

Obesity is characterized by dysfunctional alterations in the ability of the adipose tissue to store fat. In fact, as the adipose tissue expands it becomes resistant to insulin and increase the release of lipids into the circulation. This exposes skeletal muscle and liver to overwhelming amounts of fat, which also leads to insulin resistance in these peripheral tissues. The expansion of the visceral fat depot seems to be the most deleterious, since it has been associated with the development of type 2 diabetes, hypertension, and cardiovascular disease. In this context, this study was designed to investigate the molecular and physiological mechanisms underlying the dysfunctional alterations that occur in visceral (VC) and subcutaneous (SC) adipose tissues in diet-induced obesity. We found that the molecular machinery involved in lipid storage and release is defective in both VC and SC adipose tissues in obesity, leading to chronically elevated levels of fat in the blood. We have also identified specific signaling steps of the lipid breakdown cascade that are disrupted in obesity. Identification of this defective regulatory mechanisms can help in the development of novel therapeutic strategies to treat obesity and its related metabolic disorders.

Gaidhu MP, Anthony NM, Patel P, Hawke TJ, Ceddia RB. Dysregulation of lipolysis and lipid metabolism in visceral and subcutaneous adipocytes by high-fat diet: role of ATGL, HSL, and AMPK. *Am J Physiol Cell Physiol.* 2010 Apr;298(4):C961-71.

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