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# Epidemiology of Abdominal Obesity

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Maria Teresa Anselmo Olinto,  
Heloísa Theodoro and Raquel Canuto

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## Abstract

Abdominal obesity (AO) is associated with endothelial dysfunction, inflammation, insulin resistance, diabetes mellitus, hypercholesterolemia, metabolic syndrome, and cancer. AO is a multifactorial disorder arising from genetic, environmental, socioeconomic, and behavioral factors. Thus, in this chapter, we devote ourselves to the exercise of trying to explain the epidemiology of AO in adults. We showed the increasing prevalence of AO around the world, and a gender difference in this determination was observed. Among women, the population group who is the most affected by AO, a higher prevalence of AO is observed in individuals living in low- or middle-income countries (LMIC), who are older, multiparous, and in the menopausal transition, and who belong to the poorest strata and have lower educational level. While among men, the risk of AO is positively associated with socioeconomic status, particularly in LMIC. Regarding behavioral factors (eating frequency, sleep duration, physical activity, and smoking), gender differences are difficult to be detected due to the lack of studies investigating their association with AO according to sex. However, the current evidence suggests that men benefit more from consuming a greater number of meals a day and women are more affected by the harmful effects of physical inactivity. We argued AO, despite biological conditions associated with behavior factors, should be examined as an important issue of gender inequality in health, possibly mediated by socioeconomic and behavioral differences between men and women.

**Keywords:** abdominal obesity, waist circumference, epidemiology, gender, income

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## 1. Introduction

Obesity is a worldwide epidemic. Beyond the fat mass per se, the pattern of fat distribution has a profound influence on cardiometabolic risk. Visceral abdominal fat (VAF) is metabolically

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active and pro-inflammatory and presents a higher cardiometabolic risk association and calcification of the coronary arteries than the body mass index (BMI) and has more impact on health than subcutaneous fat, presenting a risk factor for increased incidence of metabolic syndrome [1, 2].

Abdominal obesity (AO) is directly associated with increased VAF, and it is also associated with endothelial dysfunction, inflammation, insulin resistance, diabetes mellitus, hypercholesterolemia, metabolic syndrome [MetS], and cancer [1, 3].

There are several methods available to measure AO. Waist circumference (WC) provides an indicator of central adiposity that is the most practical and easiest method used in large-scale epidemiological studies [4]. It is a good predictor of cardiometabolic morbidity and mortality, and it has also a positive association with visceral abdominal fat. However, WC does not allow us to differentiate between visceral fat and subcutaneous fat; methods such as absorptiometry by dual energy X-ray (DEXA), impedance, or densitometry can be used to handle this differentiation [5–7].

WC measurement requires correct and standardized procedures, which depend mainly on training and adequate equipment. A standardized technique requires that the person being measured removes bulky or tight garments, as well as shoes with heels, empties their bladder then stands in the upright position, with arms loosely positioned to the side. The tape is passed around the body and positioned mid-way between the iliac crest and costal margin of the lower rib, ensuring that it

Population	Organization (References)	Recommended waist circumference threshold for abdominal obesity	
		Men	Women
Europid	IDF	≥94 cm	≥80 cm
Caucasian	WHO	≥94 cm [increased risk] ≥102 cm [still higher risk]	≥80 cm [increased risk] ≥88 cm [still higher risk]
United States	AHA/NHLBI [ATP III]	≥102 cm	≥88 cm
Canada	Health Canada	≥102 cm	≥88 cm
European	European cardiovascular societies	≥102 cm	≥88 cm
Asian [including Japanese]	IDF	≥90 cm	≥80 cm
Asian	WHO	≥90 cm	≥80 cm
Japanese	Japanese obesity society	≥85 cm	≥90 cm
China	Cooperative task force	≥85 cm	≥80 cm
Middle East, Mediterranean	IDF	≥94 cm	≥80 cm
Sub-Saharan African	IDF	≥94 cm	≥80 cm
Ethnic Central and South American	IDF	≥90 cm	≥80 cm

**Table 1.** Waist circumference cutoffs recommended for the diagnosis of abdominal obesity according to ethnicity and gender [9].

is horizontally oriented and untwisted. The subject is asked to look ahead and breathe out; the measurement is taken at the end of expiration; then, the procedure is repeated [7, 8].

In addition, the definition of cutoffs should consider the characteristics of the study population. In 2009, a method to standardize the diagnosis of metabolic syndrome was established, upon discussions held by the International Diabetes Federation (IDF) and the American Heart Association/National Heart, Lung, and Blood Institute. In this context, it was suggested that ethnicity and gender should be considered for the diagnosis of AO [9] (**Table 1**).

AO is a multifactorial disorder arising from genetic, environmental, socioeconomic, and behavioral factors. These factors differ in their respective contributions to the AO epidemic. In this chapter, we devote ourselves to the exercise of trying to explain the epidemiology of AO. First, we describe the worldwide prevalence of AO. Then, the possible biological and socioeconomic factors that are associated with AO are demonstrated, according to the sex/gender differences. Finally, we describe the role of important behavioral factors determining AO.

## 2. Prevalence of abdominal obesity

Populations worldwide have faced a growing “epidemics” of AO. Overweight and obesity across low- and middle-income countries (LMIC) have reached levels found in higher-income countries (HIC). Despite its high prevalence, there are differences among regions and countries, and these need to be taken into account for us to understand the etiology of AO.

To better elucidate this picture, **Table 2** depicts the prevalence of AO according to gender in selected high and LMIC. LMIC showed the highest prevalence of AO, compared with HIC; in several studies, an increasing trend of AO in the past 10 years [10, 16, 17] has been observed. For example, in the study called China Health and Nutrition Survey (1993–2009), with 52,621 Chinese adults, the prevalence of AO increased from 8.5 to 27.8% among men and from 27.8 to 45.9% among women [17]. Similarly, in the USA, data from the National Health and Nutrition Examination Survey [NHANES] from 1999 to 2010 identified an increase over time, and the difference between genders was 20% higher for females [10].

Regarding gender, both in LMIC and in HIC, it was observed that women had a higher prevalence of AO than men. Also, recent studies show a higher prevalence of AO in women than men, in all ages. There is a proportional increase in the accumulation of fat in the abdominal region as people get older, but a stabilization or even a small decrease in the prevalence of AO in men after 60 years of age could be perceived [12, 18, 20]. In Brazil, in a study involving data from 3117 subjects, the prevalence of AO was found to be 26 and 73%, respectively, in women aged 24–34 years and 55–65 years. In men, the prevalence of AO was found to be 16.9% (24–34 years) and 27.2% (55–65 years) [20].

But why do women have a higher prevalence of AO than men? On one hand, sex hormones strongly influence body fat distribution and adipocyte differentiation between females and males, showing a physiological difference in the AO determination between sexes. However, in part AO is due to a social construction, since socioeconomic, cultural, and behavioral characteristics play an important role in its causal chain.

Author	Country (year)	Prevalence (%)	Men (%)	Women (%)
High-income countries				
Beltrán-Sánchez et al. [10]	USA (2009–2010) <sup>1</sup>	56.0	46.4	65.4
Gutiérrez-Fisac et al. [11]	Spain (2008–2010) <sup>2</sup>	36.0	32.0	39.0
Riediger and Clara [12]	Canada (2007–2009) <sup>1</sup>	35.0	29.1	40.0
Schienkiewitz et al. [13]	Germany (1997–1999) <sup>1</sup>	33.9	29.7	38.0
Sardinha et al. [14]	Portugal (2008–2009) <sup>2</sup>	–	19.3	37.9
Low-/middle-income countries				
Barquera et al. [15]	Mexico (2010–2012) <sup>3</sup>	74.0	64.5	82.8
Misra and Shivastava [16]	South Asians (2011–2012) <sup>4</sup>	68.9	17.6–62.2	23.7–74.8
Xi et al. [17]	China (2009) <sup>3</sup>	37.4	27.8	45.9
Linhares et al. [18]	Brazil (2010) <sup>1</sup>	30.0	19.5	37.5
Chukwuonye et al. [19]	Nigeria (2013) <sup>1</sup>	21.8	3.2	39.2

Cutoffs of WC for AO = <sup>1</sup>≥102 cm (males) and ≥88 cm (females); <sup>2</sup>>102 cm (males) and >88 cm (females); <sup>3</sup>>90 cm (males) and >80 cm (females); <sup>4</sup>≥90 cm (males) and ≥80 cm (females).

**Table 2.** Prevalence of AO according to gender in selected high- and low-middle-income countries.

To better understand this topic, it is important to elucidate the differences between sex (biology) and gender (the social). According to Annandale and Hunt [21], this distinction was essential to make it clear that gender inequalities in health were, in the most part, socially produced, rather than biologically given. Olinto [22] also called attention to the operationalization of the gender category in epidemiological studies. Sex only means the genetic, anatomical, and physiological characterization of human beings. However, gender roles are socially constructed and usually framed as an extension of biologically determined social functions. The pioneering feminist Simone de Beauvoir said in her famous quote: “One is not born, but rather becomes, a woman.” Though the positions of women and men are not simply parallel, the principle is also true for men: One is not born masculine, but has to become a man [23]. Thus, gender is related to differences in patterns of employment, education, family, and household structure, leisure and consumption at the societal level, and in the everyday experience of individual men and women [21]. In this sense, we describe the possible biological and social factors associated with AO according to gender differences.

### 3. Physiological differences between the sexes and the characteristics of reproductive life in women

Among the physiological factors related to sex differences, there is a hormonal issue: Sex hormones strongly influence body fat distribution and adipocyte differentiation. Estrogens and testosterone differentially affect the physiology of adipocytes. Visceral fat is higher in men than in premenopausal women. In men, visceral fat accrual generally increases with the

amount of total body fat, whereas in women, visceral fat accumulation is less a function of total adiposity. Women had less visceral fat despite having a higher total body fat, BMI, and subcutaneous fat [24–27].

As women reach menopause, depot differences in adipocyte size are attenuated due to the express increase in omental cell size. The propensity of postmenopausal women toward visceral fat accumulation and presence of larger adipocytes suggests that the decline of estrogen may stimulate adipocyte hypertrophy in this depot. In men, adipocytes of the visceral and abdominal subcutaneous fat compartments have similar sizes across the range of adiposity values [24, 28].

In men, a meta-analysis found that those with low concentrations of total testosterone (TT), sex hormone-binding globulin (SHBG), and free testosterone (FT) were more likely to develop metabolic syndrome compared to those having high sex hormone concentrations. The revealed associations were independent of age and lifestyle factors. Associations with TT were strongest for prevalent AO [OR (odds ratio) per quartile decrease = 1.58] (95% CI 1.51–1.66). Low FT concentrations were associated with incident AO [HR (hazard ratio) = 1.13] (95% CI 0.98–1.29), although the latter was not statistically significant [29].

Studies show that the prevention of AO should be focused on lifestyle. However, there are different hormonal factors between the sexes that must be considered, especially for women, whose menopausal transition goes through hormonal changes that favor the redistribution of body fat, being more susceptible to abdominal fat accumulation. Moreover, the life expectancy of women generally exceeds that of men, with a significant increase in the number of women experiencing the menopausal transition phase, which makes this an increasingly relevant issue in terms of public health.

Menarche and menopause set the beginning and the end of women's reproductive life and are important risk factors for chronic diseases, including obesity and cardiovascular disease. Early menarche, before the age of 12, has been associated with a higher prevalence of AO [30–33]. Both early menarche and early menopause can be considered as increased risk factors for cardiovascular disease.

The association between early menarche and obesity is still controversial in the literature. However, some longitudinal studies demonstrated that childhood obesity is the trigger to early pubertal development because of increased exposure to reproductive steroids [estradiol level] [30, 32, 34–36]. Furthermore, many genetic variants associated with the timing of menarche are in or near genes associated with childhood and adulthood obesity, so this fact should be considered [32].

Reproductive life characteristics are directly associated with AO. For example, a cross-sectional study with a sample of 617 women from southern Brazil observed that women with a history of three or more pregnancies and menarche at the age of 11 or earlier had a 25% higher prevalence of AO compared to nulliparous or primiparous women with menarche at 14 years or older [31]. Studies show that the number of parturitions leads to a tendency of decreased hip circumference and increased WC [37–39].

Pregnancy was associated with visceral adiposity gains and central obesity in the study with 122 premenopausal women monitored for 5 years. Throughout the monitoring period,

nulliparous women had a 14% increase in the visceral adipose tissue, while those with at least one parturition increased by 40%. Parturition was associated with increased WC. It is suggested that after pregnancy, there is a preference for fat accumulation in the visceral adipose tissue [40]. Similarly, in a sample of 170 American women aged 18–76 years, it has been found that the intra-abdominal fat tissue increased as the number of parturitions increased, regardless of age, body fat percentage, physical activity, and smoking [41].

Changes in the distribution of body fat and AO as a consequence of pregnancy require further investigation. As a modifiable risk factor, weight gain during the pre- and postpartum periods can provide a critical window for performing interventions to prevent substantial weight gain and the development of obesity in women [42].

Epidemiological studies are consistent in the sense that characteristics of reproductive life may have a strong influence on body fat buildup in women during the menopausal transition. The transition from the reproductive phase to the nonreproductive phase is characterized by endocrine changes due to the decline of ovarian activity, biological changes due to decreased fertility, and consequent clinical changes due to menstrual cycle changes, as well as a variety of symptoms [43].

The change in metabolism that accompanies menopausal transition occurs at the expense of a reduction in lipoprotein lipase, responsible, along with estrogen, for regulating fat accumulation and distribution in tissues [44]. Testosterone seems to be a factor influencing the accumulation of visceral adipose tissue, and it seems to be related to the state of hyperandrogenism in women. The accumulation of visceral fat was higher in women after menopause compared to premenopausal women [45].

Several studies have shown a high prevalence of AO in postmenopausal women, ranging from 50 to 85% [31, 46–49]. A systematic review by Mendes et al. [49] found a high prevalence of AO in the menopausal transition, mainly in studies performed in clinics. Among these, the highest prevalence rates were reported in a study based on the Northeast region of Brazil, with 76.6% of premenopausal women and 85.2% of postmenopausal women. The lowest prevalence was found among Asian women, who had prevalence of 16.4% among premenopausal women and of 29.1% among postmenopausal women, suggesting that ethnicity is an outlier factor as far as the accumulation of fat in the abdominal region is concerned, once Western women have a higher prevalence than Eastern women.

The relationship involving women's experience in their reproductive period, their individual characteristics, such as age of menarche and number of parturition, hormonal changes during the menopausal transition, and the accumulation of fat in the abdominal region deserves more attention from health professionals.

#### **4. Socioeconomic status and gender**

Gender, as a social construct, plays an important role in the association between socioeconomic characteristics and AO. Socioeconomic status (SES) is a complex and multidimensional construct,

in which individuals are classified by being compared to other individuals, based on material and nonmaterial attributes [50]. SES influences the individual access to goods and services regarding nutrition, physical activity, and other healthy practices and environmental conditions, which influence the relationship between socioeconomic position and AO.

Studies have used different SES indicators; nonetheless, we focus primarily on individual characteristics, such as income, education and occupation, and economic development of countries, because they may have greater importance regarding interpretations of linkages between SES and AO which emphasize behavior.

Classic studies showed the direction of association between SES and obesity varied by population and economic status of countries. In developed countries, individuals with lower socioeconomic status were more likely to be obese than those in the higher socioeconomic group. However, in some LMIC the prevalence of obesity has increased among low SES groups, mainly among women. However, Is the association between SES and obesity similar to the association between SES and AO?

Most of these global analyses did not evaluate AO as outcome. This paper aims to show findings on the association between SES and WC in HIC and LMIC. In HIC, a recent study analyzed 50 years of socioeconomic inequities in WC among US-born black and white Americans, using data from the National Health Examination Surveys (NHES) I-III (1959–1970), National Health and Nutrition Examination Surveys (NHANES) I-III [1971–1994], and NHANES 1999–2008. WC increased in socioeconomic strata among both black and white Americans. Regarding income, white people in the 20th [low] income percentile have greater mean WC compared with people in the 80th [high] income percentile [51]. In the same direction, a cross-sectional study carried out with 12,883 individuals representing the Spanish population found that the frequency of obesity and AO decreased as the educational level increased [11]. In addition, a prospective study in the United Kingdom with 8312 subjects and three follow-ups over ten years showed that a lower adult occupational position predicted adverse changes in WC [52]. Finally, 56,556 participants from seven population-based German cohort studies (CARLA, SHIP, KORA, DEGS, EPIC-Heidelberg, EPIC-Potsdam, PopGen) were analyzed by meta-analysis. Men and women in the low education group had a 0.1% point greater annual increase in WC than participants in the high education group. Women with low income had a 0.1% point higher annual increase in WC than women with high income [53].

On the other hand, in LMIC, associations of risk factors with AO differ between men and women. The Thai National Health Examination Survey investigated this association in 64,480 adults. Compared with primary education, the odds of obesity [range I] were higher in men with university education. For women, the association was inverse, the odds of obesity ranges I and II were higher in those with primary education [54]. In Northeast China, a representative sample of 25,196 adults was evaluated. Analysis stratified by gender showed that men with a higher educational level, white-collar job, or cadre job were part of the high risk group, and women with a higher level of education or higher family income were in the low risk group [55]. Finally, in Brazil, a cross-sectional study with 1,720 Brazilian adults found results that point to the same direction. The WC was 4.67 cm higher in women who live in low education

neighborhoods compared to the residents of high education areas. In the same group, the chance of AO was 2.05 times higher [56].

Thus, the findings suggest that the association between SES and AO is similar to the association with overall obesity. Separate studies of individual nations showed that high status people tend to have a smaller WC than others, in HIC, and this association is the same for women, in LMIC. On the other hand, among men from LMIC, the association between SES and AO is positive. First, this reversal in the relationship of SES and AO across levels of economic development highlights the importance of the national socioeconomic context of AO. Second, the high prevalence of AO among low SES women, in LMIC, shows an important health inequality. In some LMIC countries, low SES groups may now have sufficient access to cheap, calorie-dense, and processed food, as a consequence of the globalization of the fast food industry and agriculture.

## 5. Behavioral characteristics

We explored the role of eating frequency, sleep duration, physical activity, and smoking on AO. We also draw a parallel between gender differences and these associations.

### 5.1. Eating frequency

Since the 1960s, studies have suggested an inverse association between the consumption of a greater number of meals per day and body weight maintenance [57]. Since then, the media, health professionals, and guidelines for health and weight management have followed this recommendation [58]. However, studies that have attempted to determine the effects of eating frequency on weight and body composition have reached different conclusions, and some scientists have called attention to the lack of solid evidence to justify such recommendation.

Thus, several review studies have been recently conducted in order to determine whether there is association between eating frequency and body weight and body composition [59–62]. However, a few studies that investigated AO or WC or waist-to-hip ratio as outcome were retrieved in these reviews [63].

A negative association between eating frequency was found in several observational studies [64–68]. Most of them found that having three or fewer meals/day is associated with excessive abdominal fat, when compared with having more than three meals/day; however, this association can only be seen among men [64–67]. In general, increased eating frequency has been postulated to increase metabolism [69], appetite control, and food intake and to improve glucose and insulin control [70–72]. However, this association only among men could be due to the fact that men who have a high eating frequency also have a healthier lifestyle, including the practice of physical activity and healthier eating habits, which results in reduced body fat and WC [73].

On the other hand, a recent cross-sectional study used data from the NHANES 2003–2012 and found that eating frequency was positively associated with central obesity in men and women.

Compared with the lowest eating frequency ( $\leq 3$  times/d), the odds for AO in the highest category ( $\geq 5$  times/d) were OR 1.42 (95% CI 1.15;1.75) in men and OR 1.29 (95% CI 1.05;1.59) in women [74]. This finding seems plausible, given the observed positive association between eating frequency and energy intake [67, 75, 76]. In addition, the authors called attention to the apparent inverse relation between eating frequency and adiposity measures, such as an artifact that in part can be attributed to the underreporting of eating frequency concomitant with the underreporting of energy intake by obese or overweight subjects [74].

Regarding experimental evidence, a meta-analysis study published in 2015 evaluated experimental research on meal frequency with respect to changes in fat mass and lean mass, and its results suggest a small potential benefit of increased feeding frequencies for fat mass and body fat percentage. In studies retrieved in this meta-analysis, only the Arciero's study measured the outcome as abdominal fat. This randomized trial investigated the effects of consuming traditional (15% of total energy) versus higher [35% of total energy] protein intakes as three or six meals/day on abdominal fat. Six meals per day in a high-protein condition were superior to three meals per day with a high-protein or traditional protein intake [15%] for decreasing abdominal fat in the absence of significant differences in total energy intake or expenditure. However, the study design did not rule out the possibility of an interaction between the two [eating frequency and protein intake] playing a role in mediating these responses [63].

Although observational studies suggest that men may benefit from a potential protective effect of a high eating frequency, women may not experience this protective effect. However, there is no sufficient evidence to establish a clear and strong association between eating frequency and AO, and it is necessary to conduct more observational and experimental studies.

## 5.2. Sleep duration

Over the past several decades, the prevalence of chronic sleep deprivation has grown to epidemic proportions. According to the National Sleep Foundation, by 1998 only 38% of American adults were obtaining 8 h of sleep and that number had fallen to 28% by 2009 [77]. Evidence has grown over the past decades supporting the role of sleep duration as a notable risk factor for overall obesity [78, 79]. More recently, studies have suggested that measures of central adiposity, such as WC, are also associated [80].

Heorell-Haglöw's [80] cross-sectional study investigated the association between sleep duration and WC in 6,461 Swiss women. The study showed a U-shaped association: Both short sleeping ( $< 5$  h) and long sleeping ( $\geq 10$  h) women had greater means of WC compared to normal sleepers (7–8 h) in younger women ( $< 50$  years old). Other cross-sectional study involving men and women found a similar U-shaped association, considering short sleepers ( $< 7$  h) and long sleepers ( $> 8$  h) [81]. However, several studies found that only short sleep, and not long sleep duration, is a risk factor for central obesity [82–84].

A few longitudinal studies have investigated the association between sleep duration and AO. A prospective study with a 10-year follow-up in women confirmed the U-shaped association found in some cross-sectional studies. Among younger women (aged  $< 40$  years), both habitual short ( $< 6$  h) and long sleep durations ( $\geq 9$  h) were risk factors for central obesity. In addition,

decreased sleep duration from normal to short duration during the follow-up was a risk factor for increased WC [85]. Another prospective cohort investigated the association of short sleep duration ( $\leq 5$  h) among women in the first year postpartum with adiposity status at 3 years postpartum. The authors demonstrated that postpartum sleep of  $\leq 5$  h/day was associated with a higher WC at 3 years postpartum, compared to sleeping  $> 5$  h [86].

In order to assess the magnitude and consistency of the relation of insufficient sleep and WC, a meta-analysis of cross-sectional studies was conducted in 2014. The analyses included 21 studies (total of 56,259 participants); the results confirmed a significant negative relation between sleep duration and WC, and longer sleep duration was not investigated [86]. However, What is short sleep and long sleep? Grandner et al. summarized the laboratory findings and epidemiological studies that investigated the consequences of acute reductions in sleep time, and they proposed to describe short sleep duration as  $< 6$  h/day [87]. However, there is no robust definition of long sleep duration, although several studies have defined long sleepers as those who have a habitual sleep duration of more than 8 h.

Finally, clinical trials that measured AO as an outcome are sparse, probably because it is difficult to maintain individuals on sleep deprivation for a long period of time. In a randomized study of adults, sleep curtailment was shown to undermine dietary efforts to reduce adiposity, after 14 days of moderate caloric restriction with 8.5- or 5.5-h nighttime sleep opportunity. It reduced the fraction of weight loss by 55% and increased the loss of fat-free body mass by 60% [88]. The hormonal regulation that occurs during sleep and its multiple peripheral effects depend on sleep duration and quality, indicating that sleep deprivation has deleterious health effects. In a randomized study, acute sleep deprivation in adults has been associated with changes in thermoregulation and, consequently, reductions in total energy expenditure in humans. Sleep-deprived individuals may also increase their total caloric intake due to the impact of sleep deprivation on peripheral regulators of satiety. Studies have associated sleep deprivation with lower leptin and higher ghrelin levels, with consequent increases in appetite and weight gain [89–92].

Furthermore, reduced sleep duration is known to have consequences on individuals' social routines and lifestyles, particularly regarding dietary habits and level of physical activity. In this sense, short sleep duration is associated with irregular eating habits, such as a dominance of snacks over meals, increased intake of fat and sweets, increased energy intake and, on the other hand, reduced intake of fruits and vegetables, and lower physical activity level [93–95]. These findings can be explained in part by a disinhibited eating behavior [tendency toward overeating and eating opportunistically], which is associated with less healthy food choices, thus contributing to weight gain. Because short sleep duration inevitably results in more time and opportunities for eating, short-duration sleepers who also have a high disinhibition eating behavior trait will eat more and gain more weight than short-duration sleepers who have a low disinhibition eating behavior trait [96].

After more than three decades of studies about the metabolic consequences of changes in the patterns of sleep duration, we can conclude that there is sufficient evidence to support that short sleep duration plays an important role in the AO etiology in adults. However, the association between long sleep and AO lacks more solid evidence, and explanations about its

causal chain are weak. Nonetheless, disrupted eating patterns, such as having a dominance of snacks over meals, a higher intake of fat and sweets, and a lower intake of fruits and vegetables, are associated with both short and long sleep duration [95].

### 5.3. Physical activity

Strong evidence shows that physical inactivity increases the risk of many adverse health conditions, including coronary heart disease and type 2 diabetes mellitus, and it also shortens life expectancy [97]. The therapeutic role of physical activity in cardiovascular and metabolic disease involves multiple mechanisms, and one of these pathways appears to be via its effect on abdominal fat [98]. Physical activity has a role in the prevention, treatment, and control of AO. However, this chapter has focused on the epidemiology of AO, and thus we have decided to describe the predictor role of physical activity on the occurrence of AO.

Several prospective studies have evaluated the association between physical activity and AO. In the European Prospective Investigation into Cancer and Nutrition [EPIC] prospective cohort study, 84,511 men and 203,987 women were followed for 5.1 years. Physical activity predicted a lower WC in men and in women, regardless of baseline body weight, baseline WC, and other confounding factors [99]. A nationally representative population-based cohort study, The Australian Diabetes, Obesity and Lifestyle Study [AusDiab] found similar results. It followed 2,191 men and 2,650 women for 5 years. Men and women with AO showed the odds of reducing physical levels from baseline to follow-up for 1.40 (95% CI 1.10;1.79) and 1.44 (95% CI 1.16;1.80), respectively, compared to those with a normal WC [100].

In the same direction, long-term longitudinal studies (follow-up  $\geq 20$  years) have shown that youth physical activity had an effect on AO through the maintenance of physical activity in adulthood [101, 102]. A longitudinal study data by Finns on the cardiovascular risk in young people followed 1319 men and women for 21 years. Structural equation analysis indicated that the prevalence of AO in adulthood was directly affected by adult physical activity and indirectly via youth physical activity. Obesity was significantly reduced from youth to adulthood. The model accounted for 19% of AO in men and 13% in women [103]. Another long-term longitudinal study has showed sex differences in the association between physical activity and AO. The Coronary Artery Risk Development in Young Adults study (CARDIA), a 20-year follow-up prospective longitudinal study with 3,554 men and women, showed that maintaining high levels of activity was associated with smaller gains in WC compared with low activity levels. Men maintaining high activity gained 3.1 fewer centimeters in WC, and women maintaining higher activity gained 3.8 fewer centimeters [104].

Studies only among women showed similar results. In a prospective cohort with 233 middle-aged women, after a 20-month follow-up, changes in physical activity remained as an independent predictor of abdominal fat change after adjusting for changes in total body fat and total energy intake. Compared with women who maintained or decreased physical activity, women who increased physical activity had approximately half the risk [RR (relative risk) = 0.52 95% CI: 0.27;0.98] of gaining abdominal fat [105]. In Southern Brazil, a case-control study investigated the association between the practice of physical activities in adolescence and AO in adulthood among women who were shift workers. Women who participated in five

or more physical activities in adolescence were 50% less likely to have AO than women who participated in one activity or no physical activities (OR = 0.50 95% CI: 0.27;0.93) [106].

Finally, evidence from clinical trials can help us understand how physical activity reduces body fat, preferentially from the abdominal area. In the past decade, several systematic reviews have summarized results from randomized controlled and nonrandomized controlled trials [107–109]. They support moderate physical activity as an effective intervention for reducing abdominal fat in overweight or obese subjects. Also increasing physical activity expressed as energy expended per week was positively related to reductions in total adiposity in a dose-response manner. The gender influence presents controversial results due to low numbers for comparison among those reviews. However, the most recent review showed a stronger relationship between physical activity and visceral fat in women than in men. However, it is difficult to compare differences in the amount of visceral fat reduction by aerobic exercise between men and women, as women generally store a greater total fat mass relative to body weight than men. Also, energy cost of exercise interventions and energy expenditure associated with sedentary behavior may differ considerably between genders [109].

Furthermore, the interplay between physical activity and AO may differ by gender. Although the practice of physical activity may be more beneficial for women than men, it has been shown that inactive women with a large WC had a greater risk of cardiovascular heart diseases (CHD) than inactive men with a large WC [98, 110]. In this sense, Kin and Han [98] examined the interaction between physical activity and AO in relation to the Framingham Risk Score in 2,112 adults, using data from the 2007 Korean National Health and Nutrition Examination Survey (NHANES IV). The findings suggested that the risk of CHD associated with physical inactivity and AO was much stronger in women than in men. Inactive men with a large WC had an OR for CHD risk of 2.91 (95% CI 1.63;5.22), compared with physically active men with a normal WC, while inactive women with a large WC had an OR of 6.37 (95% CI 3.44;11.80), compared with active women with a normal WC [98]. Thus, at the same time that women could have greater benefits from the practice of physical activity compared with men, they also have more deleterious effects from physical inactivity.

#### 5.4. Smoking

Smoking is one of the major causes of preventable death in developed countries and presents a complex interaction with abdominal and general obesity. People with both conditions are at high risk of cardiovascular disease and have a substantially reduced life expectancy. Unfortunately, for many smokers, the fear of weight gain is a considerable barrier to cessation. Thus, understanding the role of smoking in the occurrence of AO is a very important issue.

Smokers have a lower body weight and BMI than nonsmokers. Nevertheless, the association between smoking and WC is inverse. Large cross-sectional studies have supported the hypothesis that current smokers tend to have a larger WC than nonsmokers, suggesting that smoking may increase central fat accumulation [111–113]; prospective studies showed that the number of cigarettes plays an important role in this association [111, 114, 115]. A cross-sectional study

with a nationally representative sample of 9047 Scotland adults found that in women who were current smokers, the mean WC was higher compared with nonsmokers [111]. In the same way, a prospective study of five consecutive birth cohorts of Finnish twins ( $n = 4,296$ ) analyzed the effect of adolescent smoking on AO in early adulthood. Smoking at least 10 cigarettes a day when aged 16–18 years increased the risk of adult AO in 34%, regardless of current smoking status [114]. A cohort of 79,236 white, non-Hispanic healthy adults estimated the effects of behaviors on a 10-year change in odds ratios for WC gain. Women who continued smoking up to a pack a day experienced a small protection against WC gain, but this effect could not be confirmed for women smoking more than a pack a day.

In addition, studies of smokers have shown a dose-response association between total smoking amount during a lifetime and AO. A cross-sectional study with 6,123 Caucasians from Switzerland found that compared with light-smoking men, the odds ratio for AO was 1.28 (95% CI 0.78–2.10) for moderate smokers and 1.94 (95% CI 1.15;3.27) for heavy smokers and 1.07 (95% CI 0.72–1.58) and 2.15 (95% CI 1.26;3.64) in women who were moderate and heavy smokers, respectively [115]. A prospective study in South Korea, with 283 subjects, found results that point to the same direction. WC and VAF area showed a J- or U-shaped association with total smoking during lifetime. And, after restricting the analyses to past/current smokers, they found positive dose-response associations between smoking pack-years and AO [116].

Recently, epigenetic studies have shown that genetic factors may have an important role in this association. A genetic variant in chromosome 15 CHRNA5-CHRNA3-CHRNA4 gene region (rs16969968) was associated with smoking quantity. It was also associated with a lower BMI and a greater WC. In this sense, a Mendelian randomization of the meta-analyses of 29 studies comprising almost 150,000 participants using this genetic variant as a proxy for smoking heaviness was conducted. The results suggested that for every copy of the minor allele associated with cigarette consumption (i.e., increasing cigarette per day consumption by approximately one cigarette), WC will be increased by 0.14%, if BMI were to remain constant. These findings strengthen the hypothesis that a preferential redistribution toward central adiposity is associated with higher cigarette consumption.

The physiological mechanism linking smoking and AO was not fully elucidated, but cortisol and sex hormones could play a possible role in greater WC among smokers. Smokers have higher fasting plasma cortisol concentrations than nonsmokers, and VAF is influenced by the cortisol concentration. In addition, an imbalance between male and female sex hormones [estrogens/androgen in women] and a decrease in testosterone, in men, are observed in smokers [117]. At the same time, unhealthy behavior was also observed in subjects who smoked. Some researchers have pointed to the existence of dangerous behaviors that included smoking, lower physical activity, and unhealthy eating habits in adults, and these behaviors were usual among low SES subjects [106, 118].

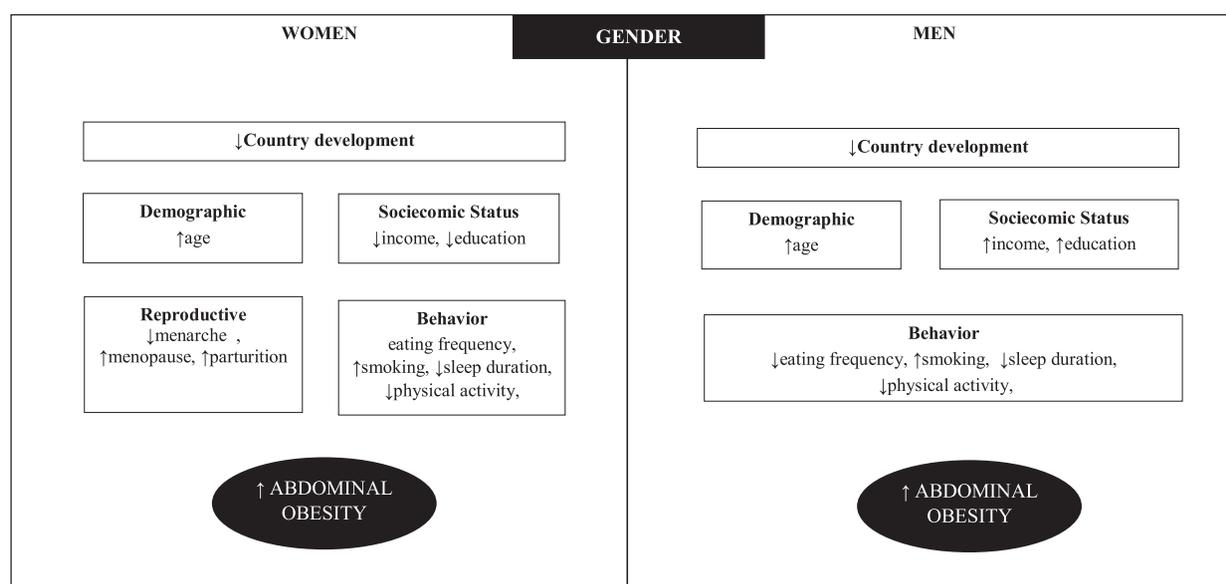
Unfortunately, smoking cessation also has deleterious effects on the body composition. Several studies have reported an increase in weight after smoking cessation [119, 120]. A prospective study Inter99 (1-year follow-up) investigated changes in WC after smoking cessation. Smoking cessation resulted in substantial increase in weight and central fat. Women quitters gained

more weight and had a higher increase in WC than men [121]. Kahn's cohort study showed that quitting smoking was associated with approximately doubling the likelihood of WC gain compared with those who were current smokers or those who never smoked [122]. However, the increase in WC may be explained by the increase in weight; consequently, future studies are needed to elucidate this issue.

The role of smoking in increased overall obesity is well documented in the literature, while its relationship with AO has received less attention. However, current evidence suggests a causal relationship between smoking and fat distribution. Smoking increases the risk of AO and, among smokers, the number of cigarettes smoked per day was also positively associated with AO. We still need further evidence in order to understand the real impact of smoking cessation in the body composition. Thus, smoking should be avoided in order to prevent AO; current smokers should be informed that they are more prone to central fat accumulation and to the inherent additional health risks; the increase in unhealthy central fat after smoking cessation needs attention, mainly among women, who pay more attention to body shape and could avoid smoking cessation.

## 6. Conclusion

In this report, we have showed how demographic, socioeconomic, and behavioral characteristics influence the occurrence of AO in adults. **Figure 1** depicts the mainly associated factors of AO in men and in women. Among women, the population group who is the most affected by AO, a higher prevalence is observed in individuals living in LMIC, who are older, multiparous, and in the menopausal transition, belong to the poorest strata, and have lower educational level, while, among men, the risk of AO is positively associated with social position, particularly in LMIC. The characteristics of women's reproductive life are highlighted in the



**Figure 1.** Causal pathway of abdominal obesity according to gender.

figure, such as menarche, menopause, and parturition, because these risk factors only affect women. Regarding behavioral factors [eating frequency, sleep duration, physical activity, and smoking], gender differences are difficult to be detected due to the lack of studies investigating their association with AO according to sex. However, the current evidence suggests that men benefit more from consuming a greater number of meals a day and women are more affected by the harmful effects of physical inactivity.

Finally, this chapter showed the increasing prevalence of AO around the world, mainly in LMIC, and explored the associated factors. We also argued AO, despite biological conditions associated with behavior factors, should be examined as an important issue of gender inequality in health, possibly mediated by socioeconomic and behavioral differences between men and women.

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## Author details

Maria Teresa Anselmo Olinto<sup>1,2\*</sup>, Heloísa Theodoro<sup>1,3</sup> and Raquel Canuto<sup>4</sup>

\*Address all correspondence to: mtolinto@gmail.com

1 University of Vale do Rio dos Sinos, São Leopoldo, Brazil

2 Department of Nutrition, Federal University of Health Science of Porto Alegre, Porto Alegre, Brazil

3 Department of Nutrition, University of Caxias do Sul, Caxias do Sul, Brazil

4 Department of Nutrition, Federal University of Rio Grande do Sul, Porto Alegre, Brazil

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