PHARMACIST & TECHNICIAN CE:

When a Loss Becomes a Win - Overview of Weight Loss Pharmacotherapy

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Obesity is a complex chronic disease that presents variably across patient populations. The World Health Organization (WHO) defines overweight and obesity as abnormal or excessive fat accumulation that presents a risk to health.[1] Obesity screening tools include body mass index (BMI) and waist circumference, but each has its own limitations based on patient-specific factors.[2] While obesity is a widely used term, the condition is sometimes referred to as adiposity-based chronic disease. In the United States, obesity has steadily increased in prevalence since 1999.[3] Data in figure 1 shows that about 70.7% of Wisconsin residents are impacted by overweightness or obesity.[4] Looking back half a century, the average BMI in the United States was 25.7 kg/m^2, just barely passing the Centers for Disease Control and Preventions (CDC) overweight threshold of 25.0 kg/m^2 in 1971.[5,6] By 2020, the average BMI in the US had grown to 30.0 kg/m^2, which is the threshold for Class I obesity according to the CDC.

CE FOR **PHARMACISTS & TECHNICIANS**

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Learning Objectives

- Define obesity and classify obesity and risk of obesity related comorbidities.
- Describe the pathophysiology and impact of obesity.
- List comprehensive lifestyle interventions recommended for weight loss.
- Identify the general treatment approach and the role of weight loss pharmacotherapy in obesity management.
- Discuss the mechanism, efficacy, safety, monitoring, and place in therapy of pharmacologic agents for the treatment of obesity.
- Describe the role of the pharmacy team in optimizing obesity management.

Abstract

Obesity is a complex metabolic condition that needs chronic management. Incidences of obesity are increasing in the United States, leading to an increase in many obesity-related comorbidities that result in poor health outcomes. Obesity originates from multiple factors, both intrinsic and extrinsic to a person. A holistic approach, including comprehensive lifestyle interventions, pharmacological agents, and metabolic surgery or devices should be considered for the chronic management of obesity. Recognizing the chronic nature of this disease, a growing number of new pharmacologic agents are now approved by the Food and Drug Administration or in the pipeline. Along with the classification, risk stratification, pathophysiology, etiology, and lifestyle interventions, this article aims to provide a detailed overview of the approved pharmacotherapy for weight loss.

Impact of Obesity

Alongside this marked growth in obesity prevalence, the US has seen similarly increasing rates in comorbid conditions associated with obesity.[7,8] Health conditions with notable growth on par with average BMI include asthma, coronary artery disease, chronic kidney disease, diabetes mellitus, hypertension, heart failure, major depressive disorder, and metabolic dysfunction-associated steatotic liver disease. Additionally, several forms of cancer have been linked to increases in obesity, as well as an increased risk of cancer-related mortality.[9] Cancers most frequently linked to obesity in the US include breast, colorectal, esophageal, kidney, gallbladder, uterine, pancreatic, and liver cancers.

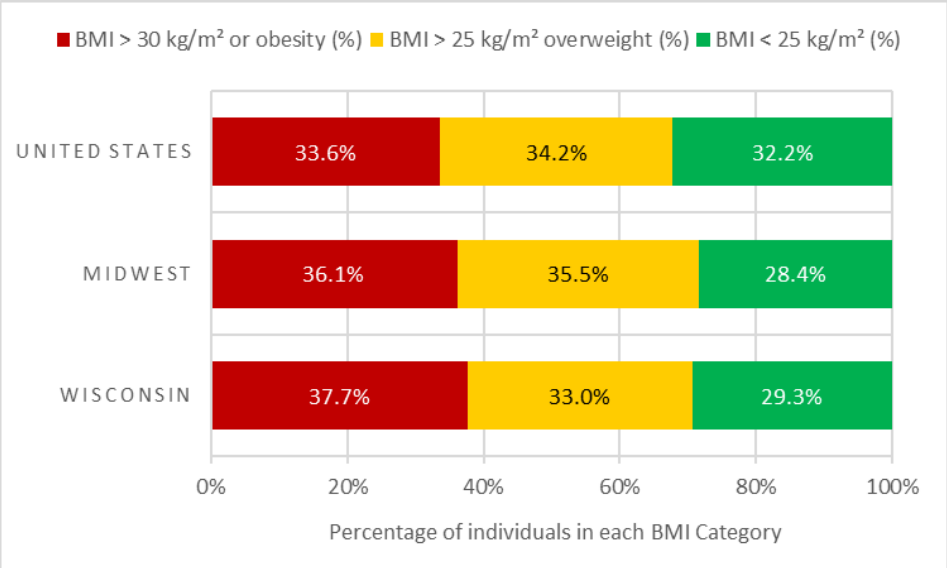
Obesity has a multifactorial etiology, with a significant impact on physiologic functions including those that are immunologic, inflammatory, and hormonal.[10] Due to its complex etiology and health consequences, obesity should be treated as a chronic disease.[11] Although healthcare payers are slow to accept obesity as a chronic disease, the scientific and medical communities largely consider obesity a chronic condition. Recently, there has been an increased number of pharmacotherapeutic agents approved by the Food and Drug Administration (FDA) for chronic obesity management, as well as increased utilization of these agents for the treatment of obesity. This article aims to provide an overview of obesity classification, risk stratification, pathophysiology, etiology, and lifestyle interventions, as well as an assessment of safety, efficacy, and place in therapy of the pharmacotherapeutic options available for weight loss.

Pathophysiology and Etiology

Obesity features an excess of adiposity, a physiologic state contributing to metabolic dysfunction in lipids, glucose, cardiac, endocrine, hepatic, immunologic, intestinal, pulmonary, and reproductive systems.[12] In an abundance of food, the body enhances its ability to store fat, leading to enhanced lipolysis and the release of excessive fatty acids. This induces lipotoxicity, increasing oxidative stress and inflammation in both adipose tissue and surrounding organs.[11] An excess of stored fat may not seem inherently harmful, but its debilitating effects on metabolic functions make obesity a significant threat to one's overall health.

Causes of obesity are multifold. There are genetic conditions that may cause hyperphagia and extreme obesity; examples include leptin receptor and proopiomelanocortin deficiencies.[13] Health conditions such as Cushing's syndrome and various psychiatric conditions like depression, eating disorders, and schizophrenia can lead to weight gain. Environmental factors such as abundance of food supply, Western civilization, advances in technology and automation, pervasive food advertising, etc. are external factors contributing to obesity. Additionally, many medications from drug classes such as antipsychotics, antidepressants, glucocorticoids, injectable progestins, anticonvulsants, and anticholinergics can increase the risk of weight gain.[14] Aside from fluid retention and related weight gain, the main etiology of obesity is the imbalance in the hunger and satiety hormones.[15] Increase in hunger hormones such as ghrelin and decrease in satiety hormones such as amylin, leptin, peptide YY, glucose-dependent insulinotropic polypeptide, glucagon like peptide-1 (GLP-1), and cholecystokinin can lead to increased weight.

FIGURE 1. Body Mass Index (BMI) Distribution in the United States, Midwest, and Wisconsin[4,7]



Obesity Classification and Comorbitity Risks

Obesity screening tools include BMI and waist circumference, but each when used alone has its own limitations.[2] Body mass index is solely used by the WHO to classify obesity. Various disease states or body composition variance may affect and lead to misclassification of individuals' BMI, including those with higher muscle mass, frailty, and heart failure. Therefore, it is recommended to assess both BMI and waist circumference (in inches) together to determine a patient's obesity-related comorbidity risk.[13] Table 1 describes the risk of obesity-related comorbidities for various BMI and waist circumference levels. While not an exhaustive list, some of the obesity-related comorbidities include prediabetes, diabetes, hypertension, dyslipidemia, cardiovascular disease, non-alcoholic fatty liver disease, polycystic ovarian syndrome, female infertility, male hypogonadism, obstructive sleep apnea, asthma or other reactive airway disease, osteoarthritis, gastroesophageal reflux disease, urinary stress incontinence, and depression.

Guidelines and Treatment Approach

The recommendations for how to manage obesity as a chronic condition are published by the American College of Cardiology/American Heart Association/ The Obesity Society, the American Association of Clinical Endocrinologists/ American College of Endocrinology, American Diabetes Association (ADA), and most recently by the American Gastroenterology Association (AGA).[13,16-18]

These guidelines recommend the use of pharmacologic agents as an adjunct to comprehensive lifestyle interventions when BMI is ≥ 30 kg/m^2 with or without risk factors or when BMI is ≥ 27 kg/m^2 with at least one comorbidity such as hypertension or diabetes. Selection of pharmacotherapy is generally recommended based on the weight loss efficacy, contraindications, tolerance, presence of comorbidities, administration route and frequency, cost, and patient preference via shared decision making. Concomitant medications that increase weight should also be addressed. Patients with BMI of ≥ 35 kg/m^2 regardless of comorbidities can be recommended for bariatric surgery or placement of weight loss devices.[19] These interventions can also be considered for patients with BMI 30-34 kg/m^2 with metabolic diseases.

Generally, the goal of treatment is to achieve 5-10% of total body weight loss, but depending on the obesity-related comorbidities, additional clinical goals can be set and achieved.[13,16-18] Examples of such goals include prevention of type 2 diabetes, lowering blood pressure or hemoglobin A1c, decreasing the number of medications used for management of a chronic condition, and improvement in general well-being.[18]

Comprehensive Lifestyle Interventions

Comprehensive lifestyle interventions (CLI) are a very important aspect of obesity management at all stages. These interventions include healthy diet, adequate physical activity, and consistent sleep routine, as well as behavioral modifications.[13,16,18] No specific diet is recommended over others; however, it is recommended that dietary changes should consider patient preference, health status, and ability to consistently adhere to a diet. The ADA guidelines for management of obesity in patients with diabetes recommend prioritizing adequate intake of protein and healthy fats, while limiting intake of rapidly digested carbohydrates.[18] Aiming for 200-300 minutes of moderate to high intensity aerobic activity along with resistance training 2-3 times per week is recommended for promotion of weight loss.[13,16] Behavioral modification strategies include collaborative goal setting, accountability, cognitive restructuring, problem solving, stimulus control, self-monitoring of diet, weight, and activity level, motivational interviewing, and relapse prevention.[20] While there is no consensus on a set number of hours of sleep per day bringing health benefits, circadian desynchrony has been associated with an increased risk of metabolic syndrome and cardiovascular diseases.[21] Therefore, it is important to prioritize a consistent, restful sleep schedule. With overwhelming data demonstrating how nonoptimal diet, sedentary lifestyle, and chronic sleep disturbances increase the risk of obesity, interventions in these areas are necessary for all patients at risk to reduce health complications and improve clinical outcomes.

TABLE 1. Obesity Classification and Risk of Obesity-related Comorbidities[3,5,6]

Classification	BMI (kg/m²)	Comorbidity Risk	Waist Circumference	
			Men ≤ 40 inches Women ≤ 35 inches	Men > 40 inches Women > 35 inches
Underweight	< 18.5	Low		
Normal	18.5 - 24.9	Average		
Overweight	25.0 - 29.9	Increased	Increased	High
Class I	30.0 - 34.9	Moderate	High	Very High
Class II	35.0 - 39.9	Severe	Very High	Very High
Class III	≥ 40	Very severe	**Extremely High**	**Extremely High**

Pharmacotherapeutic Options and Clinical Evidence

Pharmacotherapy for weight loss includes short-term (≤ 12 weeks) options, such as phentermine, and long-term options, such as orlistat, phentermine-topiramate, bupropion-naltrexone, liraglutide, semaglutide, and tirzepatide.[17,22] All of the long-term agents except for tirzepatide are approved for patients ≥ 12 years of age, providing treatment options for management of pediatric obesity. These agents are contraindicated in pregnancy and not recommended for use during breastfeeding. Efficacy of these agents has been established when compared with placebo in clinical trials. Additionally, the use of multiple anti-obesity pharmacotherapy agents or use with bariatric surgery and weight loss devices is not supported by evidence. Table 2 summarizes available agents, dosing, contraindication, common adverse events, and monitoring for the FDA-approved weight loss drugs. Table 3 details efficacy of these agents in comparison to placebo. Although not seen or used commonly, metreleptin and setmelanotide are approved for very specific patient populations suffering from obesity. Metreleptin is approved for use in patients with leptin deficiency in congenital or acquired generalized lipodystrophy.[23] Setmelanotide is approved for use in patients ≥ 6 years with obesity with suspected pathogenic proopiomelanocortin, proprotein convertase 1, or leptin receptor deficiencies.[24]

Amphetamine Derivatives

Agents like phentermine and diethylpropion are sympathomimetic amines approved by the FDA for short-term (≤ 12 weeks) use for obesity treatment. They work by increasing levels of norepinephrine in CNS, increasing sympathetic tone which results in anorexigenic effects.[25] While approved for short-term use, practitioners prescribe these medications off-label for longer terms to treat obesity.[17] The more commonly used agent out of the two is phentermine, which is available in two different formulations: capsules in 15 mg, 30 mg, and 37.5 mg and tablets in 8 mg and 37.5 mg.[25,26] Taken before meals, the maximum daily dose for phentermine is 37.5 mg. Severe renal dysfunction prohibits use and eGFR between 15-29 would limit

the daily dose to 15 mg per day.

The evidence for phentermine comes from eight short-term randomized controlled trials (RCTs) ranging from 12 to 28 weeks in duration.[17] Patients in these studies were 34-46 years of age, with a BMI of 29-38 kg/m^2 with no or controlled comorbidities. The meta-analysis of three studies reporting percent total body weight loss (TBWL) showed that patients taking phentermine had a mean difference (MD) in TBWL of 3.63% compared to placebo (95% CI, 2.97-4.29%), equaling 4.74 kg weight loss from baseline. In clinical trials, the common reasons for discontinuation of the drug were side effects such as elevated blood pressure and heart rate, palpitations, irritability, insomnia, headache, dry mouth, nausea, and constipation. The observational data, however, do not show significant increase in blood pressure or heart rate.[27] The general recommendation is to avoid use of phentermine in patients with a history of cardiovascular disease and uncontrolled hypertension and periodically monitor blood pressure and heart rate.[17] Phentermine should also be avoided in patients with concerns of arrhythmias, seizures, and untreated hyperthyroidism. It should be separated from use of monoamine oxidase inhibitors by 14 days. While it is prescribed as a chronic medication for weight loss, due to the lack of long-term studies assessing its safety and efficacy, the AGA guidelines recommends phentermine use for weight loss with low certainty evidence.

Lipase Inhibitor

Orlistat is a lipase inhibitor that works by inhibiting absorption of dietary fats when taken within one hour of eating a meal containing fat.[28] The prescription version of the product is 120 mg capsules while the over-the-counter version is available in 60 mg capsules. No renal or

TABLE 2. FDA-Approved Pharmacotherapeutic Options for Weight Loss

Class	*Medication*	*Dosing*	*Contraindications*	*Common Adverse Effects*	*Monitoring Parameters*
Amphetamine Derivatives[25]	**Phentermine** Adipex-P® Lomaira®	15-37.5 mg PO daily in the morning 8 mg PO TID 30 minutes before meals	CV disease, HTN, hyperthyroidism, history of drug abuse, glaucoma, use within 14 days of MAOI	Dizziness, constipation, xerostomia, tremor, palpitations, tachycardia, psychosis, insomnia, irritability, anxiety	Weight and waist circumference every month, CNS effects, HR, BP
Lipase Inhibitor[28]	**Orlistat** Xenical® Alli® (OTC)	120 mg (Rx) or 60 mg (OTC) PO TID with meals containing fat	Chronic malabsorption syndrome, cholestasis	Oily spotting, flatus with discharge, fecal urgency, increased defecation, fecal incontinence	Weight
Combination Agents	**Phentermine-topiramate**[29] Qsymia®	3.75/23 mg PO daily for 14 days then 7.5/46 mg PO daily	Use within 14 days of MAOI, glaucoma, hyperthyroidism	Tachycardia, decreased serum bicarbonate, constipation, xerostomia, headache, paresthesia	Weight, serum bicarbonate, serum creatinine, potassium, glucose, BP, mood disturbances, symptoms of glaucoma and acidosis
	Naltrexone-bupropion[30] Contrave®	8/90 mg PO daily for 7 days, 8/90 mg PO BID for 7 days, 16/180 mg PO QAM and 8/90 mg PO QPM for 7 days, then 16/180 mg PO BID thereafter	Chronic opioid use, acute opioid withdrawal, hypertension, seizure disorder, eating disorder, discontinuation of alcohol, use within 14 days of MAOI	Headache, insomnia, nausea, constipation, vomiting	Weight, BP, HR, blood glucose, renal and liver function; mental status assessment for depression, suicidal ideation, anxiety, mania
GLP-1 Receptor Agonists	**Liraglutide**[31] Saxenda®	0.6 mg SC daily for 1 week, then increase by 0.6 mg every week to the target dose of 3 mg SC daily	History of MTC, MEN2	Diarrhea, nausea, vomiting, constipation, abdominal pain, hypoglycemia (in type 2 diabetes patients taking other anti-hyperglycemics)	Weight, serum creatinine, triglycerides, signs and symptoms of pancreatitis and gallbladder disease, mood/behavior, HR; in patients with diabetes - A1c, blood glucose, and diabetic retinopathy screening
	Semaglutide[32] Wegovy®	0.25 mg SC weekly for 4 weeks, 0.5 mg SC weekly for 4 weeks, 1 mg SC weekly for 4 weeks, 1.7 mg SC weekly for 4 weeks, 2.4 mg SC weekly thereafter			
GLP-1/GIP Receptor Agonists	**Tirzepatide**[22] Zepbound®	2.5 mg SC weekly for 4 weeks, increase dose by 2.5 mg SC every 4 weeks; recommended maintenance dose: 5 mg, 10 mg, or 15 mg weekly			

Abbreviations: MD - mean difference, % TBWL - percent total body weight loss, PO - by mouth, TID - three times per day, BID - two times per day, CV - cardiovascular, HTN - hypertension, MAOI- monoamine oxidase inhibitor, HR - heart rate, BP - blood pressure, CM - centimeters, SC - subcutaneous, MTC - medullary thyroid carcinoma, MEN2 - multiple endocrine neoplasia type 2, A1c - hemoglobin A1c

hepatic dose adjustments are recommended.

Twenty-eight RCTs ranging from 48 weeks to four years have evaluated the outcomes of the 120 mg orlistat.[17] Patients in these trials were 42 to 58 years of age with a BMI range of 33-36 kg/m^2. Sixteen of these 28 trials assessed data for percent TBWL. A meta-analysis of these trials showed that orlistat 120 mg regimen resulted in an MD in TBWL of 2.78% when compared with placebo (95% CI, 2.36-3.20%). Compared to placebo, significant number of patients achieved ≥ 5% and ≥ 10% TBWL. Of note, 60 mg orlistat was not evaluated in these studies. Benefits of this drug come with sizable side effects including flatulence, oily stools and spotting, fecal urgency, fecal incontinence, and risk of cholelithiasis.[28] Taking a fiber supplement can help decrease these side effects. Because it inhibits absorption of fats, it is recommended that patients on this treatment take multivitamins containing fat soluble vitamins A, D, E, and K, two hours apart from orlistat. This is also the reason why patients with chronic malabsorption, inflammatory bowel disease, celiac disease, and history of bariatric surgery should avoid using orlistat. Contrasting the weight loss achieved with orlistat with its gastrointestinal (GI) side effects, the AGA guidelines recommend against use of orlistat for weight loss with the caveat that it can be considered in patients who determine the benefit/risk ratio by placing high value on the potential weight loss and low value on the GI side effects.[17]

Phentermine-topiramate

Phentermine's anorexigenic effects are augmented when combined with topiramate.[29] Topiramate was first approved as an anti-epileptic, but anorexia was recognized to be a common side effect of the drug. This was due to topiramate's effects on appetite suppression and satiety enhancement. The extended-release oral capsule, available as 3.75 mg/23 mg, 7.5 mg/46 mg, 11.25 mg/69 mg, and 15 mg/92 mg strengths, is taken once daily. Use in severe renal or hepatic dysfunction is not recommended, but in moderate dysfunction, daily dose is limited to 7.5 mg/46 mg.

Evidence for phentermine-topiramate 15 mg/92 mg comes from three RCTs.[17] Ranging from 52 to 56 weeks in length, the studies included patients 42-51 years of age with BMI 27-45 kg/m^2 with one or more comorbidities including diabetes. Compared to placebo, phentermine-topiramate resulted in an MD in TBWL of 8.45% (95% CI, 7.98-9.01%). About 68% of patients taking the 15 mg/92 mg dose achieved ≥ 5% and ≥ 10% of weight loss compared to 19.4% of patients taking placebo. In one of the three trials, 31.5% of patients taking the 15 mg/92 mg dose were able to lose ≥ 15% of total body weight compared to 3.3% of patients who took placebo. Patients of childbearing age should be advised to use appropriate contraception while taking this drug due to topiramate's teratogenicity.[29] Additionally, due to inhibition of carbonic anhydrase, it can cause metabolic acidosis and hypercalciuria resulting in increased risk of nephrolithiasis. The AGA guidelines recommend phentermine-topiramate alongside lifestyle modifications, with it being a preferred treatment option in people who have comorbid migraines.[17] With the phentermine entity, this combination should be avoided in patients with uncontrolled hypertension, existing cardiovascular disease, and untreated hyperthyroidism. Blood pressure and heart rate should be monitored in patients taking this drug combination. While not FDA-approved, the AGA guidelines state that some physicians may use topiramate monotherapy or another antiepileptic, zonisamide, off-label as anti-obesity medications.

Naltrexone-bupropion

Exact mechanism of how naltrexone—an opioid antagonist—and bupropion—a weak inhibitor of dopamine and norepinephrine—assist with weight loss is not fully understood.[30] Impact on satiety centers in the hypothalamus and on the dopamine reward pathway is believed to regulate one's food intake. Available as an extended release tablet, it follows a weekly titration schedule to reach to the maximum of 32 mg/360 mg daily dose. In severe renal and hepatic dysfunction, use is not recommended while the maximum daily dose is 8 mg/90 mg in patients with moderate renal or hepatic dysfunction.

Five RCTs assessed naltrexone-bupropion efficacy against placebo for 56 weeks.[17] Patients were on average 43-61 years of age with a mean BMI of 36 kg/m^2. Meta-analysis of the five studies show that naltrexone-bupropion resulted in an MD in TBWL of 3.01% (95% CI, 2.47-3.54%), equivalent to 3.01 kg weight loss. Significantly more patients achieved ≥ 5% and ≥ 10% TBWL in the naltrexone-bupropion group compared to placebo. One study reported that significantly more patients taking naltrexone-bupropion achieved ≥ 15% TBWL. Most commonly reported side effects were constipation, nausea, vomiting, headache and insomnia. Taking the second dose of the day a little earlier may minimize insomnia. It should not be used in patients with active opioid treatment, seizure disorders, and history of eating disorders.[17,30] Especially in the first 12 weeks of treatment, there should be periodic measurement of blood pressure and heart rate. The AGA recommends considering naltrexone-bupropion in patients who are also attempting smoking cessation and/or in patient with depression.

Glucagon Like Peptide-1 Receptor Agonist

Liraglutide and semaglutide are glucagon-like peptide-1 receptor agonists (GLP-1 RAs).[31,32] They induce glucose dependent insulin release, delay gastric emptying, and improve satiety. These mechanisms result in decreased caloric intake and weight loss. Both are administered via subcutaneous injection using a pen. Liraglutide's starting dose is 0.6 mg daily, titrated to a maintenance dose of 3.0 mg daily over four weeks based on tolerance. Semaglutide's starting dose is 0.25 mg weekly, titrated to a maintenance dose of 2.4 mg weekly over 16 weeks. More than two missed doses may require lowering the next dose due to increased risk of GI side effects. Dose adjustments for renal or hepatic dysfunctions are not provided; however, patients with renal dysfunction may experience more GI side effects needing to reassess dosing.

Eleven RCTs evaluated the efficacy of liraglutide against placebo when used for 52 weeks.[17] These studies included patients with a mean age of 43-59 years and a BMI range of 31.3-40.1 kg/m^2 with comorbidities like type 2 diabetes. Out of 11, eight studies provided percent TBWL data. The meta-analysis showed that liraglutide resulted in an MD in TBWL of 4.81% (95% CI, 4.23-5.39%) when

compared with placebo. Significantly more patients taking liraglutide demonstrated ≥ 5%, ≥ 10%, and ≥ 15% weight loss. Semaglutide was assessed against placebo in eight RCTs from 52 to 72 weeks. Patients in these trials were 46-59.5 years of age with a BMI range of 32-39.9 kg/m^2. Patients taking semaglutide resulted in an MD in TBWL of 10.76% (95% CI, 8.73–12.80%) when compared with placebo. A pooled analysis showed that significantly more patients taking semaglutide had ≥ 5%, ≥ 10%, and ≥ 15% TBWL compared to placebo. The GLP-1 RAs are associated with common GI adverse events such as nausea, vomiting, abdominal pain, flatulence, dyspepsia, eructation, diarrhea, and constipation as well as headache, fatigue, and dizziness.[31,32] Breaking down meals in small quantities only until just full and eating more frequently throughout the day can help decrease some of these GI adverse effects. They are contraindicated in patients with personal or family history of medullary thyroid carcinoma or multiple endocrine neoplasia type 2 syndrome and should be used cautiously in patients with acute pancreatitis or gallbladder disease. Cases of acute kidney injury and increased heart rate are reported with the use of GLP-1 RAs. The package inserts for these GLP-1 RAs include warning for increased risk of suicidal behavior and ideation; however, FDA issued an update of the ongoing evaluation in January 2024.[33] The preliminary evaluation does not link this risk to GLP-1 RAs, but results from a large meta-analysis and postmarketing data analysis remain pending. Both of these agents are studied in patients with diabetes, but when used along with other anti-hyperglycemic agents, due to risk of hypoglycemia, dose adjustment and necessary blood glucose monitoring should be exercised. Due to the magnitude of weight loss, the AGA recommends prioritizing semaglutide 2.4 mg over other approved weight loss medications.[17] The AGA guidelines also recommend liraglutide for weight loss with moderate certainty evidence. These medications should not be used with other GLP-1 RAs or with dipeptidyl peptidase-4 inhibitors.

Tirzepatide

Tirzepatide is a dual GLP-1 and glucose-dependent insulinotropic polypeptide (GIP) receptor agonist first approved for the treatment of type 2 diabetes mellitus.[22] It gained an additional FDA-approved indication for obesity management in late 2023 following the results of SURMOUNT-2 trial.[34] It is available as subcutaneous injection pen in four different strengths.[22] The starting dose is 2.5 mg weekly for four weeks then increasing to 5 mg thereafter. Per patient response, the dose can be increased by 5 mg to a maximum of 15 mg per week.

The evidence for tirzepatide is available from four SURMOUNT trials ranging from 72 to 88 weeks.[35-38] Patients in these trials were 32 to 65 years old with or without comorbidities and with BMI range of 29.5-45 kg/m^2. The mean difference in TBWL when compared to placebo was 11.9-17.8% (p< 0.001) in SURMOUNT-1, 9.6-11.6% (p<0.0001) in SURMOUNT-2, 20.8% (p<0.001) in SURMOUNT-3, and 19.4% (p<0.001) in SURMOUNT-4 trial. Significantly more patients in all tirzepatide groups achieved TBWL of ≥ 5%, ≥ 10%, ≥ 15%, and ≥ 20%. The safety profile of tirzepatide is similar to the GLP-1 RAs. Additionally, it decreases serum concentration of hormonal contraceptives, needing to use non-oral contraceptives or a barrier method for four weeks after initiation and four weeks after each dose escalation.[22] While these weight loss drugs are not compared head to head, tirzepatide's weight loss magnitude from the SURMOUNT trials seems to be higher than that from other weight loss drugs. At the

TABLE 3. Efficacy of Weight Loss Medications in Comparison to Placebo[17,35-38]

Medication	*MD % TBWL[a]*	*Mean weight loss (kg)*	*≥ 5% TBWL[b]*	*≥ 10% TBWL[b]*	*≥ 15% TBWL[b,c]*	*≥ 20% TBWL[b,c]*
Phentermine	3.63% (2.97-4.29)	4.74	4.12 (3.04-5.59)	5.10 (3.02-8.61)	Not available	Not available
Orlistat	2.78% (2.36-3.2)	2.81	1.71 (1.55-1.88)	1.94 (1.70-2.22)	Not available	Not available
Phentermine-topiramate	8.45% (7.89-9.01)	7.73	3.48 (3.13-3.87)	6.33 (5.26-7.61)	9.51 (5.86-15.44)	Not available
Naltrexone-bupropion	3.01% (2.47-3.54)	3.01	2.18 (1.41-3.37)	3.04 (1.80-5.14)	3.88 (2.13-7.08)	Not available
Liraglutide	4.81% (4.23-5.39)	5.30	2.09 (1.80-2.42)	2.67 (2.14-3.34)	3.04 (2.25-4.12)	Not available
Semaglutide	10.76% (8.73-12.8)	10.81	2.74 (2.21-3.40)	5.25 (3.61-7.64)	7.82 (5.19-11.76)	Not available
Tirzepatide	9.6 - 20.8% (p<0.001)[d-g]	9.7-25[d-g]	34.6 (19.2-62.6)[h]	34.7 (17.6-68.3)[h]	48.2 (19.2-121.0)[h]	40.4 (12.2-133.8)[h]

Abbreviations: MD - mean difference, TBWL - total body weight loss, kg - kilogram
[a] Mean difference (MD) in percent total body weight loss (TBWL) (95% CI)
[b] Risk ratio (95% CI)
[c] Studies of certain drugs did not evaluate ≥ 15% or ≥ 20% TBWL
[d] SURMOUNT-1: MD % TBWL 11.9-17.8% (p<0.001); mean weight loss 10.1 kg
[e] SURMOUNT-2: MD% TBWL 9.6-11.6% (p<0.0001); mean weight loss 9.7-11.6 kg
[f] SURMOUNT-3: MD% TBWL 20.8% (p<0.0001); mean weight loss 25 kg
[g] SURMOUNT-4: MD% TBWL 19.4% (p<0.0001); mean weight loss 15.8 kg
[h] SURMOUNT-3 results; ≥5 to ≥20% results reported as odds ratio (95% CI)

time the AGA guidelines were published, tirzepatide only had an FDA-approved indication for type 2 diabetes and therefore it has not been given a recommendation as weight loss medication; however, the guidelines do discuss the impact of this medication on weight loss per the clinical trials analyzed.[17]

Current Studies and Future Developments

Oral semaglutide is approved for treatment of type 2 diabetes, but not for weight management. A phase 3 randomized, double blinded, placebo-controlled trial, OASIS-1, evaluated oral semaglutide 50 mg daily against placebo in patients without type 2 diabetes.[39] Patients taking oral semaglutide showed an MD in TBWL of 12.7% compared to placebo (95% CI, 11.3-14.2%). Significantly more patients also achieved ≥ 5%, ≥ 10%, ≥ 15%, and ≥ 20% TBWL compared to placebo.

Oforglipron is an oral nonpeptide GLP-1 receptor agonist that was evaluated for weight loss against placebo in the GZGI trial.[40] The primary endpoint was percent change in total body weight at 26 weeks with four different oforglipron doses. All four doses showed significant reduction in body weight compared to placebo at 26 weeks. Oforglipron is not currently approved for weight loss.

In addition to these two agents, several other drug classes such as amylin receptor agonists, sodium-glucose transport protein 2 inhibitors, a glabridine analogue, leptin sensitizers, oxytocin, botulinum toxin type A, methylphenidate, taste receptor activators, and GIP/GLP-1/glucagon triple agonists are being investigated for the possible use as weight loss agents.[41]

Inappropriate Medication Use for Weight Loss

There are a handful of pharmacologic and herbal and complementary agents that people may use to achieve weight loss. Most commonly, these are sympathomimetics other than phentermine, levothyroxine, metformin, laxatives, and over the counter supplements for weight loss.[42] Some medications even have a warning against the use for weight loss; for example, levothyroxine includes a boxed warning against the use for the treatment of obesity or weight loss.[43] An evaluation of emergency department (ED) visits in the United States from 2004 through 2013 showed that herbal and complementary products for weight loss resulted in estimated 25.5% of ED visits (95% CI, 23.1-27.9%).[44] Patients might be using certain herbal supplements and complementary medications with weight loss benefits or claims; therefore, obtaining a full medication history, reviewing the risks/benefits of these agents, and providing patient education is imperative. Being familiar with these agents and recognizing when they are being prescribed or used for weight loss is critical to reducing harmful misuse.

Role of the Pharmacy Teams

Pharmacists can affect outcomes for patients through management of anti-obesity as well as concurrent medications. Implementation of a clinical pharmacist in an interdisciplinary weight loss service has been shown to significantly improve the magnitude of weight loss in comparison to primary care-driven care.[45] Anti-obesity medications have various safety concerns and contraindications; pharmacist-led interventions can address these safety concerns by assessing appropriateness of drug, monitoring and managing side effects, and avoiding drug interactions. For example, orlistat may decrease absorption of fat-soluble vitamins A, D, E, and K and patients should be advised to take a multivitamin containing these vitamins, administered at least two hours apart. Naltrexone-bupropion may enhance seizure potentiating medications and may also result in acute opioid withdrawal if used with chronic opioid therapy. The GLP-1 RA medications can lower blood sugar when taken with other glucose lowering therapies which may increase the likelihood for hypoglycemia. Such nuances can be recognized as part of full medication evaluation by the pharmacist and appropriate interventions can be recommended to providers and patients to assure increased benefits while reducing harm from anti-obesity medications. Additionally, many of these medications require a patient to learn how to use them with a variety of patient education techniques, including demonstration and teach-back.

Furthermore, patients may face coverage issues when prescribed anti-obesity medications because payers have not completely accepted the fact that obesity is a chronic condition. Facing the high demand of GLP-1 RAs, there has been a prolonged shortage of these agents.[46] These reasons may divert patients to source their medications from non-FDA-approved agencies, risking use of counterfeit or illegal products. Pharmacy teams can educate patients to source medications safely, assist with insurance coverage or financial assistance when available, advise to avoid use of non-approved medication or supplement use in lieu of approved medications, and suggest approved therapeutic alternatives that are safe and effective. Pharmacy teams can additionally play a role in positive reinforcement, motivation, medication adherence, as well as proper dosing and monitoring of these agents. With the knowledge, trust, and relationships the pharmacy teams have with their patients, they are able to optimize care in a patient taking weight loss medications.

Conclusion

Rates of obesity are on a constant rise and so are the rates of obesity-related comorbidities. Treating overweightness and obesity as a chronic disease has been shown to reduce the onset or complications from obesity-related comorbidities. Various pharmacotherapy agents are FDA-approved for the management of obesity in conjunction with comprehensive lifestyle interventions. In clinical trials, these agents have demonstrated a varying magnitude of weight loss when compared with placebo. There are considerable safety concerns that are specific to each drug and therefore the recommendation is to select an appropriate medication not only based on efficacy, but also based on contraindications and safety concerns. Pharmacists and pharmacy teams can play a significant role in safe and optimal use of anti-obesity medications, assuring more benefits over potential harm.

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Assessment Questions

1. Which of the following parameters is included in the current classification of obesity?
   a. Body mass index
   b. Muscle mass
   c. Body fat
   d. Waist circumference

2. Which of the following are considered to be obesity-related comorbidities?
   a. Cancer
   b. Depression
   c. Diabetes
   d. All of the above

3. In obesity, the hunger hormone, _____ may be increased and the satiety hormone, _____ may be decreased.
   a. peptide YY, dopamine
   b. ghrelin, leptin
   c. amylin, GLP-1
   d. serotonin, cholecystokinin

4. JK is a 54-year-old patient with a past medical history of diabetes, hypertension, depression, and recent UTI. She is taking metformin 1000 mg PO BID, lisinopril 20 mg PO daily, paroxetine 40 mg PO daily, and cephalexin 250 mg PO Q6H. She presents for the first evaluation visit for obesity management. Reviewing her current medications, which medication has higher potential for causing weight gain?
   a. Metformin
   b. Lisinopril
   c. Paroxetine
   d. Cephalexin

5. Which should be included in the comprehensive approach to obesity?
   a. A low-calorie diet between 600 to 800 calories per day
   b. Moderate intensity exercise for 150 minutes per week
   c. Incorporate eating behavior change
   d. At least 6 hours of sleep per night

6. SA is a 44-year-old patient presenting with obesity. She was recently diagnosed with hypertension, which is managed with 2 anti-hypertensive agents. Her body mass index is 31 kg/m^2. Which of the following is an optimal nonpharmacologic recommendation to achieve health outcomes?
   a. Decrease fat intake by 10% daily.
   b. Increase physical activity to 450 minutes per week.
   c. Reduce carbohydrate intake to 60 grams per meal.
   d. Lose 5% of body weight.

7. According to guidelines and FDA-approved indications, which of the following patients is recommended to initiate a weight loss medication alongside lifestyle interventions?
   a. A 34-year-old with BMI of 28 kg/m^2 and depression
   b. A 25-year-old with BMI of 29 kg/m^2 and no other chronic conditions
   c. A 54-year-old with BMI of 25 kg/m^2 and hypertension
   d. A 48-year-old with BMI of 28 kg/m^2 and diabetes

8. Which medication is FDA-approved for short-term treatment of obesity?
   a. Phentermine
   b. High-dose semaglutide
   c. High-dose liraglutide
   d. Orlistat

9. According to the available clinical data, which medication is considered to promote the most weight loss against placebo?
   a. Orlistat
   b. Tirzepatide
   c. Liraglutide
   d. Phentermine

10. A pharmacy team member may do the following to optimize care for a patient presenting with obesity:
   a. Recommend that a patient prescribed liraglutide should take a multivitamin tablet 2 hours apart to reduce the risk of fat soluble vitamin deficiency.
   b. Discuss importing semaglutide from another country due to shortages to decrease gaps in therapy
   c. Suggest a patient start high intensity interval training to maximize weight loss
   d. Identify that sitagliptin, a DPP-4 inhibitor, needs to be discontinued before starting weight loss therapy with tirzepatide.

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