



Adipose Tissue Regulator in Obesity During Fibrosis

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DESCRIPTION

Obesity is causally linked with the development of cardiovascular disease. Accumulated evidence indicates that cardiovascular disease is the collateral damage of obesity-induced adipose tissue dysfunction that promotes a chronic inflammatory state in the body. Adipose tissues secrete bioactive compounds called adipokines, which largely function as modulators of inflammation. The microenvironment of the adipose tissue will affect the adipokine secretome, with actions on distant tissues. Obesity typically leads to upregulation of pro-inflammatory adipokines and downregulation of anti-inflammatory adipokines, contributing to the pathogenesis of cardiovascular disease. In this study, we focus on the microenvironment of adipose tissue and how it affects cardiovascular disease, including atherosclerosis and ischemic heart disease, through the systemic effects of adipokines.

Obesopathy, regional obesity and cardiovascular risk

Although adipose tissue quantity is undoubtedly associated with cardiovascular risk, recent human data indicate that differences in adipose tissue quality, which can be assessed directly by immunohistochemistry or noninvasively by radio density attenuation imaging by computed tomography, are closely related to insulin resistance, cardio metabolic risk, and all the others cause mortality regardless of total fat volume. Adipose tissue abnormalities may be key factors regulating systemic metabolism and leading to Cardio Metabolic Diseases (CMD). Although animal models of obesity tend to produce fairly uniform phenotypes, the degree of adipose tissue dysfunction in obese humans shows significant heterogeneity, with lower degrees of adiposopathy being associated with systemic metabolic profiles and function. This inter individual variability in adipose tissue quality may be partly related to lifestyle differences, as physical activity impacts adipose tissue physiology and Temporomandibular Disorders (TMD) risk. Differences in adipose tissue quality are also closely related to the observation that different fat deposits have different effects on the propensity to develop TMD.

Numerous clinical studies using obesity measures such as waist circumference and waist-to-hip ratio as markers of central obesity, as well as abdominal cross-sectional imaging, have found clear associations between fat load total and systemic dysmetabolism, with generally stronger associations for visceral disorders. It is now recognized that visceral fat expansion is strongly associated with increased cardio metabolic risk, while subcutaneous fat expansion makes a small contribution or, in some cases, even reduces the risk of metabolic disorders. Therefore, it has been hypothesized that visceral fat is lower quality than subcutaneous deposits and has specific properties associated with higher cardio metabolic risk. The subcutaneous fat is made up of the total body fat mass, while the abdominal visceral adipose tissue constitutes from 5% to 20%. Although visceral fat is not the predominant store of white adipose tissue, inflammatory markers, including interleukin, C-reactive protein, and Tumor Necrosis Factor (TNF), tend to circulate in higher concentrations in subjects with abdominal obesity compared to peripheral and visceral fat has been shown to be an important source of circulating free fatty acids. Although arterial disease tends to worsen with increasing total weight load in adults and children, computed tomography or magnetic resonance imaging studies of fat compartments identify visceral fat volume as more strongly associated with systemic endothelial dysfunction than subcutaneous fat.

Deposits of visceral and subcutaneous fat arise during development from different origins and this may partly explain the tendency of visceral fat to develop different metabolic, inflammatory, angiogenic and lipolytic properties that contribute to CMD compared to the subcutaneous. The mechanisms by which fat stores expand in response to excess calorie intake are a critical determinant of the risk of metabolic dysfunction and Cardiovascular Disease (CVD). This expansion is mediated by an increase in the number of adipocytes (hyperplasia) and an increase in the size of adipocytes (hypertrophy). It is classically accepted that hyperplasia allows healthy expansion of adipose tissue because it is mediated by the formation of functional adipocytes from progenitor cells (adipogenesis). In contrast,

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adipocyte hypertrophy typically results in dysfunctional lipid-laden adipocytes that undergo cell death and contribute to inflammation, dysfunction, and associated pathologies of adipose tissue. Different adipose tissue depots contribute differently to disease processes, which may be associated with

dysfunctional expansion of different adipose depots. It has been suggested that in many people, subcutaneous fat has limited extensibility due to deficient adipogenic capacity, leading to hypertrophy of subcutaneous adipocytes (hypertrophic obesity) and ultimately promoting fat storage in depots.