14 - LEPTIN BEHAVIOR IN DIFFERENT METHODS OF EXERCISES

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INTRODUCTION

Leptin was discovered by Zhang et al. (1994), and has been studied because it is related to obesity and other risk factors for growth and human development. It is a hormone that acts directly on the central nervous system, providing satiety after eating. It is completely influenced by the level of other hormones such as ghrelin and insulin, thereby controlling appetite and fat accumulation.So, diet and exercise are factors limiting for regulation by interfering directly in the energy balance, which in turn tells the hypothalamus to the condition of adipose tissue, deciding its production (CIOLAC et al., 2003).

Leptin has a unique behavior in females and can cause symptoms such as delayed menarche and even amenorrhea disease according to the studies of Yu et al. (1997) because it will influence the production of ovarian hormones.

It is a hormone coming from the Ob gene, consisting of 167 peptides. The relationship of leptin with adipose tissue leads to understand that it interferes too much in body fat factor according to the study of Zhang et al. (1994), for developing resistance to this hormone, and be influenced by constant adrenal steroid hormones. Ciolac et al. (2003) found that leptin values changed due to the level of insulin signaling to the central nervous system the level of fat in it and an appropriate response to the ingestion of the individual, controlling appetite (MATTHWES and WALLACE, 2002).

Who (1985) and Pardini et al. (1998) pointed that contained in plasma leptin binds to receptors in the choroid plexus which sends to the brain and binds to hypothalamic receptors, forming an axis leptin - sympathetic nervous system (SNS) /gonadotropin - leptin /leptin -mediators of appetite /leptin - thermogenesis. Hall (2001) complete, the leptin - SNS may cause changes in renal excretion of sodium, interfering with the cardiovascular system, because this maneuver increases blood pressure.

Ciolac (2003) added more information about leptin beyond its relationship with obesity and appetite, showing their interaction with fertility and growth. Another function was presented by Garg (2004): it is believed that leptin is a modulator of insulin sensitivity and glucose distribution. From there, using animal models, Berg (2000) affirm that resistance to this hormone was linked to hipoleptinemia, proven by experience with the introduction of subcutaneous leptin resulting in there stabilization of insulin sensitivity. For this reason, the person takes longer to feel its effect, still eating too much, not getting information to the hypothalamus that nutritional needs were met, and may also be related to the activation of ghrelin receptors that activate hunger.

Thinking about the amount of leptin, how much higher the level, higher will be the fat layer, lower will be the insulin and lower will be the appetite. However, the study of Pelleymonter et al. (1995) showed there was a "state of resistance" found, for example, in obesity, breaking the simplicity of the above process.

The discussion of the hormone leptin and exercise gives the caloric expenditure as the determining factor, being able to change the constitution of the individual through the mobilization of fatty acids during and after exercise. For weight loss to occur, the American Heart Association (2001) considers necessary a caloric expenditure between 1000 and 2000 kcal/week from exercise may be aerobic or resisted.

The aerobic exercises can be cyclic and acyclic, moving large muscle groups, requiring the organism as a whole in a complex and fast to achieve the goal of weight loss, and an alleged lipolytic area for more fat loss in that period (FIELDS, 200). The resistance exercise, the action involves voluntary skeletal muscle against some resistance, composed of concentric and eccentric moments (Fleck and Kraemer, 2006) as well as aerobic exercise but with different physiological requirements. Therefore the higher energy produced by resistance exercise is the eccentric phase and after the implementation of training, verified for the post-exercise oxygen consumption (EPOC) increased for longer than aerobic exercise (BELINSKY, 1985).

As noted in the studies leading up to the moment, leptin as a hormone that directly interferes in the affairs of human development and body composition. It is necessary to understand how their intervention occurs in physical exercises.

Given this initial context, this article aims to discuss the behavior of leptin in addressing different types of training methods in the form of a literature review supported by the search of articles with keywords such as leptin, exercise and hormones.

EXERCISE AND LEPTIN

Miller's research and Sobral Filho (2004), suggests that physical exercise causes various physiological effects that can be immediately classified as immediate acute (after exercise), late acute (between 24 hours and 72 hours after exercise) and chronic (after several sessions of exercise), which lead the body to training adaptations and secretion of hormones such as GH (growth hormone), catecholamines, glucagon, insulin, endorphin, cortisol, among others. The binding of leptin with other hormones could contribute to the understanding of their behavior during exercise. Fisher et al. (2001) found results in circulating concentration of glucose and insulin, 86% on the variation in the circulating concentration of leptin, ending dependence on glucose homeostasis and insulin.

Analysing Hulver e Houward (2003) is not evident if only the exercise without change in the situation of body composition, may alter the concentration of leptin and in need of betters cientific data. The intensity of exercise triggers a specific response to the concentration of plasma leptin in the body though. studies still are not sure on getting an answer (SABIA et al., 2004; HOUWARD e HULVER, 2003).

However, we can make a bridge with hungry after strenuous exercise which in turn requires an energy expenditure too, cause it really hungry after his execution. Thus, after exercise, blood glucose level is lower, which may lead to an increase in circulating leptin, or action of their receptors with no change in body composition by acute exercise.

Sabia et al. (2004) reflect on it and compare the effect of continuous aerobic exercise and intermittent associated with a diet in obese subjects trained three times a week for three months, say both trainings have been successful in changing body

composition, decreasing 1, 5% of total body mass, BMI and 4.6% in the continuous aerobic exercise, and intermittently there was a reduction of 3.7% in total body mass and 7.6%in BMI. Considering how the modification of body composition by exercise has a direct action at the level of leptin.

The concentrations of body fat, the decrease in adipose tissue by exercise alters the long leptin receptor, Ob-Rb. With this idea, Kimura et al. (2004) studied mice that had spontaneous exercises for 12 weeks. The response was the reduction of body fat, decrease in concentration of leptin, as well as the concentration of receptor down regulation.

Otherwise, researchers such as Dubuc et al. (1998) found that the loss of 3 kg of body fat through physical training leads to a decrease in the level of leptin in women approximately 7ng/ml. Okazaki et al. (1999) presented a study of BMI and leptin concentration, which has decreased the percentage of fat and leptin after an aerobic training for 50% of VO2max (30 minute stationary bike or walking, plus 30 minutes of aerobic exercise with low impact) built into the diet for 12 weeks. In contrast, Torjman et al. (1999) and Dusrtine et al. (2001) did not achieve the same reduction in the concentration of leptin with exercise in a sample of individuals who exercised for 60minutes on the treadmill at 50% VO2max. It was shown no difference during 4 hours after exercise. Thus, affirming that the low-intensity acute exercise has no impact on the concentration of leptin.

Kraemer et al. (1999) also found no changes in leptin concentration after delivering a 30 minutes treadmill exercise at 80% VO2max in sedentary post menopausal women. These same authors in 2002 found that exercising for 60 minutes or less, does not alter the concentration of leptin in healthy men and women, but when exceeded one hour of activity, leptin values were normalized, suggesting that it is not changeable by the action of short duration of exercise.

In training books as Fleck and Kraemer (1999) has the required time to 12 weeks to start having results for the reduction of adipose tissue. Maybe this time for waiting for the modification in leptin levels before the exercise is due to changes in body composition.

During the exercise perceive besides leptin, ghrelin action which in turn acts in the formation of GH (growth hormone), which is of paramount importance for the lipolytic effect of the body according to Lange (2004). Then, during exercise, ghrelin increases, decreases GH and leptin (since it is inversely proportional to ghrelin). Nevertheless, GH is an activator of lipolysis, decreasing the level of body fat, necessarily, less leptin. However, it is known that ghrelin acts in reverse form of leptin, it interferes with food intake, increasing it. So the complex reflection during exercise, the question arises: with ghrelin increases GH, leptin decreases, insulin decreases, increases glucose supply, lipolysis increases?

Essig et al. (2000) found a 30% reduction in leptin 48 hours after the exercise session.Tuominen et al. (1997) found 34% decrease in serum leptin measured 44 hours after 2 hours of exercise at 75% VO2 max, revealing evidence that training decreases the concentration of leptin independent of changes in fat mass.

Articles also address the level of leptin involving diet and exercise together. Sandoval et al. (2003) studied situations of stress to the body caused by prolonged fasting and strenuous exercise, and found the decrease of leptin, thus confirming the role of the CNS in controlling the amount of leptin produced by adipocytes. In athletes, hipoleptinemia and hypoinsulinemia are used as markers of chronic stress to the intense training (MANTZOROS, 1999; KRAEMER et al., 2002).

The high-fat diet failed to attenuate central visceral obesity in individuals who participated in physical training, but the exercise was notable to prevent the development of hyperleptinemia (RACETTE et al., 1997). In this regard, Kraemer et al. (2002) assume that the exercise has no power to modify the hyperleptinemia, but add that the exercise leads to decreased amounts of tissue.

Koutsari et al. (2003) submitted to exercise and diet with a high percentage of carbohydrates (macronutrients in the amount of 70% of the total energy intake) and a control group who consumed low carbohydrate intake, and obtained as a result of a higher concentration of leptin in fasting and post prandial groups with high consumption of carbohydrates.

By contrast, Hickey et al. (1997) reported that leptin might reduce their numbers without changing adipose tissue, a study in sedentary women during 12 weeks of aerobic training four days a week and 30 to 40 minute session. Pasman et al. (1998) follows the line of previous author, the study confirms that in obese women, practitioners of aerobic exercise training for 8 and 16 weeks and low-calorie diet, as result, the long-term training decreases plasma leptin concentrations independent of change in insulin levels and body fat percentage.

With inverse data, Pasman et al. (1998), with the sample of obese men during 16 months of aerobic training and dietary intervention, resulted in a decrease in plasma levels of leptin for the active group. In a similar study done by Reselenad et al. (2001), men with metabolic syndrome who underwent aerobic exercise intervention and control food for a year experienced a decrease in leptin and weight reduction.

Most articles refer to this hormone aerobic exercise. Few studies were found with respect to the resisted work on leptin levels. Gippini et al. (1999) addressed this issue and selected young bodybuilders and sedentary overweight or normal weight. Their study showed that leptin levels were similar, proving that training for muscle hypertrophy does not affect this hormone, independent of body composition sampled. Afterwards, Kanaley et al. (2001),with a sample of diabetics noted the reduced level of leptin in the acute phase of the program of weight training, but over the long term (six weeks) there was no change. Ryan et al. (2000) to establish the protocol resisted analyzed obese postmenopausal women with and without weight loss after 16 sessions, rolling resistance exercises that led to an increase in lean body mass and resting metabolic rate in both groups, resulting in a drop of 36 % of leptin.

Zafeiridis et al. (2003) created different protocols, using three distinct maximum force (4 sets, 5 reps, 88% of 1RM - repetition maximum), muscle hypertrophy (4 sets, 10 reps, 75% of 1RM), endurance (4 sets; 15 repetitions, 60% of 1RM). As Noland et al. (2000), who auditioned in swimmers at different intensities in the two studies there was no difference in leptin values immediately after exercise and there was no difference after 30 minutes of completion.

CONCLUSION

Analyzing some studies presented above, each with its own special on individuals in different age groups and physical characteristics, duration and intensity of exercise, there are many differences regarding the level of leptin values found in every situation of effort.

The literature shows significant changes in body composition after 3 months of training, then that period should be assumed as a result of chronic exercise. The work must be done in the form of training for weight loss, as used by obese or over weight, targeting the aerobic interval (ALMEIDA e PIRES, 2008) and resisted circuited (SON et al., 2008). Thus, the percentage of fat will necessarily diminish and the body will adapt to the new structure of adipose tissue to release their hormones dependent.

Despite this review conducted in this study, we can not achieve to a standardization of results, which can be justified due to the biological individuality.

We leave in alert a subject that must bring the research methodologies to real people who necessarily need these results. With own protocols for these variables we can find a real answer and take a firm position with the data that only research

can provide.

Thus, we can consider, in such analysis that prolonged training have a better response to leptin levels. Being totally suitable for people with hormonal disorders, obesity, hypertension, metabolic syndrome and diabetes, among others, in relation to the control of leptin.

From this, we can raise a question, some authors have identified a decrease in leptin after intense exercise, others have found leptin decline after a few hours of effort to a lesser degree. Those values of decreased leptin are related to the intake post exercise oxygen (EPOC)? Leaving a question about when the EPOC starts its activities in the body and its duration at different levels of intensity and volume of exercise, and finally correlate it with plasma leptin.

Assume a position on the change in percentage of fat that will influence the level of leptin is still impossible due to many differences in the studies presented in this review, requiring more studies with better methods.

REFERENCIAS

ALMEIDA PA, PIRES CMR. A importância do treinamento intervalado em programas de redução de peso e melhoria da composição corporal. **Dig Efdeportes**, ano 13, n. 19, 2008. Disponível em: <http://www.efdeportes.com/efd119/treinamento-intervalado-em-programas-de-reducao-de-peso.htm>. Acesso: 13 out 2010.

AMERICAN HEART ASSOCIATION. Exercise standards for healthcare professionals from the American Heart Association. **Circulation**, 104(14):1694-740, 2001.

BERG, JP.Leptin is a potent anti-diabetic in mice with lipodystrophy and insulin resistance**. Europ J Endocrinol**, 2000; 142:114-6.

CAMPOS, M.A. **Musculação: diabéticos, osteoporóticos, idosos, crianças, obesos.** Rio de Janeiro: Sprint; 2001.

CIOLAC, EG; et al. Exercício intervalado é melhor que exercício contínuo para diminuir a pressão arterial 24 horas pós-exercício em hipertensos. **Rev Soc Cardiol** Est São Paulo, 2003; 13 (2 Supl):48.

DUBUC, GR; et al. Changes of serum leptin and endocrine and metabolic parameters after 7 days of energy restriction in men and homen. **Metabolism**. v.47, p.429-434, 1998.

DURSTINE J.L.; et al. Leptina y Ejercício: Nuevas Direcciones. **Departament of Excercise Sciense University of South Carolina Columbia.** 2002.

ESSIG, DA; et al. Delayed effects of exercise on the plasma leptin concentration**. Metabolism 2000**;49(3):395-9.

FILHO ADR, et al. Efeitos do treinamento em circuito ou caminhada após oito semanas de intervenção na composição corporal e aptidão física de mulheres obesas sedentárias. **Rev Bras de Ob, Nut e Em,** v.2, n.11, p. 498-507, 2008.

FISHER, JS; et al. Acute exercise effect on postabsorptive serum leptin. **J. Appl Physiol**. v. 91, p.680-686, 2001.

FLECK, S. J. E KRAEMER, W. J. Fundamentos do treinamento de força muscular. 2. ed. **Porto Alegre: Editora Artmed,** 1999.

GIPPINI, A; et al. Effect of resistance exercise (body building) training on serum leptin levels in young men. Implications for relationship between body mass index and serum leptin. **J Endocrinol Invest**, 1999;22(11):824-8.

HALL, JE; HILDEBRANDT, DA; KUO, J. Obesity hypertension: role of leptin and sympathetic nervous system. A**m J Hypertens,** 14: 103S-115S; 2001.

HICKEY, MS; et al. Gender-dependent effects of exercise training on serum leptin levels in humans. **Am. J. Physiol**. v .272, p.562-566, 1997.

HULVER, M.W; HOUWARD, J.A. Plasma leptin and exercise. **Sports Medicine,** v. 33, p. 473-482, 2003.

KANALEY, JA; et al. Resting leptin responses to acute and chronic resistance training in type 2 diabetic men and women. **Int J Obes Relat Metab Disor**d, 2001;25(10):1474-80.

KIMURA, M; et al. Long term exercise down regulates leptin receptor mRNA in the arcuate nucleus. **Neuroreport.** v. 15, p. 713-716, 2004.

KOUTSARI, C; et al. Plasma leptin is influenced by diet composition and exercise. **Int J Obes Relat Metab Disord** 2003;27(8):901-6.

KRAEMER, RR; et al. Serum leptin concentration in response to acute exercise in posmenopausal womem with na without hormone replacement therapy. **Proc. Soc. Exp. Biol. Med.** v .221, n.3, p.171-177, 1999.

KRAEMER, RR; CHU, H; CASTRACANE, VD. Leptin in exercise. Exp Biol Med., 2002; 227: 701-8.

LANGE, KH. **Fat metabolism in exercise – with special reference to training and growth hormone administration. Scand J Med Sci Sport**s. 2004;14(2):74-99.

MANTZOROS, C.S. The role of leptin in human obesity and disease: a review of current evidence. **Annals of Internal Medicine**.v.130, p.671-680, 1999.

MONTEIRO, MF; SOBRAL FILHO, DC. Exercício físico e o controle da pressão arterial. **Rev Bras Med Esporte**, 2004; 10(6):513-6.

NOLAND, RC; et al. Effect of intense training on plasma leptin in male and female swimmers. **Medicine & Science in sport & exercise. p. 227-231, 2000.**

OKAZAKI, T; et al. Effects of mild aerobic exercise and mild hypocaloric diet on plasma leptin in sedentary females. Clin Exp. **Pharmacol Physiol**. v .26, p.415-420, 1999.

PASMAN, WJ; WESTERTERP-PLANTENGA, MSW; SARIS, H; M. The effect of exercise training on leptin levels in obese males. **The American Physiological Society.** p.280-286,1998.

PELLEYMONTER, MA; et al. Effects of the obese gene product on body weight regulation in ob/ob mice. Science. 1995;269(5223):540-3.

RACETTE, SB; COPPACK, SW; LANDT, M; KLEIN, S. Leptin production during moderate intensity aerobic exercise. **J Clin Endocrinol Metab.,** 1997; 82: 2275-7.

RYAN, AS; PRATLEY, RE; GOLDBERG, AP. Changes in plasma leptin and insulin action with resistive training in posmenopausal females. **Int. J. Obes. Relat. Metab. Disord.** v. 24, p. 27-32, 2000.

SANDOVAL, DA; DAVIS, SN. Leptin: metabolic control and regulation. **J Diab Compl.** 2003; 17(2): 108-13.

TORJMAN, MC; et al. Serum leptin during recovery following maximal incremental and prolonged exercise. **J. Sports Med.** v. 20, p.444-450, 1999.

TUOMINEM, JA; et al. Serum leptin concentration and fuel homeostasis in healthy man. **Eur J Clin Invest. 1997**;27(3):206-11.

ZAFEIRIDIS, A; SMILIOS, IR; CONSIDINE, V; TOKMAKIDIS, SP. Serum leptin responses after acute resistence exercise protocols. **J. Appl Physio**l. v.94, p.591-597, 2003.

ZHANG, Y; et al. **Positional cloning of the mouse obese gene and its human homologue. Nature**, v. 372, p. 425-

432, 1994.

WALLACE, TM; MATTHEWS, DR. The assessment of insulin resistance in man. **Diabetic Medicine 2002**; 19:527-534.

WHO STUDY GROUP ON DIABETES MELLITUS. Report .Tech Rep Ser WHO No. 727. Geneva: **World Helath Organization,** 1985.

YU, WH; et al. Role of leptin in hipothalamix-pituitary function**. Proc Natl Acad Sci.** v.94n.102, 1997.

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LEPTIN BEHAVIOR IN DIFFERENT METHODS OF EXERCISE

ABSTRACT

Advances in endocrinologic studies point to the adipocyte as more than a tissue, he comes to synthesize substances such as tumor necrosis factor alpha, interleukin-6 and leptin. In this case, here the leptin deserves the attention for being allocated to food intake, thus characterizing the satiety hormone food. However, this literature review will look at the exercise as a way of modulating production of leptin in the body, discussing different methodologies of exercise tested by researchers in different populations.

KEYWORDS: leptin, satiety, exercise.

COMPORTEMENT DE L´LEPTINE DANS DIFFERENTES METHODES DE EXERCICE

RÉSUMÉ

Les progrès dans les études endocriniennes point à l'adipocyte que plus d'un tissu, ils'agit de synthétiser des substances telles que l'alpha du facteur de nécrosetumorale, l'interleukine-6 et la leptine. Dans ce cas, la leptine mérite ici d'être le point culminant attribuée à l'apport alimentaire, caractérisant ainsi la nourriture hormone de satiété. Néanmoins, cette revue de la littérature abordera l'exercice comme un modulateur de la production de leptine par le corps, de discuter des approches différentes pour exercer testé par les chercheurs dans différentes populations.

MOTS-CLÉS: leptine, la satiété, de l'exercice.

COMPORTAMIENTO DEL LA LEPTINA EN DIFERENTES MÉTODOS DE EJERCICIOS

RESUMEN

Los avances en la endocrina estudios señalan que el adipocito como algo más queuna tela, se trata de sintetizar sustancias como el factor de necrosis tumoral,interleukina-6 y leptina. En este caso, la leptina se merece aquí para ser el punto culminante atribuye a la ingesta de alimentos, lo que caracteriza la comida hormona de la saciedad. Sin embargo, esta revisión de la literatura se dirigirá el ejerciciocomo un modulador de la producción de leptina en el cuerpo, discutir diferentes enfoques para el ejercicio probado por los investigadores en las diferentes poblaciones.

PALABRAS-CLAVE: la leptina, la saciedad, el ejercicio.

COMPORTAMENTO DA LEPTINA EM DIFERENTES METODOLOGIAS DE EXERCÍCIOS

RESUMO

Avanços em estudos endocrinológicos apontam para o adipocito como mais que um tecido, ele vem a sintetizar substâncias como o fator de necrose tumoral alfa, interleucina-6 e a leptina. Nesse caso, aqui a leptina merece o destaque por ser atribuída à ingestão de alimentos, assim caracterizando o hormônio da saciedade alimentar. Não obstante, essa revisão bibliográfica abordará o exercício como forma moduladora da produção da leptina pelo organismo, discutindo diferentes metodologias de exercício testadas por pesquisadores em diferentes populações.

PALAVRAS-CHAVE: leptina, saciedade, exercício.