EDITORIAL ARTICLE

THE ROLE OF LEPTIN ON WEIGHT MANAGEMENT

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Obesity represents one of the most serious global health issues and according to the World Health Organization there are an estimated 1 billion adults who are overweight (body mass index>25 kg/m^2) and 300 million of these are considered clinically obese (body mass index>30 kg/m^2).[1] Taken for granted that this great threat imposes deleterious repercussions on the individuals' outcome and affects negatively their quality of life, the underlying etiology of obesity has received a great deal of attention by the health science, over the last decades.[2]

Although dietary choices and physical exercise may control obesity, a strong body of evidence demonstrates that body-weight is, up to some extend, genetically pre-determined. In regard to general population, it is estimated that genetic factors are responsile for the development of obesity up to 25-40%. Other parameters such as age, gender, ethnicity, socio-economic status, e.t.c., act as additive factors to the development of obesity.[3,4]

During the last decade, regarding the heredity aspect of obesity, there has been a growing recognition of the significant role of leptin as well as the leptin receptor to the balance of body weight. This protein was named leptin from the Greek, "leptos" which means thin, because it is proved to be a key-factor in the control of energy expenditure, food intake and body weight.[5-8]

Leptin, is predominantly although not exclusively, secreted by adipocytes. It monitors the size of the adipose tissue mass, circulates in the plama and generates signals to the brain about the body energy stores. More in detail, leptin provides a system signaling the amount of adipose tissue to the hypothalamus of brain where several orexigenic or anorexigenic neuropeptides are stimulated in order to manage body weight.[5-8]

When energy intake and energy expenditure are equal, leptin levels reflect total bodyfat mass. Conversely, when the weight is not maintained (negative or positive balance) and accumulation of fat exceeds the genetically pre-determined point, the levels of leptin change (leptin secretion or plasma concentration) in order to function as a sensor of this energy imbalance to the brain. Under normal conditions, the hypothalamic centers after having received and recognized these signals, regulate energy balance by reducing appetite when fat accumulation is exceeding, or growing appetite when fat accumulation is beneath normal. For these reasons, leptin is frequently cited by the literature as a "lipostatic" regulation system, depending on the size and the number of adipocytes in human body.[9-12]

This process was discovered after it had been well documented in mice which are carrying the homozygous obese mutation and develop a syndrome that resembles human morbid obesity. Although, this feedback regulatory loop is well established in rodents, the nature of this multidimensional and complex relation still remains partially unclear in human.[13]

Given the fact that leptin plays significant role on weight management, it is not understandable why obese individuals, who

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ISSN:1108-7366 www.hsj.gr

have high body weight and consequently high leptin secretion and plasma concentration, cannot manage to reduce food intake and loose weight. Many researchers have commented on this negative association and emphasised the importance of resistance to leptin. According to the literature, the alternative suggestion is that a small minority of obese individuals may have broken lipostats and thus maintain high body weight. [2,3]

A commonly held view is, that the science of genetics in conjunction with social and educational policies can provide the required basis to promote positive health and well-being throughout human life and significantly minimize the increased frequency and prevalence of obesity as well as its serious consequences on health.

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