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Mini Review

Approaching Obesity and Its Metabolic Changes

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Abstract

For many years, adipose tissue was only considered an energy storage organ, however, it is now known that it plays a key role in the integration of systemic metabolism, and this metabolic function is mediated, in part, by its ability to secrete several proteins, which are called adipokines. Although adipokines are didactically grouped into distinct categories, their actions occur in an integrated manner, causing the participation of TAB in energy homeostasis, immunity, inflammatory response, insulin sensitivity, angiogenesis and blood pressure. When adipose tissue accumulates, the expression of pro-inflammatory cytokines increases, thus causing inflammation and the development of insulin resistance, which are triggering factors for chronic non-communicable diseases.

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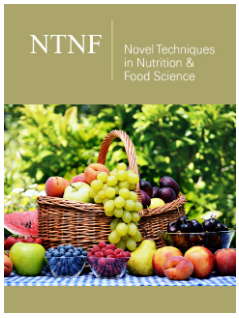

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Adipose cells originate in the embryonic stage from the differentiation of mesenchymal cells, forming adipoblasts and myoblasts that will give rise to pre-adipocytes, then forming adipose tissue, whose main primary functions are the isolation and protection of the organism, storage of Free Fatty Acids (FFA) after food intake and release during the fasting period or high energy requirements to ensure energy supply [1,2]; (Figure 1). Adipose tissue formed from the differentiation of myoblasts, known as brown adipose tissue, is present in large quantities in fetuses and newborns, and in more delimited regions in adults (neck region and around the kidneys). The brown color is due to the large number of mitochondria in this tissue, and favors greater cellular activity regulated by the hormone norepinephrine, being responsible for the regulation of body temperature [3]. White adipose tissue is the best known in the literature, as its main function is to store triglycerides (85-90%), as well as it releases them when in times of high energy demand or prolonged fasting in the form of long-chain fatty acids to be used in energy production, it is found in the subcutaneous (abdominal, gluteal and femoral) and visceral regions, being considered the adipose tissue with the greatest metabolic activity, playing an important role in mechanical protection [4,5].

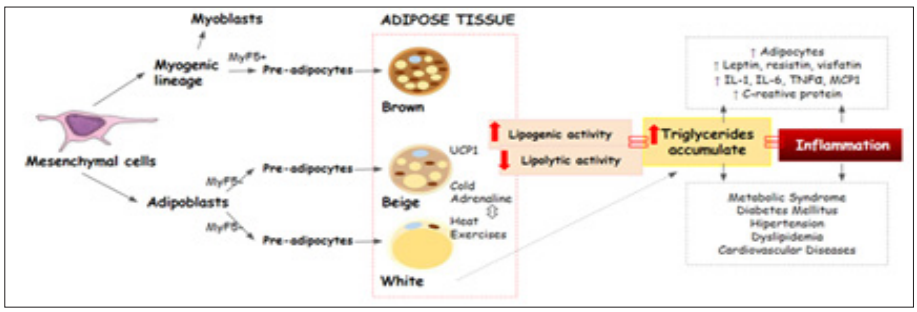

Figure 1: Adipose tissue formation and pathological complications triggered by the abnormal accumulation of fat in adipocytes.

Beige adipose tissue was discovered more recently. Beige cells may not have the same embryonic expression profile in all white fat deposits, and thus may have different origins and, consequently, different characteristics such as number of nerve fibers, vascularization and conditions of environmental exposures [6]. The presence of beige adipose tissue is stimulated

by cold and β3-adrenergic receptors and has a key component for energy burning called Uncoupling Protein 1 (UCP1), as well as having the ability to increase its expression, thus favoring increased energy expenditure. There is no specific location, for beige adipose tissue [2]. Mesenchymal cells differentiate into adipoblasts and myoblasts, with the action of MyF5+, transforming them into pre-adipocytes that later form brown, beige and white adipose tissues. As a result of greater lipogenic activity and less lipolytic activity, triglycerides accumulate in adipocytes, causing hypertrophy and/or hyperplasia thereof, favoring the elevation of leptin, resistin, vistatin, Interleukin 1 and 6 (IL-1, IL-6), Tumor Necrosis Factor α (TNF-α), monocyte and Macrophage Chemotraction Protein (MCP1) and C-Reactive Protein (CRP), thus intensifying an inflammatory process. Events can trigger factors for the onset of diabetes mellitus, hypertension, dyslipidemia, cardiovascular diseases, among others [7,8].

In this sense, it appears that the adipose tissue has several physiological functions and intense metabolic activity, contributing to energy balance, hormonal regulation (insulin and catecholamines), as well as the nutritional status in conditions of prolonged fasting or increased energy expenditure [9], mediated by the biosynthesis, incorporation and storage of triglycerides from food, as well as by non-lipid substrates, such as carbohydrate, which favors anabolic action, also called lipogenic activity [10]; further the release of stored triglycerides benefit the hydrolysis of TG into long-chain fatty acids or glycerol, to be mobilized into the tissues and promote catabolic action, that is, lipolytic activity [11]. Changes in the dynamics of energy homeostasis, that is, greater lipogenic activity and less lipolytic activity triggers the abnormal accumulation of fat producing hypertrophy and/or hyperplasia of adipocytes, thus naming obesity [5].

Obesity is a chronic health risk and disease, triggered by several etiological factors, including social, behavioral, environmental and genetic factors, defined as an abnormal or excessive accumulation of fat [12]. Over the past three decades, the prevalence of overweight and obesity has increased to 27.5% in adults and 47.1% in children worldwide [13]. The accumulation of adipose tissue is directly related to the expression of several proteins, and when referring to white adipose tissue, it is known that it is capable of producing about 50 cytokines and other molecules that are involved in various physiological or pathological processes acting through autocrine, paracrine and endocrine mechanisms [8,14].

Among the adipokines produced, leptin is a hormone that is directly related to energy metabolism by controlling food intake [7], and obese individuals produce high amounts of this hormone; however, it cannot act on the central nervous system , thus losing its ability to intervene in food consumption, and also has pro-inflammatory activity, that is, its presence causes an increased expression of Tumor Necrosis Factor-alpha (TNF-α) and Interleukin-6 (IL- 6) by monocytes and macrophages [15]. Other cytokines that show increased concentrations in the presence of adipose tissue are Monocyte Chemotactic Protein (MCP-1/CCL-2), Tumor Necrosis Factor alpha (TNF-α) and Interleukin-6 (IL-6) which are pro-inflammatory, produced by macrophages and lymphocytes, and are directly related to the development of Insulin Resistance (IR) [11,15,16]. Pro-inflammatory cytokines (TNF-α and IL-6) are able to induce the expression of resistin, visfatin and C-reactive protein, which is directly related to IR and is associated with the activation of inflammatory processes through a pathway dependent on the nuclear factor κB (NF-κB) [11,15,17]. Thus, uncontrolled food consumption, the intensification of the inflammatory process caused by increased concentrations of cytokines and insulin resistance, will favor the emergence of comorbidities, which currently represent 7.4% of diabetics and 24.5% of hypertensive individuals who are overweight, all of which increases the risk of morbidity and mortality along with a higher cost to health care [13].

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