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Opinion

Does Adiposis Tissue have an Endocrıne Organ Effect?

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Obesity can include many different disease phenotypes, such as fatty liver, hypertension, insulin resistance, and abdominal adipose tissue deposition associated with low-grade systemic and chronic inflammation. In obese individuals; It increases the risk of developing type II diabetes mellitus, cardiovascular disease, osteoarthritis, and developing certain cancers such as endometrial, breast, and colon.

Chronic high-fat diet consumption leads to excessive lipid transport to metabolic organs, rapid weight gain, decreased insulin sensitivity, and impaired glucose homeostasis. Visceral obesity is associated with the development of chronic metabolic diseases such as insulin resistance, type 2 diabetes, and cardiovascular disease. Studies have shown that a diet high in fat and carbohydrates causes a significant increase in oxidative stress and inflammation in people with obesity. Oxidative stress occurs under physiological conditions and in many diseases and causes direct or indirect damage to different organs. Obesity and the resulting production of oxidative stress have been associated with the development of other pathologies such as diabetes mellitus, and cardiovascular diseases, the simplest of which is metabolic syndrome. Therefore, it is hypothesized that adipose tissue inflammation plays a critical role in the pathogenesis of obesity-related complications in obese individuals. It has been reported that obesity can induce systemic oxidative stress associated with dysregulated adipokine production that contributes to the development of metabolic syndrome.

Adipose tissue synthesizes and secretes various peptides, hormones and adipokines. These biologically active compounds play critical roles in the regulation of adipogenesis and energy metabolism. In contrast, accumulation of adipose tissues, especially excess visceral fat, causes dysregulation of these adipokines and adverse metabolic responses such as elevated inflammation and insulin resistance. Previous studies have reported that some molecules associated with adiposity are associated with obesity. Therefore, the determination of the levels of these molecules can be used as a tool to predict future risks. Considering the various biological functions of obesity-related molecules, it will be possible to explain the metabolic consequences of obesity and its relations with molecules. Although several cross-sectional studies have reported strong correlations between molecules associated with obesity, a limited number of studies have reported few associations between changes in molecules. Therefore, the long-term direction of their relations remains largely uncertain. For example, it is unknown whether increased inflammation is associated with changes in insulin resistance or leptin resistance. Adiposity-associated molecules have been shown to predict the future risk of obesity-related diseases.

Although the primary function of adipose tissue is to store excess nutrients as triacylglycerols and secrete free fatty acids during fasting, it also secretes large amounts of hormones and signaling molecules. These adipose tissue products are collectively called adipokines and may act as mediators between dietary patterns and disease risk. Although not all human adipokines are still fully characterized, it has become clear that adipose tissue is a potential source of more than 600 secreted proteins. Adipokines contribute to regulating appetite and satiety, fat distribution, insulin secretion and sensitivity, energy expenditure, endothelial

function, inflammation, blood pressure, and hemostasis. Indeed, an imbalance of pro- and anti-inflammatory adipokines secreted by white adipose tissue (WAT) due to the expansion of fat mass in obesity has been found to have potential effects on obesity-associated metabolic disorders. The increase in levels of these adipokines leads to the development of a dysfunctional WAT and chronic low-grade inflammation characterized by inflammation as well as infiltration of pro-inflammatory immune cells and inappropriate extracellular matrix remodeling and impaired angiogenesis in WAT. The released adipokines can modulate various cell signals, which can prevent or exacerbate metabolic complications, depending on the type at systemic or intracellular levels.

Obestatin is a 23 amino acid peptide hormone derived from the ghrelin gene, produced in the stomach a several other tissues in the body. While it was originally reported that obestatin opposes the effects of ghrelin on appetite and food intake, it is now clear that obestatin is not an endogenous ghrelin antagonist, but a multifunctional peptide hormone in its own right. Scientists showed that Humanin peptides can lower many markers associated with age-related metabolic disorders. It shows that the effects of humanin on cardiovascular function, insulin sensitivity, and anti-inflammation are promising in obesity. Kisspeptin and Spexin are peptides that play a role in the regulation of body weight and metabolism. They correlate negatively with obesity, insulin resistance indices, and hormones, which are known to affect insulin sensitivity in women. Both Kisspeptin and Spexin may be related to the pathophysiology of obesity and insulin resistance. Asprosin is a recently discovered fasting hormone that stimulates hepatic glucose production. Plasma asprosin has been shown to cross the blood-brain barrier and directly activate orexigenic AgRP+ neurons in a cAMP-dependent pathway. Genetic deficiency of asprosin in humans results in a syndrome characterized by anorexia and emaciation, phenocopied by mice carrying similar mutations, and completely salvaged by asprosin expression. It has also been found that obese humans and mice pathologically exhibit high concentrations of asprosin in their serum, and neutralization of plasma asprosin using a monoclonal antibody reduces appetite and body weight in obese mice. Therefore, asprosin, besides performing a glucogenic function, is a centrally acting orexigenic hormone and may be a potential therapeutic target to treat both obesity and diabetes.

Future studies need to investigate the relationship between some obesity-related peptides and adipokines. For example, changes in serum levels of Preptin, Adropin, Adiponectin, Irisin, Vaspin, Leptin, Resistin Obestatin, Humanin, Kisspeptin, Spexin, Asprosin, etc molecules depending on body mass indexes can be investigated. It is thought that the data to be obtained will contribute to important results by combining the previous studies on obesity molecule relations. Therefore, systemic or intracellular control of these factors has been an ideal therapeutic target aimed at preventing obesity or ameliorating its associated complications. In terms of increasing the success rate for the treatment of obesity, it is thought that discovering how these hormones are related to nutrition will benefit the treatment. In addition, it is anticipated that such studies will lead to the elucidation of the molecule-mediated pathways linking obesity and obesity-related diseases, and will reveal the potential value of these molecules in predicting obesity-related conditions. It is thought that it will be effective in determining the molecules to be targeted and in the development of drugs for them.