International Journal of Home Science 2021; 7(3): 152-155

International Journal of Home Science

ISSN: 2395-7476
IJHS 2021; 7(3): 152-155

www.homesciencejournal.com
Received: 04-07-2021
Accepted: 07-08-2021

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Abdominal obesity and health risk

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Abstract
Abdominal fat or truncal obesity consists of both subcutaneous and visceral fat. It has been found that visceral fat is more metabolically active than subcutaneous fat. The low-level inflammation linked with abdominal fat is associated with insulin resistance and with increases in the release of inflammatory adipokines and cytokines. As a result of these changes, abdominal fat can cause a variety of health conditions. In this review, we focus on the adverse effects of abdominal fat on the body and how it can lead to the development of cardiovascular disease, hypertension, type 2 diabetes mellitus, hyperlipidemia, stroke and cancer. Additionally, we discuss how abdominal fat can be reduced as a result from correction of hormonal deficiencies. Abdominal (visceral) obesity is thought to be the predominant risk factor for metabolic syndrome and as predictions estimate that 50% of adults will be classified as obese by 2030 it is likely that metabolic syndrome will be a significant problem for health services and a drain on health economies. The most widely used measures for abdominal obesity include waist circumference (WC) and waist-to-hip ratio (WHR), which are determined by both environmental and genetic factors.

Keywords: Adipokines, cytokines, fat, inflammation, obesity, abdominal obesity

1. Introduction
Abdominal obesity, also known as central obesity and truncal obesity, is a condition when excessive abdominal fat around the stomach and abdomen has built up to the extent that it is likely to have a negative impact on health. Abdominal obesity has been strongly linked to cardiovascular disease, (Yusuf S, *et al.* 2004) [38]. Alzheimer's disease, and other metabolic and vascular diseases (Razay G, *et al.* 2006) [31].

Visceral and central abdominal fat and waist circumference show a strong association with type 2 diabetes (Anjana M, *et al.* 2004) [1].

Visceral fat, also known as organ fat or *intra-abdominal fat*, is located inside the peritoneal cavity, packed in between internal organs and torso, as opposed to subcutaneous fat, which is found underneath the skin, and intramuscular fat, which is found interspersed in skeletal muscle. Visceral fat is composed of several adipose depots including mesenteric, epididymal white adipose tissue (EWAT), and perirenal fat. An excess of adipose visceral fat is known as central obesity, the "pot belly" or "beer belly" effect, in which the abdomen protrudes excessively. This body type is also known as "apple shaped", as opposed to "pear shaped" in which fat is deposited on the hips and buttocks.

The obesity pandemic plagues many lives and poses a tremendous health risk. Obesity can be classified as either android or gynoid obesity Ribisl, P.M. (2004) [30]. Android obesity occurs when fat is deposited in the abdominal (truncal) region. Gynoid obesity occurs when fat is deposited in the buttocks and thighs (hips). Android obesity is more of a health concern than gynoid obesity. There are several ways in which we can measure the degree of obesity in an individual. By measuring the body weight and height, one can calculate the body mass index (BMI) specific to that person. An adult with a BMI of >30.0 kg/m2 or ≥40 kg/m2 is classified as obese or morbidly obese, respectively Racette, S.B. *et al.* 2003 [29]. By measuring waist circumference, we can associate with body weight. Women with a waist circumference >80 cm and men with a waist circumference >94 cm are at an increased health risk. Generally, obesity is caused when energy consumption exceeds the amount of energy expended. In order to com- pensate for the extra energy, adipose tissue is utilized in energy storage, and when there is a large demand of lipid storage, adipocyte hypertrophy occurs (Racette, S.B., *et al.* 2003) [29]. In adipocyte hypertrophy, fat cells increase their volume so that they can withstand the large lipid storage.

Thus, this results in weight gain in adults. Adipose tissue can be categorized into white and brown adipose tissue and brown adipose tissue. Adipose tissue mainly consists of white adipose tissue which is used in energy storage, whereas brown adipose tissue is used as a homeostatic mechanism (Fantuzzi, G. 2005) [12]. In the abdomen, fat can be stored as either visceral fat or subcutaneous fat. When normal subcutaneous adipose tissue storage sites are full, and the body needs to store excess triglycerides, the excess triglycerides are stored in abnormal subcutaneous adipose tissue storage sites. Abnormal subcutaneous adipose storage sites have a decreased ability to store fatty acids and can also cause an abundant release of free fatty acids (FFAs) (Jenson, M.D. 2008) [18]. It has been determined that upper body subcutaneous fat is more lipolytically active than lower body fat. However, visceral fat is more lipolytically active than subcutaneous fat. Thus, visceral fat is more of a health threat than sub- cutaneous fat. It has been found that higher levels of abdominal subcutaneous adipose tissue and abdominal visceral adipose tissue have been shown to increase the risk of cardiometabolic conditions (Liu, J., *et al.* 2010) [22]. Effective methods of assessing abdominal fat are dual-energy X-ray absorptiometry (DEXA) and anthropometric measures, which are instrumental in the calculation of visceral fat (Direk, K., *et al.* 2013) [8].

2. Pathophysiology

Abdominal fat causes a variety of changes throughout the body, which are elicited through triggering of the immune system. This can be categorized as low-level chronic inflammation. Specific mechanisms responsible for proportionally increased visceral fat storage when facing positive energy balance and weight gain may involve sex hormones, local cortisol production in abdominal adipose tissues, endocannabinoids, growth hormone, and dietary fructose (Andre Tchernof *et al.* 2013) [3].

2.1 Cardiovascular Disease

The occurrence of chronic inflammation is associated with an increased risk of cardiovascular disease (Fantuzzi, G. 2005) [12]. Low-level chronic inflammation disrupts cardiovascular function and it can even accelerate atherosclerosis (Alam, I., *et al.* 2012) [2]. According to the INSPIRE ME IAA Study, increased abdominal circumference associated with visceral fat is strongly associated with cardiovascular disease (Smith, J.D., *et al.* 2012) [35]. Epicardial adipose tissue is visceral fat deposited in the chest cavity, which covers the coronary arteries and muscular tissue of the heart. Epicardial adipose tissue is linked with high-risk plaques and atherosclerosis. It has been found that individuals who suffer from coronary artery disease (CAD) have increased epicardial adipose tissue thickness and volume, as compared to individuals devoid of CAD (Echavarria-Pinto, M., *et al.* 2013) [9].

2.2 Type 2 Diabetes Mellitus

Hyperinsulinemia and insulin resistance are both associated with an increased risk of type 2 diabetes mellitus (Hsu, I.R., *et al.* 2007) [14]. In the body, visceral fat is a source of lipids. These lipids are transported to the liver via the hepatic portal vein. The accumulation of these lipids interferes with insulin signaling in liver cells (Direk, K., *et al.* 2013) [8]. This occurs because excess FFAs can alter blood glucose levels through several mechanisms, including excess of acetyl coenzyme A, as well as a reduction of nicotinamide adenine dinucleotide, ATP and citrate (Jenson, M.D. 2008) [18]. Increased levels of adipokines and cytokines are also linked to visceral fat. The latter in- creased levels of cytokines can deter insulin receptor kinase activity, thus disrupting insulin signaling by inhibiting the transmission of chemical responses within cells (Alam, I., *et al.* 2012) [2]. In addition, obesity causes a decrease in insulin sensitivity of the liver and thereby leading to insulin resistance (Hsu, I.R., *et al.* 2007) [14]. Thus, the liver extracts less insulin than usual. Furthermore, visceral fat causes an increase in the amount of secreted insulin which enters the blood. The decrease in extracted insulin by the liver and increase in the amount of secreted insulin entering the blood can cause hyperinsulinemia.

2.3 Hyperlipidemia

Hyperlipidemia is a condition in which there are high levels of fat particles (lipids) in the blood. The metabolism of lipoproteins is controlled by hepatic triglyceride lipase (HTGL) (Ebbert, J.O. and Jensen, M.D. 2013) [10]. When the concentration of post-hepatic HTGL is high, then the serum concentration of high-density lipoprotein (HDL) cholesterol is low. In visceral adipose tissue there is high HTGL activity. Thus, visceral fat causes low levels of HDL cholesterol (also referred to as "good cholesterol"). Visceral fat also contains high levels of triglycerides. The abundance of triglycerides is linked with high concentrations of very-low-density lipoprotein (VLDL) as well as low- density lipoprotein (LDL). Thus, visceral fat can cause increased concentrations of VLDL and LDL in the blood.

2.4 Hypertension

Hypertension is a condition in which there is elevated systemic blood pressure. Adiponectin is an adipokines that is secreted by adipocytes; however, in obesity, there are decreased levels of adiponectin (Summer, R., *et al.* 2011) [34] Adiponectin is a vasodilator and it helps to regulate blood pressure. In obese individuals, subcutaneous adipose tissue expresses a greater amount of adiponectin than in visceral adipose tissue. Thus, excess visceral adipose tissue is a factor which can lead to the development of systemic hypertension through low-level chronic inflammation and de- creased levels of adiponectin.

2.5 Stroke

Obesity is associated with an environment that promotes inflammation and thrombosis (Zhang, X., *et al.* 2009) [39]. This type of environment can lead to the development of stroke. Specifically, it has been shown that an increase in abdominal obesity leads to an increased risk of cerebral thrombosis. In the human body, the carotid arteries provide the majority of blood to the brain. The Multicultural Community Health Assessment Trial (M-CHAT) Study demonstrated that increased abdominal girth, in particular, visceral adipose tissue is associated with the development of carotid atherosclerosis (Lear, S.A., *et al.* 2007) [21].

2.6 Cancer

Obesity is associated with low-level chronic inflammation. In this environment, macrophages are triggered and these macrophages are able to enter tumors. When the macrophages enter tumors, they produce cytokines, prostaglandins and angiogenic factors, which in turn aid tumor growth Plasminogen activator inhibitor-1 (PAI-1) is a protein that is made in visceral white adipose tissue. Increased PAI-1 levels can lead to the formation of new blood vessels, which can promote tumor cell growth and cause the tumor cells to

metastasize (Hursting, S.D. and Dunlap, S.M. 2012) [12].

Table 1: Interpreting your waist circumference

	Men	Women
Low risk	37 inches and below	31.5 inches and below
Intermediate risk	37.1 - 39.9 inches	31.6 - 34.9 inches
High risk	40 inches and above	35 inches and above

3. Diet and obesity

The currently prevalent belief is that the immediate cause of obesity is net energy imbalance—the organism consumes more usable calories than it expends, wastes, or discards through elimination. Some studies indicate that visceral adiposity, together with lipid dysregulation and decreased insulin sensitivity, (Stanhope K.L. and Havel P.J. 2010) [33] is related to the excessive consumption of fructose (Elliote S.S., *et al.* 2002, Perez-Pozo S.E., *et al.* 2010 and Choi M.E., 2009) [11, 28, 7]. Some evidence shows that in regards to juveniles, when free fructose is present as children's fat cells mature, it makes more of these cells mature into fat cells in the abdominal region. It also caused both visceral fat and subcutaneous fat to be less sensitive to insulin. These effects were not attenuated when compared to similar glucose consumption.

Intake of trans-fat from industrial oils has been associated with increased abdominal obesity in men (Koh-Banerjee P, *et al.* 2003) [19] and increased weight and waist circumference in women (Bendsen N.T., *et al.* 2011) [4] These associations were not attenuated when fat intake and calorie intake was accounted for (Micha R, and Mozaffarian D., 2008, Kavanagh K, *et al.* 2007) [24, 20] Greater meat *(processed meat, red meat, & poultry)* consumption has also been positively associated with greater weight gain, and specifically abdominal obesity, even when accounting for calories (Vergnaud A.C, *et al.* 2010 August and Vergnaud A.C, *et al.* 2010 December) [36, 37] Conversely, studies suggest that oily fish consumption is negatively associated with total body fat and abdominal fat distribution even when body mass remains constant (Hosseini-Esfahani F, *et al.* 2017 and Noreen E.E, *et al.* 2010) [16, 26]. Similarly, increased soy protein consumption is correlated with lower amounts of abdominal fat in postmenopausal women even when calorie consumption is controlled. Numerous large studies have demonstrated that eating ultra-processed food (Monteiro, *et al.* 2018) [25] has a positive dose-dependent relationship with both abdominal obesity and general obesity in both men and women. Consuming a diet rich in unprocessed food and minimally processed food is linked with lower obesity risk, lower waist circumference and less chronic disease.

Obesity plays an important role in the impairment of lipid and carbohydrate metabolism shown in high-carbohydrate diets (Ibrahim I.A., *et al.* 2011) [17]. It has also been shown that quality protein intake during a 24-hour period and the number of times the essential amino acid threshold of approximately 10 g (Cuthbertson D, *et al.* 2005) [6] has been achieved is inversely related to the percentage of central abdominal fat. Quality protein uptake is defined as the ratio of essential amino acids to daily dietary protein (Loenneke J.P *et al.* 2012) [23].

Visceral fat cells will release their metabolic by-products in the portal circulation, where the blood leads straight to the liver. Thus, the excess of triglycerides and fatty acids created by the visceral fat cells will go into the liver and accumulate there. In the liver, most of it will be stored as fat. This concept is known as 'lipotoxicity' (Harvard College, 2006) [27].

4. Conclusion

The increase in abdominal fat is associated with the risk of developing cardiovascular disease, type 2 diabetes mellitus, hypertension, stroke and certain cancers. Abdominal fat contains both visceral and subcutaneous adipose tissue, but visceral adipose tissue poses more of a health threat. It has been found that exercise; diet, prescribed drugs and surgery are viable options which can effectively reduce abdominal fat. Although maintaining a healthy weight should continue to be a cornerstone in the prevention of chronic diseases and premature death, it is equally importantly to maintain a healthy waist size and prevent abdominal obesity.

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