

Research article

## Effect of energy expenditure and training status on leptin response to sub-maximal cycling

Anissa Bouassida<sup>1</sup>✉, Jean-Claude Chatard<sup>2</sup>, Karim Chamari<sup>3</sup>, Monia Zaouali<sup>1</sup>, Youssef Feki<sup>4</sup>, Najoua Gharbi<sup>5</sup>, Abdelkarim Zbidi<sup>1</sup> and Zouhaïr Tabka<sup>1</sup>

<sup>1</sup> Laboratoire des adaptations cardio-circulatoires, respiratoires, métaboliques et hormonales à l'exercice musculaire, Faculté de Médecine Ibn El Jazzar, Sousse, Tunisie. <sup>2</sup> Laboratoire de Physiologie Clinique et de l'Exercice, Faculté de Médecine de Saint-Etienne, Saint-Etienne, France and Aspire Academy for Sports Excellence, Doha, Qatar. <sup>3</sup> Research Unit – "Evaluation, Sport, Health" National Centre of Medicine and Science in Sports (CNMSS), El Menzah, Tunisia. <sup>4</sup> Laboratoire des Sciences de Mesures et d'Explorations Fonctionnelles, ISSEP Kef, Tunisie. <sup>5</sup> Laboratoire d'Endocrinologie, Faculté des Sciences de Tunis, Tunisie.

### Abstract

We examined the leptin response and related hormones during and after two sub-maximal exercise protocols in trained and untrained subjects. During this study, plasma concentrations of leptin [Lep], insulin [I], cortisol [C], growth hormone [GH], glucose [G] and lactate [La] were measured. 7 elite volleyball trained players (TR) and 7 untrained (UTR) subjects (percent body fat:  $13.2 \pm 1.8$  versus  $15.7 \pm 1.0$ ,  $p < 0.01$ , respectively) were examined after short and prolonged sub-maximal cycling exercise protocols (SP and PP). Venous blood samples were collected before each protocol, during, at the end, and after 2 and 24 h of recovery. SP and PP energy expenditures ranged from  $470 \pm 60$  to  $740 \pm 90$  kcal for TR and from  $450 \pm 60$  to  $710 \pm 90$  kcal for UTR, respectively. [Lep] was related to body fat percentage and body fat mass in TR ( $r = 0.84$ ,  $p < 0.05$  and  $r = 0.93$ ,  $p < 0.01$ ) and in UTR ( $r = 0.89$ ,  $p < 0.01$  and  $r = 0.92$ ,  $p < 0.01$ , respectively). [Lep] did not change significantly during both protocols for both groups but was lower ( $p < 0.05$ ) in all sampling in TR when compared to UTR. Plasma [I] decreased ( $p < 0.01$ ) and [GH] increased ( $p < 0.01$ ) significantly during both SP and PP and these hormones remained lower (I:  $p < 0.01$ ) and higher (GH:  $p < 0.01$ ) than pre-exercise levels after a 2-h recovery period, returning to base-line at 24-h recovery. Plasma [La] increased ( $p < 0.01$ ) during both protocols for TR and UTR. There was no significant change in [C] and [G] during and after both protocols for all subjects. It is concluded that 1) leptin is not sensitive to acute short or prolonged sub-maximal exercises (with energy expenditure under 800 kcal) in volleyball/anaerobically trained athletes as in untrained subjects, 2) volleyball athletes showed significantly lower resting and exercise leptin response with respect to untrained subjects and 3) it appears that in these anaerobically trained athletes leptin response to exercise is more sensitive to the level of energy expenditure than hormonal or metabolic modifications induced by acute exercise.

**Key words:** Hormones, anaerobic training, acute exercise, body fat.

### Introduction

Leptin is an hormone mainly secreted in the adipocytes. It mediates body mass and body fat mass through the energy balance, itself controlled by satiety and energy intake (Campfield et al., 1995; Zhang et al., 1994). Leptin acts as a peripheral signal informing the central nervous system

of changes in the amount of adipose tissue in the body (Flier, 1998). Therefore, it has become of interest to examine whether physical exercise, through its disruptive effects on energy balance, sympathoadrenal drive, hormonal and metabolic changes may affect leptin concentrations (Zafeiridis et al., 2003).

The impact of acute exercise on leptin secretion is a subject of controversy. Differences have been evidenced due to the population investigated (men, women, trained, untrained), the exercise protocol (duration, intensity) and nutritional state (fasting or not) (Desgorces et al., 2004).

The response of plasma leptin concentration to a single bout of exercise performed by trained populations is limited and the reordered studies have focused on individual sport athletes, such as, running, swimming or rowing (Desgorces et al., 2004; Karamouzis et al., 2002; Leal-Cerro et al., 1998). In that context, leptin has been shown to be unchanged in rowers (Jurimae et al., 2005) and in long distance runners (Hickey et al., 1996; Zaccaria et al., 2002), after a 6 km maximal rowing exercise (Jurimae et al., 2005) and prolonged run, respectively (Hickey et al., 1996; Zaccaria et al., 2002). In contrast, Leptin decreases after acute heavy rowing exercise (Desgorces et al., 2004; Jurimae and Jurimae, 2005; Jurimae et al., 2006a; 2006b), after marathon swimming (Karamouzis et al. 2002) and after marathon running (Leal-Cerro et al. 1998; Zaccaria et al., 2002).

The impact of aerobic training on leptin secretion is a subject of controversy. Indeed, leptin has been shown to be either unchanged in elite endurance runners (Ishigaki et al., 2005) and in adolescent female runners (Kraemer et al., 2001) or decreased in elite young male athletes from different sport branches (Unal et al., 2005b) and in highly trained male rowers after training (Jurimae et al., 2003). These decreases have been described either without any changes in body fat content (Jurimae et al., 2003) or with a reduction in body fat content with training (Unal et al., 2005b). Nevertheless, little observation was made in anaerobically trained subjects (Fatouros et al., 2005) although anaerobic exercise induces marked hormonal and metabolic changes (Burlison et al., 1998) like acidosis, and altered carbohydrate metabolism (Mueller et al., 2000). In this context, volleyball has been described as an 'interval' sport with both anaerobic and aerobic

components (Smith et al., 1992) with an essential significant demand on anaerobic/strength efforts (Bompa, 1999).

Thus, the purpose of this study was to investigate leptin response in anaerobically trained elite volleyball players after short-term and long-term acute exercises, comparing them to a control group of untrained subjects.

## Methods

### Subjects

Subjects group of 14 healthy males participated in this study. Seven were volleyball elite players mainly anaerobically trained (TR). Indeed, their fitness training was essentially composed of pliometric and weight training. Their training duration was on average of 14 h-week<sup>-1</sup> for more than 7 years. They were compared to a control group of 7 untrained males (UTR). The subjects' physical characteristics are shown in Table 1. None of them smoked or drank alcohol or was taking any medication at the time of the experiment. Informed consent was obtained from all subjects and the study protocol that was in accordance with the principles of the Declaration of Helsinki 1964, was approved by the Tunisian Committee for Human Protection in Biomedical Research.

### Experimental design

Before the protocols took place, each subject was submitted to the following tests:

1- Anthropometric evaluation: body mass and height were determined by using standard physician's scales. Skin-fold thickness was measured by the sum of four different skin folds (biceps, triceps, sub-scapula, and supra-iliac) measured on the right side of the body with Holtain Callipers. On average, three independent measures were taken of each fold. If the second measure was not within 5 % of the first, subsequent folds were taken until two folds within 5 % were recorded. Then the mean of these two values was kept for analysis. The percentage of body fat was estimated with the equation of Siri, (1956) and the fat mass was determined using the formula of McArdle et al. (2001): Fat mass(kg) = (weight (kg)\*%fat)/100.

2- Maximal graded test: peak aerobic power (PAP) was determined on a calibrated cycle ergometer (Kettler CX1, Germany). The test consisted of a 3-min warm-up followed by increments in power output of one minute at 60 rpm until exhaustion. The loads during warm-up and increments were individually adjusted by taking into account the age and the body mass of each subject (Wasserman et al., 1987). The power was considered maximal, at exhaustion, when both following criteria were achieved: inability of the subject to maintain the

required pedaling frequency; attainment of age-predicted maximal heart rate (HR<sub>max</sub>) = 210-(0.65 x age) ± 5% (Jones and Campbell, 1982) measured with a cardio-frequency-meter (Polar S810, Kempele, Finland).

### Exercise sessions

Two protocols were carried out with a 7-day interval for each subject. A standardized breakfast (500 kcal) was taken (07:30 a.m) at the laboratory (the last meal taken by the subjects was towards 8:00 p.m and no other food or drink was taken until breakfast). Then the subjects remained seated until pre-exercise blood sampling (08:30 a.m) and exercise (09:00 a.m). The experiment was designed as follows:

**Short Protocol (SP) (45 min):** 3 min warm-up followed by two periods of cycling exercise of 21 min each at 70% and 85% of PAP, respectively, with 40 min passive recovery in-between.

**Prolonged Protocol (PP) (85 min):** a similar warm-up and cycling exercises but with a 40 min at 30% of PAP (active recovery) between the two periods of exercise. Only 6 untrained subjects participated in PP.

To maintain optimal hydration, the subjects drank four times the amount of 200 ml of water every 30 min: just before, during and after the exercise (Péronnet, 1988). The order of the protocols was randomized.

### Energy expenditure

After completion of SP and PP, energy expenditure was precisely calculated using the equations of McArdle et al. (2001).

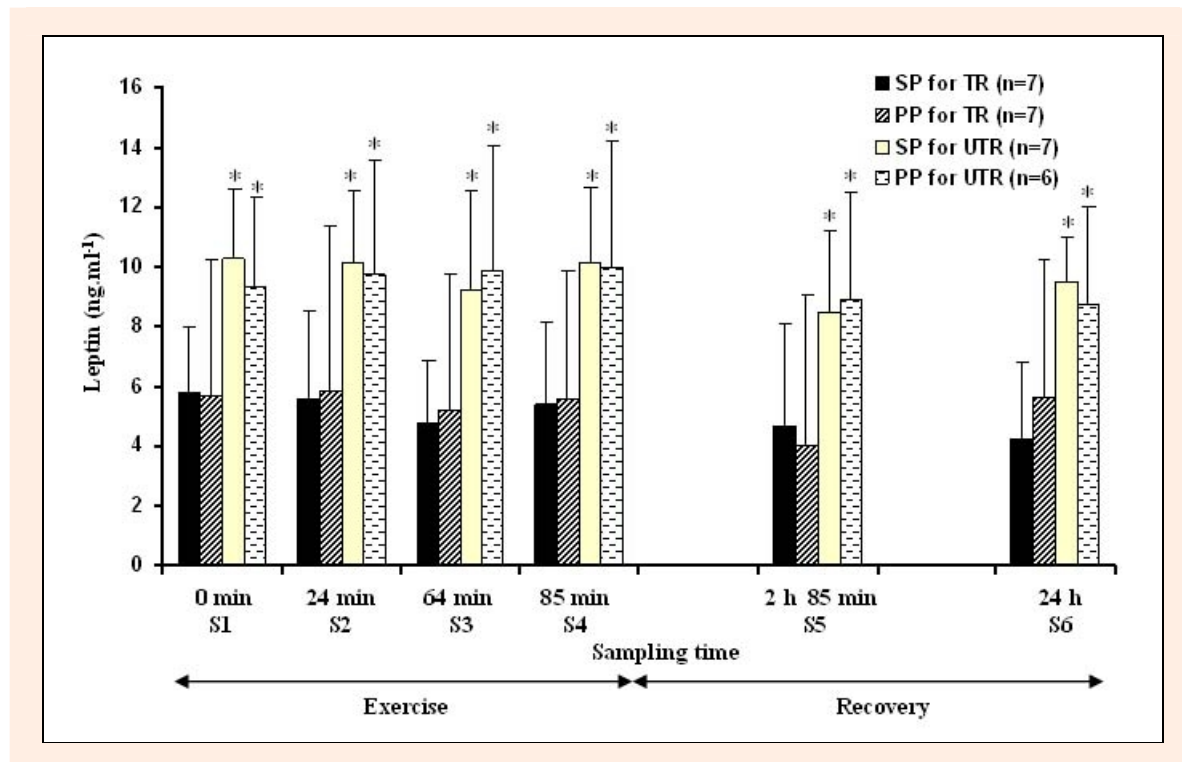
### Blood analysis

Blood samples were collected by vene-puncture on 6 occasions: before the protocol (at 08:30 a.m, pre-exercise control value, S1), at the end of the first exercise (24 min, S2), at the end of recovery period (64 min, S3), at the end of the second exercise (85 min, S4), after 2 hours (S5) and after 24 h of recovery (S6). No exercise was allowed 24-h preceding protocols and during the 24-h period of recovery post protocols. Plasma leptin concentration was measured by an immunoradiometric assay (Diagnostic system laboratories, U.S.A) (intra-assay coefficient of variation CV was 2.6% and interassay CV was 3.7%). Plasma insulin concentration was determined by an immunoradiometric method (Immunotech, France, intra-assay CV was 3.3% and interassay CV was 4.8%). Plasma cortisol concentration was estimated by a radioimmunoassay method (Immunotech, France, intra-assay CV was 2.8% and interassay CV was 5.3%). Plasma growth hormone concentration was measured by an immunoradiometric assay (Immunotech, France, intra-assay CV was 1.9% and

**Table 1. Physical characteristics of subjects. Data are means (±SD).**

	Trained (TR) (n = 7)	Untrained (UTR) (n = 7)
Age (years)	22.1 (2.5)	26.5 (3.8) **
Body mass (kg)	89.7 (10.6)	85.0 (10.9) *
Height (m)	1.92 (.04)	1.80 (.04) **
Body fat (%)	13.2 (1.8)	15.8 (1.1) **
Fat mass (kg)	11.91 (1.87)	13.38 (2.39)
BMI (kg·m <sup>-2</sup> )	24.1 (2.4)	26.3 (2.8) *
PAP (W)	308.1 (24.5)	258.5 (27.8) **

BMI: Body Mass Index; PAP: Peak aerobic power. \* p < 0.05; \*\* p < 0.01 TR vs UTR.



**Figure 1.** Mean  $\pm$  SD changes in plasma leptin concentrations during the two cycling protocols (SP and PP) in untrained (UTR) and trained (TR) subjects. SP (45 min): 3 min at 25% of PAP - 21 min at 70% of PAP - 40 min rest - 21 min at 85% of PAP; PP (85 min): 3 min at 25% of PAP - 21 min at 70% - 40 min at 30% of PAP - 21 min at 85% of PAP; CV: Control Value; PAP: Peak Aerobic Power; S: Sample. \*  $P < 0.05$  TR vs UTR during the respective protocol.

interassay CV was 13.1%). Plasma glucose and lactate were determined by using an enzymatic method (kit Randox, UK).

### Statistical analysis

ANOVA with repeated measures was performed: 2 (protocol)  $\times$  2 (group)  $\times$  6 (time). When this analysis revealed significant differences a paired Student *t*-test was used to identify significant changes between S1 to S6 and a non-paired Student *t*-test was used to locate where significant differences existed between trained and untrained subjects. Correlations of leptin to percent body fat, fat mass, insulin, cortisol, growth hormone and lactate were performed using the Pearson's method.  $P < 0.05$  was considered statistically significant.

## Results

### Energy expenditure

For SP, energy expenditure was of  $469.3 \pm 58.2$  kcal for TR and  $450.7 \pm 58.3$  kcal for UTR ( $p = 0.55$ ) while for PP, energy expenditure was of  $740 \pm 90$  kcal for TR and  $710 \pm 90$  kcal for UTR ( $p = 0.56$ ).

### Hormonal and metabolic modifications

Leptin concentrations did not significantly change during SP and PP and, neither during recovery in both groups (Figure 1). GH concentrations increased and insulin decreased significantly ( $p < 0.01$ ) during SP and PP with respectively a peak and a dip at the end of the second exercise (S4) (Table 2). After 2 hours of recovery, insulin and GH remained significantly lower ( $p < 0.01$ ) and

higher ( $p < 0.01$ ) with respect to pre-exercise and reverted close to the control values after 24 h of recovery (Table 2). As shown in Table 2, lactate concentrations increased significantly ( $p < 0.01$ ) during and after the completion of both protocols in TR and UTR, returning to control values after 2 hours recovery (Table 2). There was no significant change in plasma concentrations of cortisol and glucose during and after both protocols for both groups (Table 2).

### Comparison between TR and UTR

TR subjects had a lower percent of body fat ( $p < 0.01$ ) and BMI ( $p < 0.05$ ) although they had a higher body mass ( $p < 0.05$ ) and height ( $p < 0.01$ ) (Table 1). TR had lower leptin ( $p < 0.05$ ) and lactate ( $p < 0.05$  and  $p < 0.01$ ) concentrations than untrained (Figures 1 and Table 2). At rest, on average, leptin and lactate were significantly lower for TR than for UTR ( $5.81 \pm 3.87$  vs  $9.20 \pm 5.88$   $\text{ng}\cdot\text{mL}^{-1}$ ,  $p < 0.05$  for leptin and  $1.8 \pm 0.4$  vs  $2.6 \pm 0.6$   $\text{mmol}\cdot\text{L}^{-1}$ ,  $p < 0.05$  for lactate) (Figure 1 and Table 2).

### Correlations

In the two groups, leptin was significantly related to percent body fat ( $r = 0.84$ ,  $p < 0.05$  for TR and  $r = 0.92$ ,  $p < 0.01$  for UTR) and to body fat mass ( $r = 0.92$ ,  $p < 0.01$  for TR and  $r = 0.81$ ,  $p < 0.05$  for UTR). Leptin was not correlated to other parameters.

## Discussion

The main findings of the present study were that: 1) leptin is not sensitive to acute short or prolonged sub-maximal exercises (with energy expenditure under 800-kcal) in elite volleyball players as in untrained subjects, and 2) the

**Table 2.** Mean ( $\pm$ SD) changes in plasma cortisol, insulin, growth hormone and lactate concentrations during the two protocols (SP and PP) in trained (TR) and untrained (UTR) subjects.

Variable	Control value (S1)	24 min (S2)	64 min (S3)	85 min (S4)	2h 85 min (S5)	24 h (S6)
<b>Cortisol (nmol·L<sup>-1</sup>)</b>						
SP TR	500 (136)	521 (152)	491 (170)	515 (179)	479 (170)	521 (158)
PP TR	512 (164)	530 (176)	494 (148)	524 (136)	485 (127)	503 (155)
SP UTR	479 (155)	491 (182)	458 (179)	485 (161)	479 (152)	500 (124)
PP UTR	494 (133)	518 (182)	482 (142)	476 (176)	476 (130)	491 (136)
<b>Insulin (UI·mL<sup>-1</sup>)</b>						
SP TR	13.1 (4.2)	5.8 (2.8) **	6.5 (2.6) **	4.6 (2.5) **	8.2 (3.4) **	12.0 (4.4)
PP TR	13.0 (4.5)	4.5 (3.1) **	4.6 (2.1) **	4.0 (3.2) **	7.9 (3.6) **	12.5 (4.3)
SP UTR	15.2 (4.1)	4.1 (2.3) **	6.3 (2.0) **	3.2 (1.4) **	6.0 (3.5) **	13.9 (4.5)
PP UTR	15.4 (4.3)	3.8 (2.4) **	3.6 (2.2) **	2.6 (1.6) **	5.4 (3.1) **	13.7 (3.1)
<b>Growth hormone (ng·mL<sup>-1</sup>)</b>						
SP TR	.73 (.12)	2.83 (1.26) **	1.52 (.94) **	4.4 (2.5) **	1.84 (.96) **	.33 (.15)
PP TR	.62 (.14)	2.28 (1.44) **	1.83 (.74) **	3.6 (2.7) **	1.76 (.84) **	.32 (.10)
SP UTR	.63 (.16)	2.18 (.91) **	1.37 (.84) **	4.6 (1.5) **	1.68 (.52) **	.41 (.09)
PP UTR	.76 (.17)	1.78 (1.04) **	1.96 (.90) **	4.6 (2.1) **	1.92 (.60) **	.48 (.18)
<b>Lactate (mmol·L<sup>-1</sup>)</b>						
SP TR	1.8 (.5) ††	6.1 (2.0) **	4.5 (.8) **	7.1 (.8) **†	1.9 (1.2)	1.9 (.5)
PP TR	1.9 (.4) †	5.7 (1.5) **†	3.4 (1.4) **	6.3 (1.4) **†	1.9 (1.0)	1.8 (.5) †
SP UTR	2.7 (.6)	7.3 (2.0) **	4.3 (1.3) **	8.7 (1.3) **	2.8 (.6)	2.2 (.4)
PP UTR	2.6 (.6)	7.6 (1.6) **	3.3 (.4) **	8.0 (1.4) **	2.6 (.5)	2.8 (.8)
<b>Glucose (mmol·L<sup>-1</sup>)</b>						
SP TR	4.70 (1.38)	4.38 (1.53)	4.60 (1.56)	4.34 (1.45)	4.49 (1.24)	4.66 (1.69)
PP TR	4.62 (1.75)	4.23 (1.23)	4.53 (1.53)	4.74 (1.55)	4.66 (1.50)	4.44 (1.12)
SP UTR	4.55 (1.52)	4.33 (1.50)	4.98 (1.43)	4.73 (1.33)	5.01 (1.28)	4.70 (1.54)
PP UTR	4.21 (1.28)	4.26 (1.44)	4.83 (1.23)	4.87 (1.26)	5.08 (1.16)	4.75 (1.20)

SP (45 min): 3 min at 25% of PAP - 21 min at 70% of PAP - 40 min rest - 21 at 85% of PAP; PP (85 min): 3 min at 25% of PAP - 21 min at 70% - 40 min at 30% of PAP - 21 at 85% of PAP; CV: Control Value; PAP: Peak Aerobic Power; S: Sample. \*\*  $p < 0.01$  S1 vs other sampling; †  $p < 0.05$ , ††  $p < 0.01$  TR vs UTR during the respective protocol.

volleyball anaerobically trained players had decreased leptin concentrations at rest and during exercise when compared to the untrained subjects.

The present discussion will only focus on acute exercise studies performed by trained subjects. Hickey et al., (1996) and Jurimae et al. (2005) showed that leptin did not change significantly after a 20-mile run at 70% of  $VO_{2max}$  in male runners and after a maximal 6,000-m rowing in highly trained male rowers. In contrast, leptin concentrations significantly decreased following 1) 25-km sea swimming competition in male long-distance swimmers (Karamouzis et al., 2002), 2) 30-min maximal rowing in college level male rowers (Jurimae and Jurimae, 2005), 3) an ultra-marathon in trained runners (Zaccaria et al., 2002), 4) 2-km maximal sculling in elite rowers (Jurimae et al., 2006a), 5) a marathon in non-professional long distance runners (Leal-Cerro et al., 1998), 6) 6.5 km rowing at the individual anaerobic threshold in college level male rowers (Jurimae et al., 2006b), and 7) 90-min rowing at 70-75% of  $VO_{2max}$  in trained rowers (Desgorces et al., 2004). The present study showed that as for untrained subjects, leptin was unaltered in volley-ball trained athletes for the sub-maximal exercises performed. Although, the energy expenditure (EE) level of 740 kcal in the present study (highest level of EE observed) did not significantly alter leptin concentration, both acute sub-maximal exercises were accompanied by changes in insulin, growth hormone, and lactate concentrations. In this context, it has been suggested that Leptin secretion may be influenced by hormonal and metabolic changes induced by physical exercise (Desgorces et al., 2004; Gomez-Merino et al., 2002). Indeed, the observed decreases

in leptin concentrations by other studies were accompanied by hypoinsulinemia (Desgorces et al., 2004; Jurimae et al., 2006a; 2006b) and increased: growth hormone (Jurimae and Jurimae 2005; Jurimae et al., 2006a), glycerol (Karamouzis et al., 2002), cortisol (Jurimae et al., 2006a), FFA (Karamouzis et al., 2002, Zaccaria et al., 2002) and neuropeptide Y (NPY) (Karamouzis et al., 2002) concentrations. The present study shows that for the sub-maximal exercises performed with energy expenditure lower than 800 kcal, it is possible to observe hormonal and metabolic changes without concomitant changes in plasma leptin.

In the present study, leptin cortisol, insulin, GH and glucose responses were similar between the SP and PP. Furthermore, the PP was the most stressful, as indicated by the highest lactate levels. It has been shown, that acidosis can decrease leptin secretion from cultured adipocytes (Teta et al., 2003). It may be that the rise in lactate secondary to the PP was not a sufficient stimulus to alter leptin response. Another possible explanation for the lack of differences in plasma leptin decline after the PP is the prevailing theory of delayed leptin responses after physical activity. Previous studies that studied running and rowing exercise in healthy and trained men have reported delayed leptin reduction at 30 minutes (Jurimae et al., 2006a; 2006b), 9-13 hours (Nindl et al., 2002) and 48 hours (Olive and Miller., 2001) after exercise.

The difference of difference in leptin response could be explained by the level of energy expenditure of the exercise tasks performed by the subjects. Indeed, a 800 kcal level of energy expenditure has been proposed to be a threshold for the decrease of leptin secretion (Bouas-

sida et al., 2006; Kraemer et al., 2002) and, secondly, the amount of muscle tissue used during the acute exercise could also influence the leptin response (Jurimae and Jurimae 2005; Jurimae et al., 2006b). However, these two hypotheses can be discussed. On one hand, Zaccaria et al. (2002) showed that a half-marathon at 1400-kcal EE did not modify leptin concentrations and Zafeiridis et al., (2003) demonstrated that resistance exercise protocols designed for development of maximum strength, muscular hypertrophy and strength endurance (mainly anaerobic exercise) at relatively low levels of energy expenditure ( $213.19 \pm 6.8$ ,  $314.86 \pm 71.1$ , and  $327.58 \pm 12.8$  kcal) decreased leptin levels. On the other hand, Jurimae et al., (2005) demonstrated stable leptin after acute heavy rowing exercise.

The present study shows that in spite of hormonal and metabolic modifications, energy expended during the 85 min prolonged sub-maximal exercise seems to be insufficient to cause significant changes in leptin concentrations in elite volleyball players as in untrained subjects. Kyriazis et al., (2007) and Weltman et al., (2000) showed that leptin was not altered after 60 min and 30 min exercises with lower energy expenditure than ~600 kcal ( $567 \pm 25$  kcal) and ( $150 \pm 11$ ,  $271 \pm 23$ ,  $364 \pm 28$  and  $529 \pm 45$  kcal), respectively. In contrast, Nindl et al., (2002) observed a decrease of leptin after a  $856 \pm 114$  kcal of anaerobic work (50 total sets of squat, bench press, leg press and lat pull-down) and Olive and Miller (2001) observed a leptin decrease after a  $883 \pm 14$  kcal aerobic run (60 min at 70% of maximal oxygen consumption " $\text{VO}_{2\text{max}}$ "). The 800-kcal threshold observed by the majority of studies investigating leptin response to exercise seems thus to be applicable not only in untrained but also in volleyball players, since the present study exercises performed (under the 800-kcal level) failed to cause a decrease in leptin concentration. It has to be mentioned that Volley-ball training is a mix of aerobic and anaerobic training, but that the investigated players mainly focused on preponderantly anaerobic training (force and pliometrics) in their fitness training. It is thus shown that even in anaerobically trained athletes, at least the threshold of 800-kcal of energy expenditure seems to be applicable to obtain significant decreases of leptin concentration at exercise. Nevertheless, this has to be confirmed by subsequent studies with energy expenditures higher than 800-kcal. It could be postulated that this level of energy expenditure may be necessary to elicit an, as of yet, unidentified neural or hormonal signal that depresses leptin synthesis. Alternatively, mobilization of non-esterified fatty acids from adipose tissue for use as an energy substrate may be a controlling factor in leptin levels, consistent with results from van Aggel-Leijssen et al. (1999). Additional research would help determine the level of energy expenditure or metabolic response from the exercise that alters plasma leptin. This information could be valuable in understanding mechanisms underlying its control and consequential effects on energy balance in the body (Olive and Miller, 2001). It remains unknown how energy availability, sympathetic stimulation, and metabolites and hormones that down- and upregulate leptin concentration may interact to modulate leptin concentrations (Zafeiridis et al., 2003).

A significant correlation in each group was observed for leptin with percent body fat and with fat mass. Similarly, other investigators demonstrated that resting leptin levels were strongly related to fat mass in aerobically trained athletes (Hickey et al., 1996, Jurimae et al., 2006a; Leal-Cerro et al., 1998). Lower body fat mass was mediated by leptin and a direct relationship between leptin and body fat composition was found in both groups. These data confirmed the studies in which leptin is lower in trained with respect to untrained subjects, e.g. professional football players trained for at least five years (Unal et al., 2005b), marathon runners trained at high level (Leal-Cerro et al., 1998) and athletes trained for at least 2 years (Unal et al., 2005a). In untrained or less trained subjects, short exercise training periods have shown the same effect on leptin secretion. For example, resting leptin levels decreased after 1) 3-weeks of military training preceded by a 5-day aerobic military walk of 25-35 km (during the five days) (Gomez-Merino et al., 2002). after 2) 12-weeks of an aerobic cycling exercise program e.g.  $5 \text{ d.wk}^{-1}$  at 50% of maximal oxygen uptake during 45 min, performed by 25 obese sedentary women (Polak et al., 2006) and after 3) a 6-month anaerobic training program ( $3 \text{ d.wk}^{-1}$ , 10 strength exercises/three sets per session) in 50 inactive men (Fatouros et al., 2005). These decreases were associated with a parallel decline in body fat (Fatouros et al., 2005; Polak et al., 2006). Nevertheless, the decrease in resting leptin concentration may also occur without any decrease in fat mass. Indeed, Ishii et al., (2001) showed a reduction in leptinemia after 6-weeks of an aerobic training in type-2 diabetic subjects and this decrease was independent of the changes in fat mass. The present study lower resting and exercising leptinemia observed in the Volley-ball players with respect to the untrained subjects could be due to their lower body fat mass. Thus it appears here that independently of the type of training, mainly aerobic or mixed aerobic with an important component of anaerobic training, the resulting lower body fat mass induces lower resting and exercising leptin levels.

## Conclusion

It is concluded that the present data indicate that plasma leptin concentration is not sensitive to acute short or prolonged exercise (under 800-kcal of energy expenditure) in elite volleyball players. In addition, plasma leptin concentration was lower in volleyball/anaerobically trained athletes with respect to untrained subjects. It appears that leptin is more sensitive to energy expenditure than hormonal or metabolic modifications induced by acute exercise (hypoinsulinemia, hypersecretion of growth hormone and hyperlactatemia) in anaerobically trained athletes as in aerobically trained athletes as shown by past studies.

## Acknowledgements

The author would like to thank all the subjects who participated in this study, the staff of the Physiology Department, Faculté de Médecine Ibn El Jazzar, 4002 Sousse, Tunisia and the staff of the Biochemistry Department, Hôpital Farhat Hached, 4002 Sousse, Tunisia. We also wish to thank James Ptyce for reviewing the English of the manuscript. This work was supported by the "Ministère de l'Enseignement Supérieur, de la recherche Scientifique et de la Technologie, Tunisia".

## References

- Bompa, T.O. (1999) *Periodization training for sports*. Champaign: IL, Human Kinetics
- Bouassida, A., Zalleg, D., Bouassida, S., Zaouali, M., Feki, Y., Zbidi, A. and Tabka Z. (2006) Leptin, its implication in physical exercise and training: a short review. *Journal of Sports Science and Medicine* **5**, 172-181.
- Burleson, M.A., O'Bryant, H.S., Stone, M.H., Collins, M.A. and Triplett-McBride, T. (1998) Effect of weight training exercise and treadmill exercise on post-exercise oxygen consumption. *Medicine and Science in Sports and Exercise* **30**, 518-522.
- Campfield, A.L., Smith, F.J., Guisez, Y., Devos, R. and Burn, P. (1995) Recombinant mouse OB protein: evidence for a peripheral signal linking adiposity and central networks. *Science* **269**, 546-549.
- Desgorces, F.D., Chennaoui, M., Gomez-Merino, D., Drogou, C. and Guezennec, C.Y. (2004) Leptin response to acute prolonged exercise after training in rowers. *European Journal of Applied Physiology* **91**(5-6), 677-681.
- Fatouros, I.G., Tournis, S., Leontini, D., Jamurtas, A.Z., Sxina, M., Thomakos, P., Manousaki, M., Douroudos, I., Taxildaris, K. and Mitrakou, A. (2005) Leptin and adiponectin responses in overweight inactive elderly following resistance training and detraining are intensity related. *Journal of Clinical Endocrinology and Metabolism* **90**(11), 5970-5077.
- Flier, J.S. (1998) Clinical review 94: What's in a name? In search of leptin's physiologic role. *Journal of Clinical Endocrinology and Metabolism* **83**(5), 1407-1413.
- Gomez-Merino, D., Chennaoui, M., Drogou, C., Bonneau, D. and Guezennec, C.Y. (2002) Decrease in serum leptin after prolonged physical activity in men. *Medicine and Science in Sports and Exercise* **34**(10), 1594-1599.
- Hickey, M.S., Considine, R.V., Israel, R.G., Mahar, T.L., McCammon, M.R., Tyndall, G.L., Houmard, J.A. and Caro, J.F. (1996) Leptin is related to body fat content in male distance runners. *American Journal of Physiology* **271**(5 Pt 1), E938-40.
- Ishigaki, T., Koyama, K., Tsujita, J., Tanaka, N., Hori, S. and Oku, Y. (2005) Plasma leptin levels of elite endurance runners after heavy endurance training. *Journal of Physiological Anthropology and Applied Human Science* **24**(6), 573-578.
- Ishii, T., Yamakita, T., Yamagami, K., Yamamoto, T., Miyamoto, M., Kawasaki, K., Hosoi, M., Yoshioka, K., Sato, T., Tanaka, S. and Fujii, S. (2001) Effect of exercise training on serum leptin levels in type 2 diabetic patients. *Metabolism* **50**(10), 1136-1140.
- Jones, N.L. and Campbell, E.J.M. (1982) *Clinical exercise testing*. 2nd edition. W.B. Saunders Company, Philadelphia.
- Jürimäe, J., Mäestu, J. and Jürimäe, T. (2003) Leptin as a marker of training stress in highly trained male rowers? *European Journal of Applied Physiology* **90**, 533-538.
- Jurimae, J. and Jurimae, T. (2005) Leptin responses to short term exercise in college level male rowers. *British Journal of Sports Medicine* **39**(1), 6-9.
- Jürimäe, J., Purge, P. and Jürimäe, T. (2005) Adiponectin is altered after maximal exercise in highly trained male rowers. *European Journal of Applied Physiology* **93**(4), 502-505.
- Jürimäe, J., Purge, P. and Jürimäe, T. (2006a) Adiponectin and stress hormone responses to maximal sculling after volume-extended training season in elite rowers. *Metabolism* **55**(1), 13-19.
- Jürimäe, J., Hofmann, P., Jürimäe, T., Mäestu, J., Purge, P., Wonisch, M., Pokan, R. and von Duvillard, S.P. (2006b) Plasma adiponectin response to sculling exercise at individual anaerobic threshold in college level male rowers. *International Journal of Sports Medicine* **27**(4), 272-277.
- Karamouzis, I., Karamouzis, M., Vrabas, I.S., Christoulas, K., Kyriazis, N., Giannoulis, E. and Mandroukas, K. (2002) The effects of marathon swimming on serum leptin and plasma neuropeptide Y levels. *Clinical Chemistry and Laboratory Medicine* **40**(2), 132-136.
- Kraemer, R.R., Acevedo, E.O., Synovitz, L.B., Hebert, E.P., Gimpel, T. and Castracane, V.D. (2001) Leptin and steroid hormone response to exercise in adolescent female runners over a 7-week season? *European Journal of Applied Physiology* **86**, 85-91.
- Kraemer, R.R., Chu, H. and Castracane, V.D. (2002) Leptin and exercise. *Experimental Biology and Medicine (Maywood)* **227**(9), 701-708.
- Kyriazis, G.A., Caplan, J.D., Lowndes, J., Carpenter, R.L., Dennis, K.E., Sivo, S.A. and Angelopoulos, T.J. (2007) Moderate exercise-induced energy expenditure does not alter. Leptin levels in sedentary obese men. *Clinical Journal of Sport Medicine* **17**(1), 49-51.
- Leal-Cerro, A., Garcia-Luna, P.P., Astorga, R., Parejo, J., Peino, R., Dieguez, C. and Casanueva, F.F. (1998) Serum leptin levels in male marathon athletes before and after the marathon run. *Journal of Clinical Endocrinology and Metabolism* **83**(7), 2376-2379.
- McArdle, W., Katch, F. and Katch, V. (2001) *Physiologie de l'activité physique: Energie, nutrition et performance*. 4<sup>th</sup> Edition. Maloine/Edisem, Paris.
- Mueller, W.M., Stanphone, K.L., Gregoire, F., Evans, J.L. and Havel, P.J. (2000) Effects of metformin and vanadium on leptin secretion from cultured rat adipocytes. *Obesity Research* **8**, 530-539.
- Nindl, B.C., Kraemer, W.J., Arciero, P.J., Samatallee, N., Leone, C.D., Mayo, M.F. and Hafeman, D.L. (2002) Leptin concentrations experience a delayed reduction after resistance exercise in men. *Medicine and Science in Sports and Exercise* **34**(4), 608-613.
- Olive, J.L. and Miller, G.D. (2001) Differential effects of maximal- and moderate-intensity runs on plasma leptin in healthy trained subjects. *Nutrition* **17**, 365-369.
- Péronnet, F. (1988) La physiologie du marathonien. *La Recherche* **19**, 920-930.
- Polak, J., Klimcakova, E., Moro, C., Viguerie, N., Berlan, M., Hejnova, J., Richterova, B., Kraus, I., Langin, D. and Stich, V. (2006) Effect of aerobic training on plasma levels and subcutaneous abdominal adipose tissue gene expression of adiponectin, leptin, interleukin 6, and tumor necrosis factor alpha in obese women. *Metabolism* **55**(10), 1375-1381.
- Siri, W.E. (1956) The gross composition of the body. In: *Advances in Biological and Medical Physics*. Eds: Tobias, C.A. and Lawrence, J.H. Academic Press New York. Vol 4.
- Smith, D.J., Roberts, D. and Watson, B. (1992) Physical, physiological and performance differences between Canadian national team and universiade volleyball players. *Journal of Sports Sciences* **10**(2), 131-138.
- Teta, D., Bevington, A., Brown, J., Pawluczyk, I., Harris, K. and Walls, J. (2003) Acidosis downregulates leptin production from cultured adipocytes through a glucose transport-dependent post-transcriptional mechanism. *Journal of the American Society of Nephrology* **14**(9), 2248-2254.
- Unal, M., Unal, D.O., Baltaci, A.K. and Mogulkoc, R. (2005a) Investigation of serum leptin levels and VO<sub>2</sub>max value in trained young male athletes and healthy males. *Acta Physiologica Hungaria* **92**(2), 173-179.
- Unal, M., Unal, D.O., Baltaci, A.K., Mogulkoc, R. and Kayserilioglu, A. (2005b) Investigation of serum leptin levels in professional male football players and healthy sedentary males. *Neuro Endocrinology Letters* **26**(2), 148-151.
- van Aggel-Leijssen, D.P., van Baak, M.A., Tenenbaum, R., Campfield, L.A. and Saris, W.H. (1999) Regulation of average 24h human plasma leptin level; the influence of exercise and physiological changes in energy balance. *International Journal of Obesity and Related Metabolic Disorders* **23**(2), 151-158.
- Wasserman, K., Hansen, J.E., Sue, D.Y. and Whipp, B.J. (1987) *Principles of exercise testing and interpretation*. Lea & Febiger, Philadelphia.
- Weltman, A., Pritzlaff, C.J., Wideman, L., Considine, R.V. and Fryburg, D.A., Gutgesell, M.E., Hartman, M.L. and Veldhuis, J.D. (2000) Intensity of acute exercise does not affect serum leptin concentrations in young men. *Medicine and Science in Sports and Exercise* **32**(9), 1556-1561.
- Zaccaria, M., Ermolao, A., Roi, G.S., Englaro, P., Tegon, G. and Varnier, M. (2002) Leptin production after endurance races differing in duration and energy expenditure. *European Journal of Applied Physiology* **87**(2), 108-111.
- Zafeiridis, A., Smilios, I., Considine, R.V. and Tokmakidis, S.P. (2003) Serum leptin responses following acute resistance exercise protocols. *Journal of Applied Physiology* **94**, 591-597.
- Zhang, Y., Proenca, R., Maffei, M., Barone, M., Leopold, L. and Friedman, J.M. (1994) Positional cloning of the mouse obese gene and its human homologue. *Nature* **372**, 425-432.

### Key points

- Trials concerning acute exercise and leptin indicated discrepant results.
- Acute exercise with energy expenditure higher than 800 kcal can decrease leptinemia.
- Elite volleyball players presented decreased leptin levels than untrained subjects.

### AUTHORS BIOGRAPHY



#### **Anissa BOUASSIDA**

##### **Employment**

Associate Professor, High Institute of Sport and Physical Education, Jendouba univ., Kef, Tunisia.

##### **Degree**

PhD

##### **Research interests**

Exercise and training induced hormonal and metabolic changes.

**E-mail:** bouassida\_anissa@yahoo.fr

#### **Jean-Claude CHATARD**



##### **Employment**

Professor, ASPETAR, Qatar Orthopedic and Sports Medicine Hospital

##### **Degree**

PhD, MD.

##### **Research interests**

Exercise and training induced hormonal and metabolic changes.

#### **Karim CHAMARI**



##### **Employment**

Research Unit - Evaluation, Sport Health - National Center of Medicine and Science in Sports (CNMSS), El Menzah, Tunisia. Fitness Coach in Professional Football (soccer)

##### **Degree**

PhD, MD

##### **Research interests**

Exercise responses and training adaptations. Exercise testing.

#### **Monia ZAOUALI**



##### **Employment**

Professor, Faculty of Medicine Ibn El Jazzar, Sousse, Tunisia

##### **Degree**

PhD

##### **Research interests**

Exercise and training induced hormonal and metabolic changes.

#### **Youssef FEKI**



##### **Employment**

Professor, High Institute of Sport and Physical Education, Jendouba univ., Kef, Tunisia.

##### **Degree**

PhD

##### **Research interests**

Exercise induced cardio-circulatory, respiratory, metabolic and hormonal changes.



#### **Najoua GHARBI**

##### **Employment**

Professor, Laboratory of Endocrinology, Biology Department, Faculty of Science, Tunis, Tunisia.

##### **Degree**

PhD

##### **Research interests**

Exercise and training induced hormonal and metabolic changes.



#### **Abdelkarim ZBIDI**

##### **Employment**

Director of the Faculty of Medicine Ibn El Jazzar, Sousse, Tunisia.

##### **Degree**

PhD, MD

##### **Research interests**

Exercise induced cardio-circulatory, respiratory, metabolic and hormonal changes.



#### **Zouhair TABKA**

##### **Employment**

Professor, Faculty of Medicine Ibn El Jazzar, Sousse, Tunisia.

##### **Degree**

PhD, MD

##### **Research interests**

Exercise induced cardio-circulatory, respiratory, metabolic and hormonal changes.

**E-mail:** zouhair.tabka@rns.tn

#### ✉ **Anissa Bouassida**

Laboratoire de Physiologie, Faculté de Médecine Ibn El Jazzar, avenue Mohamed Karoui 4002, Sousse, Tunisie.