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Application of Leptin in Enhancing Immunity and Maintaining Obesity

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ABSTRACT

Leptin is predominantly generated in adipose tissue and circulates in serum both as a free and as a protein-bound entity. Leptin, a 16 kDa non-glycosylated polypeptide product of the obese (ob) gene, is an adipocyte-derived hormone which has long been recognized as a key factor in regulating a wide range of biological responses involving energy homeostasis, neuroendocrine function, angiogenesis, bone formation and reproduction. Leptin is a key afferent signal linking adiposity level and nutritional status to neuroendocrine regulation of energy homeostasis chiefly through decrement in caloric uptake and enhancement in energy expenditure. Serum levels of leptin reflect the amount of energy stored in adipose tissue. Short-term energy imbalance as well as serum levels of several cytokines and hormones influence circulating leptin levels. Leptin acts by binding to specific receptors in the hypothalamus to change the expression of several neuropeptides that regulate neuroendocrine role and energy uptake and expenditure. Leptin plays a significant function in the pathogenesis of obesity and eating disorders and is thought to mediate the neuroendocrine response to food deprivation.

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Introduction

Leptin is a hormone that comes from Greek word meaning "thin" it is also known as "Ob gene" that is located on chromosome number 7 [1]. Leptin, a 167-amino acid protein synthesized by adipocytes, circulates at a concentration proportional to the adipose tissue mass and relays a satiety signal to the hypothalamus [2]. Leptin is transported to the central nervous system from plasma by a saturable, unidirectional system, including binding of leptin to the short form of the leptin receptor located at the endothelium of the vasculature and the epithelium of choroid plexus. Leptin acts in the hypothalamus to regulate appetite, energy expenditure and sympathetic nervous system outflow [3]. Leptin is predominantly generated in adipose tissue and circulates in serum both as a free and as a protein-bound entity. The leptin receptor is described in several hypothalamic nuclei involving the arcuate nucleus, ventromedial hypothalamus, and paraventricular nucleus and dorsomedial hypothalamus [4]. Leptin acts in the hypothalamus to regulate appetite, energy expenditure and sympathetic nervous system outflow. The arcuate nucleus is can be considered as the major site of transduction of the signal from circulating leptin into a neuronal response. Central neural administration of leptin does not affect food uptake or sympathetic nerve activity after destruction of the arcuate nucleus. Main function of leptin is to achieve an energy balance in the body [5].

Leptin hormone is produced by the adipose tissue, chiefly by the white adipose tissue of the human body; it is comprised of 167 amino acids. Leptin, a 16 kDa non-glycosylated polypeptide product of the obese (ob) gene, is an adipocyte-derived hormone which has long been recognized as a key factor in regulating a wide range of biological responses involving energy homeostasis, neuroendocrine role, angiogenesis, bone formation and reproduction [6]. Leptin has been increasingly recognized as a cytokine-like hormone with pleiotropic actions in modulating immune responses and revealed to provide a proliferative signal in hematopoiesis and lymphopoiesis [7].

Leptin can activate monocytes, dendritic cells and macrophages and stimulate them to generate T helper-1 type cytokines and also exerts activating effects on neutrophils and natural killer cells and stimulate their gene expressions. Significantly, leptin has been revealed to modulate the adaptive immunity via elevating T cell survival and stimulating their generation of pro-inflammatory cytokines such as interferon-gamma and interlukin-2 [8]. Immune mechanisms play a pivotal function in atherogenesis. Atherosclerotic lesions are filled with immune cells that can coordinate and effect inflammatory responses. The common components of atherosclerotic lesions are macrophages and T lymphocytes, and perhaps also contain mast cells and dendritic cells [7]. Macrophages are seen in all phases of atherosclerosis, whereas both types of T helper cells in both cluster differentiation-4+ and cluster differentiation-8+ have been detected in human atheromas and have been revealed to be immunologically activated [9]. The initial amino acid sequence of leptin indicated that it could belong to the long-chain helical cytokine family, such as interleukin-2, interleukin-12, and growth hormone. Leptin receptor (Ob-R) reveals sequence homology to members of class I cytokine receptor (gp130) superfamily that involves the receptor for interleukin-6, leucocyte inhibitory factor, and granulocyte colony-stimulating factor [10].

Leptin is a key afferent signal linking adiposity level and nutritional status to neuroendocrine regulation of energy homeostasis chiefly through decrement in caloric uptake and enhancement in energy expenditure. Serum levels of leptin reflect the amount of energy stored in adipose tissue. Short-term energy imbalance as well as serum levels of several cytokines and hormones influence circulating leptin levels [11]. Leptin acts by binding to specific receptors in the hypothalamus to alter the expression of several neuropeptides that regulate neuroendocrine function and energy intake and expenditure. Leptin plays a significant function in the pathogenesis of obesity and eating disorders and is thought to mediate the neuroendocrine response to food deprivation [12].

Conclusion

Leptin is a hormone that comes from Greek word meaning "thin" it is also known as "Ob gene" that is located on chromosome number 7. More hormones present in the body that upregulate or downregulate the level of leptin. Leptin is upregulated by insulin and cortisol and downregulated by catecholamines. The initial amino acid sequence of leptin indicated that it could belong to the long-chain helical cytokine family, such as interleukin-2, interleukin-12, and growth hormone. Leptin also autoregulates its own expression by glucose and fatty acids also influence leptin expression. Leptin's effects on body weight are mediated through effects on hypothalamic centers that control feeding behavior and hunger, body temperature and energy expenditure.

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