Obesity Algorithm®

2019

obesitymedicine.org

Obesity Medicine Association

CLINICAL LEADERS IN OBESITY MEDICINE®

Table of Contents*

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

Supporting documents in reference section

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Purpose

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

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Intent of Use

- The Obesity Algorithm is intended to be a “living document” updated once a year (as needed). It is intended to be an educational tool used to translate the current medical science and the experiences of obesity specialists to better facilitate and improve the clinical care and management of patients with overweight and obesity.

- This algorithm ***is not*** intended to be interpreted as “rules” and/or directives regarding the medical care of an individual patient.

- While the hope is many clinicians may find this algorithm helpful, the final decision regarding the optimal care of the patient with overweight and obesity is dependent upon the individual clinical presentation and the judgment of the clinician who is tasked with directing a treatment plan that is in the best interest of the patient.

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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 these guidelines must be made in light of local resources, individual patient circumstances, and in partnership with patient preferences.

Permissions

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Major Updates Included in the 2019 Version

- Obesity and cardiovascular disease
- Obesity and diabetes mellitus
- Obesity and dyslipidemia
- Obesity and cancer
- Expansion of investigational anti-obesity pharmacotherapy
- Treatments for lipodystrophy
- Pharmacokinetics and obesity
- General updates and text edits
- Updated references

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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, Wells S, Long J, Shah NN, Primack C. Obesity Algorithm eBook, presented by the Obesity Medicine Association. www.obesityalgorithm.org. 2019. 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, Seger J, Wells S, Long J, Shah NN, Primack C. Obesity Algorithm Slides, presented by the Obesity Medicine Association. www.obesityalgorithm.org. 2019. 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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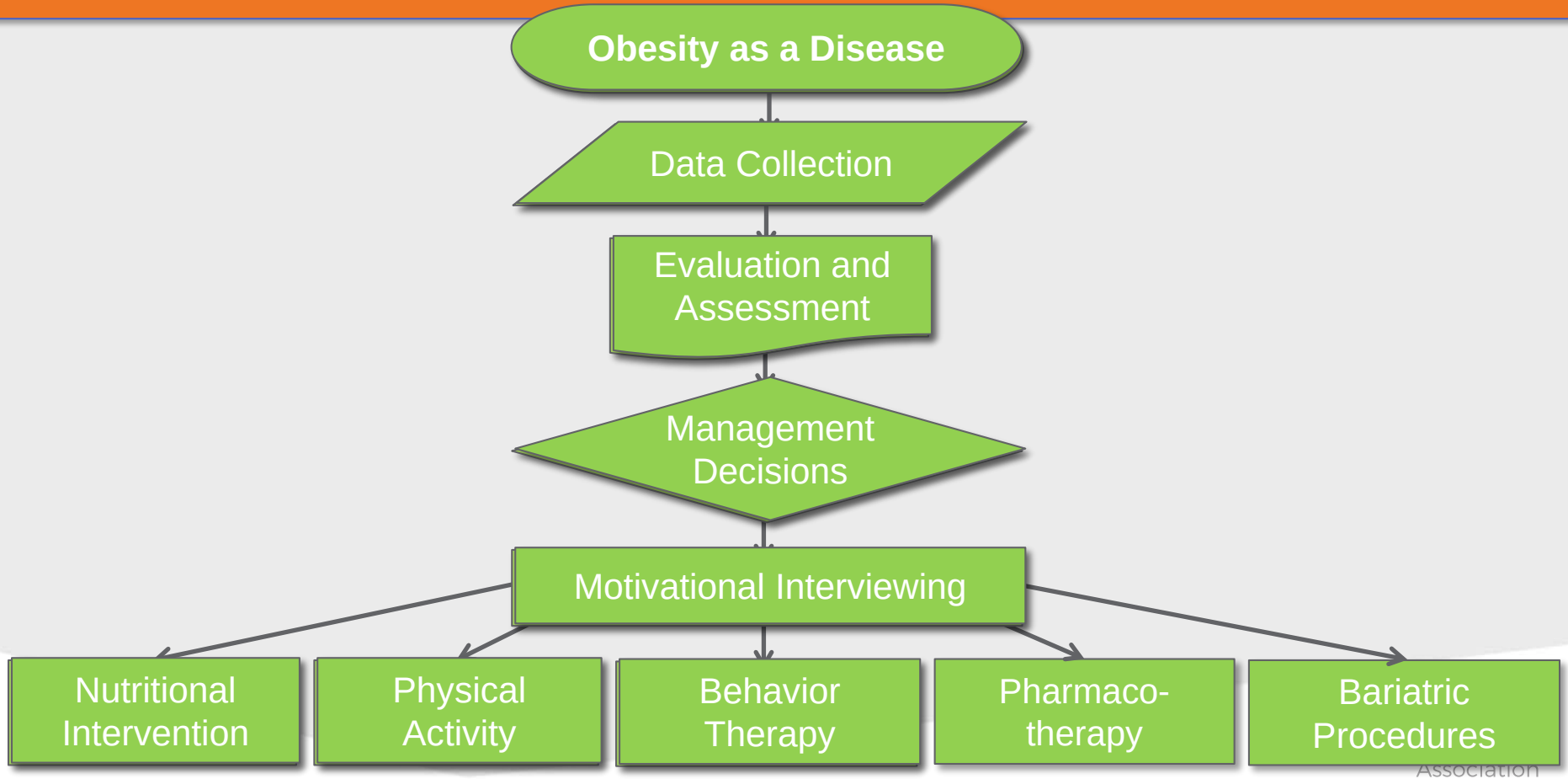
The Disease of Obesity

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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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 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

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Obesity Health Care Office Environment

Clinicians and staff should be trained to avoid hurtful comments, jokes, or being otherwise disrespectful, as patients with obesity may be ashamed or embarrassed about their weight.

Positive Office Space

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

Appropriate Medical Devices

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

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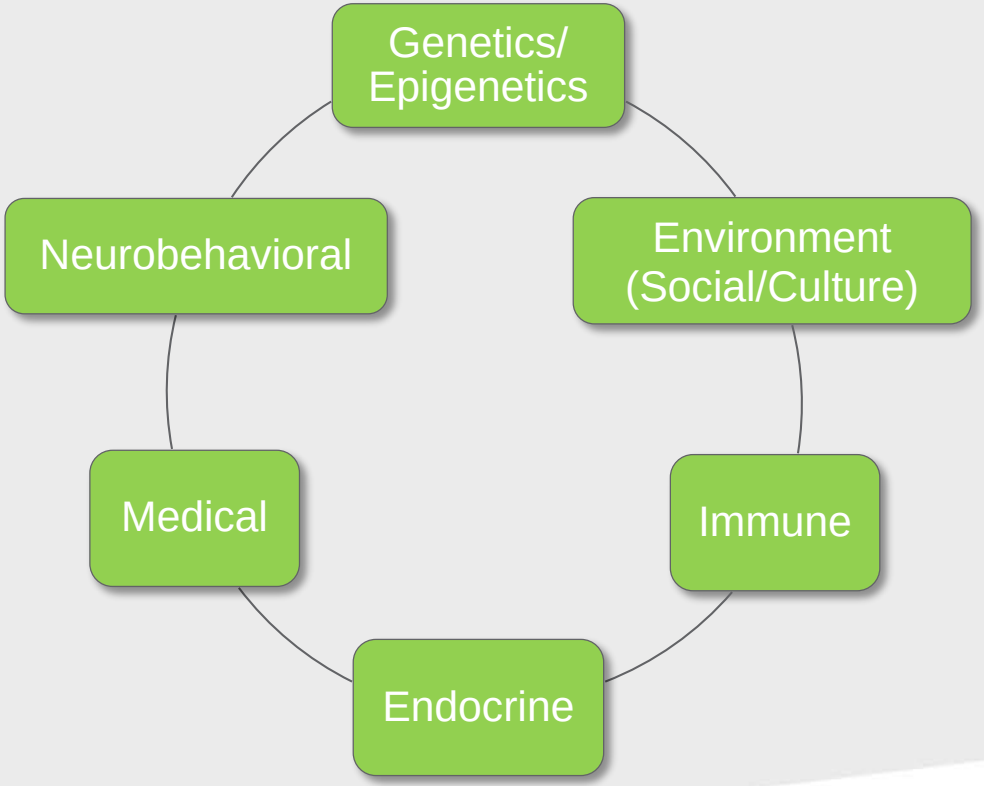
Obesity is a Multifactorial Disease



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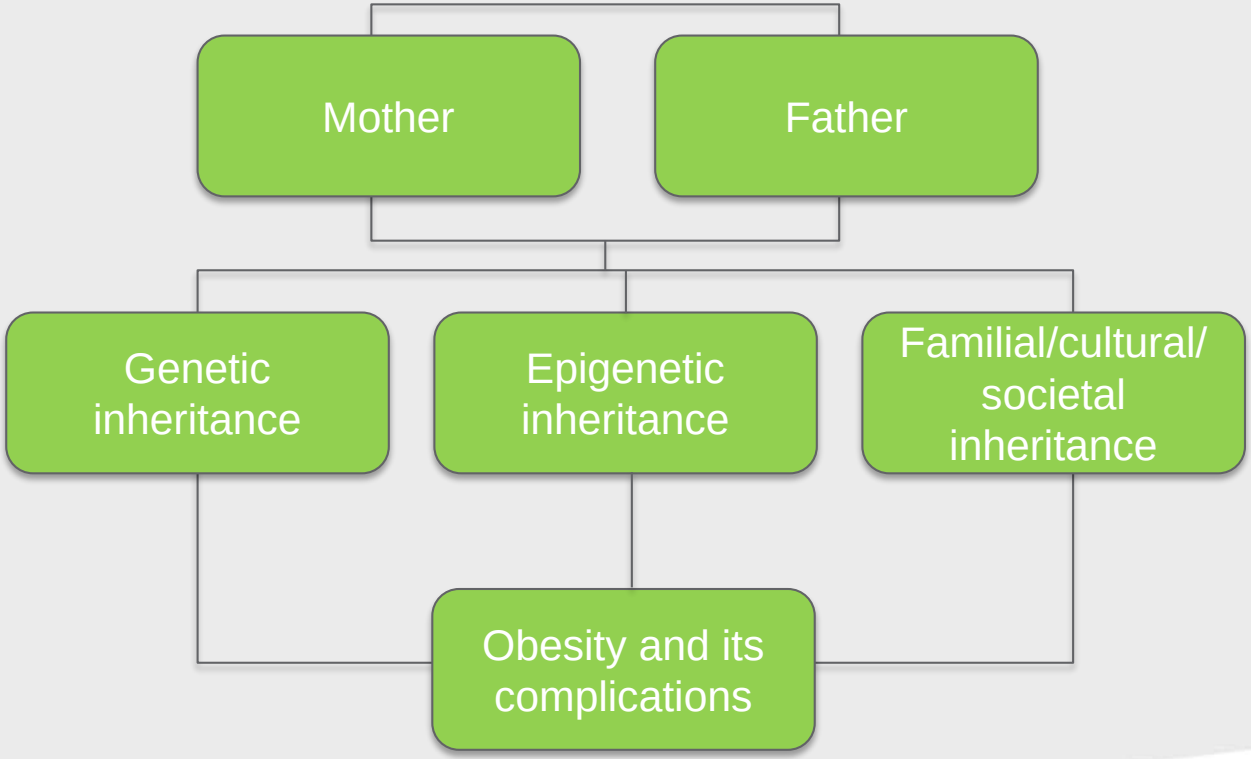
Multifactorial Inheritance Factors Contribute to Obesity



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Obesity: Extragenetic Etiology/Causes

Extragenetic

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

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Obesity: Epigenetic Etiology/Causes

Epigenetics: Alterations in gene expression without alteration in the genetic code

Pre-pregnancy

- Pre-conception paternal or maternal overweight/obesity may influence epigenetic signaling during subsequent pregnancy:
  - Increased risk of overweight/obesity in offspring
  - Increased risk of other diseases (e.g., cardiovascular disease, cancer, diabetes mellitus, etc.) in offspring

Pregnancy

- Especially in the presence of gestational diabetes mellitus, unhealthy maternal nutrition in women who are pregnant and overweight or with obesity may increase placental nutrient transfer to fetal circulation:
  - Glucose
  - Lipids and fatty acids
  - Amino acids
- Increased maternal nutrient transport may alter fetal gene expression:
  - Covalent modifications of deoxynucleic acid and chromatin
  - May impact stem cell fate
  - May alter postnatal biologic processes involved in substrate metabolism
  - May increase offspring predisposition to overweight/obesity and other diseases

Post-pregnancy

- Adverse effects of epigenetic pathologies may help account for generational obesity
- Improvement in generational obesity in offspring will likely require generational change in nutrition and physical activity in prior generations of parents

Within Subsets of Patients with Overweight and/or Obesity

Deranged endocrine and immune responses

↓

Sick Fat Disease (SFD) (Adiposopathy)

Endocrine/metabolic:
- Elevated blood glucose
- Elevated blood pressure
- Dyslipidemia
- Other metabolic diseases

Abnormal and pathologic physical forces

↓

Fat Mass Disease (FMD)

Biomechanical/structural:
- Stress on weight-bearing joints
- Immobility
- Tissue compression (i.e., sleep apnea, gastrointestinal reflux, high blood pressure, etc.)
- Tissue friction (i.e., intertrigo, etc.)

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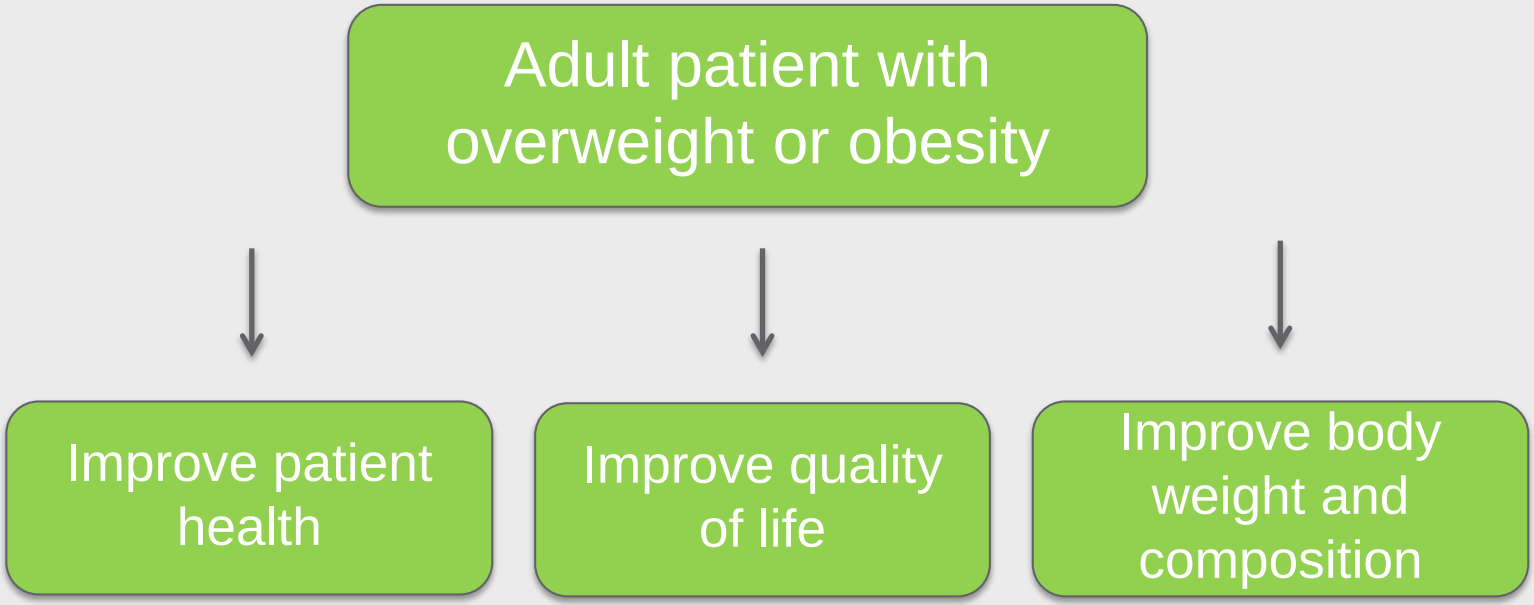
Overall Management Goals



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Classification of Obesity

Body Mass Index: Increase Body Fat (Adiposity)

Body mass index (BMI) in kilograms per meters squared (kg/m^2)*

Normal Weight	Overweight	Class I Obesity	Class II Obesity	Class III Obesity
18.5-24.9	25.0-29.9	30.0-34.9	35.0-39.9	≥ 40

*Different BMI cut-off points may be more appropriate based upon gender, race, ethnicity, and menopausal status. For example, some suggest a BMI >23 kg/m^2 may be a more appropriate cut-off point to screen for type 2 diabetes mellitus among Asians, and BMI may underestimate percent body fat in postmenopausal women.

Percent Body Fat: American Council on Exercise Classification

American Council on Exercise Classification:
Percent body fat*

Essential Fat	Athletes	Fitness	Acceptable	Obesity
Women: 10-13% Men: 2-5%	Women: 14-20% Men: 6-13%	Women: 21-24% Men: 14-17%	Women: 25-31% Men: 18-24%	Women: ≥ 32% Men: ≥ 25%

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

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Waist Circumference: Increased Body Fat (Adiposity)

Obesity classification:
Waist circumference (WC)*

Abdominal Obesity - Men
≥ 40 inches
≥ 102 centimeters

Abdominal Obesity - Women
≥ 35 inches
≥ 88 centimeters

*Different WC abdominal obesity cut-off points are appropriate for different races (e.g., ≥ 90 centimeters for Asian men and ≥ 80 centimeters for Asian women)

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Obesity: Summary Diagnostic Metrics and Diagnostic Codes

Body Mass Index
≥ 30 kg/m²

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

Abdominal Obesity: Women
≥ 35 inches
≥ 88 centimeters

Abdominal Obesity: Men
≥ 40 inches
≥ 102 centimeters

Overweight and Obesity E66

- Code first obesity complicating pregnancy, childbirth and the puerperium, if applicable (O99.21-)
- Use additional code to identify body mass index (BMI), if known (Z68.-)

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

E66 Overweight and obesity*

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

* Coding choice should not only accurately reflect the diagnosis, but also consider the impact on patients who may read the diagnosis in their medical records (i.e., codes including the terms "morbid obesity," and/or perhaps even "excess calories")

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

BMI*	Total Body Fat	< 25 kg/m^2	25 – 29 kg/m^2	30 - 34 kg/m^2	>= 35 kg/m^2
Men % Body Fat (mean)	28%	23%	28%	32%	37%
Women % Body Fat (mean)	40%	34%	41%	44%	48%

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

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

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

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Which Is the “Best” Measure of Obesity?

Population Assessment

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

Individual Assessment

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

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Fat Mass Disease:

Abnormal and Pathologic Physical Forces

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Clinical Manifestations: Fat Mass Disease

Cardiovascular

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

Pulmonary

- Dyspnea
- Obstructive sleep apnea
- Hypoventilation/Pickwickian syndrome
- Asthma

Neurologic

- Intracranial hypertension (pseudotumor cerebri) due to increased intra-abdominal pressure and sleep apnea, with impaired central venous return.
- Stroke (see “cardiovascular”)
- Nerve entrapment (i.e., meralgia paresthetica, carpal tunnel syndrome)

Clinical Manifestations: Fat Mass Disease

Musculoskeletal

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

Gastrointestinal

- Gastroesophageal reflux
- Hernias

Integument

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

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Clinical Manifestations: Fat Mass Disease

Psycho-Social

- Depression
- Hopelessness
- Low self-esteem
- Body-image dissatisfaction
- Diminished sex drive
- Impaired intimacy and sexual relationships
- Decreased work productivity
- Increased work absenteeism

General Biases

- Society
- Family
- Workplace
- Harassment
- Bullying

Heath Care Bias

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

Negative Self or External Perceptions

- “Unmotivated”
- “Weak-willed”
- “Less intelligent”
- “Less attractive”
- “Unsuccessful”
- “Overindulgent”
- “Lazy”

Weight bias internalization

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

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

Metabolic Manifestations of Adiposopathy

- High blood glucose (prediabetes mellitus, type 2 diabetes mellitus)
- High blood pressure
- Metabolic syndrome
- Adiposopathic dyslipidemia
  - Increased triglyceride levels
  - Decreased high-density lipoprotein cholesterol levels
  - Increased atherogenic particle number (increased apolipoprotein B)
  - Increased proportion of small, dense, low-density lipoprotein particles
  - Increased triglyceride-rich lipoproteins
  - Increased lipoprotein-remnants
- Insulin resistance
- Hepatosteatosis (fatty liver)
- Hyperuricemia and gout
- Cholelithiasis
- Acanthosis Nigricans
- Nephrolithiasis
- Glomerulopathy
- Pro-thrombotic predisposition
- Neuropsychiatric diseases (such as worsening depression or loss of gray matter due to adiposopathic immune and endocrine responses)
- Asthma (due to adiposopathic immune and endocrine responses)
- Worsening of other inflammatory diseases (osteoarthritis, atherosclerosis, etc.)

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Gender-specific Manifestations of Adiposopathy

Women

- Hyperandrogenemia
- Hirsutism
- Acne
- Polycystic ovarian syndrome
- Menstrual disorders
- Infertility
- Gestational diabetes mellitus
- Preeclampsia
- Thrombosis

Men

- Hypoandrogenemia
- Hyperestrogenemia
- Erectile dysfunction
- Low sperm count
- Infertility

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Obesity and Adiposopathy Increases the Risk of Cancers

- Bladder cancer
- Brain cancer
- Breast cancer (postmenopausal)
- Cervical cancer
- Colon cancer
- Endometrial/uterine cancer
- Esophageal cancer
- Gallbladder cancer
- Head and neck cancer
- Kidney/renal cancer
- Leukemia
- Liver cancer
- Multiple myeloma
- Non-Hodgkin lymphoma
- Ovarian cancer
- Pancreatic cancer
- Prostate cancer (prognosis is worse, not necessarily increased risk)
- Stomach cancer
- Thyroid cancer

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Adiposopathic and/or Fat Mass Pathologies:
Genitourinary and Reproductive Manifestations

Genitourinary

- Urinary stress incontinence
- Pelvic prolapse (e.g. cystocele, rectocele, uterine prolapse, vault prolapse)

Reproductive Pre-pregnancy

- Men
  - Buried or hidden penis
  - Erectile dysfunction
  - Psychological barriers to sexual behavior
  - Infertility
- Women
  - Psychological barriers to sexual behavior
  - Infertility, anovulation, polycystic ovary syndrome

Reproductive Pregnancy

- Gestational diabetes mellitus
- Preeclampsia
- Increased risk of miscarriage and stillbirth
- Overdue pregnancy
  - Increased need for induction
  - Increased need and complications of cesarean section in women (delayed healing and wound infection)
- Large for gestational age offspring
- Thrombosis
- Obstructive sleep apnea

Obesity Paradox

Obesity Paradox

ANATOMIC OBESITY PARADOX

- Are some fat depots protective while others are “paradoxically” pathogenic?

PHYSIOLOGIC OBESITY PARADOX

- Are some individuals who are overweight or with obesity “paradoxically” healthy?
- Do some individuals who are normal weight, or only mildly overweight, “paradoxically” have metabolic disease?

DEMOGRAPHIC (GENDER AND RACE) PARADOX

- Are women at a “paradoxically” lower age-adjusted cardiovascular disease risk than men?
- Are some races “paradoxically” at increased risk for metabolic diseases for the same amount of body weight?

THERAPEUTIC OBESITY PARADOX

- Can adding body fat “paradoxically” treat metabolic diseases typically associated with too much body fat?
- Does an increase in fat mass always predispose to metabolic disease?
- Does a decrease in fat mass always improve metabolic disease?

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Obesity Paradox

CARDIOVASCULAR OUTCOMES OBESITY PARADOX

- Why are individuals who are modestly overweight often “paradoxically” reported to have a better prognosis after cardiovascular disease (CVD) events and cardiovascular procedures?

STROKE OBESITY PARADOX

- Why do individuals with obesity “paradoxically” seem to have a better outcome with stroke?

ACUTE RESPIRATORY DISTRESS OBESITY PARADOX

- Why do individuals with obesity “paradoxically” seem to have better outcome with acute respiratory distress?

KIDNEY DISEASE OBESITY PARADOX

- Why do patients with chronic kidney disease and increased body mass index “paradoxically” have lower risk of end-stage renal disease and death?

THERAPEUTIC APPROACH OBESITY PARADOX

- How do clinicians best navigate the apparent paradox of “blame” versus “accountability” in obesity management?

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Obesity Paradox: General Concepts

- Obesity increases morbidity and mortality
- Obesity may be more closely correlated with increased mortality if maximum body mass index (BMI) is assessed rather than a single baseline BMI
- Obesity increases morbidity, including increased risk of “fat mass” diseases and “sick fat” diseases (e.g., metabolic diseases such as diabetes mellitus, hypertension, and dyslipidemia - all major cardiovascular disease risk factors)
- Obesity increases risk of cardiovascular disease even without major metabolic cardiovascular risk factors
- Individuals with the highest body weight and lowest body weight have higher mortality
- The increase in mortality with lowest body weight is often due to the confounding effect of concurrent illnesses that not only contribute to low body weight, but also to increased mortality
- Obesity increases the risk of cancer
- More than one “obesity paradox” exists
- Obesity paradoxes are less paradoxical when viewed from the perspective of both fat mass ***and*** fat function

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Obesity and Stress:
Cause and Effect

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Psychological or Medical Stress

Stress Responses

- Cognitive changes
  - Increased (e.g., some cases of emergent stress)
  - Decreased (e.g., some cases prolonged stress)
- Physiological changes
- Behavioral changes
- Pain
  - Potential analgesia with emergent stress
  - Potential worsening of pain with chronic stress

Psychological or Medical Stress: Endocrine Response

Emergent *"Fight or Flight"* Response

(Increased Sympathomimetic Activity)

- Increase in short-term sympathetic nervous system activation
- Increased catecholamines (e.g., norepinephrine and epinephrine)
- Cardiovasculopulmonary responses
  - Increased blood pressure
  - Vasoconstriction
  - Increased heart rate and contractility
  - Impaired blood flow to kidney
  - Bronchial dilation
- Short-term metabolic responses
  - Potential increase in glucose levels (increased insulin resistance, increased hepatic glycogenolysis, and increased hepatic gluconeogenesis)
  - Increased adipose tissue lipolysis

Psychological or Medical Stress: Endocrine Response

Chronic *"Submit and Stay"* Response

(Increased hypothalamic pituitary axis activity)

Increased Longer-term Stress Hormone Release

- Increased corticotropin-releasing hormone
- Increased adrenocorticotropin
- Increased arginine, vasopressin, and oxytocin
- Increased blood cortisol

Metabolic Responses to Increased Cortisol

- Potential increase in glucose levels (increased insulin resistance and increased hepatic gluconeogenesis)
- Increased blood pressure
- Increased food craving with increased body fat
- Increased adipose tissue lipolysis and muscle tissue wasting (cortisol is a catabolic hormone), with disproportionate accumulation of abdominal fat

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Medical or Psychological Stress: Immune Response

Acute Response (Catecholamine-mediated)

- Immune effects can be mixed, but in general, acute stress response may enhance immune response:
  - Demargination of leukocytes from vascular endothelia increases leukocyte blood concentration
  - Increased:
    - Innate immune response
    - Adaptive immune response
    - T-lymphocyte cytokine response

Prolonged Response (Glucocorticoid-mediated)

- Prolonged stress response may dysregulate immune response:
  - Increase total white blood cell count suggesting increased systemic inflammation
  - May decrease antibody-producing B lymphocytes with decreased:
    - Innate immune response
    - Adaptive immune response
    - T-lymphocyte cytokine response

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Chronic Psychological Stress and Eating Behavior

Limbic System

(Thalamus, hypothalamus, amygdala, hippocampus)

- Chronic stress-induced endocrinopathies and immunopathies may adversely affect the limbic system
- Hypothalamic dysfunction (such as with trauma) is an important cause of obesity

Cerebrum

(Frontal, parietal, occipital, and temporal lobes)

- Priority replacement: personal, work, or emotional priorities may overtake priorities relative to nutrition, physical activity, and/or health
- Chronic stress-induced endocrinopathies and immunopathies may adversely affect the cerebrum
- Gourmand Syndrome
  - While not necessarily a stress disorder, Gourmand Syndrome is illustrative of how cerebral disorders may affect eating behaviors
  - Occurs with damage to right frontal lobe (trauma/stroke)
  - Post-injury passion for gourmet foods

Enhanced desire for hyperpalatable foods

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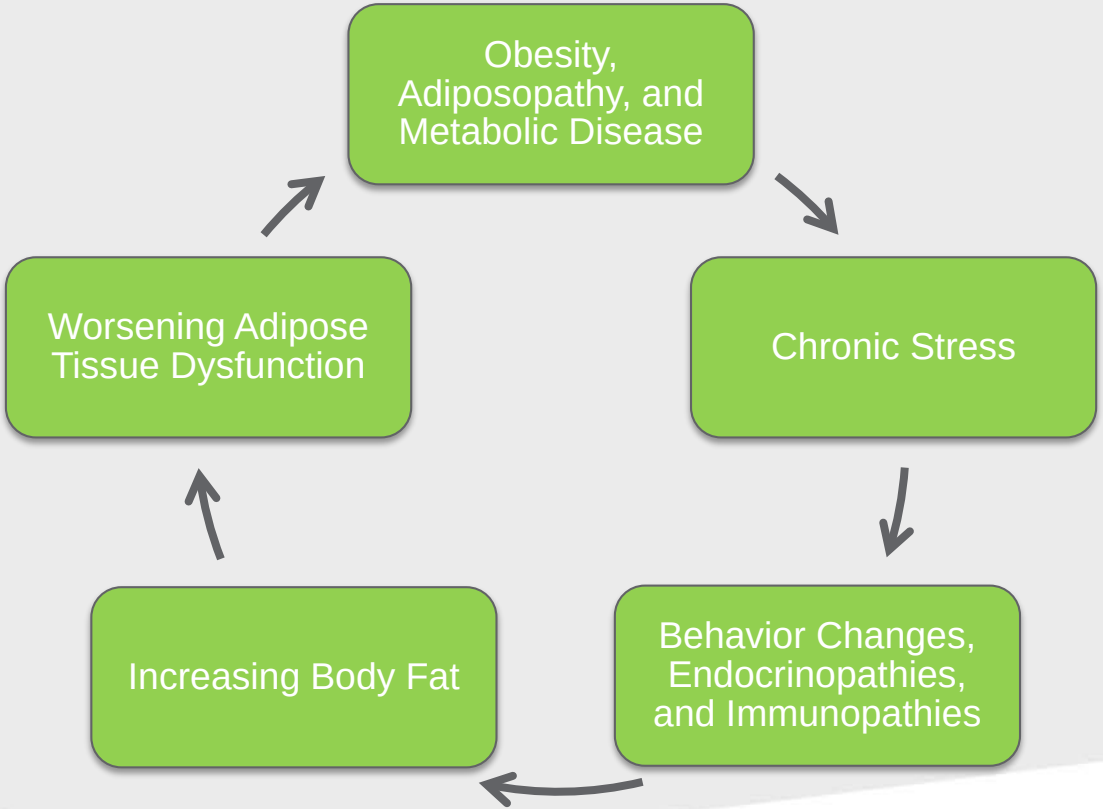
Adiposopathy Stress Cycle



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Evaluation and Treatment Overview:

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

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History

Medical History and Review of Systems

- Age, gender, race, ethnicity
- Fat mass disease (i.e., osteoarthritis, sleep apnea)
- Adiposopathy (i.e., type 2 diabetes mellitus, high blood pressure)
- Eating disorders
- Mental stress
- Sleep pattern
- Other medical and surgical conditions
- Medication and food allergies
- Medications that may affect body weight
- Cigarette smoking
- Alcohol intake
- Recreational drug use (e.g., marijuana, cocaine)

Family History

- Family members affected by obesity
- Applicable familial medical diseases

Support Systems

- Person who selects and purchases food
- Availability and involvement of family and friends
- Educational access to healthy nutrition and physical activity (e.g., current knowledgebase, availability of Internet, knowledge centers, etc.)

Socioeconomic and Cultural History

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

Examination of weight pattern over patient's lifetime

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Nutrition History

Meals and Snacks

- Timing
- Frequency (via questionnaire)
- Nutritional content
- Preparer of food
- Access to foods
- Location of home food consumption (i.e., eating area, television, computer, etc.)
- Location of away food consumption (i.e., workplace restaurants, fast food, etc.)

Behavior

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

Records

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

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Physical Activity History

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

Supporting documents in reference section

Obesity Medicine Association

Physical Activity History

Examples of common medical conditions that should be evaluated before prescribing an exercise program:

- Diseases of the heart, lung, musculoskeletal, and other body systems
- Metabolic diseases having potential risks with increased physical activity:
  - Atherosclerotic coronary heart disease (worsening ischemia)
  - Diabetes mellitus (hypoglycemia)
  - High blood pressure (increase blood pressure with resistance training)

Routine Preventive Medical Care

Ensure individuals with overweight or obesity receive standard preventive medical care, which, depending upon gender and age, may include:

- Breast cancer screening
- Gynecological exam with Pap smear (which may include assessment of human papilloma virus)
- Testicular and prostate cancer screening
- Colorectal cancer screening
- Immunizations

Physical Exam

Vital Signs

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

General Physical Exam

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

Supporting documents in reference section

Obesity Medicine Association

Laboratory: Routine

Adiposity-relevant Blood Testing

- Fasting blood glucose
- Hemoglobin A1c
- Fasting lipid levels
  - Triglycerides
  - Low-density lipoprotein (LDL) cholesterol
  - High-density lipoprotein (HDL) cholesterol
  - Non-HDL cholesterol
- Liver enzymes and other liver blood tests
  - Aspartate aminotransferase (AST)
  - Alanine aminotransferase (ALT)
  - Alkaline phosphatase
  - Total bilirubin
- Electrolytes (i.e., potassium, sodium, calcium, phosphorous, etc.)
- Renal blood testing (i.e., creatinine, blood urea nitrogen, etc.)
- Uric acid
- Thyroid stimulating hormone (TSH)
- Vitamin D levels (blacks may commonly have lower Vitamin D levels than whites)

General Laboratory Testing

- Complete blood count
- Urinalysis
- Urine for microalbumin

Laboratory: Individualized Blood Testing

- Glucose tolerance testing
- Fasting insulin testing
- Fasting proinsulin, C-peptide, and insulin if hyperinsulinemia is suspected as a secondary cause of obesity (e.g. insulinoma, nesidioblastosis, etc.)
- One milligram (mg) overnight dexamethasone cortisol suppression test, 24-hour urine collection for (free) cortisol, or repeated measures salivary cortisol collection at 11:00 PM if endogenous hypercortisolism is suspected as a secondary cause of obesity
- Prolactin, estradiol, follicle-stimulating hormone, luteinizing hormone, and pregnancy test in women with unexplained oligomenorrhea or amenorrhea
- Testosterone and other androgen levels (i.e., dehydroepiandrosterone sulfate/DHEAS) for women with hirsutism or polycystic ovarian syndrome
- Testosterone (and if low to a clinically significant degree: possibly prolactin, follicle-stimulating hormone, and luteinizing hormone) for men with impotence or physical findings of hypogonadism
- Apolipoprotein B and/or lipoprotein particle number, especially if triglyceride levels are elevated
- Iron studies (iron, total iron binding capacity, ferritin)
- High-sensitive C-reactive protein (hs-CRP)

Diagnostic Testing: Individualized

- Magnetic-resonance imaging or computed tomography of the brain if a structural lesion of the pituitary/hypothalamus is suspected (i.e., craniopharyngioma, pituitary tumor)
- Resting electrocardiogram
- Cardiac stress testing
- Echocardiogram
- Coronary calcium scores
- Cardiac positron emission tomography imaging (computed tomography)
- Ankle-brachial index
- Sleep studies
- Imaging studies of the liver (i.e., ultrasound)
- Anaerobic threshold/VO_2 testing
- Resting metabolic rate (RMR)

Diagnostic Testing: Individualized

Body Composition

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

Emerging Science Testing

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

Obesity Medicine Association

Identify and Manage Secondary/Contributing Causes of SFD and FMD

Conditions that may promote fat mass gain:

Genetic Syndromes

- Isolated (i.e., Prader Willi)
- Familial (melanocortin 4 receptor deficiency)

Medical Conditions

- Hypothalamic damage
- Immobility
- Insulinoma
- Some cases of untreated hypothyroidism
- Hypercortisolism (Cushing's disease)
- Sleep disorders

Psychological and Behavioral Conditions

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

Supporting documents in reference section

Obesity Medicine Association

Treatment of Adult Patients with Overweight or Obesity

Medical Management and Coordination

- Nutrition
- Physical Activity
- Behavior Therapy
- Pharmacotherapy
- Bariatric Surgery

Supporting documents in reference section

Obesity Medicine Association

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

Supporting documents in reference section

Obesity Medicine Association

Body Composition

Body Compartments: Fat-free Mass versus Lean Body Mass

Fat free mass* is total body mass (e.g. muscles, internal organs, water, bones, ligaments, and tendons) less any body fat. It includes:

- Water
- Mineral
- Protein and glycogen

*DXA measures fat, soft tissue, and bone. It often reports:

FFM = total mass – fat mass

Lean body mass* is total body mass (e.g. muscles, internal organs, water, bones, ligaments, and tendons), less nonessential or storage adipose tissue. It includes:

- Water
- Mineral
- Protein and glycogen
- Essential fat in organs, central nervous system, and bone marrow

*Using this definition, lean body mass usually differs from fat-free mass by only ~5%, slightly less in men, slightly more in women. Reports of "lean mass" (e.g., some DXA reports) can differ from the definition above, with bone mineral content (BMC) sometimes excluded, as in:

Total body mass = fat mass + lean mass + bone mass

Lean mass = total mass - fat mass – BMC

% body fat = fat mass / (total body mass – bone mass)

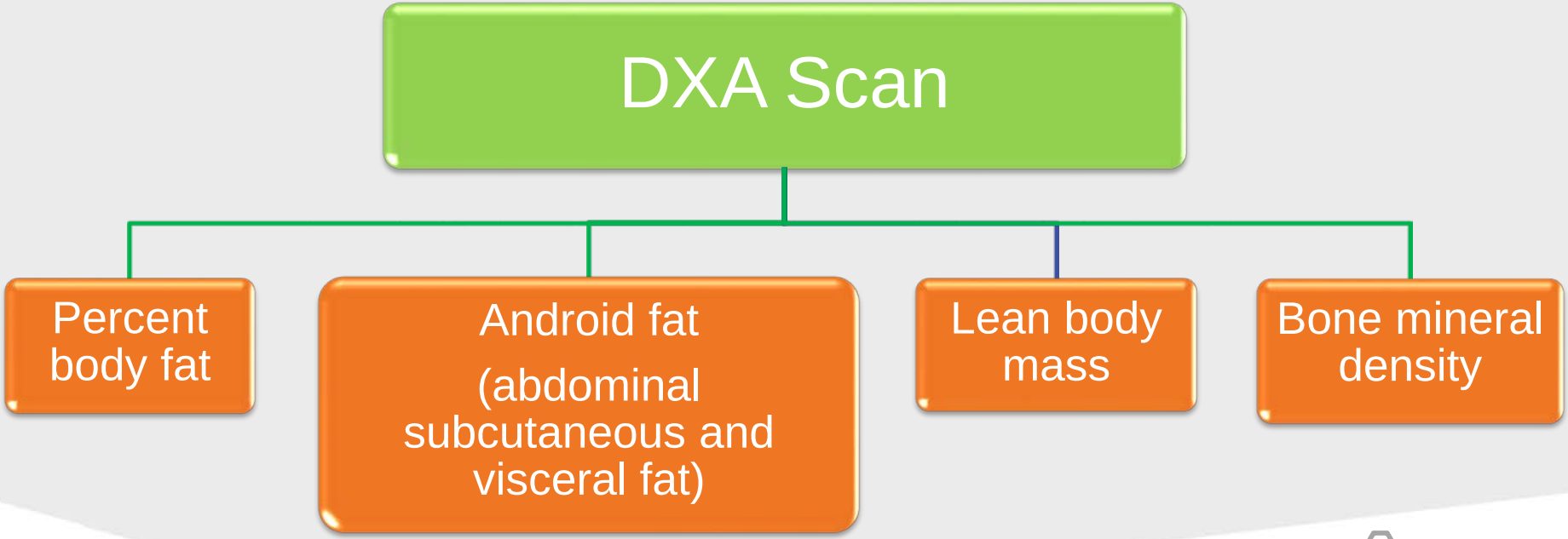
Body Compartments: Measurement Summary

Method	Accuracy*	Expense	Limitations
Calipers	User dependent, may substantially vary from direct measures of %BF	Inexpensive	Not optimal measuring technique for patients with very high body mass index
Dual-energy X-ray absorptiometry (DXA)	Accurate	Relatively inexpensive to patient – machine expensive to provider	Not all DXA (1) distinguish visceral versus subcutaneous fat, or (2) accommodate patients with very high body mass index
Air displacement (BOD POD)	Accurate with some potential variability	Inexpensive –BOD POD machine may be expensive to provider	Clothing and hydration dependent
Bioelectrical impedance	Accurate with some potential variability	Inexpensive – most machines relatively inexpensive	Hydration dependent
Under water weighing densitometry	Accurate	Relatively inexpensive	Time consuming, requires water submersion, and depends upon adequate lung exhalation
Computerized Tomography / Magnetic Resonance Imaging	Accurate	Expensive	Not all CT & MRI can accommodate individuals with very high body mass index
Deuterium dilution hydrometry	Accurate	Relatively inexpensive	Not readily available for commercial use

*The accuracy of all methods depends on the degree of training, and quality of equipment.

Dual Energy X-Ray Absorptiometry (DXA): Clinical Relevance

In living beings, due to accuracy, scope of measures, convenience and safety, DXA is often considered a *"gold standard"* for body composition analysis



Supporting documents in reference section

Obesity Medicine Association

Energy Expenditure

Energy Expenditure: Components Overall

In individuals with moderate physical inactivity, components of total energy expenditure:

- 70% resting metabolic rate
- 20% physical activity
- 10% diet-induced thermogenesis

Supporting documents in reference section

Obesity Medicine Association

Concomitant Medications

Concomitant Pharmacotherapy That Might Alter Body Weight: CVD and DM medications

Cardiovascular Medications

May increase body weight:

- Some beta-blockers
  - Propranolol
  - Atenolol
  - Metoprolol
- Older and/or less lipophilic dihydropyridine ("dipine") calcium channel blockers may increase body weight gain due to edema, compared to non-dihydropyridines and lipophilic dihydropyridines. The increased edema may exacerbate obesity-related edema (and sleep apnea related peripheral edema), and also confound body weight as a measure of body fat
  - Nifedipine
  - Amlodipine
  - Felodipine

Diabetes Mellitus Medications

May increase body weight:

- Most insulins
- Sulfonylureas
- Thiazolidinediones
- Meglitinides

May decrease body weight:

- Metformin
- Glucagon-like peptide-1 agonists
- Sodium glucose co-transporter 2 inhibitors
- Alpha glucosidase inhibitors
- Pramlintide

Supporting documents in reference section

Obesity Medicine Association

Concomitant Pharmacotherapy That Might Alter Body Weight: Hormones & Seizures

Hormones

May increase body weight:
- Glucocorticoids
- Estrogens

Variable effects on body weight:
- Progestins
  - Injectable or implantable progestins may have greatest risk for weight gain
  - May be dependent upon the individual
- Testosterone
  - May reduce percent body fat and increase lean body mass, especially if used to replace testosterone deficiency in men

Anti-seizure Medications

May increase body weight:
- Carbamazepine
- Gabapentin
- Valproate
- Pregabalin

May decrease body weight:
- Lamotrigine
- Topiramate
- Zonisamide

Supporting documents in reference section

Obesity Medicine Association

Concomitant Pharmacotherapy That Might Alter Body Weight: Anti-depressants

May increase body weight:

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

May decrease body weight:

- Bupropion
- Fluoxetine (variable)

Variable effects on body weight:

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

Concomitant Pharmacotherapy That Might Alter Body Weight: Mood & Migraine Tx

Mood Stabilizers

May increase body weight:

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

Variable/neutral effects on body weight:

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

Migraine Medications

May increase body weight:

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

May decrease body weight:

- Topiramate

Supporting documents in reference section

Obesity Medicine Association

Concomitant Pharmacotherapy That Might Alter Body Weight: Antipsychotics & Hypnotics

Antipsychotics

May substantially increase body weight:

- Olanzapine
- Quetiapine
- Clozapine
- Risperidone
- Zotepine

May somewhat increase body weight:

- Asenapine
- Chlorpromazine
- Iloperidone
- Paliperidone
- Sertindole
- Lithium
- Bexipiprazole

Variable/neutral effects on body weight:

- Amisulpride
- Aripiprazole
- Haloperidol
- Lurasidone
- Ziprasidone
- Cariprazine

Hypnotics

May increase body weight:

- Diphenhydramine

May have limited effects on body weight:

- Benzodiazepines
- Melatonergic hypnotics
- Trazodone

Supporting documents in reference section

Obesity Medicine Association

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

Pain relievers:

- Nonsteroidal anti-inflammatory drugs = Generally no weight change
- Acetaminophen = No weight change
- Opioids = New persistent opioid users may lose less weight after bariatric surgery

Anti-seizure medications used for treatment of neuropathy/pain:

- Gabapentin = Weight gain
- Pregabalin = Weight gain

Antidepressants used for neuropathy/pain:

- Weight gain = amitriptyline, doxepin, duloxetine, venlafaxine
- No weight change = nortriptyline

Topical treatments used for neuropathy/pain:

- Capsaicin = No weight change
- Lidocaine patches = No weight change

Identify and Manage Concomitant Pharmacotherapy That Might Alter Body Weight

Human Immunodeficiency Virus (HIV) Medications

May increase body weight:

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

May decrease body weight:

- Some highly active antiretroviral therapies (HAART) protease inhibitors with HIV lipodystrophy

Chemotherapies and Anti-Inflammatory Agents

May increase body weight:

- Tamoxifen
- Cyclophosphamide
- Methotrexate
- 5-fluorouracil
- Aromatase inhibitors
- Corticosteroids

May decrease body weight:

- Apremilast

Supporting documents in reference section

Obesity Medicine Association

Nutrition Therapy for Obesity

General Nutrition

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

Supporting documents in reference section

Obesity Medicine Association

Carbohydrates

- Carbohydrates contain 4 kcal/gram
- Carbohydrates can serve as a source of energy and as well cellular structural elements such as hyaluronic acid and proteoglycans
- Carbohydrates may contain sugars, starch and/or fiber
- The digestion and absorption of carbohydrates results in monosaccharide (glucose, fructose, galactose) molecules
- Carbohydrates are not an essential macronutrient, as the liver and kidney can synthesize glucose
- Calorie deficiency can lead to marasmus (insufficient calories), but there is *no known carbohydrate deficiency*
- USDA DRI for carbohydrate is 130 grams/day

Fat

- Fat contains 9 kcal/gram
- Fats or lipids are a diverse group of compounds used as an energy source and for many metabolic processes:
  - Immune response (omega-3 fatty acids)
  - Cell membrane structure (phospholipids)
  - Brain tissue (cerebrosides)
  - Synthesis of bile acid, cholesterol, vitamin D, steroid hormones
  - Insulation
- Several fatty acids cannot be made by the body and these “essential” fatty acids must be consumed in the diet
- Fatty acid deficiency can lead to a disease state
- USDA DRI for fat is at least 30 grams/day
- Replacing saturated fats with polyunsaturated or monounsaturated fats may reduce cardiovascular disease risk
- Replacing saturated fats with refined carbohydrates and sugar is not associated with reduced cardiovascular disease risk

Protein

- Protein contains 4 kcal/gram
- Protein contains amino acids and serves as the major structural building blocks of the human body: bone, muscle, skin, brain, nucleic acids
- Essential amino acids are those which cannot be made by the human body and must be consumed in the diet
- Some amino acids can be used as an energy source (converted to glucose or ketones when needed)
- Protein deficiency can lead to a disease state (Kwashiorkor is sufficient calories but insufficient protein)
- USDA DRI (Dietary Reference Intake) for protein is 0.8 to 2.0 grams/kg/day depending upon age, gender, physical activity

Insulin Controls Fat Metabolism

- Insulin promotes fatty acid and triglyceride synthesis (lipogenesis) and storage, and it inhibits fat breakdown (lipolysis)
- Foods that cause a rise in blood glucose, such as sugars, starches, or amino acids will stimulate the secretion of insulin from the pancreas
- A diet that lowers the amount of insulin secreted is beneficial for weight loss

Principles of Healthy Nutrition

Limit:

- Highly processed foods of minimum nutritional value: sweets, “junk foods,” cakes, cookies, candy, pies, chips
- Energy-dense beverages: sugar-sweetened beverages, juice, cream

Encourage:

- Consumption of healthy proteins and fats, vegetables, leafy greens, fruits, berries, nuts, legumes, whole grains
- Complex carbohydrates over simple sugars: Low glycemic index over high glycemic index foods
- High-fiber foods over low-fiber foods
- Reading labels rather than marketing claims

Managing the *quality* of calories is important when reducing the quantity of calories, such as during weight loss.

Nutrition Therapy for Obesity

Factors related to improved outcomes:

- Evidence-based
- Quantitative
- Patient adherence
- Patient preference
- Qualitative

Supporting documents in reference section

Obesity Medicine Association

Choosing Nutrition Therapy for Obesity

The most appropriate nutritional therapy for weight loss should be safe, effective, and one to which the patient can adhere.

- Encourage foods that result in a negative caloric balance to achieve and maintain a healthy weight
- Consider the following:
  - Individual food preferences, eating behaviors, and meal patterns
  - Cultural background, traditions, and food availability
  - Time constraints and financial issues
  - Nutritional knowledge and cooking skills

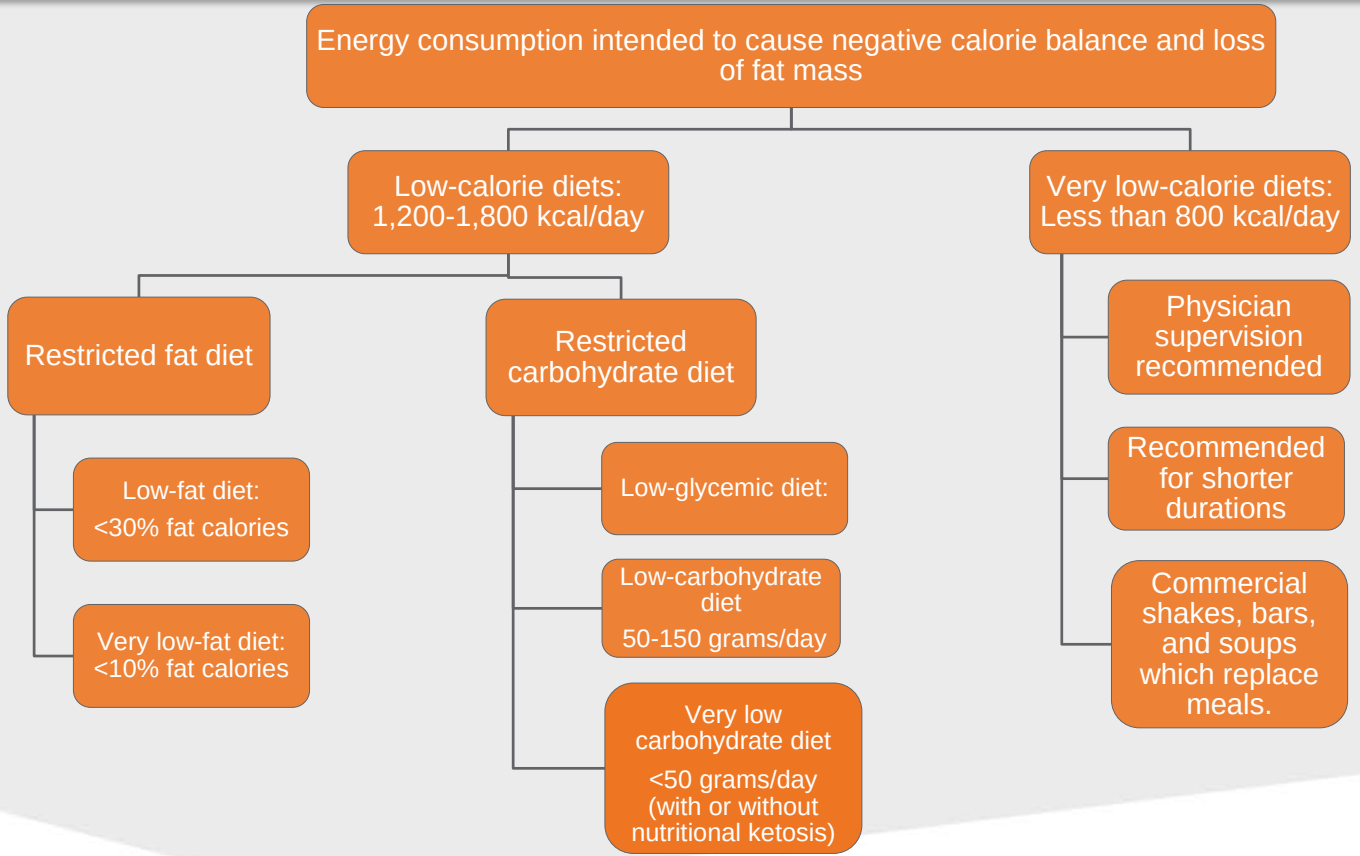
Supporting documents in reference section

Obesity Medicine Association

Choosing Nutrition Therapy for Obesity

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

Nutrition Therapy for Obesity



Supporting documents in reference section

Obesity Medicine Association

Low-calorie Diets: Restricted-carbohydrate Diet

Low-carbohydrate diet defined as 50-150 grams of carbohydrates per day.
Very low-carbohydrate diet defined as <50 grams of carbohydrates per day.

Weight Loss

- May produce modestly greater weight loss compared to fat-restricted dietary intake for the first 6 months, wherein afterwards, the net weight loss may be similar to other calorie restricted nutritional interventions
- May assist with reducing food cravings

Metabolic Effects

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

Risks

- May produce carbohydrate cravings within the first few days of implementation, which may be mitigated by adding low-glycemic-index carbohydrate foods
- May induce gout flare if history of gout
- May present challenges in patients undergoing dietary protein restriction (severe kidney disease)
- May result in malaise

Obesity Medicine Association

Low-calorie Diets: Restricted-fat Diet

Defined as 10-30% of total calories from fat.

Weight Loss

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

Metabolic Effects

- May reduce fasting glucose and insulin levels
- Modestly decreases low-density and high-density lipoprotein cholesterol levels
- May modestly reduce blood pressure

Risks

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

Very Low-calorie Diets

Defined as less than 800 kcal/day, typically implemented utilizing specifically formulated meal-replacement products *supervised by a trained clinician.*

Weight Loss

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

Metabolic Effects

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

Risks

- Fatigue, nausea, constipation, diarrhea, hair loss, and brittle nails
- Cold intolerance, dysmenorrhea
- Small increase in gallstones, kidney stones, gout flare
- If insufficient mineral intake, then may predispose to palpitations and cardiac dysrhythmias, muscle cramps
- Weight regain *will* occur if patients are not taught how to maintain healthy eating when transitioning to non-meal replacement

Trans Fats

Trans fats are created through a process of artificially hydrogenating polyunsaturated fats (vegetable oils) into more saturated fats, allowing for higher melting temperatures more desirable for processed foods, cooking and frying.

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

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

Supporting documents in reference section

Obesity Medicine Association

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 sweets*

* Olive oil is a staple of most definitions of the Mediterranean diet; however, some Mediterranean cuisine includes lard and butter for cooking, and olive oil for dressing salads and vegetables

Supporting documents in reference section

Obesity Medicine Association

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

Obesity Medicine Association

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:
- Processed and refined foods
- Foods with a high glycemic index
- 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

Obesity Medicine Association

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 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
- Processed foods
- Refined sugar, refined vegetable oils, and salt

Supporting documents in reference section

Obesity Medicine Association

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

* While plant-based nutritional intake is generally associated with weight loss, reduced risk of heart disease, other metabolic diseases, some cancers, and possibly all cause mortality, these potential benefits may be negated when healthier plant-based whole foods (i.e., with natural fiber and nutrients) are replaced by processed foods, fried foods, and refined carbohydrates.

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

Supporting documents in reference section

Obesity Medicine Association

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
  - May reduce caloric intake with preservation of lean body mass
  - May reduce body weight and potentially improve other metabolic parameters
- Potential disadvantages
  - Does not necessarily emphasize healthy 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)
  - May promote gout, urate nephrolithiasis, postural hypotension, and cardiac dysrhythmias
  - Most long-term evidence of efficacy and reported safety in animal studies

Physical Activity and Obesity

Physical Activity to Improve Health

Adiposopathy (Sick Fat Disease)

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

Non-adipose Parameters

- Improve metabolic health
- Improve musculoskeletal health
- Improve cardiovascular health
- Improve pulmonary health
- Improve mental health (e.g., mood, happiness, sense of well-being)
- Improve sexual health
- Improve cognitive heath

Medical Evaluation to Ensure Safety before Beginning New Exercise Program

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

Supporting documents in reference section

Obesity Medicine Association

Assess Mobility

Unable to Walk

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

Limited Mobility, Able to Walk

- Walking
- Swimming/aquatic exercises (e.g., shallow or deep water exercises)
- Gravity-mediated physical activity
- Assess for special equipment needs

No Substantial Limitations to Mobility

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

Obesity Medicine Association

Priority: Increase Energy Expenditure

Dynamic (Aerobic) Training

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

Resistive (Anaerobic) Strength Training

- Percent body fat better assessment of body composition than BMI
- Utilize appropriate weight-lifting technique
- Emphasize “core” muscle exercises
- Core = Midsection of the body, muscles related to abdomen, back, hips – important for posture and balance stabilization
- Using a variety of free weights, machines, and resistance bands may elicit less boredom and provide greater flexibility regarding scheduling and location
- Short-term sore muscles may be expected
- Sore joints suggests poor technique, with possible need for medical evaluation and physical activity modification
- Prioritize muscle mass metrics (e.g., myotape measurements) versus amount of weight lifted

Priority: Increase Energy Expenditure and Decrease Physical Inactivity

Leisure Time Physical Activity

- Engage in competitive sport activities involving substantial physical activity, best if on a routine basis
- Engage in non-competitive sports such as running, hiking, cycling, cross-fit training, etc.
- Outdoor warm-weather physical activity in sunlight may facilitate negative caloric balance and have other health benefits, but need to avoid excessive sun exposure
- Engage in physical activity sport-alternatives, such as dancing

Transportational/Occupational Non-exercise Activity Thermogenesis (NEAT)

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

Exercise Prescription

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

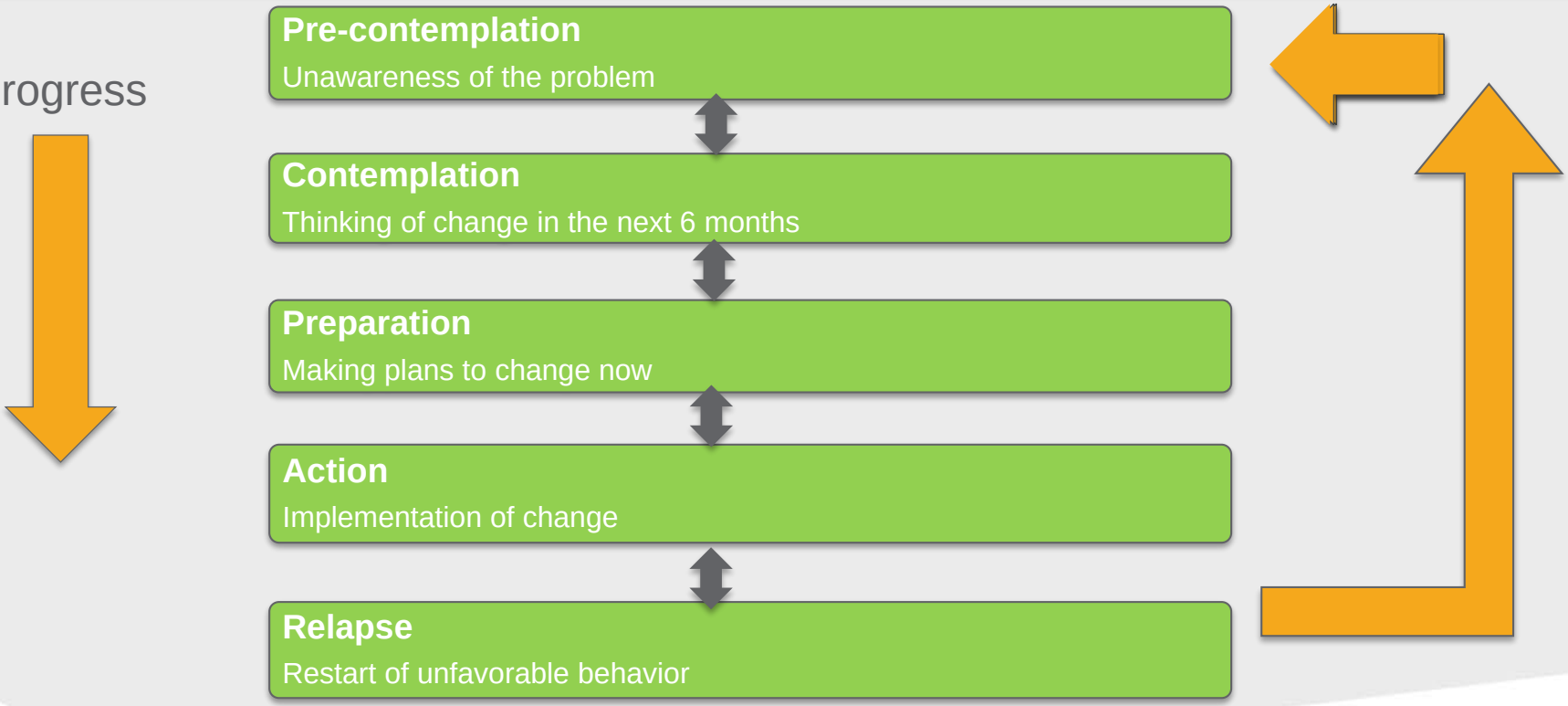
- Exercise prescription (FITT-VP)
  - ***F***requency
  - ***I***ntensity
  - ***T***ime or duration
  - ***T***ype or mode
  - ***V***olume or total energy expenditure of the exercise
  - ***P***rogression of the exercise

Supporting documents in reference section

Obesity Medicine Association

Motivational Interviewing

Motivational Interviewing: Stages of Change



Supporting documents in reference section

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Motivational Interviewing: Focus

Collaboration

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

Evocation

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

Autonomy

- Empowering the patient to own the solution
- Not the authoritarian power of the clinician

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

Obesity Medicine Association

Behavior Therapy

Why Do People Eat Like They Do?

Physiologic

- Strong biologic forces that resist weight loss
- Weak biologic forces that resist weight gain
- Hypothalamic dysfunction
  - Trauma
  - Inflammation
- Hunger before meals
- Lack of satiety after meals
- Eating to facilitate sleep

- Five senses central nervous system signaling:
  - Sight of food
  - Smell of food
  - Hear talk of food, sounds of food (cooking, wrapper opening)
  - Taste of food
  - Feel of food (texture, size, esophageal passage, stomach fullness) and feel of lack of food (e.g., vibration of empty stomach - borborygmi)

Why Do People Eat Like They Do?

Environment

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

Information Gap

- Lack of education about proper nutrition
- Challenges regarding access to nutritional information, especially when eating out
- Caloric content
- Nutritional content
- Marketing messages
  - “low fat”
  - “whole grain”
  - “no added sugar”
  - “natural sugar”
  - “cholesterol free”

Supporting documents in reference section

Obesity Medicine Association

Why Do People Eat Like They Do?

Reward

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

Why Do People Eat Like They Do?

Eating Disorders

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

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

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

Why Don’t People Engage in Routine Physical Activity?

Physiologic

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

Lack of Time

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

Supporting documents in reference section

Obesity Medicine Association

Why Don’t People Engage in Routine Physical Activity?

Disinterest

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

Environment

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

Supporting documents in reference section

Obesity Medicine Association

Why Do People Regain Body Weight?

Physiologic Priority Imbalance

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

Neurobiology

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

Supporting documents in reference section

Obesity Medicine Association

Why Do People Regain Body Weight?

Energy Expenditure

- Decrease in resting energy expenditure with weight loss
- Greater muscle efficiency occurs with weight loss, resulting in less energy expenditure with physical activity

Behavior

- Commitment amnesia
  - Forgetfulness of the degree of change and effort required to achieve initial weight loss success
  - Lack of maintaining accountability logs
- Altered priorities:
  - Intervening stress
  - Changing life circumstances
  - Changing health status
- Priority fatigue
  - Lack of maintaining healthy body weight priorities
  - Resorting to previous nutritional and/or physical activity habits after achieving initial weight-loss success
- Setpoint fallacy
  - The mistaken belief that once achieved, maintenance of weight loss will persist, irrespective of behavior, nutrition, and physical activity
  - "I know if I could just get the weight off, I could keep it off"

Supporting documents in reference section

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Behavior Therapy: Encounters and Education

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

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

Education

- Medical health
- Mental health
- Nutrition
- Physical activity
- Establish healthy sleep habits
- Establish healthy eating habits (i.e., reduce speed of eating, drink water between meals, choose and have available healthy snacks, etc.)
- Recognize and anticipate inevitable weight-loss plateaus

Behavior Therapy: Stimulus Control and Cognitive Restructuring

Stimulus Control

- Avoid eating for reasons other than hunger
- Avoid frequent snacking
- Avoid binge eating
- Utilize portion control
- Environmental removal of foods identified as especially tempting for the individual patient
- Being habitually mindful of eating stimuli may allow best chance for stimulus control

Cognitive Restructuring

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

Behavior Therapy: Goal Setting and Self-Monitoring

Goal Setting

- Patients are given step-by-step instructions to accomplish goals (i.e., nutrition and physical activity prescriptions)
- SMART
  - **S**pecific
  - **M**easurable
  - **A**ssignable
  - **R**ealistic
  - **T**ime-related
- Goals beyond body weight alone may include overall improvement in physical and mental health

Self Monitoring

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

Behavior Therapy: Behavioral Contracting and Problem Solving

Behavioral Contracting

- Tokens of reward
- Financial incentives

Problem Solving, Social Support, and Other Reinforcement Contingencies

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

Anti-obesity Medications

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.

Supporting documents in reference section

Obesity Medicine Association

Food and Drug Administration (FDA) Principles

FDA-approved Anti-obesity Medication Indications:

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

Other Principles

- Anti-obesity medications promote variable weight loss over variable duration in patients with overweight or obesity.
- Patients have an average of around 5 – 10% weight loss, with greater weight loss in hyper-responders, and less than 5% weight loss (or even weight gain) in hypo-responders.
- If no clinical improvement (e.g., at least 4 - 5% loss of baseline body weight) after 12-16 weeks with one anti-obesity medication, then consider alternative anti-obesity medication or increasing anti-obesity medication dose (if applicable).

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

Pregnancy and Lactation Categorization

Update to FDA Pregnancy and Lactation Labeling

- In December 2014, the FDA issued its “Pregnancy and Lactation Labeling Final Rule” (PLLR), which went into effect on June 30, 2015.
- The PLLR removed letter pregnancy categories - A, B, C, D, and X.
- Due to the fact that the prescribing information materials for most anti-obesity medications have yet to be updated to reflect the new rules, the Obesity Algorithm continues to include pregnancy and lactation categories.
- **In general, anti-obesity drugs are contraindicated in pregnancy, and should not be administered to, nor taken by women who are pregnant or trying to become pregnant**

Anti-Obesity Drug 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
Lorcaserin	Selective, serotonin (5-hydroxytrptamine) 2c receptor agonist that is a DEA Schedule IV agent that improves the sense of fullness. Some patients may lose 5 – 10% of body weight.	Lorcaserin is a generally well-tolerated drug, with headache, dizziness, fatigue, nausea, dry mouth, and constipation occurring more frequently compared to placebo. Warnings and Precautions include serotonin syndrome, neuroleptic malignant syndrome-like reactions, heart failure, psychiatric disorders, and priapism.	Serotonergic (SSRI's, SNRI's, MAO inhibitors) or anti-dopaminergic medications, St John's wort, triptans, bupropion, dextromethorphan, CYP 2D6 substrates

Supporting documents in reference section

Obesity Medicine Association

Anti-Obesity Drug 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, hypoglycemia, diarrhea, constipation, vomiting, headache, decreased appetite, dyspepsia, fatigue dizziness, abdominal pain, increase lipase, and renal insufficiency. Contraindicated with personal of family history of 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	This is a combination of phentermine (anti-obesity drug) and topiramate (used to treat seizures and migraine headaches). This DEA Schedule IV drug is approved as a weight management pharmacotherapy. Some patients may lose 5 – 10% of body weight.	Phentermine / topiramate can cause paresthesia (tingling or numb feelings to extremities), abnormal taste, insomnia, constipation, dry mouth, and acute angle glaucoma. Should not be used in patients with glaucoma, uncontrolled high blood pressure, heart disease, or hyperthyroidism. Topiramate can cause birth defects. Therefore, phentermine / topiramate should not be started until a pregnancy test is negative, unless the woman is using acceptable contraception, and pregnancy tests should be done monthly during use.	Monoamine oxidase inhibitors. May alter oral contraceptive blood levels.

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.

Potential for Publication Bias

Potential publication bias

- Clinicians should be cautious of the published literature regarding supplements or other therapies (including drugs), when the only available evidence is via infrequent, and/or small studies.
- The disproportionate publication of positive or significant results compared to negative or non-significant results potentially compromises the objectivity of literature review and meta-analyses.
- Negative or non-significant study results may not be submitted for publication, are often less likely to be accepted by journals for publication, and potentially less likely to be cited by other journals and the media compared to studies with positive results

Drugs are regulated differently than supplements

- Supplements (do not require a clinical trial development program acceptable to the FDA):
  - Can be marketed without FDA approval
  - Are generally considered safe until proven unsafe
- Drugs (requires a development program acceptable to the FDA):
  - Cannot be marketed until FDA approved
  - Not considered safe until proven safe

Once approved based upon clinical trial efficacy and safety, the FDA assigns an "indicated use" for pharmaceuticals. While not similarly applicable to the health benefits, efficacy or advisability of supplement consumption, independent organizations such as United States Pharmacopeia Dietary Supplement Verification Program (USP verified logo) provide voluntary processes to all for supplement quality indicators (monographs), supporting that what is in the supplement matches what the label says is in the supplement. Independent testing is also performed by companies such as ConsumerLab.com.

Definitions

	Prescription Drugs	Over-The-Counter Medications (OTC)
Definition	A therapeutic medicine intended for the diagnosis, cure, mitigation, treatment, or prevention of disease	Drugs the FDA considers to be safe and effective, but that do not require a prescription by a health professional (e.g., orlistat)
Approval process	Requires FDA approval before administered and/or prescribed to patients	Requires FDA approval for OTC use via the regulatory process of an OTC drug monograph
Marketing	Regulated by FDA*	Regulated by Federal Trade Commission*

* The FDA Office of Prescription Drug Promotion / OPDP (formerly DDMAC or Division of Drug Marketing, Advertising and Communications). Prescription Drug Advertising: Questions and Answers https://www.fda.gov/Drugs/ResourcesForYou/Consumers/PrescriptionDrugAdvertising/ucm076768.htm (accessed April 23, 2018)

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Definitions

	Supplements*	Functional Foods
Definition	Substances taken in addition to dietary intake, such as concentrated form of a nutrient (e.g., vitamins), isolated formulations of a nutrient (e.g., herbs or botanicals), minerals, and amino acids.	Nutrients with potentially favorable effects beyond basic nutrition, such as oatmeal or foods high in substances that may have health benefits (e.g., many phytochemicals)
Approval process	Not applicable. The FDA considers supplements more of a food than drug.	Not applicable
Marketing	Supplements are not permitted to be marketed for the purpose of treating, diagnosing, preventing, or curing diseases. Supplement manufacturers are responsible for ensuring the supplement is safe, claims of benefit are not false or misleading. **	Not applicable

*US Food & Drug Administration. Dietary Supplements. https://www.fda.gov/Food/DietarySupplements/ (accessed April 23, 2018)

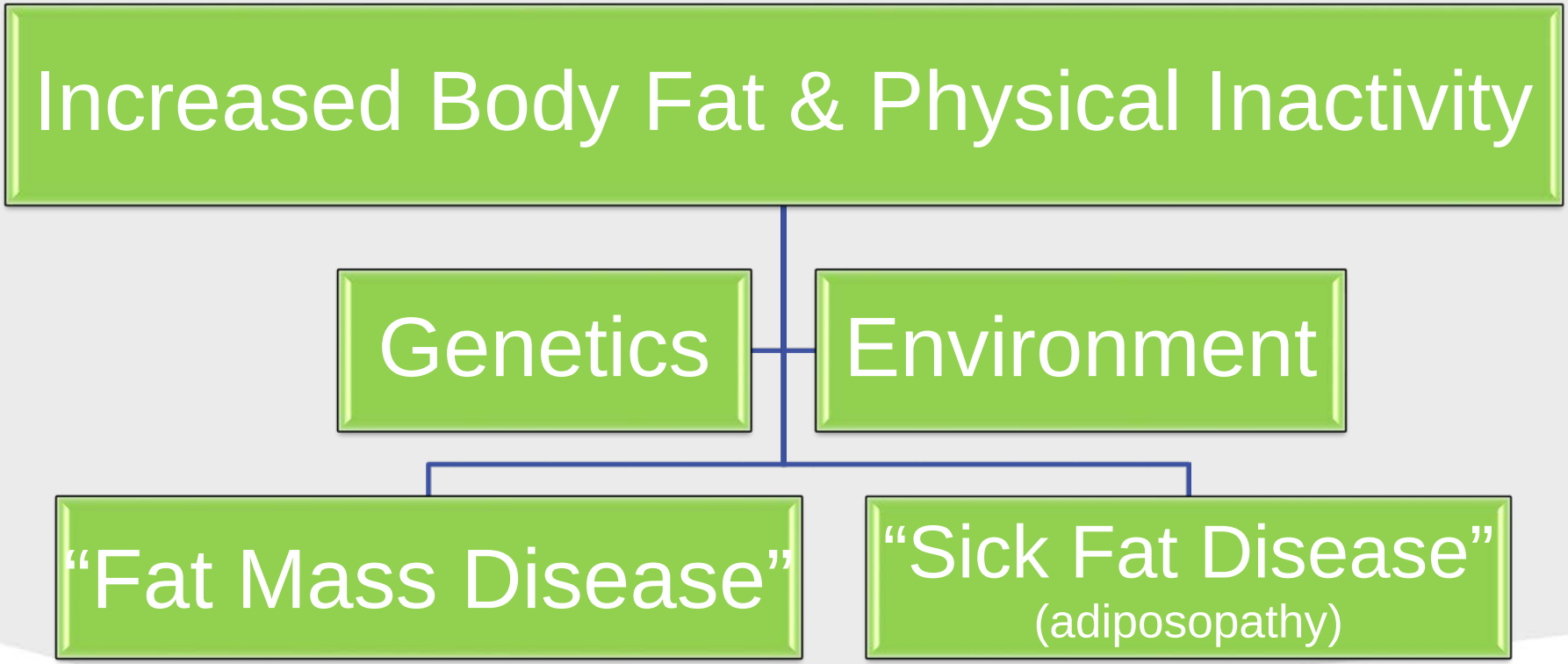
**USDA Nutrition.gov. https://www.nutrition.gov/subject/dietary-supplements (accessed April 23, 2018)

Supplements – Hepatotoxicity

- Increased herbal and dietary supplement (HDS) use is directly proportional to increased HDS-induced liver injury
- HDS-induced liver injury accounts for 20% of cases of hepatotoxicity in the US
- Major implicated agents include anabolic steroids and green tea extract
- Majority of cases of HDS-induced liver injury are from multi-ingredient nutritional supplements

Obesity and Metabolic Disease

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



Supporting documents in reference section

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Caveat: Obesity is not the only cause of metabolic disease

Non-Obesity-Related Causes of Metabolic Disease

Type 2 diabetes mellitus

- Hemochromatosis
- Hypercortisolism
- Excessive growth hormone
- Pancreatic insufficiency due to pancreatitis or surgical excision
- Side effects of concomitant therapies
- Genetic syndromes of insulin resistance
- Genetic syndromes of limited pancreatic insulin secretion

High blood pressure

- Pheochromocytoma
- Primary hyperaldosteronism
- Hypercortisolism
- Hyperthyroidism
- Renal artery stenosis
- Kidney diseases
- Side effects of concomitant therapies
- Familial or genetic syndromes

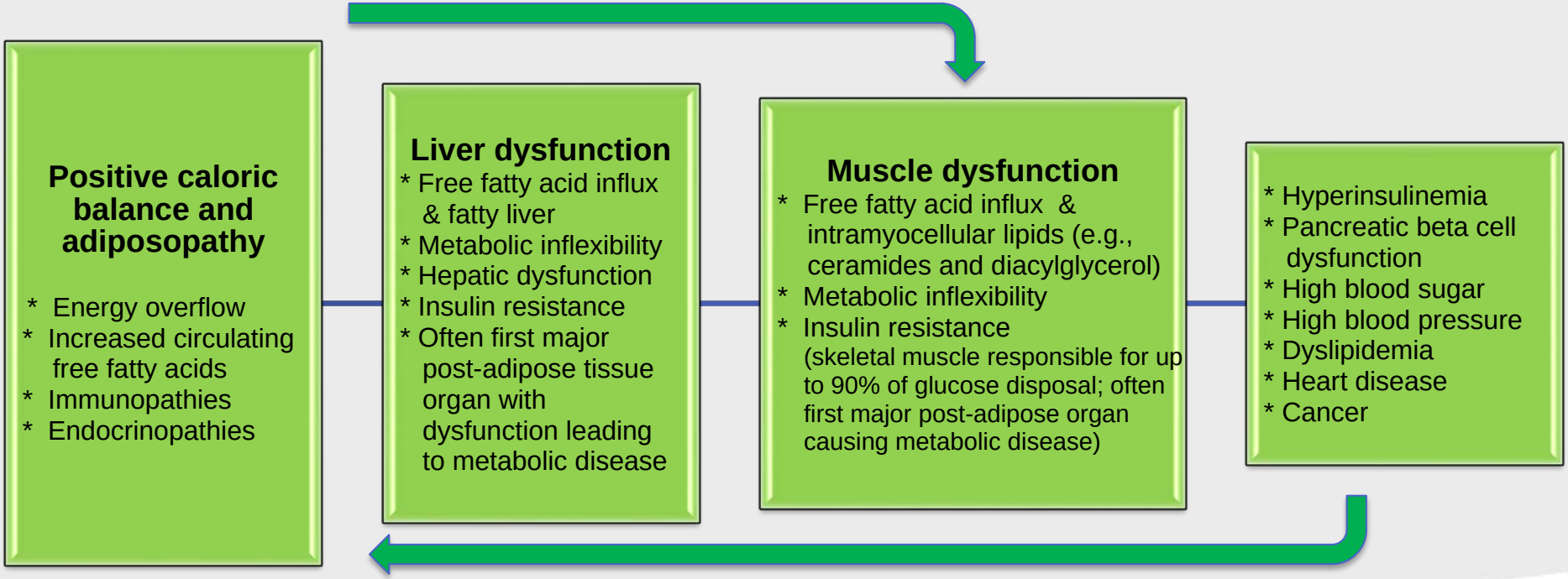
Dyslipidemia

- Untreated hypothyroidism
- Poorly controlled diabetes mellitus
- Liver disease
- Kidney disease
- Side effects of concomitant therapies
- Genetic dyslipidemias

Supporting documents in reference section

Obesity Medicine Association

Simplified mechanism of obesity, insulin resistance and metabolic disease



Hyperglycemia and hyperinsulinemia with potential worsening of obesity and adiposopathy

Supporting documents in reference section

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Obesity and Cardiovascular Disease

Obesity causes heart disease: Body fat distribution

Positive caloric balance, unhealthy nutrition & physical inactivity

Inadequate adipocyte proliferation and differentiation in peripheral subcutaneous adipose tissue

Energy overflow, adipocyte hypertrophy, & adiposopathy

Increased pericardial (paracardial and epicardial) fat

Increased intracardial fat

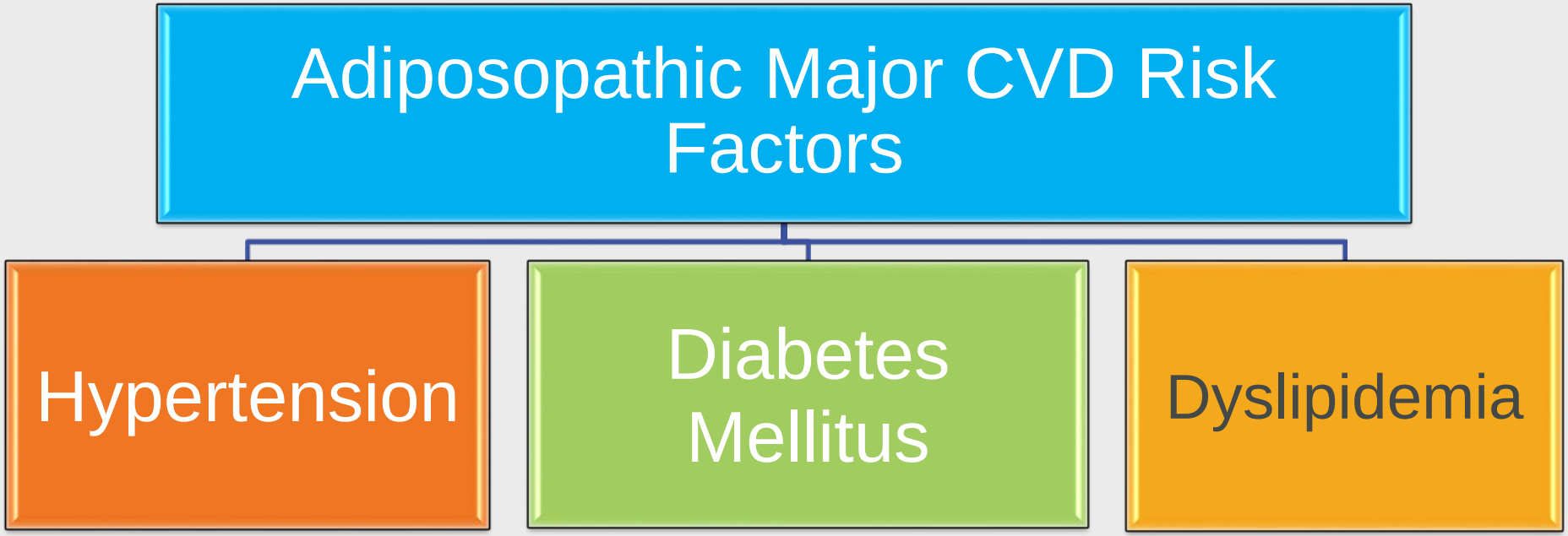
Increased visceral fat

Increased hepatic and skeletal muscle fat

Supporting documents in reference section

Obesity Medicine Association

INDIRECT Adiposopathy (“Sick Fat”) Cardiovascular Disease Effects



Supporting documents in reference section

Obesity Medicine Association

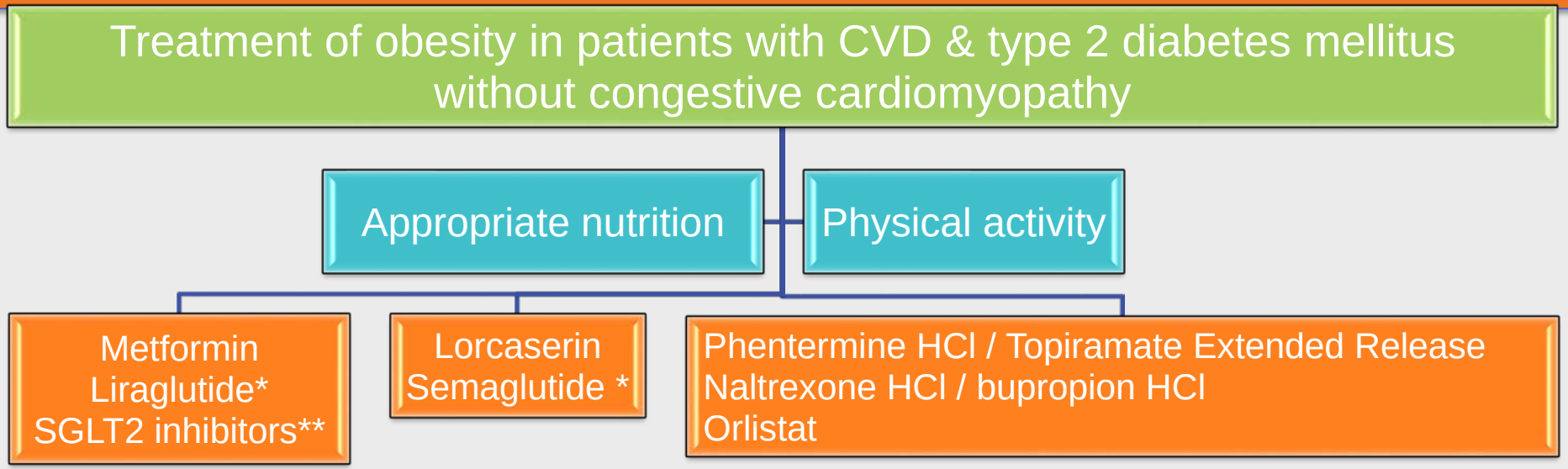
Obesity and cardiovascular disease (CVD) pharmacotherapy principles

- CVD is the most common cause of mortality among patients with obesity
- Patients with obesity should undergo global CVD risk reduction (e.g., healthy nutrition and physical activity, stop smoking, as well as optimal control of blood pressure, lipids, and blood sugars)
- 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
- Lorcaserin does not increase the risk of CVD among patients with obesity, but may reduce onset of diabetes mellitus by mechanisms independent of weight loss and reduce the rate of new onset or progressive renal impairment
- Retrospective data suggests phentermine & topiramate may not increase the risk of major adverse cardiac events
- Glucagon-like peptide 1 agonists with clinical outcome trial evidence to support CVD benefits in patients with diabetes mellitus (e.g., liraglutide, semaglutide) are being evaluated in CVD outcomes trials in patients with obesity
- Metformin and SGLT2 inhibitors decrease CVD among patients with diabetes mellitus. While they do not have an indication as anti-obesity agents, they modestly reduce body weight in patients with and without diabetes mellitus.
- Most anti-obesity agents do not have CVD outcome data to support improved CVD risk reduction when specifically evaluated in patients with obesity.
- When accompanied by weight loss, many anti-obesity drugs reduce CVD risk factors; orlistat, lorcaserin, liraglutide, naltrexone/bupropion, and phentermine/topiramate are not contraindicated in patients with cardiovascular disease
- When evidence is lacking, “expert opinion” may be useful to help provide generalized treatment options
- Obesity should be treated via a “patient-centered” approach, unique for each patient, and which may warrant individualized treatment options that differ from generalized treatment options

Supporting documents in reference section

Obesity Medicine Association

Pharmacotherapy for patients with obesity and cardiovascular disease (CVD)



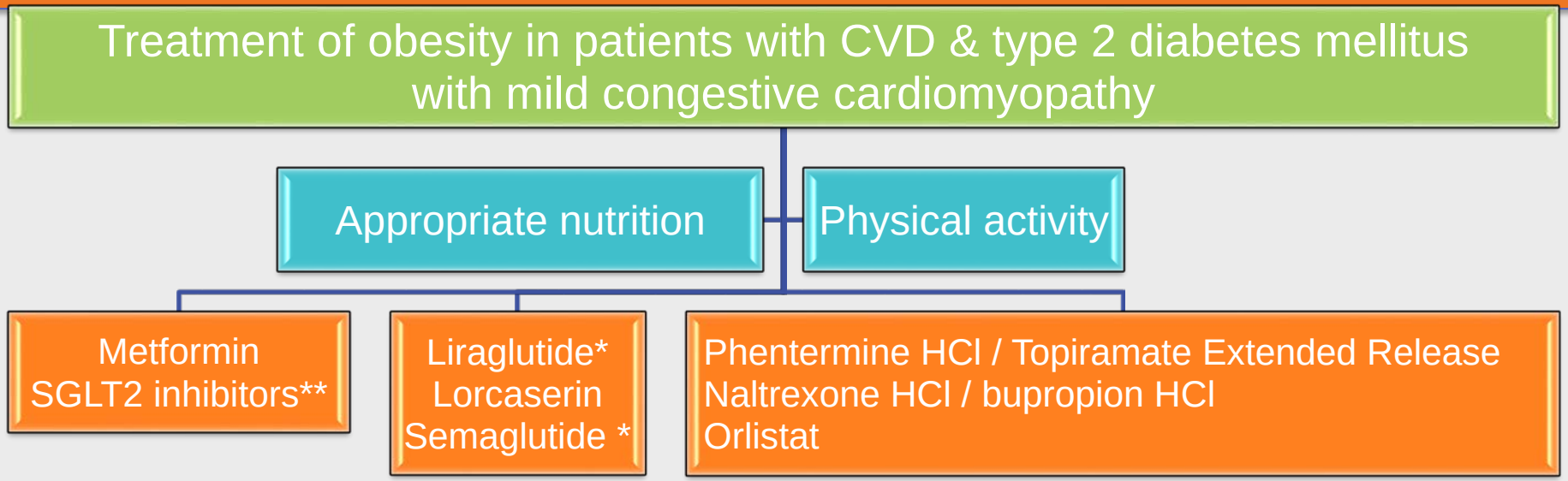
* Liraglutide at lower doses (i.e., 1.8 mg per day injectable) is indicated to treat type 2 diabetes mellitus (T2DM), and reduce CVD in patients with T2DM. Liraglutide at higher doses (i.e., 3.0 mg per day injectable) is indicated to treat obesity. Semaglutide is indicated to treat type 2 diabetes mellitus, and is undergoing clinical trials (PIONEER) for oral administration, as well as a CVD outcomes trials specifically in patients with obesity (SELECT).

** Sodium glucose transporter-2 (SGLT2) inhibitors reduce morbidity and mortality among patients with diabetes mellitus and CVD, especially those with congestive cardiomyopathy. Metformin should not be used in patients with heart disease (cardiomyopathy) resulting in hypoxia. Reports suggest metformin may be beneficial in patients with mild congestive cardiomyopathy; however, clinical trial evidence is limited. Liraglutide may not improve left ventricular systolic function in patients with heart failure. SGLT2 inhibitors may also produce mild weight loss. Therefore, while SGLT2 Inhibitors do not have an indicated use to treat obesity, they may be the anti-diabetes mellitus pharmacotherapy of choice to not only lower blood sugar, but to reduce the risk of future CVD events among patients with obesity – especially in patients having signs or symptoms of congestive cardiomyopathy.

Supporting documents in reference section

Obesity Medicine Association

Pharmacotherapy for patients with obesity and cardiovascular disease (CVD)



* Liraglutide at lower doses (i.e., 1.8 mg per day injectable) is indicated to treat type 2 diabetes mellitus (T2DM), and reduce CVD in patients with T2DM. Liraglutide at higher doses (i.e., 3.0 mg per day injectable) is indicated to treat obesity. Semaglutide is indicated to treat type 2 diabetes mellitus, and is undergoing clinical trials (PIONEER) for oral administration, as well as a CVD outcomes trials specifically in patients with obesity (SELECT).

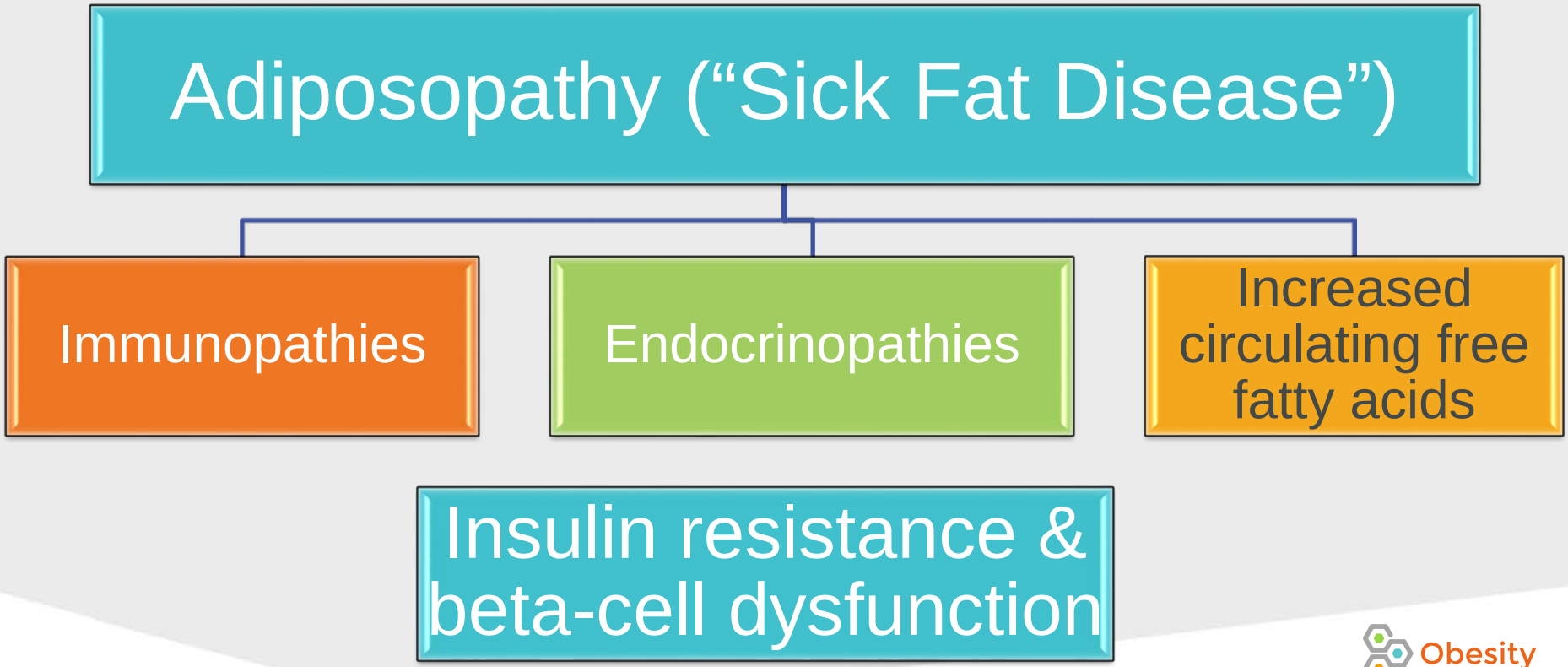
** Sodium glucose transporter-2 (SGLT2) inhibitors reduce morbidity and mortality among patients with diabetes mellitus and CVD, especially those with congestive cardiomyopathy. Metformin should not be used in patients with heart disease (cardiomyopathy) resulting in hypoxia. Reports suggest metformin may be beneficial in patients with mild congestive cardiomyopathy; however, clinical trial evidence is limited. Liraglutide may not improve left ventricular systolic function in patients with heart failure. SGLT2 inhibitors may also produce mild weight loss. Therefore, while SGLT2 Inhibitors do not have an indicated use to treat obesity, they may be the anti-diabetes mellitus pharmacotherapy of choice to not only lower blood sugar, but to reduce the risk of future CVD events among patients with obesity – especially in patients having signs or symptoms of congestive cardiomyopathy.

Supporting documents in reference section

Obesity Medicine Association

Obesity and Elevated Blood Sugar

How does obesity (adiposopathy) cause diabetes?



Supporting documents in reference section

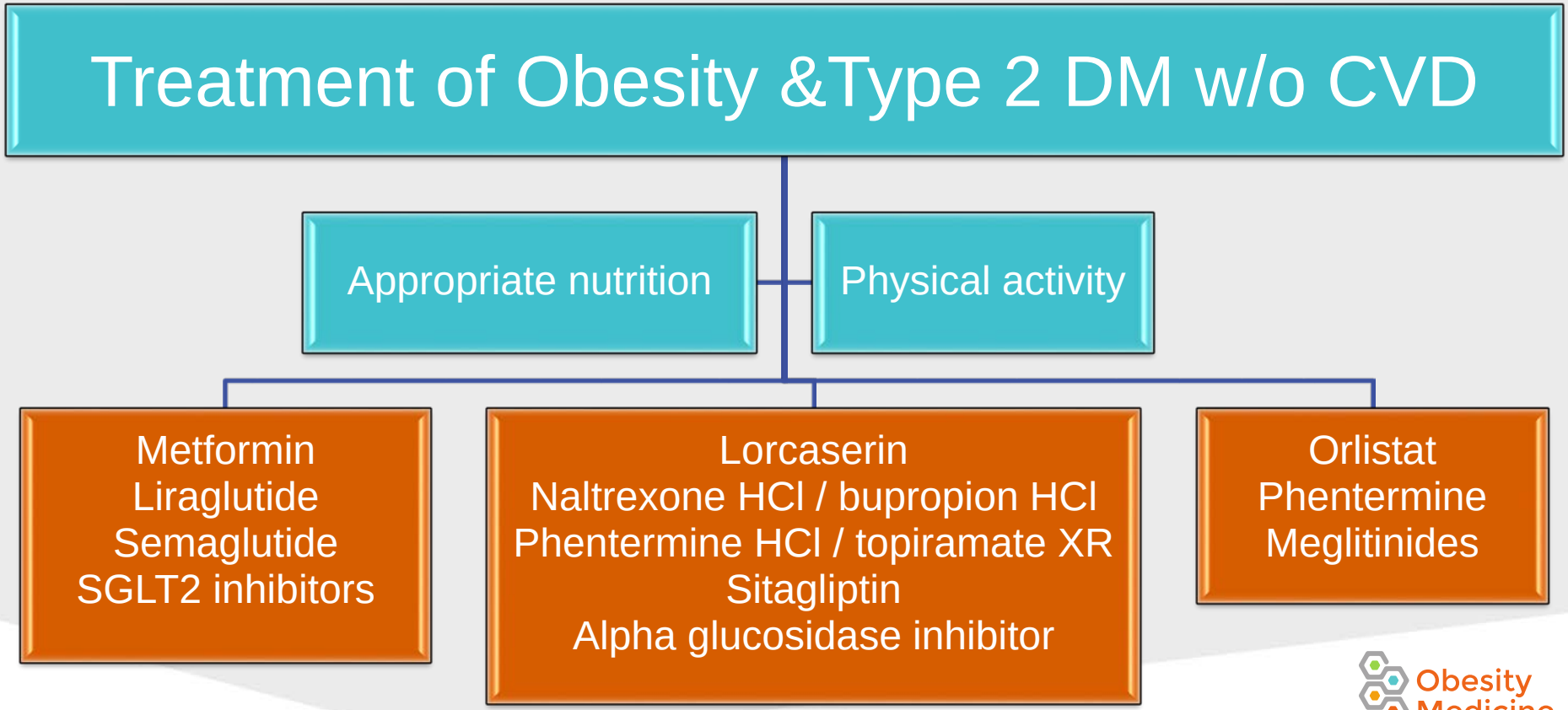
Obesity Medicine Association

Obesity and diabetes mellitus pharmacotherapy principles

- CVD is the most common cause of morbidity and mortality among patients with obesity and diabetes mellitus
- Diabetes mellitus is a major risk factor for CVD
- The disease of obesity is an important contributor to the disease of type 2 diabetes mellitus
- Patients with obesity and diabetes should undergo global CVD risk reduction (e.g., healthy nutrition and physical activity, stop smoking, as well as optimal control of blood pressure, lipids, and blood sugars)
- While CVD outcomes trials are ongoing with anti-obesity agents, no drug and dose having an indication to treat obesity has proven to reduce CVD events
- In a CVD study among patients with overweight or obesity, which included patients with diabetes mellitus, lorcaserin did not increase the risk of CVD
- Sulfonylureas and many insulins may increase body weight and increase the risk for CVD
- Based upon cardiovascular outcome trial data of patients with type 2 diabetes mellitus (consisting mostly of patients with CVD), 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. The benefits of SGLT2 inhibitors seem to be similar among patients with body mass index >= 30 kg/m2 versus < 30 kg/m2.
- Liraglutide at the 1.8 mg dose to treat diabetes reduces CVD among patients with diabetes mellitus, and reduces body weight and blood pressure
- Metformin decreases CVD among patients with diabetes mellitus, and modestly reduces body weight in patients with diabetes mellitus
- 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 CVD risk factors
- Both liraglutide and lorcaserin may lower blood sugar through weight dependent and weight independent mechanisms
- When evidence is lacking, “expert opinion” may be useful to help provide generalized treatment options
- Obesity should be treated via a “patient-centered” approach, unique for each patient, and which may warrant individualized treatment options that differ from generalized treatment options

Obesity Medicine Association

Pharmacologic treatment of patients with obesity and type 2 diabetes mellitus (T2DM) without cardiovascular disease (CVD)



Supporting documents in reference section

Obesity Medicine Association

Obesity and High Blood Pressure

How does obesity cause high blood pressure?

Fat Mass Disease and High Blood Pressure

Sleep apnea
- Cardiac pressure overload due to frequent changes in thoracic pressure
- Increased cardiac output due to hypoxia
- Increased cardiovascular inflammation

Kidney and renal vessel compression
- Increased abdominal pressure due to increased abdominal fat
- Potential restriction to renal vessel wall expansion
- Impaired naturesis

Perivascular adipose tissue
- Potential restriction to vessel wall expansion

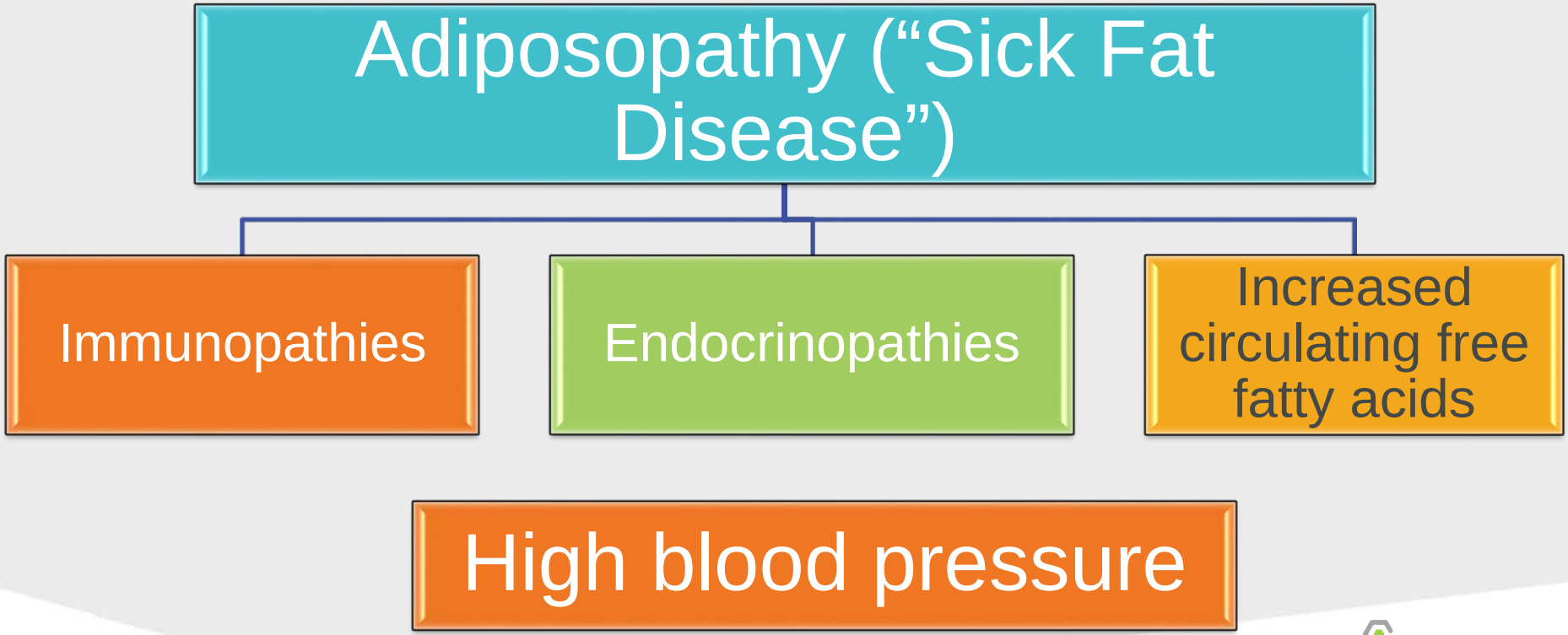
Increased cardiac output (heart rate x stroke volume)
- Increased oxygen and nutrient demands of increased adipose (and lean) body tissues increases cardiac output

High blood pressure

Supporting documents in reference section

Obesity Medicine Association

How does obesity cause high blood pressure?



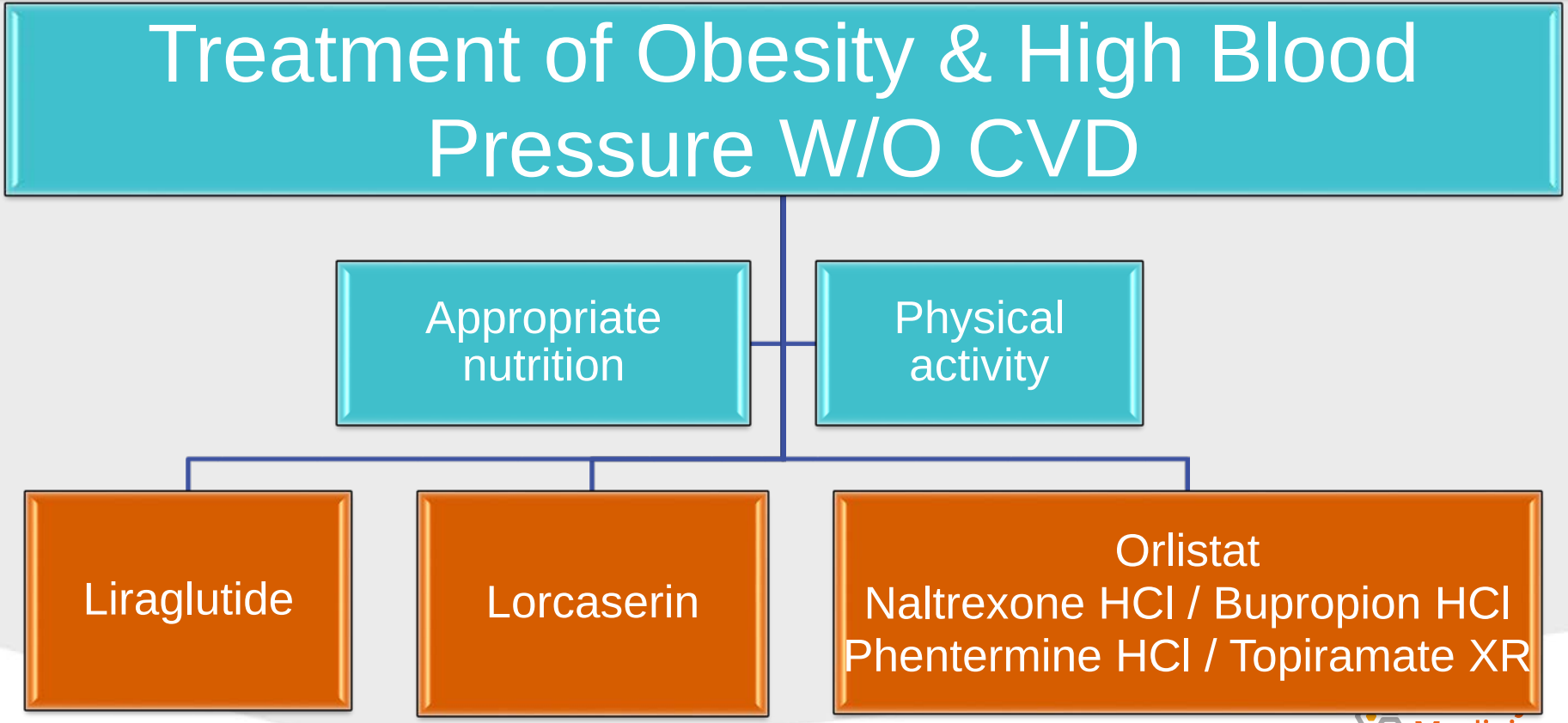
Supporting documents in reference section

Obesity Medicine Association

Obesity and high blood pressure pharmacotherapy principles

- CVD is the most common cause of mortality among patients with obesity
- Hypertension is a major risk factor for CVD
- The disease of obesity is an important contributor to the disease of hypertension
- Patients with obesity and hypertension should undergo global CVD risk reduction (e.g., healthy nutrition and physical activity, stop smoking, as well as optimal control of blood pressure, lipids, and blood sugars)
- While CVD outcomes trials are ongoing with anti-obesity agents, no drug and dose having an indication to treat obesity has proven to reduce CVD events
- In a CVD study among patients with overweight or obesity, which included patients with hypertension, lorcaserin did not increase the risk of CVD
- When accompanied by weight loss, many anti-obesity agents decrease blood pressure
- Some anti-obesity agents may increase blood pressure
- When evidence is lacking, “expert opinion” may be useful to help provide generalized treatment options
- Obesity should be treated via a “patient-centered” approach, unique for each patient, and which may warrant individualized treatment options that differ from generalized treatment options

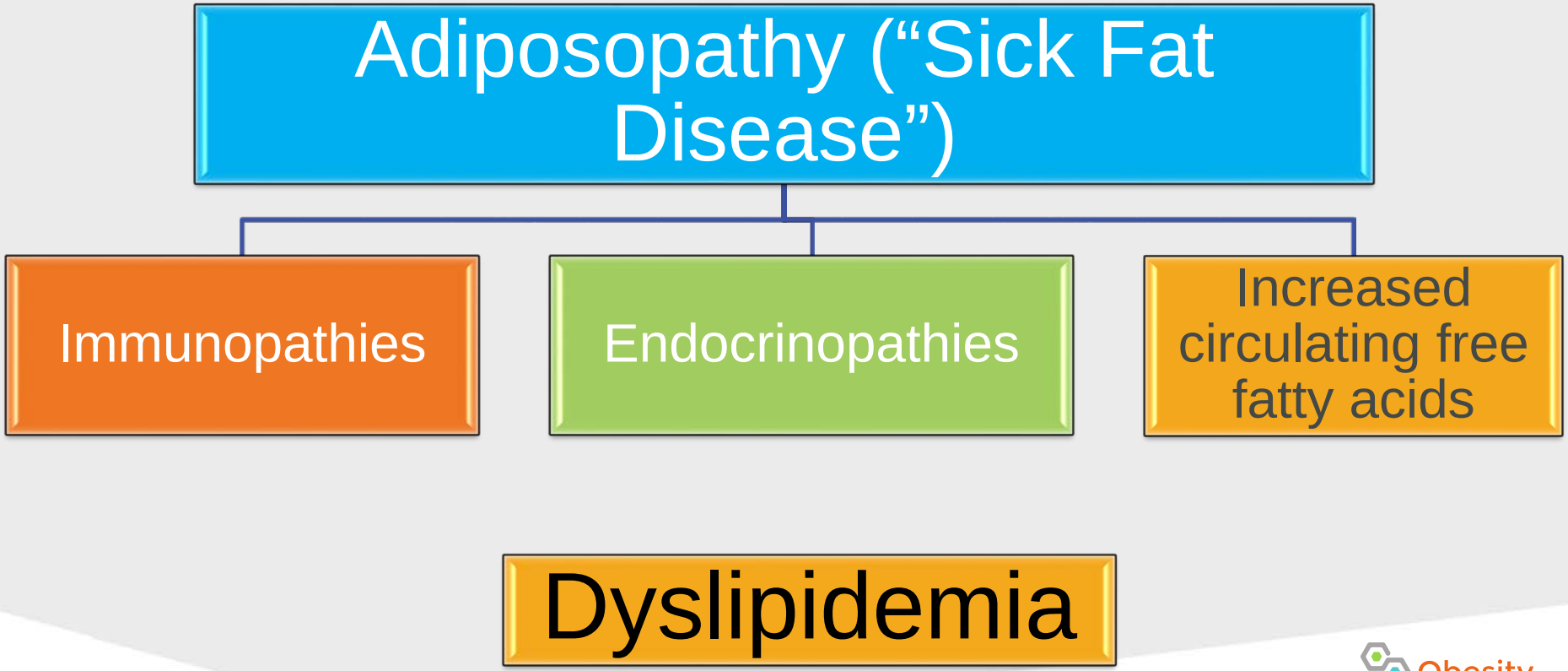
Anti-obesity pharmacologic treatment of patients with obesity and high blood pressure (CVD)



Supporting documents in reference section

Obesity and Dyslipidemia

How does obesity (adiposopathy) cause dyslipidemia?



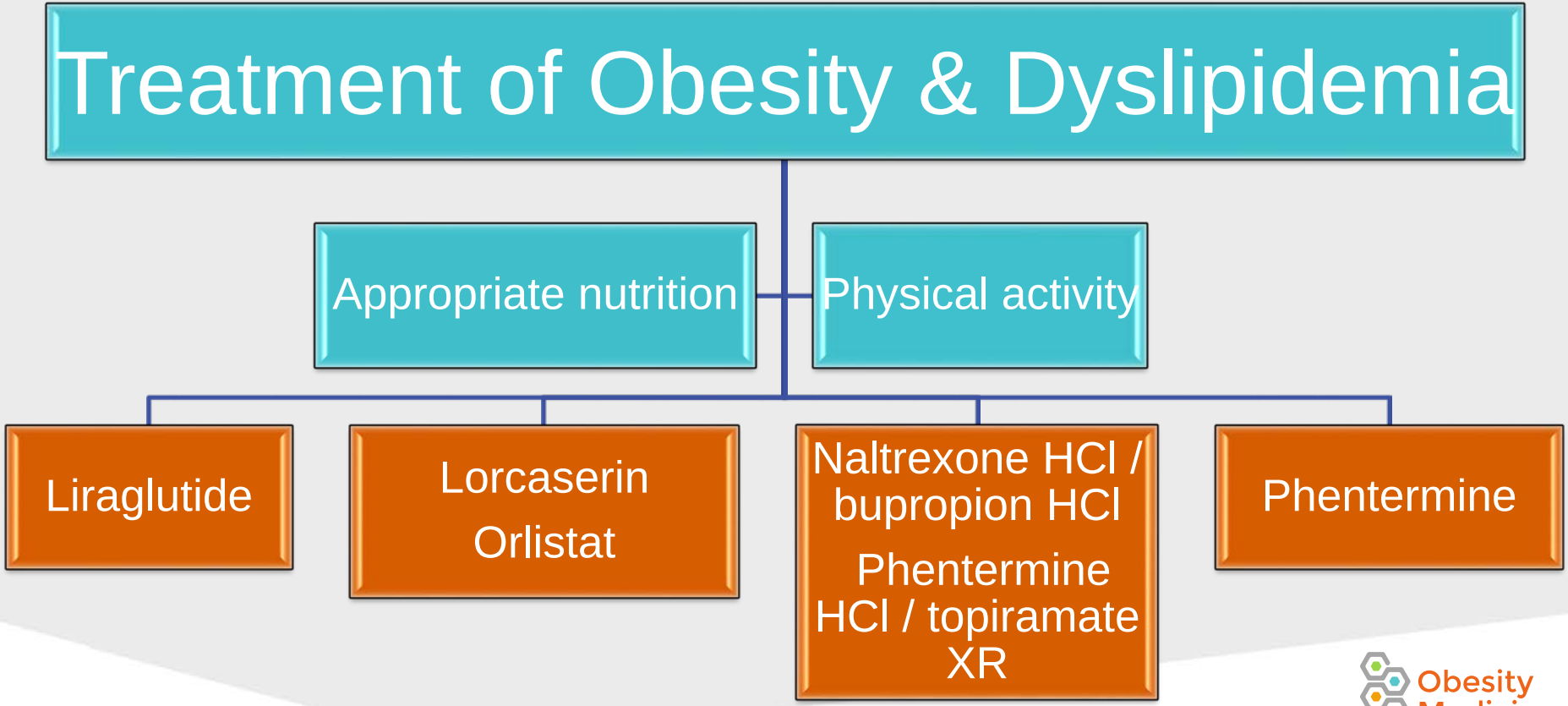
Supporting documents in reference section

Obesity Medicine Association

Obesity and dyslipidemia pharmacotherapy principles

- CVD is the most common cause of mortality among patients with obesity
- Dyslipidemia is a major risk factor for CVD
- The disease of obesity is an important contributor to the disease of dyslipidemia
- Patients with obesity and dyslipidemia should undergo global CVD risk reduction (e.g., healthy nutrition and physical activity, stop smoking, as well as optimal control of blood pressure, lipids, and blood sugars)
- While CVD outcomes trials are ongoing with anti-obesity agents, no drug and dose having an indication to treat obesity has proven to reduce CVD events
- In a CVD study among patients with overweight or obesity, which included patients with dyslipidemia, lorcaserin did not increase the risk of CVD
- When accompanied by weight loss, many anti-obesity agents improve dyslipidemia
- When evidence is lacking, “expert opinion” may be useful to help provide generalized treatment options
- Obesity should be treated via a “patient-centered” approach, unique for each patient, and which may warrant individualized treatment options that differ from generalized treatment options

Pharmacologic treatment of patients with obesity and dyslipidemia without cardiovascular disease (CVD)

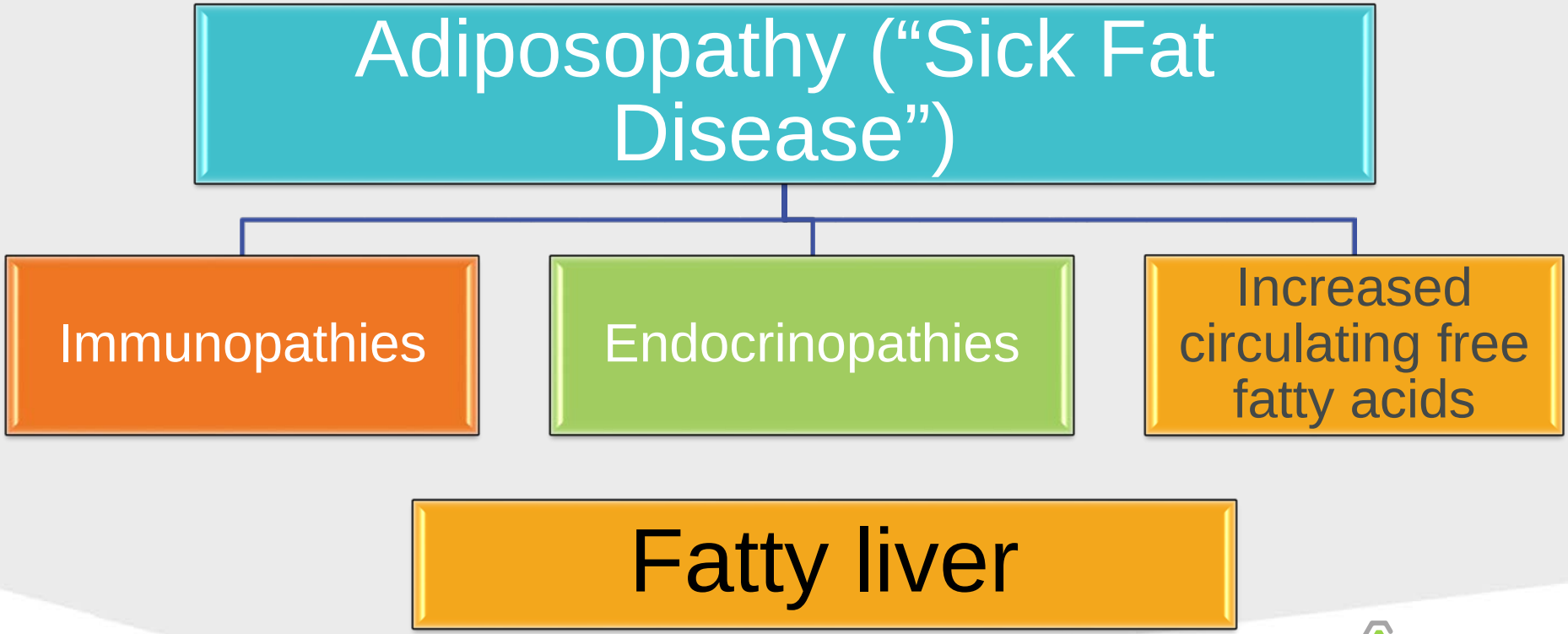


Supporting documents in reference section

Obesity Medicine Association

Obesity and Nonalcoholic Liver Disease (NAFLD)

How does obesity (adiposopathy) cause nonalcoholic liver disease?



Supporting documents in reference section

Obesity Medicine Association

Obesity and nonalcoholic fatty liver disease (NAFLD) treatment principles

- NAFLD is the most common cause of chronic liver disease (~25% of adults)
  - ~45% Hispanics
  - ~33% Caucasians
  - ~24% Blacks
- More than 2/3 of patients with NAFLD have obesity
- NAFLD is a risk factor for CVD
- Hepatosteatosis is fatty liver without clinical inflammation
- Hepatosteatitis is fatty liver with inflammation (nonalcoholic steatohepatitis or NASH)
- 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%
- NAFLD is an important cause of end stage liver disease, hepatocellular carcinoma and by 2020, may be the leading indication for liver transplant
- No drug has an approved indication to treat fatty liver
- When accompanied by weight loss, many anti-obesity agents can reduce hepatic fat
- When evidence is lacking, “expert opinion” may be useful to help provide generalized treatment options
- Obesity should be treated via a “patient-centered” approach, unique for each patient, and which may warrant individualized treatment options that differ from generalized treatment options

Obesity Medicine Association

Nonalcoholic fatty liver disease (NAFLD): Treatment

Fatty Liver Treatment

Manage common secondary causes of NAFLD
* Obesity / adiposopathy
* Type 2 diabetes mellitus
* Dyslipidemia
* Insulin resistance
* Possibly hypothyroidism

Manage medications that may contribute to NAFLD
* Corticosteroids (systemic)
* Highly active antiretroviral therapy (HAART)
* Amiodarone
* Tamoxifen
* Methotrexate

Manage uncommon secondary causes of NAFLD
* Substantial and rapid surgical weight loss
* Starvation
* Total parenteral nutrition
* Hepatitis C infection
* Environmental toxicity
* Wilsons disease
* Celiac disease
* Lipodystrophy
* Disorders of lipid metabolism (e.g., abetalipoproteinemia, hypolipoproteinemia, familial combined hyperlipidemia, glycogen storage disease, Weber-Christian syndrome)

Supporting documents in reference section

Obesity Medicine Association

Nonalcoholic fatty liver disease (NAFLD): Treatment

Fatty Liver Treatment

Nutrition

* Achieve a healthy body weight among patients with overweight or obesity
* Implement an evidenced-based meal plan that limits saturated fats and processed/refined carbohydrates, such as the Mediterranean diet or other carbohydrate/saturated fat restricted nutritional interventions

Dynamic ("aerobic") and resistance physical activity

* Helps achieve and maintain a healthy body weight
* Increases peripheral insulin sensitivity, reduces circulating free fatty acids and glucose, and reduces their delivery to the liver
* Increases intrahepatic fatty acid oxidation, decreases fatty acid synthesis, and helps prevent mitochondrial and hepatocellular damage

Medications

* No pharmacotherapy has an approved indication to treat NAFLD
* Some drugs may reduce hepatic fat
- Metformin
- Peroxisome proliferator activated receptor gamma agonists
- Glucagon Like Protein – 1 agonists
- Vitamin E

Supporting documents in reference section

Obesity Medicine Association

Obesity and Cancer

Obesity and Adiposopathy Increase the Risk of Cancers

- Bladder cancer
- Brain cancer
- Breast cancer (postmenopausal)
- Cervical cancer
- Colon cancer
- Endometrial/uterine cancer
- Esophageal cancer
- Gallbladder cancer
- Head and neck cancer
- Kidney/renal cancer
- Leukemia
- Liver cancer
- Multiple myeloma
- Non-Hodgkin lymphoma
- Ovarian cancer
- Pancreatic cancer
- Prostate cancer (prognosis is worse, not necessarily increased risk)
- Stomach cancer
- Thyroid cancer

Supporting documents in reference section

Obesity Medicine Association

How does obesity (adiposopathy) cause cancer?

Adiposopathy ("Sick Fat Disease")

Immunopathies

- Adiposopathic cytokines (e.g., tumor necrosis factor, interleukin 6) damage cellular DNA, promote gene mutations, enhance angiogenesis, promote cell proliferation, and contribute to mitochondrial and endoplasmic reticulum stress, increasing release of reactive oxygen species (ROS) that damage (or futher damage) cellular DNA
- Adiposopathic cytokines may also promote endothelial dysfunction, extracellular matrix abnormalities and intravasation (the rate-limiting step of metastasis)

Endocrinopathies

- Increased potentially cancer promoting hormones (e.g., estrogen, leptin, and androgens in women) and decrease in anti-carcinogenic adiponectin
- Insulin resistance may increase insulin and insulin growth factor-1, which are growth factors that may stimulate tumor growth

Hypoxia

- Adipose tissue hypoxia (due to growth beyond vascular supply and/or increased interstitial pressure from limited expansion of extracellular matrix) promotes secretion of angiogenic factors, which if localized around nascent tumor cells, may accelerate surrounding blood vessel growth, provide increased oxygen and nutrients, accelerate the growth of deranged tumor cells, and facilitate progression towards more malignant cancer

Carcinogenesis

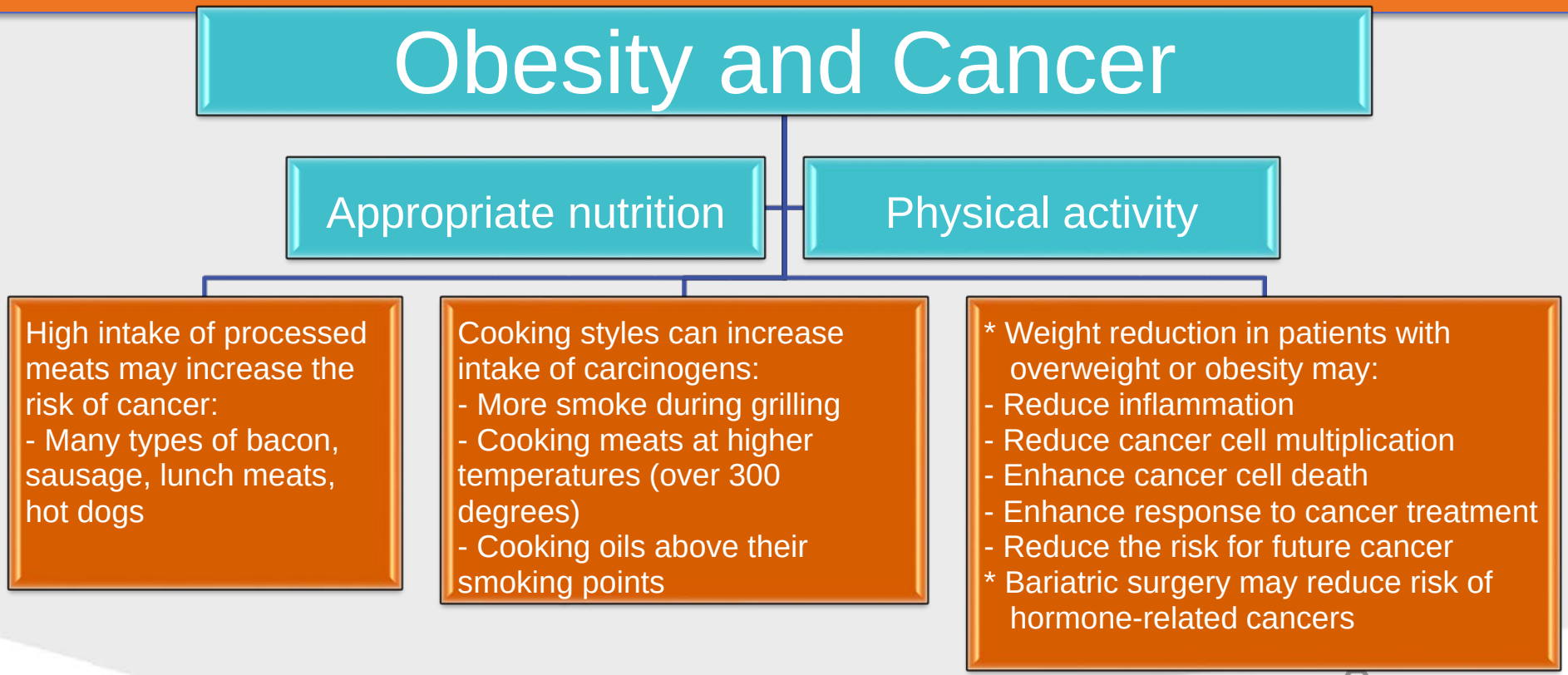
Supporting documents in reference section

Obesity Medicine Association

Obesity and cancer treatment principles

- Obesity is the second most common preventable cause of cancer, and may soon overtake smoking as the most common preventable cause of cancer
- 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
- No drug has an indication to treat obesity and prevent or treat cancer
- 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
- When evidence is lacking, “expert opinion” may be useful to help provide generalized treatment options
- Obesity should be treated via a “patient-centered” approach, unique for each patient, and which may warrant individualized treatment options that differ from generalized treatment options

Nutrition in patients with obesity and at risk for cancer or with cancer



Supporting documents in reference section

Obesity Medicine Association

Nutrition in patients with obesity and at risk for cancer or with cancer

Obesity and Cancer

Appropriate nutrition

* Attaining healthy body weight is a primary goal for nutritional and physical activity based cancer management in patients with obesity
* Another goal is to reduce hyperinsulinemia (a growth factor), through weight loss & possibly metformin

Physical activity

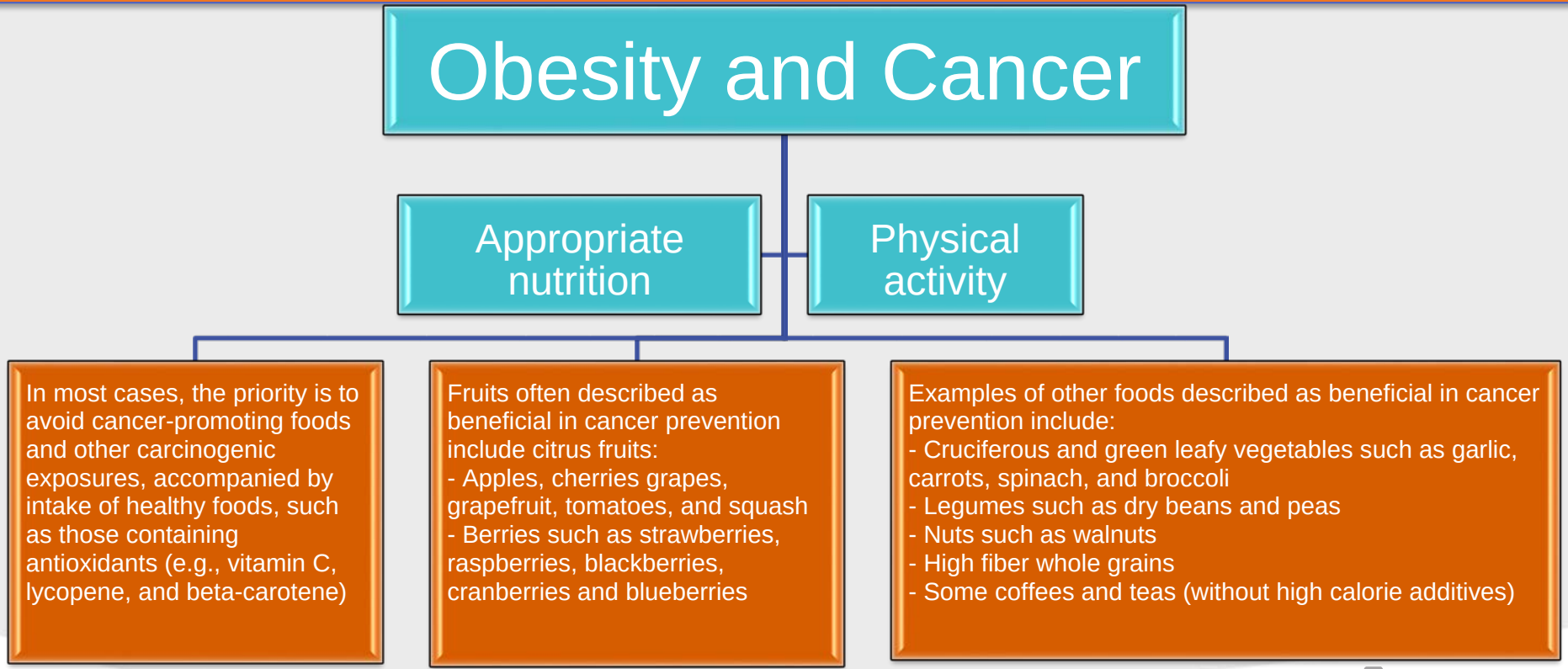
* Oxidation is when a substance gives away electrons (and oxygen gains electrons) - opposite of reduction (can be either harmful or helpful to health)
* Common examples of the natural consequences of oxidation:
- Oxidation of iron is rust (corrosion)
- Oxidation of fish oils causes rancidity
- Oxidation of a cut apple causes it to turn brown
* Obesity, adiposopathy, smoking, and decreased energy expenditure promote “oxidative stress,” which is the imbalance in the creation of unstable reactive oxygen species (ROS), relative to the body’s ability to detoxify these radicals (i.e. via “anti-oxidants”)
- ROS (e.g., superoxide) oxidize and damage DNA, and contribute to cancer
- Increased ROS contributes to aging
* Anti-oxidants counterbalance oxidation

* Foods thought most beneficial for cancer:
- Phytochemicals
- Fiber
- Antioxidants
* Antioxidants may be most beneficial when obtained from healthy food, compared to vitamin pills or supplements

Obesity Medicine Association

Supporting documents in reference section

Nutrition in patients with obesity and at risk for cancer or with cancer



Supporting documents in reference section

Obesity Medicine Association

Investigational Anti-obesity Pharmacotherapy

Priorities of Anti-Obesity Drug Development: Objectives

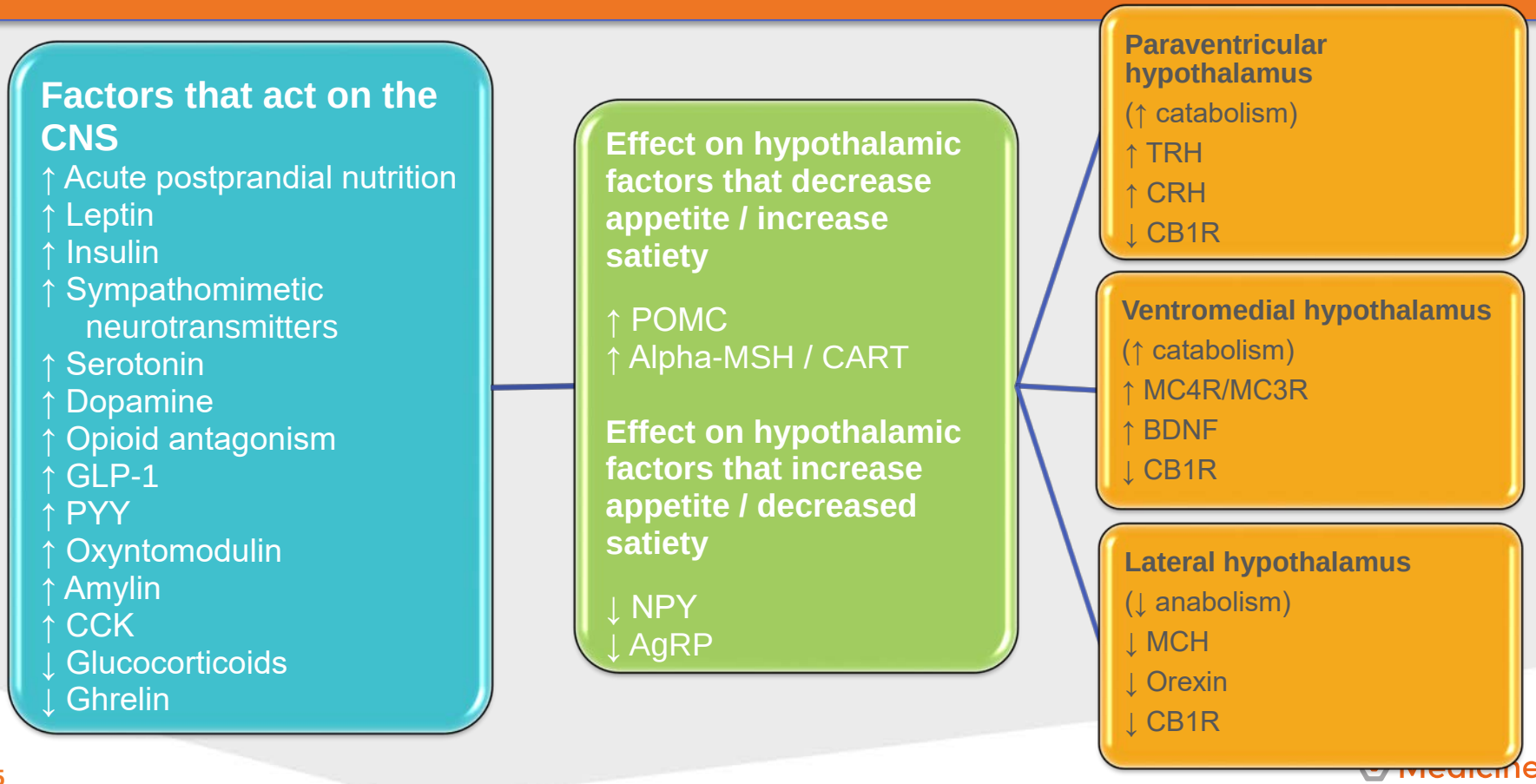
- **Improve the health of patients**
  - Improve hyperglycemia, high blood pressure, and abnormal lipid levels
  - Reduce cardiovascular events
  - Improve other adverse metabolic, biomechanical, and psychosocial health consequences, with improved quality of life.
  - Reduce mortality

- **Improve the weight of patients**
  - Weight loss to a clinically meaningful degree that patients and clinicians will embrace initiation of anti-obesity therapy
  - Weight loss maintenance to a degree that patients and clinicians will persist in adhering to long-term anti-obesity therapy

Supporting documents in reference section

Obesity Medicine Association

Illustrative Targets of Anti-Obesity Therapy



Supporting documents in reference section

Early versus Late Weight-Management Intervention: Illustrative Consequences

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

Obesity Medicine Association

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…

Supporting documents in reference section

Obesity Medicine Association

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

Obesity Medicine Association

Bariatric Surgery

Physiology, Procedures, Micronutrients, Microbiome, Complications

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Bariatric Surgery

- Regardless of the bariatric surgical procedure chosen, the surgery is best performed by an appropriately trained surgeon at an accredited surgery center.

- The accreditation of a bariatric surgery center is determined by the Metabolic and Bariatric Surgery Accreditation and Quality Improvement Program (MBSAQIP).

Potential Bariatric Surgery Candidate

What is the patient's BMI (in kg/m^2)?

Does clinical evidence exist confirming the presence of adverse health consequences (AHC) due to excessive and/or dysfunctional body fat?

- BMI ≥ 35 with one or more AHC
- BMI ≥ 40 with or without AHC

*BMI 30-34.9 with one or more AHC: Mounting evidence supports surgical intervention as a treatment option in this group

Supporting documents in reference section

Obesity Medicine Association

Bariatric Surgery Pre-operative Evaluation

- Medical evaluation by a clinician specializing in the care of patients with overweight or obesity
- Surgical consultation by bariatric surgery specialist
- Cardiology, pulmonary, gastroenterology, and/or other specialty consultation as indicated
- Mental health assessment: underlying eating disorders; mood disorders; substance abuse; history of physical or emotional trauma; education regarding potential for increased suicide risk and transfer addictions post op; evaluation of existing coping mechanisms
- Nutritional assessment (e.g., dietitian)
- Educational support (e.g., pre-operative seminar)

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)

Other FDA-approved Bariatric Technologies

Aspiration Therapy via Modified Percutaneous Endoscopic Gastrostomy (PEG)

- Mechanism: Drains 30% of ingested meal
- Indication: Body mass index 35-55 kg/m^2
- Efficacy: 12% excess weight loss at one year
- Safety: Potential tube site inflammation/infection

Electrical Vagal Blocking System

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

Other FDA-approved Bariatric Technologies

Intragastric Balloons

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

Endoscopic Plication Devices

- Mechanism: Endoscopic suturing of the stomach reduces gastric volume
- Indication: Investigational
- Efficacy: 30-50% excess weight loss for up to 1-2 years
- Safety: Stitch failure with weight regain

Bariatric Surgery: Early Complications (First 30 Days)

The complications listed here are unique to bariatric surgery and not inclusive of more general post-operative complications that can occur (e.g., pneumonia, pulmonary emboli).

Leak or Perforation (Typically after RNY, BPD/DS, VSG):

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

Bariatric Surgery: Early Complications (First 30 Days)

The complications listed here are unique to bariatric surgery and not inclusive of more general post-operative complications that can occur (e.g., pneumonia, pulmonary emboli).

Bleeding at the Surgical Site or Rarely Intraluminal/Gastrointestinal (More Likely with RNY, BPD/DS, and VSG):

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

Wound Infection (Possible after All Procedures):

- Abdominal pain, excessive drainage, fever/chills, decreased appetite, leukocytosis, change in bowel pattern
- Presence of intra-abdominal infection/abscess may require drainage percutaneously or by re-operation

Supporting documents in reference section

Obesity Medicine Association

Bariatric Surgery: Late Complications (Beyond 30 Days)

The complications listed here are unique to bariatric surgery and not inclusive of more general post-operative complications that can occur (e.g., pneumonia, pulmonary emboli).

Gastro-gastric Fistula (RNY):

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

Band Erosion through Gastric Wall into the Lumen (LAGB):

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

Bariatric Surgery: Late Complications (Beyond 30 Days)

The complications listed here are unique to bariatric surgery and not inclusive of more general post-operative complications that can occur (e.g., pneumonia, pulmonary emboli).

Incisional Hernias (More Common with Open Procedures):

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

Internal Hernias (RNY/BPD-DS):

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

Bariatric Surgery: Early *or* Late Complications

The complications listed here are unique to bariatric surgery and not inclusive of more general post-operative complications that can occur (e.g., pneumonia, pulmonary emboli).

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

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

Stricture (Stomal Stenosis) (RNY or BPD-DS):

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

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

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

Bariatric Surgery: Early *or* Late Complications

The complications listed here are unique to bariatric surgery and not inclusive of more general post-operative complications that can occur (e.g., pneumonia, pulmonary emboli).

Dumping Syndrome (RNY):

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

Gallbladder or Gallstone Disease:

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

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

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

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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-op.

For a complete explanation of micronutrient deficiencies, refer to “Clinical Practice Guidelines for the Perioperative Nutritional, Metabolic, and Nonsurgical Support of the Bariatric Surgery Patient” at www.asmbs.org.

Supporting documents in reference section

Obesity Medicine Association

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

Micronutrient Deficiency Replacement after Bariatric Surgery: Vitamins/Minerals

Vitamin/ Mineral	Assessment	Replacement of Deficiency & Maintenance
Vitamin A	Retinol	• If deficiency with corneal keratinization, ulceration or necrosis: 50-100,000 IU IM for 3 days, followed by IU IM for 2 weeks; if no corneal changes: 10,000 - 25,000 IU orally for 1-2 weeks • Further treatment depends on persistent malabsorptive effects, as may most be a concern with biliopancreatic diversion/duodenal switch, which may require maintenance oral vitamin A at least 5000 IU per day
Vitamin B1 (Thiamine)	Thiamine	• If hyperemesis, then 100mg IV for 7 days, then 50 mg/d until thiamine in normal range, and then maintenance oral vitamin B1 of at least 3 mg per day.
Vitamin B9 (Folate)	Red blood cell (RBC) folate	• If daily multivitamin has 400ug of folic acid, then replacement dose for deficiency is an additional 800 ug/d orally (total of 1200 ug/d of folic acid until RBC folate in normal range), and then a multivitamin with at least 500 ug/d of folic acid
B12 (Cobalamin)	Vitamin B12	• A typical dose to treat B12 deficiency 1000 ug/mo IM, 1000 ug/wk sublingually, or 350-500 ug/d orally until B12 in normal range. Maintenance dose may include 500 – 1000 ug oral vitamin B12 per day.
Calcium	Calcium	• In addition to ensuring adequate vitamin D, calcium deficiency is typically treated with calcium citrate 1200-1500 mg/d. Calcium citrate may be better absorbed than calcium carbonate • Calcium should be taken at least 1 hour apart from other supplements, especially iron (which competes for absorption)
Iron	Ferritin, iron, total iron binding capacity	• For moderate deficiency, menstruating women, or patients at risk for iron deficiency anemia, total elemental iron oral intake (including in a multivitamin) is often 150 - 200 mg/d • Iron supplementation may be more effective with vitamin C supplementation 500 mg/d • For severe deficiency, IV iron is sometimes required, which is provided in multiple different formulations, some of which require test doses.

Micronutrient Deficiency Replacement after Bariatric Surgery: Vitamins/Minerals

Vitamin/Mineral	Assessment	Replacement of Deficiency & Maintenance
Vitamin D	25-hydroxyl-(OH)-vitamin D	• A typical oral dose for mild deficiency of vit. D3 is 3000 IU/d, followed by at least 1000 IU/d after gastric bypass and 2000 IU/d after biliopancreatic diversion/duodenal switch once normal range vitamin D levels are achieved • For severe deficiency (e.g., biliopancreatic diversion), IM 100,000 IU vitamin D3 once per month, or otherwise, vitamin D2 50,000 IU/wk orally until vit. D levels in normal range, then D3 3000 IU if still with substantial malabsorptive signs and symptoms, or if stable with vitamin D values in the normal range, then at least D3 1000 IU/d after gastric bypass and D3 2000 IU/d after biliopancreatic diversion/duodenal switch. • Regarding formulation, vit. D2 (ergocalciferol) is a form of dietary vit. D found in plants. Vit.D3 (cholecalciferol) is found in foods of animal origin and is similar to the vit. D3 generated when 7-dehydrocholesterol in the skin is converted by ultraviolet radiation from sunlight. Both D2 and D3 are reported as 25-hydroxyvitamin D, which is then converted by the kidneys into the more active 1,25 dihydroxyvitamin D (calcitriol). Vit. D3 may be preferred (longer half-life and potentially more potent) than vit. D2. Although the most potent, calcitriol is more rarely used (.25 or .50 mcg/d orally)
Vitamin E	A-Tocopherol	• A typical dose to treat vitamin E deficiency is 400 to 800 IU/d orally, with oral vitamin E 400 IU/d especially for biliopancreatic diversion.
Vitamin K	Prothrombin time	• If vitamin K deficiency occurs during substantial gastrointestinal malabsorption, then vitamin K can be replaced 10 mg by slow IV. Otherwise, typical oral replacement dose is 4 mg or 300 ug/d. Continued treatment depends on persistent malabsorptive effects, as may most be a concern with biliopancreatic diversion/duodenal switch.
Zinc	Zinc	• A typical replacement dose for zinc deficiency is 60 mg of elemental zinc twice daily. Zinc consumption may impair copper absorption, thus 1 mg of copper should be given per each 10 mg of zinc administered. Once zinc is in normal range, if malabsorption remains a risk, a typical supplemental dose is zinc 30 mg/d. If malabsorption less of a risk, then a common dose of zinc is 8 – 15 mg per day.

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Microbiome: Gut Flora Basics

Microbiome = Collection of micro-organisms

Microbiota = Organisms themselves

Microbiome: Gut Flora Bacteria

- Over 1,000 bacterial species, with over 90% anaerobic
- Substrates: Sloughed intestinal cells, plant polysaccharides, starch cellulose, and bile components
- Functions include:
  - Metabolizing essential nutrients
  - Synthesizing vitamin K
  - Fermentation of sugars to acids, gasses, or alcohol
  - Digesting cellulose
  - Promoting angiogenesis
  - Enhancing enteric nerve function

Microbiome: “Favorable” Weight and Metabolic Effects of Bariatric Surgery

Bariatric Surgery May:

- Reduce availability of nutrients delivered to the gut
- Reduce lipogenic signaling (gut and systemic)
- Reduce inflammation (gut and systemic)
- Alter bile-acid metabolism and increase bile-acid pool favoring metabolic processes involving glucose and lipids
- Alter gut hormones favoring metabolic processes involving glucose and lipids
- Decrease the Firmicutes:Bacteroidetes ratio, potentially reducing the efficiency of extracting calories from gut carbohydrates

Executive Summary

Obesity Medicine Association

CLINICAL LEADERS IN OBESITY MEDICINE®

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

Obesity Medicine Association

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 very low-calorie diets. Obesity is a chronic medical disease and often requires lifelong treatment.

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Evaluation and Treatment Summary

Comprehensive Evaluation of the Patient with Overweight/Obesity

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

*lab and diagnostic testing should be individualized

Individualized Treatment Plans*

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

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

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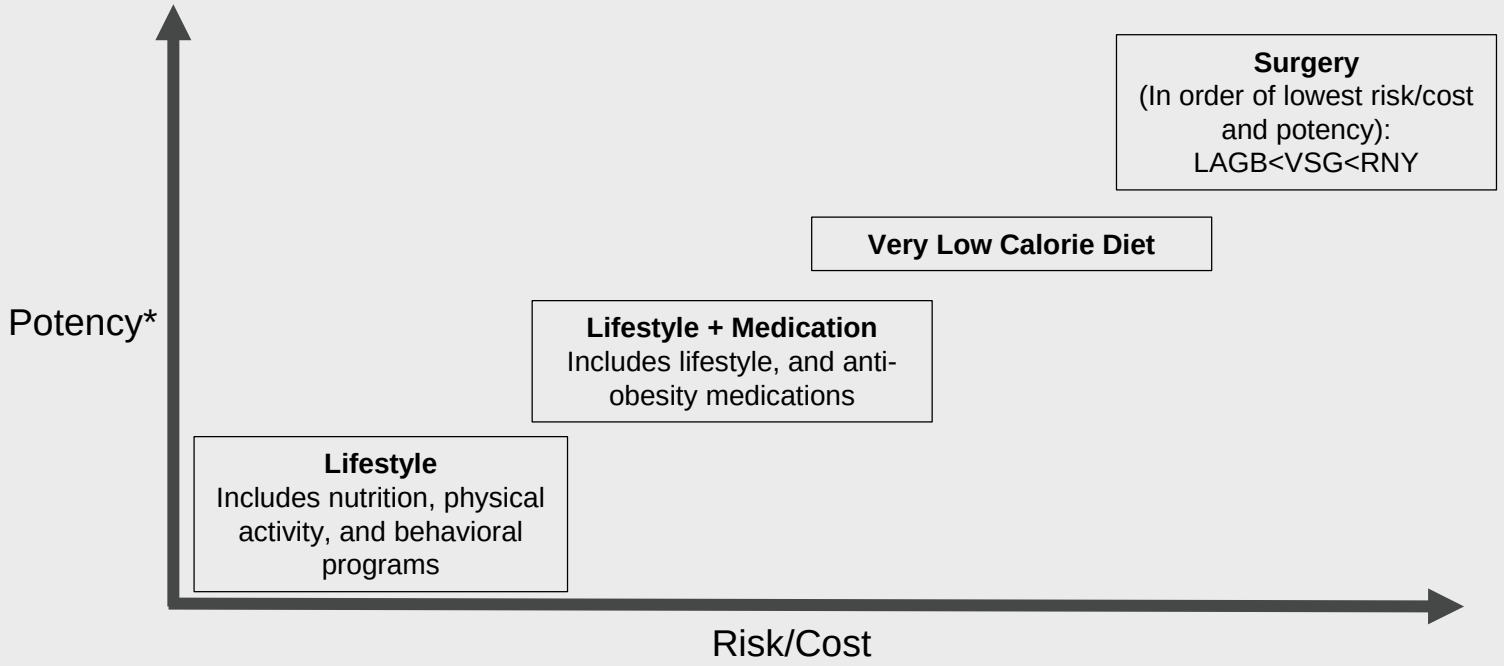
Current Treatment Options for Obesity



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

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Disclosures

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Disclosures

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