Obesity Algorithm®

Abridged Edition

2023

obesitymedicine.org

Obesity Medicine Association

CLINICAL LEADERS IN OBESITY MEDICINE®

Table of Contents: Section # 1*

*Sections and pages not found in the free downloadable slides are found in the eBook.

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Purpose

To provide clinicians an overview of principles important to the care of patients with increased and/or dysfunctional body fat, based upon scientific evidence, supported by medical literature, and derived from the clinical experiences of members of the Obesity Medicine Association.

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Intent of Use

- The Obesity Algorithm is intended to be a "living document" updated once a year (as needed). It is intended to be an educational tool used to translate the current medical science and the experiences of obesity specialists to better facilitate and improve the clinical care and management of patients with overweight and obesity.
- This algorithm *is not* intended to be interpreted as "rules" and/or directives regarding the medical care of an individual patient.
- While the hope is many clinicians may find this algorithm helpful, the final decision regarding the optimal care of the patient with overweight and obesity is dependent upon the individual clinical presentation and the judgment of the clinician who is tasked with directing a treatment plan that is in the best interest of the patient.
- Throughout this resource is mention of "weight loss" or "reduction in body weight." In most cases, this is intended to convey a reduction in unhealthful increases in body fat (overweight and obesity), as implicit in an obesity algorithm sponsored by the Obesity Medicine Association.

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Disclaimer and Permissions

Disclaimer

- Since the original presentation by the Obesity Medicine Association (OMA) in 2013, the Obesity Algorithm® has undergone yearly updates to include the latest trends in the field of obesity medicine. The OMA Obesity Algorithm was developed to assist health care professionals provide care for patients with overweight and obesity. The Obesity Algorithm is not intended to be a substitute for a medical professional's independent judgment and should not be considered medical advice. The content herein is based on medical literature and the clinical experiences of obesity medicine specialists. In areas regarding inconclusive or insufficient scientific evidence, the authors used their professional judgment.
- The Obesity Algorithm is a working document intended to represent the state of obesity medicine at the time of publication. OMA encourages medical professionals to use this information in conjunction with, and not as a replacement for, their best clinical judgment. The presented recommendations may not be appropriate in all situations. Any decision by practitioners to apply the principles of the OMA Obesity Algorithm should be made in light of local resources, individual patient circumstances, in partnership with patient agreement, and with the knowledge of federal, state, and local laws.

Permissions

- The Obesity Medicine Association owns the copyright to the Obesity Algorithm but invites you to use the slide set. Access to the Obesity Algorithm content and/or permission for extensive quoting or reproducing excerpts and for the reproduction and use of copyrighted text, images, or entire slides will not be granted until the requestor has signed the copyright consent and permission agreement available at https://obesitymedicine.org/obesity-algorithm-powerpoint/ OMA reserves the right to deny a request for permission to use the Obesity Algorithm.

Writing Process

Transparency

This "Writing Process" section describes the processes by which the OMA Obesity Algorithm electronic documents were developed and funded. The Obesity Medicine Association (OMA) Obesity Algorithm is provided in two electronic formats.

- **Downloadable slides:** These are free to providers who visit the OMA website. They are intended to be an educational tool to assist Obesity Medicine providers better educate themselves and others. Omitted slide numbers represent content included in the eBook, but not the free downloadable slides.
- **eBook:** This more extensive resource is free for OMA members. The OMA Obesity Algorithm eBook is available for a fee for OMA non-members and intended to be a yearly updated "living textbook" of Obesity Medicine.

Managing disclosures, dualities of interest, and funding

- The writing, editing, and publishing of the OMA Obesity Algorithm slides and eBook have never received outside funding for their development.
- Authors of the OMA Obesity Algorithm slides and eBook have never received payment for their work in developing these documents.
- Potential dualities of interest of the authors are disclosed in the relative Disclosure sections of each respective Obesity Algorithm document.
- For sections of the OMA Obesity Algorithm slides and eBook wherein substantive conflicts of interest may exist, members of the writing team having no potential conflicts of interest review these sections and have final approval of these sections.

Writing Process

Group Composition

- Authors of the OMA Obesity Algorithm slides and eBook represent a diverse range of clinicians, allied health professionals, clinical researchers, and academicians, intended to reflect a multidisciplinary and balanced group of experts in obesity science, evaluation, and treatment.

Evidence Foundation

- The content of the OMA Obesity Algorithm slides and eBook is supported by citations. These citations are derived from literature searches, as well as updates from data reported within the year prior to each annual update.
- Fully referenced citations are listed within the OMA Obesity Algorithm slides and eBook.

Review

- The OMA Obesity Algorithm slides and eBook undergo repeated reviews and approvals by authors having diverse perspectives of obesity medicine.
- Comments and suggested edits received from OMA members and outside sources are considered with each year's updated revisions.
- Comments and suggested edits originating from those with significant conflicts of interest are vetted by authors without similar conflicts of interest, prior to including the potential edits in revisions.
- The OMA Obesity Algorithm slides and eBook also undergo independent review and approval by the OMA Board of Trustees. The authors then consider all external reviewer comments and respond with a written rationale for modifying or not modifying the documents.

Writing Process

Recommendations

- These documents are intended to provide an overview of principles important to the care of patients with unhealthful increases in body fat.
- These documents are not intended to be interpreted as "rules" and/or directives regarding the medical care of an individual patient.
- The final decision regarding the optimal care of the patient with overweight and obesity is dependent upon the individual clinical presentation and the judgment of the clinician who is tasked with directing a treatment plan that is in the best interest of the patient.

Updating

- Both the OMA Obesity Algorithm and eBook are planned for yearly reviews and updates. During each year, the literature is routinely monitored to identify potentially relevant information applicable to forthcoming updates.
- If during the interim of scheduled updates, areas in need of urgent clarification are discovered, or if new evidence suggests the need for urgent modifications, then interim changes to the OMA Obesity Algorithm slides and eBook are made prior to each year's scheduled update.

Limitations

Prior OMA Obesity Algorithm versions

- The Obesity Medicine Association (OMA) Obesity Algorithm® (first released in 2013) undergoes yearly review with updates to clarify and amend text to reflect the latest research and perspectives in the specialty field of Obesity Medicine.
- Due to ever evolving new science, re-evaluation of older science, and yearly editing to improve clarity, older OMA Obesity Algorithm versions may not reflect up-to-date information.
- No single resource should solely determine patient care. The OMA Obesity Algorithm should be considered an adjunct to the totality of medical resources, as well as an adjunct to the clinical judgment regarding the management of patients with overweight and obesity.
- Upon each new release, it is recommended readers replace outdated OMA Obesity Algorithm versions with the most current version.
- If you find areas that may benefit from clarification or correction, then please contact and notify the Obesity Medicine Association.

Major Updates Included in the 2023 Version

- Simplified genetic syndromes
- Added Asian BMI threshold
- Added Ethnicity specific waist circumference thresholds
- New section on Obesity and Women
- Revised laboratory evaluation
- Simplified body composition testing
- Simplified Energy Expenditure section
- Added non-nutritive sweeteners
- Removed lorcaserin
- Added semaglutide
- Simplified pharmacology
- Simplified Investigational Anti-Obesity Pharmacology
- Significant revision of Bariatric Surgery section
- Reorganized overall order
- General text edits and updates
- Updated references

OMA Obesity Algorithm eBook, Slides, Authors and Citations

Adult Obesity Algorithm eBook: Detailed overview of Obesity Medicine

Citation: Tondt J, Freshwater M, Christensen S, Iliakova M, Weaver E, Benson-Davies S, Younglove C, Afreen S, Karjoo S, Khan N, Thiara D, Whittle C. Obesity Algorithm eBook, presented by the Obesity Medicine Association. www.obesityalgorithm.org. 2023. https://obesitymedicine.org/obesity-algorithm/ (Accessed = Insert date)

Adult Obesity Algorithm free downloadable slides: General overview of Obesity Medicine (content omitted in the downloadable slides can be found in the eBook)

Citation: Tondt J, Freshwater M, Christensen S, Iliakova M, Weaver E, Benson-Davies S, Younglove C, Afreen S, Karjoo S, Khan N, Thiara D, Whittle C. Obesity Algorithm Slides, presented by the Obesity Medicine Association. www.obesityalgorithm.org. 2023. https://obesitymedicine.org/obesity-algorithm-powerpoint/ (Accessed = Insert date)

The Obesity Algorithm is listed by the American Board of Obesity Medicine as a suggested resource and study-aid for the obesity medicine certification exam. (https://www.abom.org/exam-resources-2/)

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Executive Summary

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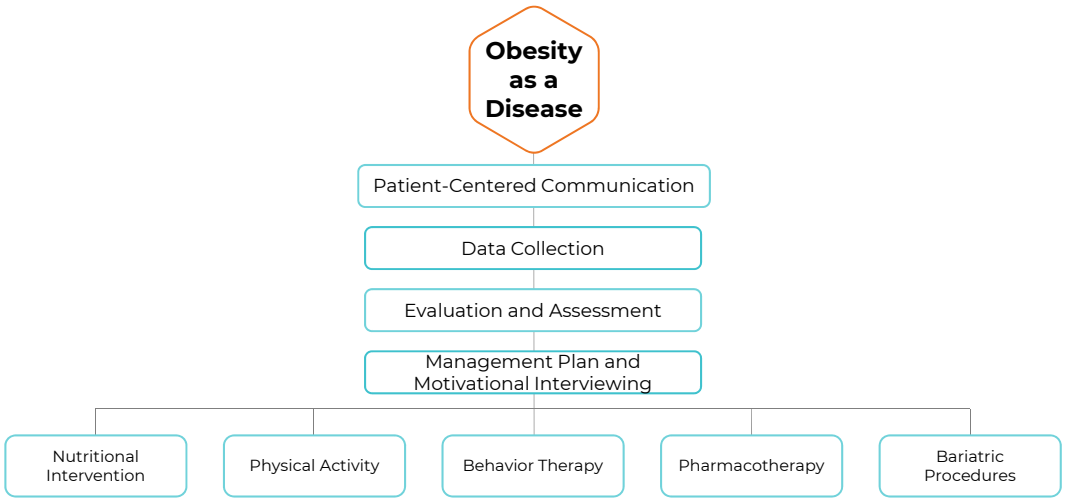
The Obesity Medicine Association's Definition of Obesity

"Obesity is defined as a chronic, progressive, relapsing, and treatable multi-factorial, neurobehavioral disease, wherein an increase in body fat promotes adipose tissue dysfunction and abnormal fat mass physical forces, resulting in adverse metabolic, biomechanical, and psychosocial health consequences."

The OMA Obesity Algorithm



Assess for the Presence of Obesity, Adiposopathy, Fat Mass Disease

Obesity may be assessed using several criteria (Thresholds vary based on gender and ethnic differences):

Body Mass Index (BMI)	18.5-24.9 kg/m^2	25.0-29.9 kg/m^2	≥30 kg/m^2
Percent Body Fat	Male: <25% Female: <32%		Male: >25% Female: >32%
Waist Circumference	Male: <40 in. Female: <35 in.		Male: >40 in. Female: >35 in.
Edmonton Obesity Staging System	Stage 0, 1, 2, 3, 4		

No Obesity	**Overweight**	**Obesity** Class I: BMI 30.0-34.9 Class II: BMI 35-39.9 Class III: BMI ≥ 40.0
Prevention	Primary care provider or dietitian	
	If treatment is ineffective, refer to an obesity medicine specialist.	Consider referring to an obesity medicine specialist.

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Assess for the Presence of Obesity, Adiposopathy, Fat Mass Disease

Body Mass Index	BMI = (Weight in kg)/(height in m)^2 OR 703 x (Weight in pounds)/(height in inches)^2
Percent Body Fat	Can be assessed by DXA scan, bioelectrical impedance, whole body air-displacement plethysmography, etc.
Waist Circumference	Can be measured by tape measure around the abdomen at the level of the anterior superior iliac crests, parallel to the floor. Tape should be snug against skin without compressing.
Edmonton Obesity Staging System	STAGE 0: No apparent risk factors, no physical symptoms, functional limitations, and/or impairment of well-being STAGE 1: Presence of obesity-related subclinical risk factors, mild physical symptoms, mild psychopathology, mild functional limitations, and/or mild impairment of well-being STAGE 2: Presence of established obesity-related chronic disease, moderate psychopathology, moderate functional limitations, and/or impairment of well-being STAGE 3: Established end-organ damage, significant psychopathology, significant functional limitations, and/or impairment of well-being STAGE 4: Severe (potentially end-stage) disabilities from obesity-related chronic diseases, severe disabling psychopathology, severe functional limitations, and/or severe impairment of well-being

Obesity medicine specialists, certified by the American Board of Obesity Medicine, dedicate a portion or all of their practice to the treatment of obesity. They perform a medical evaluation (history, physical, laboratory, body composition) and provide medical supervision for lifestyle change (nutrition, activity, behavior change), medications, or other nutritional interventions. Obesity is a chronic medical disease and often requires lifelong treatment.

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Evaluation and Treatment Summary

Comprehensive Evaluation of the Patient with Overweight/Obesity	
History	Weight history, past medical history, family history, social history, screening for weight-promoting medications, food intake, activity, review of systems
Physical Examination	Height, weight, blood pressure, body composition analysis, waist measurement, complete physical examination
Laboratory Tests*	Complete blood count, electrolytes, liver function, kidney function, fasting lipid profile, thyroid tests, hemoglobin A1c, uric acid, vitamin D
Diagnostic Testing*	EKG, echocardiogram, exercise stress test, sleep study, barium swallow or esophagoduodenoscopy

**Lab and diagnostic testing should be individualized*

Individualized Treatment Plans*	
Nutrition	Use calorie restriction, carbohydrate restriction, food journaling, very low-calorie diet programs
Activity	Give exercise prescription, use pedometers, limit TV and computer time, decrease sedentary time, initial goal of 150 minutes per week of moderate-intensity physical activity
Counseling	Eliminate provider bias and stigma, identify self-sabotage, develop strong support, address stress management, sleep optimization, other psychological support as needed
Pharmacotherapy	Use pharmacotherapy as part of a comprehensive program
Referral	Consider referral to an obesity medicine specialist

**If ineffective, consider referral to a metabolic and bariatric surgeon. Optimal pre- and post-operative care includes an obesity medicine specialist.*

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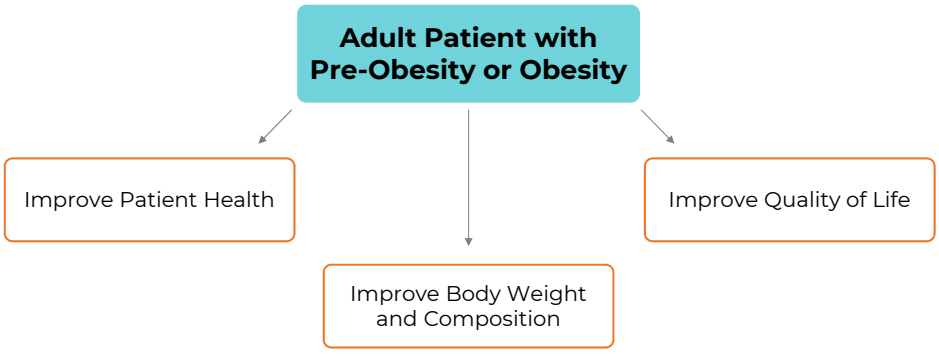
Overall Management Goals



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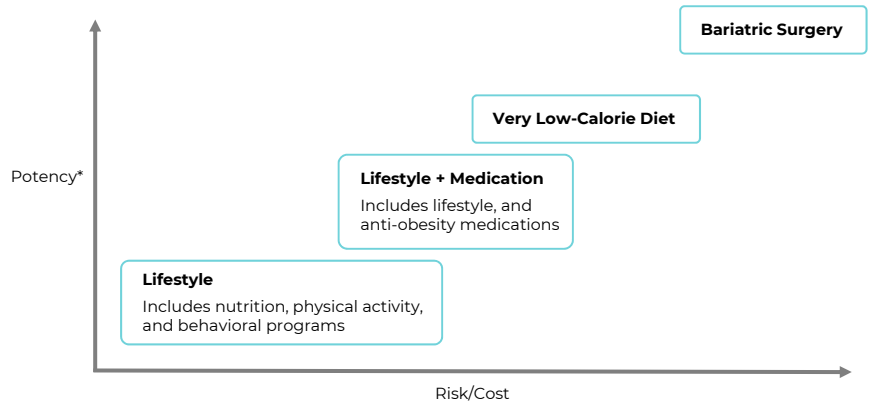
Current Treatment Options for Obesity



**Potency includes many factors, such as the amount, rate, and sustainability of weight loss, and the long-term resolution of adiposopathy and fat mass disease. Potency varies greatly for each individual (i.e., long-term adherence to a lifestyle program can be as potent as gastric bypass surgery).*

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The Disease of Obesity

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Obesity is a Multifactorial Disease that Requires a Multifaceted, Patient-centered, Individual Approach ("one size does not fit all")

Data from randomized clinical trials (RCT) in populations may not always apply to the care of an individual patient.

Multiple contributors to obesity may account for RCT variances (sometimes wide variances) in individual responses based upon:

- Age
- Race
- Gender
- Genetics
- Individual physiology
- Economic status
- Nutrition
- Physical activity
- Concomitant medications
- Concurrent illnesses
- Home environment (e.g., food access, stress, family, culture, school, travel)
- Work environment (e.g., food access, stress, nature of work, wellness programs, travel)
- Nutrition and physical activity environment
- Behavior modification
- Motivational interviewing
- "Apps," text messages & social media
- Wearable technologies
- Anti-obesity medications
- Bariatric surgery
- Natural individual variances which may not be reflected by the reported mean value of a clinical trial

Top 10 Benefits of Treating Obesity as a Disease

1. Healthful nutrition and regular physical activity often improves anatomic, physiologic, inflammatory, and metabolic body processes
2. Medically managed weight reduction in patients with obesity often improves glucose and lipid metabolism, reduces blood pressure, and reduces the risk of thrombosis
3. Medically supervised weight management programs for patients with obesity have the potential for statistically significant and clinically meaningful weight loss maintenance
4. Weight loss in patients with obesity may reduce premature mortality
5. Weight loss in patients with obesity may have favorable cardiac hemodynamic effects
6. Weight loss in patients with obesity may improve obstructive sleep apnea and osteoarthritis
7. Weight loss in patients with obesity may reduce the onset of certain cancers, improve response to cancer treatments, and reduce the onset/recurrence of new cancers
8. Weight loss in women with obesity may improve polycystic ovary syndrome, as well as improve obesity-related gynecologic and obstetric disorders; weight loss in men may increase testosterone levels when hypogonadism
9. Weight loss in patients with obesity may improve quality of life, improve body image, and improve symptoms of some psychiatric disorders (e.g., depression)
10. Weight loss in child-bearing women (and men) with overweight or obesity may help mitigate epigenetically transmitted increased risk of obesity and metabolic disease in future generations

Top 10 Takeaway Messages: Obesity is a Disease

1. The signs, symptoms, and pathophysiology of obesity fulfill the definition of a disease
2. Obesity can be due to inherited (genetic, epigenetic) factors and/or environmental factors
3. Obesity may result in cellular and organ anatomic abnormalities
4. Obesity may result in cellular and organ functional abnormalities
5. Obesity may result in pathogenic adipocyte and/or adipose tissue endocrine and immune dysfunctions that contribute to metabolic disease (adiposopathy or "sick fat" disease)
6. Obesity may result in pathogenic physical forces from excessive body fat, promoting stress damage to other body tissues ("fat mass disease")
7. Many diseases are promoted by unhealthful behavior, and obesity is no less of a disease when it is promoted by unhealthful behavior
8. Data from 2017 – 2018 estimate that approximately 42% of U.S. adults have obesity; 19.3% of youths have obesity
9. As with other diseases, obesity is best discussed using "people-first" language
10. Obesity is promoted by genetic predisposition, and shares similar pathophysiologies as aging

Obesity is a Disease When…

1. The patient has excessive body fat, as assessed by reliable measures
2. Excessive body fat is caused by genetic or developmental errors, infections, hypothalamic injury, adverse reactions to medications, nutritional/energy imbalance, and/or unfavorable environmental factors
3. Excessive body fat results in pathogenic structural or functional abnormalities resulting in increased patient morbidity and mortality
4. Multiple pathogenic adipocyte and/or adipose tissue endocrine and immune dysfunctions contribute to metabolic disease (adiposopathy or "sick fat" disease)
5. Multiple pathogenic physical forces from excessive body fat cause stress damage to other body tissues (fat mass disease)

The adverse health consequences of increased body fat are not simply **"co-morbidities"** or **"associated risk factors".**

Obesity Prevalence

The US adult obesity prevalence was **41.9% in 2017 – March 2020.**

From 1999 –2000 through 2017 –March 2020, **US obesity prevalence increased from 30.5% to 41.9%**. During the same time, the **prevalence of severe obesity increased from 4.7% to 9.2%.**

Non-Hispanic Black adults (49.9%) had the highest age-adjusted prevalence of obesity, followed by Hispanic adults (45.6%), non-Hispanic White adults (41.4%) and non-Hispanic Asian adults (16.1%, not accounting for lower BMI threshold).

The obesity prevalence was **19.3% among youths** (2-19 years), **39.8% among adults** aged 20 to 39 years, **44.3% among adults aged 40 to 59 years, and 41.5% among adults aged 60 and older.**

The prevalence of metabolic syndrome in US adults is >30%. **Only <20% of US adults have "optimal" metabolic markers.**

Obesity Terminology

"People-first" language recognizes the potential hazards of referring to or labeling individuals by their disease. Thus, **"patient who has pre-obesity or obesity"** or **"patient with overweight or obesity"** are preferred over "obese patient." This is similar to the standard with other diseases, such as cancer, wherein "patient with cancer" is preferred over "cancerous patient."

Encouraged Terms

- Weight
- Excess weight
- Unhealthy weight
- Overweight
- Body mass index
- Affected by obesity

Discouraged Terms

- Morbidly obese
- Obese
- Fat
- Heaviness
- Large size

Obesity Health Care Office Environment

Patients with obesity may have negative feelings or prior experiences regarding their weight. Clinicians and staff may need training to avoid weight bias and provide a supportive environment.

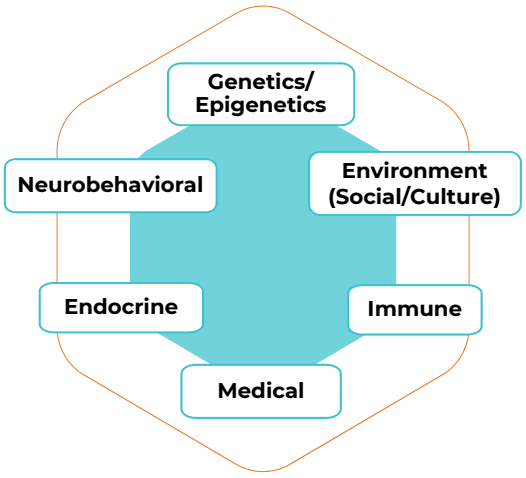
Positive Office Space

- Sturdy, armless chairs, wide chairs with arms, and/or firm sofas in waiting rooms and exam rooms
- Sturdy, wide exam tables that avoid or prevent tipping
- Sturdy stool or step with handles to help patients climb onto the exam table
- Tables/chairs/toilet seats best sustain higher body weights
- Extra-large patient gowns
- Split toilet seat; provide a specimen collector with a handle
- Providing reading materials in the waiting room that focus on healthy habits rather than dieting or body image

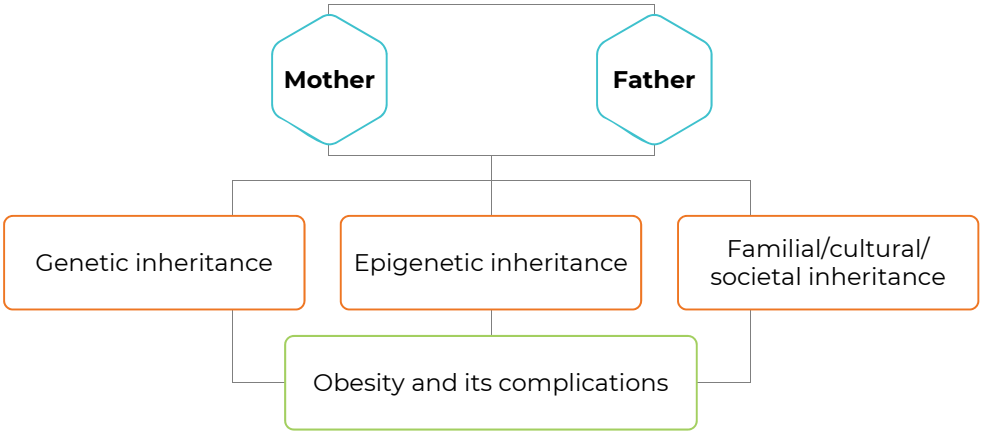
Appropriate Medical Devices

- Large adult blood pressure cuffs or thigh cuffs on patients with an upper-arm circumference greater than 34 cm
- Extra-long needles to draw blood
- Large vaginal specula
- Weight scales with the capacity to measure patients who weigh more than 400 pounds
- Weight scales are optimally located in a private area wherein the value is only seen by the patient and provider

Obesity is a Multifactorial Disease



Multifactorial Inheritance Factors Contribute to Obesity



Obesity: Extragenetic Etiology/Causes

Extragenetic

- Environment (family, home, geographic location)
- Culture
- Lack of optimal nutrition and physical activity
- Disrupted sleep (e.g., poor quality, too little, or too much)
- Adverse consequences of medications
- Mental stress
- Neurologic dysfunction (central nervous system trauma, hypothalamic inflammation, leptin resistance)
- Viral infections
- Gut microbiota neurologic signaling and transmission of pro-inflammatory state
- Toxins/obesogens (e.g., pollutants, endocrine disrupters)

Classification of Obesity

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1 2 3 4 5 6 7

Top 10 Takeaway Messages: Obesity Classification and Consequences

1. For the general population, body mass index (BMI) ≥ 25 kg/m^2 is considered pre-obesity/overweight; BMI ≥ 30 kg/m^2 is considered obesity
2. BMI has limitations in assessing adiposity in individuals with increased muscle mass, decreased muscle mass, men vs women
3. For individuals, accurately determining percent body fat, android fat, and visceral fat is a better assessment of adiposity compared to BMI alone
4. Central obesity is defined as waist circumference ≥ 40 inches (102 cm) for men and ≥ 35 inches (88 cm) for women [≥ 90 cm for Asian men; ≥ 80 cm for Asian women]
5. Waist circumference is well-correlated with the risk of metabolic and cardiovascular disease
6. Fat mass disease results in pathologic mechanical and physical forces leading to adverse clinical outcomes (e.g., sleep apnea, orthopedic problems)
7. Sick fat disease (adiposopathy) results in pathologic endocrine and immune responses that promote the most common metabolic diseases encountered in clinical medical practice (e.g., diabetes mellitus, high blood pressure, dyslipidemia)
8. Anatomic adiposopathic changes with obesity include adipocyte hypertrophy, adipose tissue expansion, increased energy storage in multiple fat depots and increased fat deposition in body organs
9. Functional adiposopathic changes with obesity include adipose hypoxia, increased reactive oxygen species, extracellular matrix abnormalities, intra-organelle dysfunction, neurological changes, and immunopathic/endocrinopathic responses
10. The degree by which adiposopathy results in metabolic disease largely depends on the interactions and crosstalk with other body organs

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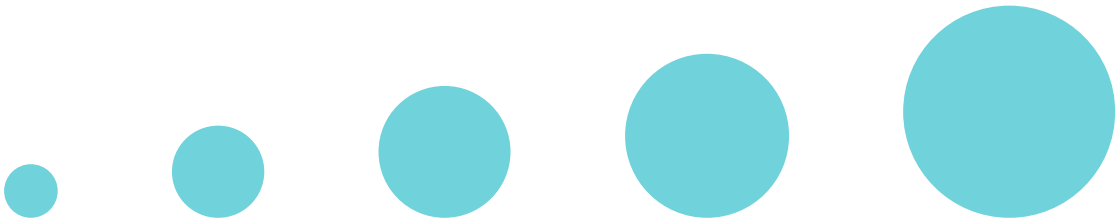
Body Mass Index: Increased Body Fat (Adiposity)

Body Mass Index (BMI) in kilograms per meters squared (kg/m²)*

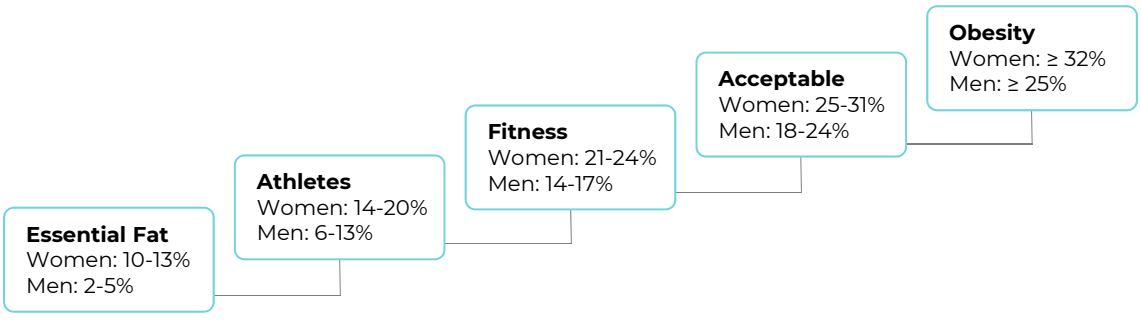


Normal Weight	Pre-obesity / overweight	Class I Obesity	Class II Obesity	Class III Obesity
18.5-24.9	25.0-29.9	30.0-34.9	35.0-39.9	≥ 40

*Different BMI cut-off points may be more appropriate based upon gender, race, ethnicity, and menopausal status. For example, among Asians, a BMI >23 kg/m2 may indicate pre-obesity, a BMI >27.5 kg/m2 may indicate class I obesity, a BMI >32.5 kg/m2 may indicate class II obesity, and a BMI >37.5 kg/m2 may indicate class III obesity

Percent Body Fat: American Council on Exercise Classification

American Council on Exercise Classification: Percent body fat*



**Based on "expert opinion;" cut-off points not scientifically validated*

Obesity Medicine Association (OMA) Classification of Percent Body Fat

Obesity Medicine Association Classification of Percent Body Fat in Adults as Assessed by DXA

	Women	Men
Essential fat	< 15%	< 10%
Athlete	15 - 19%	10 – 14%
Fitness	20 - 24%	15 – 19%
Acceptable	25 - 29%	20 – 24%
Pre-obesity	30 - 34%	25 – 29%
Obesity	≥ 35%	≥ 30%

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Obesity Medicine Association (OMA) Classification of Visceral and Android Fat

Obesity Medicine Association Classification of Visceral and Android Fat in Adults via DXA*

	Women	Men
Optimal visceral fat	< 1 lb. (500 grams/0.5 kg)	< 1 lb. (500 grams/0.5 kg)
Optimal android fat	< 3 lbs. (1400 grams/1.4 kg)	< 3 lbs. (1400 grams/1.4 kg)
Average total fat for adults	70 lbs. (30 kg)	80 lbs. (35 kg)
Average visceral fat for adults	2 lbs. (1000 grams/1 kg)	3 lbs. (1400 grams/1.4 kg)
Average android fat for adults	7 lbs. (3000 grams/3 kg)	7 lbs. (3000 grams/3 kg)

- lbs. = pounds; kg = kilograms; the pounds, grams, and kilograms listed represent approximate conversion values for the sake of simplicity
- DXA = Dual X-ray Absorptiometry
- Classifications are based upon (a) expert opinion, (b) research data regarding DXA, and (c) practical/clinical experience performing DXA
- Total body fat is widely variable, and correlates to body weight, height, and gender
- Visceral and/or android fat directly correlate to adiposopathic metabolic diseases (e.g., diabetes mellitus, hypertension, dyslipidemia, cardiovascular disease, and cancer), perhaps more so than waist circumference alone
- Some individuals (especially women) may have higher percent body fat with limited (and sometimes negligible) visceral fat
- US adults typically have visceral and android fat at least > 2-3 x "optimal"
- Visceral adipose tissue often increases linearly 5-fold from age 18 to 82

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Percent Body Fat (%BF)*: U.S. Army Regulations (Active Duty)

Men %BF Calculator

(Age, height, & tape measure of neck and waist)

Maximum allowable %BF

Age 17-20	20%
Age 21-27	22%
Age 28-39	24%
Age 40+	26%

Women %BF Calculator

(Age, height, & tape measure of neck, waist, and hip)

Maximum allowable %BF

Age 17-20	30%
Age 21-27	32%
Age 28-39	34%
Age 40+	36%

**Changes to "tape test" may be forthcoming*

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Waist Circumference: Increased Body Fat (Adiposity)

Obesity classification: Waist circumference (WC)*

Abdominal Obesity - Men	Abdominal Obesity - Women
≥ 40 inches	≥ 35 inches
≥ 102 centimeters	≥ 88 centimeters

**Different WC abdominal obesity cut-off points are appropriate for different races*

Different waist circumference methodologies include measurements made at the level of the umbilicus, at the midpoint between highest point of iliac crest and lowest rib, and at the level of the iliac crest. According to the National Institutes of Health, waist circumference is best measured at the highest point of the iliac crest. From a longitudinal standpoint, waist circumference response to obesity treatment is best assessed by consistency in how waist circumference is measured. Various waist circumference ratios (i.e., ratio to height and hip) may provide additional predictive risk information. However, the degree these additional ratio metrics influence patient care to a clinically meaningful degree is uncertain.

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Waist Circumference: Thresholds for Abdominal Obesity - Ethnic Differences

Population	Men	Women
Europid	≥94 cm	≥80 cm
Caucasian	≥94 cm (increased risk) ≥102 cm (still higher risk)	≥80 cm (increased risk) ≥88 cm (still higher risk)
United States, Canada & European	≥102 cm	≥88 cm
Asian (including Japanese)	≥90 cm	≥80 cm
Japanese	≥85 cm	≥90 cm
China	≥85 cm	≥80 cm
Middle East, Mediterranean & Sub-Saharan African	≥94 cm	≥80 cm
Ethnic Central and South American	≥90 cm	≥80 cm

**A waist to height ratio of 0.5 may be a simplified threshold common to all ethnicities.*
It may also be a better screening tool for cardiometabolic risk than BMI

Metabolic Syndrome: The NCEP ATP III Definition*

Patient must meet **three or more** of the following five risk factors:

Diagnostic Criteria	Defining Level
Abdominal obesity • Men • Women	Waist circumference >102 cm (>40 in) >88 cm (>35 in)
Triglycerides *	≥150 mg/dL (1.7 mmol/L)
HDL cholesterol * • Men • Women	 <40 mg/dL (1.04 mmol/L) <50 mg/dL (1.30 mmol/L)
Blood pressure *	≥130/ ≥85 mmHg
Fasting glucose *	≥100 mg/dL (5.6 mmol/L)

Metabolic syndrome (MetSyn):
- MetSyn is not a disease; it is a descriptive clustering of atherosclerotic cardiovascular risk factors
- Abdominal obesity is the only diagnostic physical finding
- Diagnostic criteria does NOT include low density lipoprotein cholesterol
- Diagnostic criteria may vary, depending on the organization crafting the diagnostic criteria
- Waist circumference diagnostic criteria may vary, depending upon race and gender
- Central obesity is a clinical marker of adiposopathy; increased visceral adiposity is a surrogate for global (integrative) fat dysfunction

**Or receiving drug treatment for these diagnostic criteria*

Obesity: Summary Diagnostic Metrics and Diagnostic Codes

Body Mass Index
≥ 30 kg/m^2

Percent Body Fat
Women: ≥ 32%
Men: ≥ 25%

Abdominal Obesity: Women
≥ 35 inches
≥ 88 centimeters

Abdominal Obesity: Men
≥ 40 inches
≥ 102 centimeters

Overweight and Obesity E66

- Begin with coding the complications (rather than a body mass index code), such as obesity complicating pregnancy, childbirth and the puerperium, if applicable (099.21-)
- Then consider additional / secondary code to identify body mass index (BMI), if known (Z68.-)

Excludes

- Adiposogenital dystrophy (E23.6)
- Lipomatosis NOS (E88.2)
- Lipomatosis dolorosa [Dercum] (E88.2)
- Prader-Willi syndrome (Q87.1)

E66 Overweight and Obesity*

- E66.0 Obesity due to excess calories **
- E66.01 Morbid (severe) obesity due to excess calories
- E66.09 Other obesity due to excess calories
- E66.1 Drug-induced obesity
- E66.2 Morbid (severe) obesity with alveolar hypoventilation
- E66.3 Overweight
- E66.8 Other obesity
- E66.9 Obesity, unspecified

- Code choice is best focused on the diagnosis and complications, rather than BMI alone
- Consider impact on patients who may read the diagnosis in their medical records (i.e., codes that include the terms "morbid obesity" and "excess calories") which may impact stigma and biases.

*** E66.0 is a non-billable code. Better to code one of the listed subcategory codes ("child code"). Unless drug-induced obesity, preferred codes might include E66.8 if the cause is known, or E66.9 if the cause is unclear*

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Body Mass Index
(BMI = kg/m^2 or weight in kilograms/height in meters squared)

Advantages

- Increased BMI generally correlates with metabolic and fat mass diseases in population studies
- Commonly used
- Reasonably reproducible
- Low cost
- Adequate measure for epidemiological studies
- Adequate screening metric for most patients

Disadvantages

- While BMI can estimate percent body fat in populations, BMI may not always correlate well with body composition, metabolic disease, and fat mass diseases in an individual patient
- Does not account for muscle mass
- May over-diagnose obesity in muscular individuals, under-diagnose patients with sarcopenia
- BMI cut-off points do not always distinguish between men and women, nor ethnic and racial considerations
- May not be an appropriate indicator of body fat in postmenopausal women
- Should not be the sole measure of adiposity for all patients

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Percent Body Fat

Advantages

- More specific assessment of body fat
- May be a reasonable longitudinal measure, especially in patients who may not be losing weight, but engaged in resistance exercise training, and thus may be losing body fat, and increasing muscle

Disadvantages

- Some measurement techniques are not always accurate, nor easily reproducible. For example, even with proper placement and multiple measures, skinfold calipers can vary from more accurate measures of percent body fat by 10% or more.
- Electronic body fat measurements may be more expensive than calipers
- The accuracy and reproducibility of electronic body fat measurements are dependent upon the equipment and software, technique, the expertise of the technician, and with some measures, the condition of the patient at time of measurement (e.g., state of hydration)
- Cut-off points of percent body fat not validated to correlate to metabolic disease
- While percent body fat may provide diagnostic information more useful than body mass index for many individuals, it is the amount of android and visceral fat (abdominal obesity) that best correlates with metabolic disease and cardiovascular disease risk

General Percent Body Fat Correlation with Body Mass Index (BMI): DXA Measurements U.S. Adults from NHANES 1999 – 2004

BMI*	Total Body Fat	< 25 kg/m²	25 – 29 kg/m²	30 - 34 kg/m²	> 35 kg/m²
Men % Body Fat (mean)	28%	23%	28%	32%	37%
Women % Body Fat (mean)	40%	34%	41%	44%	48%

**Not adjusted for age, race, or ethnicity, which can contribute to variability in percent body fat. Correlations of BMI to percent body fat in an individual patient depends on the amount of muscle mass.*

Reference values for centile percent body fat are often based on databases over a decade old.
Age < 40 years generally with lower % body fat than > 40 years. An analysis of DXA performed in U.S. Caucasian adults from 2003 – 2015 reported that depending on age:

- The upper 50th centile of % body fat > 30% - 43% for women and > 20% - 32% for men
- The upper 10th centile of % body fat > 43% - 52% for women and > 32% - 41% for men

Waist Circumference

Advantages

- Well-correlated to metabolic and cardiovascular disease
- Individualized anatomical measure of adipose tissue deposition, with an increase in waist circumference reflective of adipose tissue dysfunction
- Correlates well with total abdominal fat
- Low cost
- Waist to height ratio is more predictive of metabolic disease than BMI

Disadvantages

- Measurement not always reproducible
- Waist circumference is not superior to BMI in correlating to metabolic disease in patients with BMI $\geq$ 35 kg/m^2
- Racial/ethnic differences in the cut-off points that correlated to an increased risk of metabolic disease
- May not correlate well with intraperitoneal (visceral) fat, which can vary depending on gender and ethnicity
- May not correlate well with intraperitoneal (visceral fat) in patients with prior abdominal liposuction

Which is the "Best" Measure of Obesity?

Population Assessment

- An increase in body mass index (BMI), waist circumference (WC), and percent body fat (%BF) all correlate with an increased prevalence of metabolic syndrome

Individual Assessment

- BMI is a reasonable initial screening measurement for most patients
- WC provides additional information regarding adipose tissue function/dysfunction and predisposition to metabolic disease among individuals with BMI<35 kg/m^2
- %BF may be especially useful in patients with extremes in muscle mass (i.e., individuals with sarcopenia or substantial increases in muscle mass), and thus may be a more accurate measure of body composition when assessing the efficacy of interventions directed towards change in muscle mass
- %BF accuracy is not compromised by differences in gender, race, ethnicity, or individual variations in body fat distribution
- %BF may provide more detailed information regarding body composition, which if accompanied by other measurements (e.g., android fat, visceral fat, lean body mass), may assist clinical assessment, and potentially better help with motivation

Fat Mass Disease:
Abnormal and Pathologic Physical Forces

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Clinical Manifestations: Fat Mass Disease

Impacts multiple body systems

Cardiovascular

- Congestive heart failure and cor pulmonale
- Heart failure with preserved ejection fraction or HFpEF
- Varicose veins
- Thromboembolic events (i.e., pulmonary embolus, stroke)
- Hypertension (i.e., compression of kidney by increased visceral fat)

Pulmonary

- Dyspnea
- Obstructive sleep apnea (OSA)
- Hypoventilation/Pickwickian syndrome
- Asthma
- Increased risk for upper respiratory infection (URI)
- Increased severity of URI

Neurologic

- Reduced subcortical grey matter
- Intracranial hypertension (pseudotumor cerebri) due to increased intra-abdominal pressure/OSA with impaired central venous return.
- Stroke (see "cardiovascular") Nerve entrapment (i.e., meralgia paresthetica, carpal tunnel syndrome)

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Clinical Manifestations: Fat Mass Disease

Impacts multiple body systems

Musculoskeletal

- Immobility
- Osteoarthritis (e.g., knees, hips)
- Low back pain
- Myalgias
- Altered center of gravity
- Impaired balance

Gastrointestinal

- Gastroesophageal reflux
- Hernias

Integument

- Striae distensae (skin stretch marks)
- Stasis pigmentation
- Venous stasis ulcers
- Cellulitis
- Skin tags
- Intertrigo (i.e., bacterial, fungal skin fold infections)
- Carbuncles

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Clinical Manifestations: Fat Mass Disease Pulmonary Complications

Obesity Increases the Risk for, and Increases the Severity of Upper Respiratory Tract Infections (URI), Including Viral (Influenza and Coronavirus) Infections

Patients with obesity may have fat mass disease, such as baseline compromise of lung function, with limited margin for further impairment of lung function:

- Reduced tidal volume
- Reduced forced expiratory volume (FEV 1)
- Sleep apnea
- Day and nighttime hypoxia

Other fat mass disease factors complicating outcomes with infections include:

- Debilitation
- Immobility and orthopedic challenges
- Genetic predisposition

Patients with obesity may also have sick fat disease (adiposopathy) which can predispose to infection and worse outcomes:

- Disruption of innate and acquired immunity
- Pro-inflammatory responses
- Competing financial resources (e.g., polypharmacy, medical office visits) from treatment of common adiposopathic metabolic complications (e.g., diabetes mellitus, hypertension, dyslipidemia) may impair urgent and chronic medical care for URI's
- Fear of contracting URI (coronavirus) may limit urgent and chronic medical care for obesity, cardiovascular disease events, and metabolic diseases

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Clinical Manifestations: Fat Mass Disease Musculoskeletal Complications

Obesity-Related Adverse Orthopedic Complications

- Fat mass disease biomechanical stress
- Adiposopathic chronic inflammation contributes to osteoarthritis
- Obesity limits mobility & can contribute to orthopedic disabilities
- Obesity increases risk of orthopedic surgery complications:
  - Infection and sepsis
  - Thromboembolism
  - Trauma and musculoskeletal damage
  - Organ impairment/failure
  - Reduced long-term implant viability
  - Mortality

Advantages of Weight Loss in Patients with Obesity, Pre and Post Orthopedic Surgery

- May reduce operative difficulties and complications
- May reduce stress to joints and reduce pain and disability

Clinical Manifestations: Fat Mass Disease

Psychosocial complications

Weight Bias
- Society
- Family/Friends
- Workplace (bullying, harassment)
- Health care

Psychosocial Impact On Patient
- Depression/Hopelessness
- Low self-esteem
- Body-image dissatisfaction
- Impaired intimacy and sexual relationships
- Decreased work productivity and increased work absenteeism

Weight Bias Internalization
- Increased body fat can contribute to self-stigmatization
- Weight stigma may contribute to mental stress, leading to adiposopathic stress responses and metabolic disease

Clinical Manifestations: Fat Mass Disease

Weight Bias

Negative Self or External Perceptions
- "Unmotivated"
- "Weak-willed"
- "Less intelligent"
- "Less attractive"
- "Unsuccessful"
- "Overindulgent"
- "Lazy"

Heath Care Bias
- Provider negative attitudes and stereotypes about people with obesity can affect medical perceptions, judgment, interpersonal behavior, and decision making – all leading to compromised care
- Patient experiences of poor and/or demeaning interactions with health care providers may cause patient stress, avoidance of care, mistrust, and diminished adherence to treatment

Sleep Disruption and Obesity

Sleep disorders (e.g., too little or too much sleep) or Mistiming of: Sleep Patterns, Food Intake and/or Light Exposure

Altered Circadian System*

- **Altered Feeding May Promote Obesity**
- **Unhealthy Effects on Body Composition**
- **Hyperglycemia**
- **High Blood Pressure**
- **Dyslipidemia**
- **Increased Risk of Heart Disease and Stroke**

**A diurnal rhythm is an endogenous or exogenous response synchronized to day/night 24-hour day. A circadian rhythm (sleep/wake cycle) is endogenously generated response synchronized to a 24-hour day.*

Sleep Disorders and Obesity: Obstructive Sleep Apnea*

History

- Snoring (usually loudly)
- Insomnia
- Restless sleep
- Sudden wakening with choking or gasping
- Headaches
- Daytime sleepiness
- Fatigue
- Motor vehicle accidents (a potential complication of sleep disorders)
- Forgetfulness
- Mood changes
- Lack of interest in sexual behavior
- Gastroesophageal reflux

**Other sleep disorders associated with obesity include insomnia and restless leg syndrome*

Sleep Disorders and Obesity: Obstructive Sleep Apnea

Physical Findings

Increased neck circumference

- Men ≥ 17 inches; Women ≥ 16 inches
- Neck circumference cut-off points that define increased obstructive sleep apnea risk can vary, with some cut-off points being > 17 inches for men and > 15 inches for women

Head abnormalities

- Modified Mallampati score of 3 or 4
- Retrognathia
- Lateral peritonsillar narrowing
- Macroglossia
- Tonsillar hypertrophy
- Enlarged uvula
- High arched/narrow palate
- Nasal abnormalities
- Overbite

Cardiopulmonary abnormalities

- Peripheral edema
- Cardiac dysrhythmia
- High blood pressure

Sleep Disorders and Obesity: Obstructive Sleep Apnea (OSA)

Diagnosis

Questionnaires

- Berlin Sleep Questionnaire
- Epworth Sleepiness Scale
- STOP-BANG Questionnaire*
  - **STOP- S**noring, **T**iredness, **O**bserved apnea and high blood, **P**ressure
  - **BANG- B**MI, **A**ge, **N**eck circumference, **G**ender

Testing

- In-lab overnight sleep studies
  - Apnea hypopnea index (AHI)
    - 5-15/hour = mild sleep apnea
    - 15-30/hour = moderate sleep apnea
    - >30/hour = severe sleep apnea
- Home sleep test
- Multiple sleep latency test

**STOP-BANG Questionnaire may have the highest sensitivity in diagnosing mild moderate, and severe OSA*

Adverse Consequences of Untreated Obstructive Sleep Apnea Consequences of Untreated Obstructive Sleep Apnea

- Worsening obesity
- Congestive heart failure
- Atrial fibrillation
- Nocturnal dysrhythmias
- Stroke
- High blood pressure
- Type 2 diabetes mellitus
- Pulmonary hypertension

Sleep Disorders and Obesity: Obstructive Sleep Apnea

Treatment

- Reduction of fat mass
- Behavior therapy to improve sleep patterns
- Oral appliances
  - Mandibular reposition devices
  - Tongue retaining devices
- Nasal expiratory positive airway
- Continuous positive airway pressure
- Adaptive servo-ventilation
- Surgery
  - Laser-assisted uvulopalatoplasty
  - Radiofrequency ablation
  - Palatal implants
  - Electrical stimulation of upper airway muscles
  - Skeletal surgery procedures

Adiposopathy (Sick Fat Disease):
Abnormal Endocrine & Immune Responses

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Anatomic Changes

- **Positive caloric balance may lead to adipocyte hypertrophy with variable increases in adipocyte number, as regulated by intracellular:**
  - Sterol regulatory element binding protein-1 (SREBP1)
  - Peroxisome proliferator-activated receptor (PPAR) gamma
  - Ccaat-enhancer binding proteins (C/ebps)

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Anatomic Changes

- **When adipogenesis (proliferation and differentiation) is impaired in peripheral subcutaneous adipose tissue (SAT), inadequate storage of excess energy in SAT may result in energy overflow and increased circulating free fatty acids**
  - Worsening adipocyte hypertrophy and adipocyte dysfunction
  - Increasing ("ectopic") fat deposition in other depots
    - Visceral fat
    - Abdominal SAT
    - Pericardiac fat
    - Perivascular fat
  - Increasing ("ectopic") fat deposition in other body organs
    - Liver
    - Muscle
    - Pancreas
    - Heart
    - Kidney

Functional Changes

- **Increased adipocyte hypertrophy and adipose tissue accumulation may contribute to:**
  - Adipocyte and adipose tissue hypoxia
  - Increased adipose tissue immune cell infiltration
  - Increased adipocyte apoptosis
  - Increased reactive oxygen species and oxidative stress
  - Extracellular matrix abnormalities
  - Intraorganelle dysfunction (e.g., mitochondrial and endoplasmic reticulum stress)
  - Changes in adipose tissue neural network and innervations

Evaluation and Treatment Overview:

History, Physical Exam, Laboratory, Diagnostic Testing, Treatment Priorities

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Top 10 Takeaway Messages: Obesity Evaluation

1. Patients with obesity often do not receive standard preventive medical care
2. Useful nutrition monitoring approaches include recording food and beverage intake using a diary
3. Body systems to be evaluated before prescribing a physical activity program include cardiac, pulmonary, and neuro-musculoskeletal systems, as well as metabolic processes (diabetes mellitus, hypertension)
4. Routine laboratory assessment may include measures of glycemia (fasting glucose levels, HbA1c), lipid levels, liver enzymes, electrolytes, creatinine & blood urea nitrogen, thyroid stimulating hormone, complete blood count, urine for albumin, and possibly vitamin D
5. Individual testing may include evaluation for insulin resistance, insulinoma or nesidioblastosis, hypercortisolism, oligomenorrhea/amenorrhea, hyperandrogenemia & polycystic ovary syndrome in women, and hypogonadism in men.
6. Other diagnostic tests in patients with overweight or obesity might include magnetic-resonance imaging or computed tomography of the pituitary, resting electrocardiogram, cardiac stress testing, echocardiogram, coronary calcium scores, ankle-brachial index, sleep studies, and imaging studies of the liver.
7. Methods to measure body composition include dual-energy x-ray absorptiometry (DXA), bioelectrical impedance, whole body air displacement plethysmography, measuring tape, or skinfold calipers
8. Prader Willi is the most common non-inherited, non-polygenic genetic syndrome that may promote obesity
9. Melanocortin 4 receptor deficiency (autosomal dominant or recessive) is the most common inherited, non-polygenetic syndrome that may promote obesity
10. Medical conditions that may promote fat mass gain include hypothalamic damage, immobility, insulinoma, hypercortisolism, sleep disorders, untreated hypothyroidism, and adverse effects of concurrent medications

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History

Body Weight History
(Assessment of lifetime body weight)

- Pattern of body weight gain over lifetime (e.g., slow & gradual, rapid & sudden, or combination)
- Factors influencing weight change
  - Physical health
  - Mental health
  - Medications, surgery, other treatments
  - Life circumstances (family, marriage, newborn, work, moving, finances, abuse)
  - Past strategies/behaviors/interventions proven effective and ineffective
  - Effect of current and past body weight on mobility, interaction with family and friends, work, instances of bias & discrimination
  - Current or anticipated barriers to future weight loss

History

Medical History
- Age, gender, race, ethnicity
- Fat mass disease (i.e., osteoarthritis, sleep apnea)
- Adiposopathy (i.e., type 2 diabetes mellitus, high blood pressure, dyslipidemia)
- Other medical and surgical conditions
- Eating disorder screening
- Mental health & stress screening
- Sleep pattern disorder evaluation & screening

Medications
- Medication and food allergies
- Medications that may affect body weight

Review of Systems (ROS)
- Include history of other conditions potentially relevant to anti-obesity medications (e.g., glaucoma, pancreatitis, kidney stones, chance of getting pregnant, seizures)
- Conditions that may warrant treatment with drugs having favorable weight effects (e.g., migraine headaches, depression)

Family History
- Family members with obesity
- Applicable familial metabolic medical diseases
- Family history of cardiovascular disease and/or cancer

Social History & Support Systems
- Cigarette smoking
- Alcohol intake
- Recreational drug use (e.g., marijuana, cocaine)
- Person who selects and purchases food
- Availability and involvement of family and friends
- Educational access to healthful nutrition and physical activity (e.g., current knowledge base, availability of Internet, knowledge centers, etc.)

Socioeconomic and Cultural History
- Economic status
- Social status
- Cultural background
- Occupation
- Family structure and support for weight loss
- Parenting behavior
- Marital status
- Living situation
- Abuse (physical, mental, sexual)
- Geographic location (e.g., urban food desert)

Nutrition History

Meals and Snacks

- Timing
- Nutritional content and amount
- Planning and preparation of food
- Physical/financial access to foods
- Location of food consumption (i.e., eating area, television, workplace, fast food, etc.)
- Family/cultural influences
- Community influences

Behavior

- Previous nutritional attempts to lose weight and/or change body composition
  - If unsuccessful or unsustained, what were short- and long-term barriers to achieving or maintaining fat weight loss
- Triggers (hunger, cravings, anxiety, boredom, reward, etc.)
- Nighttime eating
- Binge eating
- Emotional eating
- Readiness for change

Records

- Food frequency questionnaire
- Food and beverage diary, including type of food or beverage consumed and amount consumed
  - 24 or 72-hour recall
  - Keep food and beverage record for a week and return for evaluation
- Electronic application tools

Physical Activity History

- Success and/or failure of previous physical activity/exercise efforts
- If no longer engaged in a routine physical activity/exercise regimen:
  - When? (Date of change)
  - What? (Cause of change)
  - Why? (Identify barriers to re-engagement)
- Current physical activity (FITTE)
  - Frequency
  - Intensity
  - Time or Duration
  - Type
  - Enjoyment (physical activity/exercise preferences)
- Current fitness level, endurance capacity, mobility limitations, and equipment needs
- Access to locations amenable to increased physical activity/exercise (e.g., gym, workplace, exercise facilities, bicycle paths and walkways, urban or rural home setting)
- Physical, social, financial, and perceived barriers to increased physical activity

Physical Activity History

Examples of Common Medical Conditions Best Evaluated Before Prescribing an Exercise Program:

- Diseases of the heart, lung, neuromusculoskeletal, and other body systems
- Metabolic diseases having potential risks with increased physical activity:
  - Atherosclerotic coronary heart disease (worsening ischemia)
  - Diabetes mellitus (hypoglycemia)
  - High blood pressure (acute increase during exercise but decrease post-exercise and chronically)

Routine Preventive Medical Care

Individuals with Obesity often do not Receive the same Preventive Standards of Care as those without Obesity. Examples of Preventive Medical Care (Depending upon Gender and Age) may Include:

- Breast cancer screening
- Gynecological exam with Pap smear (which may include assessment of human papilloma virus)
- Prostate cancer screening
- Colorectal cancer screening
- Immunizations
- Other standard preventive health screening

Physical Exam

Vital Signs

- Height with bare or stocking feet measured with a stadiometer
- Weight using calibrated scale and method consistent from visit to visit (i.e., light indoor clothing or gown)
- Body mass index
- Waist circumference
  - Standing using superior iliac crest
  - May not provide additional diagnostic information among patients with BMI > 35 kg/m^2
- Blood pressure using appropriately sized cuff
- Pulse
- Neck circumference

General Physical Exam

- Comprehensive physical exam
- Special emphasis on physical exam of the nose, throat, neck, lung, heart, abdomen, body shape, neuromusculoskeletal system, and integument

Laboratory: Routine

- Complete blood count (CBC)
- Comprehensive metabolic panel (CMP)
  - Fasting blood glucose
  - Liver enzymes
  - Renal function and electrolytes
- Fasting lipid levels
  - Triglycerides
  - Low-density lipoprotein (LDL) cholesterol
  - High-density lipoprotein (HDL) cholesterol
  - Total cholesterol
- Hemoglobin A1c (HbA1c)
- Thyroid stimulating hormone (TSH)
- 25-hydroxyvitamin levels

Laboratory: Individualized Blood Testing

- Urinalysis and/or urine for microalbumin/creatinine in those with at risk for kidney disease (e.g., diabetes, hypertension, nephrotic syndrome, etc.)
- Glucose tolerance testing
- Fasting insulin testing or other indices of insulin resistance
- Fasting proinsulin, C-peptide, and insulin if hyperinsulinemia is suspected as a secondary cause of obesity (e.g., insulinoma, nesidioblastosis, etc.)
- One milligram (mg) overnight dexamethasone suppression test, 24-hour urine collection for (free) cortisol, or repeated measures salivary cortisol collection at 11:00 PM if endogenous hypercortisolism is suspected as a secondary cause of obesity
- Prolactin, estradiol, follicle-stimulating hormone, luteinizing hormone, and pregnancy test in women with unexplained oligomenorrhea or amenorrhea. Additionally, testosterone and dehydroepiandrosterone sulfate (DHEAS) in women with hirsutism or polycystic ovary syndrome
- Testosterone (and if low to a clinically significant degree: possibly prolactin, follicle-stimulating hormone, and luteinizing hormone) for men with impotence or physical findings of hypogonadism
- Iron studies (iron, total iron binding capacity, ferritin), zinc, copper, thiamine, folate, B12, and fat-soluble vitamins in patients with a history of bariatric surgery
- Apolipoprotein B and/or lipoprotein particle number especially if triglyceride levels are elevated. High-sensitive C-reactive protein (hs-CRP) can also be used for cardiovascular risk stratification
- Uric acid in those with a history of gout

Diagnostic Testing: Individualized

- Magnetic-resonance imaging or computed tomography of the brain if a structural lesion of the pituitary/hypothalamus is suspected (i.e., craniopharyngioma, pituitary tumor)
- Resting electrocardiogram
- Cardiac stress testing
- Echocardiogram
- Coronary calcium scores
- Cardiac positron emission tomography imaging (computed tomography)
- Ankle-brachial index
- Sleep studies
- Imaging studies of the liver
- Indirect calorimetry (anaerobic threshold/VO2 testing)
- Resting metabolic rate (RMR)

Diagnostic Testing: Individualized

Body Composition

- Dual-energy X-ray absorptiometry (DXA), ideally with android fat assessment (abdominal subcutaneous and visceral fat assessment)
- Bioelectric impedance
- Near-infrared interactance
- Whole-body air displacement plethysmography (BOD POD)
- Tape measurements (to assess muscle mass as well as wrist and neck size for use in percent body fat equations)
- Caliper percent body fat measurements (e.g., three-site skinfold calculations)
- Underwater weighing
- Quantitative magnetic resonance (QMR)
- Computerized tomography (single slice or volume method)
- Deuterium dilution

Emerging Science Testing

- Leptin
- Adiponectin
- Leptin-to-adiponectin ratio
- Free fatty acids
- Immune markers
  - Tumor necrosis factor
  - Interleukin 1 and 6
- Infectious testing
  - Gut microbiota
  - Adenovirus assays
  - Evaluation for other microbes

Identify and Manage Secondary/Contributing Causes of SFD and FMD

Conditions that May Promote Fat Mass Gain:

Genetic Syndromes

- Isolated, not inherited (i.e., Prader Willi)
- Familial (melanocortin 4 receptor deficiency)

Medical Conditions

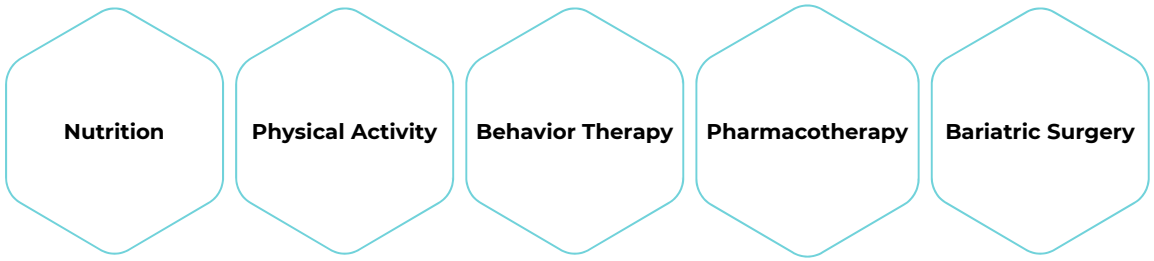
- Hypothalamic damage
- Immobility
- Insulinoma
- Untreated hypothyroidism
- Hypercortisolism (Cushing's disease)
- Sleep disorders
- Adverse effect of some medications

Psychological and Behavioral Conditions

- Mental stress
- Depression
- Anxiety
- Post-traumatic stress syndrome
- Binge-eating disorder
- Night-eating syndrome
- Eating disorders not otherwise specified

Treatment of Adult Patients with Overweight or Obesity

Medical Management and Coordination



Treatment of Adult Patients with Overweight or Obesity

- Treat adipocyte and adipose tissue dysfunction, which treats sick fat disease (SFD or adiposopathy)
- Treat excessive body fat, which treats fat mass disease (FMD)
- Treating diseases due to increased body fat and its adverse metabolic and biomechanical consequences may improve patient health, quality of life, body weight, and body composition

Nutrition Therapy for Obesity

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1 2 3 4 5 6 7

Top 10 Takeaway Messages: Obesity and Nutrition

1. Health outcomes are most improved with nutrition therapy when the dietary interventions are evidence-based, quantitative, qualitative, and facilitate patient adherence
2. Low calorie diet is ~ 1200 to 1800 kcal/day; very low-calorie diet is generally < 800 kcal/day
3. Fat restricted diet is often defined as 10 – 30% of total calories from fat
4. Low carbohydrate diet is generally defined as 50 – 150 grams of carbohydrates per day; very low-carbohydrate diet is < 50 grams of carbohydrates per day
5. The isocaloric substitution of ultra-processed refined carbohydrates with saturated fats does not improve cardiovascular disease risk; the isocaloric substitution of saturated fats with unhealthful ultra-processed carbohydrates does not improve cardiovascular disease risk
6. A ketogenic dietary pattern restricts carbohydrate intake and typically discourages ultra-processed and refined foods, foods high in glycemic index/load, and foods rich in trans fatty acids. Ketosis may reduce hunger.
7. A Mediterranean dietary pattern encourages olive oil, vegetables, fruits, legumes, whole grains, nuts, seeds, seafood, fermented dairy products, poultry, eggs, and red wine. It discourages high amounts of red meats, meat products, and ultra-processed carbohydrates
8. A DASH dietary pattern encourages vegetables, fruits, whole grains, fat-free or low-fat dairy products, fish, poultry, lean meats, nuts, seeds, legumes, fiber, foods containing calcium, potassium and magnesium. It discourages sodium > 2300 mg per day, total fat > 27% of total daily calories, cholesterol > 150 mg per day for 2100 Calorie eating plan, red and ultra-processed meats, sugar-sweetened beverages, and foods with added sugars
9. A vegetarian dietary pattern encourages vegetables, fruits, whole grains, legumes, seeds, nuts and discourages meats
10. Fasting (alternative day, intermittent, or time-restricted eating) may contribute to overall caloric restriction and weight reduction

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General Nutrition

The principles outlined pertain to general nutrition and **may not apply to the individual patient.**

Carbohydrates

- Carbohydrates contain 4 kcal/gram
- Carbohydrates can serve as a source of energy and as well cellular structural elements such as hyaluronic acid and proteoglycans; carbohydrates may contain sugars, starch and/or fiber
- The intestinal digestion of carbohydrates results in breakdown products of monosaccharides such as glucose, galactose, and fructose. Glucose and galactose are actively transported into intestinal cells by sodium glucose cotransporter-1 (SGLT-1). Fructose is absorbed across intestinal cells by facilitated diffusion via glucose transporter 5 (GLUT-5)
- The satiation from carbohydrates in foods (e.g., fruits) is substantially dependent upon the presence of fiber
- Carbohydrates are generally not considered an essential macronutrient, as liver, kidney, and possibly small intestine can synthesize glucose (i.e., gluconeogenesis). However, with genetic defects affecting glucose metabolism and/or storage (e.g., glycogen storage disease), carbohydrates may be considered conditionally essential
- Calorie deficiency can lead to marasmus (insufficient calories), but there is no known carbohydrate deficiency
- The Dietary Reference Intake (DRI) for carbohydrate is 130 grams/day

Fat

- Fat contains 9 kcal/gram
- Fats or lipids are a diverse group of compounds used as an energy source and for many metabolic processes:
  - Immune response (omega-3 fatty acids)
  - Cell membrane structure (phospholipids)
  - Brain tissue (cerebrosides)
  - Synthesis of bile acid, cholesterol, vitamin D, steroid hormones
  - Insulation
- Two fatty acids cannot be made by the body and these "essential" fatty acids must be consumed in the diet [i.e., omega-3 alpha linolenic acid (ALA) and omega-6 linoleic acid (LA)]
- Omega-3 fatty acids eicosapentaenoic acid (EPA)and docosahexaenoic acid (DHA) and gamma linolenic acid (an omega-6 fatty acid) are sometimes considered "conditionally essential," meaning they can be endogenously derived on the condition of no lack of intake of essential fatty acids. Given humans are only able to produce small amounts of EPA and DHA, oral intake of EPA and DHA is often recommended from cold water marine fish
- Dietary Reference Intake (DRI) for fat is at least 30 grams/day
- Replacing saturated fats with polyunsaturated or monounsaturated fats may reduce cardiovascular disease risk
- Replacing saturated fats with refined carbohydrates and sugar is not associated with reduced cardiovascular disease risk

Trans Fats

Trans Fats are Created Through a Process of Artificially Hydrogenating Polyunsaturated Fats (Vegetable Oils) into more Saturated Fats, Allowing for Higher Melting Temperatures (Which is more Desirable for Processed Foods, Cooking and Frying)

- **Partially hydrogenated vegetable oils** were developed because they favorably affected taste in applicable foods and were less expensive than saturated fats from animals (lard)
  - Some early shortenings (fats) made from partially hydrogenated vegetable oil (cottonseed and soybean oil) originally contained 50% trans fats, and were marketed as being a more healthful alternative to animal fat, because they were derived from "vegetables"
  - Although they contains partially hydrogenated palm and soybean oils, common shortenings now contain minimal trans fats, soybean oil, fully hydrogenated palm oil (i.e., 3 grams saturated fats, 6 grams polyunsaturated fats, 2.5 monounsaturated fats)
- Trans fats may increase low-density lipoprotein cholesterol, reduce high-density lipoprotein cholesterol, and increase the risk of cardiovascular disease (myocardial infarction and stroke), type 2 diabetes mellitus, and certain cancers
- While the FDA banned partially hydrogenated oil in 2018, trans fats are sometimes reportedly still found in ***some*** cakes, pies, cookies (especially with frosting), biscuits, microwavable breakfasts, stick margarine, crackers, microwave popcorn, cream-filled candies, doughnuts, fried fast foods, and frozen pizza
- Conjugated linoleic acid (CLA) is a naturally occurring trans / cis fat derived from ruminants (fermentation of plant-based foods via microbes in the stomach prior to digestion) which is not proven to be detrimental to health; conjugated trans linkages are not included as trans fats for nutritional regulations and food labeling

Saturated Fats

Podcast : Dairy
Lydia Alexander MD

Podcast : Fats
Krishna Doniparthi MD

- Carbon chain fatty acids with no double bonds
- Solid or semisolid at room temperature
- Many natural foods containing saturated fats also contain polyunsaturated and monounsaturated fat
- Coconut and palm oils have a high percent of saturated fats (both medium and large chain), and are commonly found in snack foods
- Long chain saturated fatty acids (>12 carbons) are found in meats, dairy products, tropical oils (coconut and palm oil), and hydrogenated vegetable oils (shortening)
- Medium chain saturated fatty acids (8-12 carbons) are found in coconut and palm oil
- Stearic acid (C18:0) represents about 25% of saturated fats in the U.S. adult diet, and has minimal effects upon low density lipoprotein (LDL) cholesterol
- Palmitic acid (C16:0) represents about 50% of saturated fats in the U.S. adult diet, and increases LDL cholesterol
- Saturated fat consumption may impair vascular endothelial function; polyunsaturated fats such as omega-3 fatty acids may improve endothelial function
- Saturated fats are less likely than unsaturated fats (e.g., with cooking) to become oxidized or become rancid
- Reports are inconsistent regarding the relationship of saturated fat-containing dairy products and cardiovascular disease; dairy food intake is included in "diets" generally considered to be healthful (e.g., Mediterranean Diet). Some reports suggest that low-fat fermented dairy products (e.g., cheeses and yogurt) may be more healthful than other high fat dairy products (e.g., butter)

Polyunsaturated Fats

- Carbon chain fatty acids with multiple double bonds
- Liquid at room temperature
- Examples: Vegetable and fish oils
- Nuts (e.g., walnuts, almonds, macadamia nuts, hazelnuts, pecans) contain both polyunsaturated and monounsaturated fats, and thought to reduce the risk of cardiovascular disease
- Omega-3 fatty acids found in oceanic cold-water fish are often considered cardioprotective
- Prescription dose omega-3 fatty acids (4 grams per day of predominantly eicosapentaenoic acid) can reduce triglyceride levels and reduce the risk of CVD
- Cooking oils beyond their individual smoking points or repeated use of the same cooking oils may increase oxidation, generate unhealthful free radicals, and have some minor potential to create trans isomers; more likely with unrefined, unbleached, undeodorized, raw, pure, virgin polyunsaturated fats

Monounsaturated Fats

- Carbon chain fatty acids with one double bond
- Liquid at room temperature
- Examples: Olive and canola oils
- Smoking points can vary widely depending on the oil
- While some polyunsaturated fats have among the lowest smoking points (e.g., unrefined safflower and sunflower oils 200 – 300 degrees F), some sources of monounsaturated oils have among the higher smoking points (e.g., pomace and extra light olive oil and avocado oil ~ 500 degrees F)
- Stoves and ovens can be as hot as 500 degrees F
- Baking and stir-frying with canola oil does not significantly increase the generation of trans fats

Protein

- Protein contains 4 kcal/gram
- Protein contains amino acids and serves as the major structural building blocks of the human body: bone, muscle, skin, brain, nucleic acids
- Essential amino acids are those which cannot be made by the human body and must be consumed in the diet
  - These include histidine, isoleucine, leucine, lysine, methionine, phenylalanine, threonine, tryptophan, and valine
- Some amino acids can be used as an energy source (converted to glucose or ketones when needed)
- Protein deficiency can lead to a disease state (Kwashiorkor is sufficient calories but insufficient protein)
- Dietary Reference Intake (DRI) for protein is 0.8 to 2.0 grams/kg/day depending upon age, gender, physical activity
- Although in healthy adults, a high protein intake does not adversely affect the kidney, protein restriction may delay progression to dialysis in those with chronic kidney disease

Insulin Controls Fat Metabolism

- Insulin promotes fatty acid and triglyceride synthesis and storage (lipogenesis), and inhibits fat breakdown (lipolysis)
- Foods that cause a rise in blood glucose, such as sugars, starches, or amino acids will stimulate the secretion of insulin from the pancreas
- A nutritional plan that lowers insulin levels may decrease ectopic fat deposition (visceral adipose tissue, intrahepatic fat, and intrapericardial fat), and improve components of the metabolic syndrome independent of weight loss

Principles of Healthful Nutrition

Limit:

- Unhealthful ultra-processed foods of minimum nutritional value such as “sweets,” “junk foods,” cakes, cookies, candy, pies, chips, and ultra-processed meats such as bacon, sausage, hot dogs, pastrami
- Energy-dense foods high in calories
- Energy-dense beverages: sugar-sweetened beverages, juice, cream
- Avoid trans fats and excessive sodium

Encourage:

- Consumption of healthful proteins and fats, vegetables, leafy greens, fruits, berries, nuts, legumes, whole grains
- Complex carbohydrates over simple sugars: Low glycemic index over high glycemic index foods
- High-fiber foods over low-fiber foods
- Many dairy products (while being mindful of caloric content)
- Reading labels rather than marketing claims

Principles of Caloric Intake
(Hunger, Appetite, Satiety, Satiation)

- Hunger = Physiologic craving or need for food (e.g., increased ghrelin, neuropeptide Y, asprosin or decreased leptin)
- Appetite = Desire to eat food, which may be physiologic due to hunger, or may be independent of hunger via responses to psychosocial environments and/or cued responses to senses such as touch, sight, hearing, smell, and taste.
- Satiation = Feeling of fullness within a meal
- Satiety = Feeling of fullness between meals
- Cravings or desire to eat food can be measured by validated scales
- Factors that influence satiety and satiation and thus affect daily *ad libitum* calorie intake include the volume of food, food form and texture, food palatability, protein intake, dietary fiber intake, food availability, sensory specific satiety, sleep deprivation and circadian rhythm alignment/malalignment, physical activity, stress, ketosis, muscle and fat mass, and hedonic drive
    - Example: Whole apples are more satiating than pureed apples, and pureed apples are more satiating than apple juice even with equated amounts of dietary fiber. This may be due to oropharyngeal or incretin response differences from the processing
- Sleep deprivation not only increases hunger but also increases cravings for energy dense foods and decreases physical activity

Principles of Caloric Intake
(Energy Partitioning)

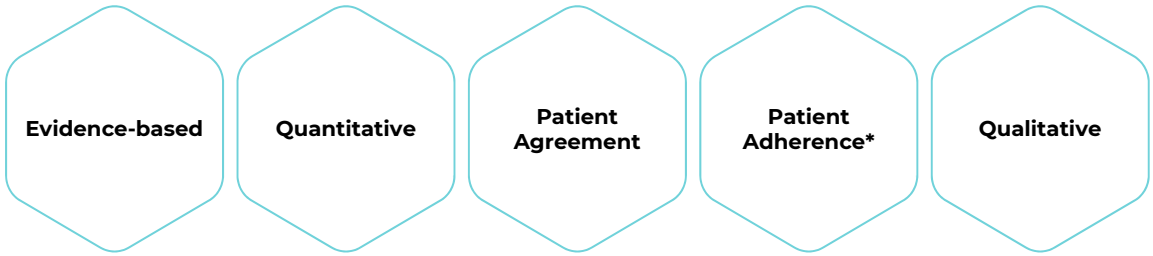
- Caloric partitioning is the distribution of consumed energy to specific tissues
    - Proteins may be preferentially delivered, utilized and stored in muscle tissue
    - Fats are utilized in muscles (i.e., fatty acids) at lower levels of physical activity, and predominantly stored in adipose tissue during periods of positive caloric balance
    - Carbohydrates are preferentially utilized in muscle for immediate energy needs. During positive caloric balance, carbohydrates are stored in liver and muscle as glycogen and ultimately converted to, and stored as fat in adipose tissue
    - Exercise increases partitioning of consumed energy to muscle tissue whereas during physical inactivity increases partitioning of consumed energy to adipose tissue
        - During negative caloric balance, the derivation of energy from muscle tissue is less during concomitant resistance training, which can promote muscle retention or growth during adipose tissue loss
    - Sleep deprivation increases partitioning of consumed energy to adipose tissue (particularly abdominal or visceral) and promotes lean mass wasting

Low Calorie and Non-Nutritive Sweeteners

- In general, low-calorie and non-nutritive sweeteners appear comparable to water in comparison to sugar-sweetened beverages in weight loss interventions
- They may also have physiologic and/or metabolic effects of unknown clinical significance
- There may be heterogeneity response:
  - Saccharin may increase body weight compared to aspartame, rebaudioside A, and sucralose
  - Food cue reactivity from non-nutritive sweeteners may have a gender or weight interaction
- They may also have physiologic and/or metabolic effects of unknown clinical significance

Nutrition Therapy for Obesity

Factors Related to Improved Health Outcomes:



**While possibly counterintuitive, randomized clinical trials do not necessarily support improved weight loss when based upon patient food preference. In fact, meta-analyses suggest that patient choice in weight loss strategies have no significant effect on duration or attrition, with greater weight loss often occurring in the control groups. However, effectiveness of any therapeutic intervention is likely enhanced when patients are engaged and agree to treatment plans.*

Choosing Nutrition Therapy for Obesity

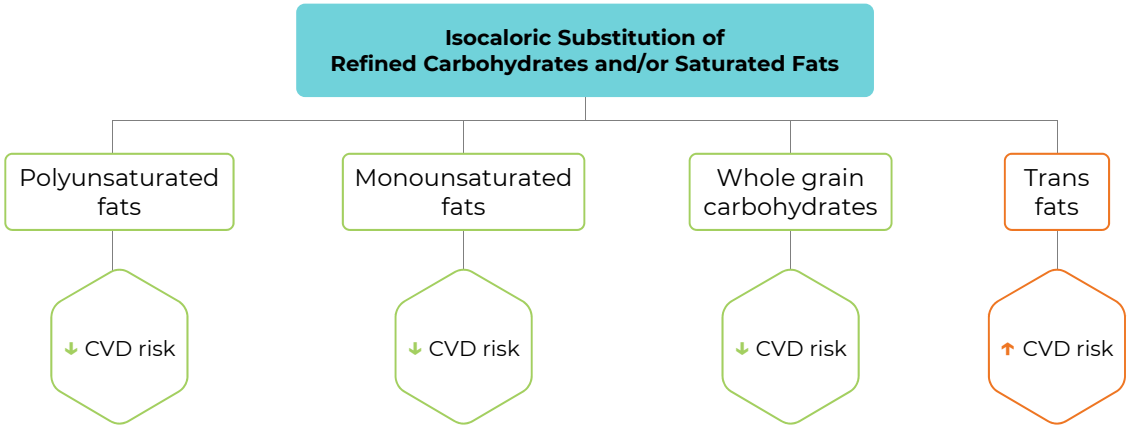
The most Appropriate Nutritional Therapy for Weight Loss is One that is Safe, Effective, and One to which the Patient will Adhere.

- Encourage foods that result in a negative caloric balance to achieve and maintain a healthy weight
- Consider the following:
  - Eating behaviors, and meal patterns
  - Cultural background, traditions, and food availability
  - Time constraints and financial issues
  - Nutritional knowledge and cooking skills
  - Medical conditions potentially affected by the nutrition plan

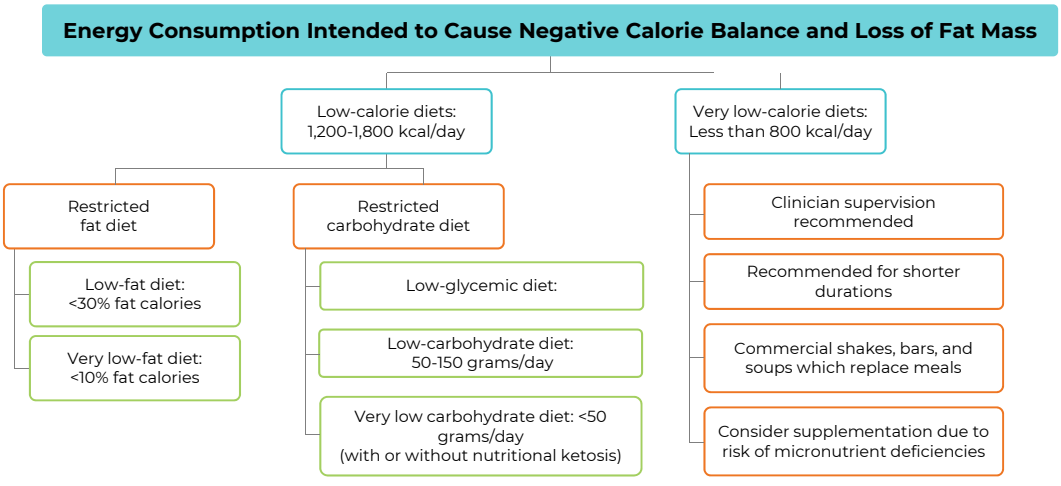
Choosing Nutrition Therapy for Obesity

- Nutritional approaches for weight loss typically focus on the caloric manipulation of the three macronutrients: carbohydrate, fat, or protein
- Very low-calorie diets contain less than 800 kcal/day and require close medical supervision for safety reasons
- Low calorie diets range from 1200-1800 kcal/day (1200-1500 for women, 1500-1800 for men)
- Restricting dietary fat leads to a greater reduction in total and LDL cholesterol, whereas restricting dietary carbohydrate leads to a greater reduction in serum triglycerides and an increase in HDL-cholesterol
- Reduction of carbohydrates can lead to a greater reduction in serum glucose and hemoglobin A1C
- Reduction of carbohydrates and/or saturated fats may promote visceral and liver fat reduction

Choosing Nutrition Therapy for Obesity



Nutrition Therapy for Obesity



Low-calorie Diets: Restricted-carbohydrate Diet

Low-carbohydrate Diet defined as 50-150 grams of Carbohydrates Per Day.
Very Low-carbohydrate Diet defined as <50 grams of Carbohydrates Per Day.

Weight Loss

- May produce modestly greater weight loss compared to fat-restricted dietary intake for the first 6 months
- After 6 months, the net weight loss may be similar to other calorie-restricted nutritional interventions

Metabolic Effects

- Reduces fasting glucose, insulin and triglycerides
- Modestly increases high-density lipoprotein cholesterol levels
- May increase low-density lipoprotein cholesterol levels
- May modestly reduce blood pressure
- The metabolic effects noted above may occur with or without weight loss
- In patients with epilepsy, a very low carbohydrate ketogenic diet (VLCKD) may reduce seizures
- LCKD may possibly improve diabetes mellitus complications (i.e., nephropathy)
- May help increase energy expenditure during weight loss maintenance

Risks

- May produce carbohydrate cravings within the first few days of implementation, which may be mitigated by adding low-glycemic-index carbohydrate foods, may induce gout flare if history of gout, and may result in malaise
- May present challenges in patients undergoing dietary protein restriction (severe kidney disease)
- Due to the possibility of hypoglycemia and hypotension in patients treated for diabetes mellitus and hypertension respectively, blood sugar and blood pressure should be monitored for potential adjustment in applicable metabolic drug treatments

Podcast: Obesity - A Disease
Episode 7

Low-calorie Diets: Restricted-fat Diet

Defined as 10-30% of Total Calories from Fat.

Weight Loss

- After six months, fat-restrictive, low-calorie nutritional intervention generally produces similar weight loss compared to the “low-carb diet”

Metabolic Effects

- If accompanied by weight loss, may reduce fasting glucose, insulin level and modestly reduce blood pressure
- Modestly decreases low-density and high-density lipoprotein cholesterol levels

Risks

- Hunger control may present challenges, which may be mitigated with anti-obesity pharmacotherapy
- If fat restriction results in a substantial increase in carbohydrate consumption, and if weight loss is not achieved, then an increase in dietary carbohydrate intake may contribute to hyperglycemia, hyperinsulinemia, hypertriglyceridemia, and reduced levels of high-density lipoprotein cholesterol

Very Low-calorie Diets

Defined as Less than 800 kcal/day, Typically Implemented Utilizing Specifically Formulated Meal-replacement Products Supervised by a Trained Clinician.

Weight Loss

- Produces more rapid weight loss than low calorie (low-fat or carbohydrate restricted) diets due to lower energy intake

Metabolic Effects

- Reduces fasting glucose, insulin and triglycerides
- May modestly increase high-density lipoprotein cholesterol levels
- May modestly decrease low-density lipoprotein cholesterol
- Reduces blood pressure

Risks

- Fatigue, nausea, constipation, diarrhea, hair loss, brittle nails, cold intolerance, dysmenorrhea
- Small increase in gallstones, kidney stones, gout flare
- Due to the possibility of hypoglycemia and hypotension in patients treated for diabetes mellitus and hypertension respectively, blood sugar and blood pressure should be monitored for potential adjustment in applicable metabolic drug treatments
- Potential insufficient micronutrient intake, which may predispose to cardiac dysrhythmias and muscle cramps. Consider screening for vitamin D, iron, thiamine, folate, and vitamin B12
- Weight regain will occur if patients are not taught how to maintain healthful eating when transitioning to non-meal replacement

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Popular Dietary Patterns for Obesity

Includes many Dietary Patterns but must be Calorically Restricted to Effectively Treat Obesity. Weight Loss and Metabolic Effects Vary.

- Mediterranean diet
- Therapeutic lifestyle diet
- DASH (Dietary Approaches to Stop Hypertension)
- Ketogenic (modified Atkins) diet
- Ornish diet
- Paleo diet
- Vegetarian or vegan diet
- Intermittent fasting / time restricted eating
- Commercial diet programs

The OMA does not endorse any particular dietary pattern

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Mediterranean Diet

The Mediterranean Diet is not a defined "diet," but rather a generalized term to described several meal pattern variants often found in Greece, Italy, and Spain. The Mediterranean Diet has the most robust scientific support in reducing cardiovascular disease risk, although it has only been compared to a few diets.

Encouraged

- Olive oil as main source of fat
- Vegetables, fruit, legumes, whole grains, nuts, and seeds
- Moderate intake of red wine
- Moderate consumption of seafood, fermented dairy products (cheese and yogurt), poultry, and eggs

Discouraged

- Limit consumption of high amounts of red meat, meat products, and ultra-processed carbohydrates

** Saturated fats are often discouraged with the Mediterranean Diet; olive oil is a staple of most definitions of the Mediterranean Diet. However, some Mediterranean cuisine may include lard and butter for cooking, and olive oil for dressing salads and vegetables*

Therapeutic Lifestyle Change Diet (TLC)

The TLC Diet is a low-fat meal-plan variant that was recommended by the National Cholesterol Education Program, Adult Treatment Panel. It is the "diet" classically utilized in the conduct of lipid clinical trials.

Encouraged

- Total fat: 25–35% of daily calories
  - Polyunsaturated fat: Up to 10% of total daily calories
  - Monounsaturated fat: Up to 20% of total daily calories
- Carbohydrate: 50% to 60% of total calories
- Soluble fiber: At least 5-10 grams a day, preferably 10-25 grams a day
- 2 grams per day of plant stanols or sterols through foods or dietary supplements

Discouraged

- Limit saturated fat: < 7% of total calories
- Limit cholesterol: < 200 mg a day
- Avoid foods with *trans* fatty acids.

Ketogenic Diet (Keto or Modified Atkins Diet)

The Ketogenic Diet is illustrative of a carbohydrate-restricted nutritional intervention that promotes utilization of fat for energy and generates ketosis, which may reduce hunger.

Encouraged

- **The induction phase** allows no more than 20 grams of carbohydrate per day from non-starchy vegetables and leafy greens; encourages adequate protein, and higher proportion of dietary fat to reduce insulin levels and generate a state of nutritional ketosis.
- **The ongoing weight loss phase** allows a wider variety of vegetables, seeds and nuts, and low-glycemic fruits (i.e., strawberries and blueberries).
- **The pre-maintenance phase**, after the goal weight is achieved, allows carbohydrate intake to be slowly increased - provided weight gain does not occur.
- **In the maintenance phase**, 60 to 90 grams of carbohydrates per day is allowed if weight and health benefits are maintained, which may allow legumes, whole grains, and fruits.
- All phases encourage a balance of saturated, monounsaturated, and polyunsaturated fatty acids.

Discouraged

Avoid:

- Ultra-processed and refined foods
- Foods with a high glycemic index / glycemic load
- Foods rich in *trans* fatty acids

In all but the maintenance phase, limit:

- Cereals, breads, and grains
- Dairy products, except cheese
- Starchy vegetables
- Most fruits

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Ketogenic Diet (Keto or Modified Atkins Diet)

The Ketogenic Diet is illustrative of a carbohydrate-restricted nutritional intervention that promotes utilization of fat for energy and generates ketosis, which may reduce hunger.

Advantages

- May contribute to clinically meaningful weight loss in patients with overweight or obesity
- May reduce hunger
- Lower carbohydrate food intake will typically result in lower postprandial glucose and insulin levels
- If associated with weight loss, a ketogenic diet may improve glucose metabolism with improved insulin sensitivity, reduced fasting glucose, and reduced fasting insulin levels
- May lower diastolic blood pressure
- May reduce triglyceride and increase high density lipoprotein cholesterol levels
- Ketonemia may help treat seizures
- Effects upon physical exercise performance are inconsistent
- Possible patient-specific adjunct to multifactorial therapy for certain kinds of cancers

Disadvantages

- May increase low density lipoprotein (LDL) cholesterol levels, sometimes substantially so in patients with genetic hypercholesterolemia
- An increase in LDL cholesterol with the ketogenic diet may be mitigated by consumption of polyunsaturated fats versus saturated fats
- May not improve insulin sensitivity in patients not experiencing weight loss
- May cause transient fatigue and mild decrease in mental cognition upon start of a ketogenic diet
- Effects upon physical exercise performance are inconsistent

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Ketogenic Diet (Keto or Modified Atkins Diet)

Health Perspective: Saturated Fats

Most studies suggesting saturated fats are unhealthful evaluated isocaloric substitution for other nutrients, not during clinically meaningful weight reduction. However, many patients with obesity who use carbohydrate restricted diets are attempting to achieve clinically meaningful weight reduction

Carbohydrate restricted diet may help reduce body weight with improvement and/or remission of adiposopathic metabolic diseases and major cardiovascular disease (CVD) risk factors:

- Diabetes mellitus
- High blood pressure
- Hypertriglyceridemia

Other benefits include reduced:

- Insulin levels
- Risk of certain cancers
- Hunger
- Seizure episodes in patients with seizure disorders

Some patients with genetic dyslipidemias: may have moderate to marked increases in low density lipoprotein cholesterol with saturated fat consumption, which if excessive or uncontrolled, should prompt replacement of saturated fats with poly or monounsaturated fats

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Ketogenic Diet (Keto or Modified Atkins Diet)

Management of the Rare Patient with Marked Increases in LDL Cholesterol and/or LDL Particle Number with Ketogenic Diet

- The ketogenic diet is generally associated with a null to small increase LDL-c and/or LDL particle number. However, in rare cases, individuals may have a marked increase in LDL-c and/or LDL particle number.

Confirmation and Evaluation

- Repeat the lipid testing
- Evaluate for new onset or worsening of secondary causes of hypercholesterolemia (e.g., diabetes mellitus, hypothyroidism, nephrotic syndrome, liver disease)
- Evaluate for recent change in medications that may worsen cholesterol levels (e.g., some beta-blockers, corticosteroids, amiodarone, cyclosporin, anabolic steroids, protease inhibitors, some diuretics)
- Evaluate for diet-sensitive genetic dyslipidemias (e.g., sitosterolemia)

Management

- Replace dietary saturated fats with polyunsaturated or monounsaturated fats
- Reduce dietary cholesterol
- Consider a trial period off the ketogenic diet to determine if elevated lipid levels resolve
- Consider a trial of ezetimibe (i.e., if suspected hyperabsorption of intestinal cholesterol)
- Consider cholesterol-lowering drug treatment (e.g., statins)

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Ketogenic Diet (Keto or Modified Atkins Diet)

Sitosterolemia: Illustrative Example of a Diet-sensitive Genetic Condition that can Result in High Cholesterol

- Beta-sitosterolemia (phytosterolemia) is a rare autosomal recessive disorder that may phenotypically resemble heterozygous familial hypercholesterolemia
- A loss of function mutation impairs the efflux of absorbed consumed plant sterols and animal cholesterol from intestinal and hepatic cells into the intestinal and biliary lumen
- Clinical findings include tendon xanthomas and increased cardiovascular disease risk out of proportion to the patient's lipid profile. Conversely, some patients may exhibit marked hypercholesterolemia, despite no immediate family history of hypercholesterolemia, with potentially wide fluctuations of cholesterol levels during nutritional changes
- Diagnosis is made by measuring plant sterol levels (sitosterol, campesterol) or genetic testing
- Patients may respond poorly to statins but may respond well to reduced dietary plant sterol and cholesterol consumption, bile acid sequestrants, and/or ezetimibe

Ornish Diet

The Ornish Diet is illustrative of a fat-restricted nutritional intervention.

Encouraged

- Foods are best eaten in their natural form
- Vegetables, fruits, whole grains, and legumes
- One serving of a soy product each day
- Limited amounts of green tea
- Fish oil 3-4 grams each day
- Small meals eaten frequently throughout the day

Discouraged

- Limit dietary fat: < 10% of total daily calories
- Limit dietary cholesterol: ≤ 10 mg per day
- Limit sugar, sodium, and alcohol
- Avoid animal products (red meat, poultry, and fish) and caffeine (except green tea)
- Avoid foods with *trans* fatty acids, including vegetable shortening, stick margarines, and commercially prepared foods, such as frostings; cake, cookie, and biscuit mixes; crackers and microwave popcorn; and deep-fried foods
- Avoid refined carbohydrates and oils

Dash Diet

The "Dietary Approaches to Stop Hypertension" (DASH) is a diet pattern promoted by the U.S. National Heart Lung and Blood Institute, primarily to treat high blood pressure.

Encouraged

- Vegetables, fruits, and whole grains
- Fat-free or low-fat dairy products
- Fish, poultry, and lean meats
- Nuts, seeds, and legumes
- Fiber and the minerals calcium, potassium, and magnesium

Discouraged

- Limit sodium: 1,500-2,300 mg per day
- Limit total fat: ~27% of total daily calories
- Limit saturated fat: <6% of total daily calories
- Limit cholesterol: ≤ 150 mg per day for a 2,100-Calorie eating plan
- Avoid red and processed meats
- Avoid sugar-sweetened beverages
- Avoid foods with added sugars

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Paleolithic Diet

Paleolithic nutritional intervention is based upon a diet pattern presumed to exist during the Paleolithic period (lasting 3.4 million years and ending 6000-2000 BC). It differs from some other diets in that it excludes grains, dairy, and ultra-processed foods.

Encouraged

- Fresh vegetables, fruits, and root vegetables
- Grass-fed lean red meats
- Fish/seafood
- Eggs
- Nuts and seeds
- Healthful, naturally-produced oils (olive, walnut, flaxseed, macadamia, avocado, and coconut)

Discouraged

Avoid:

- Cereal grains
- Legumes, including peanuts
- Dairy products
- Potatoes
- Ultra-processed foods
- Refined sugar, refined vegetable oils, and salt

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Vegetarian Diet*

A vegetarian nutritional intervention includes a meal plan consisting of foods that come mostly from plants.

Encouraged

- Vegetables
- Fruits
- Whole grains
- Legumes
- Seeds
- Nuts
- May include eggs and milk

Discouraged

- Fowl
- Fish
- Beef
- Pork
- Lamb

**Plant-based nutritional intake is generally associated with weight loss, reduced risk of heart disease (including heart failure), and beneficial effects on metabolic diseases, some cancers, and possibly all cause mortality. However, these potential benefits may be negated when more healthful plant-based whole foods (i.e., with natural fiber and nutrients) are replaced by ultra-processed foods, fried foods, and refined carbohydrates. Vegetarian diets may also result in deficiencies of micronutrients such as vitamin B12, which may require monitoring and replacement when appropriate.*

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Vegetarian Diet Variants

Vegan ("Total Vegetarian"): Only plant-based foods (e.g., fruits, vegetables, legumes, grains, seeds, and nuts) with no animal proteins or animal by-products, such as eggs, milk, or honey

Lacto-vegetarian: Plant foods plus some or all dairy products (e.g., cheese)

Lacto-ovo Vegetarian (or Ovo-lactovegetarian): Plant foods, dairy products, and eggs

Semi or Partial Vegetarian: Plant foods and may include chicken or fish, dairy products, and eggs, but not red meat

Pescatarian: Plant foods and seafood

Flexitarian: Mostly plant-based foods (minimally processed), with occasional fish, meat, and animal products in moderation

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Fasting (e.g., alternative day, intermittent, time-restricted eating)

- As effective as continuous caloric restriction for overall caloric restriction
- Potential advantages:
  - Reducing "decision fatigue" regarding food selection
  - Quickly reversible
  - May better fit in day-to-day patient scheduling (including Ramadan)
  - May reduce caloric intake with variable effects on lean body mass, resting metabolic rate, and total energy expenditure, often dependent upon physical activity
  - May reduce body weight and improve metabolic parameters (e.g. improve insulin sensitivity, blood pressure, lipids, and inflammatory markers)
- Potential disadvantages
  - Does not necessarily emphasize healthful meal quality
  - May not be appropriate for patients with eating disorders (e.g., bulimia or binge-eating disorder)
  - Increases the risk of hypoglycemia among patients with diabetes mellitus who do not appropriately adjust their hypoglycemic anti-diabetes drug treatments (e.g., insulin, sulfonylurea)
  - Unclear if sustainable on a lifetime basis for a lifelong disease (i.e., obesity)
  - Most long-term evidence of efficacy, health benefits and safety are derived from animal studies
  - Prolonged fasting (not intermittent fasting) may promote gout, urate nephrolithiasis, postural hypotension, and cardiac dysrhythmias

Podcast : Obesity - A Disease Episode 6

Obesity Medicine Association

Physical Activity and Obesity

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Top 10 Takeaway Messages: Obesity and Physical Activity

1. Routine physical activity may improve body composition
2. Routine physical activity may improve adiposopathic endocrine and immune body processes
3. Physical activity may improve metabolic, musculoskeletal, cardiovascular, pulmonary, mental, sexual, and cognitive health
4. Dynamic training promotes weight loss and may help prevent weight gain or regain
5. Resistance training may improve body composition, prevent muscle loss during weight loss, and increase resting energy expenditure
6. In addition to physical exercise, increased energy expenditure can be achieved via increased leisure time physical activity and non-exercise activity thermogenesis (NEAT)
7. A common physical exercise prescription (FITTE) includes frequency, intensity, time spent, type, and enjoyment
8. Metabolic equivalent tasks (METS) are used to assess the intensity of physical exercise, with one MET equal to the amount of energy expended for one minute while lying down at rest [equal to 3.5 milliliters of oxygen consumption per kilogram of bodyweight per minute (3.5 ml/kg/min)]
9. Standing = 2 METS; walking 4 miles per hour = 4 METS; running 10 miles per hour = 16 METS
10. Progress can be measured through tracking activity patterns over time via various activity logs, or can be measured by using a reliable technique to measure body composition

Physical Activity to Improve Health

Adiposopathy (Sick Fat Disease)

- Assist with weight maintenance
- Assist with weight loss
- Improve body composition
- Improve adiposopathic physiologic disturbances
- Possibly improve adipocyte function (“train” fat cells)
  - Improve insulin sensitivity
  - Increase mitochondrial biogenesis
  - Increase browning (“beiging”) of fat cells

Non-adipose Parameters

- Improve metabolic health
- Improve musculoskeletal health
- Improve cardiovascular health
- Improve pulmonary health
- Improve neurological health
- Improve mental health (e.g., improve mood, promote happiness & sense of well-being, reduce stress)
- Improve sexual health
- Improve cognitive heath
- Reduce risk of cancer, and improve response to cancer treatments

Medical Evaluation to Ensure Safety before Beginning New Exercise Program

- Assess current physical activity level
- Assess readiness (e.g., PAR-Q)
- Agree upon patient expectations and goals (with optional written "contract")
- Assess potential need for medical testing/evaluation (i.e., cardiac stress testing, pulmonary function tests, musculoskeletal assessment, etc.)
- Assess mobility, fitness, and potential equipment needs or modifications
- Potential adjustment of medications
  - Before start of physical activity plan (e.g., diabetes and blood pressure medications)
  - During implementation of physical activity plan
- Optimal default
  - Back-up plan

Assess Mobility

Unable to Walk

- Seated exercise program
- Arm exercises (i.e., arm cycling)
- Swimming/aquatic exercises (e.g., shallow or deep-water exercises)
- Resistance/gravity-mediated physical activity (seated leg raises, band exercises, dumbbells) Consider physical therapy evaluation
  - Recommend rehabilitation & physical therapy guided activity program
  - Set physical activity goals
  - Assess special equipment needs

Limited Mobility, Able to Walk

- Walking
- Swimming/aquatic exercises (e.g., shallow or deep-water exercises)
- Resistance/gravity-mediated physical activity (seated leg raises, band exercises, dumbbells)
- Balance exercises (walk straight line, standing on one foot, standing/sitting up and down, target "core" muscles)
- Assess for special equipment needs

No Substantial Limitations to Mobility

- Exercise/physical activity prescription plan driven by patient and guided by clinician
- Assess for special equipment needs

Priority: Increase Energy Expenditure

Dynamic (Aerobic) Training

- Some physical activity is better than none
- At least 150 minutes (2.5 hours) per week of moderate physical activity or at least 75 minutes (1.25 hours) per week of vigorous intensity aerobic exercise = most health benefits, promote modest weight loss, and prevent weight gain
- Some may further benefit from at least 300 minutes (5 hours) per week of moderate physical activity or at least 150 minutes (2.5 hours) per week of vigorous intensity aerobic exercise = promote more robust weight loss and prevent weight regain after weight loss

Resistive (Anaerobic) Strength Training

- Muscle strengthening of major muscle groups two or more times per week, with an emphasis in increasing total muscle mass, most efficiently achieved by training large muscle groups – all which may increase percent of lean body mass
- Utilizing appropriate weight-lifting techniques, using a variety of free weights, machines, and resistance bands may elicit less boredom and provide greater flexibility regarding scheduling and location
- Development of "core" muscles is important for posture and balance stabilization, and include muscles located at the midsection of the body (i.e., abdomen, back, hips)
- During negative caloric balance, resistance training can mitigate muscle loss and limit reduced resting metabolic rate
- Short-term sore muscles may be expected
- Sore joints suggest poor technique, with possible need for medical evaluation and physical activity modification
- Prioritize muscle mass metrics (e.g., myotape measurements) versus amount of weight lifted

Priority: Decrease Physical Inactivity

Leisure Time Physical Activity

- Engage in competitive sport activities involving substantial physical activity, best if on a routine basis
- Engage in non-competitive sports such as running, hiking, cycling, cross-fit training, etc.
- Outdoor warm-weather physical activity in sunlight may facilitate negative caloric balance and have other health benefits, but need to avoid excessive sun exposure
- Engage in physical activity sport-alternatives, such as dancing
- Use of apps that encourage physical activity and/or tracking of this activity have been shown to increase overall physical activity and can lead to significant changes in body weight.

Transportational/Occupational Non-exercise Activity Thermogenesis (NEAT)

- Walk short distances instead of automated transportation
- Take stairs instead of elevator
- Carry overnight travel bags instead of using rollers (akin to farmers walk)
- Active work environment (i.e. standing desks, walking desks)
- Avoid prolonged inactivity
    - Take breaks from inactivity
    - Walk, stand, incidental movements

Exercise Prescription

- Exercise prescription (FITTE)
  - **F**requency
  - **I**ntensity
  - **T**ime spent
  - **T**ype
  - **E**njoyment level

- Exercise prescription (FITT-VP)
  - **F**requency
  - **I**ntensity
  - **T**ime or duration
  - **T**ype or mode
  - **V**olume or total energy expenditure of the exercise
  - **P**rogression of the exercise

Metabolic Equivalent Tasks (METS)

- METS are used to assess the intensity of physical exercise: Kcal = METS x weight x time
- Equal to the amount of energy expended in one minute while lying down at rest
- Equal to ~3.5 milliliters of oxygen consumption per kilogram of bodyweight per minute (3.5 ml/kg/min)
- Oxygen consumption may be decreased with increased age
- Standing = 2 METS
- Walking 4 miles per hour = 4 METS
- Running 10 miles per hour = 16 METS

Tracking Progress

- Daily activity logs (written or electronic)
- Pedometer/accelerometer logs
- Dynamic training metrics (i.e., miles run, laps swam, etc.)
- Resistance training metrics (i.e., muscle-circumference measurements, reps, sets, etc.)
- Percent body fat measurements

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Motivational Interviewing

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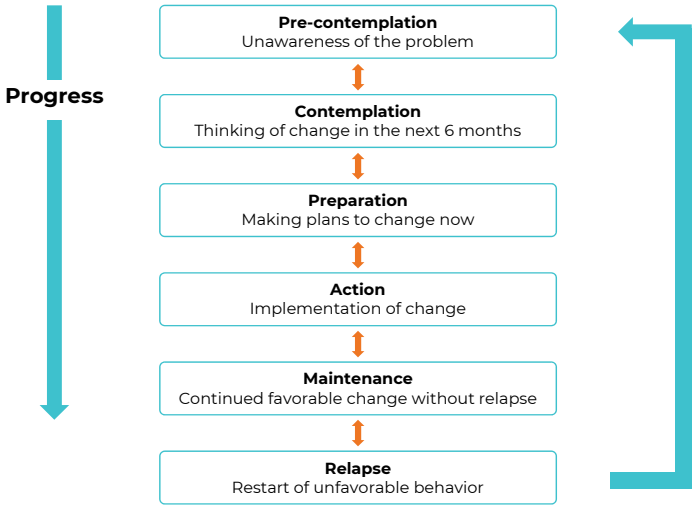
Top 10 Takeaway Messages: Obesity and Motivational Interviewing

1. **Motivational interviewing** is a collaborative, patient-centered goal-directed counseling approach intended to guide people toward positive behavior change, which in the context of obesity medicine, promotes a healthier body weight and a healthier body composition among patients with pre-obesity/obesity
2. **Stages of change** that may be evaluated during motivational interviewing include pre-contemplation, contemplation, preparation, action, maintenance, and relapse
3. **General motivational interviewing principles** include empathy, avoiding arguments, developing discrepancy, resolving ambivalence, and supporting self-efficacy
4. **Empathy** involves communication, understanding, collaboration, support, encouragement and listening
5. **Avoiding arguments** involves recognizing types of resistance (arguing, denying, ignoring, interrupting) and then "rolling with resistance" through reflection, shifting focus, reframing, and/or siding with the negative
6. **Developing discrepancy** explores the mismatch between where the patient is today, and where the patient says he/she wants to be in the future
7. **Resolving ambivalence** is amplifying discrepancy and addressing the uncertainty for the desire for change.
8. **Supporting self-efficacy** is affirming favorable results though focusing on patient successes and highlighting patient skills and strengths
9. **The 5A's** of motivational interviewing include Ask, Assess, Advise, Agree, and Arrange or Assist
10. **FRAMES** is a common motivational interviewing acronym that stands for Feedback, Responsibility of the patient, Advice to change, Menu of strategies, Empathy, and Self-Efficacy; OARS is a common motivational interviewing acronym that stands for Open-ended questions, Affirmation, Reflections, and Summaries

Motivational Interviewing: Stages of Change



Motivational Interviewing: Focus

Collaboration

- Working together to find and implement pragmatic solutions
- Not focusing on who is right and who is wrong

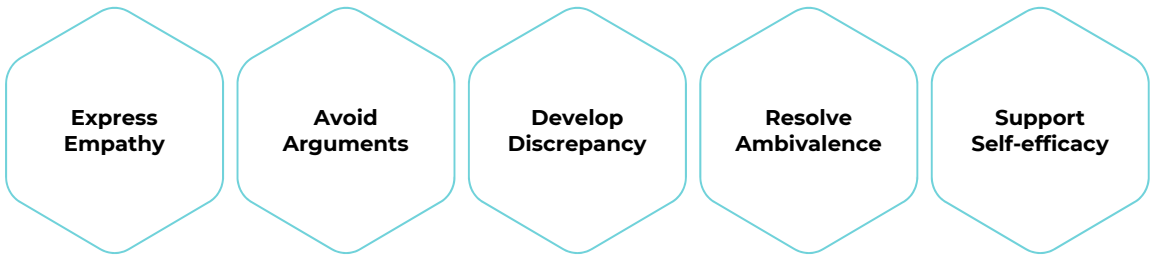
Evocation

- Drawing out the patient's thoughts and ideas regarding solutions
- Not telling the patient what to do

Autonomy

- Empowering the patient to own the solution
- The patient is the expert on his/her own life, not the clinician

Motivational Interviewing: Principles



Express Empathy

- Communicate
- Understand
- Collaborate
- Support
- Encourage
- Listen

Avoid Arguments: Resistance

Types of Resistance

- Resistance in changing behavior may arise when the patient:
  - Views the problem or solution differently than the clinician
  - Feels the clinician is being too judgmental and/or authoritative
- Types of resistance
  - Arguing
  - Denying
  - Ignoring
  - Interrupting

Roll with Resistance

- Rolling with resistance avoids arguments and confrontations by choosing not to challenge patient actions and statements that suggest resistance to change
- May be especially useful during initial interactions with the patient

Therapeutic Paradox

- Therapeutic paradox is analogous to "reverse psychology," wherein the clinician makes a statement seemingly in support of no change in hopes the patient will make an argument for change
  - *"It sounds like now is not the best time for you to make changes."*
  - *"You seem to be saying you have a lot going on right now that keeps you from making changes, so what do you think is the best way for us to move forward at this time?"*

Avoid Arguments: Roll with Resistance Examples

Reflection

- Simple reflection: *"You don't think you can lose weight right now."*
- Amplified reflection: *"People worry too much about your weight; your current body weight is not really a problem."*
- Double-sided reflection: *"You had previously suggested you were committed to weight loss, but now you no longer feel commitment is necessary."*

Shifting Focus

- *"Your conflict with your contractor is obviously stressful to you; but for now, perhaps we should focus on other issues that led to the entries in your food journal."*

Reframing

- *"You say you get angry when your family and friends express concern about your body weight. To what degree do you believe they intend to frustrate you, and to what degree do you believe their concern reflects how much they care for you and your health?."*

Siding with the Negative

- *"It sounds like now is not the best time for you to make changes"*

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Discrepancy and Ambivalence

Identify Discrepancy

- The process of exploring the mismatch between where patients are today and where they want to be in the future
- Contrast current behavior and life goals
- Acknowledge positive and negative aspects of current behavior
- Promotes motivation for change

Amplify Discrepancy

- Amplifying discrepancy can help resolve ambivalence
- May facilitate thoughts of change

Resolve Ambivalence

(defined as uncertainty in the desire for change)

- Resolution of ambivalence helps facilitate change
- Can involve discussing:
  - Benefits for change
  - Risks of change
  - Benefits and risk of not changing

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Motivational Questioning: Evoking Change-talk Examples

Elicit Talk of Change

- *"Why do you want to change?"*
- *"How important is it that you change?"*
- *"What values are most important to you?"*
- *"How do your actions fit your values?"*
- *"How do you plan to change?"*
- *"How confident are you that you can change?"*

Exploring Past and Future

- *"How were things better in the past?"*
- *"What may happen if things stay the same?"*
- *"How would you like for things to change within the next year?"*
- *"What are the best ways for you to change in the next year?"*

Query Extremes

- *"If you were completely successful in making this change, how would things be better in the future?"*
- *"What is the worst-case scenario if you do not change?"*
- *"What is the best-case scenario if you do change?"*

Change Metric Examples

Importance of Change

- *"On a scale of 1-10, where one is not important and 10 is most important, how important is it for you to change?"*
- *"Why are you not at a lower/higher number?"*

Readiness to Change

- *"On a scale of 1-10, where one is not ready to change and 10 is absolutely ready to change, how ready are you to change?"*
- *"Why are you not at a lower/higher number?"*

Confidence in Ability to Change

- *"On a scale of 1-10, where 1 is not at all confident and 10 is absolutely confident, how confident are you in your ability to change?"*
- *"Why did you choose this number and not a lower/higher number?"*

Decision-balancing Examples

- *"Write down some of the positive things and negative things about your current eating and physical activity levels."*
- *"It sounds like you are frustrated by your current behavior but feel change would be too difficult. It might be helpful to make a chart of the pros and cons of each option – keeping things the same and changing."*

Self-efficacy: Affirmation

Supporting Self-efficacy

- Motivational interviewing assumes the patient is capable of making change
- Change is promoted by focusing on past patient successes and highlighting existing patient skills and strengths

Evoking Questions

- How might you go about taking the first steps in making a change?"
- "What obstacles might you encounter and how would you overcome them?

Affirmation Statements

- *"I see you have a real commitment toward improving your health."*
- *"Your spirituality and family are two strengths that seem to be helping you stick with your plan.."*
- *"It seems that despite a lot of things happening, you have managed to stay on course, and that is really impressive."*
- *"Although you have not seen the results you were hoping for on the scale, the fact you have returned reflects how serious you are about reducing your weight."*

Self-efficacy: Advice/Feedback and Summary Examples

Advice/Feedback

- *"What do you know about how body fat can affect your…?"*
  - Blood sugar/pressure/cholesterol
  - Heart
  - Breathing
  - Bones and joints
  - Possible pregnancy
  - Quality of life
  - [Other clinical consequences experienced by the patient]

Summary

- *"From what you've said, you want to lose weight mainly because you are concerned about your health and because your family is concerned."*
- *"It seems that with your commitment to the weight-management plan, and with support from your family, most everyone agrees that overall you are making great progress."*
- *"Although you had made progress in the past, your weight went up a bit this time. But it is good you did not get so discouraged as to cancel your appointment."*

Motivational Interviewing Techniques: Micro-Counseling (OARS)

Open-ended Questions

- Avoids binary answers such as "yes" or "no"
- Invites expression of elaborative thoughts
- May help patient explore reasons for and possibility of change

Affirmation

- An expressed recognition of the patient's strengths and how these strengths can be applied to implement favorable change
- Affirmations to the patient by the clinician should be:
  - Relevant
  - Genuine

Reflections

- Careful listening can often be the most effective form of empathy
- After careful listening, the clinician is better able to:
  - Facilitate evocation
  - Develop discrepancy
  - Amplify and resolve ambivalence
  - Offer collaboration
  - Support self-efficacy

Summaries

- Each counseling session should conclude with a summary of:
  - What was discussed
  - Shift attention from negative past failures and toward positive but realistic future goals
  - Establish metrics to measure success of future goals
  - Outline follow-up plans

Motivational Questioning: General Approach Examples

Open-ended Questions

- *"If you don't mind, can you tell me why are you here today?" (Incorporates permission.)*
- *"What do you hope we can accomplish today?"*
- *"What do you realistically think we can accomplish today?"*

Reflective Listening

- *"From what you are telling me, it sounds like you (or your family/friends) want you to lose weight, but you..."*
  - *"... have concerns."*
  - *"... are unsure how."*
  - *"... are unsure if you need to."*
  - *"... are unsure if you want to."*
  - *"... are unsure if you can."*
  - *"... are unsure if you are committed to change."*

Normalizing

- *"While no situation is the same, in general, many people often have problems losing weight."*
- *"Many people feel like you: they want to lose weight but find it difficult."*
- *"Many people have repeatedly tried to lose weight in the past before they were finally successful."*

Motivational Interviewing Techniques: 5A's of Obesity Management

Ask

- Ask for permission to discuss body weight.
- Explore readiness for change.

Assess

- Assess BMI, waist circumference, and obesity stage.
- Explore drivers and complications of excess weight.

Advise

- Advise the patient about the health risks of obesity, the benefits of modest weight reduction (i.e., 5-10 percent), the need for long-term strategy, and treatment options.

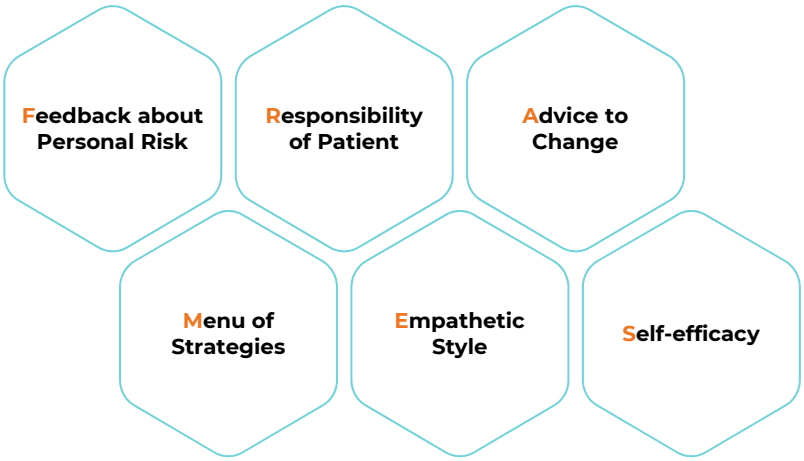
Agree

- Agree on realistic weight reduction expectations, targets, behavioral changes, and specific details of the treatment plan.

Arrange/ Assist

- Assist in identifying and addressing barriers; provide resources; assist in finding and consulting with appropriate providers; arrange regular follow up.

Motivational Interviewing Techniques: FRAMES



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Behavior Therapy

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Top 10 Takeaway Messages: Obesity and Behavior Therapy

1. Eating behavior in patients with increased body fat often reflects the imbalance in physiologic forces that strongly resist weight reduction and weakly resist weight gain. This is analogous to the imbalanced physiologic response between hypoglycemia (marked symptoms and strong signals to immediately consume food) and hyperglycemia (often no symptoms and often no signal to change eating behavior)
2. Eating behavior is affected by all 5 senses (sight, smell, hearing, taste, and feel)
3. Eating behavior can be affected by genetic predisposition, mental stress, emotions, habitual time cues, environment, information gap, reward factors, and psychiatric disease
4. Eating behavior can be affected by eating disorders (e.g., binge-eating disorder, bulimia nervosa, sleep-related eating disorder and night-eating syndrome)
5. Physical inactivity behavior may be due to patient musculoskeletal, neurologic, pulmonary, cardiac, and other health disorders
6. Physical inactivity behavior may be related to fatigue, disinterest, and unhealthful environment (e.g., availability and excessive utilization of conveniences).
7. Behavior related to weight regain may be related to personal and physiologic priority imbalances (i.e., "lack of time") as well as physiologic changes of a weight reduced state
8. Behavior therapy elements for optimal success include promoting behaviors that are doable, efficacious, measurable, and which engage self-ownership
9. Behavior therapy implementation optimally includes frequent encounters with qualified medical professionals, education, stimulus control, cognitive restructuring, goal setting, self monitoring, behavioral contracting, problem solving, social support, and other contingencies
10. For patients ready for change, healthful nutrition and physical activity may be aided by weight management technologies, access to healthful nutrition and physical activity resources and/or knowing the existence of social media resources applicable to healthful nutrition and physical activity.

Why Do People Eat Like They Do?

Physiologic

- Strong biologic forces that resist weight reduction
- Weak biologic forces that resist weight gain
- Hypothalamic dysfunction
  - Trauma
  - Inflammation
- Hunger before meals
- Lack of satiety after meals
- Eating to facilitate sleep
- Food as an addiction in susceptible individuals
- Five senses central nervous system signaling:
  - Sight or images of food
  - Smell of food
  - Hear talk of food, sounds of food (cooking, wrapper opening)
  - Taste of food
  - Feel of food (texture, size, esophageal passage, stomach fullness) and feel of lack of food (e.g., vibration of empty stomach - borborygmi)

Why Do People Eat Like They Do?

Mental Stress

- Chronic stress-induced limbic (e.g., hypothalamic) endocrinopathies and immunopathies
- Chronic stress-induced cerebral endocrinopathies and immunopathies
- Chronic stress-induced priority replacement of personal, work, or emotional priorities that overtake nutritional and physical activity priorities
- Mental stress may impair self-regulation and promote choosing unhealthful (immediately rewarding ultra-processed) foods over more healthful (delayed-gratification unprocessed) foods

Timing and Emotions

- Timing
  - It's mealtime
  - Special occasions
  - Holidays
- Emotions
  - Surrogate for love and/or affection
    - For self
    - For others (children and friends)
  - Celebrate happiness
  - Soothe sadness
  - Avoidance: Cooking or eating can be a successful accomplishment, preferable to more challenging activities or situations
  - Treat:
    - Boredom
    - Fatigue
    - Stress

Why Do People Eat Like They Do?

Environment

- Others are eating
- Food is available
- Offers of free food
- Highly researched and effective advertisements for energy dense foods
- Perceived obligations
  - Family gatherings
  - Business meetings
  - Clean-plate syndrome

Information Gap

- Lack of education about proper nutrition
- Challenges regarding access to nutritional information, especially when eating out
- Caloric content
- Nutritional content
- Marketing messages that, while sometimes clinically relevant, can sometimes be misleading
  - "low fat"
  - "multigrain"
  - "no added sugar"
  - "natural sugar"
  - "cholesterol free"
  - "heart healthy"
  - "organic'
  - "gluten-free"
  - "fortified/enriched"

Why Do People Eat Like They Do?

Reward

- Eating as a remuneration / reward for an accomplishment or "good day"
- Eating as compensation for a "bad day"
- Eating for pleasure, not because of hunger
- Over-consumption of ultra-refined, hyperpalatable food may affect the brain's reward system
  - Stimulates opioid release
  - Decreases biologic stress response
  - May ultimately simulate addiction-like reward deficits, which promotes compulsive eating

Why Do People Eat Like They Do?

Eating Disorders

- Binge-eating disorder
- Bulimia nervosa
- Night-eating syndrome
- Sleep-related eating disorder

Eating Disorders and Obesity: Binge-eating Disorder

Diagnosis

- Frequent episodes of consuming large amounts of food more than once per week for at least three months
  - No self-induced vomiting (purging)
  - No extra exercising
  - Feelings of lack of self control, shame, and guilt
- Occurs in up to 3% of U.S. adults
- May occur in up to 50% of patients with severe obesity
- Eating Attitudes Test or Binge-Eating Scale may assist with diagnosis

Severity Based Upon Episodes Per Week:

- Mild = 1 – 3; Moderate = 4 – 7; Severe = 8 – 13; Extreme = ≥ 14

Treatment

- Often requires treatment by a qualified clinician
- Cognitive behavior therapy
- Lisdexamfetamine dimesylate is the only pharmacotherapy with an FDA indication to treat binge-eating disorder
- Although not FDA indicated for this use, clinical trials suggest other pharmacotherapies may be efficacious
  - Some selective serotonin reuptake inhibitors
  - Topiramate
  - Topiramate-Phentermine

Lisdexamfetamine Dimesylate

Indications and Use:

- Lisdexamfetamine dimesylate is a central nervous system stimulant indicated for the treatment of:
  - Moderate to severe binge-eating disorder (BED)
  - Attention Deficit Hyperactivity Disorder (ADHD)
- Limitations:
  - Not indicated for weight loss; safety and effectiveness for the treatment of obesity have not been established
- Drug Enforcement Agency Schedule II drug
- Dosing for BED: Once in the morning with or without food. Avoid afternoon doses. Capsule may be opened and mixed with yogurt, water, or orange juice (see drug interactions).
  - Starting dose = 30 mg every morning for one week
  - Titration dose = 50 mg every morning for one week
  - Top dose = 70 mg every morning
  - Recommended dose = 50-70 mg every morning
  - GFR 15-<30: Max dose 50 mg daily
  - GFR <15: Max dose 30 mg daily

Most Common Adverse Reactions:

- Anorexia
- Anxiety
- Decreased weight & appetite
- Diarrhea
- Dizziness
- Dry mouth
- Irritability
- Insomnia
- Nausea / Vomiting
- Upper abdominal pain
- Increased heart rate
- Constipation
- Feeling jittery

Lisdexamfetamine Dimesylate

Contra-Indications

- Risk of abuse should be assessed prior to prescribing. Patients should be monitored for signs of abuse while on therapy and it should be discontinued if concerns of abuse arise
- Known hypersensitivity (e.g., anaphylactic reactions, Stevens-Johnson Syndrome, angioedema, and urticarial) to amphetamine products or other ingredients in lisdexamfetamine dimesylate
- Use with monoamine oxidase (MAO) inhibitor or within 14 days of the last MAO inhibitor dose

Warnings

- Serious cardiovascular reactions
  - Due to reports of sudden death in children and adolescents with serious heart problems, as well as sudden death, stroke, and myocardial infarction in adults, avoid use in patients with known structural cardiac abnormalities, cardiomyopathy, serious health arrhythmia, or coronary artery disease.
- Blood pressure or heart rate increases
  - Blood pressure and pulse should be monitored. Benefits and risks should be considered before use in patients for whom blood pressure increases may be problematic.
- Psychiatric adverse reactions
  - May cause psychotic or manic symptoms in patients with no prior history or exacerbation of symptoms in patients with pre-existing psychosis. Evaluate for bipolar disorder prior to stimulant use.
- Suppression of growth
  - Height and weight should be monitored in pediatric patients during treatment.
- Peripheral vasculopathy, including Raynaud's phenomenon
  - Stimulants are associated with peripheral vasculopathy, including Raynaud's phenomenon. Careful observations for digital changes is necessary during treatment with stimulants.
- Risk of serotonin syndrome when combined with other serotonergic medications

Eating Disorders and Obesity: Bulimia Nervosa

Diagnosis

- Cycle of recurrent binge eating and compensatory purging, laxative abuse, diuretic abuse, extra exercising, fasting, or strict food restriction
- Occurs in approximately 3% of adults (mostly women), and reportedly higher (as much as 10%) among college-aged women
- Signs and physical findings:
  - Russell sign: Calluses and abrasions on dorsum of the hands caused by repeated contact with the teeth during self-induced vomiting
  - Enamel erosion of the teeth (usually lingual surface)
  - Sialadenosis (enlargement of the salivary gland, such as the parotid gland)
- Laboratory:
  - Hypokalemia (promoted by hypomagnesemia), hypochloremia, metabolic alkalosis
  - Elevated amylase suggests possible vomiting and salivary gland irritation

Screening

- Screen for Disordered Eating (SDE), Eating Disorders Screen for Primary Care (EDSPC), Eating Disorder Inventory (EDI) & Eating Attitudes Test (EAT)
- Sick (vomiting), Control (loss of control), One Stone (loss of ~ 15 pounds in 3 months), Fat (disturbance in body fat image), Food (obsession with eating behavior) = SCOFF

Treatment

- Cognitive behavior therapy, possibly in combination with drug treatment
- Fluoxetine is an FDA-approved pharmacotherapy for bulimia nervosa
- Although not FDA-indicated for this use, topiramate and naltrexone may be efficacious in treating bulimia nervosa

Eating Disorders and Obesity: Night-Eating Syndrome (NES)

Diagnosis

- At least 25% of daily food consumption (often greater than 50%) consumed after evening meal
- Recurrent awakenings from sleep that require eating to go back to sleep, often involving carbohydrate-rich snacks
- Little interest in breakfast (morning anorexia)
- NES may occur in as much as 5% of the U.S. population
- Awareness of evening or nocturnal ingestions help differentiate NES from sleep-related eating disorder (SRED). SRED is a parasomnia (undesired events accompanying sleep) with "sleep-walking" resulting in repeated episodes of compulsive binge eating and drinking after waking up at night. SRED usually occurs while partially awake and often with no memory of the event afterward

Treatment

- Behavioral therapy regarding nutritional timing and content
- While not an indicated use, selective serotonin re-uptake inhibitors (e.g., sertraline), or anti-migraine/seizure topiramate may be useful for NES

Why Don't People Engage in Routine Physical Activity?

Physiologic

- Musculoskeletal, neurologic, pulmonary, cardiac, and other health disorders
- Pain or soreness
- Fatigue
- Conveniences which limit the physiologic need for physical activity
  - Automated transportation (i.e., cars, buses, etc.)
  - Elevators and escalators
  - Online shopping
  - Automated equipment that lessens manual labor

Lack of Time

- Work commitments
- Family responsibilities
- Time preferentially allotted for other entertainments with minimal energy expenditure (screen time)
  - Television
  - Movies
  - Video games
  - Internet surfing, email, texting, apps
  - Watching sports

Why Don't People Engage in Routine Physical Activity?

Disinterest

- "Exercise is boring"
- Past failures to achieve exercise goals
- Past failures in observing body changes
- Concerns of being seen:
  - In workout clothes
  - In gyms surrounded by others more fit
- Desire to avoid perspiration
  - General appearance
  - Hair
  - Odor

Environment

- Lack of:
  - Others engaged in physical activity (family, friends, etc.)
  - Safe environment
  - Parks or other areas for leisure activity
  - Accessible gym
  - Workplace exercise equipment
- Inadequate maintenance of increased physical activity, once started
- Insufficient education on physical activity
  - Benefits
  - Risks
  - Techniques
  - Recommendations

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Why Do People Plateau with Weight Reduction or Regain Body Weight?

Physiologic Priority Imbalance

- Neuro-biologic processes strongly resist under-nutrition (starvation)
- Neuro-biologic processes weakly resist over-nutrition
- Analogous example:
  - Hypoglycemia can be profoundly symptomatic and may promote physiologic and behavioral priority for immediate caloric intake
  - Hyperglycemia is often asymptomatic and rarely promotes physiologic and behavioral priority for immediate reduced caloric intake

Neurobiology

- Weight loss may decrease neuroendocrine factors, which in turn may increase hunger
  - Leptin
  - Insulin
  - Cholecystokinin
  - Peptide YY
- Weight loss may increase ghrelin, which in turn may increase hunger
- To the extent that within the central nervous system, insulin and leptin "resistance" limits hunger reduction and negative caloric balance, an increase in physical activity may increase the brain's sensitivity to insulin and leptin
- A lack of maintaining routine physical activity after weight loss may contribute to body fat regain

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Why Do People Plateau with Weight Reduction or Regain Body Weight?

Energy Expenditure (dynamic energy balance)

- Decrease in resting energy expenditure with weight loss due to loss of body tissue
- Adaptive thermogenesis wherein reduction in resting metabolic rate exceeds that predicted by loss of body tissue
- Greater muscle efficiency occurs with weight loss, resulting in less energy expenditure with physical activity

Behavior

- Commitment amnesia
  - Forgetfulness of the degree of change and effort required to achieve initial weight loss success
  - Lack of maintaining accountability logs
- Altered priorities
  - Intervening stress
  - Changing life circumstances
  - Changing health status
- Setpoint fallacy
  - The mistaken belief that once achieved, maintenance of weight loss will persist, irrespective of behavior, nutrition, and physical activity
  - "I know if I could just get the weight off, I could keep it off"
- Priority fatigue
  - Lack of maintaining healthy body weight priorities
  - Resorting to previous nutritional and/or physical activity habits after achieving initial weight-loss success
- Decision fatigue
  - Mental stress or multiple higher priority decision-making may impair self-regulation regarding health, and may facilitate choosing unhealthful, immediately rewarding and immediately available ultra-processed foods over more healthful, delayed-gratification unprocessed foods

Behavior Therapy Techniques: Elements for Optimal Success

Doable

- Practical
- Accessible
  - Frequency
  - Consistency

Efficacious

- Evidence-based

Measurable / Accountable

- Trackable
- Verifiable
- Record keeping
- Feedback (positive & negative reinforcement)
- Supervised
- Social support

Self-ownership

- Autonomous stakeholder
- Personal stakeholder

Behavior Therapy: Encounters and Education

Frequent Encounters with Medical Professional or Other Resources Free from Provider Bias

- Clinician (e.g., Physician, Nurse Practitioner, Physician Assistant)
- Dietitian
- Nurse educator
- Physical activity professional trainer (i.e., trainer, physiologist, etc.)
- Mental-health professional
- Certified health coach
- Web-based programs
- Mobile access (i.e., text messages, applications, etc.)
- Multidisciplinary approach
  - Clinicians with professional expertise
  - Patient with self expertise

Education

- Obesity as a disease ("sick fat" and "fat mass" disease)
- Medical health
- Mental health & stress management
- Nutrition
- Physical activity
- Establish healthful sleep habits
- Establish healthful eating habits (i.e., reduce speed of eating, drink water between meals, choose and have available healthful snacks, etc.)
- Recognize and anticipate inevitable weight-loss plateaus

Behavior Therapy: Stimulus Control and Cognitive Restructuring

Stimulus Control

- Eating patterns that maximize satiety such as meal timing, nutrient composition (high fiber, moderately high protein, moderately low glycemic load, higher volume), and appetite awareness training
- Avoid eating for reasons other than hunger
- Avoid frequent snacking
- Avoid binge eating
- Utilize portion control
- Environmental removal of foods identified as especially tempting for the individual patient
- Being habitually mindful of eating stimuli may allow best chance for stimulus control

Cognitive Restructuring

- Address matters of body image
- Identify and establish a plan to counteract unhelpful or dysfunctional thinking leading to unhealthful behaviors and actions
- Emphasize rationale of aggressive yet realistic weight-reduction expectations through an emphasis on weight-reduction as a matter of medical and mental health
- Encourage patient to:
  - Acknowledge he/she is capable of positive thoughts and behaviors
  - Replace unhelpful thoughts and behaviors with more productive ones
  - Practice behavior therapy skills between clinician encounters

Behavior Therapy: Goal Setting and Self-Monitoring

Goal Setting

- Patients are given step-by-step instructions to accomplish goals (i.e., nutrition and physical activity prescriptions)
- SMART
  - **S**pecific
  - **M**easurable
  - **A**ssignable
  - **R**ealistic
  - **T**ime-related

Non-scale victories: Celebrate goal attainments beyond body weight and other number-focused metrics. Acknowledge:

- Better able to focus on things other than food
- Better able to engage in physical activity of daily living (e.g., walking up stairs)
- Better sleep quality
- Better able to fit into clothes previously worn before weight gain
- Reduction in medications

Self Monitoring

- The frequency of self-monitoring is significantly related to weight loss
- Daily or weekly body weights
- Other routine self-anthropometric measurements (i.e., calipers or other home testing for percent body fat, tape measure for waist circumference, MyoTape for muscle mass, etc.)
- Food diaries (including online services or mobile applications)
- Physical activity logs and pedometer/accelerometer measures
- Sleep monitoring
- Photo journaling

Behavior Therapy: Behavioral Contracting and Problem Solving

Behavioral Contracting (Non-food Related Rewards)

- Tokens of reward
- Financial incentives

Problem Solving, Social Support, and Other Reinforcement Contingencies

- Stress management
- Establish alternative back-up procedures to engage during times that challenge adherence to agreed upon plans (e.g., stressful periods, life changes, etc.)
- Health care team support
- Mental-health professional
- Faith-based interventions
- Other group or social support
- Commercial weight loss/maintenance programs
- Encourage interactions with others that may provide positive recognitions for successes

Concomitant Medications

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Top 10 Takeaway Messages: Obesity and Concomitant Medications

1. Anti-hypertensive medications most associated with body weight gain include some beta-blockers (propranolol, atenolol, and metoprolol) and calcium channel blockers (mainly through edema = nifedipine and amlodipine)
2. Anti-diabetes medications that most promote body weight gain include most insulins, sulfonylureas, thiazolidinediones, and meglitinides
3. Hormone therapies that most promote body weight gain include glucocorticoids and injectable progestins
4. Anti-seizure medications most associated with body weight gain include carbamazepine, gabapentin, valproate, and pregabalin
5. Antidepressants most associated with body weight gain include some tricyclic antidepressants (amitriptyline, doxepin, imipramine), some selective serotonin reuptake inhibitors (paroxetine), some selective serotonin and norepinephrine reuptake inhibitors (venlafaxine), some irreversible monoamine oxidase inhibitors (isocarboxazid, phenelzine), as well as mirtazapine, brexpiprazole, and trazodone
6. Mood stabilizers most associated with body weight gain include gabapentin, divalproex, lithium, valproate, vigabatrin, cariprazine, carbamazepine
7. Migraine medications most associated with body weight gain include amitriptyline, gabapentin, paroxetine, valproic acid, and some beta blockers
8. Among antipsychotics most associated with body weight gain include clozapine, olanzapine, chlorpromazine, brexpiprazole, iloperidone, lithium, quetiapine, risperidone, thioridazine, and zotepine.
9. Chemotherapeutic and anti-inflammatory agents most associated with body weight gain include tamoxifen, cyclophosphamide, methotrexate, 5-fluorouracil, aromatase inhibitors, and corticosteroids
10. Other drugs associated with body weight gain include the hypnotic diphenhydramine, some anti-seizure & antidepressants used for treatment of neuropathy, and some highly active antiretroviral therapies (HAART) protease inhibitors when not accompanied by lipodystrophy

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Concomitant Pharmacotherapy: Limitations in the Reported Effects on Body Weight

The Reported Weight Effects of Concomitant Non-Anti-Obesity Medications should be Interpreted with Caution, because the Data Describing these Weight Effects:

- Are mostly derived from observations of studies not specifically designed to evaluate the effects of these medications on body weight
- Are mostly derived from comparisons of the reported effects from different studies, rather than derived from a direct head-to-head comparison within the same controlled clinical trial
- Studies often report variable weight effects, depending on the condition being treated (e.g., psychiatric medications being used for multiple psychiatric conditions), dataset, analysis [head-to-head versus meta-analysis, and specific agents having especially inconsistent reported effects (e.g., haloperidol, sertraline)]
- The reported effect is mostly expressed as mean values, with the potential for wide variances in individual weight responses to a particular drug

Concomitant Pharmacotherapy that Might Alter Body Weight: CVD and DM Medications

Cardiovascular Medications

May Increase Body Weight:

- Some beta-blockers
  - Propranolol may increase body weight
  - Atenolol may increase body weight
  - Metoprolol may increase body weight
  - Carvedilol may not increase body weight
- Older and/or less lipophilic dihydropyridine ("dipine") calcium channel blockers may increase body weight due to edema possibly because they are more vasodilatory, compared to non-dihydropyridines and lipophilic dihydropyridines. The increased edema may exacerbate obesity-related edema (and sleep apnea related peripheral edema), and also confound estimates of body fat
  - Nifedipine
  - Amlodipine

Diabetes Mellitus Medications

May Increase Body Weight:

- Most insulins
- Sulfonylureas and Meglitinides (e.g., nateglinide, repaglinide)
- Thiazolidinediones ("-zones")

May Decrease Body Weight:

- Metformin
- Glucagon-like peptide-1 (GLP-1) receptor agonists (e.g., "-tides")
- Dual GLP-1 and glucose-dependent insulinotropic (GIP) agonist
- Sodium glucose co-transporter 2 inhibitors (e.g., "-flozins")
- Alpha glucosidase inhibitors (e.g., acarbose, miglitol)
- Amylin mimetic (pramlintide)

Neutral Effects on Body Weight:

- Dipeptidyl peptidase-4 (DPP4) inhibitors (e.g., "-gliptins")

Metformin

- May help improve adiposopathic disorders:
  - Insulin resistance
  - Polycystic ovary syndrome
  - Cardiovascular disease (especially when compared to sulfonylurea)
- May help treat complications of concurrent drug treatments:
  - Antipsychotic-related weight gain
  - Human immunodeficiency virus (HIV) protease inhibitor-associated abnormalities (i.e., HIV lipodystrophy)
- May help reduce the overall cancer rate and help improve the treatment of multiple cancers:
  - Colon
  - Ovary
  - Lung
  - Breast
  - Prostate
- May improve insulin sensitivity and reduce hunger via multifactorial effects such as enhancing the effects of gastrointestinal hormones applicable to weight loss (e.g., increased glucagon-like peptide-1 levels and receptors, increased peptide YY, decreased neuropeptide Y) all which may facilitate long-term weight loss

Concomitant Pharmacotherapy that Might Alter Body Weight: Hormones & Seizures

Hormones

May Increase Body Weight:
- Glucocorticoids

Variable Effects on Body Weight:
- Progestin contraceptives
  - Injectable or implantable progestins may have greatest risk for weight gain
- Testosterone
  - May reduce percent body fat and increase lean body mass, especially if used to replace testosterone deficiency in men

Neutral Effects on Body Weight:
- Combined oral contraceptives
- Progestin intrauterine devices

Anti-seizure Medications

May Increase Body Weight:
- Carbamazepine
- Gabapentin
- Valproate
- Pregabalin

May Decrease Body Weight:
- Topiramate
- Zonisamide

Concomitant Pharmacotherapy that Might Alter Body Weight: Antidepressants

May Increase Body Weight (Reports on body weight not always consistent):

- Some tricyclic antidepressants (tertiary amines)
  - Amitriptyline
  - Doxepin
  - Imipramine
  - Dosulepin
- Some selective serotonin reuptake inhibitors (e.g. paroxetine, citalopram)
- Some selective serotonin and norepinephrine re-uptake inhibitors (e.g., venlafaxine)
- Some irreversible monoamine oxidase inhibitors (e.g., isocarboxazid, phenelzine)
- Trazodone
- Mirtazapine
- Brexpiprazole

May Decrease Body Weight:

- Bupropion
- Fluoxetine (variable)

Variable Effects on Body Weight:

- Some tricyclic antidepressants (secondary amines)
  - Desipramine
  - Nortriptyline
  - Protriptyline
- Some selective serotonin reuptake inhibitors
  - Escitalopram
  - Sertraline
- Some serotonin and norepinephrine re-uptake inhibitors
  - Desvenlafaxine
  - Duloxetine
- Some irreversible monoamine oxidase inhibitors (i.e., tranylcypromine)
- Some other serotonergic agents
  - Vortioxetine

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Concomitant Pharmacotherapy that Might Alter Body Weight: Hormones & Seizures

Mood Stabilizers

May Increase Body Weight:

- Gabapentin
- Divalproex
- Lithium
- Valproate
- Vigabatrin
- Cariprazine
- Carbamazepine

Variable/Neutral Effects on Body Weight:

- Lamotrigine (sometimes reported to decrease body weight)
- Oxcarbazepine

Migraine Medications

May Increase Body Weight:

- Amitriptyline
- Gabapentin
- Paroxetine
- Valproic acid and Divalproex
- Some beta-blockers

May Decrease Body Weight:

- Topiramate
- Zonisamide

Neutral Effects on Body Weight:

- Non-steroidal anti-inflammatory drugs (NSAIDs)
- Dihydroergotamine
- Triptans (e.g., sumatriptan, rizatriptan, zolmitriptan)
- 5-HT 1F receptor agonist (i.e., Lasmiditan)
- Calcitonin gene-related peptide receptor (CGRP) receptor antagonists (e.g., ubrogepant, rimegepant, galcanezumab, atogepant)

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Concomitant Pharmacotherapy that Might Alter Body Weight: Antipsychotics & Hypnotics

Antipsychotics

Most Consistently Increase Body Weight:

- Clozapine
- Olanzapine
- Chlorpromazine
- Brexpiprazole
- Iloperidone
- Lithium
- Quetiapine
- Risperidone
- Thioridazine
- Zotepine (not available in US)

Neutral/Variable Effects on Body Weight:

- Amisulpride
- Aripiprazole
- Asenapine
- Cariprazine
- Haloperidol
- Loxapine
- Lurasidone
- Ziprasidone
- Paliperidone
- Perphenazine

Hypnotics

May Increase Body Weight:

- Diphenhydramine
- Zolpidem (may increase risk of sleep-related eating disorder)

May Have Limited Effects on Body Weight:

- Benzodiazepines
- Melatonergic hypnotics
- Trazodone

Podcast:
Anti-psychotic agents
Bill McCarthy MD

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Concomitant Pharmacotherapy that Might Alter Body Weight: Neuropathy & Pain Treatments

Pain Relievers:

- Nonsteroidal anti-inflammatory drugs (NSAIDs) and acetaminophen = May not lead to weight change (unless edema occurs due to NSAID-induced kidney damage)
- Opioids = New persistent opioid users may lose less weight after bariatric surgery

Neuropathy:

- **May Increase Body Weight**
  - Gabapentin
  - Pregabalin
  - Amitriptyline
  - Doxepin
  - Duloxetine
  - Venlafaxine
- **Neutral Effect on Body Weight**
  - Nortriptyline
  - Topical capsaicin
  - Topical lidocaine

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Identify and Manage Concomitant Pharmacotherapy That Might Alter Body Weight

Human Immunodeficiency Virus (HIV) Medications

May Increase Body Weight:

- Some highly active antiretroviral therapies (HAART) protease inhibitors without HIV-associated lipodystrophy

May Alter Body Composition:

- May increase abdominal and visceral fat
- Some highly active antiretroviral therapies (HAART) protease inhibitors with HIV-associated lipodystrophy

Chemotherapies and Anti-Inflammatory Agents

May Increase Body Weight:

- Tamoxifen
- Cyclophosphamide
- Methotrexate
- 5-fluorouracil
- Aromatase inhibitors
- Tumor necrosis factor alpha inhibitors
- Corticosteroids

May Decrease Body Weight:

- Apremilast

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Identify and Manage Concomitant Pharmacotherapy That Might Alter Body Weight

Organ Transplant Medications

- **Corticosteroids (e.g., prednisone)**
  - Corticosteroids may increase body weight (as well as increase blood sugar, blood pressure, and blood lipids)
  - Rapid discontinuation of prednisone (prior to discharge after hospitalization for transplant) may have improved survival, without much change in body weight compared to patients with maintenance prednisone.
- **Calcineurin inhibitors (cyclosporin, tacrolimus)**
  - Calcineurin inhibitors may increase body weight as well as components of the metabolic syndrome
- **Mammalian target of rapamycin (mTOR) inhibitors (sirolimus, everolimus, temsirolimus)**
  - mTOR inhibitors may increase body weight, as well as contribute to the components of the metabolic syndrome

Obesity & Organ Transplantations

- Obesity is the most common promoter metabolic disorders leading to organ failure and post-transplant medications may additionally contribute to metabolic disorders
- Obesity increases the difficulty, time, and rate of complications of surgical procedures. Patients with obesity undergoing organ transplant may benefit from pre-transplant weight management interventions with continued weight management interventions post-transplant
- Increased caloric intake after organ transplant is often related to post transplant improvement in health, symptoms, and quality of life. Steroid avoidance alone does not mitigate increase in body weight after organ transplant.

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Anti-obesity Medications

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Top 10 Takeaway Messages: Anti-obesity Medications

1. Phentermine is a sympathomimetic amine with possible adrenergic side effects and contraindicated in patients with cardiovascular disease
2. Phentermine hydrochloride (HCl) 8 – 37.5 mg prescribed in the U.S. is generally equivalent to 6.4 – 30 mg of phentermine resin marketed outside the US
3. Although not consistent with the prescribing information indicated use, phentermine administration for longer than 12 weeks is supported by clinical data and opinion leaders
4. Orlistat is a gastrointestinal lipase inhibitor with possible adverse experiences that include oily rectal discharge and flatus; it is contraindicated in patients with chronic malabsorption syndrome and cholestasis
5. Liraglutide is a glucagon-like peptide-1 receptor agonist (GLP-1 RA) approved at 1.8 mg per day for treatment of type 2 diabetes mellitus, and at 3.0 mg per day for treatment of obesity with possible gastrointestinal side effects; it is contraindicated in patients with personal or family history of medullary thyroid cancer or Type 2 Multiple Endocrine Neoplasia syndrome
6. Semaglutide is an injectable GLP-1 RA approved at 2.0 mg weekly for treatment of type 2 diabetes mellitus and at 2.4 mg weekly for treatment of obesity. It has similar side effects and contraindications as Liraglutide.
7. Naltrexone/bupropion is a combination of an opioid antagonist and antidepressant, with possible gastrointestinal side effects; it is contraindicated in patients with uncontrolled hypertension, chronic opioid use, seizure disorders, and abrupt discontinuation of alcohol, benzodiazepines, barbiturates and antiepileptic drugs
8. Phentermine/topiramate is a combination of a sympathomimetic amine and anti seizure/migraine medication with side effects that include paresthesias, dysgeusia; it is contraindicated in women who may become pregnant
9. GLP-1 RAs and phentermine/topiramate can be taken with or without meals
10. Orlistat should be taken three times a day with each meal that contains fat; bupropion/naltrexone should not be taken with high fat meals due to increased absorption

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Anti-obesity Medications

Adjunct to Nutritional, Physical Activity, and Behavioral Therapies

Objectives:

- Treat disease
  - Adiposopathy or sick fat disease (SFD)
  - Fat mass disease (FMD)
- Facilitate management of eating behavior
- Slow progression of weight gain/regain
- Improve the health, quality of life, and body weight of the patient with overweight or obesity
- May be an effective adjunct to bariatric surgery in enhancing weight reduction or preventing weight regain

5-10 Percent Weight Reduction May Improve Both Metabolic and Fat Mass Disease

Pharmacokinetics and Obesity

Obesity May Affect a Drug's:

- Absorption
  - Obesity may decrease SQ absorption due to reduced subcutaneous blood flow per unit volume of SQ tissue; high volume of subcutaneous drug (e.g., insulin) may decrease its absorption per unit of drug
  - Intramuscular administration may require longer needles with obesity; however, intramuscular injections generally have faster absorption than subcutaneous administration in patients with obesity
- Metabolism
- Distribution
  - In general, obesity does not seem to consistently affect protein binding
  - Often times, no systematic relationship exists between the degree of lipophilicity of markedly lipophilic drugs and their distribution in individuals with obesity
- Excretion

Pharmacotherapy Absorption and Obesity

- Liraglutide and Semaglutide = injectable (with or without meals)
- Phentermine = capsule or tablet (with or without food recommendations vary, depending on formulation)
- Bupropion/naltrexone = tablet (should not be taken with high fat meal due to increased absorption)
- Phentermine/topiramate = capsule (with or without food)
- Orlistat = capsule (should be taken three times a day with fat containing meal)

Pharmacotherapy Excretion and Obesity

- Measurement of glomerular filtration rate (GFR) represents the blood filtered by the kidney, often assessed by fructose-based polysaccharide (i.e., inulin) that is neither secreted nor reabsorbed across tubules
- Some estimates of GFR are not validated for obesity; patients with obesity often have increased GFR
- Complications of obesity may affect excretion (renal insufficiency due to diabetes mellitus or hypertension)
- Salazar-Corcoran is specific for obesity, but not commonly used
- GFR equations most often used include:
    - **Chronic Kidney Disease Epidemiology Collaboration (CKD-EPI)** equation is frequently used by commercial labs, and the formula includes age, gender, race, and blood creatinine (not weight)
    - **Abbreviated Modification of Diet in Renal Disease (MDRD)** equation is frequently used in clinical trials, may be less accurate at higher GFR, and the formula includes age, gender, race and blood creatinine (not weight)
    - **Cockcroft-Gault** equation is rarely used in clinical practice, but is the historic standard used in for renally-adjusted drug dosing studies, especially when the formula of blood creatinine, age, and gender is adjusted for body weight

Excretion and Obesity: Kidney Stones

- Obesity alone is a risk factor for kidney stones
  - Insulin resistance alters renal acid-base metabolism, lowers urine pH, and increases risk of uric acid stones
  - Increased nutritional intake of high-oxalate foods, salt, processed foods and animal proteins increases risk of kidney stones
- Orlistat increases unabsorbed intestinal fat which binds to intestinal calcium, with less calcium available to bind to intestinal oxalate This allows for increased intestinal oxalate absorption that may ultimately cause hyperoxaluria, promoting calcium oxalate stones
- The topiramate component of phentermine HCl / topiramate ER decreases renal tubule reabsorption of HCO_3 and decreased excretion of H, contributing to metabolic acidosis, and increased urinary pH, which may increase risk of calcium phosphate stones
- If associated with dehydration, then very low-calorie diets may increase the risk of kidney stones
- Gastric bypass bariatric surgery may contribute to fat malabsorption, increased urinary oxalate absorption and urinary excretion, decreased urinary volume, and increased risk of calcium oxalate stones

Pharmacotherapy Excretion and Obesity: Drug Testing for Amphetamines

The Following may Test Positive for Amphetamines on Drug Screens:

- **Amphetamine Derivatives** (Including Lisdexamfetamine)
- **Phentermine**
- **Bupropion**
- **Fluoxetine**
- **Metformin**

Food and Drug Administration (FDA) Principles

FDA-approved Anti-obesity Medication Indications:

- Patients with obesity (e.g., BMI ≥ 30kg/m^2)*
- Patients with overweight (e.g., BMI ≥ 27kg/m^2) with presence of increased adiposopathic complications (e.g., type 2 diabetes mellitus, hypertension, dyslipidemia)*
- Anti-obesity medications are contraindicated in patients hypersensitive to the drugs

Other Principles

- Anti-obesity medications promote variable weight reduction over variable duration in patients with overweight or obesity
- Patients have an average of 5 – 10% weight reduction, with greater weight reduction in hyper-responders, and less than 5% weight loss (or even weight gain) in hypo-responders
- If no clinical improvement (e.g., at least 3 - 5% loss of baseline body weight) after 12-16 weeks with one anti-obesity medication, then consider alternative anti-obesity medication or increasing anti-obesity medication dose (if applicable)**

**While body mass index (BMI) is the only measure listed in the prescribing information for anti-obesity medications, BMI has limitations. Especially in muscular individuals or those with sarcopenia, overweight and obesity are more accurately assessed by other measures.*

***Once anti-obesity drug therapy is initiated with an anti-obesity agent not having a prescribing information time limitation for use, the decision to continue or discontinue anti-obesity drug treatment is best based upon the individual patient response, and clinical judgment regarding the risks of further or recurrent weight gain*

Pregnancy and Lactation Categorization

Update to FDA Pregnancy and Lactation Labeling

- In December 2014, the FDA issued its "Pregnancy and Lactation Labeling Final Rule" (PLLR), which went into effect on June 30, 2015.
- The PLLR removed letter pregnancy categories - A, B, C, D, and X.
- Due to the fact that the prescribing information materials for most anti-obesity medications have yet to be updated to reflect the new rules, the Obesity Algorithm continues to include pregnancy and lactation categories
- **In general, anti-obesity drugs are contraindicated in pregnancy, and best avoided in women who are pregnant, trying to become pregnant or who are breastfeeding**

Anti-Obesity Medication Summary

(All Have Contraindications for Hypersensitivity and Pregnancy)

Drug	Description	Main Side Effects	Illustrative Drug Interactions
Phentermine (and other sympathomimetic amines)	Sympathomimetic amine approved in 1959. It is a DEA Schedule IV stimulant agent approved for short-term use (12 weeks). Some patients may lose about 5% of body weight.	Side effects include headache, high blood pressure, rapid or irregular heart rate, overstimulation, tremor, and insomnia. Should not use with overactive thyroid or uncontrolled high blood pressure or seizure disorder. Contraindicated in patients with history of cardiovascular disease, within 14 days of monoamine oxidase inhibitors, glaucoma, agitated states, drug abuse	During or within 14 days following monoamine oxidase (MAO) inhibitors, sympathomimetics, alcohol, adrenergic neuron blocking drugs, and possibly some anesthetic agents
Orlistat	Gastrointestinal lipase inhibitor that impairs digestion of dietary fat. Lower doses are approved over-the-counter. Some patients may lose about 5% of body weight.	Side effects include oily discharge with flatus from the rectum, especially after fatty foods. (May help with constipation.) May promote gallstones and kidney stones. May cause malabsorption of fat-soluble vitamins (A, D, E, K). Need to take a multivitamin daily. Contraindicated in chronic malabsorption syndrome and cholestasis. Rare cases of severe liver injury and pancreatitis.	Cyclosporine, hormone contraceptives, seizure medications, thyroid hormones, warfarin

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Anti-Obesity Medication Summary

(All have contraindications for hypersensitivity and pregnancy)

Drug	Description	Main Side Effects	Some Drug Interactions
Liraglutide	Glucagon-like peptide-1 receptor agonist that is an injectable drug. At lower doses (1.8 mg per day), liraglutide is indicated to lower blood sugar among patients with type 2 diabetes mellitus. Liraglutide 3.0 mg per day is approved for treatment of obesity. Some patients may lose 5 – 10% of body weight, especially with the liraglutide higher dose.	Adverse reactions include nausea, diarrhea, constipation, vomiting, headache, decreased appetite, dyspepsia, fatigue, dizziness, abdominal pain, increased lipase, and renal insufficiency. Contraindicated with personal or family history or medullary thyroid cancer or Type 2 Multiple Endocrine Neoplasia syndrome. Discontinue with suspected pancreatitis, gall bladder disease, or suicidal behavior and ideation. May promote hypoglycemia, particularly in patients with diabetes mellitus treated with insulin or sulfonylureas.	May slow gastric emptying, which may impact absorption of concomitantly administered oral medication.
Naltrexone / Bupropion	Combination of naltrexone (opioid antagonist used for addictions) and bupropion (used for depression and smoking cessation). Some patients may lose 5 - 10% of body weight.	Naltrexone / bupropion can cause nausea, constipation, headache, vomiting, dizziness, insomnia, dry mouth, diarrhea, and acute closure glaucoma. The bupropion component is an antidepressant, and antidepressants can increase the risk of suicide thinking in children, adolescents, and young adults; monitor for suicidal thoughts and behaviors. Should not be used in patients with uncontrolled high blood pressure, seizure disorders, or drug/alcohol withdrawal.	Opioid pain medications, anti-seizure medications, MAO inhibitors, and possible drug interactions with other drugs.
Phentermine / Topiramate	Combination of phentermine (sympathomimetic amine, anti-obesity drug) and topiramate (used to treat seizures and migraine headaches). DEA Schedule IV drug. Some patients may lose an average of 5 – 10% of body weight.	Can cause paresthesia (tingling or numb feelings to extremities), dizziness, dysgeusia (abnormal taste), insomnia, constipation, or dry mouth. Monitor for increased heart rate, suicidal behavior/ideation, mood and sleep disorders, cognitive impairment, metabolic acidosis, elevated creatinine, and low blood sugars in patients on anti-diabetes medications. Discontinue with acute myopia and secondary angle glaucoma. Should not be used with glaucoma or hyperthyroidism. Topiramate can cause birth defects. Phentermine / topiramate should not be started until a pregnancy test is negative. Thereafter, the FDA recommends women use effective contraception and have monthly pregnancy tests during treatment with phentermine / topiramate.	Should not be taken during or within 14 days of monoamine oxidase inhibitors. Avoid use with alcohol, due to potentiation of depressant effects. May potentiate hypokalemia when used with non-potassium sparing diuretics.
Semaglutide	Glucagon-like peptite-1 receptor agonist that is an injectable drug. Semaglutide is used at doses up to 2.0 mg weekly for type 2 diabetes. Semaglutide 2.4 mg weekly is approved for treatment of obesity. Average weight loss at one year is 16%.	Adverse reactions include nausea, diarrhea, vomiting, constipation, abdominal pain, headache, fatigue, dyspepsia, dizziness, abdominal distension, eructation (belching), flatulence, gastroenteritis, and gastroesophageal reflux disease. Contraindicated in patients with personal or family history of medullary thyroid carcinoma or in patients with Multiple Endocrine Neoplasia syndrome type 2 or known hypersensitivity to semaglutide. **Warnings and precautions:** Acute pancreatitis, acute gallbladder disease, acute kidney injury especially in patients with severe adverse gastrointestinal reactions, diabetes retinopathy, heart rate increase, suicidal behavior and ideations. Associated with hypoglycemia in patients with type 2 diabetes treated with concomitant hypoglycemic medications such as sulfonylureas or insulin.	May slow gastric emptying, which may impact absorption of concomitantly administered oral medication.

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Pharmacotherapy

Anti-obesity /medications Approved for Short-term Use

- Phentermine
- Diethylpropion
- Phendimetrazine
- Benzphetamine

Anti-obesity Medications Approved for Chronic Use

- Phentermine HCl/topiramate extended release
- Naltrexone HCl/bupropion HCl extended release
- Liraglutide
- Orlistat
- Semaglutide

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Sympathomimetic Amines

- Examples: Phentermine, diethylpropion, phendimetrazine, benzphetamine
- Increases satiety
- Drug Enforcement Agency (DEA) Schedule weight-management agents
  - DEA IV for phentermine and diethylpropion
  - DEA III for phendimetrazine and benzphetamine

- Potential adverse experiences include:
  - Palpitation
  - Tachycardia
  - Increased blood pressure
  - Overstimulation
  - Tremor
  - Dizziness
  - Insomnia
  - Dysphoria
  - Headache
  - Dryness of mouth
  - Dysgeusia
  - Diarrhea
  - Constipation
  - Pregnancy category X

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Phentermine

Indications and Use

- Sympathomimetic amine for short-term treatment of obesity (FDA approved for use over a few weeks)
- Drug Enforcement Agency Schedule IV drug
- Dose (In the US, phentermine is almost exclusively available in the HCl formulation – available in 15 mg and 30 mg strength)
- Phentermine HCl = 37.5 mg (or 18.75mg) once per morning; sometimes 18.75 mg twice a day
  - Phentermine HCl (different formulation than above) = 8 mg (or 4 mg) three times a day before meals
  - Phentermine resin = 30 mg (or 15 mg) once per morning

Potential Drug Interactions

- Monoamine Oxidase Inhibitors: Use of phentermine is contraindicated during or within 14 days following the administration of monoamine oxidase inhibitors because of the risk of hypertensive crisis
- Alcohol: Concomitant use of alcohol with phentermine may result in an adverse drug reaction
- Insulin and Oral Hypoglycemic Medications: A reduction in insulin or oral hypoglycemic medications in patients with diabetes mellitus may be required
- Adrenergic Neuron Blocking Drugs: Phentermine may decrease the hypotensive effect of adrenergic neuron blocking drugs

Pharmacokinetics

- Urinary excretion may be 62-85%; use with caution when administering phentermine to patients with renal impairment
- Phentermine hydrochloride (HCl) 8–37.5 mg marketed in the U.S. is **generally equivalent** to 6.4–30 mg of phentermine resin marketed outside the U.S.
- The portion of phentermine in phentermine HCl is often termed “free base,” and is an amount similar found in phentermine resin.
- Complexed drugs (e.g., phentermine ion-exchange resin) often require metabolism by gastric enzymes or intestinal flora to become activated

Phentermine

Most Common Adverse Reactions:

Headache, high blood pressure, rapid or irregular heart rate, overstimulation, tremor, and insomnia.

Contra-indications

- Hypersensitivity & Pregnancy / Nursing
- History of cardiovascular disease (e.g., coronary artery disease, stroke, arrhythmias, congestive heart failure, uncontrolled hypertension)
- Administration during or within 14 days following the administration of monoamine oxidase inhibitors
- Hyperthyroidism
- Glaucoma
- Agitated states
- History of drug abuse

Warnings and Precautions

- According to the Prescribing Information, the safety and efficacy of combination therapy with phentermine and any other drug products for weight loss including prescribed drugs, over-the-counter preparations, and herbal products, or serotonergic agents such as selective serotonin reuptake inhibitors (e.g., fluoxetine, sertraline, fluvoxamine, paroxetine), have not been established. Therefore, coadministration of phentermine and these drug products is not recommended according to the Prescribing Information.
- Primary Pulmonary Hypertension (PPH) – a rare, frequently fatal disease of the lungs – has been reported to occur in patients receiving a combination of phentermine with fenfluramine or dexfenfluramine.
- Valvular Heart Disease - Serious regurgitant cardiac valvular disease, primarily affecting the mitral, aortic and/or tricuspid valves, has been reported in otherwise healthy persons who had taken a combination of phentermine with fenfluramine or dexfenfluramine for weight loss.
- When tolerance to the anorectant effect develops, the recommended dose should not be exceeded in an attempt to increase the effect; rather, the drug should be discontinued.
- Phentermine may impair the ability of the patient to engage in potentially hazardous activities such as operating machinery or driving a motor vehicle
- Phentermine is related chemically and pharmacologically to amphetamine (d- and dll-amphetamine) and other related stimulant drugs that have been extensively abused. The possibility of abuse of phentermine should be kept in mind when evaluating the desirability of including a drug as part of a weight reduction program.
- Use caution in prescribing phentermine for patients with even mild hypertension (risk of increase in blood pressure).

Phentermine

- Although not studied in a prospective, large, randomized, controlled, clinical outcomes trial, the use of phentermine for longer than 12 weeks is supported by other data and opinion leaders – especially in patients at low cardiovascular disease risk
  - Phentermine hydrochloride (HCl) 8–37.5 mg is generally equivalent to 6.4–30 mg of phentermine resin
  - Phentermine HCl is available in the US; phentermine resin can be found outside the US
  - Phentermine HCl formulations ("salts") typically have faster gastrointestinal absorption compared to phentermine resin formulation
  - More rapid gastrointestinal absorption would be expected to have faster onset of action

Orlistat

Indications and Use

- Gastrointestinal lipase inhibitor
- Not a Drug Enforcement Agency Scheduled drug
- Dose = One 120-mg capsule three times a day with each main meal containing fat (during or up to 1 hour after the meal).
- An over-the-counter formulation is available at 60 mg capsule with each meal containing fat

Potential Drug Interactions

- Cyclosporine plasma levels may be reduced when orlistat is co-administered with cyclosporine; cyclosporine should be taken at least 3 hours before or after orlistat in patients taking both drugs
- A 30% reduction in beta-carotene supplement absorption when concomitantly administered with orlistat.
- Orlistat inhibits absorption of a vitamin E acetate supplement by approximately 60%
- Vitamin K levels tended to decline in subjects taking orlistat; therefore, patients on chronic stable doses of warfarin who are prescribed orlistat should be monitored closely for changes in coagulation parameters
- Post-marketing reports of hypothyroidism with orlistat and levothyroxine; levothyroxine and orlistat should be administered at least 4 hours apart

Pharmacokinetics

- Systemic exposure to orlistat is minimal
- It is likely the metabolism of orlistat occurs mainly within the gastrointestinal wall

Orlistat

Most Common Adverse Reactions: Oily discharge from the rectum, flatus with discharge, increased defecation, fecal incontinence

Contra-indications

- Orlistat is contraindicated in patients with chronic malabsorption syndrome or cholestasis, and in patients with known hypersensitivity to orlistat or to any component of this product.

Warnings and Precautions

- Gastrointestinal events may increase when orlistat is taken with a diet high in fat (>30% total daily calories from fat). The daily intake of fat should be distributed over three main meals.
- Patients should be strongly encouraged to take a multivitamin supplement that contains fat-soluble vitamins to ensure adequate nutrition because orlistat has been shown to reduce the absorption of some fat-soluble vitamins and beta-carotene. In addition, the levels of vitamin D and beta-carotene may be low in patients with obesity. Vitamin supplements should be taken once a day at least 2 hours before or after the administration of orlistat, such as at bedtime.
- May increase risk of cholelithiasis
- May increase risk of urinary oxalate and kidney stones
- Rare post-marketing reports of severe liver injury and pancreatitis

Liraglutide*

Indications and Use**

- Liraglutide is a glucagon-like peptide-1 (GLP-1) receptor agonist
- Drug Enforcement Agency Schedule: Not a scheduled drug
- Solution for subcutaneous injection, pre-filled, multi-dose pen that delivers doses of 0.6 mg, 1.2 mg, 1.8 mg, 2.4 mg, or 3.0 mg
- Inject subcutaneously in the abdomen, thigh, or upper arm; the injection site and timing can be changed without dose adjustment
- The lower dose of liraglutide 1.8 mg per day is approved for the treatment of type 2 diabetes mellitus (not type 1 diabetes mellitus or diabetes ketoacidosis). The recommended dose of liraglutide for treatment of obesity is 3.0 mg daily, any time of day, without regard to the timing of meals
- Dosing:
  - Week 1 = 0.6 mg per day
  - Week 2 = 1.2 mg per day
  - Week 3 = 1.8 mg per day
  - Week 4 = 2.4 mg per day
  - Week 5 and onward = 3.0 mg per day
  - Slower dose titration may improve tolerability and gastrointestinal side effects

**Evaluate the change in body weight after 16 weeks and discontinue liraglutide for obesity if the patient has not lost at least 4% of baseline body weight since it is unlikely that the patient will achieve and sustain clinically meaningful weight loss with continued treatment.*

***The liraglutide prescribing information for treatment of type 2 diabetes mellitus (top dose 1.8 mg per day) also has an indication to reduce the risk of major adverse cardiovascular events in adults with type 2 diabetes mellitus and established cardiovascular disease.*

***The liraglutide prescribing information for treatment of obesity (top dose 3.0 mg per day) has an indication as an adjunct to reduced-calorie diet and increased physical activity for chronic weight management in adult patients with an initial body mass index of 30 kg/m² or greater, or 27 kg/m² or greater in the presence of at least one adverse consequence of increased adiposity (e.g., type 2 diabetes mellitus, hypertension, or dyslipidemia). Liraglutide at the 3.0 mg per day dose is not indicated to treat type 2 diabetes mellitus and should not be used with any other GLP-1 agonist. The safety of coadministration with other weight loss products has not been established.*

Liraglutide

Potential Drug Interactions

- Liraglutide delays gastric emptying. This may impact absorption of concomitantly administered oral medications.
- Liraglutide has low potential for pharmacokinetic drug-to-drug interactions related to cytochrome P450 and plasma-protein binding

Pharmacokinetics

- Unlike native GLP-1, liraglutide is stable against metabolic degradation by both neutral endopeptidase and dipeptidyl peptidase IV and has a plasma half-life of 13 hours after subcutaneous administration
- Liraglutide exposures are similar among three subcutaneous injection sites (upper arm, abdomen, and thigh); absolute bioavailability of liraglutide following subcutaneous administration is approximately 55 percent
- Liraglutide is endogenously metabolized similar to large proteins without a specific organ as a major route of elimination
- Following a [3H]-liraglutide dose, intact liraglutide is not detected in urine or feces, with only a minor part excreted as liraglutide-related metabolites in urine or feces (6 percent and 5 percent, respectively)

Liraglutide

Most Common Adverse Reactions

- Nausea
- Hypoglycemia
- Diarrhea
- Constipation
- Vomiting
- Headache
- Decreased appetite
- Dyspepsia
- Fatigue
- Dizziness
- Abdominal pain
- Increased lipase

Contra-indications

- Personal or family history of medullary thyroid carcinoma or Multiple Endocrine Neoplasia syndrome type 2
- Hypersensitivity to liraglutide or any product components
- Pregnancy

Liraglutide

Warnings

- **Prescribing information boxed warning**: Liraglutide causes dose-dependent and treatment-duration-dependent thyroid C-cell tumors at clinically relevant exposures in both genders of rats and mice. It is unknown whether liraglutide causes thyroid C-cell tumors, including medullary thyroid carcinoma (MTC), in humans, as the human relevance of liraglutide-induced rodent thyroid C-cell tumors has not been determined. Liraglutide is contraindicated in patients with a personal or family history of MTC and in patients with Multiple Endocrine Neoplasia syndrome type 2 (MEN 2). Counsel patients regarding the potential risk of MTC with use of liraglutide and inform them of symptoms of thyroid tumors (eg, a mass in the neck, dysphagia, dyspnea, persistent hoarseness). Routine monitoring of serum calcitonin or using thyroid ultrasound is of uncertain value for early detection of MTC in patients treated with liraglutide.
- Discontinue promptly if pancreatitis is suspected; do not restart if pancreatitis is confirmed
- If cholelithiasis or cholecystitis are suspected, gallbladder studies are indicated
- Serious hypoglycemia can occur when liraglutide is used with an insulin secretagogue (i.e., a sulfonylurea)
  - Consider lowering the dose of anti-diabetes drugs to reduce the risk of hypoglycemia
- Monitor heart rate at regular intervals to evaluate for possible heart rate increase
- Renal impairment has been reported post-marketing, usually in association with nausea, vomiting, diarrhea, or dehydration, which may sometimes require hemodialysis
  - Use caution when initiating or escalating doses of liraglutide in patients with renal impairment
- Post-marketing reports exist regarding serious hypersensitivity reactions (e.g., anaphylactic reactions and angioedema)
  - If these occur, then liraglutide and other suspect medications should be discontinued, and the patient instructed to promptly seek medical advice
- Monitor for depression or suicidal thoughts and discontinue liraglutide if symptoms develop

Semaglutide (Wegovy®)

Indication	• BMI ≥ 30 kg/m^2 • BMI ≥ 27 kg/m^2 + at least 1 ORC • Age: 12 years and older with a body weight above 132 pounds (60kg) and obesity
Dosage	• Titrate: 0.25mg SC weekly x 4 then 0.5mg x 4 weeks, 1mg x 4 weeks, 1.6mg x 4 weeks then 2.4mg weekly continuing
Mechanism of action (MOA)	• GLP-1 agonist • Central satiety, ↓gastric emptying
Contraindications	• Pregnancy • Personal or FH medullary thyroid CA • MEN type 2
Precautions	• Acute pancreatitis, acute gallbladder disease • Serious hypoglycemia with insulin secretagogue • HR increase • Renal impairment
Common adverse drug reactions (ADR)	• N/V, dyspepsia, diarrhea, constipation • Headache, dizziness, fatigue • Hypoglycemia, abdominal pain, increased lipase
Weight Loss	• WL -14.9% vs -2.4% Placebo • ≥5% WL – 83.5%; Placebo 31.1% • ≥10% - 66.1%; Placebo 12%

Naltrexone HCl / Bupropion HCl Extended Release

Indications and Use

- Naltrexone is an opioid antagonist
- Bupropion is an aminoketone antidepressant with relatively weak inhibition of neuronal reuptake of norepinephrine and dopamine
- Drug Enforcement Agency Schedule: Not a scheduled drug
- Tablets = 8 mg/90 mg (naltrexone HCl/bupropion HCl extended release)
- Dosing:
  - Week 1 = 1 tablet in AM, no tablets in PM
  - Week 2 = 1 tablet in AM, 1 tablet in PM
  - Week 3 = 2 tablets in AM, 1 tablet in PM
  - Week 4 and beyond = 2 tablets in AM, 2 tablets in PM

Naltrexone HCl / Bupropion HCl Extended Release

Potential Drug Interactions

- Should not be administered with opioids due to naltrexone component, which is an opioid receptor antagonist (all opioids should be discontinued at least 7 days prior to start of naltrexone HCl/Bupropion HCl)
- Monoamine oxidase inhibitors may increase risk of hypertensive reactions when used concomitantly
- Bupropion is metabolized by CYP2B6 into an active metabolite, 4-hydroxybupropion, which may be the major active form
  - CYP2B6 inhibitors or inducers may affect bupropion levels with unclear consequences of its efficacy given simultaneous changes in 4-hydroxybupropion levels
- Bupropion inhibits CYP2D6 which may increase the concentration of drugs metabolized by CYP2D6:
  - Antidepressants, (e.g., selective serotonin reuptake inhibitors and many tricyclics), antipsychotics (e.g., haloperidol, risperidone and thioridazine), beta-blockers (e.g., metoprolol) and Type 1C antiarrhythmics (e.g., propafenone and flecainide)
- Digoxin levels may be decreased
- Drugs that lower seizure threshold should be used with caution
- Dopaminergic Drugs (levodopa and amantadine) can cause central nervous system toxicity
- Naltrexone HCl / Bupropion HCl Extended Release can cause false positive urine test results for amphetamines

Naltrexone HCl / Bupropion HCl Extended Release

Pharmacokinetics

- Both parent and the 6-beta-naltrexol metabolite are active
- Naltrexone and 6-beta-naltrexol are not metabolized by cytochrome P450 enzymes
- Naltrexone and its metabolites are excreted primarily by the kidney
- Bupropion is extensively metabolized
- CYP2B6 is the principal isozyme involved in the formation of hydroxybupropion (bupropion metabolite), whereas cytochrome P450 isozymes are not involved in the formation of the other active metabolites
- Bupropion and its metabolites inhibit CYP2D6
- Following oral administration of 200 mg of 14C-bupropion in humans, 87 percent and 10 percent of the radioactive dose were recovered in the urine and feces, respectively

Naltrexone HCl / Bupropion HCl Extended Release

Most Common Adverse Reactions

- Nausea
- Constipation
- Headache
- Vomiting
- Dizziness
- Insomnia
- Dry mouth
- Diarrhea

Contra-indications

- Uncontrolled hypertension
- Seizure disorders, anorexia nervosa or bulimia, or undergoing abrupt discontinuation of alcohol, benzodiazepines, barbiturates, and antiepileptic drugs
- Use of other products containing bupropion
- Chronic opioid use
- During or within 14 days of taking monoamine oxidase inhibitors
- Known allergy to any of its ingredients
- In August 2020, pregnancy was removed as a contraindication. (https://contrave.com/content/pdf/Contrave_PI.pdf), However, the prescribing information recommends to discontinue naltrexone HCl/Bupropion HCL ER when pregnancy is recognized

Naltrexone HCl / Bupropion HCl Extended Release

Warnings

- Monitor for depression or suicidal thoughts and discontinue naltrexone HCl/bupropion HCl if these symptoms develop
- Patients may experience changes in mood (including depression and mania), psychosis, hallucinations, paranoia, delusions, homicidal ideation, aggression, hostility, agitation, anxiety, and panic, as well as suicidal ideation, suicide attempt, and completed suicide. Patients who develop neuropsychiatric adverse events should discontinue naltrexone HCl/bupropion HCl and contact a healthcare provider.
- Bupropion is used for the treatment of depression. Antidepressant treatment can precipitate a manic, mixed, or hypomanic episode, with apparent increased risk in patients with bipolar disorder, or who have risk factors for bipolar disorder. No activation of mania or hypomania was reported in the clinical trials of naltrexone HCl/bupropion HCl patients for treatment of obesity; however, patients receiving antidepressant medications and patients with a history of bipolar disorder or recent hospitalization because of psychiatric illness were excluded from naltrexone HCl/bupropion HCl clinical trials. Prior to initiating naltrexone HCl/Bupropion Cl ER, patients should be screened for bipolar disorder and bipolar disorder risk factors. Naltrexone HCl/bupropion HCl is not approved for treating bipolar depression.
- Risk of seizure may be minimized by adhering to the recommended dosing schedule and avoiding co-administration with high-fat meals
- Monitor blood pressure and heart rate in all patients, especially those with cardiac or cerebrovascular disease
- Hepatotoxicity: Cases of hepatitis and clinically significant liver dysfunction observed with naltrexone exposure
- Angle-closure glaucoma has occurred in patients with untreated anatomically narrow angles treated with antidepressants
- Weight reduction may cause hypoglycemia in patients treated with anti-diabetes mellitus medications. Glucose levels should be monitored.

Phentermine HCl / Topiramate Extended Release

Completion of Risk Evaluation and Mitigation Strategy (REMS) program to inform prescribers and female patients about the increased risk of congenital malformations (especially orofacial clefts) in infants exposed to phentermine HCl/topiramate extended release during the first trimester of pregnancy*

Indications and Use

- Drug Enforcement Agency Schedule IV drug
- Phentermine is a shorter-acting sympathomimetic amine approved as monotherapy as a weight-management drug
- Topiramate is a longer-acting neurostabilizer approved as monotherapy for seizure disorders and migraine headache prevention
- Doses = Once daily in the morning with or without food
  - Starting dose = 3.75 mg/23 mg (phentermine/topiramate extended release)
  - After 14-day intervals, and as clinically indicated, escalate doses to:
    - Recommended dose = 7.5 mg/46 mg
    - Titration dose = 11.25 mg/69 mg
    - Top dose = 15 mg/92 mg
  - Gradually wean dose from the top dose (15 mg/92 mg) to help avoid potential seizures

**Completion of the FDA-mandated REMS program is optional and not required prior to prescribing phentermine HCl/topiramate extended release. Implementation of a REMS program by clinicians and pharmacies is intended to provide appropriate safety information to females of reproductive potential.*

Phentermine HCl / Topiramate Extended Release

Potential Drug Interactions

- May alter the exposure to oral contraceptives, causing irregular menstrual bleeding but not an increased risk of pregnancy
  - Oral contraceptives should not be discontinued if spotting occurs
- May potentiate central nervous system depressants such as alcohol
  - Patients should avoid concomitant alcohol
- May potentiate hypokalemia of non-potassium-sparing diuretics
- Phentermine is contraindicated during or within 14 days following the administration of monoamine oxidase inhibitors because of the risk of hypertensive crisis

Pharmacokinetics

- Phentermine is metabolized by the liver, with most excreted by the kidney
- Topiramate is excreted mainly by the kidney

Phentermine HCl / Topiramate Extended Release

Most Common Adverse Reactions

- In clinical trials, adverse reactions occurring more than or equal to 5 percent of the time include:
  - Paresthesia
  - Dizziness
  - Dysgeusia (taste distortion/perversion)
  - Insomnia
  - Constipation
  - Dry mouth

Laboratory Abnormalities May Include

- Metabolic acidosis
- Elevated creatinine
- Lowering of glucose levels

Phentermine HCl / Topiramate Extended Release

Contra-indications

- Pregnancy
- Glaucoma
- Hyperthyroidism
- During or within 14 days of taking monoamine oxidase inhibitors
- Hypersensitivity or idiosyncrasy to sympathomimetic amines

Warnings and Precautions

- Pregnancy testing is recommended before initiating phentermine HCl/topiramate ER and monthly during therapy. Women who can become pregnant should be advised of the potential risk to a fetus and to use effective contraception during phentermine HCl/topiramate ER therapy
- Monitor for increased heart rate in all patients (especially patients with cardiovascular disease), depression or suicidal thoughts, acute myopia and secondary angle closure glaucoma, mood and sleep disorders, cognitive impairment (with cautioned initial use in patients operating automobiles or hazardous machinery), metabolic acidosis, elevated creatinine, and possible low blood sugar in patients treated with anti-diabetes medications
- Phentermine HCl / topiramate extended release should be discontinued or considered for discontinuation in patients with increases in adrenergic responses, such as increase in heart rate (especially in those with cardiac and/or cerebrovascular disease), suicidal behavior and ideation, acute myopia and secondary angle-closure glaucoma, unacceptable mood and sleep disorders, cognitive impairment, pregnancy or nursing
- May increase risk of kidney stones and hypokalemia
- May increase the risk of oligohidrosis and hyperthermia

Nonsystemic Oral Hydrogel

Description and Mechanism of Action

- Biodegradable oral non-systemic superabsorbent hydrogel made from cross-linked carboxymethylcellulose and citric acid that promotes fullness and may help to increase satiety to help with weight management
- Each capsule contains thousands of superabsorbent hydrogel particles (0.75 grams per capsule), and each particle is approximately the size of a grain of salt
- The capsules disintegrate in the stomach and release the enclosed hydrogel particles, which can then hydrate up to 100 times their original weight
- When fully hydrated, the individual non-clustering hydrogel particles occupy about a quarter of average stomach volume
- The gel particles mix with ingested foods, creating a larger volume with higher elasticity and viscosity in the stomach and small intestine, promoting satiety and fullness
- The hydrogel particles are partially degraded enzymatically in the colon, releasing most of the absorbed water, and subsequently being excreted in the feces
- Ingested orally similar to drugs, but regulated by the FDA as a class II medical device because it acts through mechanical modes of action

Nonsystemic Oral Hydrogel

Indications and Use

- Indicated to aid in weight management in adults with overweight and/or obesity having a body mass index (BMI) 25 – 40 kg/m^2, when used in conjunction with appropriate nutrition ("diet") and physical activity ("exercise").
- Three capsules (2.25 g/dose) are administered with water 20 – 30 minutes before lunch and dinner
- Each individual pod holds a single dose of three (3) capsules, to be administered with water before lunch and dinner/supper
- Fourteen (14) pods are supplied in a weekly tube
- Patients should follow these steps:
  - Swallow 3 capsules with water
  - After taking the capsules, drink 2 additional glasses of water (8 fl oz/250 mL each)
  - Wait 20-30 minutes to begin the meal
- If a pre-meal dose is missed, the hydrogel capsules should be taken immediately after that meal

Potential Drug Interactions

- The effect of the hydrogel capsule device on all concomitant medications is not known; all medications that are taken once daily should be taken in the morning (fasting or with breakfast) or at bedtime
- If a patient is taking the concomitant medication with meals or close to meals, the prescriber should consider if the risk of incorrect dosing, especially for narrow therapeutic drugs, is outweighed by the potential benefit
- For all medications that should be taken with food, the concomitant medication should be taken after the meal has started
- For patients who take metformin with meals, glycemic control should be monitored after initiation of the hydrogel capsule device to determine if changes are indicated for glucose control, because the hydrogel may have an affect on metformin absorption similar to the effect of concomitant food

Nonsystemic Oral Hydrogel

Pharmacokinetics

- The hydrogel capsule device passes through the digestive system, maintaining its three-dimensional structure in the stomach and small intestine before breaking down in the colon. The water is then released and reabsorbed by the body.
- The hydrogel capsule device particles are eliminated through normal bowel movements (not systemically absorbed)

Most Common Adverse Reactions

- Common side effects are mainly gastrointestinal, including abdominal pain, constipation, flatulence, infrequent bowel movements, abdominal distention, diarrhea, and nausea. Most of these side effects are mild or moderate in intensity, occurring within the first 3 months and resolving in 2 weeks.
- Clinical trials support that the adverse experiences with the hydrogel capsule device are similar to the placebo group

Contra-indications

- Pregnancy
- History of allergic reaction to cellulose, citric acid, sodium stearyl fumarate, gelatin, or titanium oxide

Warnings

- Keep out of reach of children
- May alter the absorption of medications
- Do not use after expiration date printed on the product packaging.

Nonsystemic Oral Hydrogel

Precautions

- Patients should contact a healthcare provider (HCP) immediately if a severe or continued adverse event occurs
- If severe allergic reactions, severe abdominal pain, or severe diarrhea occurs, then patients should discontinue the product and speak with an HCP
- Patients with dysphagia upon swallowing other capsules, are likely to have difficulty swallowing these capsules
- Patients should not consume the hydrogel capsule device if the package is damaged
- Capsules should be discarded if any capsules are broken, crushed, or damaged
- Use with caution in patients with active gastrointestinal conditions such as gastro-esophageal reflux disease (GERD), ulcers, or heartburn
- Avoid using in patients with the following conditions:
    - Esophageal anatomic anomalies, including webs, diverticula, and rings.
    - Suspected strictures (such as patients with Crohn's disease).
    - Complications from prior gastrointestinal surgery that could affect GI transit and motility.
- The hydrogel capsule device is not a food substitute; it is not absorbed by the body and therefore has no nutritional or caloric value
- The hydrogel capsule device should be taken under the direction of an HCP as part of a structured weight loss program. Failure to adhere to prescribed dietary and exercise instructions may result in failure to lose weight

Obesity Medicine Association

Metabolic and Bariatric Surgery

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Top 10 Takeaway Messages: Gastrointestinal (GI) Hormones

1. GI hormones regulate caloric balance, hunger/satiety food digestion, and nutrient utilization via central nervous system signaling, effects on GI motility, and GI enzyme release
2. Common GI hormone action in response to eating include decrease in hunger and facilitative digestion (delayed gastric emptying, digestive enzyme release, and post-absorptive nutrient metabolism)
3. The jejunum is the second longest segment of the small intestine, and absorbs the greatest amount of simple sugars, fatty acids, proteins, minerals and vitamins
4. The ileum is the longest segment of the small intestine, and absorbs bile salts, bile acids, vitamin B12, some vitamins and some minerals
5. After food intake, most GI hormones decrease hunger/increase satiety.
6. Among the few GI hormones that increase hunger between meals are ghrelin ("hunger hormone") and neuropeptide Y; positive caloric balance may not always be hunger-related
7. Illustrative GI hormones produced by the stomach include ghrelin and gastrin
8. Illustrative hormones produced by the pancreas include insulin, glucagon, pancreatic polypeptide, amylin, and somatostatin
9. Illustrative GI hormones produced by the small intestine include cholecystokinin, secretin, motilin, and glucose-dependent insulinotropic peptide (GIP; also known as gastric inhibitory peptide)
10. Illustrative GI hormones produced by the ileum and/or large intestine include fibroblast growth factor 19, glucagon-like peptide-1, glucagon-like peptide-2, oxyntomodulin, and peptide YY

Nutrient Absorption

Stomach (~1 foot)	Duodenum (~1 foot)	Jejunum (~8 foot)	Ileum (~12 foot)	Large Intestine (~5 foot)
• Water • Alcohol	• Fatty acids • Amino acids • Minerals (e.g., calcium during calcium deficiency, iron) • Some vitamins	The jejunum absorbs the greatest amount of: • Simple sugars • Fatty acids • Proteins • Minerals • Vitamins	• Bile salts • Bile acids • Vitamin B12 • Some vitamins • Some minerals	• Water • Sodium chloride • Potassium • Intestinally derived vitamin K

GI Hormone Regulation of Caloric Balance, Food Digestion, and Nutrient Utilization

Before Eating (During Fasting): GI Hormones May Increase Hunger

- Ghrelin
- Neuropeptide Y*

**The biologically active peptide YY (PYY) and pancreatic polypeptide (PP) are expressed by endocrine cells in the digestive system. Animal studies suggest neuropeptide Y (NPY) endocrine cells are found throughout the small and large intestine, with NPY thus being involved in both gut-brain and brain-gut axis (bidirectional signaling). NPY is mainly described as a highly abundant orexigenic peptide found throughout the brain.*

Common Gastrointestinal Hormone Actions in Response to Eating

Hunger

Digestion

- Delayed gastric emptying
- Digestive enzyme release
- Post absorptive nutrient metabolism

Podcast : Gut Hormones
Marisa Censani MD

Postprandial Gastrointestinal Hormone Actions

Hormone After Eating	↓ Hunger ↑ Satiety	↓ Motility ↓ Gastric Emptying	Stimulate Digestive Enzyme Release	Counter-Regulatory Digestive Enzyme Release	Assist in Post-Absorptive Nutrient Management
Glucagon like peptide-1	X	X			
Oxyntomodulin	X	X		X	
Peptide YY	X	X		X	
Cholecystokinin	X	X	X		
Amylin	X	X			
Gastrin			X		
Secretin			X	X	
Somatostatin	X			X	X
Glucagon like peptide-2				X	
Pancreatic polypeptide	X			X	
Insulin	X				X
Glucagon	X				X
Fibroblast growth factor 19	X				X
Motilin	X				

Illustrative Gastrointestinal Hormones and Function

Where Secreted	Gastrointestinal Hormone	Effect of Hormone on Eating Behavior	Effect of Eating on Hormone Secretion	Notes	Effect of Gastric By-pass and Sleeve Gastrectomy
Stomach	Ghrelin (secreted by P/D1 cells)	↑ Hunger	↓	↑ With fasting ↑ Gastric emptying/ GH	Ghrelin likely to decrease with sleeve gastrectomy (not so with gastric bypass)
	Gastrin (secreted by G cells in antrum)	↓ Hunger	↑	↑ HCl acid and pepsinogen	↓ Gastric bypass - / ↑ Sleeve
Pancreas	Insulin (secreted by pancreatic beta cells)	↓ Hunger	↑	↑ Hunger with hypoglycemia ↑ Glucose transporter 4 in adipose tissue/muscle, glycogenesis, lipoprotein lipase activity, lipogenesis	↓ Insulin resistance/fasting insulin ↑ Insulin sensitivity/insulin responsiveness
	Glucagon (secreted by pancreatic alpha cells)	↓ Hunger	↓	↑ Glycogen to glucose ↑ Postprandial glucagon in patients with type 2 diabetes mellitus (glucagon not suppressed)	Variable
	Pancreatic polypeptide (PP) [secreted by PP (F) cells]	↓ Hunger	↑	↓ Pancreatic exocrine secretion	Variable
	Amylin (secreted by pancreatic beta cells)	↓ Hunger	↑	↓ Gastric emptying, glucagon	- / ↓
	Somatostatin (secreted by D cells pylori antrum, duodenum, and pancreatic islets)	↓ Hunger	↑	↓ Growth hormone, gastrin, HCl, secretin, CCK, insulin, glucagon	- / ↑

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1 2 3 4 5 6 7

Illustrative Gastrointestinal Hormones and Function

Where Secreted	Gastrointestinal Hormone	Effect of Hormone on Eating Behavior	Effect of Eating on Hormone Secretion	Notes	Effect of Gastric By-pass and Sleeve Gastrectomy
Small Intestine (Mainly Duodenum and Jejunum)	Cholecystokinin (CCK) (secreted by I-cells)	↓ Hunger	↑	↑ Gall bladder contractility and bile, pancreatic enzymes; ↓ Gastric emptying	↑ Postprandial
	Secretin (secreted by S cells)	-	↑	↑ Pancreatic bicarbonate and bile ↓ Intestinal motility & gastric acid	Variable
	Glucose-dependent insulinotropic peptide (GIP; Also known as Gastric Inhibitory Peptide – secreted by K cells)	-	↑	↑ Insulin ↑ Glucagon postprandial	Variable
	Motilin (secreted by M or Mo cells)	↓ Hunger	↓ (↑ with fasting)	↑ Gastric motility, interdigestive migratory contractions (borborygmi)	?

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Illustrative Gastrointestinal Hormones and Function

Where Secreted	Gastrointestinal Hormone	Effect of Hormone on Eating Behavior	Effect of Eating on Hormone Secretion	Notes	Effect of Gastric By-pass and Sleeve Gastrectomy
Ileum and/or Large Intestine	Fibroblast growth factor (FGF) 19 [FGF 21 is produced by the liver] (secreted by ileal cells and regulated by farnesoid X receptors - FXR)	↓ Hunger	↑	↓ Bile acids, glucose production ↑ Insulin sensitivity, glycogen synthesis	↑
	Glucagon like peptide-1 (secreted by ileum/colon L-cells)	↓ Hunger	↑	↑ Insulin ↓ Glucagon, gastric emptying	↑
	Glucagon like peptide-2 (secreted by ileum/colon L-cells)	↓ Hunger	↑ ↑	↑ Glucose metabolism, intestinal mucosal growth, increases absorptive surface, epithelial brush-border nutrient transporters and digestive enzymes, intestinal blood flow, postprandial chylomicron secretion ↓ Gastrointestinal motility	↑
	Oxyntomodulin (secreted by ileum/colon L-cells)	↓ Hunger	↑	↑ GLP-1 and glucagon receptor activity ↓ Gastric acid and gastric emptying	↑ Gastric bypass - Sleeve gastrectomy
	Peptide YY (secreted by ileum/colon L-cells)	↓ Hunger	↑	↓ Gall bladder and pancreatic secretions, gastric emptying	↑

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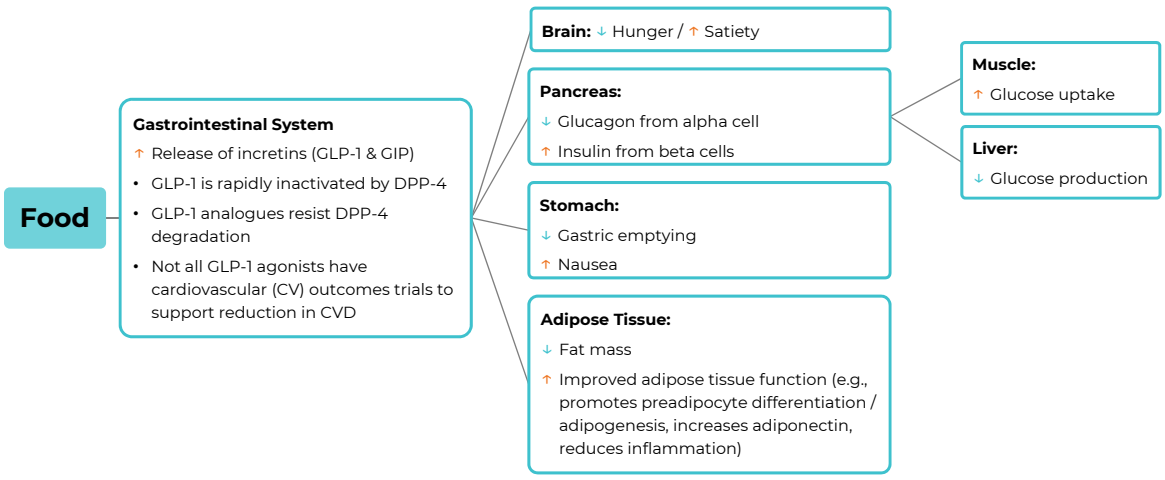
Glucagon-like Peptide-1 (GLP-1)



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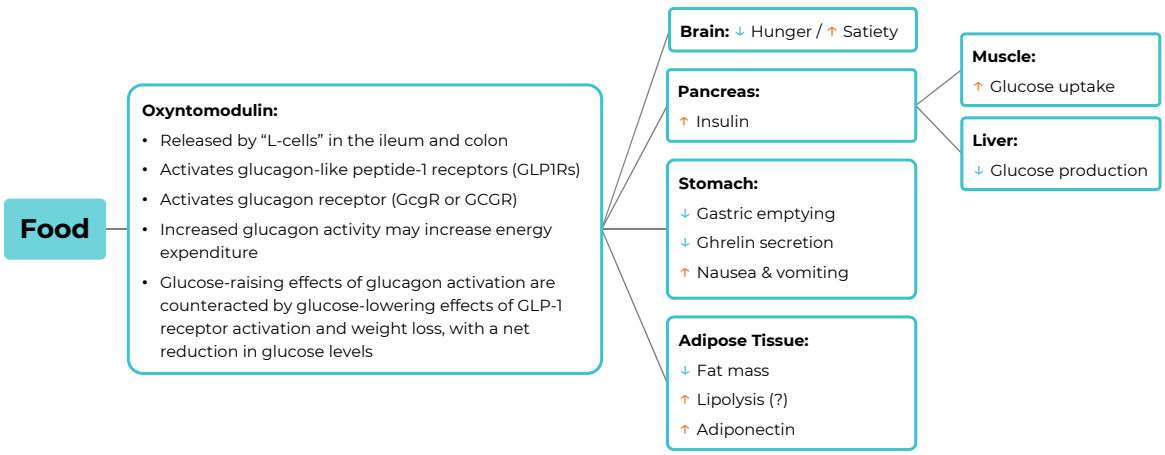
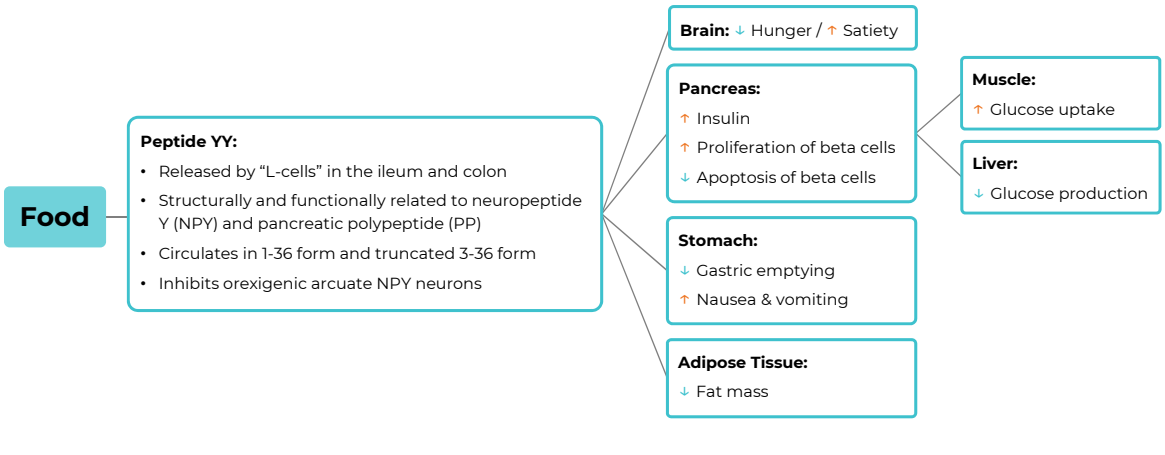
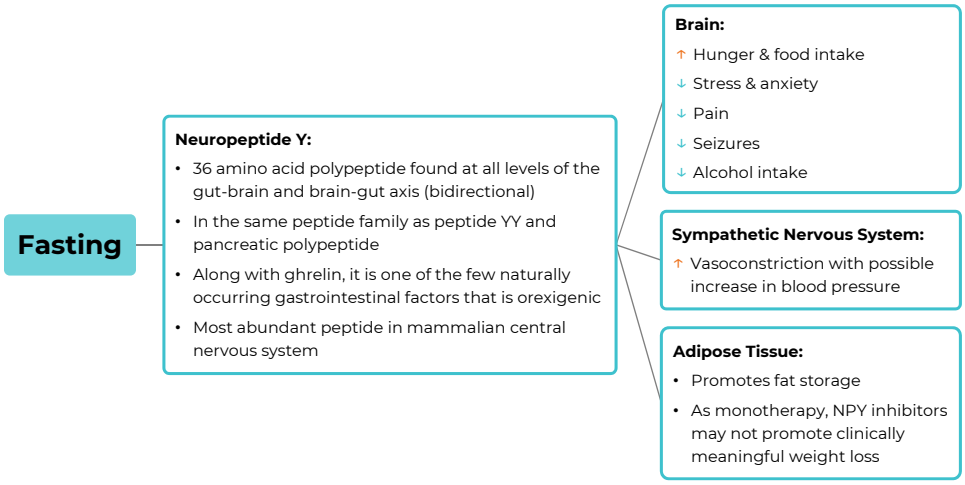
Oxyntomodulin



Peptide YY



Neuropeptide Y (NPY)



Top 10 Takeaway Messages: Bariatric Surgery

1. The two most common bariatric surgical procedures are Roux-en-Y gastric bypass and vertical sleeve gastrectomy (often performed laparoscopically), which provide clinically meaningful improvement in metabolic diseases such as type 2 diabetes mellitus
2. Gastric bypass involves dividing the stomach into a small proximal gastric pouch (leaving a large "bypassed" gastric remnant in situ) attached to a "roux" limb of small bowel jejunum, bypassing the larger gastric remnant, all of the duodenum, and a portion of the proximal small intestine
3. Acute complications of gastric bypass include leaks or perforations potentially leading to peritonitis with severe abdominal pain, fever, tachycardia, and leukocytosis; imaging may include soluble contrast for abdominal CT or upper GI study (not barium); treatment is immediate surgical exploration
4. Chronic complications of gastric bypass include gastro-gastric fistula, resulting in an increased capacity to ingest food and suboptimal weight loss or weight regain
5. Dumping syndrome is a complication of gastric bypass resulting in facial flushing, lightheadedness, reactive hypoglycemia, and postprandial diarrhea
6. Internal hernia can occur with gastric bypass, with intermittent postprandial pain and emesis
7. Sleeve gastrectomy involves removing a portion of the stomach, leaving less stomach and altering gastrointestinal hormones
8. Acute complications of sleeve gastrectomy include gastrointestinal obstruction and staple line leaks
9. Chronic complications of sleeve gastrectomy include sleeve dilation, gastrointestinal reflux disease and luminal stenosis/strictures
10. Acute complications that can accompany most any abdominal surgery include infection, dehydration, cardiac dysrhythmias, atelectasis and pneumonia, deep vein thrombosis, and pulmonary emboli

Potential Bariatric Surgery Patient

Does clinical evidence exist that the increase in body fat is pathogenic?

Did the patient make reasonable attempts to reduce body weight and improve health?

Was the patient evaluated by a clinician trained in comprehensive management of overweight and obesity (e.g., physician certified by the American Board of Obesity Medicine or provider credentialed in Advanced Education in Obesity Management)?

Does the patient demonstrate a commitment to follow post-operative recommendations, maintain necessary lifestyle changes and agree to life-long post-operative medical surveillance?

What are the specific insurance criteria that need to be met (e.g., documentation of prior unsuccessful weight loss attempts)?

Surgical Candidate	Non-surgical Candidate
Consider Bariatric Surgery and Continue Medical Obesity Management	Initiate, Continue and/or Intensify Medical Obesity Management and Consider Endoscopic Therapy

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Potential Bariatric Surgery Candidate

1991 National Institute of Health (NIH) Consensus Development Panel

BMI ≥ 35 with one or more adverse health consequence due to obesity	BMI ≥ 40

2022 American Society for Metabolic and Bariatric Surgery (ASMBS)

BMI ≥ 30 with type 2 diabetes Or BMI ≥ 30 without substantial or durable weight loss or co-morbidity improvement using nonsurgical methods	BMI ≥ 40
BMI ≥ 25 in Asian individuals	BMI ≥ 27.5 in Asian individuals

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Bariatric Surgery Poor Candidates

- Patient is not seeking to lose weight
- Medical conditions that would make surgery or anesthesia unsafe, such as end-stage lung disease, severe heart failure, portal hypertension, or coagulopathy
- Inflammatory bowel disease, untreated gastric ulcer, or GI motility disorders
- Pregnancy or planned pregnancy within the next 2 years
- Dependence on drugs or alcohol
- Uncontrolled depression, psychosis, or eating disorders
- Inability to commit to life-long lifestyle changes following bariatric surgery

Any determination to proceed with bariatric surgery should be based upon the evaluation of an individual patient by a qualified medical professional who specializes in the care of bariatric patients.

Bariatric Surgery Team

The exact composition of a bariatric surgery team will vary, but often will include the following:

- Bariatric coordinator
- Bariatric surgeon
- Dietitian
- Mental health specialist
- Bariatric nurses
- Medical bariatrician

Bariatric programs must meet strict criteria to participate in the following programs:

- MBSAQIP Center of Excellence
  - Managed by the Metabolic and Bariatric Surgery Accreditation and Quality Improvement Program of the American College of Surgeons
  - The most widely used credentialing program for bariatric surgery programs
  - Designations include ambulatory center, comprehensive center, adolescent center, and may include an obesity medicine qualification
- Health insurance programs may have their own certification programs

Bariatric Surgery Pathway Overview

Pre-operative steps

- Education and training with bariatric surgery team on bariatric surgery, nutrition, exercise, mental health
  - Pre-operative testing and/or consultations
  - Evaluation by primary care physician
  - Evaluation by bariatric surgeon
  - Evaluation by dietitian: Number of visits depends on program, health insurance requirements
  - Evaluation by mental health specialist
- Pre-authorization by patient's health insurance company (above evaluations must already be completed)

Surgery: Either same-day surgery or overnight

Post-operative steps

- Follow up with bariatric surgery team: 1 week followed by 1, 3, 6, and 12 months after surgery
- Annual follow-up with nutrition labs

Bariatric Surgery Pre-operative Evaluation

- Evaluation by primary care provider
- Pre-operative testing and/or consultations
  - Labs vary, but often include CBC, CMP, Vitamins B1, B9, B12, A, D, E, K, iron panel, TSH, HbA1c, lipid panel, pregnancy test
  - EKG, CXR, sleep study, EGD, cardiology consult, and/or pulmonary consult depending on patient comorbidities
- Evaluation by bariatric surgeon
  - Evaluate for contraindications or other special considerations for surgery
  - Discuss risks, benefits, and alternatives of surgery (often including nutrition, physical activity, behavior, and medication)
- Evaluation by dietitian
  - Education and planning for nutrition changes before and after bariatric surgery
- Evaluation by mental health specialist
  - Identification of underlying eating disorders, mood disorders, substance abuse, history of physical or emotional trauma, education regarding potential for increased suicide risk and transfer addictions post op, education on coping mechanisms

Bariatric Surgery Pre-operative Evaluation

Goals of patient education and training before surgery

- Patient forms reasonable expectations of themselves and bariatric surgery
- Patient understands limitations of bariatric surgery
- Patient can identify risks and benefits of bariatric surgery
- Patient commits to the following requirements for health maintenance after bariatric surgery:
  - Drinking at last 64 ounces of water every day after surgery
  - Taking nutrition supplements every day after surgery
  - Developing and maintaining nutrition habits that are healthy and sustainable
  - Being physically active for at least 30 minutes every day after surgery
  - Developing mental health tools to cope with challenges and setbacks
  - Regular follow-up with bariatric surgery team, including annual after surgery

Bariatric Surgery Pre-operative Evaluation

The final evaluation of a patient by their bariatric surgery team prior to surgery will determine whether the patient is ready to proceed with surgery. A patient may have surgery delayed or cancelled if the following safety considerations are not met:

- Cessation of tobacco products for at least 3 months prior to surgery
- Cessation of NSAID products, including aspirin and ibuprofen
- Adequate and completed treatment for alcohol or other substance abuse
- No active, severe health condition impairing ability to perform safe bariatric surgery
- Adequate social and mental support for patient
- Completion of bariatric pathway steps in time allotted prior to surgery

Excess Weight Loss

- "Excess weight loss" is a term, mainly used in the surgical literature, to describe the percent amount of weight lost in excess of ideal body weight
- May have variances in how "ideal body weight" is determined
- It is challenging to directly compare "excess weight loss" often described in the surgical literature to the "weight loss" described in the medical literature, which is simply the percent of weight loss from baseline
- For the same amount of actual weight loss, the percent "excess body weight loss" is often a higher reported value compared to "total body weight loss" (i.e., EBWL vs TBWL)

Total Body Weight Loss Percent =
Weight Lost / Initial Weight * 100

Excess Weight Loss Percent =
Weight Lost / (Initial Weight – "Ideal Weight") * 100

Bariatric Surgical Procedures

	Pros	Cons	Expected EBWL at two years	Optimally suited for patients with:	Other comments
Roux-en-Y Gastric Bypass	Greater improvement in metabolic disease and GERD	Increased risk of malabsorptive complications over sleeve	60-75%	Higher BMI, GERD, Type 2 DM	Largest data set
Vertical Sleeve Gastrectomy	Improves metabolic disease; micronutrient deficiencies infrequent	Can worsen GERD and Barrett's esophagus	50-70% (*3-year data)	Metabolic disease	Currently most common procedure performed
Laparoscopic Adjustable Gastric Banding	Least invasive; removable	Limited efficacy and any metabolic benefits achieved are dependent on weight loss	30-50%	Lower BMI; no metabolic disease	Performance has declined and removal rate of at least 25 percent at five years
Biliopancreatic Diversion with Duodenal Switch	Greatest amount of weight loss and resolution of metabolic disease	Increased risk macro- and micronutrient deficiencies over bypass	70-80%	Higher BMI, Type 2 DM	Most technically challenging
Loop Duodenal Switch	May be simpler & safer than BD-DS with less micronutrient deficiencies	Long-term data not available	70-80%	Higher BMI, Type 2 DM	Two step procedure: VSG followed by single anastomosis

Bariatric Surgical Procedures

Roux-en-Y Gastric Bypass (RNY)

The stomach is completely divided into a small proximal gastric pouch leaving a large "bypassed" gastric remnant in situ. The proximal gastric pouch is attached to a "roux" limb of small bowel, bypassing the large gastric remnant, all of the duodenum, and a portion of the proximal small intestine.

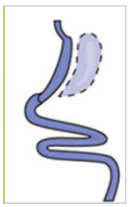
Laparoscopic Adjustable Gastric Banding (LAGB)

An adjustable band is placed around the upper stomach creating a small pouch. The band diameter is adjustable through the percutaneous introduction of saline via a subcutaneous port which is accessed in the upper abdomen.

Bariatric Surgical Procedures

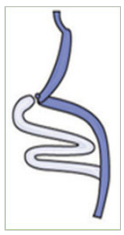
Vertical Sleeve Gastrectomy (VSG)

The stomach is reduced to about 25 percent of its original size by the surgical removal of a large portion of the stomach along the greater curvature, resulting in a narrower sleeve or tube-like structure. Can be used as the first step of staged approach of duodenal switch.



Biliopancreatic Diversion with Duodenal Switch (BPD/DS)

A partial gastrectomy (much like a sleeve) is performed, removing 70-80% greater curvature of the stomach sparing the pylorus and a small portion of the duodenum and the creation of a Roux-en-Y duodenoenterostomy bypassing a large portion of the intestine.



Bariatric Surgery Perioperative Medications

- Some medications that increase perioperative risks may be held prior to surgery, such as:
  - Anticoagulants, with risk of holding or bridging medications weighed against bleeding risk
  - Steroids and other medications that impair wound healing
  - Weight loss medications
  - Hypoglycemic medications
- Typical post-operative medications include analgesics, antiemetics, bowel regimen, and antacids.
- Patients at increased thrombotic risk may be temporarily placed on anticoagulant.
- Some medications may be decreased or stopped after surgery, such as:
  - Anti-hypertensives
  - Anti-hyperglycemics

Bariatric Surgery Post-Operative Diet

After bariatric surgery, most patients are started on a liquid diet for several reasons:

- To accomplish drinking 64 ounces of water every day
- To enable healing of gastrointestinal anatomy manipulated during bariatric surgery
- To enable patients to easily track and monitor food intake post-operatively

In general, these are the types of diets used in a staged progression from liquid to solid foods over weeks to months:

- **Clear liquid diet (CLD):** Anything liquid that is see-through and is easy to swallow
  - CLD is often the first intake following bariatric surgery.
  - CLD may last several days to multiple weeks.
- **Full liquid diet (FLD):** Any liquid or semisolid that is easy to swallow and requires minimal chewing.
  - FLD is the consistency of yogurt, oatmeal, cream of wheat, or baby food.
  - FLD is also sometimes referred to as pureed/ blended diet.
- **Soft diet:** Any food that is easy to chew and swallow.
  - Soft diets include most fish, ground meat, well-cooked vegetables, most grains, and thicker foods like peanut butter.

The specific diet plan, progression, and monitoring is unique to bariatric surgery program and patient. Each patient should learn the post-operative diet plan of their program and ask questions directly to their program dietitian.

Bariatric Surgery Long Term Dietary Changes

While there are no universal dietary guidelines for patients after bariatric surgery, commonly used recommendations include:

- Drink 64 ounces of water every day
- Take nutrition supplements containing vitamins A, D, E, K, B-complex, iron, and calcium every day
- Eat smaller meals, more often throughout the day
- Eat a balanced diet of proteins, carbohydrates, and fat with every meal
- Track food intake, at least initially, to form healthy habits and understand food intake
- Limit foods and drinks with added sugar
- Limit carbonated and caffeinated drinks
- Limit fatty and fried foods
- Specifically for patients after gastric bypass, limit spicy and acidic foods

Other FDA-Approved Bariatric Procedures

Health insurance coverage may be limited for these procedures

Intragastric Balloons

- Mechanism: Balloon is inserted into stomach and filled
- Indication: Body mass index > 30 and < 40 kg/m^2; approved for up to 6 months
- Types:
  - Intragastric fluid-filled and swallowable gas filled balloon
  - TransPyloric shuttle
- Efficacy: 12-31% excess weight loss over 6-12 months
- Safety: Stomach blockage with uncomfortable fullness, vomiting, stomach ulcer, gastric hypertrophy, and early device removal

Other FDA-Approved Bariatric Procedures

Aspiration Therapy Via Modified Percutaneous Endoscopic Gastrostomy (PEG)

- Mechanism: Drains 30% of ingested meal
- Indication: Body mass index 35-55 kg/m^2
- Efficacy: 12% excess weight loss at one year
- Safety: Potential tube site inflammation/infection
- As of April 2022, the manufacturer is no longer producing the device due to financial reasons

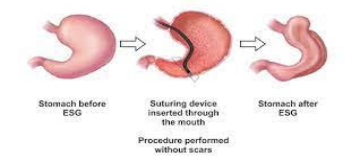
Electrical Vagal Blocking System

- Mechanism: Pacemaker-like implantable device surgically placed under skin, with lead wires placed around the vagus nerve just above the stomach; blocks vagal impulses to brain resulting in decreased hunger and increased satiety
- Indication: Body mass index > 40 kg/m^2 or > 35 kg/m^2 among those with adverse consequences of obesity
- Efficacy: 8.5% excess weight loss
- Safety: Potential gastroparesis (vagal trunk injury or entrapment)

Other FDA-Approved Bariatric Procedures

Endoscopic Sleeve Gastroplasty (ESG)

- Procedure: Endoscopic suturing of the stomach creating a full-thickness plication of stomach tissue
- Indication: BMI 30-50 kg/m^2
- Efficacy: 16% total body weight loss and 60% excess weight loss for up to 1-2 years
  - Improvement of at least 1 weight-related chronic disease in 80% of individuals
- Safety: Stitch failure, upper GI bleeding, peri-gastric leak/infection, perforation
  - No deaths observed in published ESG literature



Physiologic Mechanisms of Weight Loss with ESG

- Restriction of gastric expansion induces early satiation during meals
- Delayed emptying of the stomach promoting a prolonged sensation of satiety after meals
- Fundus is left intact, and ESG does not appear to affect ghrelin secretion the way VSG/LSG does.

Bariatric Surgery: Early Complications (First 30 Days)

General Post-operative Complications

- Nausea/vomiting
- Dehydration
- Wound infection
- Cardiac dysrhythmias
- Deep vein thrombosis and/or pulmonary embolism
- Atelectasis and pneumonia

Bleeding at the Surgical Site or Rarely Intraluminal/Gastrointestinal (RNY, Sleeve gastrectomy, BPD/DS)

- Usually within 72 hours post-op, may require early intervention or reoperation
- Symptoms: tachycardia, hypotension, drop in hemoglobin/hematocrit, oliguria
- From three to seven days out, cause is more likely due to erosions and ulcerations at the anastomoses or along staple lines

1 2 3 4 5 6 7

Bariatric Surgery: Early Complications (First 30 Days)

Leak or Perforation (RNY, Sleeve gastrectomy, BPD/DS)

- Can lead to acute peritonitis
- Technical failure within the first 72 hours (with ischemia can occur up to 14 days post-op)
- Can also occur at any time due to ulcer perforation (avoid NSAIDS, steroids, nicotine, caffeine, alcohol)
- Often with acute and severe abdominal pain (may *NOT* have peritonitis symptoms if on steroids)
- Fever, tachycardia, abdominal or back pain, and leukocytosis
- Urgent surgical exploration may be required but can sometimes be managed with endoscopic stent and drain (in selected cases)
- Imaging not always diagnostic but when performed, water soluble contrast preferred (abdominal CT or Upper GI)
- Immediate surgical consultation is critical for suspected leak or perforation *EVEN* if imaging is negative

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Bariatric Surgery: Late Complications (>30 Days)

Gastro-gastric Fistula (RNY)

- Results in increased capacity to ingest food, and/or increased passing of food into the gastric remnant (where it is more completely digested and absorbed)
- Possible contributing factor to suboptimal weight loss/weight regain and recurrence of metabolic disease
- A non-healing ulcer might raise concern for a gastro-gastric fistula

Band Erosion through Gastric Wall into the Lumen (LAGB)

- Suspect if band is full but patient perceives no restriction or obstructive symptoms with empty or minimally filled band
- Can also present as infection with pain, fevers, leukocytosis
- Pain/infection may or may not be present
- Diagnose with esophagogastroduodenoscopy (EGD), surgical consult for removal is required for eroded band

Bariatric Surgery: Late Complications (>30 Days)

Incisional Hernias (More Common with Open Procedures)

- Pain at one of the incisional sites
- Maybe be palpable defect but due to body habitus this may be difficult to ascertain on exam and CT or US is needed to confirm
- Repair usually postponed until significant weight loss unless signs of bowel incarceration/strangulation (bowel obstruction)

Internal Hernias (RNY/BPD-DS)

- Usually accompanied by intermittent, post prandial pain and emesis, sometimes only pain
- Herniation through defect in the mesentery created during the surgical procedure
- Challenging to diagnose both clinically and radiographically- if suspected, diagnostic laparoscopy often needed, and surgical consult should be considered even if imaging is negative
- *Surgical emergency if sudden/acute onset*

Sleeve / pouch dilation (RNY/Sleeve Gastrectomy)

- Longer-term expansion of the residual stomach
- May result in weight regain after sleeve gastrectomy or gastric bypass
- Treatment includes endoscopic reduction of dilated sleeve or pouch reduction

Bariatric Surgery: Early or Late Complications

Intestinal (Small Bowel) Obstruction (RNY, BPD-DS, or Open Procedure)

- Abdominal pain, nausea/vomiting, (constipation/obstipation not present if partial)
- Usually, six months or longer out from surgery but can be anytime
- May be associated with an internal hernia, narrowing of the roux limb due to scarring, intussusception, and/or adhesions
- Evaluation: CT scan abdomen most common but can also be seen on plain flat/upright abdominal x-rays

Stricture (Stomal Stenosis) (RNY, Sleeve gastrectomy, or BPD-DS)

- Post-prandial, epigastric abdominal pain and vomiting (often with frothy emesis)
- Usually, 4-6 weeks following RNY
- May result from narrowing of the anastomosis or angulation of the intestinal limbs
- May be associated with anastomotic ulcer (RNY and BPD-DS)
- EGD +/- balloon dilation. Surgery only after multiple failed dilations

Band Obstruction: Band Too Tight, Band Slip/Prolapse (LAGB)

- Abdominal pain, reflux, and regurgitation of undigested food which occurs post-prandially
- Weight gain can occur due to dependence on liquid calories
- Diagnostic testing: Can be clinical diagnosis, or upper GI imaging/EGD
- Surgery indicated for a slip which is not relieved after the complete removal of all band fluid

1 2 3 4 5 6 7

Bariatric Surgery: Early or Late Complications

Dumping Syndrome (RNY)

- Unique complication of RNY (due to bypass of the pyloric emptying mechanism), which is common in the first 18 months postoperatively
- Occurs in approximately 70-85 percent of patients with RNY
- Symptoms: Facial flushing, lightheadedness, fatigue, reactive hypoglycemia, and postprandial diarrhea
- Treatment: Often includes avoidance of foods with high glycemic index/load, avoidance of drinking fluid with meals.
- Acarbose may help alleviate symptoms of dumping syndrome

Gallbladder or Gallstone Disease

- Right upper quadrant or epigastric post-prandial or nocturnal pain (classically radiating to back or right shoulder)
- Diagnostic testing includes labs (if elevated white blood cell count, alkaline phosphatase, bilirubin, liver transaminases, or amylase lipase send to Emergency Room for urgent surgical consult)
- Imaging: Abdominal ultrasound (abdominal CT if abdominal wall thickness impairs ultrasound), consider HIDA scan if ultrasound is negative

Marginal Ulcer (at an anastomotic site-most common with RNY)

- Abdominal pain +/- vomiting
- Best to stop NSAIDS, steroids, nicotine, caffeine, alcohol, and/or illicit drugs to heal
- Proton pump inhibitor 3 times/day plus Carafate 4 times/day; optimize protein intake; surgery for failed refractory ulcer
- Diagnose with upper endoscopy, consider surgery for refractory disease

Bariatric Surgery: Early or Late Complications

Metabolic bone disease (RNY, BPD/DS)

- Secondary hyperparathyroidism and osteopenia resulting from calcium deficiency

Kidney stones (RNY, BPD/DS)

- Hyperoxaluria may lead to oxalate kidney stone

Gout

- Rapid weight loss may increase the risk of gout exacerbation

Mental health

- Risk of addiction transference
- Increased risk of suicide

Bariatric Surgery Considerations After Surgery

- **Hair loss:** Any weight loss can cause hair loss. This can be avoided and treated with dietary interventions.
- **Loose skin:** Body contour can change with weight loss. Bariatric surgery teams can refer patients to plastic surgeons for expertise in body contour changes following bariatric surgery.
- **Constipation:** Common after bariatric surgery. Recommend 64oz water daily, physical activity, fiber and dietary intake variety including fruits, vegetables and grains. Can also be treated with medications.
- **Weight regain:** Weight regain can result from many factors. This requires evaluation by bariatric surgery team for possible lifestyle, mental health, medication, endoscopic, and surgical intervention.
- **Pregnancy:** Pregnancy should be avoided for at least 1 year after bariatric surgery. This period allows for weight stabilization with identification and treatment of malnutrition. Many patients can undergo safe pregnancies following bariatric surgery with the support of their bariatric surgery team.

Bariatric Surgery Weight Regain

- Bariatric surgery was initially intended to be a once-in-a-lifetime procedure.
- Health insurance coverage of bariatric procedures often reflects the expectation.
- The prevalence of weight regain may less than 10% in patients with duodenal switch and up to 30% in patients with sleeve gastrectomy.
- In patient's that maintain nutrition and physical activity changes , the prevalence of weight regain is less than 10%.
- Revision rates are increasing for bariatric surgery and are now as high as 25% in some areas. Most common indications for bariatric surgery revision:
  - Complication of initial bariatric surgery
  - GERD
  - Weight regain

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References

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