter leptin release either during exercise itself or during 3.5 h of recovery thereafter. Other investigators have studied the effects of continuous, moderate-intensity exercise on leptin concentrations and have found either no exercise-related change in circulating leptin Perusse et al. (1997) or an exercise-related decrease in blood leptin Jürimäe; Jürimäe (2005 e Zaccaria et al. (2013).

In most of the above-stated studies, the primary training stimulus was endurance exercise. Few data exist on the impact of high intensity, intermittent exercise, which characterizes many sports match plays and recreational activities. The influence of different exercise modalities, volumes and intensities on acute leptin response is less well characterized. Earlier evidence seemed to indicate that an emerging time efficient exercise modality for health promotion is the high intensity interval exercise (HIIE) Talanian et al. (2007 e Trapp et al. (2008) and because of that, studies investigated the effects of HIIE on acute metabolic and hormonal responses Engel et al. (2014 e Hackney et al. (2012) . Leptin levels were decreased in obese males when a HIIE was performed in a 1:1 "effort-recovery" ratio with 10 × 1 min work bouts at 90% of their individual peak velocity reached on the maximal graded exercise test, interspersed by 1 min of active recovery at 30% of peak velocity. When young lean sedentary male subjects completed four intense 5 min cycling periods of 76% VO_{2max} interspersed by 3 min of 43% of VO_{2max} serum leptin was increased at the start of exercise and decreased at the end Fisher et al. (2001).

Although a great deal is known about acute leptin responses after moderate intensity continuous cycling in sedentary and obese subjects, little is known about the effects of a single bout of high intensity running in healthy subjects and which is the leptin response under different "effort-recovery" ratios. Besides that, leptin response under different intensities and durations of moderate continuous running have not been clearly established. With all of this in mind, we sought to examine the influence of high-intensity interval running in comparison to submaximal, steady-state endurance running on the serum leptin responses of healthy college students 30 min after exercise.

2. Materials and Methods

Subjects

Seven (5 men and 2 women), healthy, nonsmoking college students were recruited. All subjects marked "no" to all questions of the Physical Activity Readiness Questionnaire (PAR-Q), therefore, they were considered healthy and at very low risk of performing high intensity exercise. They were all familiar with the procedure of maximal exercise testing and either performed regular exercise on a noncompeting level (less than 5 h of sport and/or resistance training a week). All subjects signed informed consent forms and procedures were approved by the University of Southern Santa Catarina State Review Board (CAAE: 09153418.1.0000.5369). Subjects characteristics are described in Table 1.

Design

Each volunteer completed a graded treadmill test to assess peak velocity and anthropometric measures at the gym of the University of Southern Santa Catarina State. Subjects were then evaluated on two separate occasions and the order of the two study conditions was assigned in a randomized fashion. The admissions were scheduled at least 48 hs apart. During the 48 hours before the testing sessions, the subjects were instructed to avoid strenuous training sessions and caffeine ingestion during 12 hours before. All the tests were performed in

the first semester of 2018, in the morning, and were preceded by a day of rest. The subjects refrained from high-intensity exercise for 24 hours before the testing sessions. During the testing period, the subjects were asked to consume their usual meal at least 3 hours before the scheduled testing time.

Table 1. Subjects Characteristics and Maximal Graded Exercise Test results (n=7).

Variables	Mean ± SD
Age (years)	22,5 ± 3,46
Body Mass (kg)	67,3 ± 9,51
Heigh (cm)	179 ± 6,3
BMI (kg/m^2)	22,5 ± 1,83
HRpeak (bpm)	193 ±3,29
PV (km.h^{-1})	14,9 ±1,43
RPEmáx	10 ± 0
70% PV(km.h^{-1})	10, 43 ±1,07
90% PV(km.h^{-1})	13,41 ± 1,35

BMI: body mass index; PV: Peak Velocity at maximal exercise test; HRpeak: Peak Heart Rate at maximal exercise test; RPEmax: Peak Rate of Perceived Exertion at maximal exercise test

Methodology

Peak Treadmill Velocity Assessment. At the first visit, body mass (nearest 0.1 kg) was measured in kg on a calibrated electronic scale. Stretch height was measured in cm using a wall-mounted stadiometer. The participants performed a warm-up on a treadmill at a speed of 7.0 km.h-1 for 6 min. Then they started the incremental test at a speed of 8.0 km.h-1 and increments of 1.0 km.h-1 every minute until voluntary exhaustion. The Peak Velocity (PV) was determined as the highest velocity subjects could maintain for a complete stage plus the interpolated velocity from incomplete stage Kuipers et al. (1985). During testing, a Polar S610I Heart Rate (HR) monitor (Polar Electro, Kempele, Finland) was fitted around the chest and used to monitor HR. Data from the last 10 seconds of each exercise stage were considered representative measurements of each stage. The highest HR value observed during the test was considered as the Heart Rate Peak (HRpeak). Rating of perceived exertion (RPE) was also monitored and recorded at the end of each minute according to the Borg scale 6–20 (Borg, 1982). The end of the test was determined by the presence of at least one of the following criteria: (i) HR ≥ 100% estimated for age; (ii) RPE > 18; or (iii) when participants voluntarily stopped.

HIIE and MICE Protocols. At the second and third visit, 2-4 days apart, all subjects completed HIIE and MICE in random order. HIIE was performed in a 2:2 "effort-recovery." The participants performed 5 × 2 min work bouts at 90% of their individual PV reached on the maximal graded exercise test, interspersed by 2 min of passive recovery (i.e., staying out of the treadmill). The MICE session consisted of 30 min at 70 % of PV performed continuously. The participants performed a 6 min warm-up at 7 km/h before both exercise sessions. HR was continuously recorded throughout the exercise sessions. Blood samples were taken before exercise (0 min) and 30 min after the end of each test. The participants remained in a seated position for 30 min and they could read and use electronic devices such as a tablet, computer, or smartphone.

Blood Samples and Leptin Analysis. For HIIE and MICE running sessions, blood was drawn from an antecubital vein and collected in 10 mL into tubes with a separator gel for serum (BD Vacutainer, New Jersey, USA) immediately before and 30 min after exercise. To separate the plasma, samples were centrifuged at the rate of 3.600 rpm for 15 min and frozen at −80◦C until blood assays were conducted. Plasma leptin concentrations (ng/ml) were measured using enzyme-linked immunosorbent assay (ELISA) kit (Immunomat, Würzburg Germany). Plasma samples were duplicated and analyzed through a spectrophotometer (Specra Max 340, Molecular Devices, Canada) and the absorbance calculation were determined (Soft Max Software, Molecular Devices, Canada).

Statistical Analysis

Statistical analyses were performed by using IBM SPSS Statistics for Windows (Chicago,

IL). ANOVA (2x2) with repeated measures on exercise protocol (2 levels) and time (2 levels) factors were used to determine the effects of exercise protocol and time, by blood sampling time interaction on serum leptin. Bonferroni's post hoc multiple comparisons were performed to locate the statistically significant differences between exercise protocols and within each protocol. Data are presented as means ± SD with a value of $P \leq 0.05$ considered statistically significance. The Cohen's d effect size to repeated measures was applied according to the equation d = {((M1 – M2)) / ((SD1 + SD2)/ 2)} Effect size values (d) were classified as: 0.0 to 0.19 = trivial; 0.20 to 0.59 = small; 0.60 to 1.19 = moderate; 1.20 to 1.99 = large; 2.00 to 4.00 = very large Hopkins et al. (2009).

The G Power 3.1.9.7 software was used to calculate a post hoc power analysis of the t tests (Erdfelder et al., 2009). The mean and standard deviation from pre vs post HIIE and the t test (difference between two dependent means – matched pairs) were used as group parameters. The power (1-β err prob) estimated was 0,78 for our sample size of 7 subjects. When the mean and standard deviation from pre vs post MICE was applied the power estimated was 0,34.

3. Results

Subjects characteristics are shown in table 1. The acute effects of the two exercise regimens on serum leptin levels are presented in table 2. The leptin levels were not significantly different before HIIE and MICE (t= -0,03, p=0.97). A significant main effect of time (before vs 30-min after) was found ($F(1,6) = 17,52$; $p = 0,005$; $\eta2 = 2,34$) and post hoc analysis indicated that leptin decreased 30 min after HIIE (t= 2,95, p=0,025) and MICE (t=4,18; p=0,005) when compared to the pre-exercise moment. No significant main effect of condition (type of exercise) was found for leptin concentration ($F(1,6) = 0,17$; $p = 0,69$; $\eta2 = 0,03$) and no interaction (condition × time) was observed ($F(1,6) = 0,48$, $p=0,513$, $\eta2 = 0,04$). There was no difference between the HIIE and MICE conditions immediately after exercise (t=0,90; p=0,40). Leptin levels decreased 37±22,6 % after HIIE and 29,4±11,8% after MICE and these changes were not significantly different between both conditions. Individual responses of leptin after both types of exercise are shown in table 3.

4. Discussion

This study investigated the effect of HIIE compared with traditional MICE on acute leptin response after exercise. The novel design of the study allowed for an investigation of leptin responses following 2 popular running exercise modalities. It appears that HIIE and MICE induced similar responses. Herein, we found that HIIE reduced the leptin levels by 37% and had a large effect size. The observed changes in leptin following HIIE are consistent with the results of previous research that has examined diverse populations. De Souza et al., (2018) investigated leptin response in sedentary obese men in three experimental sessions (HIIE: 10 × 60 s runs at 90% of the HRmax alternated by 60 s of active recovery; (2) MICE: 20 min run at 70% of the HRmax; (3) Rest—control) and found an interaction (condition × time) for leptin with a reduction immediately after HIIE compared to the pre-exercise moment. Elias et al., (2000) reported a transient decrease in serum leptin following a treadmill exercise bout to exhaustion in

Table 2. Leptin concentrations in high intensity interval exercise (HIIE) and moderate intensity continuous exercise (MICE) sessions.

Exercise	Leptin (ng/ml)		t	p	Δ (%)	Cohen's d
	Pre	Post				
HIIE	1,64 ± 0,63	0,97 ± 0,53*	2,953	0,026	37,0±22,6	1,21 (large)
MICE	1,63±0,83	1,12±0,58**	4,182	0,006	29,4±11,8	0,76 (moderate)

HIIE: High Intensity Interval Exercise; MICE: Moderate Intensity Continuous Exercise; Δ (%): Percentage change of leptin between pre and post exercise sessions.

*p<0.05; **p<0.01

Table 3. Individual responses of Leptin concentrations in high intensity interval exercise (HIIE) and moderate intensity continuous exercise (MICE) sessions.

Subject	Sex	MICE			HIIE		
		Pre (ng/ml)	Post (ng/ml)	Δ (%)	Pre (ng/ml)	Post (ng/ml)	Δ (%)
1	Female	1,70	1,20	-29,4%	2,42	0,75	-69,0%
2	Female	3,20	2,20	-31,3%	2,38	2,11	-11,3%
3	Male	0,70	0,60	-14,3%	1,14	0,70	-38,6%
4	Male	1,70	0,90	-47,1%	2,02	0,70	-65,3%
5	Male	2,00	1,60	-20,0%	1,12	0,70	-37,5%
6	Male	1,20	0,70	-41,7%	1,51	1,19	-21,2%
7	Male	0,90	0,70	-22,2%	0,86	0,70	-18,6%
Mean±SD		1,63±0,83	1,12±0,58	29,4±11,8	1,64±0,63	0,97±0,53	37±22,6

HIIE: High Intensity Interval Exercise; MICE: Moderate Intensity Continuous Exercise; Δ (%): Percentage change of leptin between pre and post exercise sessions.

healthy sedentary males when leptin concentrations were assessed 30 minutes post-exercise and later. Guerra et al., (2010) demonstrated that leptin concentration was reduced in response to a single 30 s Wingate sprint exercise by 17% and 26% (120 and 240 min after sprint exercise) in a control group and that glucose ingestion prior to exercise accentuated this response reducing leptin by 60, 69, and 65% at 30, 120, 240 min into the recovery period, respectively. Jürimäe & Jürimäe (2005) showed that concentration of leptin and growth hormone were significantly decreased and increased respectively immediately after the maximal 30 minutes of ergometer rowing and leptin concentration remained significantly reduced after 30 minutes of recovery. It is noteworthy that in this study 95.1 (2.2)% of the mean heart rate was obtained during maximal 2000 m rowing ergometer test and blood lactate was significantly increased immediately after the test (14.9 (4.3) mmol/l), which indicates a high intensity effort.

In contrast, previous studies on the effects of short-term exercise on leptin have shown no change in leptin concentration after high intensity exercise bouts. An 1-h duration, moderate intensity exercise bout (60-min endurance run at 70% VO2max) that expended 900 kcal decreased plasma concentrations of leptin, but a short-duration, maximal graded exercise test until volitional exhaustion that expended 200 kcal had no apparent effect on plasma leptin Olive; Miller (2001) even 48 h post-exercise. Larsen et al., (2019) submitted overweight, inactive men to MICE (60% peak oxygen consumption) and HIIE (60 s of work at 100% peak oxygen consumption: 240 s of rest at 50% peak oxygen consumption) bouts and found that the leptin concentration 30 minutes after exercise was not altered when compared with pre-exercise, although there was a main effect of time, with higher concentrations the morning after exercise compared with 30 min after exercise. Thus, these short-term (<60 min) exercise studies suggested that leptin production is not acutely affected by short-term exercise, regardless of exercise intensity. According to these authors, reductions or increases can be attributed to circadian rhythms, hemoconcentration, limited energy expenditure of these exercise bouts or the protocol of these studies that excluded prolonged post-exercise blood sampling (>4 hr post-exercise).

The plasma leptin further decreased after MICE by 29,4% at 30 min post-exercise and had a moderate effect size. These finding are in agreement to Duclos et al., (1999) who demonstrated the plasma leptin decreased by approximately 30% in post-exercise recovery (2 h after the end of a 2-h run) of a 65–75% VO2max run for 120 min. In the same manner, in other study Zaccaria et al. (2013) young trained males underwent a 4-h treadmill exercise at 65% of VO2max and plasma leptin levels decreased at the end of the exercise, reaching a significant reduction already after the second hour. However, our

study did not concur with Weltman et al., (2000) who demonstrated that aerobic exercise for 30 min, at intensities ranging from well below to well above the lactate threshold with exercise caloric expenditure ranging from 150 to 529 kcal, does not alter leptin release either during exercise itself or during 3.5 h of recovery thereafter.

Zaccaria et al., (2013) demonstrated that during a prolonged moderate intensity exercise, leptin decrease is significantly related to the total energy expenditure and norepinephrine concentrations seem to play an important role in the inhibition of leptin secretion. They showed a reduction only after the second hour, with a mean reduction at the end of the test of 22.5% with respect to baseline condition. During a 4-h exercise, they observed an average 7-fold increase of plasma catecholamines and a significant negative correlation between plasma leptin and norepinephrine. Other studies showed that adrenergic agonists, primarily catecholamines, have a suppressive role on leptin secretion, both in vitro and in vivo Fritsche et al. (1998 e Scriba et al. (2000), and that sympathetic blockade increases leptin expression and plasma leptin levels Sivitz et al. (1999). This indicates that endogenous activity of the sympathetic nervous system physiologically suppresses leptin expression and secretion. It is well known that for a constant exercise intensity or VO2 the plasma noradrenaline concentration continuously increases until exhaustion of the subject, whatever the intensity of the submaximal exercise Koivisto et al. (1982). The catecholamines seem to be conditioned by relative power of exercise and for a given duration, the noradrenalin concentrations increase exponentially with the intensity of exercise and this increase becomes faster beyond 75% of maximal aerobic power Christensen et al. (1979). Given the intensity of MICE and HIIE sessions in our study were 70% and 90 % of PV, respectively, it is plausible that leptin was influenced by catecholamines response in both situations.

Another possible explanation for our findings is that an exercise-induced decrease in plasma leptin could increase hunger and stimulate the replenishment of energy stores. Tuominen et al., (1997) investigated the effects of 2 hour of treadmill exercise at 75% of VO2max and showed reduced leg muscle glycogen by 32% compared with the control clamp trial, and a 34% reduction in leptin concentrations. Serum leptin concentrations were reduced by glycogen-depleting exercise and were increased during a hyper insulinemic clamp. Leptin concentrations correlated directly with serum insulin, cortisol, and triglycerides, and inversely with growth hormone concentrations which led authors to speculate that leptin is associated with factors that govern fuel homeostasis. Endurance-trained subjects exhibited a 4 fold increased plasma free fatty acids (FFA) concentration after exercise compared to the rest session with a significant negative correlation between plasma leptin and FFA concentrations (20), which suggests the possible role of lipolysis via increased plasma FFA or glycerol on leptin secretion. It is well established that during long-term exercise and a few hours after stopping running the major source of fuel is FFA. Some authors Williams et al. (2013) demonstrated that plasma FFA was significantly elevated at 60 min after MICE (65% VO2peak for 60 min) and plasma glycerol concentrations were significantly elevated after both HIIE (4 x 30 s Wingates separated by 4.5 min of active rest) and MICE. They also showed plasma norepinephrine concentrations were significantly elevated compared with MICE following HIIE. Additionally, the reduction in leptin concentration during fasting was shown to be correlated with parameters that reflect decreased glucose availability and increased lipolysis Dubuc et al. (1998). As such, given the non-highly trained state of the subjects from our research, the HIIE and MICE protocol may have exacerbated the physiological responses in both sessions and it is plausible that participants experienced glycogen depletion and increased norepinephrine, glycerol and FFA, which may have led to leptin reduction 30 minutes after HIIE and MICE in a similar degree.

It was speculated that circulating leptin levels are only decreased by bouts of exercise with considerably high intensity and long duration and in order to alter leptin, a threshold of energy deficit must be achieved Salbe et al. (1997 e Weigle et al. (1997 e Zaccaria et al. (2013). Plasma leptin concentrations are modulated by energy balance as circulating leptin is decreased and increased in response to fasting and overfeeding, respectively Aggel-Leijssen et al. (1999). These findings rise the assumption that exercise-associated reductions in leptin may be due to alterations in nutrient availability or nutrient flux at the level of adipocytes, the primary site of leptin production and secretion. On the other hand, it is advocated that the decrease in leptin concentration that is caused by an exercise bout could increase the effectiveness of leptin receptors through binding to specific leptin receptors located throughout the central nervous system (CNS). For instance, many obese individuals are resistant to leptin's actions, which is hypothesized to result from an insensitivity of receptors to leptin because of hyperleptinemia Park; Ahima (2015), similar to non–insulin-dependent diabetics who are insensitive to insulin. Thus, it gives some indication that the exercise bout may have a significant effect on plasma leptin levels independent of the effects of exercise on energy balance allowing the leptin to function in a typical manner Olive; Miller (2001). Reinforcing these arguments, examined changes in leptin signaling cascades in human skeletal muscle after a single 30 s Wingate sprint exercise and showed that cycling sprint performed under fasting conditions stimulated signal transducer and signaling cascades activated by leptin in skeletal muscles. Increased activator of transcription 3 (STAT3), stimulated signal transducer and activator of transcription 5 (STAT5), extracellular signal-regulated kinase (ERK) phosphorylation and an increased 3.2-fold peroxisome proliferator-activated receptor- γ coactivator-1α (PGC-1α) mRNA content were observed. These authors suggested that sprint exercise behave as a leptin mimetic and could be used to stimulate the leptin signaling pathways in human skeletal muscle. It has been shown that a single bout of sprint exercise activates AMP-activated protein kinase (AMPK), which is also activated by leptin in skeletal muscle Shiver et al. (2002). These authors suggested that high intensity and/or sprint exercise might be used to avoid leptin resistance in humans and may lead to increased leptin sensitivity, whereas obese humans have reduced leptin receptors in their skeletal muscle Guerra et al. (2010).

To our knowledge, ours is the first study to examine the effects of intense, intermittent treadmill running on circulating leptin levels during recovery in healthy subjects. The present data also support the suggestions that not only relatively chronic changes in energy expenditure are required to reduce leptin production and/or clearance and/or that extremes of exercise-induced negative energy balance (such as the energy expenditure associated with the prolonged exercise) are required to lower serum leptin levels. The percentage of PV was chosen as the criterion for exercise intensity based on previous data that suggest that release of both catecholamines and GH is stimulated at exercise intensities above the anaerobic threshold, which can occurs in intensities ranging from 50-90% VO2max Binder et al. (2008 e Faude et al. (2009) depending on training status and population . Thus, MICE performed at 70% PV might be closed to anaerobic threshold and allowed subject to run for 30 minutes. The HIIE at 90% PV was applied to represent an intensity that cannot be maintained for a longer period without continuous rise in blood lactate and match an increase in blood catecholamine concentrations along the 5 running bouts. These physiological responses, like other studies cited above, could have stimulated the leptin signaling pathways in human skeletal muscle and improved its sensitivity.

However, this study did not have a control for factors such as differences in diet or exercise between individuals. Decreases in plasma leptin following a single exercise bout

have manifested themselves as a late 24-48hs hours post-exercise (Fisher et al.,2001), thus the time of blood sampling relative to exercise bouts may be a contributing factor to some disparate findings and results. The plasma leptin was obtained before starting and 30 min after the end of the exercise. However, in other studies (De Souza et al., 2018) the sample was obtained immediately after the session, and 60 min after the end of the session. We intended to investigate a faster leptin response after exercise and a possible sensitivity, caused by different intensities and durations of treadmill runs.

It is not clear from this study if the exercise bout caused the reductions in plasma leptin levels or if it was a result of changes in energy balance. Further, the small sample size (n=7), with men and women, leads to low power into the analyses (from 0,34 to 0,78 1-β error probabilities). A control trial was not conducted in this study to determine whether diurnal changes accounted for observed reductions in leptin. All subjects were healthy, active men and women thus, the findings cannot be generalized to other populations. Lastly, our study was not able to indicate other endocrine responses that could be associated with leptin responses during and after exercise.

In summary, the present study supports the very limited data that exist and support the notion that HIIE and MICE decreases the plasma leptin concentration 30 min after exercise. Specifically, we have shown that moderate intensity continuous and high-intensity interval runs, in healthy adults, is also reflected by a similar degree (%) of leptin concentration decrease. Based upon to our data, the slower leptin after exercise could stem primarily from an augmented catecholamine response, enhanced fat utilization and rapid need for replenishment of energy stores. Therefore, it appears that both exercise modalities result in physical stress which is sufficient to improve short-term leptin sensibility.

Supplementary Materials: The following are available online at www.jsc-cycling.com/xxx, Figure S1: title, Table S1: title, Video S1: title.

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