<table>
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<th>Why Can’t My Patient Lose Weight? - Effective Obesity Management in Primary Care</th>
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Main

Obesity is a leading cause of preventable morbidity and mortality in Canada. One-quarter of adult Canadians and one-in-10 children now have clinical obesity.1

Weight-related comorbidities can involve all organ systems and include hypertension, type 2 diabetes, dyslipidemia, musculoskeletal disorders, cancer, sleep apnea, chronic pain, depression, and dementia. Additionally, the social stigma associated with obesity has a profound burden on affected individuals, including employment, health status, health care, and education inequities. Much of this stigma is based in the misconception that individuals with obesity are lazy, unmotivated, or lack self-control.1

Effective management of obesity is essential to primary care. This eCME program provides a framework for the management of obesity, which is demonstrated through case-based scenarios in two vignettes. Each vignette is eligible for 1.0 MAINPRO credit.

Learning Objectives:

After completion of this program, the participant will be able to:

- Assess the root causes of obesity in patients
- Identify barriers to weight loss and assist patients in overcoming them
- Individualize treatment of obesity
Vignette Selection

1. Vignette 1: Identifying the Root Causes of Obesity
2. Vignette 2: Individualizing Obesity Management

Vignette 1: Identifying the Root Causes of Obesity

This vignette will explore the complex and multi-faceted underlying causes of obesity that can occur in individuals, and will help guide practitioners in beginning the process of addressing obesity in their patients.

Learning Objectives:

After completion of this learning module, the participant will be able to:

- Initiate a conversation about body weight with their patient
- Identify the root causes of obesity in their patient
- Make recommendations to the patient for managing their weight

Discussion Forum

Reflecting on your own patients:

1. What methods do you currently use to manage overweight and obesity in the patients in your practice?
2. How effective are the methods you currently use to manage overweight and obesity in the patients in your practice? Why?
3. What complications of obesity do you most commonly encounter?
Obesity in Primary Care
Obesity is a growing problem in primary care, as more than half of adult patients now have overweight or obesity, and obesity-related comorbidity is on the rise.\(^2\)

Obesity is a chronic, progressive disease requiring lifelong management. Thus, primary care providers play an important role in preventing and managing obesity.\(^3,4\) Current approaches to obesity management focus on behavioural interventions that address core drivers of obesity, in conjunction with pharmaceutical intervention (with orlistat or liraglutide), or bariatric surgery.

Even modest weight-loss can produce clinically significant metabolic improvements, and has the potential to substantially reduce risk of comorbidities.\(^5,6\) However, a significant care gap exists; obesity is not being managed effectively in primary care to manage body weight over the long term and improve health outcomes.\(^7\) Primary care providers have profound potential to change the course of this trend.

Although there are clinical guidelines for obesity management, many practitioner-based barriers have been identified, including:\(^4,8–12\)

- low self-efficacy (the healthcare provider’s perception of their own influence on health and nutrition of their patients, their capacity to provide nutrition education, and their ability to successfully treat overweight and obese individuals)
- poor education on obesity in medical school
- perceived time constraints
- organizational limitations
- perceptions that interventions are ineffective
- difficulty of broaching the topic with the patient

Significant weight bias and stigma exist within the healthcare system, which also presents a barrier to appropriate and individualized treatment for patients living with obesity. In this program, we will examine a few of the myths that underlie this stigma.
**Obesity Definition**

The World Health Organization (WHO) defines overweight and obesity as abnormal or excessive body fat accumulation that may impair health. These are classified (see Table 1) using the body mass index (BMI), which is an adult person’s weight in kilograms divided by the square of his height in meters (kg/m$^2$):$^2$

- $\text{BMI} \geq 25 = \text{overweight}$
- $\text{BMI} \geq 30 = \text{obesity}$

**Table 1: WHO BMI Categories$^{13}$**

<table>
<thead>
<tr>
<th>BMI (kg/m$^2$)</th>
<th>Class</th>
</tr>
</thead>
<tbody>
<tr>
<td>Below 18.5</td>
<td>Underweight</td>
</tr>
<tr>
<td>18.5–24.9</td>
<td>Normal weight</td>
</tr>
<tr>
<td>25.0–29.9</td>
<td>Overweight</td>
</tr>
<tr>
<td>30.0–34.9</td>
<td>Obesity class I</td>
</tr>
<tr>
<td>35.0–39.9</td>
<td>Obesity class II</td>
</tr>
<tr>
<td>Above 40</td>
<td>Obesity class III</td>
</tr>
</tbody>
</table>

BMI provides only a rough estimate of health-related risks, and needs to be interpreted in the context of other clinical parameters. Rather than setting weight goals, interventions should focus on improving overall health and well-being.
Body Fat Distribution

Although weight status is commonly assessed using the BMI, the heterogeneous distribution of body fat is an important factor in the health-related outcomes of obesity. Central obesity is a specific concern for obesity-related health risks. This is thought to be due to the presence of increased visceral fat stored around the body organs, which exerts hormonal and metabolic activity.14

Central obesity has been found to be a greater risk factor for cardiovascular disease, diabetes, and dementia than total body obesity.15 Table 2 shows the increased-risk categories for waist circumference in men and women.16

Table 2: Risk Cut-offs for Waist Circumference16

<table>
<thead>
<tr>
<th></th>
<th>Increased Risk</th>
<th>Substantially Increased Risk</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male*</td>
<td>More than 94 cm (37 inches)</td>
<td>More than 102 cm (40 inches)</td>
</tr>
<tr>
<td>Female*</td>
<td>More than 80 cm (31.5 inches)</td>
<td>More than 88 cm (35 inches)</td>
</tr>
</tbody>
</table>

*Some ethnic groups or people living with other risk factors may have increased risk even at lower waist circumference measurements.

Edmonton Obesity Staging System

The Edmonton Obesity Staging System (EOSS) (see Table 3) delineates 5 stages of obesity (0 through 4) based on the impact that excess weight is having on the patient’s physical, mental, and functional health. The stages describe the presence of weight-related risk factors, comorbidity, and function, for a better understanding of the weight-related effects on an individual, with corresponding management approaches.17 EOSS has been shown to be a better predictor of long-term mortality than BMI or waist circumference.18

Table 3: The Edmonton Obesity Staging System (EOSS)

<table>
<thead>
<tr>
<th>EOSS Stage</th>
<th>Description</th>
<th>Suggestions for Management</th>
</tr>
</thead>
<tbody>
<tr>
<td>STAGE 0: no apparent risk factors</td>
<td>Patient has no apparent obesity-related risk factors (e.g., blood pressure, serum lipids, fasting glucose, etc. within normal range), no physical symptoms, no psychopathology, no functional limitations and/or impairment of well-being.</td>
<td>Identification of factors contributing to increased body weight. Counselling to prevent further weight gain through behavioural measures including healthy eating and increased physical activity.</td>
</tr>
<tr>
<td>STAGE 1: pre-clinical risk factors present</td>
<td>Patient has obesity-related subclinical risk factor(s) (e.g., borderline hypertension, impaired fasting glucose, impaired glucose tolerance, etc.)</td>
<td>Investigation for other (non-weight-related) contributors to risk factors. More intense behavioural interventions, including diet and exercise to prevent...</td>
</tr>
<tr>
<td>(e.g., impaired fasting glucose)</td>
<td>elevated liver enzymes, etc.), mild physical symptoms (e.g., dyspnea on moderate exertion, occasional aches and pains, fatigue, etc.), mild psychopathology, mild functional limitations and/or mild impairment of well-being.</td>
<td>further weight gain. Monitoring of risk factors and health status.</td>
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<tr>
<td><strong>STAGE 2: established comorbidity</strong></td>
<td>Patient has established obesity-related chronic disease(s) (e.g., hypertension, type 2 diabetes, sleep apnea, osteoarthritis, reflux disease, polycystic ovary syndrome, anxiety disorder, etc.), moderate limitations in activities of daily living and/or well-being.</td>
<td>Initiation of obesity treatments including consideration of all behavioral, pharmacological and surgical treatment options. Close monitoring and management of comorbidities as indicated.</td>
</tr>
<tr>
<td><strong>STAGE 3: end-organ damage</strong></td>
<td>Patient has established end-organ damage such as myocardial infarction, heart failure, diabetic complications, incapacitating osteoarthritis, significant psychopathology, significant functional limitation(s) and/or impairment of well-being.</td>
<td>More intensive obesity treatment including consideration of all behavioral, pharmacological and surgical treatment options. Aggressive management of comorbidities as indicated.</td>
</tr>
<tr>
<td><strong>STAGE 4: end-stage (e.g., loss of sight)</strong></td>
<td>Patient has severe (potentially end-stage) disability/ies from obesity-related chronic diseases, severe disabling psychopathology, severe functional limitation(s) and/or severe impairment of well-being.</td>
<td>Aggressive obesity management as deemed feasible. Palliative measures including pain management, occupational therapy and psychosocial support.</td>
</tr>
</tbody>
</table>

Adapted from Sharma et al

**Obesity Myths**

Many common myths and dogmas undermine the successful management of obesity. In society, including in the healthcare system, these myths contribute to a culture in which people are prone to making assumptions about the health of individuals who are living with obesity. It is a common misassumption that people with a BMI above the normal range are always unhealthy.
Myth: “All People living with obesity are unhealthy”

Because of the strong association of obesity with metabolic syndrome and other comorbid disease, many individuals who are classified as having overweight or obesity on the BMI scale are assumed to be either sick or at great risk of disease. But the assessment of risk must be individualized for each person. Not everyone who is outside the “normal” BMI range is at risk, since body types are highly heterogeneous.

The Edmonton Obesity Staging System (EOSS) was designed to help healthcare providers better classify individuals according to their weight-related risk and is a better predictor of long-term mortality than BMI or waist circumference (see next).

Meet Melanie
Melanie is a 36-year-old mother of two children, ages 2 and 4 years. She has been your patient for approximately 12 years. She is seeing you today with a complaint of fatigue. She has heard that hypothyroidism causes fatigue and weight gain and she asks if she may have a thyroid test.

You inquire about potential causes of Melanie’s fatigue. She says that she is very busy now that she is back to work full-time at her hectic, high-stress job at an accounting firm. She says she finds it difficult to get out of bed when the alarm goes off. She tends to stay up until 11 or 12pm doing laundry, cleaning, and getting things organized for her children to go to daycare the next day, and she knows she isn’t getting enough sleep.

Obesity over the Lifespan
Several factors have been identified that represent landmarks in the course of obesity that influence the course of weight gain across the lifespan:

- **Obesity in Childhood**
  Children who develop obesity are likely to maintain obesity into adulthood. Children with obesity are also at higher risk of obesity-related disease from an earlier age, and thus tend to have an earlier onset of complex chronic illness.\(^\text{19}\)

- **Obesity During or after Pregnancy**
  Women who have obesity during pregnancy or who have a greater than recommended weight gain are at increased risk of perinatal complications such as gestational diabetes, fetal macrosomia, preterm birth, small-for-gestational age.\(^\text{20}\)

  High weight gain during pregnancy is associated with intergenerational obesity, with increased body weight of the child (during childhood).\(^\text{21}\)

  Many studies report an increase in maternal body weight with increasing age and parity. However, individuals vary in their susceptibility toward weight gain with pregnancies, and lifestyle changes that occur after having children may have a large effect on post-partum weight retention.\(^\text{22}\)
Increasing Obesity with Age
Cross-sectional studies show that BMI tends to increase during most of adult life, peaking by age 60, and thereafter tends to decline. However, people with obesity have higher mortality rates at younger ages, so premature mortality may decrease mean BMI in the surviving cohort. Data from longitudinal studies suggest that body weight and BMI do not change, or decrease only slightly, in older adults.23

Consequences of Weight Cycling
Because weight loss in individuals with obesity is commonly followed by weight regain, several studies have attempted to elucidate whether there is an effect on increasing obesity, psychological effects, or cardiometabolic risks, but with mixed results. This is an area of ongoing research.24–26

Melanie Revisited
Melanie had overweight prior to her first pregnancy. She has a family history of obesity (her mother). Her BMI then crossed into Class I Obesity after her first child was born. Melanie had difficulty maintaining any weight loss prior to her second pregnancy, and has continued to gain weight since the birth of her second child. At 38.4 kg/m², her BMI is now in Class II Obesity. Melanie’s blood pressure and lipids are in the normal range, but her bloodwork shows impaired fasting glucose.

Case Challenge
What is Melanie’s EOSS stage?
   a) Stage 0
   b) Stage 1
   c) Stage 2
   d) Stage 3
   e) Stage 4
The 5 As of Obesity Management
The Canadian Obesity Network provides a framework for managing obesity in primary care: **The 5 As of Obesity Management**. Adapted from the same framework used to promote smoking cessation in primary care, the 5 As include five steps for helping patients with managing weight-related health risks:

1. **ASK** for permission to discuss weight and explore readiness
2. **ASSESS** obesity-related risks and ‘root causes’ of obesity
3. **ADVISE** on health risks and treatment options
4. **AGREE** on health outcomes and behavioural goals
5. **ASSIST** in accessing appropriate resources and providers

Step 1: Ask
How could you initiate the conversation about body weight with Melanie?

Weight is a sensitive issue to discuss. People who have overweight or obesity are often stigmatized, in public and in the healthcare system, as lazy and overindulgent. So conversations about weight must be sensitive and non-judgemental.

The first step in managing obesity with the 5 As is to initiate a conversation about weight by asking the patient for permission to discuss weight, and exploring their readiness to discuss the issue.

Because obesity is intertwined with so many weight-related health problems in primary care, there are many opportunities to discuss weight. You recognize that Melanie’s concern that she might have low thyroid hormone is an opportunity to ask her permission to discuss her weight gain:

“You mentioned you are worried about your thyroid, because it can cause fatigue and weight gain. We’ve discussed a few of the reasons you might be feeling tired. Would you also like to talk about your weight?”

Obesity Myths
Many common myths and dogmas undermine the successful management of obesity. In society and in the healthcare system, these myths contribute to a culture in which people are prone to making assumptions about the values or the lifestyles of individuals living with obesity. One example is the prevailing idea that obesity is simply caused by overeating and inactivity or lack of willpower.
Myth: “Obesity is caused simply by gluttony and sloth”

This misconception assumes that those affected by obesity simply overindulge in food and neglect to exercise; that they just don’t care or lack will power. Inherent to this assumption is the idea that obesity is self-inflicted and that losing weight is as easy as exerting a bit of self-control.

But people who have had severe obesity report extreme distress. In one study of patients who had maintained weight loss after gastric surgery, almost all participants said they would prefer to be blind, deaf, or lose a limb than to have severe obesity.\(^{29}\)

If the desire to avoid obesity is so strong, and if losing weight is really as easy as eating less and moving more, how come so many of us are obese?

Obesity is not a choice, or a failing of will power. Obesity is a result of factors that have been outside the individual’s control. These might include sociocultural, behavioural, psychological, and biological factors.

Myth: “Shame and blame can motivate patients to lose weight”

There is a common misconception that shame and blame -- or provoking guilt or fear -- can motivate patients to lose weight. However, research shows that such strategies are not effective and can in fact lead to avoidance of medical consultations and increase engagement in unhealthy behaviours.\(^{30-32}\)

Obesity should be discussed in a respectful, non-judgemental, and constructive fashion to promote patient engagement and adherence.

Identifying Root Causes of Obesity in Your Patient

An individualized approach to treating obesity should attempt to identify the drivers of excess weight gain. These can be influencers of overeating (diet), reduced metabolism, or reduced physical activity (see Figure 1). Factors to consider include socio-cultural, biomedical, psychological, or iatrogenic causes. Although obesity is a disease of energy surplus, it is not remedied – as centuries of anecdotal and clinical evidence have shown – by simply prescribing an energy deficit (i.e., diet and exercise).\(^{33}\)

“Rather than just identifying and describing a behaviour (‘this patient eats too much’), clinicians should seek to identify the determinants of this behaviour (‘why does this patient eat too much?’)”

-- Sharma and Padwal 2010
Figure 1: Influencing Factors

Influences on Metabolic Rate
An individual’s energy expenditure through resting metabolism represents a large component of their caloric expenditure (60-75% in sedentary individuals). Any reduction in metabolic rate can promote weight gain or make weight loss difficult.

Resting metabolic rate can be influenced by many factors. These include:

- Age (resting metabolic rate decreases with age by a rate of approximately 150 kcal/day per decade)
- Gender (for the same BMI, women will have a 20% lower energy requirement than men)
- Genetics and epigenetics (recent studies suggest that genetics account for over 75% of BMI)
- Medications (e.g., beta-blockers can lower metabolic rate (see Table 4 in next section), while nicotine or caffeine can raise it)
- Metabolically active fat (brown adipose tissue is metabolically active, can account for 20% of daily energy expenditure, and may be increased with cold-exposure)
- Neuroendocrine factors (e.g., cortisol, growth hormone, estrogens, and progesterone)
- Prior weight loss (a 5 to 10% reduction in body weight can confer a corresponding 20% reduction in resting metabolic rate, increasing susceptibility to regain)
- Sarcopenia (changes in lean body mass can have a marked effect on metabolic rate; many diseases or conditions can result in muscle wasting)
Influences on Overeating

Overeating can involve socio-cultural, biomedical, physiologic/homeostatic, or psychological or iatrogenic factors. Although drivers of overeating may be complex and overlapping, identifying the primary factor for a given patient can provide the key to the treatment plan.33

- Socio-cultural factors: These are wide ranging and many involve tradition, habit, beliefs, peers, food availability or advertising, or food affordability.

  The prevalence of highly palatable, convenient, large-portioned, and energy-rich foods in our modern environment has been characterized as an aspect of a current “obesogenic environment” thought to contribute to overeating and weight gain.

- Physiological/homeostatic factors: Increased hunger and reduced satiety can be influenced by certain diseases or conditions, but are common regulatory mechanisms of weight regain after weight loss. Nutritional composition of foods may also play a role in these factors if after consuming high glycemic-index foods, blood sugar spikes and then falls.

- Psychological factors: the hedonic seeking of food may be used to stimulate the brain’s reward centres, and may be driven by any emotional state (stress, sadness, anger, etc.) or by boredom or a health condition (e.g., depression, ADHD, anxiety, PTSD, SAD, cognitive disorders, chronic pain, sleep deprivation, or addiction).

- Medication: appetite-stimulating medications or illicit drugs include certain oral antidiabetic agents or insulin, antidepressants, atypical antipsychotics*, anticonvulsants, corticosteroids, oral contraceptives, marihuana, and alcohol (see Table 4).41

It should be noted that the above categories of influences are overlapping, and interconnected. Homeostatic and hedonic influences have interplay on one another. See Metabolic and hedonic drives in the neural control of appetite: Who’s the boss?49

* Metformin use is not currently indicated for weight control in people taking atypical antipsychotic medications, but may be of benefit, especially in young adults when started early in the course of treatment.50
### Table 4: Selected Medications that can Cause Weight Gain\(^{41}\)

<table>
<thead>
<tr>
<th>Classes/Medications</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Antihistamines</td>
</tr>
<tr>
<td>• Psychotropic medications</td>
</tr>
<tr>
<td>o Tricyclic antidepressants</td>
</tr>
<tr>
<td>o Monoamine oxidase inhibitors</td>
</tr>
<tr>
<td>o Specific SSRIs</td>
</tr>
<tr>
<td>o Atypical antipsychotics</td>
</tr>
<tr>
<td>o Lithium</td>
</tr>
<tr>
<td>o Specific anticonvulsants</td>
</tr>
<tr>
<td>• (\beta)-adrenergic receptor blockers</td>
</tr>
<tr>
<td>• Diabetes medications</td>
</tr>
<tr>
<td>o Insulin</td>
</tr>
<tr>
<td>o Sulfonylureas</td>
</tr>
<tr>
<td>o Thiazolidinediones</td>
</tr>
<tr>
<td>• Highly active antiretroviral therapy</td>
</tr>
<tr>
<td>• Tamoxifen</td>
</tr>
<tr>
<td>• Steroid Hormones</td>
</tr>
<tr>
<td>o Glucocorticoids</td>
</tr>
<tr>
<td>o Progestational steroids</td>
</tr>
</tbody>
</table>
Influences on Physical Activity

As with eating and metabolism, the influences on physical activity are span domains. Exercise habits can vary widely, from highly sedentary (e.g., bed-ridden) individuals to endurance athletes who may expend multiple times their basal metabolic energy needs.

In sedentary individuals, the majority of energy burning through activity is in the form of non-exercise related activity, such as fidgeting, postural changes, and the activities of daily living. This non-exercise activity thermogenesis (NEAT) can change spontaneously to defend against weight changes.

- Socio-cultural factors: These are wide ranging and many involve culture, availability of opportunities for activity in the environment (e.g., gyms, sidewalks, neighbourhood safety), occupation, pregnancy, time constraints, etc.\(^3\)

- Physiological/homeostatic factors: Many forms of disease or conditions reduce physical activity. These might include pain or immobility, respiratory disease, sleep apnea, cardiovascular disease, or urinary incontinence. For example, obesity is associated with musculoskeletal stressors, such as osteoarthritis (see box below), which can limit activity.\(^3\) Psychiatric conditions (e.g., bipolar disorder) can also affect weight homeostasis and activity levels.\(^5\)

- Psychological factors: low motivation, low energy, or lack of interest may be factors, and in a previously active individual may be symptomatic of depression. Many other psychological or psychiatric conditions can also influence activity, including social anxiety, agoraphobia, sleep disorder, or substance abuse for example. Body image issues and self-efficacy can also have effects. For example, feelings of shame or low confidence can prevent an individual from going out in public to exercise.\(^3\)

- Medication: Any medication that induces drowsiness, impairs coordination, reduces cardiovascular function, or reduces energy level can be a factor in reduced physical activity.\(^3\)

---

**Obesity and the Joints:**

A 2005 study found that each pound of weight lost in individuals with obesity resulted in a 4-fold reduction in the load exerted on the knee per step during daily activities.\(^5\)

Obesity has a major effect on both loading and non-loading joints. This is thought to be due to both mechanical overload, and to systemic inflammation. There is an increased incidence and prevalence of osteoarthritis in obesity, which is more common in the presence of cardiometabolic disturbances. This osteoarthritis phenotype is now described as metabolic syndrome (MetS)-associated osteoarthritis.\(^5\)
Step 2: Assess
Melanie says that she very much wants to get control of her weight. “I’ve tried so many things over the last few years and I lose a few pounds, but then I gain them back and more,” she says.

You screen Melanie with the Patient Health Questionnaire-9 (PHQ-9), the results of which do not suggest she has depression.

You then review factors that could be influencing Melanie’s metabolism, appetite, and level of physical activity.

Melanie says that her constant tiredness makes her less motivated to go for walks like she used to before she had children. Also, because of the hectic lifestyle she has with two small children at home, she finds it difficult to find time or arrange child care. Because she is often awake with her youngest child during the night, Melanie says she tends to feel hungry when she wakes up and will eat leftovers from dinner. She frequently resorts to sugary drive-through or fast-food items for breakfast and also often skips breakfast or lunch due to time constraints. She reports no history of snoring or symptoms that may suggest sleep apnea.

Step 3: Advise
You let Melanie know that her blood tests show that her thyroid levels are in normal range, but that her blood sugars are higher than they should be. You explain that type 2 diabetes is a risk if Melanie’s weight continues to rise, since she is currently pre-diabetic.

You tell Melanie that you’re glad she’s committed to getting control of her weight, because it will have benefits for her overall health and well-being.

You let Melanie know that it sounds like her lack of sleep may be an important factor driving her weight gain, and she agrees.

“The diet and exercise methods that you’ve been trying since your children were born might create some short term weight loss,” you explain, “but to really get control, we need to address the underlying causes. Let’s set a goal, and see if we can get an extra hour of sleep everyday, and then meet again to see if that has made a difference. In addition, we can begin to help you monitor your food. Does that sound okay?”

Case Challenge
Now that you’ve competed the first 3 steps of the 5As, what will you do next to help Melanie?

a) Agree, Assist
b) Agree, Arrange
c) Arrange, Assist
d) Arrange, Agree
Key Points

- Obesity is a chronic disease that is often progressive.
- Obesity rates are rising in Canada, and obesity is a leading cause of preventable morbidity and mortality.
- Obesity is defined as BMI > 30 kg/m$^2$. Overweight is defined as BMI > 25.
- The Edmonton Obesity Staging System (EOSS) describes five stages of obesity, based on the impact excess weight is having on the patient’s health.
- Overeating and low physical activity are symptoms of obesity.
- Behavioural management strategies should be individualized to the patient and should address root causes.
- Liraglutide and orlistat are pharmaceutical options for the treatment of obesity in Canada, as is bariatric surgery, where indicated. See the next vignette for more information about these and other interventions.
- The 5 As of Obesity Management are:
  - ASK for permission to discuss weight and explore readiness
  - ASSESS obesity-related risks and 'root causes' of obesity
  - ADVISE on health risks and treatment options
  - AGREE on health outcomes and behavioural goals
  - ASSIST in accessing appropriate resources and providers
Pre/Post Test

1. What is the World Health Organization (WHO) definition of obesity?
   a) BMI > 25 kg/m²
   b) BMI > 27.5 kg/m²
   c) BMI > 30 kg/m²
   d) BMI > 32.5 kg/m²

2. What proportion of Canadian adults has obesity?
   a) One-tenth
   b) One-quarter
   c) One-third
   d) One-half

3. When a patient has been gaining weight and reaches a BMI of 30.0 kg/m², what BMI class are they now in?
   a) Overweight
   b) Obesity Class I
   c) Obesity Class II
   d) Obesity Class III

4. Up to what proportion of energy expenditure is represented by resting metabolism in sedentary individuals?
   a) 15%
   b) 30%
   c) 50%
   d) 75%

5. Which of the following types of medication is not associated with weight gain?
   a) atypical antipsychotics
   b) methylphenidate
   c) glucocorticoids
   d) insulin
6. What factor will the body decrease to defend against weight loss when an individual begins a program of reduced-calorie diet and exercise?
   a) exercise
   b) non-exercise activity thermogenesis
   c) appetite

7. Rick is a 38-year-old male patient with a BMI of 40 kg/m² and osteoarthritis of the knee. Rick has difficulty increasing his physical activity due to his knee pain. How much will each pound of weight loss reduce the load exerted on Rick’s knee per step during daily activities?
   a) 2-fold load reduction per pound of weight lost
   b) 3-fold load reduction per pound of weight lost
   c) 4-fold load reduction per pound of weight lost
   d) 5-fold load reduction per pound of weight lost

8. The 5 As of Obesity Management stand for:
   a) Ask, Assess, Advise, Agree, Assist
   b) Answer, Assess, Advise, Agree, Assist
   c) Ask, Assess, Advise, Agree, Arrange
   d) Assess, Answer, Advise, Agree, Amend

9. What is the Edmonton Obesity Stage (EOSS) of a patient with type 2 diabetes, and moderate limitations in activities of daily living and/or well-being?
   a) Stage 0
   b) Stage 1
   c) Stage 2
   d) Stage 3
   e) Stage 4

10. What treatments can be considered, based by the EOSS system, for a patient of EOSS Stage 2?
    a) behavioural interventions
    b) pharmaceutical interventions
    c) surgical interventions
    d) a and b
    e) all of the above
Vignette 2: Individualizing Obesity Management

This vignette will explore the approach to implementing an individualized treatment plan for obesity and overcoming barriers to the success of the treatment plan.

Learning Objectives:

After completion of this learning module, the participant will be able to:

- Partner with the patient in identifying the goal of and approach to treatment
- Implement an individualized plan for the treatment of obesity
- Help the patient overcome barriers to weight loss

The Global Obesity Epidemic

Obesity rates have more than doubled worldwide since 1980, becoming one of the important public health problems of the century. More than half of adults are now considered to be overweight or obese.\(^2\)

Once present in an individual, obesity must be recognized as a chronic disease requiring lifelong management. Children who develop overweight or obesity tend to also develop comorbid disease at a younger age and to continue to have excess weight into adulthood. Once considered a disease of adulthood, type 2 diabetes is on the rise in children and has been diagnosed in children as young as age 3 years.\(^19\)
Causes of Rising Obesity Rates

Obesity is a chronic disease characterized by a surplus of body fat. Many diverse factors can influence body weight.

The body’s energy balance, which is a function of food intake, metabolism, and physical activity (see Figure 2) shifts to a positive balance during periods of weight gain and a negative balance during periods of weight loss.

Commonly, the obesity epidemic is attributed to the combined availability of energy-dense food and the decrease in physical activity. While the net calorie surplus that can result for individuals whose food intake exceeds their caloric expenditure provides a simplistic and plausible explanation for weight gain, this explanation falls short in many ways.

Rather, the phenomenon of overeating-and-inactivity, as highlighted by Sharma et al., is often merely a symptom, rather than a root cause of obesity. Correspondingly, the oft-proposed remedy – “eat-less-move-more” – commonly fails to produce lasting results because it fails to address root causes and to account for the body’s own regulatory control over body weight.

The mechanisms of obesity are complex and multifactorial, involving not just diet and activity, but genetic, economic, psychosocial, reproductive, chemical, and iatrogenic factors. Research into obesity considers such putative causes as: changes in ambient temperatures, environmental endocrine disrupters, pharmacological agents, infectious microbes, gut microflora, sleep debt, maternal and developmental effects, evolutionarily selective pressures and assortive mating, and epigenetic mechanisms.

Recent studies suggest that heritable factors account for over 75% of BMI.
Figure 2: What Factors Affect Energy Balance?

The body exerts tight regulatory control over energy balance. Attempts to extrinsically alter energy balance will eventually be met by a counterbalance. For example, increases in physical activity commonly increase appetite and decrease NEAT.

Efforts to address energy balance through extrinsic factors (i.e., diet and exercise) tend to ineffectively address obesity over the long term. To treat obesity, we must look to the influences that are driving changes in energy balance.
Obesity Myths

Many common myths and dogmas undermine the successful management of obesity. And in society, and in the healthcare system, these myths contribute to a culture in which people are prone to making assumptions about the values or the lifestyles of individuals who have obesity. One example is the idea that obesity is caused simply by eating a high-fat diet.

**Myth: “Obesity is mainly caused by a high fat diet”**

Nutrition guidelines over the last half century have recommended restricting dietary fat intake. This recommendation began in the US in 1977 and within a few years was adopted across much of the developed world. Dietary fats, which contain more calories per gram than either of the other two macronutrients (protein or carbohydrates) have been implicated as a cause of a net calorie surplus (i.e., the erroneous ‘net energy’ thinking described above). Additionally, fats became the villain of the since-debunked diet-heart hypothesis, which considered dietary fat to be a direct contributor to cardiovascular disease.\(^{57,58}\) High fat diets have since become intrinsic in the social stigma associated with obesity, with degrading depictions often featuring fatty foods.

In fact, the obesity epidemic has coincided with the implementation of low-fat dietary guidelines across the world.\(^{59,60}\)

### 2015 Update

This year saw the removal of restrictions on dietary fat and cholesterol from the US guidelines.\(^{61}\) Additionally, a meta-analysis of the RCT evidence demonstrated these recommendations were never evidence-based.\(^{62}\)

Click here to listen to the *British Medical Journal* podcast with the lead author of the meta-analysis.
Goals of Treatment

There is a common misconception that the goal of an obesity management plan is simply for the patient to lose weight.

1. An effective approach to obesity treatment must first recognize that obesity is a chronic and often progressive disease. For those patients who have been in a pattern of weight gain over time, the first goal is not weight loss, but weight stabilization (i.e., the prevention of further weight gain).\(^63\)

2. Once weight has been effectively stabilized, weight loss can be approached as the next step. Because patient expectations often drastically exceed achievable results, it is important to have a full discussion to set realistic behavioural goals that will confer health benefits and reduced risks. Even a weight-loss of 5% is clinically significant, with the potential to substantially reduce risk of comorbidities.\(^5,6,63\)

3. Meta-analysis shows current weight-management interventions in primary care are not effective over the long term, achieving a mean weight loss of less than 1.5 kg at 12 and 24 months.\(^7\) A recognition of obesity as a chronic disease requiring lifelong intervention, placing a focus on maintenance of weight loss and improvement in health behaviours over the long term, is critical to the success of treatment.\(^63\)

Behavioural Management

As discussed, behavioural interventions should not be prescribed without first identifying the determinants of the individual patient’s obesity. For example, if sleep disruption is suspected to be a causative factor in their weight gain, then sleep hygiene approaches would be an appropriate behavioural approach.

Practice Resources:

- Recent studies have shown low-carbohydrate diets to be more effective for weight loss than low-fat diets:
  - Effects of low-carbohydrate diets versus low-fat diets on body weight and cardiovascular risk factors: a meta-analysis of randomised controlled trials\(^64\)
  - The effects of a low-carbohydrate diet vs. a low-fat diet on novel cardiovascular risk factors: A randomized controlled trial\(^65\)
  - (note that the precise “ideal” macronutrient ratio for weight loss has not yet been clinically defined but the carbohydrate content in the studies included above was <20% and <40g/day respectively)\(^66-69\)
- Systematic review and meta-analysis of different dietary approaches to the management of type 2 diabetes\(^70\)
- 2013 AHA/ACC/TOS Guideline for the Management of Overweight and Obesity in Adults: A Report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines and The Obesity Society\(^71\)
Pharmacological Management

The pharmaceutical agents liraglutide and orlistat are approved in Canada for use as adjunct to behavioural interventions for weight management in adult patients:

- who have 30 kg/m² or greater (obesity)
- who have an initial BMI >27 kg/m² (overweight) in the presence of weight-related comorbidity, and who have failed a previous weight management intervention

Liraglutide

Liraglutide is a once-daily injected human glucagon-like peptide-1 (GLP-1) analogue approved for use in obesity management in Canada in 2015. (The active ingredient liraglutide is also indicated under a different brand name for antihyperglycemic use in type 2 diabetes but the two products cannot be used concomitantly or interchangeably.)

Liraglutide is an analogue of naturally occurring human GLP-1, a hormone that is released in response to food intake, which lowers appetite. As with other GLP-1 receptor agonists, liraglutide stimulates insulin secretion and reduces glucagon secretion in a glucose-dependent manner. These effects can lead to a reduction of blood glucose.

In randomized clinical trials with liraglutide:

- 63.5% of patients with overweight or obesity had lost at least 5% body weight at 56 weeks compared to 26.6% in the placebo group (p < 0.0001)
- 32.8% of patients had lost at least 10% body weight compared to 10.1% in the placebo group (p < 0.0001)

Side Effects

The most common (incidence >10%) side effects with liraglutide are lowered appetite and gastrointestinal symptoms (nausea, vomiting, diarrhea, constipation). Gastrointestinal symptoms tend to be of transient duration (days or weeks).

Dosing

In adults with an initial BMI of 27 kg/m² or greater, the recommended daily maintenance dose is 3.0 mg/day. Daily doses higher than 3.0 mg are not recommended. For all patients, an initial dose of 0.6 mg should be escalated by 0.6 mg on a weekly basis to reach the 3.0 mg/day dose. This escalation schedule is used to reduce the likelihood of gastrointestinal symptoms. (If patients do not tolerate an increased dose during dose escalation, the dose escalation can be changed with a total delay of up to 7 days.)

Warnings and Contraindications
Liraglutide is contraindicated in patients with a personal or family history of medullary thyroid carcinoma or in patients with Multiple Endocrine Neoplasia syndrome type 2 (MEN 2).

In animal models (rats and mice), liraglutide was found to cause dose-dependent and treatment-duration-dependent thyroid C-cell tumours at clinically relevant exposures in both genders. It is unknown whether liraglutide causes thyroid C-cell tumours, including medullary thyroid carcinoma (MTC), in humans, as human relevance could not be ruled out by clinical or nonclinical studies.

Orlistat

Orlistat is a gastrointestinal lipase inhibitor that has been in use since the 1990s for obesity management. In the 4-year XENDOS trial. In the 4-year XENDOS trial:

... After 1 year of treatment:

- 73% of patients lost ≥ 5% of baseline body weight after 1 year of treatment compared with 45% of the placebo-treated patients (p<0.001).
- 41% of patients lost ≥ 10% of body weight after 1 year compared with 21% of the placebo-treated patients (p <0.001).

... After 4 years of treatment:

- 44.8% and 21% of the patients treated with orlistat lost ≥ 5 and ≥ 10% of body weight compared to 28.0% and 10% of the placebo treated patients, respectively (p <0.001).

Side Effects

Gastrointestinal symptoms were the most commonly (incidence ≥ 5%) observed treatment-emergent adverse events in double-blind, placebo-controlled clinical trials and were primarily a manifestation of the mechanism of action.

Dosing

The recommended dose of orlistat is one 120 mg capsule three times daily with each main meal (during or up to 1 hour after the meal). If a meal is occasionally missed or contains no fat, the dose may be omitted.

Doses above 120 mg three times daily have not been shown to provide additional benefit. No dose adjustment is necessary for the geriatric patient.

Based on fecal fat measurements, the effect of orlistat is seen as soon as 24 to 48 hours after dosing. Upon discontinuation of therapy, fecal fat content usually returns to pre-treatment levels within 48 to 72 hours.

Warnings and Contraindications

Orlistat is contraindicated in patients with chronic malabsorption syndrome or cholestasis.
No serious adverse reactions or safety hazards related to the use of orlistat have been reported during large, long-term clinical trials (up to 4 years). As with any weight-loss agent, the potential exists for misuse of orlistat in inappropriate patient populations (e.g. patients with anorexia nervosa or bulimia).

When using orlistat in combination with insulin or oral hypoglycemic agents in the treatment of type 2 diabetes, the risks of hypoglycemia, its symptoms and treatment, and conditions that predispose to its development should be explained to the patient, family members, caregiver or others.73

Practice Resource

Reviews of current and investigational drugs:

- See S Wharton’s 2015 Canadian Journal of Diabetes review of currently approved pharmaceutical interventions for obesity, and investigational agents.74
- See AK Kakkar and N Dahiya’s 2015 European Journal of Internal Medicine review of drug treatments for obesity.75
Bariatric Surgery

Bariatric surgery is a powerful adjunct tool in the management of severe obesity. It is an effective therapy for many of the metabolic and mechanical complications of obesity and is associated with better weight-loss maintenance than conservative treatment alone.\(^7^6\)

Weight loss of ~10% is beneficial in preventing future diabetes in patients with prediabetes or metabolic syndrome. In type 2 diabetes, greater amounts of weight loss lead to improvements in glycemic control. Bariatric surgery can often produce greater weight loss and more pronounced metabolic benefits than lifestyle interventions and medical treatment alone, via mechanisms that may be both weight-dependent and weight-independent (e.g., changes in gut hormones, bile acids, or gut microflora).\(^7^7\)

Bariatric surgery should be strongly considered in patients for whom other less-invasive methods have failed and who present with obesity-associated morbidity or increased mortality risk.\(^7^6\)

Current NIH guidelines recommend bariatric surgery for those patients with:

- BMI ≥ 40
- ≥ 35 kg/m\(^2\) with comorbid conditions

Roux-en-Y gastric bypass surgery (see Figure 3) affects weight-loss through several mechanisms, including reduced hunger, increased satiation, change in food preferences, and increased diet-induced energy expenditure.\(^7^8\)

Gastric banding or vertical sleeve gastrectomy (see Figure 3) likely affect weight-loss through reduced hunger, which may be mediated by vagal signalling.\(^7^8\)

Another type of bariatric surgery is biliopancreatic diversion or biliopancreatic diversion with duodenal switch (which preserves the pyloric valve).\(^7^9\)

The approach to bariatric surgery should involve a multi-disciplinary team. The patient should be fully informed of the risks and the benefits. Lifelong monitoring and follow-up after surgery is strongly recommended.\(^8^0\)
**Figure 3: Types of Bariatric Surgery**

![Diagram of bariatric surgery types](image)

a. Roux-en-Y gastric bypass, b. adjustable gastric banding, c. vertical sleeve gastrectomy


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**Meet Melanie**

Melanie is a 36-year-old mother of two children, ages 2 and 4 years. She has been your patient for approximately 12 years. Melanie came to see you recently, complaining of fatigue. She had heard that hypothyroidism causes fatigue and weight gain and asked about a thyroid test.

You have completed the first 3 steps of the 5 As with Melanie:

- **You have asked** Melanie for permission to discuss her weight, and noted that Melanie is ready and committed to getting control of her weight.
- **You’ve assessed** her obesity-related risks and ‘root causes’ of obesity, and have identified that reduced and poor quality sleep seem to be a contributing factor to Melanie’s weight gain.
- **Melanie is pre-diabetic,** and you have **advised** her on the health risks associated with continued weight gain and the treatment options available to her.
Step 4: Agree

When you ask Melanie what her weight management goal is, she tells you she would like to lose at least 60 pounds. You let her know that weight loss might be possible down the road, but that since she is in a pattern of weight gain, you’d like to suggest a starting goal of stabilizing her weight.

You let Melanie know that her health is the top priority, since you want to minimize her risk of developing type 2 diabetes. “Even a small weight loss will make a big difference in your health, but as a first step, let’s get your weight stabilized so you’re not gaining anymore.” Melanie agrees to this goal and asks how she should be eating.

You let her know that it would be a good idea to keep a food journal so that she can begin to track her eating habits. But, you also ask her to track her sleep.

“We identified that a lack of sleep might be a major factor for your weight gain, so let’s start keeping track of that. Also, I have some resources here that we can go over to find ways of improving your sleep hygiene to give you the best chance of better rest.”

Melanie smiles and says, “More sleep sounds perfect!”

Obesity Myths

Many common myths and dogmas undermine the successful management of obesity. These myths contribute to a culture, in society and in the healthcare system, in which people are prone to making assumptions about the values or the lifestyles of individuals with obesity. One example is the belief that obesity can be cured with calorie restriction and exercise.

Myth: “Diet and/or exercise (energy deficit) is the cure for obesity”

The counterpart of the gluttony-and-sloth myth (see vignette 1), it is often assumed that simply restricting caloric intake or increasing caloric output will have a direct and proportional effect on reducing body weight. (This simplistic calories-in-calories-out thinking is often voiced in the mainstream media in the form of “3500 calories = 1 pound” of body fat.)

But studies of energy-restricted diets in humans consistently find that this method generally fails to achieve sustained weight loss. Although many patients may experience an initial weight loss, this is often followed by weight regain. A recent review of the literature on the long-term success of energy restricted diets for treating obesity found that at 3-year follow-up the weight loss was less than 3% than in controls, and that weight regain was observed in the majority of individuals. We’ll explore why this happens in the next section.

The Physiology of Weight Regulation

The body exerts considerable control over the acquisition, expenditure, and storage of energy. Body weight is tightly regulated at the hypothalamus (see Figure 4). Hunger is stimulated and inhibited by
complex neuropeptide-hormone pathways between the brain, gastrointestinal tract, pancreas, and adipose tissue. Genetic studies have identified nearly 100 loci involved in body weight, affecting pathways related to adipogenesis, energy metabolism, neurotransmitter signalling, insulin secretion or action, lipid biology, and synaptic function.\(^87\)

The susceptibility of individuals to weight-gain is highly heritable and is heterogeneous across a population:\(^40\)

- Twin and adoption studies show a substantial genetic component in the predisposition to weight gain.\(^88,89\) Recent studies estimate the heritability of BMI at over 75%.\(^40\)
- Overfeeding studies demonstrate that some individuals will gain weight when overfed while others will not.\(^90\) Monozygotic twin pairs in overfeeding studies gain a highly correlated amount of weight.\(^91\)
- Adoption studies reveal that family environment has no relation to obesity levels, with adoptees and their adoptive parents showing no relation in BMI, but adoptees and biological parents showing a strong relation in BMI across all weight classes (very thin to very obese).\(^89\)

The rapid rise in obesity across the globe over the last half century precludes the possibility that this change is due to natural selection (Darwinian evolution). However, epigenetic mechanisms due to environmental or maternal effects could be exerting effects on genetics that make current generations more susceptible to obesity.\(^92\)
Figure 4: Weight regulation

AgRP = agouti-related peptide. CART = cocaine and amphetamine-regulated transcript. CCK = cholecystokinin. CRH = corticotropin-releasing hormone. GLP-1 = glucagon-like peptide. MCH = melanin-concentrating hormone. αMSH = alpha melanocyte-stimulating hormone. NPY = neuropeptide Y. NTS = nucleus of the tractus solitarius. PP = pancreatic polypeptide. PYY = peptide YY

The Basic Biology of Fat Storage

It is well established that insulin is the hormone responsible for fat storage. Insulin levels are determined by the carbohydrate content of diet (and recent evidence also suggests that low-fat dietary composition significantly predicts insulin secretion). Weight gain and weight loss are positively and directly influenced through insulin levels.

High blood glucose elicits the release of insulin in healthy individuals. Insulin both facilitates fat storage and prevents its release from body tissues. Insulin enables the uptake of glucose by tissues and favours the storage of fuels as glycogen and triglycerides. Insulin also inhibits fatty acid mobilization in adipose tissue (i.e., prevents “fat burning”).

“The activity of LPL within individual tissues is a key factor in partitioning triglycerides among different body tissues. Insulin influences this partitioning through its stimulation of LPL activity in adipose tissue. Insulin also promotes triglyceride storage in adipocytes through other mechanisms, including inhibition of lipolysis, stimulation of adipocyte differentiation and escalation of glucose uptake.”

-- Williams Textbook of Endocrinology

LPL: lipoprotein lipase

The Physiological Defenses of Body Weight

Once established, obesity rarely resolves with conventional diet-and-exercise approaches and/or commercial weight loss programs over the long term.
This is because the body exerts homeostatic control over fat storage, and changes in weight due to extrinsically imposed changes in energy balance (i.e., diet and exercise) stimulate complex and persistent corrective measures that defend against further weight loss and promote weight regain. Thus, following weight loss, biologically induced reductions in energy expenditure and increases in appetite drive weight regain. Specifically, there are hormonal adaptations that occur in response to weight loss, which include an increase in hunger hormone levels, and a decrease in satiety hormone levels. These changes persist long after weight loss has been achieved. In addition, in response to calorie restriction, energy expenditure is decreased through both the slowing of metabolism and reduced spontaneous physical activity.97–101

- Weight loss decreases the resting metabolic rate by 3% for every 10 pounds of weight lost (see Figure 5).47
- Following periods of semi-starvation (dieting) or starvation (fasting), neurochemicals act to preserve weight. After only 12 hours of fasting, there can be a 40% decrease in metabolic rate and significant overeating of energy-dense foods of large quantities with rapid intake (binge eating).101,102

These physiological defenses of weight explain the difficulty of weight loss and the problem of recidivism seen with a range of interventions, from behavioural and pharmacologic treatments to bariatric surgery.56,103–110

Figure 5: The Effect of Weight Loss on Resting Metabolism

Female, age 50 years, 5’ 7”

230 lb
BMR = 1822

170 lb
BMR = 1496
(18% less)
Barriers to Obesity Treatment
For successful obesity treatment, it is important for patients and practitioners to identify each individual’s potential barriers to weight loss.

Some common barriers are shown in Table 5, and include: the acceptance that obesity requires lifelong intervention; addressing any socioeconomic limitations (e.g., inability to access commercial treatments, or the higher cost of healthier foods); making plans to address time constraints that can interfere with weight management; recognizing and managing individuals who are saboteurs to the weight loss plan and building a system of support; and identifying and addressing any comorbidities or medications that promote weight gain or impede weight loss, including alcohol or substance abuse.63
<table>
<thead>
<tr>
<th>Barrier</th>
<th>Intervention</th>
<th>Rationale</th>
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<tr>
<td>Acceptance of obesity as a chronic disease</td>
<td>Education of patients and health care providers.</td>
<td>Obesity treatment requires lifelong management to maintain weight loss.</td>
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<tr>
<td>Socioeconomic status</td>
<td>Address limitations. Adapt strategy to socioeconomic situation.</td>
<td>Long-term adherence to the obesity treatment should be facilitated.</td>
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<tr>
<td>Time constraints</td>
<td>Reassess motivation and readiness for change. Adapt strategy to patient’s schedule.</td>
<td>- Offers portion control, availability, portability, ease of preparation. - Practical and sustainable.</td>
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<td>Support systems are essential to long-term weight maintenance.</td>
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<tr>
<td>Side effects</td>
<td>- Use of meal replacements</td>
<td>- Cognitive behavioural therapy (CBT) focusing on binge eating and associated psychopathology facilitates adherence to lifestyle habits.</td>
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<td>- Flexible exercise schedule</td>
<td>Reduction of cardiovascular risk and risks of accidents. Improved sleep may positively impact adherence to lifestyle changes and physical activity.</td>
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<td>- Improvement of depression and impulsive behavior facilitates adherence to obesity treatment.</td>
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<tr>
<td>Comorbidities</td>
<td>- Identification of problem.</td>
<td>Allows patient to be more physically active.</td>
</tr>
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<td></td>
<td>- Mental health</td>
<td>Allows patient to be more physically active.</td>
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<td>- Recognition and concurrent treatment of depression and attention deficit disorder (ADD).</td>
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<td>- Recognition and referral for specific intervention of binge eating disorder (BED)</td>
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<td>- Evaluation and treatment of obstructive sleep apnea (OSA)</td>
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<td>- Management of symptoms</td>
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<td></td>
<td>- Respiratory disease</td>
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<td>- Accurate diagnosis and treatment of the cause of dyspnea</td>
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<td>- Digestive disease</td>
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<td>- Recognition and treatment of reflux symptoms</td>
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<td></td>
<td>- Endocrine disorders</td>
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<td></td>
<td>- Recognition and treatment of insulin resistance, hyperglycaemia, Cushing and hypogonadism</td>
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</tr>
<tr>
<td>Medication</td>
<td>Choose medication with less propensity for weight gain.</td>
<td>Prevent weight gain.</td>
</tr>
<tr>
<td>Alcohol and substance abuse</td>
<td>Assessment and treatment of addictions.</td>
<td>Improve adherence to obesity treatment.</td>
</tr>
</tbody>
</table>

Step 5: Assist
You provide Melanie with options including a blank paper journal or an electronic food-tracking app (e.g., MyFitnessPal) to track her food and you recommend that she also track her sleep. You give her the resources on sleep hygiene that you’ve just reviewed, with notes about her individualized strategies.

You ask Melanie to schedule a follow up appointment for a month from now.

Note: Many electronic apps and devices (such as the FitBit) have entered the market and can be helpful in tracking food and activity patterns, as well as other measures such as heart rate. While these tools can improve self-monitoring, it should be noted that their use in the absence of a management plan that focuses on addressing the core drivers of an individual’s obesity is unlikely to result in long-term weight loss, especially if the focus is on calorie-counting (i.e., extrinsic factors in the energy balance – see Vignette 1).

Practice Resources:

The weight loss industry in Canada exceeds $6 billion dollars a year. Yet weight recidivism is virtually 100%. How can we help patients navigate and interpret information and resources?

See the Canadian Obesity Network’s guide to resources found online and in the community.

Case Challenge
If Melanie is unable to lose weight and maintain weight-loss with behavioural measures alone, you might consider recommending adjunctive pharmaceutical treatment or bariatric surgery. What pharmaceutical agents are approved by Health Canada for the treatment of obesity?

a) metformin
b) orlistat
c) liraglutide
d) bupropion
e) all but d
f) a and b
g) b and c
Key Points

- Although the factors that influence energy balance are complex, intersecting, and span domains, many of these factors are modifiable or manageable.
- By recognizing root causes of obesity in a given patient, practitioners can make individualized recommendations.
- Even small changes in key influencers of an individual’s weight gain have the potential to shift the energy balance favourably.
- Behavioural management strategies should be individualized to the patient and should address root causes.
- Pharmaceutical agents liraglutide and orlistat are approved in Canada for use as adjunct to behavioural interventions for weight management in adult patients:
  - who have 30 kg/m² or greater (obesity)
  - who have an initial BMI >27 kg/m² (overweight) in the presence of weight-related comorbidity, and who have failed a previous weight management intervention
- Bariatric surgery may be the optimal approach to management for carefully selected patients with severe obesity. This might include patients for whom other less-invasive methods have failed and who are at high risk for obesity-associated morbidity or mortality and have:
  - BMI ≥ 40
  - ≥ 35 kg/m² with comorbid conditions
- The 5 As of Obesity Management are:
  - ASK for permission to discuss weight and explore readiness
  - ASSESS obesity related risks and ‘root causes’ of obesity
  - ADVISE on health risks and treatment options
  - AGREE on health outcomes and behavioural goals
  - ASSIST in accessing appropriate resources and providers
Pre/Post Test

1. What level of weight loss is required for individuals with obesity to achieve clinically significant changes in weight-related risks?
   a) 5%
   b) 10%
   c) 15%
   d) 20%

2. What is the first goal of weight management for patients with unhealthy weight gain?
   a) To implement diet and exercise
   b) To stabilize body weight
   c) To reduce body weight
   d) To maintain weight loss

3. Weight is regulated at the:
   a) Prefrontal cortex
   b) Hippocampus
   c) Hypothalamus
   d) Amygdala

4. Is pharmaceutical management for obesity, as add-on to behavioural interventions, an appropriate consideration for a patient who has a BMI of 28 kg/m² and a blood pressure of 140/90, and who has failed to lose weight with behavioural interventions alone?
   a) Yes, pharmaceutical management can be considered
   b) No, the patient is not a candidate for pharmaceutical management

5. Globally, how much have obesity rates increased since 1980?
   a) stayed the same
   b) increased by half
   c) doubled
   d) quadrupled
6. To what extent is BMI thought to be heritable?
   a) 25%
   b) 50%
   c) 75%
   d) 100%

7. Studies show that resting metabolism decreases by 3% for every ‘x’ lost? What is x?
   a) 5 pounds
   b) 10 pounds
   c) 15 pounds
   d) 20 pounds

8. Liraglutide and orlistat are approved in Canada for use as adjunct to behavioural interventions for weight management in adult patients who, regardless of the presence of weight-related comorbidity, have a BMI of:
   a) \( \geq 25 \text{ kg/m}^2 \)
   b) \( \geq 27 \text{ kg/m}^2 \)
   c) \( \geq 30 \text{ kg/m}^2 \)
   d) \( \geq 35 \text{ kg/m}^2 \)

9. Liraglutide and orlistat are approved in Canada for use as adjunct to behavioural interventions for weight management in adult patients who have weight-related comorbidity, and who have failed a previous weight management intervention, and have a BMI of:
   a) \( \geq 25 \text{ kg/m}^2 \)
   b) \( \geq 27 \text{ kg/m}^2 \)
   c) \( \geq 30 \text{ kg/m}^2 \)
   d) \( \geq 35 \text{ kg/m}^2 \)

10. In the presence of weight-related comorbid conditions, bariatric surgery may be a good treatment option for carefully selected patients who have BMI of:
    a) \( \geq 25 \text{ kg/m}^2 \)
    b) \( \geq 27 \text{ kg/m}^2 \)
    c) \( \geq 30 \text{ kg/m}^2 \)
    d) \( \geq 35 \text{ kg/m}^2 \)
References


