Obesity Algorithm®

2020

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Obesity Medicine Association

CLINICAL LEADERS IN OBESITY MEDICINE®

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* 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.

Supporting documents in reference section

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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 that is 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.

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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.

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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.

Supporting documents in reference section

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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.

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Major Updates Included in the 2020 Version

- New Sectional: *"Top 10 takeaway messages"*
- Adiposopathic aging
- Polycystic ovary syndrome
- Ketogenic diet
- Obesity and psychiatry
- Obesity myths
- Anti-obesity agents in development table
- Table of the role of gastrointestinal hormones in energy and nutrient regulation
- Updates to bariatric surgery nutrient replacement
- Removal of lorcaserin as a marketed anti-obesity agent
- Links to educational resources
- General updates and text edits
- Updated references

Supporting documents in reference section

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OMA Obesity Algorithm eBook, Slides, Authors and Citations

Adult Obesity Algorithm eBook: Detailed overview of Obesity Medicine

Citation: Bays HE, McCarthy W, Christensen S, Tondt J, Karjoo S, Davisson L, Ng J, Golden A, Burridge K, Conroy R, Wells S, Umashanker D, Afreen S, DeJesus R, Salter D, Shah N, Richardson L. Obesity Algorithm eBook, presented by the Obesity Medicine Association. www.obesityalgorithm.org. 2020. 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: Bays HE, McCarthy W, Christensen S, Tondt J, Karjoo S, Davisson L, Ng J, Golden A, Burridge K, Conroy R, Wells S, Umashanker D, Afreen S, DeJesus R, Salter D, Shah N, Richardson L. Obesity Algorithm Slides, presented by the Obesity Medicine Association. www.obesityalgorithm.org. 2020. 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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The Disease of Obesity

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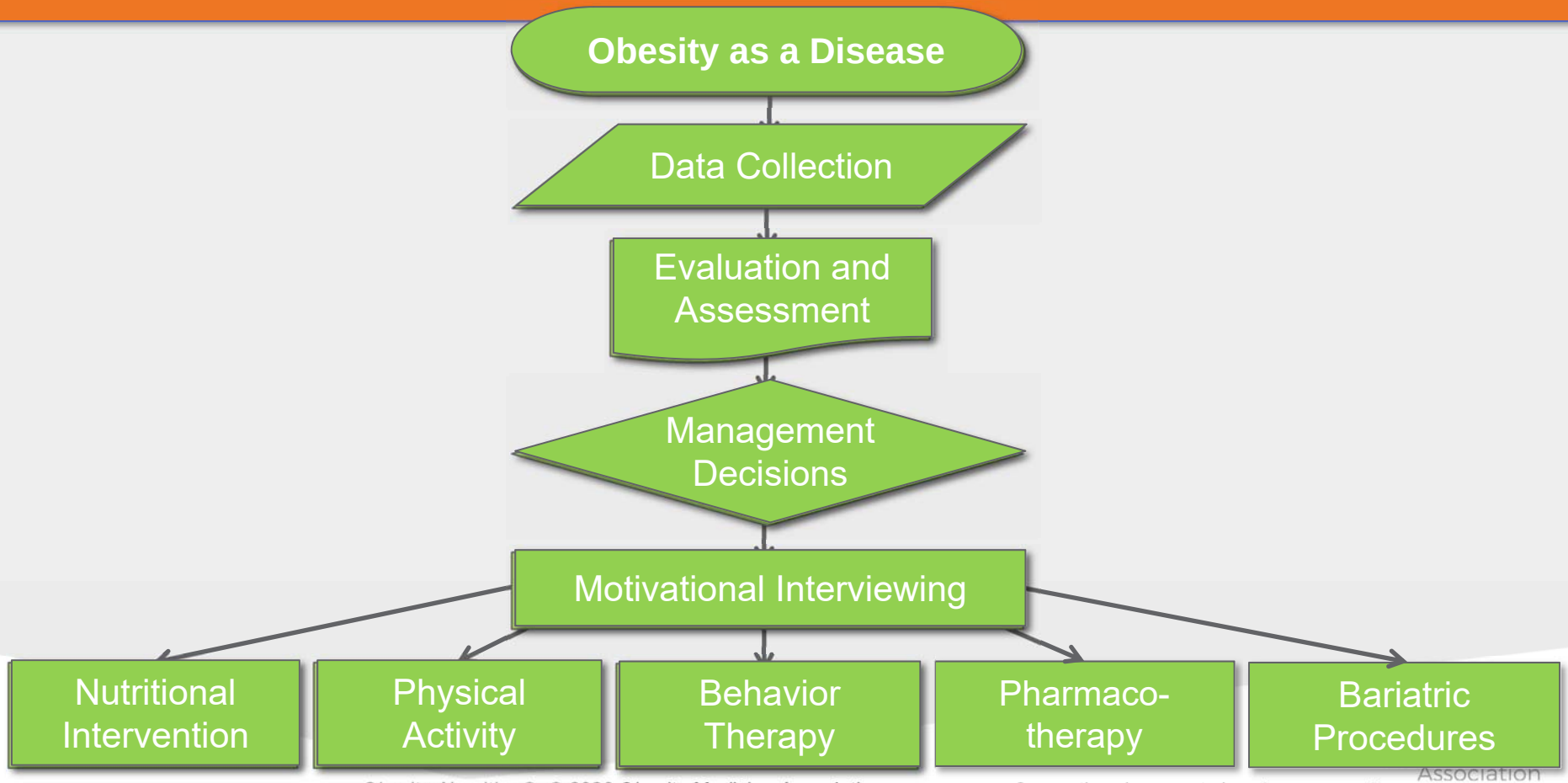
The OMA Obesity Algorithm



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The Obesity Medicine Association's Definition of Obesity

"Obesity is defined as a chronic, progressive, relapsing, 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."

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Top 10 benefits of treating obesity as a disease

1. Healthful nutrition (including negative caloric balance in patients with obesity) 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 has the potential for statistically significant and clinically meaningful weight loss maintenance
4. Weight loss in patients with obesity may reduce premature all cause 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 metabolism (polycystic ovary syndrome), as well as improve obesity-related gynecologic and obstetric disorders; weight loss in men may increase testosterone levels when hypogonadism is due to the adiposopathic consequence of obesity
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

Supporting documents in reference section

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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 substantially be due to inheritance (genetic, epigenetic, and/or environmental inheritance)
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. Even when exacerbated by unhealthful behavior, obesity is no less a disease than other diseases promoted by unhealthful behavior
8. Data from 2015 – 2016 estimate that approximately 40% of US adults have obesity; data from 2015 – 2016 support approximately 18.5% 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

Supporting documents in reference section

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Obesity Is a Disease When…

- The patient has excessive body fat, as assessed by reliable measures
- 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
- Excessive body fat results in pathogenic structural or functional abnormalities resulting in increased patient morbidity and mortality
- Multiple pathogenic adipocyte and/or adipose tissue endocrine and immune dysfunctions contribute to metabolic disease (adiposopathy or “sick fat” disease)
- 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”

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Obesity Terminology

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

Encouraged Terms

- Weight
- Unhealthy weight
- Overweight
- Body mass index
- Excessive energy stores
- Affected by obesity

Discouraged Terms

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

Supporting documents in reference section

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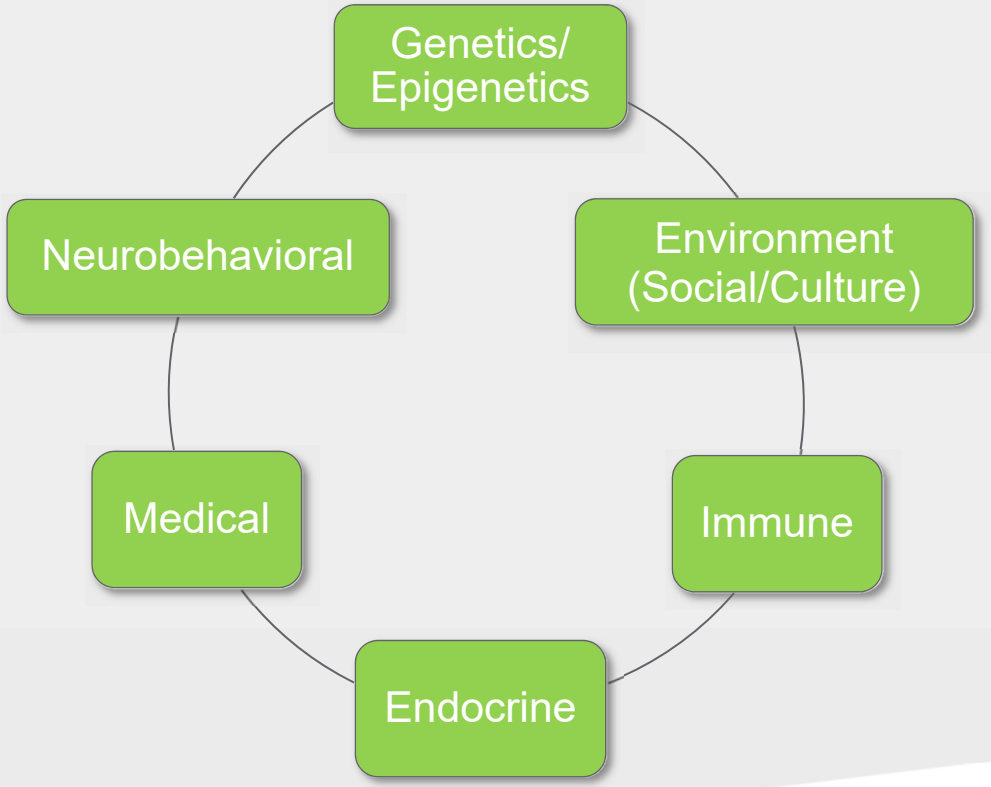
Obesity is a Multifactorial Disease



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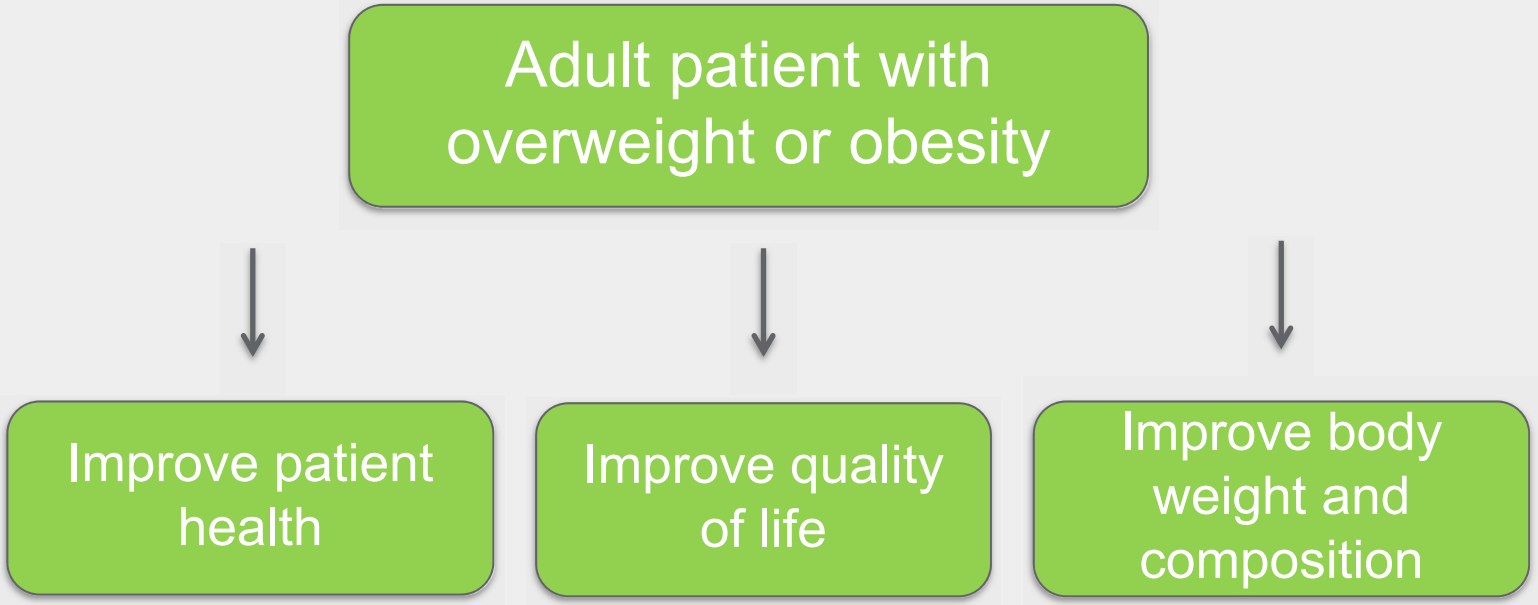
Overall Management Goals



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Classification of Obesity

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Top 10 Takeaway Messages: Obesity Classification and Consequence

1. For the general population, body mass index (BMI) ≥ 25 kg/m^2 is considered overweight; BMI ≥ 30 kg/m^2 is considered obesity
2. BMI has limitations in assessing adiposity in individuals with increased muscle mass, decrease in muscle mass, men versus women, different races, and postmenopausal status
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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Obesity Paradox

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Top 10 Takeaway Messages: Obesity Paradox

1. An Obesity Paradox exists when increased body weight is found to have an apparent favorable effect on health, which often is less paradoxical when viewed from the perspective of both fat mass ***and*** fat function
2. Many “obesity paradoxes” are reported
3. Fat depots have the potential to be protective and/or pathogenic
4. Obesity increases morbidity and mortality
5. Obesity promotes “fat mass” disease
6. Obesity promotes “sick fat” disease or adiposopathy, which is the most common cause of the most common metabolic conditions encountered in clinical practice (e.g., diabetes mellitus, hypertension, dyslipidemia and thrombosis); thus, obesity indirectly contributes to major cardiovascular disease (CVD) risk factors
7. Obesity may directly increase the risk of CVD (e.g., adiposopathic epicardial effects)
8. Individuals with the highest body weight and lowest body weight have higher mortality
9. The increase in mortality with lower body weight is often due to the confounding effect of concurrent illnesses (e.g., poor nutrition, malabsorptive syndromes, cancer & cardiac cachexia) and cigarette smoking, that not only contribute to low body weight, but also to increased mortality
10. Obesity increases the risk of cancer

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Obesity and Stress:
Cause and Effect

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Top 10 Takeaway Messages: Obesity and Stress

1. Shorter-term “fight or flight” stress response increases adrenal sympathomimetic activity
2. Shorter-term adrenergic stress responses may improve cognition, physiologic function, tolerance to pain, and immune function
3. Longer-term “submit and stay” stress may increase hypothalamic corticotropic activity
4. Longer-term hypothalamic stress responses may increase food craving, increase blood pressure, worsen glucose metabolism, promote pain intolerance, and dysregulate immune responses
5. Chronic stress-induced adiposopathic responses may adversely affect the limbic system
6. Dysregulation of the limbic system with chronic stress may affect hunger, food choice, and emotional modulation of food intake
7. Dysregulation of the limbic system with chronic stress may affect reward-seeking behavior
8. Mental stress may affect the cerebrum, which may contribute to prioritization of personal, work, or other behaviors and activities over more healthful behaviors and activities (i.e., healthful nutrition and regular physical activity)
9. Mental stress may impair self-regulation and promote choosing unhealthful (immediately rewarding ultra-processed) foods over more healthful (delayed-gratification unprocessed) foods
10. Obesity and its adverse health complications may increase mental stress, which may contribute to unhealthful behavior, endocrinopathies and immunopathies, which in turn, may further worsen obesity and its complications, resulting in an adiposopathic stress cycle

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Supporting documents in reference section

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 diaries
3. Body systems to be evaluated before prescribing a physical activity program include cardiac, pulmonary, and musculoskeletal systems, as well as body 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, near-infrared interactance, myotape, calipers, or underwater weighing
8. Prader Willi is the most common non-inherited, non-polygenic 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, some cases of untreated hypothyroidism, and adverse effects of concurrent medications

Supporting documents in reference section

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Treatment of Adult Patients with Overweight or Obesity

Medical Management and Coordination

Supporting documents in reference section

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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

Body Composition

Podcast
Obesity: A Disease
Episode 5

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Top 10 Takeaway Messages: Obesity and Body Composition

1. Lean body mass is total body mass less storage adipose tissue (i.e., water, mineral, protein, glycogen, essential organ fat)
2. In lean individuals, approximately 60% of body weight is water (i.e., water is 75% weight of muscle and body organs). In those with obesity, water weight can be as low as 40% body weight.
3. Ash weight of bone contributes a minor amount to total body weight (~3 to 10 pounds)
4. Percent body fat mass is highly variable, and may range from <5% to > 70%
5. Percent body fat mass is dependent upon both fat and muscle mass
6. Methods to measure body composition vary regarding accuracy, reproducibility, expense, and accessibility
7. Some Dual Energy X-Ray Absorptiometry (DXA) scans can measure percent body fat, android fat (abdominal subcutaneous and visceral fat), lean body mass, and bone mineral density
8. Calipers can estimate percent body fat, are user dependent, are inexpensive, and perhaps more useful for frequent longitudinal assessments once body composition is determined by more accurate measures
9. Bioelectrical impedance is a hydration-dependent body composition assessment procedure; reasonable assessment of android fat may best be achieved via a complementary tape-measured waist circumference
10. Air displacement assessment of percent body fat is clothing and hydration dependent; reasonable assessment of android fat may best be achieved via a complementary tape-measured waist circumference

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Energy Expenditure

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Top 10 Takeaway Messages: Obesity and Energy Expenditure

1. For most individuals, resting metabolic rate (RMR) represents ~70% of total daily energy expenditure
2. For most individuals without excess body fat, skeletal muscle, liver, brain, heart, and digestive system each account for ~10 - 20% of RMR (~75% of total RMR). Kidney, adipose tissue, and remaining/residual = ~25%.
3. Non-exercise Activity Thermogenesis (NEAT) varies among individuals, can range between 150 – 500 kcal/day (often greater than bouts of physical exercise), and can help account for the perception that some individuals more easily maintain a healthy body weight despite similar caloric intake and dedicated physical exercise
4. Less than 5000 steps per day is considered sedentary; $\geq$10,000 steps per day is considered active
5. Direct calorimetry estimates energy expenditure via measurement of heat generated by an organism in an enclosed chamber
6. Indirect calorimetry estimates energy expenditure via use of an electronic metabolic "cart," that measures carbon dioxide production and oxygen consumption. The respiratory quotient (RQ) = CO_2 production / O_2 consumption. The RQ for carbohydrates is 1.0; the RQ for fats is 0.7.
7. Resting energy expenditure can be estimated by calculations (Harris-Benedict & Mifflin St Jeor Equations)
8. Doubly labeled water estimates energy expenditure via oral administration of traceable hydrogen and oxygen isotope, and the estimation of carbon dioxide production, reflecting energy expenditure due to tissue respiration
9. Physical activity expenditure can be estimated by wearable technologies, such as pedometers and accelerometers
10. Energy expenditure may be increased with greater inefficiency in physiologic and behavior processes

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. Anti-depressants 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, sertindole, thioridazine, trifluoperazone, 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

Nutrition Therapy for Obesity

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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 promote 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 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. The Ketogenic Diet is a carbohydrate-restricted intervention that typically discourages unhealthful ultra-processed and refined foods, foods high in glycemic index/load, and foods rich in *trans* fatty acids. Ketosis may reduce appetite.
7. The Mediterranean Diet is not a defined diet, but rather a generalized meal pattern that 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 unhealthful ultra-processed carbohydrates
8. The DASH Diet is a diet pattern that 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 unhealthful ultra-processed meats, sugar-sweetened beverages, and foods with added sugars
9. The Vegetarian Diet 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

Supporting documents in reference section

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Dietary Patterns

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 (Atkins) diet
- Ornish diet
- Paleo diet
- Vegetarian diet
- Intermittent fasting
- Commercial diet programs

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 consistent and robust scientific support in reducing atherosclerotic cardiovascular disease risk.

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

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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” most often 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.

Supporting documents in reference section

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Ketogenic Diet (Keto or 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 appetite.

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 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 as long as weight gain does not occur.
- **In the maintenance phase,** 60 to 90 grams of carbohydrates per day is allowed, 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

Supporting documents in reference section

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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

Supporting documents in reference section

Obesity Medicine Association

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

Supporting documents in reference section

Obesity Medicine Association

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 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

Supporting documents in reference section

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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), other 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.

Supporting documents in reference section

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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

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

- May contribute to overall caloric restriction
- Potential advantages:
  - Reducing “decision fatigue” regarding food selection
  - Quickly reversible
  - May better fit in day-to-day patient scheduling (also Ramadan)
  - May reduce caloric intake with preservation of lean body mass
  - May not reduce resting metabolic rate and total energy expenditure
  - 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 history of eating disorders (e.g., bulimia)
  - 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 and reported safety in animal studies
  - Prolonged fasting may promote gout, urate nephrolithiasis, postural hypotension, and cardiac dysrhythmias

Podcast
Obesity: A Disease
Episode 6

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 body processes adversely affected by the disease of obesity (i.e., adiposopathic endocrine and immune abnormalities)
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. Tracking physical activity can be via a variety of activity logs, as well as percent body fat measurements by a reliable technique

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Motivational Interviewing

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Top 10 Takeaway Messages: Obesity and Motivational Interviewing

1. **Stages of change** that may be evaluated during motivational interviewing include pre-contemplation, contemplation, preparation, action, maintenance, and relapse
2. **General motivational interviewing principles** include empathy, avoid arguments, develop discrepancy, resolve ambivalence, and support self-efficacy
3. **Empathy** involves communication, understanding, collaboration, support, encouragement and listening
4. **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
5. **Developing discrepancy** explores the mismatch between where the patient is today, and where the patient says he/she wants to be in the future
6. **Resolving ambivalence** is amplifying discrepancy and addressing the uncertainty for the desire for change.
7. **Supporting self-efficacy** is affirming favorable results though focusing on patient successes and highlighting patient skills and strengths
8. **The 5A's** of motivational interviewing include Ask, Assess, Advise, Agree, and Arrange or Assist
9. **FRAMES** is a common motivational interviewing acronym = Feedback, Responsibility of the patient, Advice to change, Menu of strategies, Empathy, and Self-Efficacy
10. **OARS** is a common motivational interviewing acronym = Open-ended questions, Affirmation, Reflections, and Summaries

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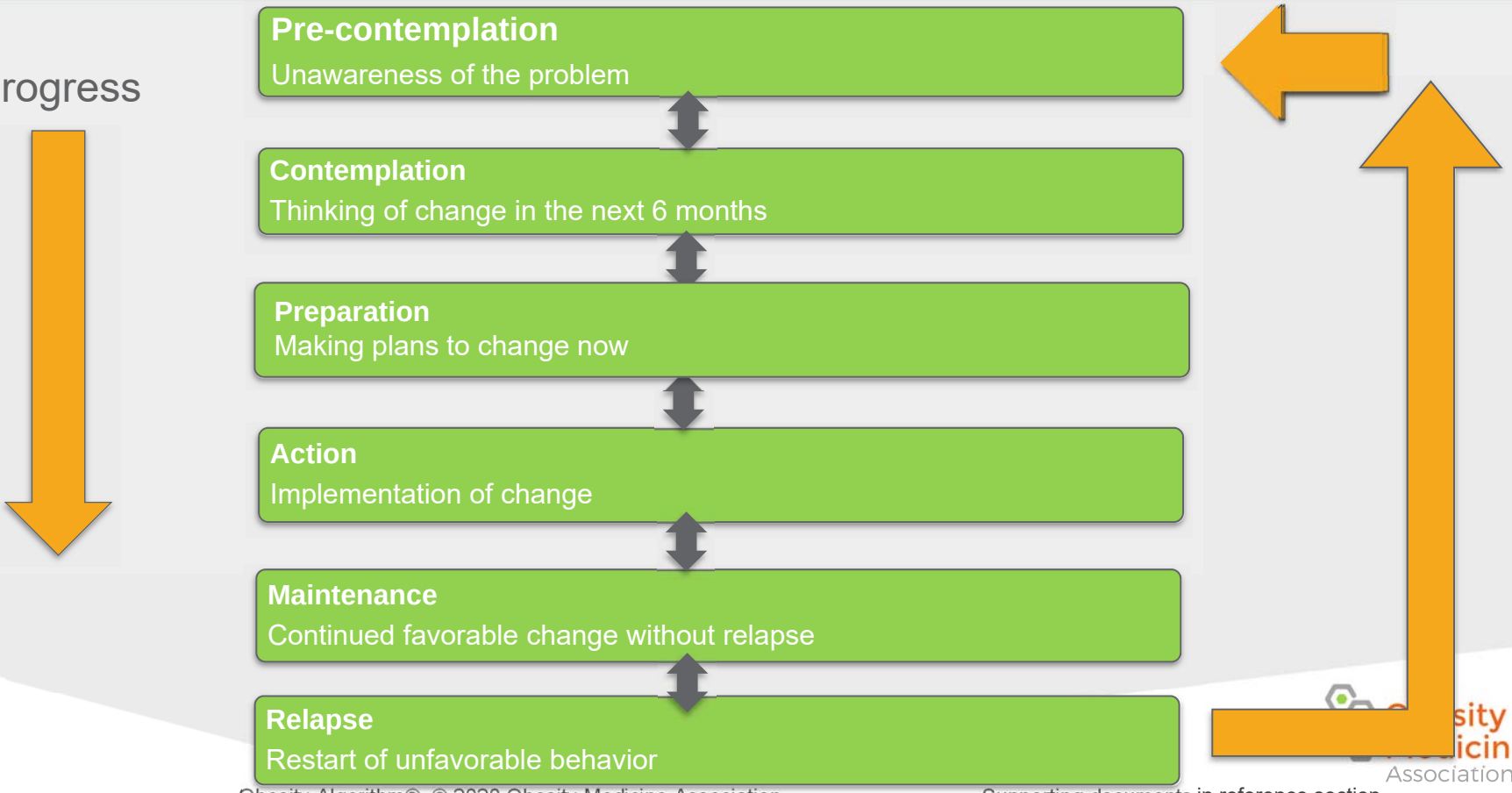
Supporting documents in reference section

Motivational Interviewing: Stages of Change



Supporting documents in reference section

Motivational Interviewing: Principles

- Express empathy
- Avoid argumentation
- Develop discrepancy
- Resolve ambivalence
- Support self-efficacy

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 loss (i.e., 5-10 percent), the need for long-term strategy, and treatment options.
Agree	• Agree on realistic weight-loss 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.

Supporting documents in reference section

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Behavior Therapy

Top 10 Takeaway Messages: Obesity and Behavior Therapy

1. Eating behavior is often the result of an imbalance in physiologic forces that strongly resist weight loss 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 mental stress, emotions, habitual time cues, environment, information gap, and reward factors
4. Eating behavior can be affected by eating disorders (e.g., binge-eating disorder, bulimia nervosa, 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 conveniences, lack of time, disinterest, and environment
7. Behavior related to weight regain may be due to physiologic priority imbalance, neurobiology, and energy expenditure
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. Purchasing or providing access to weight management technologies and social media alone may have limited benefits; however, in motivated patients ready for change, utilization of technologies and social media can help facilitate elements of behavior therapy implementation, such as record keeping, education, and social support/motivation

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 2-3 percent of U.S. adults
- Often considered the most common eating disorder; may occur in up to 50 percent of patients with severe obesity
- Eating Attitudes Test may assist with diagnosis

Severity based upon episodes per week:

- Mild = 1 – 3; Moderate = 4 – 7; Severe = 8 – 13; Extreme = $\geq$ 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

Eating Disorders and Obesity: Bulimia Nervosa

Diagnosis:

- Cycle of recurrent binge eating and compensatory purging, laxative abuse, diuretic abuse, extra exercising, fasting, or strict dieting
- Occurs in approximately 1% of adults (mostly women)
- Russell sign: Calluses and abrasions on dorsum of the hands caused by repeated contact with the teeth during self-induced vomiting
- Laboratory: Hypokalemia due to hypomagnesemia

Treatment:

- Often requires treatment by a qualified clinician
- Fluoxetine is an FDA-approved pharmacotherapy for bulimia nervosa
- Although not FDA-indicated for this use, topiramate and naltrexone may be efficacious

Eating Disorders and Obesity: Night-eating Syndrome

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)
- Night-eating syndrome may occur in as much as 5% of the U.S. population

Treatment:

- Behavioral therapy regarding nutritional timing and content

Technologies for Weight Management

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Technologies and Social Media

Applications

- Record and assess nutritional and physical activity metrics
- May be assessed and reviewed by clinicians between face-to-face evaluations

Interactive Technology

- Body-weight scales that provide interactive feedback via email or text messaging
- Wearable technologies
  - Tracks active minutes, steps, floors climbed, distance, and caloric consumption
  - Monitors heart rate and sleep patterns
  - Provides daily exercise statistics
  - Wirelessly syncs with smartphones and computers, providing interactive information to user
  - Potential benefits of wearable technology beyond standard behavioral intervention depends on the individual, and thus recommending wearable technologies is best based upon a patient-centered approach

Websites

- Websites can provide educational information regarding:
  - Nutrition
  - Caloric content of foods
  - Physical activity
  - Expected energy expenditure with certain physical activities
  - Meal plans
  - Recipes

Social Media

- Post daily meals and snacks to followers to enhance accountability (Twitter, Facebook, etc.)
- Post physical activity progress to social network group
- Obtain nutritional and physical activity advice from others, including social network support groups specific to weight management (e.g., Twitter, Facebook, blogs, forums)
- Competition or “wagers” regarding fitness metrics and goals

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 contraindications that include use in patients with cardiovascular disease
2. Phentermine hydrochloride (HCl) 8 – 37.5 mg is generally equivalent to 6.4 – 30 mg of phentermine resin
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. Lorcaserin is a selective serotonin (5-hydroxytryptamine) 2 c receptor agonist voluntarily withdrawn from the market in 2020 due to a signal of a numerical increase (<1%) in the rate of various cancers (e.g., pancreatic, colorectal, and lung). It remains unclear if lorcaserin actually increased cancer risk or had any causal relationship to increased cancer risk.
6. Liraglutide is a glucagon-like peptide-1 receptor agonist 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
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 (pregnancy tests should be performed monthly during use)
9. Liraglutide 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

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

5-10 percent weight loss may improve both metabolic and fat mass disease.

Anti-Obesity Medication Summary

(All have contraindications for hypersensitivity and pregnancy)

Drug	Description	Main Side Effects	Illustrative Drug Interactions
Phentermine	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

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 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, and not continued unless the woman uses contraception; pregnancy tests should be done monthly during use.	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.

Functional Foods, Supplements, & Over-the-counter Therapies*

*The Obesity Medicine Association has not endorsed any supplements. This section is intended to provide information the authors believe may be relevant to the clinical management of patients with obesity.

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Top 10 Takeaway Messages: Supplements & OTC Therapies

1. Prescription drugs are a therapeutic medicine intended for the diagnosis, cure, mitigation, treatment or prevention of disease. Their approval and marketing is overseen by the US Food and Drug Administration.
2. Over-the-counter (OTC) medications are drugs the FDA considers safe and effective, but do not require a prescription by health professionals. Their approval by the FDA involves the regulatory process involving an OTC drug monograph, and their marketing is overseen by the Federal Trade Commission.
3. Supplements are substances taken in addition to dietary intake (e.g., vitamins, herbs, botanicals, minerals and amino acids). The FDA does not approve supplements, as they are considered a food, and not a drug.
4. Supplements are not permitted to market for the purpose of treating, diagnosing, preventing, or curing diseases.
5. Functional Foods are nutrients with potentially favorable effects beyond basic nutrition (e.g., oatmeal may lower cholesterol).
6. Herbal and dietary supplements are a common cause of hepatotoxicity (e.g., anabolic steroids and green tea extracts), especially multi-ingredient nutritional supplements.
7. Most dietary supplements have limited to no consistent data to support long-term weight loss efficacy and safety, and have the potential for adverse supplement to drug interactions.
8. Some weight loss supplements continue to be marketed, even when banned by the FDA due to adverse effects.
9. Patients should be advised of the limited evidence supporting the efficacy and safety of many supplements and the lack of oversight by government agencies regarding the claims made about such supplements.
10. Human chorionic gonadotropin (HCG) is not effective for weight loss beyond hypocaloric intake. The FDA requires clinicians and facilities that market HCG for weight loss to post a disclaimer that "*There is no substantial evidence that HCG increases weight loss beyond that resulting from caloric restriction, that it causes a more attractive or normal distribution of fat, or that it decreases the hunger and discomfort associated with calorie restrictive diets.*"

Position Statement: Recommendations for Dietary Supplements Sold as Medicinal or Curative for Obesity*

Healthcare providers should:

- Be aware of the lack of credible evidence for efficacy and safety of many supplements promoted for the purpose of weight loss.
- Query patients who desire to accomplish weight loss regarding their use of dietary supplements for this purpose.
- Advise patients who desire to accomplish weight loss of the limited evidence supporting the efficacy and safety of many supplements and the lack of oversight by government agencies regarding the claims made about such supplements.
- Be educated on the Dietary Supplement Health and Education Act (DSHEA) and the **roles of FDA and FTC** in safety and claims monitoring of supplements promoted for the purpose of weight loss.
- Healthcare providers are strongly discouraged from engaging in entrepreneurial activities in which they directly profit from the prescribing of non-FDA approved weight-loss remedies where both safety and efficacy have not been proven.

* The Obesity Society with co-signatories = Obesity Action Coalition, Obesity Medicine Association, Academy of Nutrition and Dietetics. **www.obesity.org/publications/position-and-policies/medicinal-or-curative** (accessed October 11, 2019)

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Obesity and Metabolic Disease

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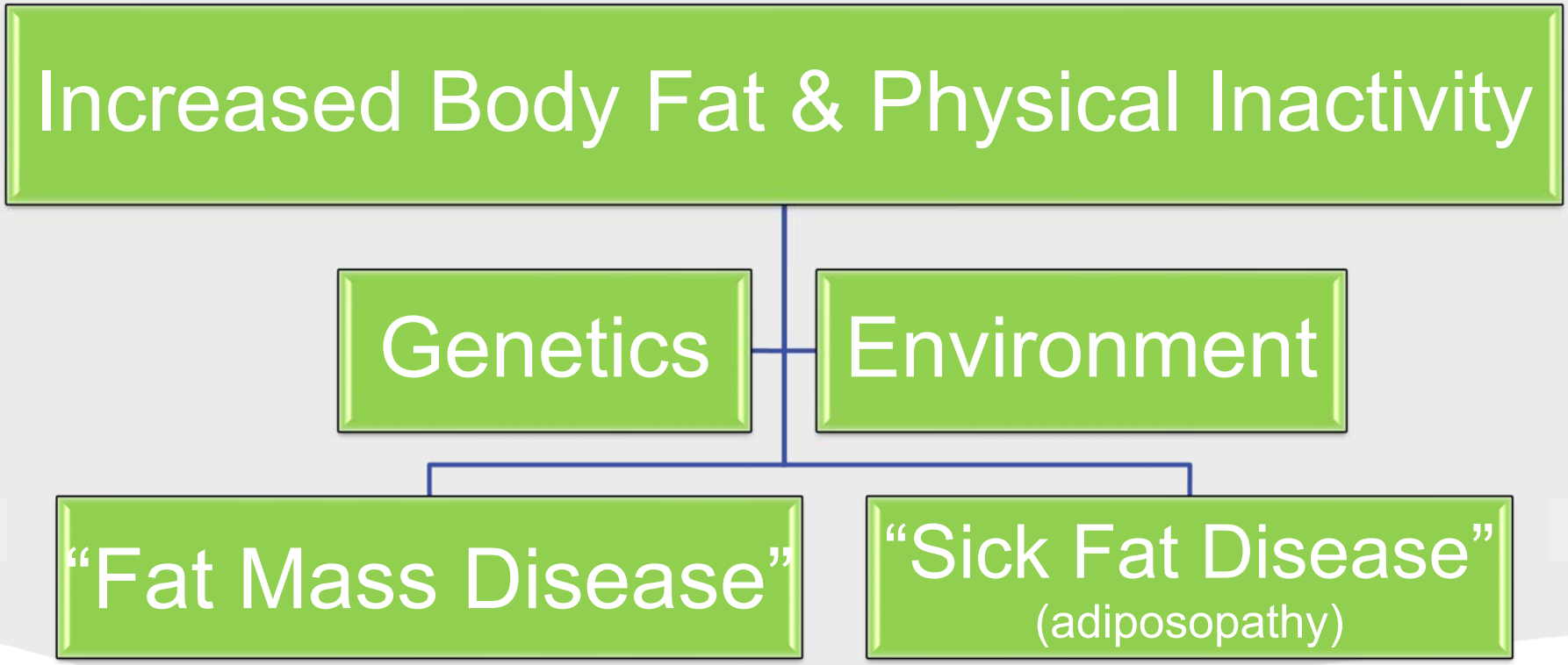
Obesity: Both “Fat Mass Disease” and “Sick Fat Disease” are pathogenic



Supporting documents in reference section

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Obesity and Cardiovascular Disease

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Top 10 Takeaway Messages: Obesity and Cardiovascular Disease (CVD)

1. Obesity adversely affects heart and vascular anatomy and function; CVD and cancer are the most common causes of mortality among patients with obesity.
2. Obesity increases the risk of CVD both directly (e.g., via the adiposopathic effects of epicardial fat), and indirectly via the adiposopathic promotion of major CVD risk factors such as diabetes mellitus, high blood pressure, dyslipidemia, and thrombosis.
3. While CVD outcomes trials are ongoing with anti-obesity agents, no drug and dose having an indication to treat obesity has proven to improve CVD outcomes; therefore, patients with obesity should undergo global CVD risk reduction (e.g., healthful nutrition and physical activity, smoking cessation, as well as optimal control of blood sugar, blood pressure, and blood lipids).
4. Obesity may increase pericardial (paracardial and epicardial) fat, intracardial fat, visceral fat, and liver and skeletal muscle fat; visceral and epicardial fat share the same mesodermal embryonic origin, both increase the risk for atherosclerosis, and both are highly correlated with coronary artery calcification.
5. Epicardial fat accumulation may directly contribute to heart failure with preserved ejection fraction (HFpEF), atherosclerotic CVD, dysrhythmias, fatty infiltration of the heart, and increased coronary calcium; HFpEF (diastolic heart failure) is especially common among patients with obesity, women, obstructive sleep apnea, older age, and CVD risk factors.

Top 10 Takeaway Messages: Obesity and Cardiovascular Disease (CVD)

6. Retrospective data suggests phentermine & topiramate may not increase the risk of major adverse cardiac events; glucagon-like peptide 1 agonists have clinical outcome trial evidence to support CVD benefits in patients with diabetes mellitus (e.g., liraglutide, semaglutide) and are being evaluated in CVD outcomes trials in patients with obesity.
7. Metformin and sodium glucose transporter (SGLT)-2 inhibitors decrease CVD among patients with diabetes mellitus; while they do not have an indication as anti-obesity agents, metformin and SGLT2 inhibitors modestly reduce body weight in patients with and without diabetes mellitus; when accompanied by weight loss, many anti-obesity drugs reduce CVD risk factors (i.e., orlistat, liraglutide, naltrexone/bupropion, and phentermine/topiramate are not contraindicated in patients with cardiovascular disease).
8. Among patients with obesity, CVD and type 2 diabetes mellitus without congestive cardiomyopathy, initial drug treatments to consider include metformin, liraglutide, and SGLT-2 inhibitors.
9. Among patients with obesity, CVD, type 2 diabetes mellitus with mild congestive cardiomyopathy, initial drug treatments to consider in include metformin and SGLT-2 inhibitors.
10. Among patients with obesity, CVD, and without type 2 diabetes mellitus and without congestive cardiomyopathy, initial treatments to consider include liraglutide.

Supporting documents in reference section

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Obesity and Elevated Blood Sugar

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Top 10 Takeaway Messages: Obesity and Diabetes Mellitus

1. The disease of obesity may promote hyperglycemia and the disease of type 2 diabetes mellitus
2. Diabetes mellitus is a major risk factor for CVD
3. CVD is the most common cause of morbidity and mortality among patients with obesity and diabetes mellitus (with or without obesity)
4. Patients with obesity and diabetes should undergo global CVD risk reduction (e.g., healthful nutrition and physical activity, smoking cessation, as well as optimal control of blood glucose, blood pressure, and blood lipids)
5. Sulfonylureas and many insulins may increase body weight and may increase the risk for CVD
6. Based upon cardiovascular outcome trial data of patients with type 2 diabetes mellitus (consisting mostly of patients with CVD), the anti-diabetes mellitus SGLT2 inhibitors (e.g., empagliflozin and canagliflozin) may reduce major adverse cardiac events (MACE), reduce heart failure, reduce cardiovascular death or heart failure hospitalization, reduce renal disease progression, and in some cases, reduce overall mortality. Body weight and blood pressure may be modestly decreased as well.
7. Liraglutide at the 1.8 mg dose to treat diabetes reduces CVD among patients with diabetes mellitus and reduces body weight and blood pressure.
8. Metformin decreases CVD among patients with diabetes mellitus, and modestly reduces body weight in patients with diabetes mellitus
9. Anti-obesity drugs do not have CVD outcome data to support improved CVD risk reduction; however, when accompanied by weight loss, many anti-obesity drugs reduce blood sugar and other CVD risk factors
10. Liraglutide lowers blood sugar through weight dependent and weight independent mechanisms

Obesity and High Blood Pressure

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Top 10 Takeaway Messages: Obesity and high blood pressure

1. The disease of obesity may promote an increase in blood pressure and the disease of hypertension
2. Hypertension is a major risk factor for CVD; CVD is the most common cause of mortality among patients with obesity and hypertension
3. Patients with obesity and hypertension should undergo global CVD risk reduction (e.g., healthful nutrition and physical activity, smoking cessation, as well as optimal control of blood glucose, blood pressure, and blood lipids)
4. In addition to food intake contributing to positive caloric balance, dietary sodium can also increase blood pressure
5. Obesity and “fat mass disease” can contribute to sleep apnea, kidney and renal vessel compression, perivascular adipose tissue (restricting blood vessel wall expansion) and increased cardiac output – all of which can increase blood pressure
6. Obesity promotes hyperleptinemia and hyperinsulinemia, both of which act upon the central nervous system to increase blood pressure
7. Obesity increases the renin-angiotensin aldosterone system (RAAS) activity in the kidney resulting in increased blood pressure
8. Obesity increases adiposopathic cytokines which increase endothelial dysfunction and increased arteriole vasoconstriction resulting in increased blood pressure
9. Obesity decreases the natriuretic effects of heart left ventricular B-type natriuretic peptide (BNP) resulting in increased blood pressure
10. When accompanied by weight loss, many anti-obesity agents decrease blood pressure; some anti-obesity agents may initially increase blood pressure (i.e., sympathomimetics such as phentermine) with possible longer-term reduction in blood pressure (compared to baseline) after weight loss

Obesity and Dyslipidemia

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Top 10 Takeaway Messages: Obesity and dyslipidemia

1. The disease of obesity is an important contributor to dyslipidemia
2. Dyslipidemia is a major risk factor for CVD; CVD is the most common cause of mortality among patients with obesity and dyslipidemia
3. Patients with obesity and dyslipidemia should undergo global CVD risk reduction (e.g., healthful nutrition and physical activity, smoking cessation, as well as optimal control of blood glucose, blood pressure, and blood lipids)
4. In addition to food intake contributing to positive caloric balance, ultra-processed carbohydrates may increase triglycerides (TGs) and reduce high density lipoprotein cholesterol (HDL-C). Saturated fats may increase low density lipoprotein cholesterol levels; however, this may depend on the effect of a carbohydrate restricted diet on body weight, and whether the patient has an underlying genetic hypercholesterolemia syndrome
5. Most of the body's energy is typically stored in the form of triglycerides in adipose tissue; the adipose tissue of patients with obesity may store over 50% of the total body cholesterol

Top 10 Takeaway Messages: Obesity and dyslipidemia

6. Lipoprotein lipase (LPL) is found on the intravascular surface of capillaries within and around body tissues (including adipose tissue), and hydrolyzes the core TG's contained in circulating TG-rich lipoproteins (e.g., very low density lipoproteins/VLDL and chylomicrons), into monoglycerides and fatty acids, thus reducing TG blood levels, and allowing for increased fatty acid transport into adipocytes; LPL is stimulated by physical exercise, insulin, fibrates, and omega-3 fatty acids
7. Hormone sensitive lipase (HSL) is located within adipocytes, and catalyzes the hydrolysis of diacylglycerol to monoacylglycerol, which is a rate limiting step in the release of free fatty acids (FFA) into the circulation. HSL is stimulated by catecholamines and inhibited by insulin. Largely through adipocyte HSL, lipolysis (i.e., adipocyte fat breakdown) is promoted by decreased insulin and increased physical activity (increased catecholamines), while lipolysis is inhibited (i.e., adipocyte fat conservation or gain) by increased insulin and decreased physical activity (decreased catecholamines)
8. The relative adiposopathic lack of uptake of FFA in peripheral subcutaneous adipose tissue (AT) results in energy overflow, with FFA deposition in abdominal subcutaneous, visceral AT, and pericardial AT, as well as FFA deposition in organs such as muscle and liver
9. Increased FFA delivery to the liver from adiposopathy and/or triglyceride-rich lipoproteins may lead to fatty liver and fatty muscle, possible "lipotoxicity" to these organs, increased circulating VLDL (with increased triglyceride levels), increased lipoprotein remnants, reduced HDL-C, and increased proportion of smaller, more dense LDL particles – all representing an adiposopathic dyslipidemia which is atherogenic
10. Weight loss decreases atherogenic apolipoprotein B-containing lipoproteins, and increases high density lipoprotein cholesterol

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Obesity and Nonalcoholic Fatty Liver Disease (NAFLD)

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Top 10 Takeaway Messages: Obesity & nonalcoholic fatty liver disease (NAFLD)

1. NAFLD includes the spectrum of fatty liver diseases, and is the most common cause of chronic liver disease (~25% of adults)
   ~45% Hispanics
   ~33% Caucasians
   ~24% Blacks
2. More than 2/3 of patients with NAFLD have obesity; NAFLD is a risk factor for cardiovascular disease
3. Hepatosteatosis is fatty liver; hepatosteatitis is fatty liver with inflammation. Nonalcoholic steatohepatitis (NASH) is the presence of ≥ 5% hepatic fat with inflammation and hepatocyte injury with or without fibrosis
4. Up to 30% of patients with NAFLD may have NASH. After 20-year follow-up, the risk of cirrhosis with hepatosteatosis is ~ 0 – 4%. After 9-year follow-up, the risk of cirrhosis with NASH = ~ 25%
5. NAFLD is an important cause of end stage liver disease, hepatocellular carcinoma and by 2020, may be the leading indication for liver transplant
6. While some drugs are suggested to improve NAFLD, no drug has an approved indication to treat NAFLD
7. Simple screening for hepatosteatosis includes otherwise unexplained elevated alanine transaminase (ALT) [and often elevated aspartate transaminase (AST)] in patients with obesity or type 2 diabetes mellitus
8. Among the more reliable and safe imaging tests for fatty liver include transient elastography and magnetic resonance imaging proton density fat fraction (MRI-PDFF) or mMR spectroscopy (MRS)
9. Excessive alcohol consumption is a common cause of fatty liver and cirrhosis. Conversely, common causes of NAFLD include obesity, adiposopathy, type 2 diabetes mellitus, insulin resistance, and some medications
10. Management of NAFLD includes treatment of secondary causes, appropriate nutrition and physical activity, and possibly peroxisome proliferator activated receptor gamma agonists, and glucagon-like protein-1 receptor agonists.

Obesity and Cancer

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Top 10 Takeaway Messages: Obesity and cancer

1. Obesity is the second most common preventable cause of cancer, and may soon overtake cigarette smoking as the most common preventable cause of cancer
2. Among US adults, the proportion of cancers attributable to excess body weight is ~ 5% for men, and ~10% for women; an increase in body weight may be contributing to an increase in cancer among young adults
3. No drug has an indication to both treat obesity and prevent or treat cancer
4. Adiposopathic consequences of obesity that promote cancer include adipose tissue cytokine production (e.g., tumor necrosis factor, interleukin-6) which may damage cellular DNA, promote gene mutations, enhance angiogenesis, promote cell proliferation and contribute to mitochondrial and endoplasmic reticulum stress, increasing reactive oxygen species (ROS) which may further damage cellular DNA
5. Additional adiposopathic immune processes that promote cancer include cytokine production, which may promote endothelial dysfunction, extracellular matrix abnormalities, and intravasation (rate limiting step of metastasis)
6. Adiposopathic endocrine processes that promote cancer include increased cancer promoting hormones, such as estrogens, leptin, androgens in women, and the growth hormones of insulin and insulin growth factor-1
7. Adiposopathic hypoxia processes that may promote cancer include growth of adipocytes and adipose tissue beyond their vascular supply, increasing immune and angiogenic responses, accelerating the growth and progression of cancer
8. Obesity, adiposopathy, cigarette smoking, and physical inactivity may promote oxidative stress, which is the imbalance in the creation of unstable ROS relative to the body's ability to detoxify these radicals (i.e., "antioxidants")
9. Beyond an increase in fat alone, among foods that may increase the risk of cancer are processed meats and cooking meats at high temperature; among foods that may decrease the risk of cancer are whole foods rich in phytochemicals, fiber, and antioxidants (e.g., citrus fruits, cruciferous and green leafy vegetables, legumes, nuts, whole grains, and some coffees and teas)
10. Among patients with obesity, weight reduction, as well as appropriate nutrition and physical activity may help prevent cancer, enhance chemotherapy for cancer, and reduce recurrent cancer

Supporting documents in reference section
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Obesity and Psychiatric Disease

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Top 10 Takeaway Messages: Obesity and Psychiatric Disease

1. Obesity and mood disorders frequently occur together
2. The relationship between obesity and depression is bidirectional. Obesity is a risk factor for mood disorders; mood disorders are a risk factor for obesity.
3. The association between depression and obesity may be stronger among women
4. Obesity and psychiatric diseases may share pathogenic pathways involving the immune and endocrine system, hypothalamic and pituitary axis, and nervous system (e.g., autonomic nervous system, monoamines, synapses, neurogenesis, and neuroinflammation)
5. Psychiatric diseases can sometimes independently contribute to overnutrition and/or consumption of foods rich in carbohydrates and fats
6. Individuals have unique body weight responses to medications used to treat psychiatric disease
7. Study populations support some psychiatric medications may generally increase body weight, while others may promote body weight neutrality or weight loss
8. Weight loss in patients with obesity may improve mood in patients without clinical depression
9. Non-surgical, intentional weight loss in patients with obesity may reduce symptoms of depression
10. Bariatric surgery often improves mental health conditions (e.g., depression and binge eating disorders); however, bariatric surgery is sometimes associated with recurring or new psychiatric disorders, alcohol or substance abuse, or eating disorders

Supporting documents in reference section

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Obesity Myths

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Obesity Myths

Common Obesity Myths

1. Obesity is not a disease; it is a lifestyle choice
2. An increase in body weight is always due to an increase in body fat
3. In patients with obesity, increased body fat is the cause of all their health conditions
4. Most people with increased body fat are generally healthy and will remain healthy
5. Increased subcutaneous adipose tissue is healthy; increased visceral adipose tissue is unhealthy
6. Obesity is due to eating too much versus obesity is not related to the caloric content of food
7. In the absence of a genetic or secondary medical cause, obesity is mostly due to a lack of willpower
8. Obesity is caused by eating processed foods
9. Obesity is caused by not eating breakfast versus caused by eating breakfast
10. Obesity is caused by a lack of breastfeeding as a child

Obesity Myths

Common Obesity Myths

11. Obesity is commonly caused by pathogens in the intestine (microbiome)
12. Lean people are “naturally skinny” because they have a higher metabolism
13. Low fat diets are the best way to lose body fat
14. Nutrition medical therapy is more effective when based upon patient preference
15. Increased physical exercise is the most efficient way to lose weight
16. Every pound of muscle that replaces fat burns an additional 50 calories per day
17. Access to exercise equipment, gym memberships, and physical activity trackers will cause weight loss
18. Setting more “realistic” obesity goals will ultimately achieve greater weight loss than more aggressive goals
19. Slow and gradual weight loss is ultimately more effective than large and rapid weight loss
20. Weight loss is difficult; maintaining weight loss is easy once the weight is lost
21. Drugs should not be used to treat obesity, because obesity is due to unhealthful diet and lack of exercise

Obesity Myth: Obesity is not a disease; it is a lifestyle choice

(See section entitled: The Disease of Obesity)

- The signs, symptoms, and pathophysiology of obesity fulfill the standard definition of a disease
- Obesity is often promoted by inheritance (genetic, epigenetic, and/or environmental inheritance)
- Obesity results in cellular and organ anatomic abnormalities
- Obesity results in cellular and organ functional abnormalities
- Obesity may result in pathogenic adipocyte and/or adipose tissue endocrine and immune dysfunctions that contribute to metabolic disease (adiposopathy or “sick fat disease”)
- Obesity may result in pathogenic physical forces from excessive body fat cause stress damage to other body tissues (“fat mass disease”)
- Even when exacerbated by unhealthful behavior, obesity is no less a disease than other diseases often promoted by unhealthful behavior

Investigational Anti-obesity Pharmacotherapy

Podcast
Obesity: A Disease
Episode 3

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Top 10 Takeaway Messages: Anti-Obesity Drug Development

1. Targets of current anti-obesity drug development are mainly focused on intervention pathways related to the central nervous system, gastrointestinal systems, and adipose tissue
2. Glucagon-like peptide receptor agonists (GLP-1 RA) increase satiety and slow gastric emptying
3. Oxyntomodulin acting agents have dual GLP-1 RA and glucagon RA activity
4. Setmelanotide is a melanocortin-4 receptor agonist
5. GLP-1 RA are being combined with other agents to create twincretins and tri-agonists (e.g., glucagon, glucose-dependent insulinotropic peptide, peptide YY, and amylin)
6. Agents that brown adipocytes have the potential to increase energy expenditure
7. Tesofensine is a triple monoamine reuptake inhibitor (e.g., inhibits dopamine, serotonin, and noradrenaline)
8. Sodium-linked glucose transporters - 2 (SGLT-2) inhibition decreases proximal renal tubule glucose reabsorption; SGLT-1 inhibition decreases gastrointestinal glucose absorption
9. With the exception of anti-obesity agents that are a combination of drugs in a single tablet or capsule, limited data exists for combination anti-obesity drug therapy
10. The development of anti-obesity pharmacotherapy is following the path of drug development of other metabolic diseases

Supporting documents in reference section

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Early versus Late Weight-Management Intervention: Illustrative Consequences

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Early Treatment/Prevention

44-year-old woman with overweight/obesity

- Pre-diabetes mellitus
- Pre-hypertension
- Mild dyslipidemia
- Discomfort to weight-bearing joints
- Mild snoring
- Low self-esteem due to increased body weight

Optimal Treatment Strategy

Decide to engage in early, proactive interventions intended to *prevent* onset of adverse health consequences from sick fat disease (diabetes mellitus, dyslipidemia, and hypertension) and fat mass disease (osteoarthritis):

- Optimize nutritional therapy and physical activity
- Initiate behavioral therapy
- Consider anti-obesity medications
- Consider bariatric surgery

Prevent onset of metabolic disease:

- Diabetes mellitus
- Dyslipidemia
- Hypertension

Prevent fat mass diseases:

- Osteoarthritis
- Sleep apnea
- Depression

Supporting documents in reference section

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Delayed Treatment

44-year-old woman with overweight/obesity

- Pre-diabetes mellitus
- Pre-hypertension
- Mild dyslipidemia
- Discomfort to weight-bearing joints
- Mild snoring
- Low self-esteem due to increased body weight

Sub-optimal Treatment Strategy

Simply tell the patient to diet and exercise and otherwise wait for the onset of diabetes mellitus, dyslipidemia, hypertension, osteoarthritis, sleep apnea, and depression. Once adverse health consequences are blatantly apparent:

- Optimize nutritional therapy and physical activity
- Initiate behavioral therapy
- Consider anti-obesity medications
- Consider bariatric surgery

Continued…

Delayed Treatment

If optimal intervention for obesity treatment and prevention is delayed, and the patient develops adverse consequences:

- Follow diabetes mellitus evaluation and treatment guidelines
  - American Diabetes Association Standards of Medical Care in Diabetes
  - American Association of Clinical Endocrinology Comprehensive Diabetes Management Algorithm
- Follow lipid evaluation and treatment recommendations and guidelines
  - AHA/ACC/AACVPR/AAPA/ABC/ACPM/ADA/AGS/APhA/ASPC/NLA/PCN Guideline on the Management of Blood Cholesterol
- Follow blood pressure guidelines
  - Report of the Joint National Committee for Management of High Blood Pressure in Adults
- Follow other disease-specific guidelines
- Utilize diabetes mellitus therapies most likely to improve adipose tissue function
- In patients with fat mass disease, utilize diabetes mellitus therapies having neutral or body weight loss effects, such as metformin, glucagon-like peptide-1 (GLP-1) agonists, sodium glucose contransporter-2 (SGLT2) inhibitors
- Utilize lipid therapies most likely to reduce atherosclerotic coronary heart disease risk and least likely to increase body weight (e.g., statins)
- Utilize blood pressure therapy most likely to reduce cardiovascular disease risk, which may also provide other health benefits (e.g. diuretics, angiotensin converting enzyme inhibitors, etc.)
- Utilize non-steroidal anti-inflammatory agents to treat osteoarthritis
- Treat sleep apnea
- Utilize anti-depressant medications least likely to promote further weight gain
- Administer additional pharmaceuticals and/or treatment modalities as indicated

Bariatric Surgery

Physiology, Procedures, Micronutrients, Microbiome, Complications

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Top 10 Takeaway Messages: Gastrointestinal (GI) Hormones

1. GI hormones play a critical role in regulation of caloric balance, food digestion, and nutrient utilization via central nervous system signaling, effects on gastrointestinal motility, and enzyme release
2. Common GI hormone action in response to eating include decrease in hunger/appetite 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 appetite/increase satiety.
6. Among the few GI hormones that increase appetite 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

Supporting documents in reference section

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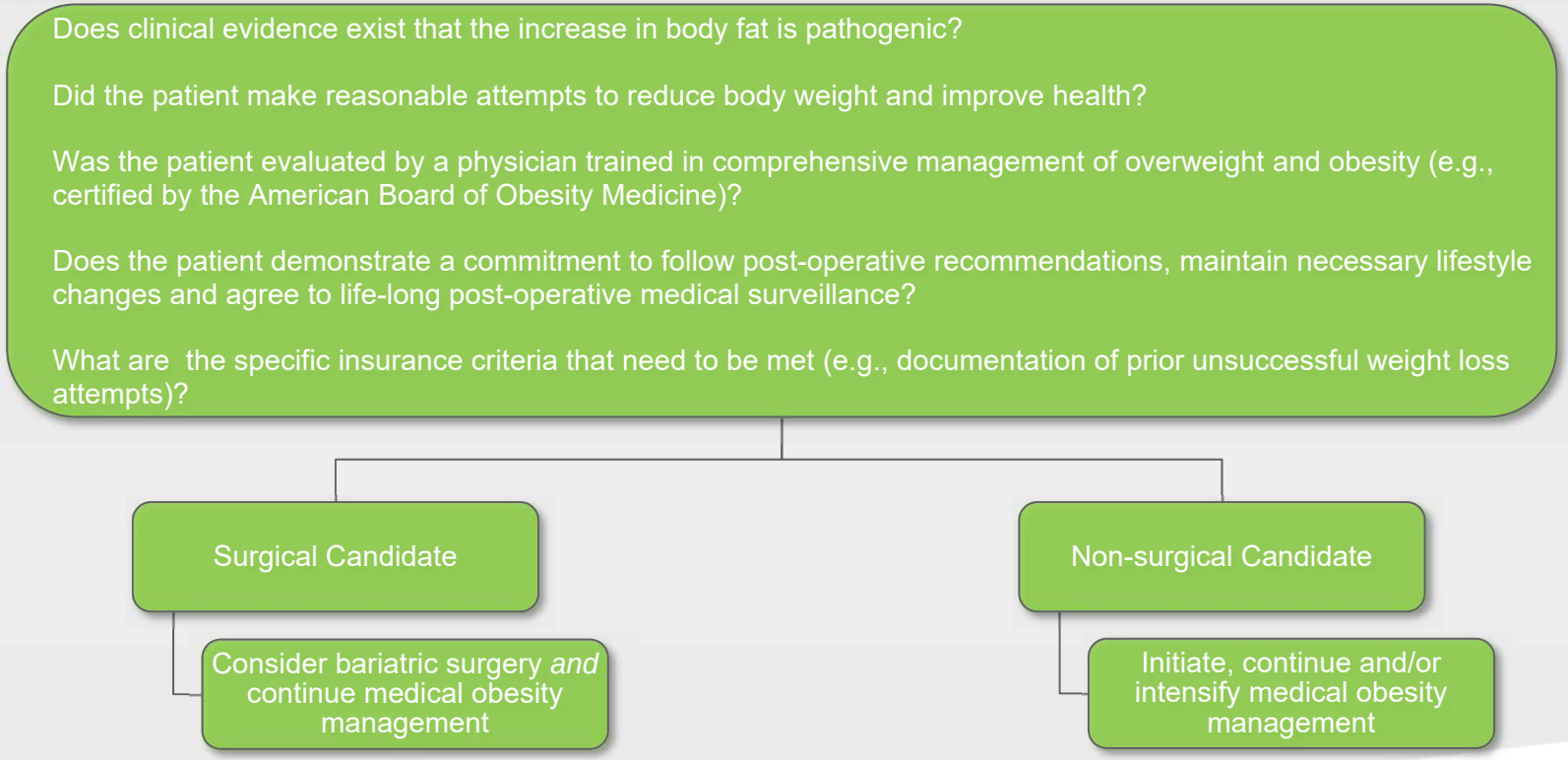
Top 10 Takeaway Messages: Bariatric Surgery

1. The two most common bariatric 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 connecting the jejunum to a proximal gastric pouch, with most of the stomach excised ("bypassed"); the distal duodenum is re-attached to a lower portion of the jejunum
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 (abdominal CT or upper GI study); 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 area
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, cardiac dysrhythmias, atelectasis and pneumonia, deep vein thrombosis, and pulmonary emboli

Potential Bariatric Surgery Patient



Supporting documents in reference section

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Bariatric Surgical Procedures

	Pros	Cons	Expected loss in percent excess body weight* at two years	Optimally suited for patients with:	Other comments
Roux-en-Y Gastric Bypass	Greater improvement in metabolic disease	Increased risk of malabsorptive complications over sleeve	60-75%	Higher BMI, GERD, Type 2 DM	Largest data set, more technically challenging than LAGB, VSG
Vertical Sleeve Gastrectomy	Improves metabolic disease; maintains small intestinal anatomy; micronutrient deficiencies infrequent	No long term data	50-70% (*3-year data)	Metabolic disease	Can be used as the first step of staged approach; most common based on 2014 data
Laparoscopic Adjustable Gastric Banding	Least invasive; removable	25-40% 5 year removal rate internationally	30-50%	Lower BMI; no metabolic disease	Any metabolic benefits achieved are *dependent* on weight loss
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

*Excess body weight (EBW) = (total body weight) - (lean body weight)

Top 10 Takeaway Messages: Bariatric Surgery Nutrient Considerations

1. While biliopancreatic diversion with duodenal switch may result in the greatest amount of weight loss, it is a procedure that has a high rate of multiple post-procedure vitamin and mineral deficiencies.
2. Common vitamin and mineral deficiencies after gastric bypass and sleeve gastrectomy include deficiencies of vitamins B1 (thiamine), B9 (folate), B12 (cyanocobalamin), and D, as well as deficiencies of the minerals iron and calcium (gastric bypass); other vitamin and mineral deficiencies are more rarely reported
3. Lower levels of vitamin D are often found pre-operatively in patients with obesity
4. High-quality multivitamins are routinely recommended after bariatric procedures, with supplements often containing higher amounts of vitamin B12, iron, vitamin C (to assist with iron absorption), vitamin D, and calcium
5. Vitamin B1 (thiamine) deficiency can cause “dry” beriberi (e.g., Wernicke-Korsakoff encephalopathy) and “wet” beriberi (e.g., congestive heart failure)
6. Vitamin B9 (folate) deficiency can cause megaloblastic anemia
7. Vitamin B12 (cyanocobalamin) deficiency can cause megaloblastic anemia and nervous system disorders
8. Vitamin D deficiency can cause osteopenia, secondary hyperparathyroidism, and hypocalcemia
9. Calcium deficiency can cause osteopenia, secondary hyperparathyroidism, and hypocalcemia
10. Iron deficiency can cause microcytic anemia

Supporting documents in reference section

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Bariatric Surgery: Common Micronutrient Deficiencies

	Vitamins							Minerals		
	A	B1	B9	B12	D*	E	K	Ca	Fe	Zn/Cu
RNY		X	X	X	X			X	X	
Sleeve		X	X	X	X				X	
LAGB		X			X					
BPD	X	X	X	X	X	X	X	X	X	X

*Vitamin D deficiency is seen in a significant number of patients with obesity at baseline. However, due to malabsorption, the risk is further increased post-operatively.

Nutritional Principles Following Bariatric Surgery

- Nutritional advice will depend upon type of bariatric procedure
- Initially three to five small meals a day, with decrease in meal number as portion size increases
- Chew small bites of food thoroughly
- Avoid consuming liquids during meals, delay for at least 30 minutes after meals
- Protein: At least 60 grams/day, optimally 1.2 to 1.5 grams/kg/day of lean mass – avoid excessive calorie intake
- Avoid concentrated sweets to minimize dumping (i.e., procedures such as gastric by-pass) and to reduce caloric intake
- High-quality multivitamins are routinely recommended after bariatric procedures, irrespective of deficiencies, which are often recommended to be chewable or liquid
- Other routine supplements often include:
  - Vitamin B12 500 µg/d tablet or sublingual, or 1000 µg/mo IM
  - Iron at least 27 mg of elemental iron daily, given with at least 500 mg vitamin C
  - Calcium citrate 1200 mg/d, preferably with vitamin D3

Top 10 Takeaway Messages: Microbiome

1. The microbiome is a collection of micro-organisms; microbiota are the organisms themselves
2. The human organism has ~10 trillion human cells; the human gut is colonized by ~100 trillion cells (bacteria, fungi, and viruses)
3. Among gut bacteria species, over 90% are anaerobic that utilize the substrate of sloughed intestinal cells, plant polysaccharides, starch cellulose, and bile components
4. The phyla Gram positive Firmicutes and gram negative Bacteroidetes make up about 90% of the bacteria in the large intestine; in obesity, Firmicutes is proportionally increased compared to Bacteroidetes
5. Some intestinal bacteria promote increased density of small intestinal villi capillaries and more efficient absorption of nutrients than other bacteria; gram positive Firmicutes may more efficiently extract calories from carbohydrates than Gram negative Bacteroidetes
6. Some intestinal bacteria may also facilitate central nervous system and other body organ signaling that may influence energy balance
7. Intestinal bacteria can also influence bile acid metabolism and gut hormone secretion, both of which can affect energy balance
8. Bariatric surgery may alter the microbiome, and reduce the efficiency of extracting calories from consumed carbohydrates
9. Individuals who are overweight or with obesity may not benefit from microbiota that promote more efficient absorption of nutrients
10. The effects of fecal transplant in humans with obesity differ compared to rodents, with some fecal microbiota transplantation to patients (humans) with obesity not leading to a reduction in body mass index, despite successful and sustained changes in the intestinal microbiome and bile acid profiles similar to the lean donor

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Executive Summary

Obesity Medicine Association

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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.

Supporting documents in reference section

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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.

Supporting documents in reference section

Obesity Medicine Association

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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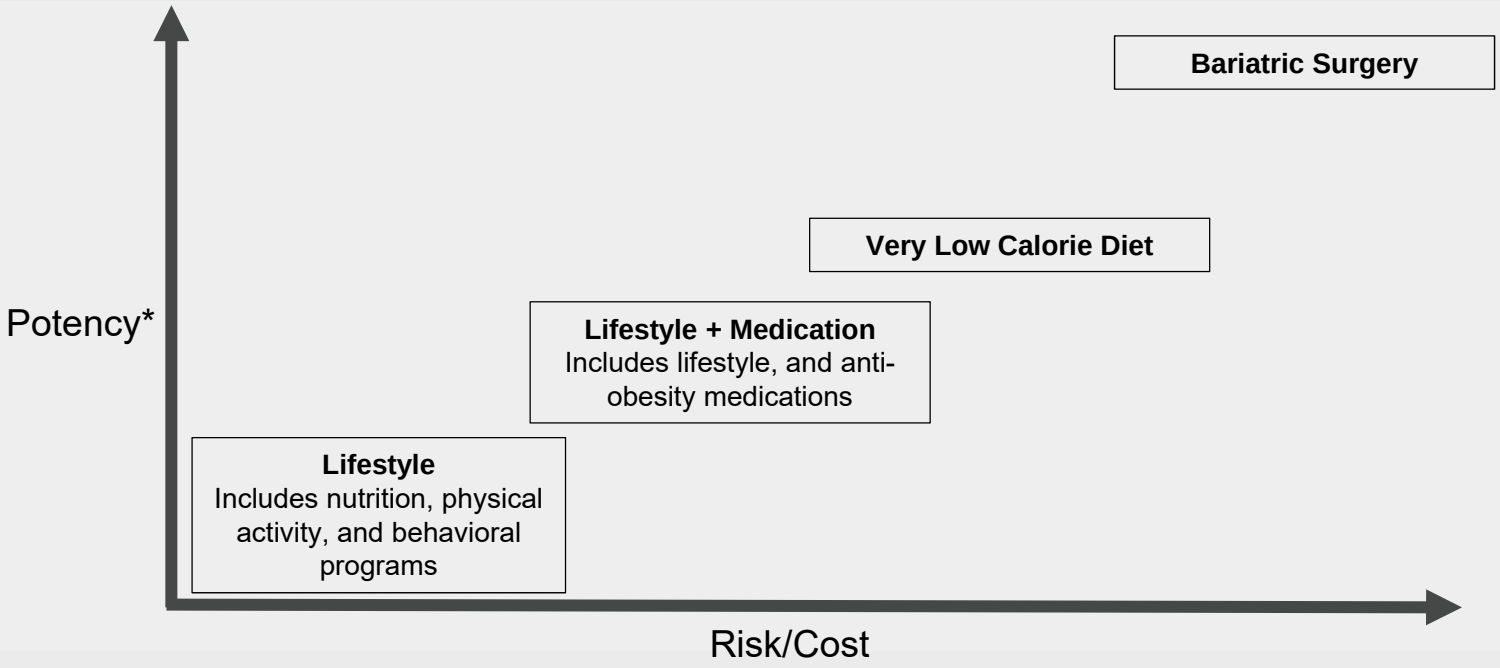
Current Treatment Options for Obesity



*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).

Supporting documents in reference section

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