Physical activity and appetite modulation: The effects of physical exercise on appetite suppression

ABSTRACT
Objective: To discuss actions of physical exercise in modulating appetite and decreasing food intake, identifying appetite suppression mechanisms and their anorexigenic actions. Method: Literature review was carried out on Google Scholar, SciELO, Epub and PubMed, using the search descriptors: obesity, appetite regulation, physical exercise, and anorexigenic to verify the state of the art of the subject. Mostly, publications in Portuguese and English, book chapters and scientific articles were included. Result: Exercise is an important tool for the prevention and treatment of obesity, being an efficient tool for reducing body fat, providing anorectic action with suppression of appetite, consequently decreasing food intake. Conclusion: The appetite suppression effect generated by the reduction of ghrelin levels is not a rule, it depends on variables. The studied population, level of conditioning and intensity of activity are decisive in obtaining the effects of suppressing appetite and decreasing food consumption.

DESCRIPTORS: Obesity; Regulation of appetite; Physical exercise; Anorexigenic.

RESAMEN
Objetivo: discutir las acciones del ejercicio físico en la modulación del apetito y la disminución de la ingesta alimentaria, identificando los mecanismos de supresión del apetito y sus acciones anorexígenas. Método: Se realizó revisión de la literatura en Google Scholar, SciELO, Epub y PubMed, utilizando los descritores de búsqueda: obesidad, regulación del apetito, ejercicio físico y anorexígeno para verificar el estado del arte de la asignatura. En su mayoría, se incluyeron publicaciones en portugués e inglés, capítulos de libros y artículos científicos. Resultado: El ejercicio es una herramienta importante para la prevención y el tratamiento de la obesidad, siendo una herramienta eficaz para reducir la grasa corporal, proporcionando acción anoréxica con supresión del apetito, disminuyendo consecuentemente la ingesta de alimentos. Conclusión: El efecto de supresión del apetito generado por la reducción de los niveles de grelina no es una regla, depende de variables. La población estudiada, nivel de acondicionamiento y la intensidad de actividad son determinantes para obtener los efectos de supresión del apetito y disminución del consumo de alimentos.

DESCRIPTORES: Obesidad; Regulación del apetito; Ejercicio físico; Anorexígeno.

RESUMO

DESCRITORES: Obesidade; Regulação do apetite; Exercício físico; Anorexígenos.
INTRODUCTION

Obesity according to the World Health Organization - WHO is characterized by the excessive accumulation of body fat, which leads to an increase in weight and overweight of the individual. WHO1 own data report that obesity is currently considered a disease, as it is a global epidemic involving aspects of public health. This excessive accumulation of fat, which occurs largely in the intra-abdominal region and specifically the visceral region, is a major risk factor for the development and appearance of several types of cardiovascular and metabolic diseases and even some types of cancer. 2

The adoption of a sedentary lifestyle along with the adoption of bad eating habits, such as high consumption of high-calorie and high-lipid diets, are factors that trigger the positive energy balance and, consequently, are linked to the accumulation of body fat. 3 Thus, controlling appetite, calorie intake and physical exercise are important tools for the prevention and treatment of obesity. 4,5

Previous studies suggest that physical exercise can be an efficient tool for reducing body fat, providing an anorexigenic action, with suppression of appetite and consequently the reduction of food intake, such action happens, because muscle contraction performed during exercise is able to produce molecules responsible for interacting with the central nervous system (CNS) and some peripheral organs acting on the regulation of food consumption. Among them, we can mention the lactate that has been characterized as a possible molecule related to the suppression of appetite and consequently the reduction of food intake 6 the same being associated with interferences in ghrelin secretion. 7

In addition to the interruption of ghrelin production, different hormones with anorexigenic properties are secreted during exercise, we can mention PYY, GLP-1 and PP that actively act in suppressing appetite and reducing food intake. 6

Diante do exposto, o objetivo desse estudo é verificar as ações do exercício físico na modulação do apetite e na diminuição da ingestão de alimentos por meio da identificação dos mecanismos de supressão do apetite e as ações anorexigenas do mesmo. Para tanto foi realizada uma revisão de literatura por meio dos portais Google Scholar, Scielo, Epup e PubMed utilizando como descritores de pesquisa os termos: obesity, appetite regulation, physical exercise, e anorexigenic para verificar o estado da arte no qual a temática se encontra.

METHODS

The present study is a narrative literature review. This type of review allows for a broad analysis of the theme, being adequate to discuss a certain topic from a theoretical point of view. The narrative review of literature is appropriate to discuss the development or the “state of the art” of a theme, constituting, for the most part, the analysis of literature published in books, printed and/or digital magazine articles and interpretations through critical analysis of the study’s author. 8

The questions that guided the research were: What are the actions of physical exercise in modulating appetite and decreasing food intake? What are the anorexigenic actions of physical exercise? What are the mechanisms by which exercise suppresses appetite? Can this appetite suppression fight obesity?

For the selection of articles that addressed the topic discussed, a search was performed on the electronic database of the Google Scholar, Scielo, Epup and PubMed portals. The research descriptors used were: obesity, appetite regulation, physical exercise, and anorexigenic, with priority being given to articles published in Portuguese and in English without limitation regarding the date of publication. The searches were carried out between July and September 2020. Repeated studies and/or those that did not address the subject to be researched were excluded.

The evaluation for the selection of articles was carried out by the author, where the selection of articles was initially carried out by analyzing the titles, followed by the summary and when selected, by reading the articles in their entirety.

RESULTS

The electronic search on the portals in question resulted in the initial identification of 82 studies. When screening these works, using the selection criteria, a total of 25 selected articles were obtained for the study, in addition, two chapters from different books, an institutional website and another 12 articles were used to conceptualize the mechanisms and phenomena involved in the topic. The highest concentration and articles related to the theme were published between 2010 and 2018.

DISCUSSION

Obesity

According to the World Health Organization - WHO Obesity is characterized by the abnormal or excessive accumulation of fat caused by the energy imbalance between the calories consumed and the calories spent. This achievement has increased for two main factors, they are: the increase in the intake of foods very caloric and rich in fat and sugar; and the increase in physical inactivity - sedentary lifestyle - due to the increasingly sedentary nature of many forms of work, changes in modes of transport and increased urbanization.

WHO released extremely worrying data on obesity worldwide. According to the entity in 2016, more than 1,9 billion adults over 18 years of age were overweight. Of these, more than 650 million adults were already considered obese. In the same year, 39% of adults (39% of men and 40% of women) worldwide were overweight. Overall, on average 13% of the world’s adult population (11% of men and 15% of women) were obese in 2016. The global prevalence of obesity almost tripled between 1975 and 2016.

In addition to adults, children are also victims of this disease. In 2019, an estimated 38.2 million children under 5 years of age were overweight or obese and more
than 340 million children and adolescents from 5 to 19 years old were overweight or obese in 2016.1

The health consequences of an overweight or obese person are numerous. WHO cites some such as: cardiovascular diseases (mainly heart disease and strokes), type 2 diabetes mellitus, musculoskeletal disorders (especially osteoarthritis) and some types of cancer, such as endometrial, breast, ovarian, prostate, liver, gallbladder, kidney and colon.

Furthermore, in children the risks are also frightening. Childhood obesity is associated with a greater chance of obesity, premature death and disability in adulthood. But in addition to increasing future risks, obese children have breathing difficulties, increased risk of fractures, hypertension, early markers of cardiovascular disease, insulin resistance and psychological and socioaffective effects.1

Hunger and appetite

Evolutionarily, human beings have adapted themselves through the ages with food scarcity. In prehistory the primitive man always had to hunt to feed himself, however, if the hunt was unsuccessful he could sometimes go days without feeding, along with this, the energy spent to obtain food through hunting was huge. For that, many men died from the energy deficit, and those who survived were those who had the greatest capacity to store energy and use it in a time of scarcity. For this reason, it can be said that we are descendants of these human beings who had this energy storage capacity, which currently results in our body’s tendency to accumulate energy.9

For that, it is known that our organism acts in a way to try in several possible ways to preserve our energy reserves, that is, to preserve our fat reserve, triggering for this act the stimulus to food consumption and the reduction of energy expenditure.9

Therefore, one of the mechanisms that our body uses is to induce hunger and appetite. According to Coelho and Lancha Jr., hunger is characterized by the physiological sensation that stimulates us to search for food, whereas appetite is only the desire to consume some type of specific food regardless of the feeling of hunger.

Appetite can be influenced by hedonic factors, related to memories and learning, acting on the brain’s reward system and also by homeostatic factors.10

To MacLean, Blundell, Mennella and Batterham11 there are four biological factors that act on the regulation of appetite, they are: the long-term energy reserves in the adipose tissue, the detection and availability of nutrients by the intestine, the metabolic requirements of the functional mass and the establishment of tastes and food preferences.

Regarding the long-term energy reserves in adipose tissue, body levels of leptin, a biological marker that varies concomitantly with the energy reserve of fat cells, and insulin, which increases its availability as peripheral resistance levels are affected by adiposity of cells, are responsible for acting on the arcuate nucleus of the hypothalamus to signal a reduction in energy consumption and an increase in energy expenditure. However, in chronic states of excess availability (as in obesity), the sensitivity of the receptors is affected and the action of these hormones on appetite control is mitigated. In addition, leptin and insulin act on other brain regions, affecting the reward system and dietary decision-making.12

Regarding the detection and availability of nutrients in the intestine, the hormones released during food ingestion (PYY, CCK and GLP-1) are responsible for the signs of satiation and satiety. In addition to hormonal secretion, there is evidence in the literature that the interaction between intestinal hormones, bile and the microbiota of the gastrointestinal tract produce effects on obesity.13

Regarding the metabolic requirements of functional mass, there is a positive relationship between the amount of lean mass, the resting metabolic rate and the regulation of energy consumption by the body.14 Regarding fat mass, this would have an inhibitory effect on appetite. However, this effect would be moderated by the levels of insulin and leptin so that in cases of excess fat mass and excessive food consumption, the inhibitory effect would be reduced.15

Finally, when it comes to the establishment of tastes and food preference, in addition to the influence of the reward system, the hedonic value of the food and several other factors related to food decision-making, the taste and preference for food trigger biological responses that influence the energy consumption. In addition, there is evidence that appetite regulating hormones have receptors in the taste buds and olfactory neurons, so that the influence of the regulation of food choice by taste also has a biological character, with indications even that the amount of PYY in the saliva can influence energy consumption.16

Ghrelin, PYY, GLP-1 and PP

Ghrelin is a polypeptide hormone, it was discovered by Kojima, Hosoda and Date17,18, being classified as a polypeptide hormone18, with 28 amino acids, and that is produced in the stomach portion, with a smaller portion being synthesized in the intestines19 its acylation - that is, the reaction that results in the introduction of an acyl group in an organic compound - is necessary for the passage in the blood-brain barrier, in addition to being an essential factor for its GH-releasing action and other endocrine activities.17,18

Still, it has orexigenic activity coupled with the control of energy expenditure; control of acid secretion and gastric motility, influence on pancreatic endocrine function and glucose metabolism and cardiovascular actions.18

The peptide YY - PYY is a peptide hormone produced in the intestine, more precisely in the ileum and colon, being secreted by the endocrine L cells of the distal portion of the small intestine and large intestine, in the postprandial period, equivalent to the amount of calories ingested. PYY decreases intestinal motility and increases satiety, which causes a decrease in appetite and consequently food intake.20
The glucagon-like peptide\(^1\) (glucagon-like peptide 1 - GLP-1), is an incretin hormone produced by the L cells of the distal ileum and colon and is always released after eating food. GLP-1 acts on pancreatic islets both in beta cells (stimulating insulin synthesis) and in alpha cells (inhibiting glucagon secretion). The aforementioned action depends on blood glucose, making hypoglycemia impossible. However, it also inhibits gastric emptying through local and hypothalamic action, thus decreasing appetite. \(^{21}\)

The pancreatic polypeptide - PP is produced in the pancreas by the islets of Langerhans, acts by stimulating the feeling of satiety by inhibiting the production of ghrelin and gastric emptying. \(^{22}\)

**Physical exercise and appetite**

Much is said about the increase in appetite after physical exercise, however, there are numerous studies that indicate the opposite to this hypothesis widely supported by common sense.

Physical exercise can modulate appetite indirectly through lactate or directly through the release of anorectic hormones during physical exercise. The hypothesis that lactate can suppress appetite comes from the assumption that its production is also modulated during high-intensity physical exercise \(^{23, 24, 25}\) lactate can decrease the feeling of appetite by cutting ghrelin secretion. \(^{23, 25}\) Lactate molecules are able to bind stomach cells, specifically to receptors coupled to protein G. \(^{26}\) These cells are linked to ghrelin secretion and such a link can compromise appetite signaling (stimulation of hunger), due to the cut in ghrelin production. This interruption, however, is transient and only happens during exercise. \(^{24, 27}\)

Regarding the secretion of anorectic hormones during physical exercise and their role in suppressing appetite, there are more concrete data in the literature for this. Since the 1970s, studies have tried to identify the appetite suppression mechanisms triggered by physical exercise through anorectic hormones. Belbeck and Critz\(^{28}\) evidenced in a classic study on the theme the occurrence of appetite suppression after a strenuous exercise session. Blood samples were collected pre and post-exercise and identified a significant increase in anorexigenic hormones in the blood plasma. In addition, he found that the intensity of the exercise is directly linked to the increase of anorectic hormones in the plasma, which would result in a net caloric deficit and in the reduction of excess caloric intake.

Bringing this discussion to the present day, According to Hazell et al\(^6\) the physiological control of appetite regulation involves circulating hormones with orexigenic (appetite stimulants) and anorexigenic (appetite suppressant) properties that induce changes in energy intake via perceptions of hunger and satiety. As the effectiveness of exercise to induce weight loss is a controversial topic, there is considerable interest in the effect of exercise on appetite regulating hormones, such as ghrelin, the YY - PYY peptide, the glucagon-like peptide-1 - GLP-1 and pancreatic polypeptide - PP being orexigenic, anorectic, anorectic and anorectic, respectively. \(^{29}\)

In general, changes in hormones that regulate appetite after acute exercise seem to be intensity-dependent, with increased intensity leading to greater suppression of orexigenic signals and greater stimulation of anorectic signals. In short, short-term appetite regulation after a single exercise session is likely to be affected by decreases in ghrelin and increases in intestinal hormones PYY, GLP-1 and PP. High intensity exercise appears to have a more consistent effect on ghrelin compared to low to moderate intensity aerobic exercise. Both PYY and GLP-1 also appear to be influenced by exercise, although duration may be more important than intensity, suggesting that there may be a threshold necessary to induce increases in these hormones. A late increase in PP secretion is likely to occur after exercise, although high-intensity exercise may result in a faster and more prolonged release of PP. In general, changes in appetite-regulating hormones after acute exercise appear to be intensity-dependent, leading to greater suppression of appetite signals and greater stimulation of satiety signals with more intense exercise. \(^6\)

Although the effects of physical exercise in suppressing appetite are proven, it is still a matter of discussion whether this effect remains and varies according to the individual's characteristics and the intensity of the exercise. There are reports that this effect of suppressing appetite and reducing food intake is transient and is offset by an increase in caloric consumption 24 to 48 hours after exercise. \(^{28}\)

In young and healthy people with controlled weight, this effect of suppressing appetite and reducing food consumption does not trigger compensatory action after moderate aerobic exercise. \(^{30, 31, 32}\) verifying that the deficit in the energy balance induced by exercise was caused by a chronic reduction in the hormonal regulation of appetite \(^33\), however, there are controversies, as Douglas et al\(^{34}\) in its systematic review and meta-analysis, combining results from different studies, it initially presents reports that, in thin individuals, acute exercise transiently suppresses feelings of appetite. On the other hand, in obese or overweight people, moderate physical exercise also provides for suppression of appetite and reduction of food intake due to the decrease in circulating acylated ghrelin \(^{35, 36}\), but this effect is transient. \(^{35, 32}\)

Panissa and colaborators\(^{37}\) identified that both with continuous exercise of moderate intensity and with high-intensity interval exercises there was a decrease in the perception of hunger and also a decrease in energy consumption. Although in women the amount of PYYs was lower in relation to men, the relative energy ingested was lower in all types of exercise, and was not affected by intensity or sex, demonstrating that although changes in food intake have not been observed due to the exercise intensity, none of the exercises generated a compensatory increase in food intake. However, the hunger felt was lower in the higher intensity exercises in relation to the control situation and the amount of acylated ghrelin was lower in the high intensity exercise.

There is also a point in the literature...
where there are situations where there are no appetite suppression effects in the period after physical exercise. This happens when a population of individuals who are already conditioned to a high performance level is submitted to an aerobic exercise program. In the case of elite athletes, in this case, there is no change in hormonal secretion or bioavailability of anorectic signals, with ghrelin concentrations being maintained at pre-activity levels and, consequently, the same appetite pattern.\(^{38,39}\)

Another turning point in the theme is reported in a study in which a group of healthy girls underwent a running and swimming training session, in which case there was a significant increase in circulating ghrelin after training, concluding that swimming training and the racing caused an increase in ghrelin, but it did not affect the hunger rate.\(^{40}\)

**CONCLUSION**

The present study investigated the actions of physical exercise in modulating appetite and decreasing food intake through the identification of appetite suppression mechanisms and anorexigenic actions. Through the literature review, it appears that there are still many inflection points on the subject.

It is known that physical exercise promotes and stimulates the secretion of anorectic hormones in the bloodstream, but its real effectiveness in suppressing appetite and reducing food intake after exercise, in the medium and long term, in overweight and overweight people is still uncertain.

In most of the studies consulted, the training protocol was based on exercises, for eutrophic people, thus presenting non-compensation results for these individuals, but for obese and overweight individuals there is still uncertainty regarding these effects of appetite suppression in a chronic way after exercise.

Seeing that the production of lactate molecules interferes with the production of ghrelin and is directly linked to the intensity of exercise, future studies should explore more high-intensity interval exercises for obese people, considering that the production of anorectic hormones such as GLP-1 PYY and PP increase proportionally with the intensity of the exercise; it would be ideal even if future studies would compare the possible relationships between lactate expression, suppression in the production of ghrelin and the possible application of this study in a group of obese individuals.

However, the present study reinforces the premise supported by the findings brought up in this review that the appetite suppression effect generated by the reduction of ghrelin levels is not a rule and depends on certain variables. The profile of the population studied, the level of fitness of that population and the intensity of physical activity are totally determinant in obtaining (or not) the effects of appetite suppression.

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