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Obesity and physical activity

Adriana Albu[1,2], Cristina Bidian[1,3], Simona Tache[1,3], Nikolaos Mavritzakis[4]
[1] *"Iuliu Hatieganu" University of Medicine and Pharmacy, Cluj-Napoca, Romania*
[2] *2nd Dept. of Internal Medicine*
[3] *Dept. of Functional Sciences, Physiology, Cluj-Napoca, Romania*
[4] *"1 Decembrie 1918" University, Dept. of Physical Education and Sports, Alba-Iulia, Romania*

Abstract
Obesity is an important health problem all over the world, leading to increased morbidity and mortality. Obesity related cardiometabolic complications are particularly associated with visceral fat deposition which induces systemic inflammation, insulin resistance and diabetes mellitus. Visceral adiposity increases the risk of hypertension, coronary artery disease and stroke. In athletes, increased body mass index does not reflect precisely fat body content because of increased muscular mass. Therefore, methods that estimate body composition are recommended to estimate nutritional status. Usually, a rigorous body weight control in athletes is required. Abnormally high body fat composition in athletes can lead to pathological complications.
Keywords: obesity, visceral fat, physical activity, body fat content.

Introduction

Overweight and obesity are two conditions characterized by an increase in fat mass accumulation. The prevalence of obesity is increasing across the world, with approximately 50% of adults expected to be obese by 2030 (***, 2014). Obesity is the result of a positive energy balance which is influenced by environmental and genetic factors. Excess obesity morbidity and mortality are related to many important obesity associated comorbidities, including hypertension, atherosclerosis with its coronary and cerebral complications, type 2 diabetes, obstructive sleep apnea, gastroesophageal reflux disease, fatty liver disease, cholelithiasis, osteoarthritis, polycystic ovary syndrome and various types of cancer (gallbladder, esophagus, kidney, uterus, colon and breast) (***, 2002). Physical exercise is an essential means to prevent and to reduce body weight.

Obesity in athletes requires a complex evaluation because the increase in muscle mass can modify classical parameters such as the body mass index (BMI). The aim of the narrative review is to emphasize the cardiometabolic complications of obesity, the therapeutic role of physical exercise, and to present some particularities of nutritional status in athletes.

Obesity and cardiometabolic related complications

BMI is the most widely used parameter to estimate clinical obesity and overweight, and is calculated as the ratio between body weight and squared height. According to the values of BMI, nutrition status is classified as obesity for BMI=30-39.9 kg/m², extreme or morbid obesity for values equal or superior to 40 kg/m². Normal weight is defined as a BMI of 18.5-24.9, while overweight is classified as BMI values of 25-29.9 kg/m² (Lopez et al., 2006). BMI is considered a surrogate marker for total body fat. It has been shown that obesity complications are related not only to the body fat content, but also to adipose tissue distribution. Central obesity, which reflects visceral fat deposits, is a more important risk factor for cardiovascular diseases than subcutaneous fat. Central obesity can be estimated by the waist circumference and waist to hip ratio (WHR). The cut points for waist circumference ≥94 cm in men and ≥80 cm in women indicate abdominal obesity, as stated by the World Health Organization/International Diabetes Foundation (Millen et al., 2014). When BMI was used as a measure of obesity, the association with cardiovascular morbidity was modest (Guarner & Rubio-Ruiz, 2015). In contrast, when using waist circumference or waist to hip ratio as a measure of abdominal adiposity, the association with cardiovascular and metabolic syndrome was strong (Czernichow et al., 2011; Vakil et al., 2012).

Visceral fat or ectopic fat may surround the heart and arteries or infiltrate organs such as the liver, muscles and kidneys. Fat surrounding the heart is located between the internal border of the mediastinum and the external surface of the parietal pericardium (pericardial fat), or within the

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Address for correspondence: Dept. of Functional Sciences, Physiology, "Iuliu Hatieganu" University of Medicine and Pharmacy, 1-3 Clinicilor Street, 400006, Cluj-Napoca, Romania
E-mail: cristina_bidian@yahoo.com
Corresponding author: Cristina Bidian; cristina_bidian@yahoo.com
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pericardial sac (epicardial fat) or the visceral epicardium, surrounding the coronary arteries (pericoronary fat). This ectopic fat tissue produces inflammatory cytokines which induce vessel wall inflammation and may stimulate the development of atherosclerosis (Yudkin et al., 2005). This fat content may be measured using echocardiography and more precisely using multidetector computed tomography or magnetic resonance imaging which quantifies adipose tissue volume (van der Kooy & Seidell, 1993; Lim, 2014).

Perivascular fat is a source of cytokines that stimulate inflammation inducing endothelial dysfunction and atherosclerosis (Yudkin et al., 2005). Perivascular fat can be measured using computed tomography examination and magnetic resonance imaging methods (Lim, 2014).

In an animal model, increased renal sinus fat was associated with increased blood pressure, probably by compression of blood vessels (Chughtai et al., 2010), while intrarenal fat was associated with inflammation, oxidative stress and renal fibrosis (Reisin & Jack, 2009). Renal sinus fat can be measured by computed tomography scan. Perirenal fat can be explored ultrasonographically (Lim, 2014).

Ectopic fat accumulation in the liver stimulates the production of inflammatory cytokines, increases insulin resistance and stimulates VLDL hepatic synthesis (Lo et al., 2011). Diagnosis of fatty liver disease can be made using ultrasonography, computed tomography or H-magnetic resonance spectroscopy (Lim, 2014).

Muscle fat infiltration is considered an important cause of insulin resistance. The alteration of fatty acid oxidation leads to diminished use of fatty acids and increased lipid storage within skeletal muscle. Fat muscle deposition can be measured using dual-energy X-ray absorptiometry or computed tomography scan (Lim, 2014).

In visceral adiposity, adipocytes are hypertrophied and dysfunctional. Abdominal obesity has been linked to insulin resistance and systemic inflammation which contribute to the development of type 2 diabetes mellitus, hyperlipidemia and cardiovascular disease (Marsland et al., 2010).

The association of obesity with impaired glucose tolerance and with diabetes mellitus has been shown in large studies in both women and men (Frank et al., 2001; Chan et al., 1994). The precise mechanism that contributes to this association is not known, but increased insulin resistance in obesity seems to play an essential role (Paley & Johnson, 2018). Adipokines secreted by adipose tissue have an important role in obesity related cardiometabolic complications. Adiponectin, which increases insulin sensitivity and protects the cardiovascular system, is reduced in obesity. Leptin regulates food intake and energy expenditure and is also involved in muscle fatty acid metabolism and hepatic glucose synthesis. Obesity is characterized by increased leptin levels and leptin resistance. Interleukin 6 and tumor necrosis factor-α are inflammatory cytokines involved in the relation between systemic inflammation, insulin resistance and obesity (Lechleitner, 2008).

Obesity is also strongly related to arterial hypertension. The relative risk of hypertension in overweight men and women was 1.46 and 1.75, respectively, after adjusting for age, in the Framingham study (Wilson et al., 2002).

Insulin resistance is an essential mechanism in the development of metabolic syndrome. The inadequately high energy intake causes overloading of adipose tissue, and lipids that cannot be stored in adipocytes accumulate in ectopic sites, such as the liver, skeletal muscle and pancreas, inducing organ functional impairment. Obesity is associated with reduced high-density lipoprotein and increased triglycerides. Insulin resistance impairs lipoprotein lipase, inducing alteration of lipid metabolism (Reisin & Jack, 2009).

Coronary heart disease has been reported to be more frequent in obese subjects (Willett et al., 1995). Chronic heart failure was 2 times more frequent among obese compared to non-obese patients (Kenchaiah et al., 2002). However, several studies have found better outcomes in overweight and obese patients with coronary heart disease and chronic heart failure compared to normal weight subjects. This phenomenon has been described as "an obesity paradox" (Segula, 2014; Hainer & Aldhoon-Hainerova, 2013). The obesity paradox mechanism is not completely understood, but it is supposed that these patients are elderly, and their obesity is not accompanied by the metabolic changes that characterize abdominal obesity (Hainer & Aldhoon-Hainerova, 2013).

An increased risk of ischemic stroke has been associated with obesity. Moreover, in central obesity, trunk adipose tissue distribution was associated with stroke mortality (Segula, 2014; Tanne et al., 2005).

In contrast, there are also obese subjects who are metabolically healthy (about a quarter of the obese adult population), with insulin-sensitive obesity, and do not develop systemic inflammation. On the other hand, unhealthy lean subjects develop metabolic and vascular complications even though they have a normal BMI. In these cases, ectopic visceral adipose tissue, particularly perivascular and pericardial fat, might be the cause of altered metabolic status (Denis & Hamilton, 2013).

Effects of exercise on body weight and metabolic alterations in obesity

Obesity and metabolic syndrome have been associated with sedentary behavior (Telford, 2007; Lakka et al., 2003). The risk for developing metabolic syndrome was 65% in men with a moderate level and 76% in those with a high level of sedentary behavior, compared to men who were active (Greer et al., 2015). Active people and athletes are less obese than sedentary people.

The combination of exercise and diet offers a method for weight control. Research indicates that exercise may increase lipid mobilization from the adipose tissue and fat utilization, thus increasing fat weight loss and, at the same time, preventing the reduction of lean tissue (Kenney et al., 2015). There are three essential ways that contribute to weight loss: reducing caloric intake below daily energy expenditure, maintaining regular food intake, and increasing energy expenditure by performing additional physical activity, and using combined methods which induce a negative energy balance by decreasing food intake and increasing energy expenditure (Westerterp, 2010).

Physical exercise has been shown to improve various markers of metabolic syndrome. In a large longitudinal study which included 4840 adults, overweight and obese individuals who increased the physical activity level improved their BMI and cardiometabolic profile with a decrease of LDL cholesterol from baseline to follow-up (536 days) (Ritti-Dias et al., 2017). In another prospective study which included 22,383 participants aged 30-64 years, without metabolic syndrome, who were followed up for a mean of 4.1 years, leisure-time activity was found to be linearly and inversely correlated with the risk of developing metabolic intensity activity associated with a lower risk of metabolic syndrome (Kuwahara et al., 2016). Studies that investigated the effects of physical training on various components of metabolic syndrome found an improved risk profile with exercise (Ostman et al., 2017; Lin et al., 2014).

The effects of exercise on abdominal obesity even in the absence of weight loss have also been studied. It has been reported that moderate intensity exercise significantly reduces total and abdominal fat, skeletal muscle lipid content, even in the absence of weight loss (Paley & Johnson, 2018; Lee et al., 2005).

At least 200 to 300 minutes of moderate to vigorous physical activity each week have been recommended to encourage long-term weight loss (Lee et al, 2013; Goldberg et al., 2009). In order to prevent weight regain and increase weight loss, 150 to 300 minutes of moderate physical activity per week or 75 to 150 minutes of vigorous physical activity weekly are recommended (Lee et al., 2013).

The mechanisms involved in the effects of exercise on obesity are not completely understood. The main theories emphasize the decline in inflammatory cytokine levels (such as interleukin-1, tumor necrosis factor-α and monocyte chemoattractant protein 1), oxidative stress, and the increase in adiponectin, as favorable effects of exercise training (Sakurai et al, 2013; Werner et al, 2013).

Body weight and fat content in athletes

Body weight in athletes depends on the type of sport. Certain sports impose a rigorous control of weight. Aesthetic sports such as rhythmic and artistic gymnastics, ice skating, or endurance sports such as long-distance running are sports which require low body weight for high quality sport activity (Ritti-Dias et al., 2017). In other sports, including wrestlers, boxers, judokas, mixed martial artists, power lifters, and weightlifters, weight classes are defined for sports competitions. Athletes usually improve muscle strength without augmenting their BMI because participating in a superior body weight class may be more difficult (Dragan, 2002; ***, 2017).

BMI is a surrogate marker of body fat and is less precise in the estimation of athletes' nutritional status, as they may have a higher BMI due to higher lean mass. Body fat content estimated as the ratio of body fat mass to body free-fat mass is essential for elite athletes because it may influence athletic performance. Thus, in athletes, methods that estimate body composition are recommended (***, 2017; Popadić Gaćeša et al., 2011).

It has been found that a body fat percentage of 7% to 9% is metabolically efficient and healthy for most males, while for females, the adequate body fat percentage is 12% to 15% (Perriello, 2001).

One example of increased BMI which does not reflect exactly the amount of fat is that of sumo wrestlers. Sumo is the national sport of Japan and has a 2000-year history. Sumo athletes have an intensive exercise regimen and consume a very high calorie diet daily (more than 5,000 cal). The frequency of meal intake is twice daily (Yamauchi et al., 2004). In one study that evaluated the BMI and fat content in sumo wrestlers compared to controls, all wrestlers were obese according to their BMI but only 40% were so when their body fat was taken into consideration. This suggests that both the BMI and fat percentage can be considered when assessing obesity in excessively muscular people.

An abnormally high body fat composition in athletes can lead to pathological complications including high blood pressure, hyperlipidemia, heart disease, diabetes, and gallbladder disease (Perriello, 2001).

In a study that included 96 wrestlers with a mean body weight of 100.4 kg, the mean serum levels of triglycerides, phospholipids, uric acid, and total protein were significantly higher in wrestlers than in age-matched healthy males. The incidence of diabetes mellitus, gout, and hypertension was considerably higher in athletes than in controls. A high calorie diet and infrequent meal intake were considered as possible causes of obesity, hyperlipidemia, and hyperuricemia in wrestlers (Nishizawa et al., 1976).

Conclusions

1. Obesity, particularly its visceral type, is associated with an increased risk of cardiometabolic diseases. Physical exercise can reduce body weight and the risk of obesity complications.
2. In athletes, obesity must be estimated not only by the measurement of BMI, but also by the body fat content.
3. The increase of fat mass reduces athletes' performance and increases the risk of cardiac and metabolic diseases.

Conflicts of interests

The authors have no conflicts of interests.

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