Progress in Nutrition 2020; Vol. 22, Supplement 2: e2020025 DOI: 10.23751/pn.v22i2-S.10587

Original article

The effect of long term exercise on leptin levels in adolescent wrestlers

Tevfik Cem Akalın[1], *Mustafa Gümüş*[1], *Taner Bayraktaroğlu*[2], *İbrahim Etem Pişkin*[3], *Faruk Yamaner*[4]

[1] School of Physical Education and Sports, Zonguldak Bülent Ecevit Üniversity, Zonguldak, Turkey
[2] Faculty of Medicine, Department of Endocrinology, Zonguldak Bülent Ecevit Üniversity, Zonguldak, Turkey
[3] Faculty of Medicine, Department of Pediatrics, Zonguldak Bülent Ecevit Üniversity, Zonguldak, Turkey
[4] Faculty of Sport Sciences, Hitit University, Çorum, Turkey

Abstract. *Study Objectives:* The implication of leptin in physical exercise and during its recovery is still unclear. The aim of this study was to examine the effect of eight-month regular well-developed wrestling training in male adolescents on leptin levels, where metabolic and endocrine changes are important. *Methods:* The sample for this study comprised 69 volunteering male individual subjects who were divided into two different groups. The training program was applied to the 49 male adolescent wrestling group age of 13–15 years (14.4±10.61 years), for 90-120 minutes/day for five days a week during the eight months. The control group consisted of 20 males age between 13–15 years (14.05±0.76 years) who did not receive any planned exercise sessions. Data about the anthropometric and metabolic characteristics including age, body weight, height, body mass index (BMI), waist circumferences, and leptin were obtained. Data were analyzed with the SPSS for Windows 21.0 software. Mann Whitney U Test was used to comparison of independent groups, Wilcoxon Signed Rank Test and Paired Samples t-Test were used to comparison of measurement times, results of analysis were evaluated with 95% confidence interval. *Results:* There were statistical significantly increase in height, weight, waist circumference, and BMI results of male adolescent wrestlers who did wrestling training for months ($p<0.05$). No statistical change was found between leptin levels ($p>0.05$), while leptin levels decreased significantly with puberty ($p<0.05$). *Conclusion:* Although some studies indicate that there is a strong relationship between BMI and leptin level, such a relationship was not found in our study. This situation is thought to be caused by physiological changes that are important and effective in adolescence.

Keywords: Wrestlers, Adolescent, Leptin, Exercise, Long term exercise

Introduction

The effect of physical exercise on leptin concentrations is currently controversial. Several investigators reported that exercise may result in reductions depending on the duration and calorie expenditure whereas others have reported no change in leptin concentrations (1, 2). Many studies have investigated the effects of training on leptin concentrations. Many studies have tended to report either no effect of training on leptin concentrations with short-term training (<12 weeks), or a reduction in leptin levels in long-term training (≥12 weeks) studies. The discordance in the literature is probably related to several factors, such as the intensity and the duration of the exercise, the nutritional status of the subject, the circadian rhythm of leptin, the hour of blood sampling and the caloric imbalance imposed by the exercise (2).

The prevalence of obesity has increased in recent years and poses an important public health problem

as some adverse health consequences are associated with increased adiposity and fat gain (3, 4) Childhood and adolescent obesity is a strong predictor of adult obesity (5) and identifying more effective weight loss intervention strategies to alleviate this financial and public health burden is paramount (1). Obesity is defined as the accumulation of excess body fat caused by the imbalance between energy intake and energy expenditure (6).

Adipose tissue is an active endocrine tissue that releases many adipokines. The alterations in the secretory profile of this adipose tissue were found to be the reason for the disturbed fat metabolism and obesity-associated complications (7). Leptin is one of the most important adipokines secreted from the adipose tissue. It has complex and multidirectional actions that mainly include suppression of appetite and regulation of body weight (8). In addition to adipose tissue, skeletal muscles and bones are important sources of circulating leptin (9) and also targets for leptin actions as leptin receptors were found to be expressed in abundance in both muscle and bone cells (10). Recent studies showed that leptin is an essential peptide hormone regulating energy homeostasis (11). Briefly, studies have shown that leptin plays a major role in regulating energy expenditure and bone metabolism (12, 13). One of the main hormones responsible for controlling energy balance and body weight by altering energy intake and energy expenditure, leptin (8) is released in response to acute energy availability during periods of intermittent fasting, calorie restriction, and overeating (8, 14, 15)

The aim of this study was to examine the effect of eight-month regular well-developed wrestling training in male adolescents on leptin levels, where metabolic and endocrine changes are important.

Material and Method

Subjects

The sample for this study comprised 69 volunteering male individual subjects who were divided into two different groups (since the leptin level of one participant in the wrestlers group and control groups could not be determined; these participants were excluded from the study): (1) training group, formed by 49 male wrestlers with suitable analyses age between 13–15 years (14.05±0.76 years) who trained wrestling and presented 1.96±0.87 years of training, 6.14±4.13 times participating in a national or regional championship and (2) control group formed by 20 adolescent males aged between 13–15 years (14.05±0.76 years) who did not receive any planned exercise sessions.

Initially, the wrestlers' medical history was taken to verify the existence of possible metabolic syndromes and health problems that would prevent the participation of these subjects in the study. It was declared that the subjects had the option to leave the study at any time, without having any responsibility. The study protocol and purpose were explained to all subjects, and a written informed consent was obtained from each participant and one of the parents. Approval was obtained from Zonguldak Bülent Ecevit University Clinical Researches Ethical Commitee.

Wrestling groups were active at a competitive level (participating in regional and/or national competitions) and were different from Wrestling Training Center affiliated by the Turkish Wrestling Federation that has well-developed training programs for children in wrestling. The wrestlers had been training for one to two years before the study. The control group, on the other hand, continued their normal daily life activities during this period and did not received any planned exercise sessions.

Training program

A regular medium and high intensity training program of 90-120 minutes / day was applied to the wrestling group, five days a week. In wrestling training centers, the wrestling federation used a weekly and monthly periodic training model based on technical tactical, and basic motoric features specific to children and adolescent athletes.

Test protocol

Anthropometric and metabolic parameters were investigated before and after the season. The control group was tested in the same period. Data about the

anthropometric and metabolic characteristics including age, body weight, height, body mass index (BMI), waist circumferences, and leptin were obtained. The body height of the subjects was measured by a metal scale with a 0.1 cm sensitivity and the body weight measurement was taken by a digital weight with a 0.1-kg sensitivity. BMI was calculated as weight (kg) divided by the square of height (m).

The maturity status of all of the children was assessed by a pediatrician experienced in the assessment of secondary sex characteristics according to the development of pubic hair, testicular volume (Prader's orchidometer) and length of the phallus as described by Marshall and Tanner in 1970, (16) to select the appropriate level.

All data were collected at the Medical Faculty laboratory at Bulent Ecevit University, Zonguldak. Blood samples were withdrawn into heparinized tubes from a cubital vein after overnight fasting and immediately stored in ice. Leptin measurements were evaluated with the Human Leptin Enzyme Linked Immuno Sorbent Assay (ELISA) Test Kit.

Statistical Analysis

Data were analyzed with the SPSS for Windows 21.0 software (SPSS Inc., Chicago, IL, USA). Descriptive statistics were given as "mean ± standard deviation" and "median (minimum - maximum). Mann Whitney U Test was used to comparison of independent groups, Wilcoxon Signed Rank Test and Paired Samples t-Test were used to comparison of measurement times, results of analysis were evaluated with 95% confidence interval.

Results

It was figured out that adolescent wrestlers aged 13-15 have, 1-5 years for active sport, their BMI were 18.93±2.80kg/m^2, waist circumferences were 70.96±7.12, and leptin level was 0.71±0.93 ng/mL (Table 1). It has been determined that the wrestler group has been doing active sports for 1.96 years, training five days a week, 1.36 hours a day, and participating in a national or regional championship 6.14 times.

When the pre-test results given in table 1 were examined, it was seen that the wrestling and control groups had homogeneous physical properties. Anthropometric characteristics of adolescent wrestlers and healthy controls were compared in terms of height, weight, BMI, waist circumference, and there was no significant difference between the two groups ($p>0.05$). Although there was no statistically significant difference in leptin levels between the groups ($p>0.05$), the leptin level of the wrestler' group was found to be lower.

The experiment and the control subjects were grouped as pubertal and prepubertal groups. According to Tanner's staging of puberty levels of wrestler and control groups physical characteristics and leptin variables were shown in table 2. 61.2% of the wrestlers were pubertal and 38.8% of wrestlers were prepubertal. There was no significant difference between serum leptin and waist circumference in pubertal and prepubertal subjects ($p>0.05$). A statistically significant difference was found between other measurement data ($p<0.05$) (Table 2).

When the results of the study were examined, body weight, height, BMI, and waist circumference increased significantly with the onset of puberty ($p<0.05$). Although waist circumference increased significantly with puberty ($p<0.05$), it did not change with exercise ($p>0.05$). The decrease in leptin levels with the onset of puberty is significant ($p<0.05$). The leptin levels did not change after exercise ($p>0.05$). After exercise, leptin levels were found to be significantly lower in the pubertal group ($p= 0.048$). Physiologically changes in hormonal, anthropometric, and adipose tissue reveal this result in puberty. We think that leptin levels and adipose tissue and muscle tissue compositions in puberty cause this. The effects of exercise are also remarkable (Table 2).

Discussion

Serum leptin level relates to the adiposity level and that it is involved in the regulation of appetite, metabolism, and energy balance are well known (17). Anthropometric characteristics of adolescent wrestlers and healthy controls were compared in terms of

Table 1. Physical characteristics and leptin variables for both the wrestlers and the controls

Variables		***Wrestlers*** *(n= 49)*	***Controls*** *(n= 20)*	***p***
Age *(year)*	*Mean ± SD*	14.41 ± 0.61	14.05 ± 0.76	0.064
	Med. (Min.-Max.)	14.00 (13.00-15.00)	14.00 (13-15)	
Body height *(cm)*	*Mean ± SD*	1.48 ± 0.10	1.52 ± 0.10	0.131
	Med. (Min.-Max.)	1.47 (1.34-1.72)	1.54 (1.30-1.64)	
Body weight *(kg)*	*Mean ± SD*	42.23 ± 11.02	45.30 ± 11.09	0.223
	Med. (Min.-Max.)	40.80 (26.00-66.00)	40.00 (33.00-73.00)	
Body Mass Index *(kg/m²)*	*Mean ± SD*	18.93 ± 2.80	19.45 ± 3.07	0.479
	Med. (Min.-Max.)	18.12 (14.06-24.06)	18.75 (15.98-27.14)	
Waist circumference *(cm)*	*Mean ± SD*	70.96 ± 7.12	68.75 ± 7.06	0.179
	Med. (Min.-Max.)	70.00 (61.00-90.00)	68.00 (61.00-87.00)	
Leptin *(ng/mL)*	*Mean ± SD*	0.71 ± 0.93	1.06 ± 1.62	0.762
	Med. (Min.-Max.)	0.40 (0.10-5.60)	0.60 (0.10-5.80)	
Active sport duration *(year)*	*Mean ± SD*	1.96 ± 0.87	-	-
	Med. (Min.-Max.)	2.00 (1.00-5.00)	-	
Training *(days/week)*	*Mean ± SD*	5.00 ± 0.00	-	-
	Med. (Min.-Max.)	0.00 (5.00-5.00)	-	
Training *(hour/days)*	*Mean ± SD*	1.36 ± 0.09	-	-
	Med. (Min.-Max.)	0.30 (1.30-1.45)	-	
Number of competition	*Mean ± SD*	6.14 ± 4.13	-	-
	Med. (Min.-Max.)	5.00 (1.00-15.00)	-	

SD; standartdeviation, *Med. (Min.-Max.); Median (Minimum-Maximum)*

height, weight, BMI, waist circumference, and there was no significant difference between the two groups (p>0.05). Although there was no statistically significant difference in leptin levels between the groups (p>0.05), the leptin level of the wrestler' group was found to be lower. Alzamil et al. (2018) and Plonka et al. (2011) reported that, in girls, the level of serum leptin was inversely correlated with the level of physical activity, and the study findings showed that girls performing higher physical activity have the lowest serum leptin level (17, 18). Previous studies found a negative correlation between leptin and physical fitness (19). Miyatake et al. (2014) found that, in clinical practice, reduced circulating leptin levels can be promoted by physical fitness in men and physical activity in women (20).

Many studies in the literature have investigated the relationship between leptin and body weight. For instance, a positive relationship between serum leptin levels and body weight has been reported in epidemiological studies (21, 22). A population-based study of Danish and Norwegian children and adolescents reported that leptin mediated the association between BMI and BP, using empirical mediation analyses (23).

Given that BMI consists not only of fat mass, but also of lean body mass, BMI is an unsuitable index for accurately assessing relationships between body fat and leptin. To accurately examine relationships between body fat, leptin, and blood pressure, an accurate and precise technique for measuring body fat, as well as studies that involve the direct evaluation of body fat, is needed (24). Fujita et al, in their study which targeted

Table 2. Comparison of the physical characteristics and leptin variables by the pubertal and pre and post training status

Variables		*Wrestlers (n= 49)*		p^1	*Total*
		Pubertal *(n= 30, 61,2%)*	***Prepubertal*** *(n= 19, 38.8%)*		
Body height *(cm)*	Pre-Training	1.52 ± 0.10 1.54 (1.36-1.72)	1.42 ± 0.72 1.40 (1.34-1.59)	**0.001**	1.48 ± 0.10 1.47 (1.34-1.72)
	Post-Training	1.62 ± 0.08 1.60 (1.42-1.75)	1.47 ± 0.07 1.45 (1.37-1.63)	**0.001**	1.56 ± 0.11 1.57 (1.37-1.75)
	p^2	**0.001**	**0.001**		**0.001**
Body weight *(kg)*	Pre-Training	46.51 ± 10.70 47.00 (26.00-66.00)	35.47 ± 7.80 33.00 (26.00-57.30)	**0.001**	42.23 ± 11.02 40.80 (26.00-66.00)
	Post-Training	54.61 ± 10.14 53.50 (37.00-75.00)	39.19 ± 7.18 37.00 (30.00-59.00)	**0.001**	48.63 ± 11.79 47.00 (30.00-75.00)
	p^2	**0.001**	**0.001**		**0.001**
Body Mass Index *(kg/m²)*	Pre-Training	19.80 ± 2.74 19.48 (14.06-24.77)	17.55 ± 2.34 17.01 (14.27-24.20)	**0.002**	18.93 ± 2.80 18.12 (14.06-24.06)
	Post-Training	20.84 ± 2.47 20.87 (17.09-27.14)	18.09 ± 1.93 17.72 (15.53-24.56)	**0.001**	19.77 ± 2.63 18.87 (15.53-27.14)
	p^2	**0.004**	**0.009**		**0.001**
Waist circumference *(cm)*	Pre-Training	74.23 ± 6.74 72.00 (64.00-90.00)	65.79 ± 3.99 66.00 (61.00-75.00)	**0.001**	70.96 ± 7.12 70.00 (61.00-90.00)
	Post-Training	75.42 ± 5.68 73.00 (64.00-87.00)	66.16 ± 2.44 67.02 (60.70-73.00)	**0.001**	70.79 ± 3.10 70.01 (60.70-87.00)
	p^2	0.872	0.695		0.788
Leptin *(ng/mL)*	Pre-Training	0.46 ± 0.56 0.15 (0.10-2.30)	1.11 ± 1.23 0.90 (0.10-5.6.30)	**0.006**	0.71 ± 0.93 0.40 (0.10-5.60)
	Post-Training	0.51 ± 0.66 0.16 (0.10-3.20)	0.86 ± 1.04 0.60 (0.10-4.70)	**0.048**	0.64 ± 0.84 0.30 (0.10-4.70)
	p^2	0.608	0.356		0.602

Data presented as Mean ± SD, Med. (Min-Max), Pre= Before the training, Post= After the training
p^1: Between pubertal and prepubertal, p^2: Between before the training and after the training
** $p<0.05$*

school-aged children, found that leptin mediates the relationship between body fat and blood pressure. Their findings suggest that leptin mediates the relationship between body fat and blood pressure, thereby highlighting leptin as a mediator that links body adiposity with blood pressure elevation (24).

The studies suggested that leptin and adiponectin responses and adaptations differ in acute and chronic exercises. Acute exercise studies: for leptin, it appears that a lowered leptin concentration was observed after long-term exercise (>60 min) that stimulates FFA release, or after exercise that generates an energy

expenditure higher than 800 kcal. Therefore, it seems that the leptin concentration is not modified after short-term exercise (60 min) or exercise that generates an energy expenditure lower than 800 kcal (25).

In terms of different exercise regimes, Lin et al meta-analysis demonstrated that exercise training of 8 weeks' duration or more might have a greater effect on improving adiponectin levels (26); this finding is in line with the finding of Salles et al., who concluded in their meta-analysis that training duration may affect the response of adipose tissues, with greater change occurring after 16 weeks or more of training (27). The duration and frequency of the exercise also seem to be crucial for leptin, since the participants with more than 8 weeks of intervention and 60 min or more per week of training yielded a higher profit of exercise on reducing leptin levels (26).

Further studies and reviews should be conducted on understanding the risks and benefits of exercise as thoroughly as possible. Only in this manner the right dose of physical activity can be achieved (28). The decrease in leptin levels with the onset of puberty is significant ($p<0.05$). The leptin levels did not change after exercise ($p>0.05$). After exercise, leptin levels were found to be significantly lower in the pubertal group ($p= 0.048$). Physiologically changes in hormonal, anthropometric, and adipose tissue reveal this result in puberty. We think that leptin levels and adipose tissue and muscle tissue compositions in puberty cause this. The effects of exercise are also remarkable. The increase of growth hormone, testosterone, and cortisol levels after a short bout of maximal exercise in athletes is often observed and described in the literature (29, 30). Taking into account that immediate post exercise increases in growth hormone and testosterone values were significantly higher after the 24-week heavy training period and that immediate post exercise increases in these anabolic hormones were also higher after the 24-week heavy training period. Alzamil et al in their research It appears that resting adiponectin does not change as a result of the heavy training period in highly trained rowers with different performance levels. Specifically, heavy training periods caused significantly higher post exercise values in rowers with better performance capacity, whereas significant decreases in post exercise adiponectin and leptin values were observed in rowers with lower performance capacity. This was thought to reflect the inadequate recovery capacities of these rowers (17).

When the results of the study were examined, body weight, height, BMI, and waist circumference increased significantly with the onset of puberty ($p<0.05$). Although waist circumference increased significantly with puberty ($p<0.05$), it did not change with exercise ($p>0.05$). There are several reports of leptin concentrations being related to changing body composition (BMI, body fat) and waist circumference in children and adolescents (31, 32). Johns et al found that leptin concentrations were significantly correlated with pre- and post-training weights and with pre- and post-waist circumferences which did not change with long-term exercise training. In their study, percent body fat correlated with leptin concentration before long-term exercise training ($r= 0.832$, $p\leq0.001$) and remained correlated ($r= 0.872$, $p\leq0.001$) after a significant decrease in percent body fat (2.2%) (33). It has been demonstrated that leptin concentrations remain correlated with fat mass after a small fat loss in children (mean age 9 years) (34).

The growth hormone appears to have a negative feedback loop with leptin as leptin treatment stimulates the production of growth hormone from the pituitary by inhibiting hypothalamic somatostatin production and stimulating the production of growth hormone releasing hormone (35). Since our study group was adolescent, the decrease in leptin levels after eight months of exercise can be thought to be caused by growth hormones.

The implication of leptin in physical exercise and during its recovery is still unclear. Several reasons that can explain the modification of the response of leptin to muscular exercise. Physical exercise and or training can reduce fat mass, play a significant role in energy expenditure and affect hormonal concentrations (insulin, cortisol, growth hormone, catecholamine, testosterone, etc.) and metabolites (free fatty acids, lactic acid, triglycerides, etc.). For all these reasons physical exercise and training could modify the leptin response depending on several factors (2).

Conclusion

In our study, there was statistical significantly increase in height, weight, waist circumference, and BMI results of male adolescent wrestlers who did wrestling training for eight months ($p<0.05$). No statistical change was found between leptin levels ($p>0.05$), while leptin levels decreased significantly with puberty ($p<0.05$). Although some studies indicate that there is a strong relationship between BMI and leptin level, such a relationship was not found in our study. This situation is thought to be caused by physiological changes that are important and effective in adolescence.

The discrepancy in the literature is probably related to several factors such as gender, the intensity and duration of exercise, the nutritional status of the subject, the circadian rhythm of leptin, the sample group and the number of the population studied, the hour of blood sampling, and the calorie imbalance brought about by exercise.

More research and investigation should be done to understand the risks and benefits of exercise as comprehensively as possible. Only in this way the correct dose and effect of physical activity can be achieved.

Acknowledgement

The study was financially funded by Zonguldak Bülent Ecevit Üniversitesi, Scientific Research Coordination Department with number 2011-Y1-00-01.

Blood analyzes were performed at Zonguldak Bülent Ecevit University Health Application and Research Center Biochemistry Laboratory, at the cost of payment. We would like to thank Prof. Dr. Murat CAN for his assistance in this process and his sensitivity for the quality and reliability of the measurements.

Conflicts of Interest

The authors declare that there is no conflict of interest about this manuscript.

References

1. Fedewa MV, Hathaway ED, Ward-Ritacco CL. et al. The Effect of Chronic Exercise Training on Leptin: A Systematic Review and Meta-Analysis of Randomized Controlled Trials. Sports Med 2018; 48: 1437–1450.
2. Bouassida A, Zalleg D, Bouassida S, et al. Leptin, its implication in physical exercise and training: a short review. J Sports Sci Med. 2006; 5(2): 172–181.
3. Kelly T, Yang W, Chen C, Reynolds K, He J. Global burden of obesity in 2005 and projections to 2030. Int J Obes. 2008; 32(9): 1431–7.
4. Benjamin RM. The Surgeon General's vision for a healthy and fit nation. Public Health Rep. 2010; 125(4): 514.
5. Spruijt-Metz D. Etiology, Treatment and Prevention of Obesity in Childhood and Adolescence: A Decade in Review. J Res Adolesc. 2011; 21(1): 129–152.
6. Meldrum DR, Morris MA, Gambone JC. Obesity pandemic: causes, consequences, and solutions-but do we have the will?. Fertil Steril. 2017; 107: 833–9.
7. Reilly SM, Saltiel AR. Adapting to obesity with adipose tissue inflammation. Nat Rev Endocrinol. 2017; 13: 633–43.
8. Klok MD, Jakobsdottir S, Drent ML. The role of leptin and ghrelin in the regulation of food intake and body weight in humans: a review. Obes Rev. 2007; 8(1): 21–34.
9. Hamrick MW. Role of the cytokine-like hormone leptin in muscle-bone crosstalk with aging. J Bone Metab. 2017; 24: 1–8.
10. Guerra B, Santana A, Fuentes T. et al. Leptin receptors in human skeletal muscle. J Appl Physiol. 2007; 102: 1786–92.
11. Sinha MK, Sturis J, Ohannesian J. et al. Ultradian oscillations of leptin secretion in humans Biochem Biophys Res Commun. 1996; 228: 733–738.
12. Ducy P, Amling M, Takeda S. et al. Leptin inhibits bone formation through a hypothalamic relay: a central control of bone mass. Cell. 2000; 100: 197–207.
13. Turner RT, Kalra SP, Wong CP. et al. Peripheral leptin regulates bone formation. J Bone Miner Res. 2013; 28; 22–34.
14. Galic S, Oakhill JS, Steinberg GR. Adipose tissue as an endocrine organ. Mol Cell Endocrinol. 2010; 316(2): 129–39.
15. Hilton LK, Loucks AB. Low energy availability, not exercise stress, suppresses the diurnal rhythm of leptin in healthy young women. Am J Physiol Endocrinol Metab. 2000; 278(1): E43–9.
16. Marshall WA, Tanner JM. Variations in the pattern of pubertal changes in boys. Arch Dis Child 1970; 45: 13–23.
17. Alzamil H, Aldokhi L, Habib SS. Physical Fitness and Its Relationship to Plasma Leptin, Leptin Soluble Receptor, and Free Leptin Index in a Saudi Population: A Comparison Between Diabetic and Non-Diabetic Individuals. Medical science monitor basic research. 2018; 24: 113–119.

18. Plonka M, Toton-Morys A, Adamski P, Suder A, Bielanski W, Dobrzanska MJ, Kaminska A, Piorecka B, Glodzik J. J Physiol Pharmacol. 2011; 62(6): 647–56.
19. Jiménez-Pavón D, Ortega FB, Artero EG, et al. HELENA Study Group. Physical activity, fitness, and serum leptin concentrations in adolescents. J Pediatr. 2012; 160(4): 598–603.
20. Miyatake N, Murakami H, Kawakami R. et al.. Circulating leptin levels are associated with physical activity or physical fitness in Japanese. Environmental health and preventive medicine. 2014; 19(5): 362–366.
21. Considine RV, Sinha MK, Heiman ML, et al. Serum immunoreactivity-leptin concentrations in normal-weight and obese humans. N Engl J Med 1996; 334: 292–5.
22. Hirose H, Saito I, Kawai T, et al. Serum leptin level: possible association with hematopoiesis in adolescents, independent of body mass index and serum insulin. Clin Sci (Lond). 1998; 94: 633–6.
23. Grontved A, Steene-Johannessen J, Kynde I, et al. Association between plasma leptin and blood pressure in two population-based samples of children and adolescents. J Hypertens. 2011; 29: 1093–100.
24. Fujita Y, Kouda K, Ohara K. ae al. Leptin mediates the relationship between fat mass and blood pressure: The Hamamatsu School-based health study. Medicine. 2019; 98(12): e14934.
25. Bouassida A, Chamari K, Zaouali M, et al. Review on leptin and adiponectin responses and adaptations to acute and chronic exercise. British Journal of Sports Medicine. 2010; 44: 620–630.
26. Lin H, Hu M, Yan Y. et al. The effect of exercise on adiponectin and leptin levels in overweight or obese subjects: a meta-analysis of randomized controlled trials. Sport Sci Health. 2017; 13. 303–314.
27. de Salles BF, Simao R, Fleck SJ, et al. Effects of resistance training on cytokines. Int J Sports Med. 2010; 31: 441–450.
28. Bird L. Exercise lowers leptin and leukocytosis. Nat Rev Immunol. 2020; 20(1): 2–3.
29. Kraemer RR, Aboudehen KS, Carruth AK. et al. Adiponectin responses to continuous and progressively intense intermittent exercise. Med Sci Sports Exerc. 2003; 35: 1320–1325.
30. Hoogeveen AR, Zonderland ML. Relationships between testosterone, cortisol and performance in professional cyclists. Int J Sports Med. 1996; 17: 423–428.
31. Pilcova R, Sulcova J, Tepesi M. et al. Leptin levels in obese children: effects of gender, weight reduction and androgens. Physiol Res. 2003; 52(1): 53–60.
32. Tsolakis C, Vagenas G, Dessypris A. Growth and anabolic hormones, leptin, and neuromuscular performance in moderately trained prepubescent athletes and untrained boys. J Strength Cond Res. 2003; 17: 40–46.
33. Jones TE, Basilio JL, Brophy PM. et al. Long-term exercise training in overweight adolescents improves plasma peptide YY and resist in. Obesity (Silver Spring, Md.). 2009; 17(6): 1189–1195.
34. Gutin B, Ramsey L, Barbeau P et al. Plasma leptin concentrations in obese children: changes during 4-mo periods with and without physical training. Am J Clin Nutr 1999; 69: 388–394.
35. Cocchi D, De Gennaro Colonna V, Bagnasco M. et al. J Endocrinol. 1999; 162(1): 95–9.

Correspondence:
Tevfik Cem Akalin
School of Physical Education and Sports, Zonguldak Bülent Ecevit University, Zonguldak, Turkey
E-mail: tc.akalin@hotmail.com
Mobile phone: +905323414259