

The Practice of Health Promotion for Preventing and Managing Chronic Disease

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Pre-Obesity and Obesity Prevention and Management

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Contradictory as it seems, malnutrition is a crucial contributor to obesity.

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OBJECTIVES

This chapter will enable the reader to:

- **1.** Discuss the incidences and prevalence of pre-obesity and obesity.
- 2. Examine the pathophysiology of obesity.
- **3.** Describe energy homeostasis and weight regulation.
- **4.** Demonstrate knowledge of pre-obesity and obesity risk and etiology.
- **5.** Examine the health consequences of pre-obesity and obesity.
- **6.** Identify valid measures to evaluate pre-obesity and obesity.
- 7. Apply practical obesity-centered history and physical exam skills.
- **8.** Utilize evidence-based treatment guidelines to develop a person-centered obesity management plan.
- **9.** Apply A-SMART and behavioral lifestyle interventions to pre-obesity and obesity management.

Overview

Pre-obesity (also known as overweight) and obesity are the world's most significant public health threats, reaching epidemic proportions, and ranked as the fifth most common causes

of mortality globally (Ritchie & Roser, 2017; World Health Organization [WHO], 2021). This fundamentally results from excess energy balance attributed to multiple factors such as genetics, personal behavior, environment, and social determinants of health. Obesity is

not only a disease but also a risk factor implicated in many of the leading causes of mortality (WHO, 2021). It is primarily an acquired disease that is highly influenced by lifestyle behaviors, such as poor nutrition, overeating, and sedentary habits, which make pre-obesity and obesity prevention realistic, though challenging (Bischoff et al., 2017; WHO, 2021). Due to the complex etiology of pre-obesity and obesity, many factors must be considered when assessing and managing these conditions. This chapter provides an overview of the pathophysiology, etiology, and strategies for preventing and managing pre-obesity and obesity using a lifestyle management approach. It aims to offer the advanced practice provider (APP) the knowledge, skills, and practical evidence-informed strategies to care for patients with pre-obesity and obesity.

Definition of Pre-Obesity and Obesity

Among the various definitions, pre-obesity and obesity are defined as an "abnormal or excessive fat accumulation that may impair health" (WHO, 2021). Over the years, there have been debates regarding classifying obesity as a disease. According to De Lorenzo et al. (2020), in 1998, obesity was declared a disease by the

National Institutes of Health and in 2008 and 2013, endorsed as a disease by The Obesity Society and the American Medical Association, respectively. In 2018, The Obesity Society confirmed that obesity is unequivocally a chronic disease (Jastreboff et al., 2019). The Obesity Medicine Association (OMA) defines obesity as a chronic disease (Bays et al., 2021). See **Table 17.1** for the definitions of obesity from various organizations. In this chapter, pre-obesity represents overweight, and excess weight is used interchangeably with pre-obesity/obesity.

Effect of Pre-Obesity and Obesity

Pre-obesity and obesity are emergent public health concerns that predispose adults to chronic diseases. According to the Global Burden of Disease study conducted in 2017, nearly 5 million premature deaths in 2017 were attributed to obesity (Ritchie & Roser, 2017). This was equivalent to almost four times the number of individuals who died due to motor vehicle accidents and nearly five times those who succumbed to HIV/AIDs in 2017. Globally, over 1.9 billion adults aged 18 and older have excess weight, including 650 million who are obese (WHO, 2021). In addition, more than 340 million children and adolescents were

| Table 17.1 De | finitions of Obesity |
|------------------------------------|---|
| Organization | Definition |
| World Health Organization | "Overweight and obesity are defined as abnormal or excessive fat accumulation that may impair health" (WHO, 2021). |
| The Obesity Society | "Obesity is a multi-causal chronic disease recognized across the lifespan resulting from long-term positive energy balance with development of excess adiposity that over time leads to structural abnormalities, physiological derangements, and functional impairments" (Jastreboff et al., 2019, p. 8). |
| Obesity Medicine Association | "Obesity is defined as a chronic, progressive, relapsing, and treatable multifactorial, neurobehavioral disease, wherein an increase in body fat promotes adipose tissue dysfunction and abnormal fat mass physical forces, resulting in adverse metabolic, biomechanical, and psychosocial health consequences" (Bays et al., 2021). |

pre-obese or obese, including 39 million under 5 years old. Worldwide, since 1975, obesity has increased almost threefold in adults and over six- to eightfold in children (5–19 years old), reaching pandemic proportions (Abarca-Gómez et al., 2017; WHO, 2021). Based on the current trends, pre-obesity and obesity are expected to increase to one in two individuals in the United States by 2030 (Ward et al., 2019).

Though pre-obesity and obesity affect all populations, these conditions are increasing in developing countries and disproportionately affecting minority populations in developed countries. For example, in the United States, Hispanics (25.8%) and non-Hispanic Blacks (22%) had higher incidences of obesity compared to non-Hispanic Whites (14.1%) (Hales et al., 2018). In underresourced communities, there tends to be increased availability of lowcost foods with low nutrients, high calories, and high fat and increased sedentary behaviors, resulting in a double burden of obesity and undernutrition in children (WHO, 2021). However, globally, incidences of overweight and obesity exceed underweight. Therefore, APPs must assess minority populations and those from developing countries for risk factors related to pre-obesity and obesity.

Consequences of Pre-Obesity and Obesity

Excess weight is implicated in the leading causes of mortality and decreased life expectancy of populations affecting nearly all body systems in children and adults. Obesity is a risk factor for many chronic diseases, including cardiovascular disease (CVD), type 2 diabetes (T2D), nonalcoholic fatty liver disease, chronic kidney disease, some cancers, various musculoskeletal problems, sleep apnea, poor mental health, and Alzheimer's disease (Blüher, 2019; Chooi et al., 2019). It is interesting to note that the recent World Cancer Research Fund/American Cancer for Research report (2018) identified strong evidence that excess body fat throughout adulthood

correlated with a higher risk of 12 types of cancers by location: mouth, pharynx, larynx, esophagus, stomach, pancreas, gallbladder, liver, colon, breast (during postmenopause), ovaries, endometrium, prostate (advanced), and kidney. Further, obesity promotes an inflammatory cellular environment that stimulates cell growth and influences anti-apoptotic effects to promote the life of cancer cells.

Children who are obese are at risk for premature adverse health outcomes that typically affect adults with obesity. In addition, they may experience bronchial hyperactivity, asthma, shifting of the femoral bone, social isolation, depression, and anxiety (Camacho et al., 2019). Furthermore, children and adolescents who are obese are more likely to be obese in adulthood and may experience teasing and victimization behaviors due to their weight (Grossman et al., 2017). See **Figure 17.1** and **Figure 17.2** for the common health consequences of obesity in children and adults, respectively.

The Canadian Adult Obesity Clinical Practice Guidelines (Rueda-Clausen et al., 2020) and Sharma (2010) describe the many consequences of being overweight or obese across four components described as the four Ms: mental, mechanical, metabolic, and monetary. For example, mental represents the cognitive and emotional effect of obesity, and mechanical relates to factors such as osteoarthritis, sleep apnea, and gastroesophageal reflux. The metabolic consequences include T2D, hypertension (HTN), gout, cardiac dysfunction, and cancer. Monetary effects of obesity are related to food, occupation, and disability. Refer to the Canadian Adult Obesity Clinical Practice Guidelines for a comprehensive description of the mental, mechanical, metabolic, and monetary consequences of obesity (Rueda-Clausen et al., 2020).

Obesity and Mortality

Several epidemiological studies have explored the association between obesity and

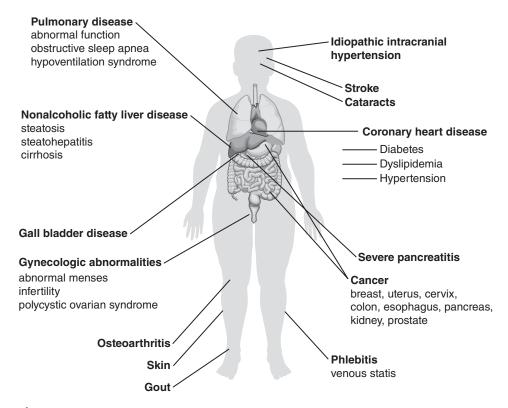


Figure 17.1 Health consequences of childhood obesity.

mortality (Nimptsch et al., 2019). Specifically, in the Prospective Studies Collaboration with 894,576 participants, mortality after adjustment for variables such as smoking was the lowest for those with body mass index (BMI) values of 22.5 to 25 kg/m² (MacMahon et al., 2009). The researchers indicated that every 5 kg/m² increase in BMI correlated with 30% higher all-cause mortality (120% higher diabetes, 80% higher kidney, and 10% higher neoplastic mortality). In another study, the European Prospective Investigation into Cancer and Nutrition, with a sample of 359,387 participants, general adiposity (measured as BMI) and abdominal adiposity (measured as waist circumference [WC] and waist-hip ratio [WHR]) were associated with increased risk of death. However, higher abdominal adiposity was more strongly related to the risk

of death with lower BMI levels, suggesting that abdominal obesity is a better predictor of mortality in individuals with normal BMI (Pischon et al., 2008).

Similarly, a study with a large cohort of nearly 2 million people indicated that most causes of death occurred in BMI ranges classified as underweight, overweight, or obese (Bhaskaran et al., 2018). The highest mortality rates for conditions such as cancer, heart disease, and respiratory diseases were found in individuals who were obese; the lowest risk of death occurred at 21 to 25 kg/m² (Bhaskaran et al., 2018). It is important to note that although there is a significant risk of ill health with excess weight, there are individuals with excess weight without pertinent health conditions (Rueda-Clausen et al., 2020). For these individuals, the recommendation is to

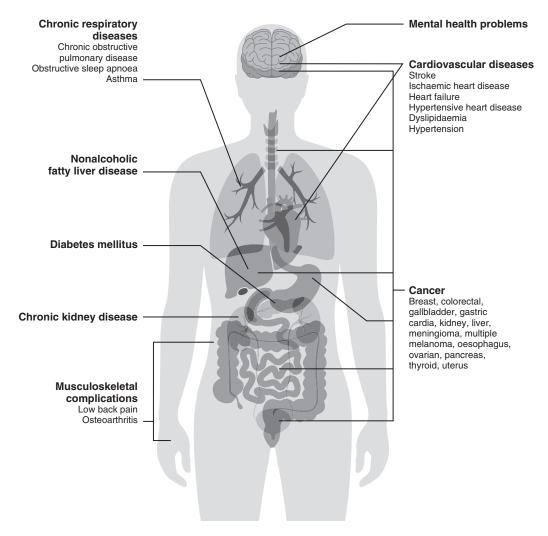


Figure 17.2 Health consequences of obesity for adults.

World Health Organization. (2022). WHO European Regional Obesity Report 2022. Fig 1.10, Medical conditions associated with obesity. WHO Regional Office for Europe. https://apps.who.int/iris/bitstream/handle/10665/353747/9789289057738-eng.pdf

encourage healthy behaviors to prevent additional weight gain and to decrease the progression of relevant complications.

Cost of Obesity

Obesity and associated complications create a significant financial burden on the U.S. economy. According to Waters and Graf (2018) of the Milken Institute, the economic burden of chronic diseases in the United States from

complications of pre-obesity and obesity resulted in \$480.7 billion in direct health care and \$1.24 trillion in indirect costs. Obesity as a risk factor contributes the most to the cost of chronic diseases in America, nearly 50% of the total cost (Waters & Graf, 2018). Additionally, obesity decreases productivity and increases absenteeism and disability—many complications related to obesity significantly influence the cost of health care at the individual and population levels.

Pathophysiology of Obesity

The pathogenesis of obesity is complex and includes biological, behavioral, and environmental factors. These and many other factors contribute to the energy imbalance resulting in obesity. Regulating energy intake and energy expenditure (EE) is the main phenomenon by which energy balance is obtained (Gadde et al., 2018). In a review of seminal studies about obesity, Gadde et al. (2018) concluded that the physiological process associated with obesity included primarily two corresponding features: energy homeostasis and nutrition.

The three factors contributing to energy balance are energy intake, EE, and energy storage (Kadouh & Acosta, 2017). EE involves three metabolic mechanisms, resting metabolic rate (RMR), food thermogenesis, and physical activity EE. RMR is the energy required to maintain essential body organs at rest and is related to fat-free mass, the components of the body that do not accumulate fat, such as the muscles, water content, vital organs, and bones. Food thermogenesis includes all energy expended in eating, digesting, and metabolizing food. Physical activity is expended with daily living, including but not limited to intentional physical exertion. Physical activity is the most varied of the three EE mechanisms because it is relative to the duration of the movement (Westerterp, 2018). Therefore, energy intake that exceeds EE over time leads to a positive energy balance and significantly contributes to excess body weight (Oussaada et al. 2019).

Body weight is well ordered for survival during times of food abundance and starvation. Therefore, a slight increase in energy balance can add up over time to substantial weight gain. A daily excess of as little as 20 calories (comparable to 1 teaspoon of sugar) greater than EE can result in about 1 kilogram (2.2 pounds) of fat per year, which, if sustained over 20 years, would result in

about 20 kilograms of weight gain (Boron & Boulpaep, 2017, as cited in Oussaada et al., 2019).

Though it is well established that obesity results from an imbalance between energy intake and EE (Kadouh & Acosta, 2017; WHO, 2021), it is crucial that APPs explore the contributing factors to energy balance and not merely focus on energy in and out, which puts the blame solely on the individual. If underlying factors are not addressed and the focus is only on behavior, patients may experience shame and weight bias, which may ultimately defy the efforts of the APP to promote weight loss (Kadouh & Acosta, 2017; Tomiyama, 2019). The following sections will highlight the association among biological, behavioral, and environmental factors and excess weight.

Biological Factors

The biological factors associated with obesity include genes, epigenetics, brain–gut axis, neuroendocrine conditions, medications, and the gut microbiome (Kadouh & Acosta, 2017). One or more of these factors interact with environmental and behavioral factors to promote the expression of obesity. Clinicians should be aware of the multiple biological factors influencing obesity as they make treatment decisions. This section describes the most salient biological factors to increase the knowledge, skills, and attitude of the APP caring for patients with obesity.

Genes and Obesity

Genome-wide association studies have identified 140 genes relevant to adiposity (Fall et al., 2017). Genes are the blueprints given at birth, instructing the body to respond to environmental changes (Yanovski & Yanovski, 2018). The relationship between genetics and obesity is complex, genome-wide research is ongoing, and more evidence is needed to support genetic testing in the clinical setting to predict obesity risk (Loos & Yeo, 2021). The genetic

implications of obesity are based on the type of gene involved (Huvenne et al., 2016), such as the more common polygenic or the rare monogenic. Rare genetic variations are monogenic obesity and are identified as syndromic or oligogenic obesity, related to mutations in the gene coding the leptin receptor and many components of the melanocortin pathway.

Syndromic and oligogenic obesity have different characteristics. Syndromic is severe early-onset obesity associated with other phenotypes, including mental retardation and malformations of features and organs; the most commonly known syndromic obesity are Prader-Willi and Bardet-Biedl (Huvenne et al., 2016). Oligogenic obesity is associated with variation in the severity of obesity attributed to 2% to 3% of adults and children who are obese. It is relatively dependent on environmental elements and nonexistent of a specific phenotype. Overall, monogenic obesity affects a tiny subset of the population worldwide, ranging from less than 10 to 100 patients worldwide (Huvenne et al., 2016). Congenital leptin deficiency causes hyperphagia in infants who, at birth, had weight ranges within the normal limits, resulting in severe obesity within the first months of life (Yanovski & Yanovski, 2018). If identified through genetic testing, individuals with leptin deficiency may be treated with leptin to decrease obesity (Yanovski & Yanovski, 2018).

The rare forms of monogenic obesity differ from the most common form, polygenic obesity. *Polygenic* obesity is highly correlated to "obesogenic" lifestyle factors, including overeating, lack of physical activity, and stress (Huvenne et al., 2016). One of the genes associated with polygenic obesity is the fat mass and obesity-associated (FTO) gene (Hess & Brüning, 2014). Of all the known genes, FTO is the most strongly associated with BMI in children and adults, irrespective of gender. There are ongoing studies to elucidate the genetic contribution to BMI. See **Table 17.2** for differences in characteristics of monogenic and polygenic obesity.

To further elucidate the influence of the FTO gene on obesity, researchers conducted a study with 359 healthy males with normal BMI levels to determine how those with highrisk FTO genes and those with low-risk FTO genes modulate the neural and hormone responses to eating and food images (Karra et al., 2013). The study results identified that men with the high-risk FTO gene had more

| Table 17.2 | Characteristics of | of Monogenic and | Polygenic Obesity |
|-------------------|--------------------|------------------|-------------------|
|-------------------|--------------------|------------------|-------------------|

| Characteristics | Monogenic | Polygenic |
|-------------------------|---------------------------------|------------------------------------|
| Onset of obesity | Early, severe | Common obesity |
| Genetic contribution | High | Modest |
| Mutation | Single gene mutation | Hundreds of variants or many genes |
| Genetic effect | Large | Small effect in each variant |
| Frequency | Rare | Common |
| Penetrance | High | Low |
| Environmental influence | None | High |
| Gene name (examples) | Leptin, melanocortin 4 receptor | |

Data from Loos, R. J. F., & Yeo, G. S. (2021). The genetics of obesity: From discovery to biology. *Nature Reviews Genetics*, 23(2), 120–133. https://doi.org/10.1038/s41576-021-00414-z

of the hunger hormone ghrelin after eating than men with the low-risk FTO gene. Additionally, the FTO gene increased more rapidly after eating, indicating that the men with the high-risk FTO gene did not appropriately decrease ghrelin after meals. The researchers also identified that men with high-risk FTO genes had an increased response in the brain's reward center and the hypothalamus, which regulates appetite, resulting in a desire to eat more. These results provide insightful findings on the influence of the FTO genotype on the complex regulation of metabolism at the physiological level.

The influence of healthy lifestyle behaviors on the genetic tendency to develop obesity is highlighted by a meta-analysis of over 200,000 adults with the FTO gene (Graff et al., 2017). Those who were physically active reduced the odds of being obese by about 30% compared to inactive adults. According to a recent study, increased vegetable and fruit intake can mitigate elevated BMI in individuals genetically susceptible to obesity (Wang et al., 2019). Thus, environmental factors that promote healthy eating and physical activity may alleviate the polygenic effects of obesity.

These findings highlight the importance of multifactorial lifestyle behaviors in attenuating the risk of obesity.

Genetic Obesity Risk Index

In 2002, researchers tested a classic model to detect the genetic risk of obesity in individuals to help guide treatment (Thirby & Randall, 2002). The findings revealed that 85% of bariatric surgery candidates had a history suggesting a genetic risk for morbid obesity and 15% indicated an exceedingly strong genetic obesity risk index. The two most likely factors to correlate with multifactorial genetic risk are familial history and age of onset of obesity. See the following box for the classic genetic obesity risk index questionnaire and scoring.

Epigenetics and Obesity

Epigenetics is the effect of behaviors and environment on gene expression and can turn genes on and off (National Human Genome Research Institute, 2022). Epigenetics vary with age due to the usual developmental process and in response to behavioral and

Genetic Obesity Risk Index Screening Questionnaire

Patient Instructions: Answer the following four questions to see whether genetics can be the reason for your weight gain. For measurement purposes, pre-obese/overweight means having a BMI of 25–29.9; obese is a BMI of 30–39.9, and very obese is a BMI of \geq 40. BMI calculators are available online and are calculated based on weight and height.

1. Were either or both of your parents obese or very obese for most of their lives?

| | Po | oints |
|-----------------------|-------|------------|
| | Obese | Very Obese |
| Neither/don't know/no | 0 | 0 |
| Yes, one parent | 7 | 14 |
| Yes, both parents | 14 | 28 |

Do you have any second-degree relatives who have been obese most of their lives?
 Score 2 points for every first-degree relative with obesity up to a maximum of 10 points.

3. How would you describe the average BMI of your siblings?

| | Points |
|---|--------|
| No siblings with obesity (BMI <30) | 0 |
| Average sibling with obesity (BMI ≥30) | 6 |
| Average sibling is very obese (BMI ≥40) | 12 |

4. When did you first become pre-obese/overweight and/or obese?

| | Points | | |
|---------------|----------------------|-------|--|
| | Pre-Obese/Overweight | Obese | |
| Never | 0 | 0 | |
| Before age 10 | 20 | 30 | |
| Before age 20 | 10 | 20 | |
| Before age 30 | 5 | 10 | |

| Sum | the points. | |
|-------|-------------|--|
| Total | . score | |

Interpreting your score

- <20: Your weight problem does not appear to be significantly related to genetics. This means it is related to lifestyle and could be solved with committed lifestyle change.
- 20–50: There appears to be a moderate hereditary component to your weight problem. This means you may find losing weight more challenging than people you know. You may need help from a dietitian, nutritionist, health professional with expertise in weight management, or health coach.
- 30–100: There appears to be a significant hereditary component to your weight problem. This means you need additional help and closer attention from a dietitian or professional in weight management. With the proper approach and a long-range plan, you should be able to overcome your inherited start.

Data from Rossner, S., Egger, G., & Binns, A. (2017). Overweight and obesity: The epidemic's underbelly. In G. Egger, A. Binns, S. Rossner, & M. Sagner (Eds.), Lifestyle medicine: Lifestyle, the environment and preventive medicine in health and disease (3rd ed., pp 131–132). Academic Press.

environmental factors. There is evidence that prenatal behavior and ecological conditions can affect the epigenetics of offspring. Among the salient epigenetic contributions to excess weight in first- and second-generation offspring is the effect of maternal nutrition and weight status before conception and during pregnancy (Chen et al., 2021; Kadouh & Acosta, 2017). Additionally, there is evidence that poor paternal nutrition in animal models and humans increases the risk of excess weight

(Ozanne, 2015). A review of the findings of studies regarding men and women whose mothers during pregnancy experienced the Dutch famine from 1944 to 1945 revealed that undernutrition increased the risk of chronic conditions, including obesity (Roseboom, 2019). A study identified that mothers with increased intake of pro-inflammatory foods were more likely to have children at increased risk of overweight and obesity (Chen et al., 2021). A pro-inflammatory diet includes a

lower intake of fruits, vegetables, and nuts/ seeds/legumes and higher intakes of red and processed meats and sugar-sweetened beverages. These findings have clinical significance for APPs to help prospective mothers increase their intake of a high-quality diet that promotes an anti-inflammatory body state and overall weight management.

The environmental influence on epigenetics may commonly influence obesity development, as demonstrated by the findings of obesity from a population with the same genetics but with different ecological exposure. For example, 60% of Pima Indians in Arizona are obese compared to 20% of the genetically similar Pima Indians in Sierra Madre, Mexico, two regions with marked environmental differences in access to food and physical activity levels (Schulz et al., 2006). The U.S. Pima Indians reported having a high percentage of calories from fat, lower fiber intake, and significantly lower occupational and leisure physical activity levels than the Pima Indians in Mexico. These results indicate an evident influence of the environment on obesity (Yanovski & Yanovski, 2018). However, even among individuals surrounded by obeseenhancing factors, there is variability among those who will become obese (Yanovski & Yanovski, 2018). These differences may result from environmental, socioeconomic, psychological, and gene-environmental connections or genetics and epigenetics.

The expression of genes varies across tissue type and time sequence and can also be influenced by DNA methylation, a physiological process of adding a methyl group to DNA. This is done by DNA methyltransferase and is a heritable (epigenetic) modification leading to malignancy, arteriosclerosis, CVD, and chromosomal defect neurological disorders such as Prader-Willi syndrome (Kandi & Vadakedath, 2015). Genetic defects can influence methylation, inflammation, nutrition, physical activity, and aging, among many other factors (Fall et al., 2017). These factors can modulate genetics and epigenetics to promote obesity and obesity-related diseases (Fall et al., 2017), as reflected in Figure 17.3. An epigenetic predisposition to develop obesity can be prevented or treated through a consistent efficacious plan that includes nutrition, movement, and behavior approaches (OMA, 2018; Rohde, 2019).

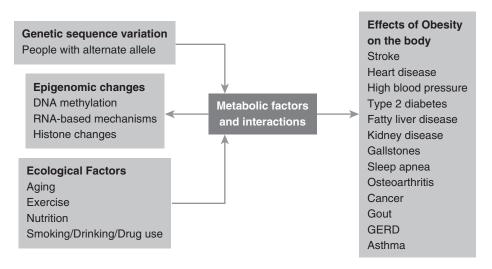


Figure 17.3 Genetic and epigenetic variation influences gene expression.

Brain-Gut Axis and Obesity

The brain is the most significant player in excess weight and energy balance (Lau & Wharton, 2020). The brain–gut axis is impaired in people with obesity compared to normal-weight individuals (Kadouh & Acosta, 2017). The following section will describe the neurobiology of obesity as described in the *Canadian Adult Obesity Clinical Practice Guidelines* (Lau & Wharton, 2020). A broad overview of three areas of the brain—the hypothalamus, mesolimbic area, and cognitive lobe, all of which regulate weight—will provide a basic understanding of the neurobiology of obesity.

Hypothalamus Role in Energy Homeo-

stasis. The hypothalamus plays a vital role in energy homeostasis by controlling energy intake and expenditure (Lau & Wharton, 2020). The arcuate nucleus of the hypothalamus, also referred to as the hunger center, controls eating habits. Signals from hormonal and neuronal activity in the gut, adipose tissue, and peripheral organs activate neurons in the arcuate nucleus to stimulate the feeling of hunger and trigger behaviors to seek food. When there is access to food, there is a downregulation of the activity of the neurons. Many other factors influence appetite control in the arcuate nucleus, including the individual's nutritional state, sensing for nutrients, taste, smell, and food preferences.

Mesolimbic System. The mesolimbic dopamine pathway drives motivation and incentive to repeat pleasurable activities such as eating, known as hedonic eating (Lau & Wharton, 2020). *Hedonic eating* occurs when a satiated person eats food mainly because of its tasty reward rather than for nutritional need (Meye & Adan, 2014). It is associated with the sight, smell, and taste of food and under ideal conditions controls a person's response to natural rewards such as food. Some individuals with obesity have a malfunctioning mesolimbic dopamine system, leading to increased

anticipation (wanting) of food, which results in a need to overeat to fulfill the level of expectation (Bello et al., 2010, and Volkow et al., 2010, as cited in Lau & Wharton, 2020). Multiple factors may trigger excess food intake.

In addition to overeating as a response to emotions, mood disorders can promote unhealthy eating behaviors (Singh, 2014). In some situations, excessive eating, particularly foods high in simple carbohydrates that affect the feel-good hormones of serotonin and the reward system, may be used as self-medication to improve symptoms of atypical depression. Other neuropsychological health conditions influencing excessive eating include attention deficit disorder, sleep disorders, chronic pain, anxiety disorders, addictions, seasonal affective disorders, and cognitive disorders (Singh, 2014). Medications, hormone management, and cognitive behavior therapy counseling may control serotonin dysregulation and treat obesity. This accentuates the need for an interprofessional healthcare team to manage overweight and obesity.

Cognitive Lobe and Executive Func**tioning.** The cognitive lobe is responsible for mental skills such as working memory, flexible thinking, and self-control, also referred to as executive functioning (Yang et al., 2018). Working memory manages relevant incoming stimuli and updates the information in memory as needed. Flexible thinking shifts attention or mental rules when situationally appropriate, and self-control suppresses impulsiveness. The cognitive lobe functions best under optimum states, such as adequate rest, decreased stress, and proper social support (Lau & Wharton, 2020). Therefore, overeating when one feels fatigued and stressed at the end of the day is not unusual. Encouraging mindful eating may increase awareness of factors triggering poor eating habits. Individuals who are obese may have a malfunctioning connection between the cognitive lobe and other parts of the brain resulting in a lack of control over eating. For example, when faced with the

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choice between eating a donut or an apple, individuals with impaired executive function may choose the donut even with the knowledge of the ill effects and that it will hinder their weight loss goal.

Influence of Hormones on Appetite

Several hormones are major players in maintaining homeostasis, including leptin, ghrelin, and insulin (Adamska-Patruno et al., 2018). Leptin and ghrelin are the hormones most associated with satiety and appetite (Yeung & Tadi, 2022). In simple terms, leptin is released from adipocytes (fat cells), sending signals to the hypothalamus. The primary purpose of leptin is to maintain body weight by controlling the desire to eat. Leptin, the satiety hormone, deters hunger when the body is at an optimum function, so the body does not desire to eat when it does not need energy. Leptin positively correlates to a person's amount of adiposity. As adiposity increases, leptin levels will increase, and if a person decreases the proportion of body fat, the leptin levels will decrease (Obradovic et al., 2021). However, when individuals are obese, they will have excess leptin levels, resulting in leptin resistance. With leptin resistance, there is decreased satiety and an increase in appetite. Hence, overeating and craving may occur, resulting in difficulty maintaining weight loss.

In obese patients, central leptin and insulin resistance develop from excess high-fat foods, caloric surplus, and increasing adiposity (Timper & Brüning, 2017). Furthermore, these factors and a dietary increase in saturated fats contribute to hypothalamic inflammation. Hence, obesity contributes to insulin and leptin resistance in the peripheral and central nervous systems (Timper & Brüning, 2017). Together, these disruptions contribute to hypothalamic inflammation and impairment of the insulin and leptin pathways, leading to increased hunger and exacerbating the development of obesity (Timper & Brüning, 2017).

Ghrelin is an appetite stimulant secreted mainly from the mucosa of the stomach fundus and pancreatic cells (Verdeş et al., 2017). The function of ghrelin is to stimulate appetite and prepare the body to eat, and it has been called the hunger hormone. Ghrelin influences short-term food intake by increasing levels before eating and decreasing levels with food intake.

In a recent study to evaluate the ratio of leptin and ghrelin in men based on meals with various macronutrients, researchers determined differences in men of normal weight compared to men who were pre-obese/obese (Adamska-Patruno et al., 2018). In normalweight men, the leptin/ghrelin ratio was more favorable after a high-carbohydrate fat-free meal than the high-fat normal amount of carbohydrate meal. Additionally, the study results indicated that compared to individuals with normal weight, males with pre-obesity and obesity had a significantly elevated leptin/ghrelin ratio in a fasting state and after eating each meal. These results provide clinical relevance that a high carbohydrate meal may be more favorable when offering nutrition counseling to men of normal weight. In contrast, in men with pre-obesity or obesity, lower carbohydrate meals may be preferable. In individuals with pre-obesity/obesity, it is ideal to have an elevated leptin/ghrelin ratio to promote satiety and decrease appetite stimulation to improve health outcomes through weight loss.

Endocrine Function of Adipocytes

The physiological link between obesity and chronic diseases is due to the endocrine function of adipocytes in adipose tissue, particularly visceral or abdominal adipose tissue, which secretes cytokines and adipokines (Nimptsch et al., 2019). Also, adiposity results in a disproportional accumulation of adipocytes associated with increased secretion of pro-inflammatory cytokines (e.g., interleukin-1 β , interleukin-6, and tumor

necrosis factor α) and adipokines (e.g., leptin and resistin) and a decrease in anti-inflammatory adipokines such as adiponectin (Forny-Germano et al., 2019; Nimptsch et al., 2019). The increased release of pro-inflammatory substances and the reduction of anti-inflammatory substances during obesity result in the body's being in a steady state of low-grade inflammation, which leads to insulin resistance, T2D, HTN, coronary artery disease, some cancers, and arthritis, among many other health consequences of obesity (Forny-Germano et al., 2019; Odegaard & Chawla, 2013).

Medications

Many prescribed medications are known to have an obesogenic, or weight-promoting, effect (Rueda-Clausen et al., 2020). **Table 17.3** provides a list of commonly used obesogenic medications across various drug classes and alternative medicines to consider. APPs must consider the implications of medications when evaluating and treating patients with or at risk for pre-obesity and obesity.

Microbiome and Obesity

The role of the microbiome in obesity is not fully understood (Aoun et al., 2020). However, evidence supports that the microbiota promotes digestion and metabolism. A more diverse gut microbiota is favorable to lean weight and may have a protective effect on weight gain in healthy individuals. In contrast, diets high in processed fat and carbohydrates and low in whole-plant foods decrease the variety of the gut microbiome. Dietary eating patterns high in fiber promote a diverse gut microbiome (Aoun et al., 2020). Based on studies using mice, there is emerging evidence that fecal microbiota transplant from lean mice to obese mice resulted in more weight loss and prevention of weight gain in the long term (Thaiss et al., 2016). The results of studies using fecal microbiota transplants with humans have revealed mixed results, but this has the potential to change the future management of obesity (Thaiss et al., 2016).

Behavioral Factors

Behavioral elements encompass high-caloric intake, stress, physical inactivity, alcohol use, inadequate sleep, and former smokers. Besides the rare types of obesity that originate from genetic causes or factors that negatively impair EE, individual lifestyle behaviors are vital in triggering the interaction between biological and environmental influences to produce a state of obesity (Kadouh & Acosta, 2017). Many individuals' choices affect how the environment will determine their weight status. Lifestyle behaviors such as sedentary habits and sleep patterns are ultimately personal choices. Environmental factors promoting healthy options will deter poor decisions, making the healthy choice easy. A brief description of the effect of selected behaviors on weight status will be described next.

Dietary Patterns and Excess Weight

Long-standing evidence supports that dietary patterns high in processed foods, including refined sugars and fats, are linked with excess weight. In a 4-year longitudinal prospective study, researchers identified the relationship between multiple lifestyle behaviors and weight gain over the long term in men and women who were not obese (Mozaffarian et al., 2011). The study results revealed that weight gain was considerably related to eating potato chips, potatoes, beverages sweetened with sugar, unprocessed red meat, and processed meat and was inversely related to eating vegetables, whole grains, fruits, nuts, and yogurt. The results regarding the effect of food on weight are supported by Smith et al. (2015). These researchers argue that the heterogeneity between food and weight change indicates differences in how foods determine satiety, resting EE, microbiome, liver fat content, and other metabolic factors.

Further, Smith et al. (2015) indicate that varied foods of similar caloric value support that not all calories equivalently affect health

| Table 17.3 | Common Weight-Pr | Table 17.3 Common Weight-Promoting Medications | | | | |
|--|--|--|--|--|---|---|
| Classification | Anticonvulsants | Antidepressants | Antihyperglycemics | Antihypertensive | Antipsychotics | Corticosteroid |
| Weight- Promoting Medications | Valproic Acid *** Carbamazepine*** Gabapentin*** | Amitriptyline*** Doxepin*** Impramine** Mortriptyline** Atypical Mirtazapine** MAOIs Phenelzine*** Tranylcypromine*** SSRIs Sertraline* Paroxetine** Citalopram*** Escitalopram*** Fluoxetine*** | Insulin** Insulin** Thiazolidinedione Pioglizaone** Sulfonylureas Glipizide* Glyburide** Glimepiride** | Clonidine* Propranolol* Metoprolol* Atenolol** | Haloperidol** Loxapine** Clozapine** Risperidone** Quetiapine** | Prednisone *** Prednisolone *** Cortisone *** |
| Medication substitutes for weight- promoting medications | Topiramate Zonisamide Lamotrigine | Bupropion Nefazodone Duloxetine Venlafaxine Desvenlafaxine Trazodone Vilazodone Vortioxetine Selegitine (topical MAOIs) Fluvoxamine (variable | Metformin DPP4i (alogliptin, linagliptin, axagliptin, axagliptin) GLP-1 (exenatide, liraglutide, semaglutide, dulaglutide, semaglutide) AGI (acarbose, miglitol) SGLI-2 analogs (canagliflozin, dapagliflozin, empagliflozin, empa | Prazosin ACEi ARBs Diuretics CCBs (may cause fluid retention) | Ziprasidone Lurasidone Aripiprazole | Budesonide NSAIDs |

Angiotensin-converting inhibitors; ARBs: Angiotensin II receptors blockers; Calcium channel blockers; MAOIs: Monoamine oxidase inhibitors; SRIs: Selective serotonin reuptake inhibitors;+Combination therapy is less likely to cause weight gain; ** 5-10 kg weight gain; ** DPP4i: Inhibitors of dipeptidy peptidase 4; GLP-1: Glucagon-Like peptide-1 receptor agonists, NSAIDs: Nonsteroidal anti-inflammatory drugs: SGL7-2: Sodium glucose co-transporter 2; AGI: Alpha-glucosidase inhibitor; ACEI:

and weight. Some calories may increase weight, and others may promote weight loss. For example, nuts were thought to promote weight gain since they are high in calories per portion size; however, both Mozaffarian et al. (2011) and Smith et al. (2015) identified a decrease in weight with eating nuts, similar to Tan et al. (2014).

Stress and Obesity

Both stress and obesity are ubiquitous issues in society and are interrelated in a cyclical pattern (Tomiyama, 2019). Stress is a well-known contributor to decision-making and affects many lifestyle behavior patterns, resulting in poor eating behaviors. Most recently, Americans have attributed the COVID-19 pandemic as a source of stress for one-third of the adult population affecting fundamental decisions about what to eat (American Psychological Association, 2021). Notably, parents with children under 18, compared to those without children, reported experiencing more stress than before the pandemic.

Chronic or repeated acute stressors are associated with obesity through interactions that span cognition, physiology, biochemistry, and behaviors (Tomiyama, 2019). Stress impairs the cognitive process of self-regulation, which would influence behaviors that prevent obesity, such as dietary choices and physical activity. Impaired self-regulation may then lead to unhealthy behaviors. Further, stress consistently activates many physiological systems, such as activation of the hypothalamicpituitary-adrenal axis, leading to increased cortisol levels known to promote eating and fat deposition with an emphasis in the abdominal area. There is evidence that the stress-obesity-stigma-stress cycle results from the stigma of excess weight, leading to an increase in stress, which exacerbates weight gain through an interplay of physiology and behaviors (Tomiyama, 2019). A detailed discussion of stress and its relationship to obesity is beyond the scope of this chapter. However, addressing stress as an upstream causal factor in obesity would be effective and valuable (Tomiyama, 2019).

Physical Inactivity and Obesity

Physical inactivity is linked to excess weight (Park et al., 2020). It may be exacerbated by conditions that encourage prolonged sitting, such as working on the computer, playing video games, and excessive TV watching. In particular, sedentary time is associated with increased WC. It is essential to highlight a classic finding that a 10% increase in physical inactivity increased WC by 1.2 in. (Healy et al., 2008). According to the WHO (2022), by 2030, nearly 500 million individuals will develop chronic conditions such as obesity and obesity-related disorders due to physical inactivity if there are no urgent public health measures to promote physical activity globally.

Alcohol and Obesity

Until recently, the prevailing belief was that drinking in moderation was safe. However, most recently, evidence has linked not only excessive drinking with poor health outcomes (Traversy & Chaput, 2015) but also drinking in small amounts. Drinking less than a standard alcoholic drink increased the risk of obesity in a study of 27 million (European Association for the Study of Obesity, 2020). The study defined a standard drink as 14 g (0.6 oz) of alcohol per day, equivalent to 5 oz of wine and 8 oz of beer. This is consistent with the definition of a standard drink by the Centers for Disease Control and Prevention (n.d.).

Compared to nondrinkers, men and women who consumed between 50% and 100% of a standard drink were 22% and 3% more likely to develop obesity, respectively (European Association for the Study of Obesity, 2020). However, the odds increased considerably for obesity for men (34%) and women (22%) who drank more than two drinks compared to those who did not drink. Furthermore, researchers identified an increased risk of illness

and death related to nonalcoholic fatty and alcoholic fatty liver disease in individuals who were overweight or obese and who drank more than 14 g per week (Inan-Eroglu et al., 2022). Individuals in the normal range did not have the same response to drinking alcohol. Evidence is emerging that alcohol contributes to obesity, and individuals who are already overweight may increase their risk of liver disease by drinking even small amounts (Inan-Eroglu et al., 2022). Inconclusive evidence exists regarding intake and obesity. The preponderance of evidence taken as a whole suggests that even when consumed in small quantities, alcohol may be a risk factor for obesity in some individuals.

Sleep and Obesity

There is evidence that insufficient and too much sleep can similarly affect weight status. A plethora of evidence indicates that inadequate sleep is associated with increased weight, particularly in children, adolescents, and younger adults (Bonanno et al., 2019; Li et al., 2017; Wu et al., 2014). Individuals who consistently slept less than 7 hours were more likely to have excess weight (Cooper et al., 2018). However, sleeping 9 or more hours was also associated with excess weight (Theorell-Haglow et al., 2014). Also, the relationship between sleep deprivation and increased weight is bidirectional, and it may be challenging to determine what came first, as in the chicken or the egg. Likewise, insomnia can be a barrier to physical activity, and a lack of physical activity can cause sleep impairment (Tomiyama, 2019). Additionally, insufficient sleep is associated with the increased hunger hormone ghrelin, the decreased satiety hormone leptin, and enhanced hedonic signaling—the drive to eat for pleasure without being hungry (Cooper et al., 2018).

Other hormonal imbalances resulting from sleep deficiency include increased cortisol levels and decreased growth hormone, both associated with obesity (Fry, 2022). Further, less than ideal sleep impairs the metabolism

of food. The mechanism for long sleep duration and obesity needs further investigation. Still, research findings support an association with factors such as poor sleep quality, physical inactivity, unhealthy food intake, an imbalance between the sleep—wake cycle, or disease comorbidities such as depression and HTN (Tan et al., 2018). Assessing patients for short and prolonged sleep is essential to obesity management.

Tobacco Use and Obesity

It is well known that quitting smoking tends to be associated with weight gain, but this is not conclusive. In a study to determine the relationship between smoking and obesity, the results indicated that former heavy smokers were more likely to be obese than former light smokers (Dare et al., 2015). However, heavy smokers who were younger and those who lived in the most affluent areas were more likely to be obese than those who never smoked. Interestingly, the study revealed that the risk of weight gain after quitting smoking was time limited. Former smokers after 20 years were at the same risk of obesity as those who had never smoked. The APP should put interventions in place to assist patients who quit smoking to prevent weight gain.

Environmental Factors

Environmental determinants of obesity include food abundance, food insecurity, built environment, socioeconomic status, culture, and bias (Kadouh & Acosta, 2017). Though an individual may have a biological tendency toward obesity, it is usually manifested due to an interplay with an obesogenic environment. There are two primary factors of an obesogenic environment. One is an overabundance of foods, and the other is the built environment. Fox et al. (2019) support the theory that modernization has led to economic advances that have promoted the transition from lower-calorie, plant-based diets to processed foods and meats high in calories, resulting in excess weight and

ill health. With uncontrolled urban expansion, the built environment has resulted in increased use of cars for transportation, making walking less appealing (Kadouh & Acosta, 2017). It is well established that dietary patterns high in animal meat protein and processed foods and an environment that promotes sedentary behaviors, such as driving, have directly correlated to the rise in overweight and obesity (Fox et al., 2019; Kadouh & Acosta, 2017). Most environmental factors are modifiable through government policies that promote access to healthy foods and areas for recreation and play.

Assessment of Pre-Obesity and Obesity

Many types of measures are available to determine adiposity, with varying utility. There are direct quantitative measures to assess total body adipose tissue, including dual-energy X-ray absorptiometry (DEXA) and bioelectrical impedance (Garvey et al., 2016). However, these direct measures are costly and require

specialized personnel; they are not practical for clinical settings and are typically reserved for research. Therefore, measurements of obesity in this chapter will focus on those feasible for the clinical practice or community setting.

There is a long-standing debate about which anthropometric measurement—BMI, WC, or WHR-is the best predictor of disease risk in adults, including CVD, T2D, and all-cause mortality. BMI and WC are interrelated. However, WC is an independent predictor of risk factors and illness beyond BMI (National Heart, Lung, and Blood Institute [NHLBI], n.d.). WC approximates visceral adipose tissue and is the most common and easiest measurement of abdominal obesity (Garvey et al., 2016). Elevated BMI, WC, and WHR values usually indicate an increased risk of CVD, T2D, and all-cause mortality. Individuals with a normal BMI but large WC or WHR are at higher risk of health problems than those with WC or WHR levels within the normal ranges (Huxley, 2010). In general, BMI and WC demonstrate the most substantial evidence for anthropometric criteria for diagnosing pre-obesity and obesity in the clinical setting (Garvey et al., 2016). See Table 17.4

Table 17.4 Level of Evidence for Anthropometric Criteria for the Assessment of Pre-Obesity and Obesity in the Clinical Setting

| Anthropomorphic Criteria | *Recommendation Grade |
|---|-----------------------|
| BMI to confirm excess adiposity and diagnose pre-obesity or obesity. | А |
| Other adiposity measurements such as dual-energy X-ray absorptiometry (DEXA) and bioelectrical impedance. Air/water displacement plethysmography may be used at the clinician's discretion if BMI and physical exam results are equivocal or additional evaluation is required. | С |
| WC should be measured in all patients with BMI $<$ 35 kg/m 2 to determine adiposity-related disease risk. | А |

^{*} Grades may be interpreted as being based on strong (Grade A), intermediate (Grade B), weak (Grade C), or no (Grade D) scientific substantiation.

Data from Garvey, W. T., Mechanick, J. I., Brett, E. M., Garber, A. J., Hurley, D. L., Jastreboff, A. M., Nadolsky, K., Pessah-Pollack, R., & Plodkowski, R. (2016).

Data from Garvey, W. T., Mechanick, J. I., Brett, E. M., Garber, A. J., Hurley, D. L., Jastreboff, A. M., Nadolsky, K., Pessah-Pollack, K., & Plodkowski, R. (2016). American Association of Clinical Endocrinologists and American College of Endocrinology comprehensive clinical practice guidelines for medical care of patients with obesity. *Endocrine Practice*, 22, 1–203. https://doi.org/10.4158/EP161365.GL

for the evidence supporting anthropometric standards to assess weight status in the clinical setting.

Body Mass Index

In adults and children 6 years and older, BMI is the most common clinical measure of weight status and risk associated with weight in the clinical setting, even though it has limitations. For adults, height and weight screening to calculate BMI should be conducted annually or more frequently (Jensen et al., 2014). In children, BMI is calculated the same as in adults, but the weight status for children is based on age- and sex-specific BMI percentiles (U.S. Preventive Services Task Force [USPSTF], 2017). Using BMI measurements to determine weight status and disease risk varies depending on ethnic groups and muscular development (Rueda-Clausen et al., 2020). As such, BMI measures may miscalculate obesity in certain conditions or groups, such as volume overload, sarcopenia, ascites, athletes, and populations of a more petite body frame. For example, Bill, a 49-year-old bodybuilder with a BMI of 33, may not be at the same disease risk as Rick, a 65-year-old adult with sarcopenia who does minimal exercise and has a BMI of 30. Bill, the bodybuilder, has more muscle mass and will weigh more; therefore, APPs should consider other factors related to body structure when interpreting BMI. Though BMI is limited, it continues to be a helpful screening measurement for individuals and populations. Screening patients for excess weight using BMI should be a routine part of most healthcare encounters. WC should be added to the assessment to identify the risk of increased visceral adiposity and the associated health risk. See Table 17.5 for the BMI formulas in metric and imperial English.

Waist Circumference

WC is positively associated with abdominal adiposity and is a reliable measure to

Table 17.5 Body Mass Index Formula

| Unit | Formula |
|---------------------|---|
| Metric | BMI = weight (kg)/height (m²) |
| Imperial English | BMI = weight (lb)/[height (in.]] ² x 703 |

determine abdominal fat content before and during weight loss treatment. *Central obesity* is defined as a WC >40 in. (>102 cm) in men and >35 in. (>88 cm) in women (NHLBI, n.d.). WC is considered more beneficial for individuals with BMI <24.9 or BMI 25.0 to 29.9 than for individuals who are obese on the BMI scale (NHLBI, n.d.). Measuring the WC at BMI values ≥35 may not add a predictive value of disease risk or change the management (NHLBI, n.d.).

However, WC may be more predictive of disease risk in older adults and ethnic groups with a shorter stature, including Asian Americans or individuals of Asian descent. Additionally, WC may provide pertinent information regarding the efficacy of weight loss treatment for long-term follow-up. In some patients, central adiposity changes may be initially more noticeable than BMI. Continuing with the scenario regarding Bill and Rick, Bill, the bodybuilder, has a WC of 36 in., and Rick has a WC of 43 in. Rick is at increased risk of cardiometabolic disorders based on the two measurements. WC within the high-risk categories is correlated with an increased risk of T2D, dyslipidemia, HTN, and CVD (NHLBI, n.d.).

Shape Matters

Over six decades ago, a French physician, Jean Vague, identified that body shape could predict disease risk (Harvard T. H. Chan School of Public Health, n.d.). Individuals shaped like an apple or with a larger waist had an increased risk of early heart disease and death than those shaped like a pear, who had a smaller waist or carried more weight around their hips and thighs (Harvard T. H. Chan School of Public

| Table 17.0 Weight classifications by DMI, WC, and Associated Kisk | | | | | | |
|---|--|-----------|-----------------------------|--------------------|---------------------|--|
| Weight Classification Category | Underweight | Normal | Overweight (Pre-obesity) | Obesity Class I | Obesity Class II | Obesity Class III (Morbid Obesity) |
| BMI (kg/m²) | < 18.5 | 18.5–24.9 | 25.0-29.9 | 20.0-34.9 | 35.0–39.9 | ≥ 40 |
| Comorbidity Risk | Low, but can lead to other health issues | Average | Increased | Moderate | Severe | Very severe |
| WC and Comorbidity Risk | | | High | Very High | Very High | Extremely High |
| Men: ≥ 40 in. (102 cm) | | | | | | |
| Female: ≥ 35 in. (88 cm) | | | | | | |

Table 17.6 Weight Classifications by BMI, WC, and Associated Risk

Health, n.d.). In overweight individuals, having a large waist may indicate an additional risk of health problems compared to someone with a normal waist measurement. The Nurses' Health Study, one of the largest longitudinal studies that has followed individuals to determine the association of abdominal obesity with chronic disease mortality, supports this relationship (Zhang et al., 2008). When the study began, over 44,000 healthy female volunteers enrolled. After a decade and a half, women with a WC of 35 in. or more had nearly two times the risk of death from heart disease than those with the lowest WC of <28 in.

Furthermore, the study results revealed that women with the largest waists had a higher risk of death from cancer or any cause than women with smaller waists (Zhang et al., 2008). The risk increased consistently with every inch added to the waist. Women with normal BMI were at high risk if they had more waist adiposity. Women with a BMI <24.9 and a WC of ≥35 in. had triple the risk of death from CVD compared to women with a normal

BMI and whose waist was <35 in. WC is an important indicator of disease risk that should be used in the clinical setting to inform the management of patients (see **Table 17.6**).

Obesity in Children and Adolescents

Obesity in children and adolescents is an age-gender-specific BMI ≥95th percentile. According to the USPSTF (2017), every child and adolescent is at risk for obesity and should be screened. Risk factors comprise parents who are obese, insufficient nutrition, low levels of physical activity, inadequate sleep, sedentary behaviors, ethnic minority, and families with limited resources. There is moderate evidence to support that children and adolescents who are obese should be referred to or provided comprehensive, intensive behavioral intervention. See Table 17.7 for BMI-for-age weight status categories and corresponding percentiles. The BMI percentile cutoff for obesity in children defines a level that a child is at

^{*}In individuals of South, Southeast, or East Asian ethnicity, comorbidity risk is observed at lower BMI levels and WC (e.g., Pre-obesity BMI 23-27.9, Obesity BMI > 28. men WC \geq 33 in. [\geq 85 cm], female WC \geq 31in. [\geq 74-80 cm])

| Table 17.7 Children and Adolescents Weight Status and Percentile Range | | |
|--|---|--|
| Weight Status Percentile Range | | |
| Underweight | Less than the 5th percentile | |
| Healthy weight | 5th percentile to less than the 85th percentile | |
| Overweight 85th percentile to less than the 95th percentile | | |
| Obesity 95th percentile or greater | | |
| Centers for Disease Control and Preventio | n. (2023). Defining child BMI categories. https://www.cdc.gov/obesity/childhood/defining.html | |

higher risk of developing significant obesityrelated health complications, as reflected in Figure 17.1.

Comprehensive Obesity Assessment

The assessment of obesity requires a comprehensive approach to determine the underlying factors associated with an imbalance in energy homeostasis. It is imperative that APPs not only focus on treating the consequences of obesity but also determine the contributing factors and underlying diseases that promote obesity (van der Valk et al., 2019). Assessment of people with obesity should consider multiple factors, as with any chronic illness. These include vital signs with anthropometrics, a comprehensive review of medications to identify those who are obesogenic (see Table 17.3), a detailed obesity-centered medical history (see Table 17.8), an obesity-focused physical exam (see Table 17.9), and relevant laboratory and diagnostic testing (see Table 17.10). Assessing any physical, mental, and psychosocial limitations is vital. Other recommended measures are fasting glucose or hemoglobin A1C levels, a lipid panel to evaluate for metabolic risk factors, and possibly alanine aminotransferase to screen for nonalcoholic fatty liver disease (Rueda-Clausen et al., 2020). The ultimate goal of the assessment is to determine the root causes of excess weight in a nonjudgmental manner and to develop effective management strategies that are person centered.

Lifestyle Management of Pre-Obesity and Obesity

Pre-obesity and obesity necessitate a multifaceted management plan; as such, there is no one-size-fits-all approach (Fitch & Bays, 2022). Management will depend on the underlying cause and should be individualized. This chapter will focus primarily on behavioral management that comprises the A-SMART (adopt healthy eating, stress less, move often, avoid alcohol, rest more, and treat tobacco use) lifestyle behaviors supported by the OMA (n.d.) pillars of eating habits, activity level, and behavior. The OMA pillar related to medication is beyond the scope of this chapter; however, indications for considering medications in an obesity management program when lifestyle behaviors are ineffective will be identified. Also, a person-centered strategy in conjunction with an interprofessional team is required for optimum outcomes. APPs are well suited to work with stakeholders to develop and implement strategies to manage pre-obesity and obesity that may address upstream issues to decrease the incidences of pre-obesity and obesity in the most likely affected individuals.

Pre-obesity and obesity should be managed as a long-standing chronic condition that involves an interprofessional team approach (Gonzalez-Campoy et al., 2013). Primordial,

Table 17.8 Components of an Obesity-Centered Medical History

History Component

Weight

- Age at onset of obesity and previous attempts to lose or gain weight and outcome
- Assess satisfaction with current weight

Nutrition

- Determine the ability to comprehend nutrition facts
- Assess caloric intake
- Identify dietary restrictions (gluten intolerance, vegan, vegetarian, lactose intolerance, food allergies

Physical Activity

- Determine routine physical activity habits
- Assess barriers (e.g., pain in joints, personal or environmental factors) to being active
- Assess facilitators of physical activity

Mental Health Disorders

 Conduct mental health screening (e.g., Physical Health Questionnaire-9 and General Anxiety Disorder Assessment), trauma, and hyperactivity

Substance Abuse

- Evaluate
- Tobacco use
- Alcohol use
- Opiates and street drug use
- Stimulants (e.g. coffee)
- Sweetened beverages

Abuse

Assess for physical, mental, and sexual abuse

Sleep

- Screen for adherence to recommended hours of sleep, use of prescribed or over-the-counter sleep aids
- Assess for symptoms of sleep apnea (snoring, daytime fatigue)

Medications

Identify use of medications that can increase weight

Social

- Determine shift work (e.g. night shift), support systems
- Financial support, health insurance coverage, and availability of facilities for physical activity such as parks, walking, or bike trails.
- Determine the level of functional ability (e.g., activity of daily living and instrumental activities of daily living

Family

 Determine family history of overweight/obesity or related complications (particularly, parents and siblings)

Interpersonal Assessment

Assess:

Readiness to change

For a comprehensive outline of recommended components of an obesity-centered medical history, please see: Rueda-Clausen, C. F., Poddar, M., Lear, S. A., Poirier, P., & Sharma, A. M. (2020). Assessment of people living with obesity. *The Canadian Adult Obesity Clinical Practice Guidelines*. https://obesitycanada.ca/quidelines/assessment/

Table 17.9 Obesity Focused Physical Exam

| Evaluation | Findings | Complication/Recommendations | |
|------------------|---|---|--|
| Anthropometrics | Weight, BMI, Waist Circumference, neck circumference, Mallampati score | Metabolic syndrome (WC), Obstructive Sleep Apnea | |
| Vital signs | Blood pressure, heart rate | Hypertension | |
| General Exam | Physical activity | Disability resulting from excess weight | |
| Skin | Acanthosis nigricans, hirsutism, acne Xanthelasmata | Insulin resistance, polycystic ovarian syndrome (hormonal testing) Hyperlipidemia | |
| Cardiovascular | Heart Sound (S3), heart rate and rhythm, peripheral edema | Heart Failure/consider BMI interpretation | |
| Respiratory | Wheezing, prolonged expiratory phase, rales | Asthma | |
| Gastrointestinal | Liver enlargement, firm | Non-Alcoholic fatty liver disease | |
| Musculoskeletal | High muscle mass, weakness, decreased range of motion in joints, swelling, tenderness, and crepitus in joints | Consider interpretation of BMI, osteoarthritis, sarcopenia (joint-x-ray) | |
| Endocrine | Hyper/hypothyroid findings, signs of hypercortisolism | Hypo/hyperthyroidism (TSH), Cushing disease (salivary and 24-hr urine cortisol) | |

Khattak ZE, Zahra F. Evaluation of Patients With Obesity. [Updated 2023 Apr 27]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2023 Jan-Available from: https://www.ncbi.nlm.nih.gov/books/NBK576399/

Table 17.10 Obesity-Related Laboratory and Diagnostic Test

| Laboratory and Diagnostic Tests | Complications |
|---|---|
| Fasting glucose, HbA1C, 2hrs OGTT | Prediabetes, metabolic syndrome, Diabetes |
| Lipid Profile: Total cholesterol, triglycerides, high-density lipoproteins (HDL), low-density lipoproteins (LDL), non-HDL | Dyslipidemia Metabolic syndrome Cardiovascular disease risk |
| Complete Metabolic Panel (eGFR) | Renal impairment, electrolyte abnormality |
| Liver function tests: Transamines (AST, ALT); hepatic imaging, biopsy | Non-alcoholic fatty liver disease |
| Thyroid Function Tests: TSH, T3, T4 | Hypo/hyperthyroidism |

| Table 17.11 Definition, Goals, and Methods for Phases of Prevention in Obes |
|---|
|---|

| Phase of Intervention | Definition and Goals | Method of Prevention |
|--------------------------|--|---|
| Primordial | Prevent childhood obesity. | Provide prenatal, early childhood lifestyle counseling with an emphasis on nutrition and physical activity. |
| Primary | Prevent the development of overweight and obesity. | Educate the public. Develop built environments that promote healthy behaviors (e.g. physical activity). Promote healthy eating and regular physical activity. |
| Secondary | Prevent future weight gain and the development of weight-related complications in patients with overweight or obesity. | Screen using BMI. Diagnose using BMI and evaluation for complications. Treat with lifestyle/behavior intervention ± weight loss medications. |
| Tertiary | Treat with weight loss therapy to eliminate or lessen weight-related complications and prevent disease progression. | Treat with lifestyle/behavior intervention plus weight loss medications if warranted. Consider bariatric surgery. |
| Quaternary | Avoid weight-enhancing treatments (e.g., medications that are obesogenic). | Review medications. |

Data from Garvey, W. T., Mechanick, J. I., Brett, E. M., Garber, A. J., Hurley, D. L., Jastreboff, A. M., Nadolsky, K., Pessah-Pollack, R., & Plodkowski, R. (2016). American Association of Clinical Endocrinologists and American College of Endocrinology comprehensive clinical practice guidelines for medical care of patients with obesity. *Endocrine Practice*, 22, 1–203. https://doi.org/10.4158/EP161365.GL

primary, secondary, tertiary, and quaternary prevention measures should be employed for the best outcomes (see **Table 17.11**). There is strong evidence that lifestyle behavior modifications that include physical activity, nutrition, and behavioral therapy interventions create the most effective changes related to obesity prevention and maintaining long-term weight loss (Vallis et al., 2020).

Evidence-Based Guidelines

Various organizations have developed evidencebased clinical practice guidelines for managing patients with pre-obesity and obesity, for example, the American College of Cardiology/ American Heart Association Task Force

(Jensen et al., 2014), Obesity Canada and the Canadian Association of Bariatric Physicians and Surgeons (Wharton et al., 2020), and the Department of Veterans Affairs/Department of Defense (U.S. Department of Veterans Affairs, 2020). These guidelines are extensive and readily available on the internet. Among the many weight loss treatment options, including popular diets, cutting-edge medications, and surgery, the role of lifestyle behavior change may be overlooked. However, for any weight loss treatment to be optimally effective, it must address the cognitive and behavioral factors through lifestyle management principles and techniques (Wadden et al., 2020). All the guidelines relevant to pre-obesity and obesity comprise changing behaviors that affect the

energy balance using decreased caloric intake and increased EE.

The Clinical Practice Guideline for Multicomponent Behavioral Treatment of Obesity and Overweight in Children and Adolescents (American Psychological Association, 2018) provides behavioral evidence-based recommendations for interventions to treat overweight and obesity in children and adolescents ages 2 to 18. The consensus is that managing overweight and obesity in children and adolescents should include parental or guardian involvement with a minimum of 26 contact hours of a multicomponent behavioral intervention. This has demonstrated effectiveness, mainly when initiated in young children who are pre-obese or obese. Several organizations, including the American Heart Association (Kelly et al., 2013) and the American Academy of Nutrition and Dietetics (Hoelscher et al., 2013), have published recommendations that support family involvement, dietary changes, and an increase in physical activity to decrease overweight or obesity as first-line treatment. Though the guidelines endorse medications or bariatric surgery as options for treatment, they caution that only a few drugs are approved for pediatric obesity, and bariatric surgery should be considered only for selective adolescents with psychological and medical complications resulting from obesity. Furthermore, the USPSTF (2017) found inadequate evidence regarding medication management in this age group. A discussion regarding medications and surgery for the treatment of obesity is beyond the scope of this chapter.

Starting the Conversation

Conversations about weight can be challenging for patients and providers because weight is a sensitive and complex issue. APPs must use people-first language to avoid labeling patients by their weight status, which promotes bias (Gallagher et al., 2021). The term patient with obesity is preferred to obese

patient because it decreases stigma and may positively influence the patient-provider relationship. Providers often cite barriers to discussing obesity with patients, such as a lack of comfort with starting the conversation, lack of training, and insufficient tools to help patients recognize obesity (Petrin et al., 2017). Despite providers' barriers to discussing weight, patients expect their providers to guide them regarding weight loss strategies. Adult patients who receive overweight or obesity counseling from primary care providers are four times more likely to make an effort to lose weight than patients who do not get weight management guidance (Rose et al., 2013). However, three-fourths of adults with pre-obesity and half of those with class 1 obesity have not received weight management counseling from a provider (Guglielmo et al., 2018). Guidance on effective conversation starters regarding pre-obesity and obesity in the clinical setting may help to overcome these barriers.

To address this issue, organizations have developed guidelines to enhance effective conversations with patients to treat obesity. Recently, the Strategies to Overcome and Prevent (STOP) Obesity Alliance (Gallagher et al., 2021) convened a dozen representatives from primary care and obesity professional organizations, such as the American Association of Nurse Practitioners and the American Academy of Physician Assistants, to develop Weight Can't Wait: Guide for the Management of Obesity in the Primary Care Setting (STOP Obesity Alliance, 2020), which offers guidance for primary care providers to speak with patients about weight. The guide provides practical approaches to caring for patients with obesity in the pre-encounter, encounter, and postencounter period of a primary care visit. The collection of information to start a weightrelated conversation happens in the preencounter period. During the pre-encounter, providers obtain patients' permission to discuss their weight, address weight bias, diagnose

Health Promotion Research Study

Whole-Food Plant-Based Lifestyle Program and Obesity

Background: Most weight loss strategies are adequate for the short term but fail to sustain weight loss. This study aimed to determine whether a whole-food plant-based (WFPB) lifestyle program would result in long-term weight loss.

Methodology: Researchers examined the obesity measures for 151 healthy community-living adults who participated in an ongoing study over a short (0.5 up to 2 years), medium (2 years to up to 5 years), or long (5 to 10 years) time frame. The WFPB lifestyle program included nutrition education, a physical activity component, and support.

Results: The body composition changes were favorable for all time frames and genders. For all participants, there were significant mean decreases in BMI (-2.5 kg/m²), body mass (-7.1 kg), and percent body fat (-6.4%; p < .001 for all). Participants with BMI within the normal range increased from baseline to 16.5% for females and 26.2% for males. For each of the three time frame groups, the number of participants in the normal BMI range increased by 25% (short), 16% (medium), and 16% (long). The proportion of participants whose body fat percentage decreased to normal was 20%, 39%, and 18% for the short, medium, and long time frame groups, respectively. Females had more significant decreases in all indices in the long-term versus short-term program. Most (85.6%) children 18 years and under whose parents were in the program adopted the WFPB lifestyle compared to only 28% of adult children. For females, energy intake was 1,842 \pm 539 calories per day, and males consumed 2,618 \pm 726 calories per day. Overall, participants were physically active, with good sleep quality and low perceived stress.

Implications for Advanced Practice: Overall, the effect of a WFPB lifestyle program on obesity measures demonstrated a significant decrease in BMI, body mass, and body fat in the individuals who participated in the short, medium, and long time frame groups. Females had more favorable outcomes in the long-term program compared to the short-term program. This study is unique because the beneficial effects were realized within the initial 2 years and sustained over the long term (5–10 years). Additionally, most of the participants' offspring adopted healthy eating and physical activity habits, which may ultimately decrease the incidence of obesity in the next generation. APPs should assist patients ready to lose weight in planning a multicomponent program that includes WFPB foods, physical activity, and a support group for sustainable weight loss.

Reference: Jakše, B., Jakše, B., Pinter, S., Pajek, J., & Fidler Mis, N. (2020). Whole-food plant-based lifestyle program and decreased obesity. *American Journal of Lifestyle Medicine*, 16(3), 1–11. https://doi.org/10.1177/1559827620949205

obesity, and include patients in decision-making (Gallagher et al., 2021).

Further, this guide includes a modified 5As (assess, advise, agree, assist, arrange) counseling framework by adding a sixth *A* (ask) to underscore that seeking permission from the patient to begin the conversation about weight is paramount to enhance the patient–provider

experience (see **Table 17.12** for features of the 6As counseling framework). The patients' decision to pursue treatment for their weight determines the *post-encounter* clinic visit. If there is interest, the APP provides patients with a plan summary and schedules a return visit to assess progress. If there is no interest in pursuing treatment for weight, the APP respects the

| Table 17.12 6As Counseling Framework | | | |
|--------------------------------------|--|--|--|
| 6As | Features | | |
| Ask | The APP asks permission to discuss weight, actively listens, acknowledges concerns, and includes the term preferred by patients to discuss their weight (e.g., overweight, unhealthy weight, elevated BMI). | | |
| Assess | The APP reviews the prescreen data (e.g., BMI, 24-hour diet recall, weight trajectory), assesses weight-related health concerns, ascertains a weight-centered history (see Table 17.8), and conducts an obesity-centered physical exam (see Table 17.9). | | |
| Advise | If patients are interested in discussing weight, the APP starts the shared decision-making process to establish the next steps. If patients are not interested in discussing their weight, the APP should respect their decision and express an openness in discussing the issue in the future (assess readiness at each encounter). | | |
| Agree | If patients agree to having a conversation about weight, then their queries and needs prompt the APP's responses. The APP collaborates with patients in specific, measurable, attainable, relevant, and time-bound (SMART) goal setting. | | |
| Assist | The APP assists patients by presenting treatment options, including intensive lifestyle intervention and referral to the interprofessional healthcare team as needed (registered dietitian, licensed social worker, certified lifestyle medicine or obesity medicine providers, and behavioral health specialist). The APP should refer as appropriate for treatment options such as anti-obesity medications and bariatric surgery. | | |
| Arrange | The APP arranges the coordination of care as appropriate. Providers unable to deliver intensive lifestyle medicine intervention that leads to significant weight loss should refer patients, organize care, and provide follow-up as needed. | | |

Data from Gallagher, C., Corl, A., & Dietz, W. H. (2021). Weight can't wait: A guide to discussing obesity and organizing treatment in the primary care setting. Obesity, 29(5), 821–824. https://doi.org/10.1002/OBY.23154

patients' decision and specifies availability for future conversations. **Figure 17.4** represents an algorithm with a suggested provider script to counsel patients with obesity. The guide can be found online and offers practical approaches for starting the conversation that will lead to effective discussions and management of patients with obesity.

Further tips for starting the conversation about weight in the primary care setting are provided by the National Institute of Diabetes and Digestive and Kidney Diseases (2017) and the STOP Obesity Alliance (2020). An abbreviated list of conversation-starter questions and clinician tips can be found in **Table 17.13**. For a detailed list of questions to consider

when discussing weight with patients, refer to Weight Can't Wait: Guide for the Management of Obesity in the Primary Care Setting (STOP Obesity Alliance, 2020).

Options for Stages of Obesity Treatment

All evidence-based clinical practice guidelines for managing patients with pre-obesity and obesity use lifestyle behavior as the first line of treatment. However, lifestyle treatment options alone may not be effective for obese patients experiencing obesity-related chronic diseases. Nonetheless, no treatment options can be effective without lifestyle measures. The

The Patient Encounter

Pre-screen: BMI and weight trajectory; 24h dietary recall; personal weight history; medications; physical activity; existing comorbidities or risk factors; stress; sleep; QOL; depression.

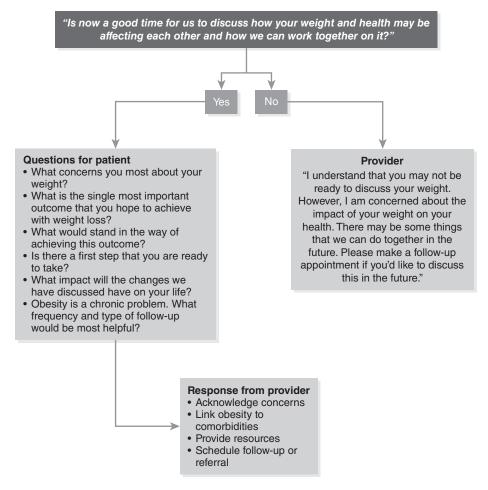


Figure 17.4 Sample questions for the patient encounter.

Edmonton Obesity Staging System (Sharma & Kushner, 2009) provides a seminal framework to guide treatment options for obesity. It considers BMI, psychopathology, metabolic and physical comorbidities, and complications in treating obesity (Sharma & Kushner, 2009). It is a simple clinical staging system that allows clinicians to consider factors beyond BMI associated with excess weight and provides

guidance for treatment measures. **Figure 17.5** lists the description and management for each stage from zero to 4.

Establishing Weight Loss Goals

The APP should work with patients to create realistic and achievable goals to help realize

| Table 17.13 Conversation-Star |
|-------------------------------|
|-------------------------------|

| Questions | Clinician Tips | | |
|--|---|--|--|
| "How are you feeling about your weight currently?" "May I discuss your health with you, including your weight?" | Before initiating screening or assessing obesity, ask patients for permission to discuss their weight and determine anthropometric values. Before discussing their weight, discuss the health issues related to excess weight. Patients prefer terms such as weight (first) or BMI (second) and are opposed to the terms fatness, excess fat, and obesity. Be sensitive to variations in cultural differences about weight, patterns of food intake, and practices. | | |
| "Are you concerned about your weight at this time?" | If patients are not interested in addressing weight, address other health concerns and ask if you can speak with them about their weight at another time. | | |
| "Would you be interested in changing lifestyle behaviors to improve your health?" | Determine readiness to begin making the change. | | |
| "I would like to learn more about your current lifestyle behaviors. What do you eat and drink on a typical day for breakfast, lunch, and dinner?" | Take an inventory of any food, drink, or snack over the past 24 hours. | | |
| "What does 'healthy eating' mean to you?" | Helps to determine person-centered care | | |
| "Do you eat only when hungry or for other reasons, such as feeling stressed or bored?" | Identifies emotional eating so interventions can be individualized | | |
| "When is the best time of day or evening for you to be active?" | Start with the health benefits of physical activity consistently, even for a short period. | | |
| "What types of activities do you enjoy? Do you prefer doing activities alone, with someone else, or in a group?" | Intervention can be customized based on group or individual activities. | | |
| "How much time do you spend sitting? Would you want to work some physical activity into your day?" | Identify sedentary behaviors and readiness for physical activity. | | |

National Institute of Diabetes and Digestive and Kidney Diseases. (2017). Talking with patients about weight loss: Tips for primary care providers. https://www.niddk.nih.gov/health-information/professionals/clinical-tools-patient-management/weight-management/talking-adult-patients-tips-primary-care-clinicians

sustainable weight loss efforts (Vallis et al., 2020). Goals that aim to improve overall health, functionality, and quality of life, not just decreased weight, will promote long-term behavior changes. Assessing patients' reasons for losing weight and their goals allows the

APP to understand where to shift the focus of the conversation, identify specific challenges related to their goals, and provide encouragement when there is progress toward meeting the goals. Because obesity is a chronic condition, it will require lifelong management.

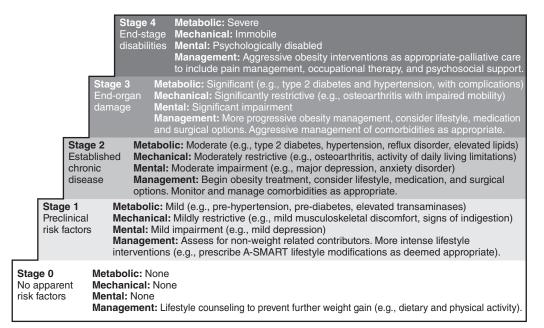


Figure 17.5 Edmonton Obesity Staging System

Health Promotion Case Study: Obesity

Case Description: C.M. is a 42-year-old male with the chief complaint of weight gain. One month ago, he was seen in the clinic for a routine physical and blood work and was noted to have increasing lipids. He was counseled on lifestyle modifications and is seeking more counseling. He has tried multiple diets and supplements in the past and has been unable to lose significant weight or keep it off. He is seeking assistance for weight loss to have more energy and to keep up with his kids for years to come.

- Past medical history: Gastroesophageal reflux disease, prediabetes, hyperlipidemia.
- Medications: Famotidine 20 mg daily, Lipitor 10 mg daily.
- Allergies: No known allergies.
- Social history: Works during the day as a realtor. Divorced with two children and one dog.
- Vital signs: BP 118/74 mmHg left arm, heart rate 76 bpm, respirations 16 bpm, temperature 98.4°F, O₂ saturation 98% on room air, weight 287.7 lb, height 70 in., BMI 41.28 kg/m².
- Review of systems: He is currently asymptomatic with occasional reflux, especially after large meals and dining out. All other systems are negative.
- Physical exam: The physical exam was unremarkable.
- Labs: Recent blood work last month. CBC is unremarkable; CMP with elevated glucose at 160 mg/dL, otherwise unremarkable; hemoglobin A1C 5.8%; total cholesterol 237 mg/dL; triglycerides 439 mg/dL; high-density lipoprotein 34 mg/dL; very-low-density lipoprotein 78 mg/dL; low-density lipoprotein 125 mg/dL; thyroid-stimulating hormone 1.230 uIU/mL.

(continues)

Health Promotion Case Study: Obesity

(continued)

Lifestyle Vital Signs

- Adopt healthy eating: He eats a lot of meat, dairy, and eggs and gets takeout several nights a week, as he does not have much time to cook. He eats fruits and vegetables once a day. He drinks 3-4 sodas a week.
- Stress less: States work is stressful at times.
- Move often: He recently joined a gym and started exercising for 30 minutes twice a week, a combination of walking and weight lifting.
- Avoid alcohol: He drinks 2–3 alcoholic drinks on the weekend and does not use drugs.
- Rest more: Not assessed.
- Treat tobacco use: He has never smoked.

Critical Thinking Questions

- Based on Table 17.8, what additional medical history do you want to obtain, and why?
- Are there any additional lifestyle questions you want to ask?
- Based on Table 17.9, what additional objective data do you want to assess, and why?
- What additional laboratory data are needed, if any?
- What lifestyle management strategies would you suggest for weight loss based on the A-SMART lifestyle behaviors?
- Write person-centered A-SMART lifestyle prescriptions using SMART goal setting.
- Find research or evidence-informed quidelines to support lifestyle management decisions.

Recommendation: Use the 6As counseling framework (Gallager et al., 2021), for example, *ask* for permission to discuss weight; *assess* pre-encounter data, weight-centered history, and weight-centered physical exam; *advise* as appropriate; *agree* on treatment using goal-setting strategies; *assist* using A-SMART lifestyle behaviors as a guide; and *arrange* follow-up.

Helping patients realize this will allow them to reframe their mindset to create sustainable lifestyle modifications, behaviors, and habits so as not to increase the risk of regaining weight with short-term fixes (Vallis et al., 2020).

Patients often wonder what their ideal weight should be, which is commonly determined using the BMI scale. Because BMI does have limitations, it is suggested to use the concept of *best weight*, which is the weight that patients can attain and maintain while still living their healthiest and most enjoyable life. The weight loss journey can be a period of discovering a person's best weight (Vallis et al., 2020).

Identifying a realistic weight loss goal is essential when clinicians have determined that patients are ready to make lifestyle changes to attain these goals. Generally, a 1 to 2 lb weight loss per week is expected, with a goal of 5% to 10% weight loss from baseline within 6 months to reduce risk factors. However, with weight reduction more substantial than 1 to 2 lb per week, there is a more significant benefit, but patients should be encouraged that even a 5% weight loss can achieve health benefits (Jensen et al., 2014). For example, a goal for a patient with a starting baseline weight of 200 lb would be to lose 10 lb (5%) to 20 lb (10%) within 6 months. These benefits include reduced CVD risk factors, improved lipid profiles, reduced hemoglobin A1C, reduced risk of developing T2D, and reduced risk of obesity-related complications (Lau & Wharton, 2020).

Strategies for Weight Loss

A successful weight loss program is multifaceted and includes lifestyle and psychological strategies. Interventions to adopt A-SMART lifestyle behaviors will enhance weight loss. The first three behaviors (adopt healthy eating, stress less, and move often) will be highlighted in this section. See **Table 17.14** for a summary of the A-SMART action plan for obesity management. In particular, an individualized weight

loss or management program that includes both a reduction in calories and an increase in physical activity has been demonstrated to be more effective than only calorie reduction or physical activity (U.S. Department of Health and Human Services [HHS], 2018; Vallis et al., 2020). Psychological strategies that will ensure successful outcomes include setting SMART goals, self-monitoring (using a journal to track food intake, physical activity, and weight), and problem-solving (Vallis et al., 2020). Effective weight management interventions will require an interprofessional healthcare team.

Table 17.14 A-SMART Recommendations with Grade and Level of Evidence

| Lifestyle Modification | Recommendation | Grade of Evidence |
|---------------------------|---|--|
| Adopt healthy eating | Medical nutrition counseling for overweight and obesity should aim to decrease fat mass and correct adipose tissue dysfunction. Include families, particularly with children. The goal of weight loss is 5–10% annually until the ideal body weight is achieved. | Grade A Best Evidence Rating Level (BEL) = 1 |
| Stress less | Spend time in recreation or play, stress reduction, and happiness, including using social support and developing coping skills for overall disease prevention. | Grade A BEL = 1 |
| Move often | Implement a lifestyle intervention to obtain a 7% weight loss and a minimum of 150 minutes per week or the equivalent of 150 minutes of aerobic activity to decrease obesity-related diabetes. | Grade A BEL = 1 |
| Avoid alcohol | Advise patients to avoid alcohol. But for those who consume alcohol, if they are of legal age and without contraindications to alcohol, the guidelines recommend daily intake of ≤ 1 drink (0.6 oz of pure alcohol) for women and ≤ 2 drinks for men. Alcohol should not be increased for any supposed beneficial effect. | Grade D BEL = 4 |
| Rest more | Advise patient to obtain at least 6 hours of sleep nightly for disease prevention (ideally 7–8 hours/night). Encourage sleep hygiene practices. | Grade A BEL = 1 |
| Treat tobacco use | Provide treatment for tobacco use. Tobacco use may not contribute to obesity, but it is well established that avoiding it supports a healthy lifestyle. | Grade not available |

Data from Gonzalez-Campoy, J. M., St. Jeor, S. T., Castorino, K., Ebrahim, A., Hurley, D., Jovanovic, L., Mechanick, J. I., Petak, S. M., Yu, Y. H., Harris, K., Kris-Etherton, P., Kushner, R., Molini-Blandford, M., Nguyen, O. T., Płodkowski, R., Sarwer, D. B., & Thomas, K. T. (2013). Clinical practice guidelines for healthy eating for the prevention and treatment of metabolic and endocrine diseases in adults: Cosponsored by the American Association of Clinical Endocrinologists/the American College of Endocrinology and The Obesity Society. *Endocrine Practice*, *19*(SUPPL. 3), 1–82. https://doi.org/10.4158/FP13155.61

Nutritional Strategies for Weight Loss

Individuals living with obesity should receive person-centered medical nutrition therapy (MNT), preferably from a dietitian or provider with experience in obesity management, to optimize weight outcomes (Brown et al., 2022). There is a myriad of evidence-based MNTs effective in improving health outcomes as described in the Canadian Adult Obesity Clinical Practice Guidelines: Medical Nutrition Therapy in Obesity Management (Brown et al., 2022). Overall, there is no one-size-fits-all eating approach for weight management. It should be noted that the best dietary pattern for managing weight is one that can be sustained over the long term. Therefore, APPs must consult with patients to develop an individualized eating plan because effective weight loss and maintenance depend on patients' long-standing adherence to a healthy nutritional regimen (Kim, 2021).

Nutritional strategies for weight loss depend on multiple factors, including the pattern, time, quality, and quantity of food intake (Kim, 2021). Reduced caloric intake was identified as the fundamental and most important weight loss factor in the scientific literature (Jenson et al., 2014; Kim, 2021). Further considerations to optimize weight loss are avoiding late-night eating and eating the largest meal of the day at breakfast (Kim, 2021). Additionally, guidelines recommend daily energy intake lower than needed for energy balance, usually 1,200 to 1,500 calories for women and 1,500 to 1,800 calories for men as well as an energy deficit of 500 to 750 calories per day, or an energy deficit of 30%, and approaches as needed to decrease caloric intake by restricting or eliminating particular foods or prescribing selected foods such as whole-plant foods, many of which are naturally lower in calories (Jensen et al., 2014).

In a recent review to determine the best dietary plan for weight loss, the Mediterranean diet, which emphasizes vegetables, fruits, whole grains, and healthy fats, was the most reliable for weight loss (Dinu et al., 2020). This was in comparison to low-fat, high-protein, intermittent energy restriction and vegetarian eating plans. Accordingly, the American College of Lifestyle Medicine (n.d.) recommends eating primarily a variety of minimally processed vegetables, fruits, whole grains, legumes, nuts, and seeds to treat, reverse, and prevent lifestyle-related chronic diseases, such as obesity. Further, patients in the initial stages of adapting to a plant-based dietary plan to decrease weight and improve health outcomes may find it more amenable to sustain adding healthy foods rather than avoiding foods. APPs may use the free online Full Plate Living Nutrition Program to teach the principle of adding high-fiber plant-based foods to increase satiety and ultimately manage weight (Full Plate Living, n.d.).

Stress and Weight Management

Stress management and coping skills should be included in a program to treat individuals with excess weight. A small, randomized controlled trial that included a stress management component to a lifestyle intervention on weight loss revealed successful outcomes after an 8-week program (Xenaki et al., 2018). The intervention had relaxation breathing exercises, progressive muscle relaxation techniques, and guided visualization, all of which decreased perceived stress and depression and improved dietary habits. Patients should be encouraged to assess for emotional triggers that spark the urge to eat even when they may not be physiologically hungry and put cues in place to eat mindfully to avoid overeating. Practical tips to offer patients regarding mindful eating include eating when hungry but before being voraciously hungry, avoiding distractions while eating, breathing deeply five times before starting a meal, eating slowly, taking small bites, stopping eating when feeling full, and having healthy snacks available when leaving home (Daniel, 2018). It is well known that physical activity can alleviate stress and enhance weight loss. Therefore, physical activity is a crucial component of a weight loss program.

Physical Activity and Weight Loss and Maintenance

Physical activity or exercise is essential to achieving and maintaining weight loss goals, particularly in conjunction with other strategies. A review of studies by Cox (2017) to determine the role of physical activity in weight management found that individuals were more likely to lose weight and sustain weight loss if they participated in physical activity for at least 1 hour per day. This finding is in keeping with the recommendation from the 2018 Physical Activity Guidelines (HHS, 2018) that some individuals will need 300 minutes or more per week of physical activity to lose weight. Further, a systematic review (Jakicic et al., 2019) expanded on the evidence that informed the development of the 2018 Physical Activity Guidelines and found that physical activity above 150 minutes of moderate to vigorous intensity per week tempered weight gain in adults. The review indicated that obesity was significantly decreased in a graded manner based on the incremental increase in vigorous physical activity per week. The effect of the association of physical activity on obesity in adults varied by age—as age increased, the effect of physical activity on obesity decreased.

Weight loss as a result of physical activity alone may be insufficient for individuals who are obese (Jakicic et al., 2019). Nevertheless, APPs should routinely assess

physical activity and guide patients who can be physically active to integrate intentional movement into their routine behaviors to attenuate weight gain. For weight loss, individuals should do at least 150 to 300 minutes of moderate-intensity or 75 to 150 minutes of vigorous-intensity physical activity per week or a comparable mixture of moderate-and vigorous-intensity aerobic physical exercise (HHS, 2018). However, some movement is better than none (Bays et al., 2021). Patients should be encouraged to include resistance training as an adjunct to their overall physical activity habits but not as the sole activity for weight management.

Multicomponent Interventions for Overweight and Obesity

The Look AHEAD (Action for Health in Diabetes) landmark study particularly underscores the effect of a multicomponent approach to weight management (Look AHEAD Research Group, 2014). Look AHEAD is a randomized controlled trial with over 5,000 individuals with obesity and diabetes. The intensive lifestyle intervention group was prescribed 1,200 to 1,800 calories daily, with no more than 30% of calories from fat (less than 10% from saturated fat) and no less than 15% from protein. Also, participants in the study were prescribed at least 175 minutes of unsupervised moderate-intensity physical activity per week and individual and group counseling at varying intervals. In year 1, participants in the intensive lifestyle intervention had an average

Health Promotion Activity: Assess Edmonton's Obesity Stage and Genetic Risk

Reread the obesity case study of C.M.

- Calculate the obesity risk index score, and determine the Edmonton Obesity Staging System stage for C.M.
- What additional information is needed from C.M. to complete the Genetic Obesity Risk Index Screening Questionnaire?

weight loss from baseline of $8.5\% \pm 0.2\%$ compared with $0.6\% \pm 0.2\%$ for the comparison group. Even at 8 years, half of those who followed the dietary and physical activity prescriptions had at least a 5% decrease in weight (Look AHEAD Research Group, 2014). Those who achieved the weight loss goals increased physical activity, decreased caloric intake, monitored their weight, and attended counseling sessions.

Interprofessional Approach

Obesity is a chronic systemic condition that requires an interprofessional approach (Bischoff et al., 2017) for prevention and management to ensure the best possible long-term outcome. The root causes of overweight and obesity are multifactorial, as are the treatment approaches and referral needs. The interprofessional healthcare team members who may be required to address the many components of pre-obesity and obesity include psychiatrists, psychologists, behavioral therapists, registered dieticians, exercise physiologists, endocrinologists, health coaches, and bariatric surgeons. A thorough history and physical exam may also reveal comorbidities or obesity-related complications that could require referral to cardiology, endocrinology, dermatology, orthopedist, sleep specialists, physical therapists, occupational therapists, physiotherapy, and other medical specialists (Bischoff et al., 2017).

The APP should consult with a specialist in obesity medicine and an interprofessional team if patients cannot achieve significant and long-term weight loss with behavior modifications alone. Weight loss medications are recommended for those with a BMI of at least 30 or a BMI of 27 with at least one obesity-related comorbidity (Jensen et al., 2014). Bariatric surgery is considered for those with a BMI of at least 40 or a BMI of 35 with obesity-related comorbidities and no response to lifestyle interventions.

Summary

Pre-obesity and obesity are serious public health threats related to energy imbalance that results in excess adiposity. In particular, obesity is not just a disease but also a risk factor for many leading causes of disability, death, and chronic conditions, such as cardiac disease, T2D, osteoarthritis, cancer, and mental disorders. Overall, excess weight can be caused by physiological (genes, epigenetics, brain-gut axis, neuroendocrine conditions, medications, and the gut microbiome), behavioral (high-calorie intake, physical inactivity, alcohol use, former smokers, and inadequate sleep), and environmental (food abundance, food insecurity, built environment, socioeconomic status, culture, and bias) factors. The multifactorial causes of pre-obesity and obesity require an interprofessional team and a comprehensive approach that includes an extensive history and a physical exam to determine the underlying factors associated with the imbalance in energy homeostasis used to guide treatment. The BMI and waist measurements are objective adiposity measurements and should be included routinely in the clinical setting to diagnose and manage treatment progress. Conversations about obesity are complex, and clinicians should use evidence-informed counseling tactics that include first-person language and seek permission to discuss weight. This will ultimately decrease the risk of bias and enhance person-centered treatment plans that likely lead to adherence. Practical weight management includes evidence-informed approaches such as the A-SMART lifestyle behaviors, emphasizing a person-centered approach that supports a plant-based diet and increased physical activity. Finally, since obesity is a multifactorial chronic disease, it will require multicomponent treatments and an interprofessional team of healthcare providers to attain sustainable weight loss goals. See Table 17.15 for evidence-based resources for assessing, diagnosing, and treating pre-obesity and obesity.

TABLE 17.15 Evidence-Based Resources for Pre-Obesity and Obesity

| Resources | URL |
|---|---|
| Alcohol Calorie Calculator | https://www.rethinkingdrinking.niaaa.nih .gov/Tools/Calculators/calorie-calculator .aspx |
| American Association of Clinical Endocrinologists and American College of Endocrinology Clinical Practice Guidelines for Medical Care of Patients with Obesity | https://www.endocrinepractice.org/article /S1530-891X(20)44630-0/fulltext |
| 2013 AHA/ACC/TOS Guideline for the Management of Overweight and Obesity in Adults | https://www.ahajournals.org/doi/epub /10.1161/01.cir.0000437739.71477.ee |
| BMI Calculator | https://www.hepatitisc.uw.edu/page/clinical -calculators/bmi |
| Canadian Adult Obesity Clinical Practice Guidelines | https://obesitycanada.ca/guidelines/chapters |
| Clinical Practice Guideline for the Treatment of Obesity and Overweight in Children and Adolescents | https://www.apa.org/obesity-guideline |
| Fibrosis-4 Calculator | https://www.hepatitisc.uw.edu/page/clinical -calculators/fib-4 |
| Weight Can't Wait: Guide for the Management of Obesity in the Primary Care Setting | https://stoppublichealth9.drupal.gwu.edu /sites/g/files/zaxdzs4356/files/2022-02/wcw -guide-for-the-management-of-obesity-in-the -primary-care-setting.pdf |
| National Weight Control Registry | http://nwcr.ws/default.htm |
| Medical Nutrition Therapy in Obesity Management | https://obesitycanada.ca/wp-content/uploads/2022/10/Medical-Nutrition-Therapy_22_FINAl.pdf |
| VA/DoD Clinical Practice Guideline for the Management of Adult Overweight and Obesity | https://www.healthquality.va.gov/guidelines/CI /obesity/VADoDObesityCPGFinal5087242020 .pdf |

Acronyms

6As: ask, assess, advise, agree, assist, arrange

APP: advanced practice provider

A-SMART: adopt healthy eating, stress less, move often, avoid alcohol, rest more, and treat tobacco use

tobacco usc

BMI: body mass index

CVD: cardiovascular disease **DNA:** deoxyribonucleic acid **EE:** energy expenditure

FTO: fat mass and obesity-associated gene **HHS:** U.S. Department of Health and Human

Services

HIV/AIDs: human immunodeficiency virus/

acquired immunodeficiency syndrome

HTN: hypertension

MNT: medical nutrition therapy

NHLBI: National Heart, Lung, and Blood

Institute

OMA: Obesity Medicine Association

RMR: resting metabolic rate

SMART: specific, measurable, attainable, rele-

vant, time-bound

STOP: Strategies to Overcome and Prevent

T2D: type 2 diabetes

USPSTF: U.S. Preventive Services Task Force

WC: waist circumference **WFPB:** whole food plant based

WHO: World Health Organization

WHR: waist-hip ratio

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A pooled analysis of 2416 population-based measurement studies in 128.9 million children, adolescents,
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