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The Endocrine Role of Adipose Tissue and Its Management of Obesity-Related Diseases

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1. Letter to Editor

Adipose tissue plays a central role in regulating whole-body energy. Moreover, adipose tissue acts as an endocrine organ and produces numerous bioactive factors called adipokines which communicate with other organs and modulate a range of metabolic pathways: proteins (adiponectin, angiopoietins, chemerin, etc.), lipids (fatty acid esters of hydroxyl fatty acids, lysophosphatidic acids and sphingolipids), metabolites (uric acid and uridine) and microRNAs. However, excessive adipose tissue is associated with a chronic state of low-grade inflammation, caused by unbalanced production or secretion of these adipokines and can contribute to the development of obesity [1]. According to the World Health Organization, the global prevalence of obesity has nearly tripled since 1975 and in 2016, more than 1.9 billion adults, 18 years and older, were overweight. Of those over 650 million were obese [2]. An understanding of the mechanism underlying the metabolic actions of adipokines is fundamental for the development of novel therapeutics for obesity. Obesity has become the largest epidemic globally.

- A few adipokines are reduced in individuals with obesity.
- Anti-obesity treatments promote weight loss with side effects.
- Adiponectin can promote beneficial effects on obesity-related diseases.

2. Adiponectin as a therapeutic target for the treatment of obesity-related diseases.

Adiponectin also known as Acrp30, AdipoQ, GBP-28 and apM1 is the most abundant peptide secreted by adipocytes, whose reduction plays a central role in obesity-related diseases, including insulin resistance/type 2 diabetes and cardiovascular disease. Adiponectin exists in the circulation as varying molecular weight forms: low molecular weight trimers, intermediate molecular weight hexamers and high molecular weight dodeca to octadecamers. However, high-molecular weight complexes have the predominant action in metabolic tissues. Adiponectin has direct effects in liver, skeletal muscle and the vasculature mediated by adiponectin receptors, which occurs as two isoforms (AdipoR1 and AdipoR2) and binds to the non-signaling interacting protein T-cadherin. Adiponectin administration in humans and rodents has insulin-sensitizing, anti-atherogenic, anti-inflammatory effects and decreases body weight. Adiponectin elicits a number of downstream signaling events implicating a cross talk with the insulin

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signaling pathway. Metabolic actions of the adiponectin are carried out by activation of multiple signaling pathways including IRS1/2, AMPK and p38 MAPK. Activation of IRS1/2 by adiponectin signaling is a major mechanism by which adiponectin sensitizes insulin action in insulin responsive tissues [3]. In the liver, the activation of AMPK acts in the same pathway of insulin, in which both repress the expression of enzymes of gluconeogenesis, such as phosphoenolpyruvate carboxykinase, resulting a decrease in hepatic glucose production and glycemic control [4]. Therefore, adiponectin replacement therapy in humans may propound potential therapeutic targets in the treatment of obesity, insulin resistance/type 2 diabetes and atherosclerosis.

3. References

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