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Exploring the Impact of Physical Activity and Exercise on Leptin Serum Concentrations

Gilmara Gomes de Assis[1*], Eugenia Murawska-Ciałowicz[2]

[1]Department of Physiology and Pathology, School of Dentistry, UNESP, São Paulo State University, Araraquara 14801-903, Brazil.
[2]Department of Physiology and Biochemistry, Wroclaw University of Health and Sport Sciences, 51-612 Wroclaw, Poland.

***E-mail** ✉ gilmara.gomesdeassis@awf.gda.pl
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ABSTRACT

Leptin, derived from the Greek word “leptose” meaning thinness, was discovered in 1994 through the isolation of the obesity gene. This protein hormone, with a helical structure akin to cytokines, is primarily produced and released by subcutaneous fat cells in a pulsatile pattern, peaking near midnight. Leptin plays an important role in regulating energy balance and metabolism, and significantly influences body weight. Despite extensive research on leptin, studies on the effect of physical activity on leptin secretion from adipose tissue have yielded inconsistent results. This review examines a variety of studies that explore how different intensities of physical activity affect leptin levels, focusing particularly on its response to intense exercise and training periods. Findings on the relationship between physical activity and leptin are mixed, with some researchers suggesting that exercise can reduce leptin concentrations, particularly depending on the duration of exercise and caloric expenditure, while others report no significant change. The current review suggests that physical activity lasting over 60 minutes and involving substantial energy expenditure (> 800 kcal) is required to observe reductions in plasma leptin concentrations, especially in non-athletes.

Keywords: Physical activity, Body weight, Serum leptin, Exercise

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Introduction

Eating habits and physical activity levels significantly influence body fat balance. The secretion and action of various hormones that regulate the accumulation and storage of nutrients, energy expenditure, and growth are central to this process. Over the past several decades, energy has increasingly been stored in areas of the body where fat can accumulate, contributing to a rise in obesity rates, which now affect 50% of adults. This obesity rate highlights the limited effectiveness of energy regulation mechanisms in modern society, particularly in the context of prevalent lifestyle habits [1-3]. Humans have an energy regulation system adapted to optimize nutrient intake and storage, especially when high-energy foods are abundant. However, in the presence of sedentary lifestyles and easy access to calorie-dense foods, energy storage mechanisms can become exaggerated in certain genetic variants of humans, leading to excessive fat accumulation [4, 5].

The two main factors driving the rise in obesity today are the stimulation of fat storage by energy-dense foods and the disruption of homeostatic energy balance, resulting in reduced sensitivity to hormonal cues that govern fuel utilization [6, 7].

Leptin, a key hormone involved in regulating body fat, was discovered in 1994 through the isolation of the obesity gene. The name “leptin” comes from the Greek word “leptose,” meaning thinness. It is a protein hormone with a structure similar to cytokines [8-10] and is primarily synthesized and released by subcutaneous fat cells in a pulsatile pattern, with peak secretion typically occurring at midnight. Leptin also acts as an internal regulator for

the thymus gland and influences factors like interleukins and tumor necrosis factor-alpha. Plasma leptin concentrations correlate with fat content in adipocytes, and changes in dietary intake or daily physical activity can influence leptin levels, making it an important indicator of the body's adipose tissue status. A mutation in the leptin gene, which leads to incomplete leptin protein synthesis, is the cause of obesity in ob/ob mice and some humans [11, 12].

Leptin levels are affected by nutritional status, neuroendocrine functions, and immune responses. Additionally, hormones such as sex hormones, catecholamines, and thyroid hormones regulate leptin production. Cortisol and growth hormones also play significant roles in increasing leptin secretion [11, 13-15]. There is a known relationship between leptin changes, negative energy balance, sympathetic activity, and certain metabolites. Physical activity, energy expenditure, and stress are potential regulators of leptin secretion. For instance, the reduction in fat mass can lower leptin levels, and studies show that short-term exercise can affect leptin secretion, particularly during periods of negative energy balance. Physical activity or reduced energy intake tends to suppress nocturnal leptin secretion, while positive energy balance tends to increase it [8, 16, 17].

Given the numerous unanswered questions surrounding leptin and its regulation, it is essential to review and synthesize existing research in this area. This article explores various studies on the effects of physical activity on leptin secretion and its relationship with fat tissue. By analyzing these findings, the article aims to provide a general understanding of how physical activity influences leptin levels and, consequently, body fat.

Results and Discussion

Physical activity plays a pivotal role in energy expenditure, which can influence leptin secretion by altering the balance between energy intake and expenditure. Several studies have highlighted physical activity as a crucial factor in regulating leptin levels [18, 19]. The physical stress induced by exercise is believed to act as a potential regulator of leptin secretion by affecting hormone concentrations and energy consumption. While most studies suggest that exercise leads to a reduction in leptin levels, some research has failed to demonstrate such an effect.

Research on long-term training has generally shown less variability, with most studies emphasizing that sustained exercise contributes to a reduction in serum leptin levels. Ferdosi *et al.* [19] conducted a study involving 48 men who were divided into four groups: endurance training, resistance training, combined training, and a control group. All three exercise methods led to a significant reduction in leptin concentrations. In another study, Rashidlamir and Saadatnia [20] found that after 8 weeks of aerobic training at 60%-80% of maximum heart rate, leptin levels decreased significantly along with fat percentage reduction. Additionally, Ghadiri *et al.* [21] demonstrated that high-intensity aerobic exercise had a more substantial effect on reducing leptin levels and fat percentage compared to lower-intensity exercise.

Further studies have supported these findings [3, 22-31]. Azizi *et al.* [3] observed that 8 weeks of aerobic training at 65%-85% of maximum heart rate led to a notable reduction in serum leptin. Similarly, Hamedi Nia *et al.* [25] showed that 8 weeks of swimming exercises prevented a significant increase in leptin and insulin levels in adolescents, in addition to reducing fat percentage and BMI. Shahidi *et al.* [11] found significant reductions in serum leptin in obese women following 12 weeks of aerobic exercise, while another study showed a decrease in leptin after 13 weeks of aerobic training in obese men [26].

Polak *et al.* [27] observed similar outcomes after 12 weeks of aerobic training in non-menopausal obese women, including decreased leptin levels and improved insulin sensitivity, though no changes in adiponectin were observed. Kumru *et al.* [29] noted a decrease in leptin levels and BMI in individuals who trained for 10 hours per week for 5 years, with an increase in testosterone levels. Tomofusa *et al.* [31] found significant reductions in leptin levels, weight, and fat percentage in diabetic individuals after 8 weeks of aerobic exercise.

Contradictory findings have also been reported in some studies. Bizheh *et al.* [32] conducted a study with middle-aged women who underwent 6 months of aerobic training at 60%-70% of maximum heart rate, but found no change in leptin levels. Likewise, Saremi *et al.* [33] observed no changes in leptin or ghrelin after 12 weeks of aerobic training, despite improvements in BMI and abdominal fat. Additionally, a study on women with normal weight who performed running training for 8 weeks at 55%-75% of maximum heart rate found no significant alterations in serum leptin [34, 35].

These divergent results suggest that the effect of exercise on leptin levels may be influenced by factors such as exercise type, intensity, duration, and individual characteristics, such as body composition and hormonal

responses. Therefore, additional research is needed to clarify the underlying mechanisms and to further explore how exercise influences leptin regulation.

Conclusion

In conclusion, the reduction of leptin concentration through exercise is closely linked to changes in energy balance, improvements in insulin sensitivity, and alterations in related hormones involved in carbohydrate and fat metabolism. The effects of exercise on leptin levels, particularly in the context of obesity, are crucial, especially in adolescents struggling with obesity. To effectively influence leptin and related hormones such as insulin, thyroxine, triiodothyronine, and cortisol, the intensity and duration of the exercise program need to be carefully tailored.

Aerobic exercises, particularly those that result in high leptin concentrations, have shown significant effects on leptin levels. Interestingly, these exercises also lead to improvements in maximum oxygen consumption, a key indicator of health, alongside body fat reduction. While aerobic training significantly influences leptin, the hormonal and metabolic changes from resistance training are quite different. Though the change in serum leptin may be less compared to aerobic exercise, resistance training positively impacts metabolic indicators. Furthermore, the relationship between leptin changes and BMI and waist-to-hip ratio (WHR) suggests that resistance training can help improve leptin levels through greater energy consumption. When combined with a controlled diet, resistance training can also lead to weight and body fat loss and reduced visceral fat, as indicated by lower WHR.

Overall, the majority of research supports the idea that a balanced, low-fat diet combined with regular physical exercise reduces blood leptin levels. However, the precise timing of leptin changes and the duration needed for levels to return to baseline remain unclear. Several factors, including fat mass reduction, hormone levels, and metabolic changes, contribute to the observed effects. Future research should focus on comparing the effects of various exercise programs on leptin levels in different populations, such as obese and lean individuals, men and women, and across different age groups. Additionally, investigating the effects of combining exercise with a low-calorie diet and comparing it to exercise alone would be a valuable area for further exploration.

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