Chapter

Introductory Chapter: Growth Hormone – Obesity and Physical Exercise

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1. Introduction

Obesity is one of the most prevalent metabolic diseases in the recent decades leading to negative effects on health. Obesity has shown a significant global increase over the past 50 years and this fact would be not desired. The World Health Organization (WHO) defines obesity as when an individual has a body mass index (BMI) greater than 30 kg/m^2. The epidemic growth of obesity represents a very serious public health problem, mainly due to its important association with the development of serious diseases that can lead to death, such as type 2 diabetes, cardiovascular diseases, arterial hypertension, metabolic syndrome, chronic kidney disease, hyperlipidemia, some types of cancer, asthma, sleep disorders, liver dysfunction, kidney disease, joint disease, depression, and infertility [1, 2].

Obesity is a universal phenomenon that occurs globally, except for parts of Asia and sub-Saharan Africa, as well as a few other countries, such as, Sri Lanka, Indonesia, Sudan, Singapore, and Djibouti [1].

It is known that several complex mechanisms can lead to obesity. The main cause would be the occurrence of an excess of energy stock when compared with the energy needed by the body. Excess energy, stored in fat cells, which enlarge, alters metabolic and hormonal factors responsible for obesity. Studies reveal that the food source and quality in the diet seem to influence the body mass control more than the amount ingested. Moreover, growing evidence suggests that obesity is a disorder of the energy homeostasis system. Furthermore, the lack of balance between food intake and physical activity, between genetic and epigenetic, environmental, and microenvironmental factors, the availability of health care services, and socioeconomic status can lead to obesity [3–5].

It is already well established that the body location of adipose tissue, mainly in the abdominal region, the so-called central obesity (CO), represents an important risk factor for metabolic dysfunction. It is significantly correlated with cardiovascular disease. CO can be classified into subcutaneous abdominal adipose tissue (SAT) and visceral adipose tissue (VAT). It is suggested that VAT is a metabolic organ that regulates fat mass, blood glucose, and nutrient homeostasis. Based on this knowledge,

it would be really important to identify clinical means capable of improving the therapeutic approach to obesity [1, 5–8].

Diet modifications and physical exercise are always included as a priority in treatments for obesity. Physical exercise would promote changes in lipolytic hormones, such as growth hormone (GH) and significant reductions in VAT. VAT, regardless of total BMI, is strongly related to cardiovascular risk [9–12].

The relevance of hormones in controlling body metabolism has been known for a long time. Among the most kinetically studied hormones is GH. Some aspects of GH physiology and its relationship with exercise, however, still remain controversial and unknown, including the knowledge of elements that regulate its synthesis, mechanisms of action and effects on protein and lipid metabolism [13, 14].

GH is secreted by the anterior pituitary in a pustile form, primarily regulated by the hypothalamic GH-releasing hormone (GHRH) and by somatostatin, neuropeptides that respectively stimulate and inhibit the secretion of this hormone. It is known that GH affects several body systems, regulating the secretion of insulin-like growth factor-1 (IGF-1), which in turn regulates its secretion. GH is a potent anabolic hormone with an important role in lipid metabolism in various body sites such as the liver, muscle skeletal muscle, and adipose tissue [15–17].

In times of fasting or stress, GH promotes the use of lipids as the first source of energy in order to preserve carbohydrate and protein stores. Several other physiological and pathological conditions interfere with GH secretion, age, gender, pubertal stage, sleep status, nutritional status, body composition and temperature, fitness, gonadal steroids, insulin, and IGF-1. In the physiology of GH and its relationship with the aforementioned factors, much remains to be clarified, and this will be highly relevant. It is known that abdominal visceral fat increases in obese individuals, which is why there is a drastic reduction in the GH response in obese individuals, both in stimulated secretion and in normal release [6, 14, 18, 19].

Since physical exercise is considered a potent stimulator of GH synthesis [20], factors such as exercise intensity, volume, and frequency may influence in GH concentration. However, studies have shown that in obese individuals, the GH elevation in response to physical exercise is reduced and that with body mass loss the response returns to that found in individuals with normal BMI [21, 22].

Regular exercise is desirable for health and studies have shown that different kinds of exercise can aid in the reduction of the body fat as well as protect the individuals against cardiovascular diseases associated with obesity [23–25].

2. Conclusion

It is known that GH acts directly through its receptor and indirectly stimulates the production of IGF-1. However, the mechanism through which physical exercise can be used to increase GH secretion is not completely understood. In this sense, this book aims to integrate scientific information about the relevance of exercises and/or GH to the management of individuals with obesity. Moreover, it is expected that this book might bring new insights into the role of growth hormone in the obesity.

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