

# White adipose tissue

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**Abstract:** White adipose tissue (WAT) or white fat is one of the two types of adipose tissue found in mammals. In healthy, non-overweight humans, white adipose tissue composes as much as 20% of the body weight in men and 25% of the body weight in women. Its cells contain a single large fat droplet, which forces the nucleus to be squeezed into a thin rim at the periphery. They have receptors for insulin, growth hormones, norepinephrine, and glucocorticoids. White adipose tissue is used as a store of energy. Upon release of insulin from the pancreas, white adipose cells' insulin receptors cause a dephosphorylation cascade that lead to the inactivation of hormone-sensitive lipase. White adipose tissue also acts as a thermal insulator, helping to maintain body temperature. The hormone leptin is primarily manufactured in the adipocytes of white adipose tissue.

**Keywords:** Function of white adipose tissue

## Introduction

White adipose tissue is used as a store of energy. White adipose tissue is the most common and is the fat that so many of us complain of acquiring. Upon release of insulin from the pancreas, white adipose cells' insulin receptors cause dephosphorylation cascade that lead to the inactivation of hormone-sensitive lipase. Fatty acids are taken up by muscle and cardiac tissue as a fuel source, and glycerol is taken up by the liver for gluconeogenesis[1]. White adipose tissue also acts as a thermal insulator, helping to maintain body temperature. The hormone leptin is primarily manufactured in the adipocytes of white adipose tissue. WAT can be found associated with numerous organs its functions are more than just insulation of the organ and a ready reservoir of fat for energy production. Depending on its location WAT serves specialized functions. Kidney associated WAT plays a role in sodium reabsorption and therefore can affect intravascular volume and hypertension[2]. WAT contains macrophages, leukocytes, fibroblasts, adipocyte progenitor cells, and endothelial cells. The presence of the fibroblasts, macrophages, and other leukocytes along with adipocytes, accounts for the vast array of proteins that are secreted from WAT under varying conditions. The highest accumulations of WAT are found in the subcutaneous regions of the body and surrounding the viscera (internal organs of the chest and abdomen). WAT is composed of adipocytes held together by a loose connective tissue that is highly vascularized and innervated[3]. White adipocytes are rounded cells that contain a single large fat droplet that occupies over 90% of the cell volume. The mitochondria within white adipocytes are small and few in number. The mitochondria and nucleus of the white adipocyte is squeezed into the remaining cell volume[4].

## Mitochondrial dysfunction in WAT

Although mitochondria in brown adipose tissue and their role in non-shivering thermogenesis have been widely studied, we have only a limited understanding of the relevance of mitochondria in white adipose tissue (WAT) for cellular homeostasis of the adipocyte and their impact upon systemic energy homeostasis[5]. A better understanding of the regulatory role that white adipocyte mitochondria play in the regulation of whole-body physiology becomes increasingly important. [6] WAT mito-biogenesis can effectively be induced pharmacologically using a number of agents, including PPAR $\gamma$  agonists. Through their ability to influence key biochemical processes central to the adipocyte, such as fatty acid (FA) esterification and lipogenesis, as well as their impact upon the production and release of key adipokines, mitochondria play a crucial role in determining systemic insulin sensitivity.[7]

## Origin of chronic inflammation in obesity and contribution of the WAT

obesity associated with a reduced production of adiponectin, usually considered as a factor with anti-inflammatory properties. In contrast to what is seen in recognised inflammatory diseases, obesity is associated with a moderate, but chronic, increase of this 'cocktail' of inflammatory factors. The relative contribution of the different organs and tissues (such as liver, lymphoid system, subcutaneous and visceral adipose tissue) in the circulating levels of inflammatory cytokines is difficult to determine in obesity and during the different phases of its evolution. Indeed, especially in cases of extreme obesity, where all the different adipose depots of the organism are enlarged, the respective contributions of both adipose tissue mass and other tissues in the production of pro-inflammatory factor.[8]

## Physiological role of WAT

The traditional role attributed to white adipose tissue is energy storage, fatty acids being released when fuel is required. The metabolic role of white fat is, however, complex. For example, the tissue is needed for normal glucose homeostasis and a role in inflammatory processes has been proposed[9]. A radical change in perspective followed the discovery of leptin; this critical hormone in energy balance is produced principally by white fat, giving the tissue an endocrine function. Leptin is one of a number of proteins secreted from white adipocytes, which include angiotensinogen, adipin, acylation-stimulating protein, adiponectin, retinol-binding protein, tumour necrosis factor  $\alpha$ , interleukin 6, plasminogen activator inhibitor-1 and tissue factor. Some of these proteins are inflammatory cytokines, some play a role in lipid metabolism, while others are involved in vascular hemostasis or the complement system[10]. The effects of specific proteins maybe autocrine or paracrine, or the site of action maybe distant from

adipose tissue. The most recently described adipocyte secretory proteins are fasting-induced adipose factor, a fibrinogen-angiopoietin-related protein, metallothionein and resistin. Resistin is an adipose tissue-specific factor which is reported to induce insulin resistance, linking diabetes to obesity. Metallothionein is a metal-binding and stress-response protein which may have an antioxidant role[11].

### **Protein secreted by WAT( Leptin)**

Leptin is 16kDa peptide whose central function is the regulation of overall body weight by limiting food intake and increasing energy expenditure. However, leptin is also involved in the regulation of the neuroendocrine axis, inflammatory responses, blood pressure, and bone mass. The human leptin gene is the homolog of the mouse "obese" gene (symbol OB) that was originally identified in mice harboring a mutation resulting in a several obese phenotype.

Leptin functions by binding to its receptor which is a member of the cytokine receptor family. Leptin and its receptors possess structural similarities to the IL-6 family of cytokines and the class I cytokine receptor family. The leptin receptor mRNA is alternatively spliced resulting in six different products. The leptin receptors are named Ob-R, OB-Rb, OB-Rc, Ob-Rd, Ob-Re, and Ob-Rf. The OB-Rb mRNA encodes the long form of the leptin receptor (also called LEPR-B) and is expressed primarily in the hypothalamus but is also expressed in cells of the innate and adaptive immune systems as well as in macrophages. The other receptor subtypes are expressed in numerous tissues including muscle, liver, kidney, adrenal glands, leukocytes, and vascular endothelium [12].

Leptin expression is under complex control and a number of transcription factor binding sites have been identified in the promoter region of the leptin gene. Leptin levels are higher in age and weight-matched females compared with males. This is partially due to the inhibition of leptin expression by androgens and the stimulation of expression by estrogens[13]. Leptin expression has been shown to be increased by sex steroids, glucocorticoids, cytokines, and toxins released during acute infection. The sympathetic nervous system triggers a reduction in circulating leptin levels via the release of catecholamines. This effect of catecholamines has been shown to be due to activation of  $\beta$ -adrenergic receptor signaling [14].

In addition to effects on appetite exerted via central nervous system functions, leptin is also known to exert effects on inflammatory processes. Leptin modulates peripheral T cell function leading to increased levels of T helper cell type 1 cytokines. In addition leptin reduces thymocyte apoptosis and increases thymic cellularity[15]. These results correlate well with observations demonstrating a reduced capacity for immunologic defense when leptin levels are low. However, too much leptin is not beneficial as high concentrations can result in an abnormally strong immune response which predisposes an individual to autoimmune phenomena. Acute stimulation with pro-inflammatory cytokines results in increased serum levels of leptin, whereas, chronic stimulation by IL-1, IL-6, or TNF $\alpha$  leads to reduced levels of serum leptin[16].

### **Effect of dietary fat on white adipose tissue metabolism**

The prevalence of obesity is increasing worldwide, and data from the literature indicate that environmental and behavioral aspects play an important causal role. Among the environmental influences, the percentage of fat energy in the everyday diet and the lack of physical activity are two important factors[17]. Obesity is often accompanied by abnormalities in carbohydrate and lipid metabolism and in insulin and leptin secretion and action .

Exposure to high-fat diets for prolonged periods results in positive energy balance and obesity in certain rodent models that can be considered an adequate model of human obesity. The hyperlipidic diet induced a more pronounced body weight gain accompanied by an increase in the adiposity, carcass lipogenesis rate and serum triglycerols, regardless of the regimen of administration, i.e., either continuous or cycled with chow[18].

It has been shown that dietetic manipulations, hormones, and cytokines induce distinct metabolic responses at different fat depots [19]. High-fat diets reduced the activity of lipogenic enzymes and lipogenesis rate in retroperitoneal and inguinal fat depots but increased lipoprotein lipase activity in visceral fat. The type of dietary fat has been shown to influence hepatic metabolism. Although it is well documented that consumption of high-fat diets can induce obesity, the impact of dietary fatty acid composition on adipose tissue lipid metabolism has been examined by some authors, with conflicting results[19].

### **Conclusion**

The WAT associated with abdominal and thoracic organs (excluding the heart), the so-called visceral fat, secretes several inflammatory cytokines and is thus involved in local and systemic inflammatory processes. WAT associated with skeletal muscle secretes free fatty acids, interleukin-6 (IL-6) and tumor necrosis factor- $\alpha$  (TNF $\alpha$ ) and as a consequence plays a significant role in the development of insulin resistance. Cardiac tissue associated WAT secretes numerous cytokines resulting in local inflammatory events and chemotaxis that can result in the development of atherosclerosis and systolic hypertension. more research may be required to know the mechanisms of action of white adipose tissue

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