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Commentary

Subcutaneous adipose tissue & visceral adipose tissue

Adipose tissue is a loose connective tissue mostly composed of adipocytes and plays a major role in storage of energy in the form of lipids. Adipose fat also serves as an important cushion and insulates the body from heat and cold. Many physiological, psychosocial and clinical factors influence the amount and distribution of the adipose tissue throughout the human body. Besides its passive function, adipose tissue has been recognized as a major endocrine organ, because it produces hormones such as leptin, estrogen, resistin and the cytokine tumour necrosis factor alpha[1]. In recent years, global obesity epidemic has enhanced interest in adipose tissue biology. The adipose tissue beneath the skin is called subcutaneous adipose tissue (SAT), whereas the one lining internal organs is termed visceral adipose tissue (VAT). There are considerable anatomical differences in the distribution of two adipose tissues in the body. VAT is present mainly in the mesentery and omentum and drains directly through the portal circulation to the liver. During development, total body fat (TBF), VAT and SAT typically increase as a child ages, though different trends are observed in males and females[2]. Sexual maturation greatly influences the distribution of adipose tissue. Girls accumulate more TBF and SAT during and after puberty, depositing fat preferentially in the gynoid and extremity regions. On the other hand, pubertal and post-pubertal boys deposit more fat in the abdominal region, particularly in the VAT depot[3]. Body fat distribution also changes according to menopausal status; SAT areas are higher in pre-menopausal women, whereas VAT areas and the subcutaneous to visceral abdominal adipose tissue (abdominal SAT & VAT) area ratios are higher in post-menopausal women. Ethnic differences in TBF are rather mixed. VAT tends to be higher in individuals of European origin and Hispanic youth, whereas SAT is typically higher in African American youth. East Asians (Chinese, Japanese and Koreans) have largest accumulation of VAT, but the lowest accumulation of deep SAT[4].

Several health risks are associated with high amounts of TBF, VAT and SAT, including insulin resistance, hepatic steatosis, metabolic syndrome and hypertension. Epidemiological studies highlighted that VAT accumulation associates with an increased metabolic risk and overall mortality, whereas SAT expansion ameliorates insulin sensitivity and decreases type 2 diabetes (T2D) risk. These risks are affected by genetic, biological and lifestyle factors including physical activity, nutrition and stress. Visceral fat is the main determinant of insulin resistance, and many investigators have established links between excessive visceral fat and an exaggerated inflammatory state[5]. A close relationship exists between high morbidity and intra-abdominal (visceral) fat obesity, rather than extra-abdominal (subcutaneous) fat obesity. The T2D is predicted by central obesity and circulating adipokines regulating inflammation. VAT in T2D expresses higher levels of adipokines involved in inflammation which is related to fasting glucose and insulin action. Increased production of these proinflammatory molecules by VAT may explain the links observed between visceral obesity, insulin resistance and diabetes risk[2]. Morbid obesity, ageing, hormonal status, nutrition, low physical activity and other environmental factors impair SAT relative resistance to dysfunctional changes and promote development of metabolic disorders[6]. Abdominal VAT and SAT are both associated with adverse cardiometabolic risk factors, but VAT remains more strongly associated with these risk factors[7].

Although both SAT and VAT express identical set of genes, the level of expression is different. The expression of critical proinflammatory genes is substantially higher in SAT than that in VAT in individuals with morbid obesity. It is believed that there is a compartment-specific adipose tissue contribution to inflammation in obesity and abdominal SAT contributes more than VAT to the proinflammatory milieu associated with severe obesity[8].

Subcutaneous fat is soft, and there are many options for treating this type of fat. Lifestyle modification works for some people. Liposuction is also an option for the removal of excess SAT. However, for visceral fat, the only option to getting rid of it is good lifestyle modification, good eating habits and physical activity. To understand the role of SAT and VAT in pathophysiology of obesity, Ronguillo *et al*[9] have published their study in this issue. Their study design has two components: discovery and validation. For the discovery phage, they obtained surgical specimens from a group of 16 patients [8 non-diabetic normal-weight individuals with body mass index (BMI) <25 kg/m^2 and 8 patients with obesity, BMI >30 kg/m^2]. SAT and VAT samples were obtained from each patient. Global gene expression in SAT and VAT was determined in RNA isolated from the surgically obtained adipose tissues, using two-Color Agilent Microarray-Based Gene Expression Analysis. The results demonstrated significant changes in the tissue transcriptomes, with decreased expression of 327 genes and increased expression of 488 genes in SAT compared with those in VAT. Microarray-based gene expression findings were validated by quantitative real-time PCR among a confirmation group of 72 patients (35 non diabetic normal weight individuals and 37 obese individuals), in addition to 16 samples from the discovery phage. Analysis of their data showed little differences in the expression of adiponectin (*ADPN*) and peroxisome proliferator-activated receptor gamma (*PPARG*) genes from obese individuals in either SAT and VAT tissues. However, the gene expression of mevalonate diphosphate decarboxylase (*MVD*) was decreased by obesity in SAT, whereas the levels of phosphatidic acid phosphatase type 2C (*PPA2C*), cytochrome P450 4A11 (*CYP4A11*) and cytochrome P450 17A1 (*CYP17A1*) were significantly increased by obesity in VAT. Similarly, the isozyme levels of the long-chain fatty acid-coenzyme A ligase gene (*ACSL3*), succinyl-CoA ligase (*SVCGL*), isocitrate dehydrogenase 2 (*IDH2*), mitochondrial glycerol-3-phosphate acyltransferase (*GPAM*), patatin-like phospholipase domain-containing protein 3 (*PNPLA3*) and cytochrome P450 1B1 (CYP1B1) were significantly downregulated, whereas *PPAP2C*, *CYP4A11* and *CYP17A1* gene expression did not differ in the VAT of obese individuals.

Pathway analysis of validated differentially expressed genes revealed that genes involved in pathways such as *ADPN* and *PPARG* exhibited altered expression in the samples of both types of adipose tissue from the obese patients, whereas genes related to lipogenesis and energy metabolism were mainly limited to visceral fat from obese individuals. The present study reinforces the fact that the expression of different genes in SAT and VAT is affected by different extents in obesity. Such differential transcriptome analysis, authors suggest, can be useful as a new approach to discover novel genes related to obesity[9].

The colon is surrounded by mesenteric visceral body fat, and there is a direct physical and vascular interface with VAT. Based on intimate connection, it has been hypothesized that VAT-produced metabolites may have a direct influence on obesity-associated colon cancer aggressiveness and progression. Therefore, gene expression profiling of VAT may have direct bearing on molecular connectivity between obesity and cancer also.

***Conflicts of Interest*:** None.

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