



Key messages on obesity care from the 2023 ASPEN Physician Preconference Course: A narrative review

Givi Basishvili MD¹ | Carolyn Newberry MD²  | Jeffrey I. Mechanick MD³  | Rocco Barazzoni MD, PhD⁴ | Sara Hennessy MD¹

¹Department of Surgery, UT Southwestern Medical Center, Dallas, Texas, USA

²Division of Gastroenterology, Weill Cornell Medical College, New York, New York, USA

³Divisions of Cardiology and Endocrinology, Icahn School of Medicine at Mount Sinai, New York, New York, USA

⁴Department of Internal Medicine, Trieste University Hospital, Trieste, Italy

Correspondence

Givi Basishvili, MD, Department of Surgery, UT Southwestern Medical Center, 5323 Harry Hines Blvd, Dallas, TX 75390-9158, USA.
 Email: givi.research.md@gmail.com

Abstract

Obesity is a challenging chronic disease process that continues to affect a large percentage of the population at large. With the advent of new therapeutic options and interventions and a deeper scientific understanding of obesity as a complex illness, there is hope in curtailing this evolving pandemic. In this article, we present key medical information to engage and empower nutrition-focused providers to manage obesity and its nutrition complications. The topics summarized here were presented during the 2023 American Society for Parenteral and Enteral Nutrition Preconference Physician Course and include pathophysiology and hormonal regulation of obesity, multidisciplinary care planning and nutrition risk stratification of patients, and common approaches to treatment, including lifestyle modifications, antiobesity medications, and procedures from the perspective of the nutrition specialist.

KEYWORDS

anti-obesity medications, bariatric surgery, malnutrition, obesity, sacrospenic obesity

THE PROBLEM

Obesity is a global pandemic with multiple biological, structural, and social determinants. Although multiple definitions exist, the current definition is based on a body mass index (BMI) >25 or 30 (or >23 or 25 for patients identifying as having Asian heritage), indicating overweight or obesity, respectively.¹ In the United States, obesity prevalence rates vary by geography and ethno-cultural classification.² These variations in prevalence result from complex interrelationships of social determinants of health (SDOH) and genetic predisposition.

OBESITY IS A COMPLEX DISEASE

Pathophysiology and impact

A fundamental pathophysiological derangement in obesity is the development of chronic inflammation in adipose tissue. Although the mechanism is still not fully elicited, inflammatory changes may be the result of chronic fat accumulation leading to cellular rupture in the setting of an enlarging adipose mass.³ In addition to generalized inflammation, obesity is a risk factor for most noncommunicable diseases as well as for frailty and disability.^{4,5}

This review was invited by the ASPEN Physicians Engagement Committee (PEC).

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The double burden of malnutrition

Historically, malnutrition represented inadequacy of energy and/or nutrient intake, resulting in a low BMI; however, a “double burden” of malnutrition that incorporates both undernutrition and obesity has more recently been identified.⁶ A combination of calorically dense, poor-quality foods in combination with a sedentary lifestyle have increased prevalence. In addition to poor lifestyle habits and genetic predisposition, increased rates of chronic disease in patients with obesity further enhances nutrition risk and loss of lean muscle mass, resulting in sarcopenic obesity.^{7,8}

SDOH

The conditions in which we live, learn, work, and play define our SDOH. These determinants translate into behaviors that implicate health.^{9–11} Substantial literature supports the attributable risk of disease associated with SDOH influencers, including educational status, social position, income, occupation, and ethnicity. Public policy and environmental factors that govern external forces on disease are additionally important¹² and enhance disparities. They also influence patient engagement and effective treatment plans for obesity and associated chronic diseases.

Hormonal regulation

Several hormones are implicated in regulation of appetite and satiety, including leptin, ghrelin, peptide YY, neuropeptide Y, and insulin. The latter is directly affected by intestinally secreted hormones glucagon-like peptide-1 (GLP-1) and glucose-dependent insulinotropic polypeptide (GIP), which govern palatability, motivation for food, and appetite in response to ingested nutrients.¹³ These hormones additionally stabilize blood glucose, regulate adipose deposition, and retard gut motility, enhancing satiety in the postprandial state.^{9,14–17}

Sleep and obstructive sleep apnea

Adults are recommended to have 7–9 h of sleep each night to maintain optimal health.¹⁸ Decreased sleep and poor-quality sleep, as seen in obstructive sleep apnea, have complex and interrelated impacts on weight gain.¹⁹ Hormonal imbalances from decreased sleep induce appetite stimulation and increase food cravings, making adherence to nutrition intervention challenging.^{20,21} Furthermore, individuals with decreased sleep have less energy for exercise.²²

Environmental factors

Exposure to the modern “obesogenic” environment where highly energy-density, nutritionally poor foods are readily available in the form of ultraprocessed snacks and fructose-sweetened beverages increases

obesity risk.²³ Reduction in physical activity is also associated with excess weight gain.²⁴ External factors that regulate nutrition choices and/or exercise opportunities include associated neighborhood socioeconomic status, crime rates, and accessible outdoor spaces and food stores.

OBESITY MANAGEMENT PRINCIPLES: THERAPEUTICS

Although the underlying principles of weight loss include induction of a moderate net energy deficit through changes in nutrition and physical activity,²⁵ achieving this goal is challenging. To have durable success, a nutrition-focused provider should personalize recommendations to individual need, health history, environment and SDOH. A combination of lifestyle and nutrition modifications with medically supervised interventions may be necessary to optimize health outcomes. Appropriate nutrition risk stratification and monitoring are important across treatment approaches.

Multidisciplinary teams

The involvement of a multidisciplinary team is important for successful management of obesity, a chronic disease with high risk of relapse.²⁶ An effective team includes an obesity-focused physician, a dietitian with weight loss–intervention expertise, and a psychologist who can screen for and address underlying disordered eating patterns and enhance patient engagement, outcomes, and appropriate monitoring.^{27,28}

Targeted lifestyle and behavioral modifications

Lifestyle modification lays the foundation of metabolic optimization because it establishes physical and mental commitment to enacting change. Patients following a lifestyle-based weight loss program are more likely to lose 5% of their total body weight (TBW) than patients without a plan are.²⁹ These modalities are best applied in the context of behavioral change. Additionally, weight loss is associated with alterations in neurohormonal pathways that favor weight regain via enhancement of appetite and adaptive thermogenesis, further highlighting importance of targeted behavioral modification.³⁰

Nutrition interventions

Many nutrition interventions associated with purposeful weight loss have been studied and successfully implemented. These focus on inducing energy deficit via alterations in macronutrient composition and/or nutrition patterns. In practice, differences between specific nutrition approaches are small. For cardiometabolic risk mitigation, nutrition patterns that emphasize a high intake of plant-based foods and limit red and processed meats, such as the Mediterranean diet, are well validated.^{31,32}

TABLE 1 List of FDA-approved antiobesity medications, dosing, weight loss, and contraindications.

Name and mechanism of action	Dosing guidelines	Weight loss, % TBW (dose; time)	Nonresponder rate, % (dose)	Contraindications
Tirzepatide ³⁸ incretin: GIP and GLP-1 agonist	Start at 2.5 mg weekly; increase by 2.5 mg every 4 weeks to a maximum of 15 mg weekly	15% (5 mg; 72 weeks); 19.5% (10 mg; 72 weeks); 20.9% (15 mg; 72 weeks)	15% (5 mg); 11% (10 mg); 9% (15 mg)	History of medullary thyroid carcinoma, or MEN II syndrome, pregnancy; and caution in patients with renal impairment, volume depletion, gastroparesis, and pancreatitis
Semaglutide ^{41,42} incretin: GLP-1 agonist	Start at 0.25 mg weekly; increase every 4 weeks to a maximum of 2.4 mg weekly	4.7% (0.5 mg; 52 weeks); 6.7% (1 mg; 52 weeks); 14.9% (2.4 mg; 68 weeks)	11% (2.4 mg)	History of medullary thyroid carcinoma or MEN II syndrome, pregnancy; and caution should be taken in patients with renal impairment, gastroparesis, pancreatitis, acute gallbladder disease, and suicidal history or ideations
Liraglutide ^{43,44} incretin: GLP-1 agonist	Start at 0.6 mg daily; increase by 0.6 mg weekly to maximum of 3 mg daily	5%–7% (3 mg; 20 weeks)	60% (1.8 mg); 46% (3 mg)	History of medullary thyroid carcinoma or MEN II syndrome, pregnancy; and caution should be exercised in patients with renal impairment, pancreatitis, gallstones, suicidal history or ideations
Phentermine/topiramate ⁴⁵ sympathomimetic amine/multiple ^a	Start at 3.75/23 mg daily; increase by 3.75/23 every 14 days to a maximum of 15/92 mg	3.75/23 mg (5.1%); 56 weeks; 15/92 mg (10.6%); 56 weeks)	55% (3.75/23 mg); 48% (7.5/46 mg); 30%; 15/92 mg)	Pregnancy, glaucoma, uncontrolled hyperthyroidism, and use of MAO inhibitors; caution should be utilized in patients with depression, suicidality, cognitive impairment, drug abuse, and tachycardia
Naltrexone/bupropion ⁴⁶ opioid receptor antagonist/norepinephrine and dopamine reuptake inhibitor	Start at 1 tablet of 8 mg/90 mg per day; increase weekly by adding 1 tablet to a maximum goal of 2 tablets twice daily	16/180 mg (5.0%); 56 weeks; 32/360 mg (6.1%); 56 weeks)	58% (32/360 mg)	Uncontrolled hypertension, seizure disorders, anorexia nervosa or bulimia, undergoing abrupt discontinuation of alcohol, benzodiazepines, barbiturates, and antiepileptic drugs, use of other bupropion-containing products, chronic opioid use, and MAO-inhibitor use within 14 days
Orlistat ⁴⁷ lipase inhibitor	120-mg capsule 3 times a day during or up to 1 h after a meal	1.7% (8 weeks); 4.9% (16 weeks); 5.6% (24 weeks)	57% (120 mg)	Pregnancy, chronic malabsorption syndrome, and cholestasis, and caution should be taken in patients with eating disorders, oxalate nephrolithiasis, and organ transplants

Note: Nonresponder rate (lost <5% of TBW).

Abbreviations: GIP, glucose-dependent insulinotropic polypeptide; GLP-1, glucagon-like peptide-1; MAO, monoamine oxidase; MEN II, multiple endocrine neoplasia, type II; TBW, total body weight.

^aMechanisms of action include augmentation of GABA-A receptors, carbonic anhydrase inhibition, antagonism of glutamate receptors.

Some medical nutrition interventions have specific disease-modifying goals, such as the Dietary Approaches to Stop Hypertension, which targets blood pressure reduction.³³ Nutrition interventions that are overly restrictive in energy (ie, <1000 kcal/day) require close medical supervision and should only be implemented for short periods of time in patients in need of rapid weight loss for medical optimization.^{34,35}

Physical activity

Physical activity should be prescribed in all weight loss programs according to patient medical conditions, comorbidities, and baseline exercise capacity. In addition, independently of its impact on energy balance, enhancement of physical activity carries a potential for reduction of obesity-associated metabolic derangements and enhancement of lean muscle mass.^{36,37}

Pharmacotherapy

Lifestyle and behavioral modifications alone are often not sufficient to achieve sustainable, clinically significant weight loss, which is defined by a loss of >5%–10% TBW³² for most obesity-related health outcomes.³⁴ In many patients, antiobesity medications (AOMs) play a role in sustainable intervention and are appropriate in patients with a BMI >30 or >27 with weight-related comorbidities.³⁵ The role of AOMs as an adjunct to metabolic surgery is under investigation and may require additional nutrition monitoring. Patients should initiate AOMs in combination with lifestyle and behavioral modifications to augment outcomes and reduce malnutrition risk. The newest and currently most efficacious AOMs are semaglutide and tirzepatide, which are long-acting GLP-1/GIP

receptor agonists.^{38–41} Additional AOMs regulate appetite, metabolism, and nutrient absorption (Table 1).^{45,48–51}

General approach

AOMs can be a useful adjunct to lifestyle modification. Older classes of medications result in an average of 5%–10% TBW loss, and newer classes result in an average of 10%–20% TBW. Medical and nutrition providers should personalize therapy recommendations based on associated medical and malnutrition risk, degree of obesity, tolerance, and patient preference. Continued follow-up should assess pace of weight loss, body composition changes, and changes in nutrition intake to achieve desired outcomes and mitigate malnutrition risk.

Procedural interventions

Metabolic and bariatric surgery (MBS) is an effective means of durable weight loss⁵² (Table 2). The criteria for eligibility for MBS had traditionally been a BMI >40 or BMI >35 with weight-related medical comorbidities, although patients with BMI as low as 30 can now be considered.^{58,59} BMI cutoffs for newer endobariatric procedures have remained >30. Although effective and safe, MBS and bariatric endoscopy can be associated with an increased nutrition risk and require careful patient selection and medical monitoring.

Bariatric endoscopy

Endoscopic options for weight loss include (1) gastric aspiration devices, (2) small intestinal bypass devices, and (3) endoscopic

TABLE 2 Metabolic procedures with corresponding indications, weight loss, and cautions/contraindications.

Procedure	Weight loss, TBW (time)	Indications	Cautions/contraindications
Endoscopic sleeve gastroplasty ⁵³	13.6% (52 weeks); 17.2% (24 months)	BMI: 30–40; select patients with a BMI >40, if not a surgical candidate	Gastric mucosal lesions (ulcers, severe acute gastritis), neoplastic lesions, hiatus hernia >3 cm, coagulopathy, and psychiatric disorders
Sleeve gastrectomy ⁵⁴	27.2% (1 year); 23.3% (3 years); 20.4% (5 years)	BMI >35 or BMI >30 with comorbidities	Uncontrolled mental disorders, substance abuse, Barrett esophagus, severe gastric reflux, severe esophageal dysmotility, and portal hypertension with gastric varices
Roux-en-Y gastric bypass	30% (1 year ⁵⁵); 25.3% (10 years ⁵⁶)	BMI >35 or BMI >30 with comorbidities	Uncontrolled vitamin deficiencies, uncontrolled mental disorders, substance abuse, active smoking, Crohn's disease, chronic need for high doses of steroids or NSAIDs, advanced cirrhosis, and need for endoscopic gastric surveillance
Biliopancreatic diversion	36.7% (2 years); 35.9% (5 years); 39.0% (10 years ⁵⁷)	BMI >50 or BMI >40 with DM II or weight regain after sleeve	Uncontrolled vitamin deficiencies, uncontrolled mental disorders, substance abuse, active smoking, Crohn's disease, advanced cirrhosis, severe coagulopathies, and frequent loose bowel movements

Abbreviations: BMI, body mass index; DM II, diabetes type II; NSAID, nonsteroidal anti-inflammatory drug; TBW, total body weight.

TABLE 3 Screening approaches after metabolic surgery or weight loss program.

Monitoring	Frequency
Clinic follow-up visits	Biweekly to monthly upon commencement, then every 3–6 months
Routine laboratory tests (CBC, BMP, LFT, magnesium, phosphorus, and lipid panel)	Every 6 months
Bone density scan	Every 2 years
24-h urine calcium	Annually
Vitamin B ₉ (folate), vitamin B ₁₂ (cobalamin), vitamin D	Annually
Thyroid testing	Annually
Fat-soluble vitamins (A, E, K)	Annually in BPD/DS if clinical suspicion because of symptoms of deficiency in sleeve or bypass
Vitamin B ₁ (thiamin)	If clinical suspicion because of symptoms of deficiency or elevated risk factors (female, African American, inconsistent follow-up, nausea/vomiting, furosemide use, excessive weight loss, alcohol abuse)
Trace elements (copper, zinc, and selenium)	If clinical suspicion because of symptoms of deficiency

Abbreviations: BMP, basic metabolic panel; BPD, biliopancreatic diversion; CBC, complete blood count; DDS, duodenal switch; LFT, liver function test.

suturing procedures, such as the primary obesity surgery endoluminal procedure and the endoscopic sleeve gastroplasty (ESG).⁵³ Among endoscopic interventions, ESG produces the most durable and significant weight loss outcomes (Table 2).

Bariatric surgery

Currently, sleeve gastrectomy (SG) is the most performed MBS. SG surgically removes approximately 80% of the stomach. The procedure accounts for a majority of bariatric surgical procedures in the United States and is appropriate for a broad range of patients (Table 2).^{60,61} Roux-en-Y gastric bypass is the oldest metabolic operation and involves creating a small gastric pouch and bypassing the duodenum as well as the proximal jejunum via a Y-shaped intestinal reconstruction (Table 2).⁵⁶ Duodenal switch is the most aggressive MBS and includes a combination of SG and a long segment intestinal bypass. Because of the large length of intestinal bypass, this procedure is highly effective for long-term weight loss and remission of type 2 diabetes but carries an enhanced nutrition risk.⁵⁷ Biliopancreatic diversion is also a therapeutic option for patients seeking further weight loss who have already had an SG.^{62–66}

MONITORING AND NUTRITION RISK ASSESSMENT

Frequent follow-up can help prevent complications of weight loss interventions. All appointments should include assessment of weight, standard nutrition guidelines and physical activity recommendations,

TABLE 4 Micronutrient supplementation s/p bariatric surgery.

Micronutrient	Supplementation schedule
Water-soluble vitamins	
Vitamin B ₁ (thiamin)	12–100 mg daily
Vitamin B ₉ (folate)	400–800 mcg daily
Vitamin B ₁₂ (cobalamin)	350–1000 mcg daily orally or 1000 mcg IM/SQ monthly
Fat-soluble vitamins	
Vitamin A	5000–10,000 IU/day
Vitamin D ₃	3000 IU/day until 25-hydroxyvitamin D > 30 ng/ml
Vitamin E	15 mg/day
Vitamin K	90–120 mcg/day (sleeve or RYGB); 300 mcg/day (BPD)
Minerals and trace elements	
Calcium citrate	1200–1500 mcg/day (s/p sleeve gastrectomy or RYGB); 1800–2400 mcg/day (s/p BPD)
Copper	1 mg/day (sleeve or RYGB); 2 mg/day (BPD)
Iron	18 mg/day (men/postmenopausal women); 45–60 mg/day (premenopausal women)
Multivitamins	1 tablet daily
Zinc	8–22 mg

Abbreviations: BPD, biliopancreatic diversion; IM, intramuscular; RDA, recommended daily allowance; RYGB, Roux-en-Y gastric bypass; s/p, status post; SQ, subcutaneous.

and appropriate medical testing (Table 3). Serial body composition analysis may be necessary, especially in the setting of rapid weight and muscle loss, significant intolerance of oral intake, and/or physical limitations.⁶⁷

Management of patients with high nutrition risk

Nutrition providers should screen patients at risk of weight regain as well as rapid and excessive loss leading to sarcopenia and malnutrition. Risk factors identified for weight regain include inadequate medical nutrition therapy, maladaptive eating behaviors, loss of follow-up, and minimal physical activity.⁶⁸ Patients at elevated nutrition risk should be monitored, including older adults, patients with preexisting nutrition deficiencies, those with significant gastrointestinal symptoms leading to poor oral intake, and those with bypassed anatomies (Table 4). Although skeletal muscle loss is inevitable, sarcopenic obesity—monitoring consensus algorithms have been developed.⁸ Approaches to minimize muscle loss during weight management programming include enhancing protein intake and weight-bearing exercise.

Patients who are unable to meet their nutrition needs via an oral diet alone or have evidence of significant malabsorption after surgery may be candidates for nutrition support, including both enteral and parenteral intervention.⁶⁰ Hospital admission may be required for nutrition initiation, especially in patients at high risk for refeeding syndrome. Revision of bariatric surgery may be required when serious complications, including malnutrition and malabsorption, occur that cannot be medically managed.⁶⁰

SUMMARY

Obesity is a chronic disease with multifactorial etiology involving social and behavioral factors, hormonal alterations, and environmental circumstances. Although initial management involves behavioral and lifestyle modifications, pharmacotherapy and procedural interventions may be necessary to produce long-lasting results. Patients are best managed in a multidisciplinary team who follow longitudinally and identify early signs of weight regain, sarcopenia, malnutrition, and nutrition deficiencies.

CONFLICT OF INTEREST STATEMENT

Carolyn Newberry has received speaking honorariums for Inbody and Baxter International. Jeffrey I. Mechanick has received a speaking honorarium for Abbott Nutrition and is on the advisory board and is on the advisory boards for AvetaLife and Twin Health. Sara Hennessy is a consultant and educational speaker for Boston Scientific.

ORCID

Carolyn Newberry  <http://orcid.org/0000-0002-0593-6436>

Jeffrey I. Mechanick  <http://orcid.org/0000-0002-0657-588X>

REFERENCES

1. Flegal KM, Carroll MD, Kuczmarski RJ, Johnson CL. Overweight and obesity in the United States: prevalence and trends, 1960-1994. *Int J Obes Relat Metab Disord*. 1998;22(1):39-47.
2. Adult Obesity Prevalence Maps. Centers for Disease Control and Prevention, National Center for Chronic Disease Prevention and Health Promotion, Division of Nutrition, Physical Activity, and Obesity. 2023. Accessed April 19, 2024. <https://www.cdc.gov/obesity/data/prevalence-maps.html>
3. Gasmi A, Björklund G, Mujawdiya PK, et al. Micronutrients deficiencies in patients after bariatric surgery. *Eur J Nutr*. 2022;61(1):55-67.
4. Cederholm T, Barazzoni R, Austin P, et al. ESPEN guidelines on definitions and terminology of clinical nutrition. *Clin Nutr*. 2017;36(1):49-64.
5. Pi-Sunyer X. The medical risks of obesity. *Postgrad Med*. 2009;121(6):21-33.
6. Popkin BM, Corvalan C, Grummer-Strawn LM. Dynamics of the double burden of malnutrition and the changing nutrition reality. *Lancet*. 2020;395(10217):65-74.
7. Barazzoni R, Gortan Cappellari G. Double burden of malnutrition in persons with obesity. *Rev Endocr Metab Disord*. 2020;21(3):307-313.
8. Donini LM, Busetto L, Bischoff SC, et al. Definition and diagnostic criteria for sarcopenic obesity: ESPEN and EASO consensus statement. *Obes Facts*. 2022;15(3):321-335.
9. Javed Z, Valero-Elizondo J, Maqsood MH, et al. Social determinants of health and obesity: findings from a national study of US adults. *Obesity*. 2022;30(2):491-502.
10. Barrientos-Gutierrez T, Moore KAB, Auchincloss AH, et al. Neighborhood physical environment and changes in body mass index: results from the multi-ethnic study of atherosclerosis. *Am J Epidemiol*. 2017;186(11):1237-1245.
11. Cummins S, Macintyre S. Food environments and obesity—neighbourhood or nation? *Int J Epidemiol*. 2005;35(1):100-104.
12. Braveman P, Paula, Gottlieb L. The social determinants of health: it's time to consider the causes of the causes. *Public Health Rep*. 2014;129(2):19-31.
13. Holt MK, Richards JE, Cook DR, et al. Proglucagon neurons in the nucleus of the solitary tract are the main source of brain GLP-1, mediate stress-induced hypophagia, and limit unusually large intakes of food. *Diabetes*. 2019;68(1):21-33.
14. Krieger J-P. Intestinal glucagon-like peptide-1 effects on food intake: physiological relevance and emerging mechanisms. *Peptides*. 2020;131:170342.
15. Holst JJ. Incretin hormones and the satiation signal. *Int J Obes*. 2013;37(9):1161-1168.
16. Zaffina I, Pelle MC, Armentaro G, et al. Effect of dual glucose-dependent insulinotropic peptide/glucagon-like peptide-1 receptor agonist on weight loss in subjects with obesity. *Front Endocrinol*. 2023;14:1095753.
17. Fujita Y, Wideman RD, Asadi A, et al. Glucose-dependent insulinotropic polypeptide is expressed in pancreatic islet α -cells and promotes insulin secretion. *Gastroenterology*. 2010;138(5):1966-1975.e1.
18. Watson NF, Badr MS, Belenky G, et al. Recommended amount of sleep for a healthy adult: a Joint Consensus Statement of the American Academy of Sleep Medicine and Sleep Research Society. *Sleep*. 2015;38(6):843-844.
19. Papatriantafyllou E, Efthymiou D, Zoubaneas E, Popescu CA, Vassilopoulou E. Sleep deprivation: effects on weight loss and weight loss maintenance. *Nutrients*. 2022;14(8):1549.
20. Taheri S, Lin L, Austin D, Young T, Mignot E. Short sleep duration is associated with reduced leptin, elevated ghrelin, and increased body mass index. *PLoS Med*. 2004;1(3):e62.
21. Greer SM, Goldstein AN, Walker MP. The impact of sleep deprivation on food desire in the human brain. *Nat Commun*. 2013;4:2259.

22. Fenton S, Burrows TL, Collins CE, et al. Behavioural mediators of reduced energy intake in a physical activity, diet, and sleep behaviour weight loss intervention in adults. *Appetite*. 2021;165:105273.
23. Lee A, Cardel M, Donahoo WT. Social and environmental factors influencing obesity. In: Feingold KR, Anawalt B, Blackman MR, et al. eds. *Endotext [Internet]*. MDText.com, Inc; 2000. <https://www.ncbi.nlm.nih.gov/books/NBK278977/>
24. Dewi RC, Rimawati N, Purbodjati I. Body mass index, physical activity, and physical fitness of adolescence. *J Public Health Res*. 2021;10(2):2230.
25. Wilson K. Obesity: lifestyle modification and behavior interventions. *FP Essent*. 2020;492:19-24.
26. Foster D, Sanchez-Collins S, Cheskin LJ. Multidisciplinary team-based obesity treatment in patients with diabetes: current practices and the state of the science. *Diabetes Spectr*. 2017;30(4):244-249.
27. Fabricatore AN. Behavior therapy and cognitive-behavioral therapy of obesity: is there a difference? *J Am Diet Assoc*. 2007;107(1):92-99.
28. Yu B, Chen Y, Qin H, Chen Q, Wang J, Chen P. Using multidisciplinary teams to treat obese patients helps improve clinical efficacy: the general practitioner's perspective. *Am J Transl Res*. 2021;13(4):2571-2580.
29. Curry SJ, Krist AH, Owens DK, et al. US Preventive Services Task Force. Behavioral weight loss interventions to prevent obesity-related morbidity and mortality in adults: US Preventive Services Task Force recommendation statement. *JAMA*. 2018;320(11):1163-1171.
30. Van Baak MA, Mariman ECM. Obesity-induced and weight-loss-induced physiological factors affecting weight regain. *Nat Rev Endocrinol*. 2023;19(11):655-670.
31. Willett W, Sacks F, Trichopoulos A, et al. Mediterranean diet pyramid: a cultural model for healthy eating. *Am J Clin Nutr*. 1995; 61(6 suppl):1402S-1406S.
32. Wadden TA, West DS, Delahanty L, et al. Look AHEAD Research Group. The Look AHEAD Study: a description of the lifestyle intervention and the evidence supporting it. *Obesity*. 2006;14(5):737-752.
33. Filippou CD, Tsioufis CP, Thomopoulos CG, et al. Dietary approaches to stop hypertension (DASH) diet and blood pressure reduction in adults with and without hypertension: a systematic review and meta-analysis of randomized controlled trials. *Adv Nutr*. 2020;11(5):1150-1160.
34. Gregg E, Jakicic J, Blackburn G, et al. Look AHEAD Research Group. Association of the magnitude of weight loss and changes in physical fitness with long-term cardiovascular disease outcomes in overweight or obese people with T2DM: a post-hoc analysis of the Look AHEAD randomised clinical trial. *Lancet Diabetes Endocrinol*. 2016;4(11):913-921.
35. Garvey WT, Mechanick JI, Brett EM, et al. American Association of Clinical Endocrinologists and American College of Endocrinology Comprehensive Clinical Practice Guidelines For Medical Care of Patients with Obesity. *Endocrine Practice*. 2016;22(suppl 3): 1-203.
36. Yumuk V, Tsigos C, Fried M, et al. Obesity Management Task Force of the European Association for the Study of Obesity. European Guidelines for Obesity Management in Adults. *Obes Facts*. 2015; 8(6):402-424.
37. Jabekk PT, Moe IA, Meen HD, Tomten SE, Høstmark AT. Resistance training in overweight women on a ketogenic diet conserved lean body mass while reducing body fat. *Nutr Metab (Lond)*. 2010;7:17.
38. Jastreboff AM, Aronne LJ, Ahmad NN, et al. Tirzepatide once weekly for the treatment of obesity. *N Engl J Med*. 2022;387(3):205-216.
39. US Food and Drug Administration. Highlights of prescribing information: MOUNJARO. Accessed April 19, 2024. https://www.accessdata.fda.gov/drugsatfda_docs/label/2022/215866s000lbl.pdf
40. Ahrén B, Masmiquel L, Kumar H, et al. Efficacy and safety of once-weekly semaglutide versus once-daily sitagliptin as an add-on to metformin, thiazolidinediones, or both, in patients with type 2 diabetes (SUSTAIN 2): a 56-week, double-blind, phase 3a, randomised trial. *Lancet Diabetes Endocrinol*. 2017;5(5):341-354.
41. Ghush W, De la Rosa A, Sacoto D, et al. Weight loss outcomes associated with semaglutide treatment for patients with overweight or obesity. *JAMA Netw Open*. 2022;5(9):e2231982.
42. US Food and Drug Administration. Highlights of prescribing information: OZEMPIC. Accessed April 19, 2024. https://www.accessdata.fda.gov/drugsatfda_docs/label/2017/209637lbl.pdf
43. US Food and Drug Administration. Highlights of prescribing information: VICTOZA. Accessed April 19, 2024. https://www.accessdata.fda.gov/drugsatfda_docs/label/2017/022341s027lbl.pdf
44. Davies MJ, Bergenstal R, Bode B, et al. Efficacy of liraglutide for weight loss among patients with type 2 diabetes: the SCALE diabetes randomized clinical trial. *JAMA*. 2015;314(7):687-699.
45. US Food and Drug Administration. Highlights of prescribing information: QSYMIA. Accessed April 19, 2024. https://www.accessdata.fda.gov/drugsatfda_docs/label/2012/022580s000lbl.pdf
46. US Food and Drug Administration. Highlights of prescribing information: CONTRAVE. Accessed April 19, 2024. https://www.accessdata.fda.gov/drugsatfda_docs/label/2014/200063s000lbl.pdf
47. US Food and Drug Administration. Highlights of prescribing information: XENICAL. Accessed April 19, 2024. https://www.accessdata.fda.gov/drugsatfda_docs/label/2012/020766s029lbl.pdf
48. Klein S, Romijn JA. Chapter 36: Obesity. In: *Williams Textbook of Endocrinology*. 13th ed. Elsevier; 2016:1633-1659.
49. Smith SM, Meyer M, Trinkley KE. Phentermine/topiramate for the treatment of obesity. *Ann Pharmacother*. 2013;47(3): 340-349.
50. Apovian CM. Naltrexone/bupropion for the treatment of obesity and obesity with type 2 diabetes. *Future Cardiol*. 2016;12(2): 129-138.
51. Jain S, Ramanand S, Ramanand J, Akat P, Patwardhan M, Joshi S. Evaluation of efficacy and safety of orlistat in obese patients. *Indian J Endocrinol Metab*. 2011;15(2):99-104.
52. Al Suwaidi J. STAMPEDE: bariatric surgery gains more evidence based support. *Glob Cardiol Sci Pract*. 2014;2014(1):8.
53. Hedjoudje A, Abu Dayyeh BK, Cheskin LJ, et al. Efficacy and safety of endoscopic sleeve gastrectomy: a systematic review and meta-analysis. *Clin Gastroenterol Hepatol*. 2020;18(5):1043-1053.e4.
54. Sepúlveda M, Alamo M, Saba J, Astorga C, Lynch R, Guzmán H. Long-term weight loss in laparoscopic sleeve gastrectomy. *Surg Obes Relat Dis*. 2017;13(10):1676-1681.
55. Corcelles R, Boules M, Froylich D, et al. Total weight loss as the outcome measure of choice after Roux-en-Y gastric bypass. *Obes Surg*. 2016;26(8):1794-1798.
56. Jiménez A, Ibarzabal A, Moizé V, et al. Ten-year outcomes after Roux-en-Y gastric bypass and sleeve gastrectomy: an observational nonrandomized cohort study. *Surg Obes Relat Dis*. 2019; 15(3):382-388.
57. Sethi M, Chau E, Youn A, Jiang Y, Fielding G, Ren-Fielding C. Long-term outcomes after biliopancreatic diversion with and without duodenal switch: 2-, 5-, and 10-year data. *Surg Obes Relat Dis*. 2016; 12(9):1697-1705.
58. Gastrointestinal surgery for severe obesity. *Consens Statement*. 1991;9(1):1-20.
59. Eisenberg D, Shikora SA, Aarts E, et al. 2022 American Society for Metabolic and Bariatric Surgery (ASMBS) and International Federation for the Surgery of Obesity and Metabolic Disorders (IFSO): indications for metabolic and bariatric surgery. *Surg Obes Relat Dis*. 2022;18(12):1345-1356.
60. American Society for Metabolic and Bariatric Surgery. Estimate of Bariatric Surgery Numbers, 2011-2020. 2022.

61. Public Education Committee. Bariatric surgery procedures. 2021. Accessed April 19, 2024. <https://asmbs.org/patients/bariatric-surgery-procedures>
62. Chierici A, Chevalier N, Iannelli A. Postoperative morbidity and weight loss after revisional bariatric surgery for primary failed restrictive procedure: a systematic review and network meta-analysis. *Int J Surg*. 2022;102:106677.
63. Di Lorenzo N, Antoniou SA, Batterham RL, et al. Clinical practice guidelines of the European Association for Endoscopic Surgery (EAES) on bariatric surgery: update 2020 endorsed by IFSO-EC, EASO and ESPCOP. *Surg Endosc*. 2020;34(6):2332-2358.
64. Marceau P, Biron S, Marceau S, et al. Long-term metabolic outcomes 5 to 20 years after biliopancreatic diversion. *Obes Surg*. 2015;25:1584-1593.
65. Verhoeff K, Mocanu V, Jogiat U, et al. Patient selection and 30-day outcomes of SADI-S compared to RYGB: a retrospective cohort study of 47,375 patients. *Obes Surg*. 2022;32(7):1-8.
66. Felsenreich DM, Steinlechner K, Langer FB, et al. Outcome of sleeve gastrectomy converted to Roux-en-Y gastric bypass and one-anastomosis gastric bypass. *Obes Surg*. 2022;32(3):643-651.
67. Mechanick JI, Apovian C, Brethauer S, et al. Clinical practice guidelines for the perioperative nutrition, metabolic, and nonsurgical support of patients undergoing bariatric procedures - 2019 update: cosponsored by American Association of Clinical Endocrinologists/American College of Endocrinology, the Obesity Society, American Society for Metabolic & Bariatric Surgery, Obesity Medicine Association, and American Society of Anesthesiologists - executive summary. *Endocr Pract*. 2019;25(12):1346-1359.
68. Kaouk L, Hsu AT, Tanuseputro P, Jessri M. Modifiable factors associated with weight regain after bariatric surgery: a scoping review. *F1000Research*. 2019;8:615.

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