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Review Article

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Investigating the Effect of Physical Activity and Exercise on Serum Leptin Levels

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ABSTRACT

Leptin is derived from the Greek word leptose, which means thinness, and was discovered in 1994 by isolating the obesity gene. This substance is a protein hormone with a helical structure similar to cytokines, which is mainly synthesized and released by the subcutaneous fat cells in a constant pulsating manner with a peak secretion near midnight. A key factor in controlling energy and metabolism, which has an impact on body weight, is leptin. Leptin, which is produced by the obesity gene, has been the subject of extensive investigation in recent times. Researchers have reported contradictory findings about the effect of physical activity in different situations on the regulation of leptin secretion from adipose tissue sources. This review article is focused on conflicting studies and the effect of different intensities of physical activity on leptin and leptin's response to intense activity and training periods. This study reviews research that has examined the effect of various types of exercise on leptin in the form of a literature review. Recent reports on the effect of physical activity and exercise on leptin concentrations are conflicting. Some researchers have determined that exercise can reduce leptin concentration, depending on the duration of physical activity and calories consumed. While some researchers have not reported any change in leptin concentration. The present review indicates that physical activity longer than 60 minutes with substantial energy expenditure (>800 kcal) of exercise is required to observe reductions in plasma leptin concentrations in non-athletes.

Key words: *Serum leptin, Physical activity, Exercise, Body weight*

INTRODUCTION

Different eating habits and amounts of physical activity affect the body fat balance. The secretion and action of several hormones that control the collection and storage of nutrients, the recall and use of fuel, or regenerative and hypertrophic growth influence this condition. In the past half-century, energy has been increasingly concentrated in areas of the body where fat can accumulate and be stored, causing obesity in 50% of adults. This obesity rate suggests that very low energy regulatory mechanisms are influenced by common lifestyle conditions [1-3]. Humans have an energy control mechanism that is adapted to optimize energy intake and nutrient storage when an abundance of foods high in energy are accessible. When a sedentary lifestyle and an abundance of rich meals replace food shortages, energy buildup and storage are maximized in certain genetic variants of humans [4, 5]. To some extent, it can be said that the two features of the long-term energy regulation mechanism, which include the reasons for the increasing prevalence of obesity in today's societies, are: first, the stimulation caused by mainly fatty nutritional meals (which enables the synthesis and storage of fat) and secondly, the non-

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homeostatic relationship of obesity with inactivity and reduced sensitivity of hormonal recall of fuel reserves [6, 7].

Leptin is derived from the Greek word leptose, which means thinness, and was discovered in 1994 by isolating the obesity gene. This substance is a protein hormone with a helical structure similar to cytokines [8-10], which is mainly synthesized and released by the subcutaneous fat cells in a constant pulsating manner with a peak secretion near midnight. According to the family to which it belongs, it can be an internal regulator of the thymus gland and the factors secreted during the reaction such as interleukin (IL (1)) and tumor necrosis factor-alpha (INF). Since the increase in plasma concentration of Leptin is proportional to the fat content of adipocytes, and the degree of obesity is affected by dietary interventions or daily exercise, leptin can report the long-term status of body adipose tissue accumulation to the brain. Obesity in ob/ob mice and some people is caused by leptin gene mutation, which results in incomplete protein synthesis [11, 12].

As mentioned, the level of leptin hormone can be affected by the nutritional status, neuroendocrine, and immune function of the body. In addition, hormones such as sex hormones, catecholamines, and thyroid hormones play a role in leptin regulation. These hormones are effective by regulating the gene responsible for leptin production. In addition, cortisol and growth hormones are the most important hormones that help increase leptin secretion [11, 13-15]. There is a relationship between leptin changes with negative energy balance, sympathetic activity, and some metabolites. Among the potential regulators of leptin secretion are stress caused by exercise, changes in fuel displacement, systemic hormone concentrations, and the effect of energy consumption. Reduction of fat mass is one of the reasons that changes leptin levels [8, 16, 17]. Although some research has shown that short-term exercise cannot affect leptin secretion, short-term changes in energy balance caused by exercise have an effect on leptin secretion at night. The negative energy balance that occurs with physical activity or reduced energy intake suppresses nocturnal leptin secretion. While the energy balance leads to an increase in the nocturnal secretion of leptin [18, 19].

Considering the many questions that exist in this connection, there is a need to review and summarize the studies done in this field. In this article, firstly, the effect of various types of physical activity on leptin and the results obtained from them are examined and analyzed, then by combining these studies, a general conclusion of the effect of activity on leptin and, consequently, fat tissue is discussed.

RESULTS AND DISCUSSION

Physical activity is the most important factor in energy consumption in humans since energy intake can positively or negatively regulate the leptin gene expression; it is possible that the change in energy consumption through exercise also affects the level of leptin. Physical activity can be considered an important determinant of leptin levels [18, 19]. Stress caused by exercise is a potential regulator of leptin secretion. Physical activity can change leptin concentrations by the changes that fuel flow creates in circulating hormone concentrations and energy consumption caused by physical activity. Most of the research has confirmed the effect of exercise on reducing leptin concentration. Some researchers have also failed to prove its effect. The research conducted about the effect of long-term training periods has less contradiction and most researchers emphasize the positive effect of this type of training period on the reduction of serum leptin levels [11]. Ferdosi *et al*. [19] conducted a study in which 48 men were placed in 4 groups endurance training (75% to 80% HR), resistance training (70% IRM), combined training, and a control group. All 3 methods caused a significant decrease in leptin levels [19]. Rashidlamir and Saadatnia [20, 21] also reported an increase in the concentration of adiponectin and resistin, a decrease in leptin concentration, and a decrease in fat percentage after 8 weeks of aerobic training 4 days a week with 60% to 80% of the maximum heart rate [20, 22]. In Ghadiri *et al*. [21] research, aerobic exercise with higher intensity had a far more favorable effect on obesity and overweight than aerobic exercise with lower intensity and was more effective in reducing fat percentage and leptin levels [21, 23]. Azizi reported a decrease in serum leptin during 8 weeks of aerobic training, 3 sessions per week and 30 minutes each session, with 65% to 85% of the maximum heart rate [3, 24]. In the Hamedi Nia *et al*. [25] study, 60 students in 4 groups participated in preliminary swimming exercises for 8 weeks. The result was the prevention of a significant increase in leptin and insulin in adolescents, and a reduction in fat percentage and BMI in children and adolescents [25]. Shahidi *et al*. observed a significant decrease in serum leptin in obese women after 12 weeks of aerobic training, 3 sessions per week [11]. In addition, in a study, after 13 weeks of aerobic training with 75-80% of the maximum heart rate, a significant decrease in serum leptin was observed in obese men [26]. Polak *et al*. [27] trained 25 non-menopausal obese women 5 days per week with 50% Vo2max intensity (2 days of aerobic exercise and 3 days of stationary bike) for 12 weeks, the

result of which was a decrease in serum leptin, an increase in Vo2max, an increase in insulin sensitivity, no change in Abdominal subcutaneous fat tissue mRNA and genes and no change in plasma adiponectin [27, 28]. In another study, Kumru *et al*. [29] investigated long-term leptin changes. In this way, the first group had 10 hours of training per week for 5 years, the second group had 10 hours of training per week for more than 5 years, and the third group was the control group, which resulted in a decrease in serum leptin levels and a decrease in BMI and an increase in testosterone levels. It was experimental in both groups [29, 30]. Tomofusa *et al*. [31] did research on diabetic people in which 2 diet groups, one only with diet therapy and the other group with full diet therapy, performed aerobic exercise at least once a week for 8 weeks, showed that it led to a significant decrease in leptin in the training group and a decrease in weight and fat percentage in all subjects [31]. On the other hand, some research had contradictory results compared to the mentioned research. For example, Bizheh *et al*. conducted a study on thin middle-aged women in which they performed aerobic exercises for 6 months, 3 sessions a week, and each session lasted 60 minutes with an intensity of 60 to 70% of the maximum heart rate, and the result was no decrease in mean heart rate. It showed the concentration of leptin, cortisol, insulin, and blood glucose [32]. Saremi *et al*. [33] also observed no change in leptin and ghrelin levels, improved sleep quality, improved body mass index, visceral fat, and total abdominal fat after 12 weeks of aerobic training, 3 days a week for 50 to 60 minutes each day [33]. After 8 weeks of running training, 4 sessions of 30 minutes each week with an intensity of 55% to 75% of the maximum heart rate in women with normal weight, there was no significant change in serum leptin in women [34, 35].

In an example of conflicting research, Houmard *et al*. [36] gave two groups of young and old a short-term exercise session with an intensity of 75 Vo2max on a bicycle, the result of which was no change in leptin concentration in young men, old men and women, and an increase in insulin action [36]. Based on the aforementioned findings, aerobic activity typically results in a reduction of leptin. Additionally, some study has indicated a number of reasons why leptin does not change. For example, in Saremi's research, appetite status was not directly measured. Although in this study, the quality of sleep improved after exercise, but still in terms of the PSQI index, which is defined as low-quality sleep, the lack of change in the leptin hormone may be related to this factor [33]. In the special research, considering that, blood sampling was done 48 hours before and 48 hours after the training period at the same time of the day and in the fasting state, therefore, the change in the circadian cycle of leptin did not affect the results of the research. The researcher attributed the lack of change in serum leptin to the reduction of BMI in thin women [32]. In Shahidi's *et al*. [11] research, the reason for the non-significance of cortisol, the intensity of exercise applied to the subjects, and the non-significance of insulin were related to the duration and intensity of the activity and the gender of the subjects. The reason for the non-significance of BMI after aerobic exercise was the low intensity and duration of the activity [11].

Also, Kumru *et al*. [29] stated that the cause of BMI decrease is the increase in energy consumption during activity and also the decrease in leptin levels, which itself leads to a decrease in fat tissue content and a decrease in BMI [29]. Bnary Carami *et al*. attributed the non-significance of insulin reduction, body fat percentage, and glucose and insulin resistance in the experimental group to the low intensity of training, age, and disease conditions of the subjects [37]. In RamezanKhani *et al*. [38] study, the reason for the contradiction with other articles may be due to the short training periods in other articles. It is possible to argue that other potential factors, such as gender, age, race, the subjects' fat percentage, the intensity and duration of the exercises, and the amount of fats received, can also alter the profile of lipids and have an impact on study results given the lack of significant changes in blood lipids following aerobic exercise and a low-calorie diet. In Hamedi Nia *et al*.'s research, the non-significance of the change in growth hormone was attributed to the lack of duration and intensity of activity [25]. Ghadiri *et al*. [21] mentioned the intensity, volume, and certain duration of activity as factors affecting leptin levels [36]. Aging is related to the possible mechanism of the effects of exercise on leptin levels in obese and lean people with differences in the sympathetic nervous system and the hypothalamic-hypothyroid-adrenal axis [21]. Tomofusa believes that since exercise leads to a greater decrease in visceral fat than subcutaneous fat, the decrease in serum leptin cannot be attributed to the decrease in the size of fat cells [31]. In general, aerobic exercise can reduce serum leptin. The mechanism of effect of resistance training on serum leptin is different compared to aerobic training. Compared to aerobic exercise, resistance training is a potent non-oxidative stimulus that produces different neurometabolic and photoendocrine responses. More specifically, non-oxidative resistance training produces higher levels of lactate, glucagon, cortisol, and growth hormone and leads to higher post-exercise oxygen consumption and higher adrenal sympathetic activity than aerobic running. Furthermore, whereas ATP synthesis during severe resistance exercise is predominantly dependent on the usage of creatine phosphate, glucose, and glycogen consumption, submaximal aerobic exercise depends on the mobility of fat. Through glycogen depletion,

suppression of glycolysis, and enhanced glucose uptake in the presence of lactate, acidosis, and ketoclamines, resistance exercise lowers serum leptin levels. However, different results have been obtained regarding the response of leptin to resistance training [21, 39, 40].

A lot of research has been done about leptin and its influencing factors. For example, Yesim Ozarda *et al*. [41] measured the levels of leptin, adiponectin, non-esterified fatty acids, phospholipids, IGF1 and the antioxidant capacity of capillaries in children aged 4 to 30 days, and the results showed that there is a positive relationship between the concentration of leptin and adiponectin, and the opposite relationship between the concentration of resistin and leptin and adiponectin concentrations, there is a positive relationship between vascular antioxidant concentrations with leptin and adiponectin concentrations and an inverse relationship with resistin concentrations [41]. Park *et al*. [42] measured the levels of leptin, insulin, adiponectin, and lipid profiles in 7-year-old children from 2005 to 2009. The results showed that the increase in leptin concentration among children is a sign of future BMI and metabolic disorders [42]. Comparing two groups of active and inactive girls aged 9 to 16 years, Plonka *et al*. [43, 44] stated that increased exercise activity decreases leptin, body fat, WHtR, and FFM. In the research of Van Weyenberg *et al*. [45], 9 healthy foals were fed two ways: 140% of the requirement and 75% of the requirement. In the high-calorie diet (140%), serum leptin increased at the beginning of the diet, but after 7 days it returned to the original state, in the low-calorie diet (75%), plasma leptin decreased. The weight did not change in any of them [45]. In determining the relationship between serum leptin concentration and psychological stress in Japanese men, Otsuka *et al*. [44, 46] showed that stress increased leptin levels and blood chemical levels [46]. In his research, Van Weyenberg *et al*. [45] considered the lack of significant changes in T and T4 due to the short period or small changes in the low-calorie diet (75%) [45]. In his cross-sectional study, Aref Hossaini *et al*. [47] stated that leptin levels and insulin resistance were not significant, and the reason for the discrepancy with other studies was the study population. Considering the age range of the studied women, perhaps the better lipid pattern and the lack of a significant relationship between leptin and the studied parameters can be justified to some extent [47].

CONCLUSION

Finally, it can be said that the reduction of leptin concentration through exercise is associated with changes in energy balance, improvement in insulin sensitivity, and changes in related hormones or carbohydrate and fat metabolism. Examining the changes of leptin with obesity is one of the topics that are of particular importance in adolescent obesity; therefore, to change the levels of leptin and related hormones such as insulin, thyroxine, triiodothyronine, and cortisol, the intensity and duration of the exercise program should be appropriate. The results obtained from the effect of aerobic exercises with high concentrations of leptin show significant changes. The interesting thing to note in this type of exercise is the improvement of maximum oxygen consumption, which can be considered an important indicator in improving the level of health along with weight loss due to the effect of this type of exercise on body fat tissue. Of course, hormonal and metabolic changes resulting from resistance training are completely different from aerobic training. Most of the research conducted using this type of exercise shows its positive effects on some metabolic indicators. Although the amount of change in serum leptin has been reported to be less compared to aerobic training, the correlation of leptin changes with BMI and WHR and on the other hand the significant reduction of these two variables, promise the possible improvement of serum leptin levels as a result of this type of training using Gives higher energy consumption. If the diet is controlled, you can expect weight and body fat loss from the resistance training program. Decreased WHR, as an indicator of visceral fat, probably reflects the effect of resistance training on reducing visceral fat. In general, most research has shown that a balanced low-fat diet and physical exercise lower blood leptin levels. The changes in leptin with exercise activity and the time to return to the initial state are still under a veil of ambiguity. Several reasons have been proposed for the changes in leptin response to exercise. Sports activity can reduce fat mass and play an important role in energy consumption. It can also affect the concentration of hormones (insulin, growth hormone cortisol, congolamines, aldosterone, sex hormones, testosterone, triiodothyronine, thyroxine, etc.) and metabolism (free fatty acids, lactic acid, triglycerides, etc.). Comparing the effects of various training programs on leptin in fat and thin people, men and women, and at different ages is considered an important research field. In addition, applying this method with a low-calorie diet at the same time and comparing it with physical activity alone can also be considered interesting topics for future research.

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