

Visceral Fat and Diabetes: A Direct Relationship?

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A long-term discrepancy in energy intake and expenditure (i.e., positive energy balance) is the important cause of obesity resulting in increased body mass such as accumulation of subcutaneous and visceral fat. Even though general obesity is a risk factor for several diseases, many human studies have shown that visceral fat, which is the fat in the viscera, is most closely linked to many health conditions, such as cardiovascular diseases, insulin resistance and type 2 diabetes mellitus [1]. The mechanism(s) of connecting visceral fat with Metabolic Syndrome is not completely clear, but its anatomical area has been recommended, resulting in a 'portal' effect of higher free fatty acid (FFA) release and glycerol discharge [2]. Evidence has also shown that adipose tissue is an efficient endocrine organ that can secrete several cytokines, sometimes linked to as adipokines, which can encourage inflammatory responses and interfere with the action of insulin [3]. In addition, some studies have demonstrated that subcutaneous and visceral fat are physiologically different, with visceral fat showing far higher pro-inflammatory properties than subcutaneous fat [4].

Various lines of evidence have recently suggested a role for accumulation of capital of visceral fat (VF) in insulin resistance pathogenesis. Therefore, VF excess was correlated with 1) reduced glucose uptake sensitivity to insulin stimulation as quantified by euglycemic insulin clamping technique [5], 2) decreased free fatty acid (FFA) reesterification rate [6] and 3) enhanced lipolysis resistance to insulin inhibitory effect in both visceral and peripheral adipocytes [7]. Furthermore, VF accumulation is frequently correlated with general adiposity [8], making it compulsory to account for obesity when looking to create an independent role for VF in metabolic control. These predictions prove that appropriate VF measurement is an essential aspect of clinical phenotyping and has rather direct implications for the metabolic control of patients with type 2 diabetes.

A progressive change in visceral adiposity is a common characteristic of ageing and epidemiological data supports its function as a notable risk factor for metabolic syndrome, diabetes and cardiovascular atherosclerotic mortality [1]. The percentage of visceral fat (VF) correlates best with the insulin sensitivity in animal models and in humans among different body fat depots. Insulin action in people with visceral obesity is significantly impaired [9] and scientific studies has shown that VF can account for most of the variability in insulin sensitivity in heterogeneous populations [10]. These researches, furthermore, are evolutionarily conserved in nature and VF could necessarily be a "marker" with more complex endocrine and metabolic abnormalities rather than playing a "causative" role in insulin resistance pathogenesis and its metabolic implications. Hypothetical systems responsible for modulating insulin action by VF also provide enhanced portal release of free fatty acids (FFAs) [11] and/or abnormal fat-derived peptide expression and secretion like resistin [12], leptin, ACRP30, and tumor necrosis factor- (TNF-) [13]. A accurate observation in ageing biology is that recurrent restriction of caloric intake in rodents greatly improves survival and helps to prevent insulin resistance from occurring. It has observed that its prevention of VF accumulation largely accounts for the positive effects of caloric restriction (CR) on the metabolic modifications in aging [1,14]. It is important to distinguish the possible effects of a significant reduction in VF per se from other nutritional, anthropometric, and metabolic effects of CR in order to evaluate specifically the contribution of VF to the insulin resistance of ageing. Regular physical activity together with restriction of energy intake is an appropriate first-line strategy for reduction of VF and managing obesity, metabolic syndrome and

diabetes [15]. Weight reduction in patients with obesity, metaboðoc syndrome and diabetes has been confirmed to optimize cardiovascular risks following such a short-term action.

However, a possible direct relationship between visceral fat and diabetes remains controversial again.

Disclosure Statement

The author declare that there are no conflicts of interest.

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