Pediatric Obesity Algorithm®

2020-2022

Pediatric Obesity Algorithm

Disclaimer
The Pediatric Obesity Algorithm, was developed to assist health care professionals in medical decision making in the management and care of patients with overweight and obesity. The Pediatric 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 the medical literature and the clinical experience of obesity medicine specialists. In areas regarding inconclusive or insufficient scientific evidence, the authors used their professional judgment.

The Pediatric Obesity Algorithm is a working document that represents 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 and individual patient circumstances.

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Purpose

To provide health care professionals an algorithm to guide the treatment of children and adolescents with increased body fat, based upon scientific evidence, supported by the medical literature, and derived from the clinical experiences of members of the Obesity Medicine Association.
The Pediatric Obesity Algorithm was derived from input by volunteer OMA members consisting of:

- Academicians
- Clinicians
- Researchers

The Pediatric Obesity Algorithm did not receive industry funding, had no input from industry, and the authors received no payment for their contributions.

Intent of Use

The Pediatric Obesity Algorithm 2020/2022 is intended to be a “living document” updated as needed. It is intended as an educational tool to assist in the translation of medical science and the clinical experience of the authors towards assisting health care professionals improve management of their pediatric patients with overweight and obesity.

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

While it is hoped many clinicians may find this algorithm helpful, the final decision regarding the optimal care of the patient with overweight or 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.
Overall Management Goals

Pediatric patient with overweight or obesity

Develop healthy habits and lifestyle patterns through adulthood

Improve health & quality of life

Improve body composition

Prevent future adverse health consequences

Improve body image and self esteem
Epigenetics

- Heritable regulation of gene expression without a change in the base sequence of DNA

Common Manifestations of Epigenetics in Childhood Obesity

**Small for Gestational Age Infants (Intrauterine Growth Restriction)**
- Tobacco abuse during pregnancy
- Use of folic acid may attenuate the effect
- Insufficient gestational weight gain

**Large for Gestational Age Infants**
- Mothers with preconception BMIs > 30 kg/m²
- Mothers with excessive gestational weight gain
- Gestational diabetes mellitus

**Very Low Birth Weight Infants (Premature infants)**
- Undergo significant periods of undernutrition following birth
- Commonly leave the NICU at a smaller size than counterparts who remained in utero
- Undergo a period of catch-up growth when they are provided with high nutrient formula or fortified human milk
- Have higher levels of visceral fat suggesting a set up for higher incidence of CV disease and T2DM
- Often receive prolonged courses of antibiotics
- Highly stressed

Reference/s: [1]
The Maternal Resource Hypothesis: Each generation produces larger and more metabolically compromised mothers

- Less maternal disease (as compared to past generations) + decreased physical activity + improved nutrition → increased maternal BMI and adiposity
- Increased maternal insulin resistance → stimulates fetal pancreatic beta cells and adipocyte hyperplasia
- Adipocyte hyperplasia establishes a competitive dominance over other tissues
- Intense postprandial insulin response and the relative number of adipocytes is so large that the competitive dominance of adipocytes is inevitable and obesity unavoidable

Factors Associated with Epigenetic Changes that Increase Risk of Childhood Obesity

Maternal Insulin Resistance
- Maternal insulin resistance (IR), not prepregnancy BMI, with or without glucose intolerance predicts weight gain and adiposity in infant from 0-12 months
- Genes for lipid, amino acids, and inflammatory pathways unregulated with maternal IR
- Maternal IR causes specific defects in maternal skeletal muscle that can persist for 12 months after birth, increasing risk for T2DM in mother and higher risk in future pregnancies

C section delivery
- “Iatrogenic Artificial Selection”
- Larger infants secondary to excessive fetal growth leads to larger infant head circumferences leading to increased number of C section deliveries
- Increased numbers of C sections has facilitated the survival of larger infants and the mothers who produced them
- C section delivery is strongly associated with childhood obesity
- The frequency of C section births is greatest in the population that is the most inactive, sedentary and obese

Neonatal Intestinal Microbiome
- Before delivery through first 2 years of life
- Colonization of the neonatal biome may be due to transvaginal migration of organisms or translocation from the maternal GI tract
- Obesity in pregnancy associated with higher levels of Bacteroides, Clostridium and Staphylococcus (associated with increased energy harvest from diet) and lower levels of bifidobacterium than normal weight
- Intestinal colonization of infants delivered by C section closely related to maternal skin microbes, vs vaginally delivered infants, which resemble vaginal microbes
Factors Associated with Epigenetic Changes that Increase Risk of Childhood Obesity

**Exposure to Environmental Toxins**
- Microbes associated with low dose penicillin exposure can induce obese phenotype
- Nutrients i.e. Folate, methionine, choline, betaine, vitamin B12 can change DNA methylation status
- Bioactive food components (curcumin, genistein, retinoic acid) can cause micro RNAs to down regulate target RNA

**Postnatal Exposures (First 1,000 days)**
- Bottle fed
- Early introduction of complementary foods
- Maternal and paternal diets high in CHOs, low in fruits and vegetables
- Coercive feeding or reward feeding
- Poor parental role models
- Inappropriate amount of sleep and physical activity

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Antibiotic Administration and the Development of Obesity in Children

**Childhood Exposure to Antibiotics**
- 33% of pregnancies
- 45% of neonates exposed in prenatal period
- Mothers with overweight more likely to receive antibiotics
- Antibiotic treatment highest with C section
- 88% of premature and low birth weight
- By age 2, 3 antibiotic courses
- By age 10, 10 courses
- By age 20, 17 courses
- Also exposed through the food chain

**Epidemiological Evidence**
- Antibiotic mediated promotion of growth in animals widely practiced
- Effect on children: greater if prescribed before 6 months of age, boys > girls, higher cumulative orders associated with progressive weight gain
- Exposure to broad-spectrum antibiotics and macrolides have a stronger association

**Microbiota Modification**
- Germ free animals (no microbiota) require 30% more calories to maintain their body mass
- Gut microbiota allow host to digest otherwise indigestible complex plant polysaccharides to monosaccharides and short chain fatty acids
- Animals in overweight category have a 50% reduction in Bacteroides and a proportional increase in Firmicutes
- Antibiotics change the milieu, disrupt immune defenses at the intestinal border, resulting in increased inflammatory and metabolic disorders

References: [5] [6] [7] [8] [9]
### The Association of Antibiotics and the Development of Obesity

<table>
<thead>
<tr>
<th>Effects on Mitochondria</th>
<th>Effect on the Microbiome</th>
<th>Circumstantial Evidence</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Antibiotics both decrease the number of mitochondria and impair their function</td>
<td>• Early exposure is associated with resistance developing in probiotic microorganisms</td>
<td>• Strong association in meta-analyses for the association between antibiotic exposure in early life and childhood adiposity</td>
</tr>
<tr>
<td>• Mitochondria are important in maintaining energy metabolism</td>
<td>• Intestinal microbiota exposed to antibiotics show reduced diversity</td>
<td>• Strong dose-response relationship between antibiotic exposure and childhood adiposity</td>
</tr>
<tr>
<td>• Evidence suggests that antibiotics cause mutations in the mitochondrial genome</td>
<td>• Data from animal studies shows antibiotic induced changes in gut microbiota can result in fat accumulation by changing host metabolism</td>
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<tr>
<td>• Mitochondrial genome shares common pathways with bacteria</td>
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### The Gut Microbiome

<table>
<thead>
<tr>
<th>What is the gut microbiome?</th>
<th>Epidemiological Evidence</th>
<th>Microbiota Modification</th>
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<tbody>
<tr>
<td>• The diverse organisms including bacteria, fungi, and viruses that inhabit our intestinal tract</td>
<td>• Low levels of Bifidobacterium and increased levels of <em>Staphylococcus aureus</em> in infancy have been associated with being overweight at age seven</td>
<td>• Early colonization influenced by:</td>
</tr>
<tr>
<td>• Gut bacteria are important to development and maturation of the immune system and in harvesting energy and essential vitamins and minerals during digestion</td>
<td>• Low dose antibiotic exposure in mice has been demonstrated to lead to increased adiposity</td>
<td>• Maternal factors including stress, antibiotic use, smoking, and diet</td>
</tr>
<tr>
<td>• Development occurs in early life and continues through adolescence before reaching a stable adult state</td>
<td>• Transfer of obese phenotype mice to germ-free mice through transfer of the microbiome</td>
<td>• Gestational age at birth</td>
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<tr>
<td></td>
<td>• In animal models, there is a relative reduction in Bacteroidetes and increase in Firmicutes in obese mice compared to lean mice leading to increased energy harvesting from the diet</td>
<td>• Birth order</td>
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<tr>
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<td>• Infant diet</td>
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<td>• Antibiotic administration</td>
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<td></td>
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<td>• External living environment</td>
</tr>
</tbody>
</table>

Reference/s: [10] [11] [12]
# Gestational Diabetes Mellitus (GDM)

## Incidence and Associations
- 18% of pregnancies
- Glycemic status of the mother usually returns to normal by day 15 postpartum
- Being born to a mother with both GDM and overweight has the highest risk for childhood obesity
- Maternal hyperglycemia leads to fetal hyperinsulinemia which increases fetal growth and birth weight
- Higher birth weight strongly associated with childhood obesity

## Impact of Breast Feeding
- GDM infants fed milk from biological mothers during 1st week of life had higher body weight at 2 years of age than GDM infants fed donor milk from non-GDM women indicating that 1st week of life is critical period for nutritional programming
  - Possibly due to breast milk composition that is altered by glycemic status but normalizes through time
- Hispanic low income children exposed to GDM required breast feeding duration of ≥12 months to reduce obesity prevalence

## Complementary Feeding
- Associated with an increase in protein intake from 5% (breast milk) to 25% of diet
- Higher protein intake in early life associated with higher BMI at 2 years
- Added sugar intake enhances preference for sweet foods
- Early complementary feeding (<13 weeks) associated with greater caloric intake at 22 weeks
- Early complementary feeding not known to be different in non-GDM vs GDM infants but may exacerbate already elevated risk for childhood obesity

## Physical Activity
- Physical activity levels are similar among non-GDM and GDM children but the impact of similar amounts of activity on a high risk population is unknown

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**Assessment**

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[obesitymedicine.org](http://obesitymedicine.org)
Weight Assessment in Children with Obesity

Weight Assessment by Age Group and Disease Severity

Weight Assessment for Children ages 0-2 years

BMI percentile in Children Ages 2-20 years

BMI for Children with Severe Obesity Ages 2-20 years

Weight Assessment for Children ages 0-2 years

Measurement

- BMI is not used until 2 years of age
- To assess weight status in an infant, use weight for length

Tool

- Growth charts for infants are available through the CDC and the WHO
  - CDC is based on a cohort of mostly white American children, mostly non-breast fed
  - WHO is based on children from diverse racial and ethnic backgrounds, mostly breast fed

Reference/s: [18] [19]
WHO Weight for Length Growth Charts

CDC Weight for Length Growth Charts
Body Mass Index Categories in Children Ages 2 to 20 years

Caveat: Not all patients with BMI 85% or above have excess adiposity, and many children and adolescents with BMI < 5% are healthy and do not need treatment.

The CDC recommends using the WHO growth charts to monitor growth for infants and children ages 0 to 2 years of age in the U.S. and using the CDC growth charts for children age 2 years and older.

Body Mass Index Percentile Ages 2 to 20 Years

- **Underweight**: < 5th percentile
- **Healthy Weight**: 5-84th percentile
- **Overweight**: 85-94th percentile
- **Obesity**: 95-99th percentile or BMI > 30
- **Severe Obesity**: BMI > 120% of the 95th percentile or BMI ≥ 35 kg/m²

Body Mass Index Charts for Children with Overweight and Obesity Ages 2-20 years

Assessing the Child with a BMI above the 95th Percentile Assessment

### Indications
- System to express severe obesity in youth as a percentage above the 95th percentile
- Statistically: unsmoothed 99th percentile similar to 120% of the 95th percentile of BMI for age
- 2012, new growth charts created to augment Centers for Disease Control & Prevention (CDC) charts allowing clinicians to track and visualize BMI values in children with severe obesity over the 99th percentile.
- These growth charts define a child/adolescent’s BMI as a “percentage of the 95th percentile” (%BMIp95)

### Definition
- Expanded definition of severe obesity includes Class I, II, and III obesity
  - Class I obesity (≥95th percentile to <120% of the 95th percentile)
  - Class II obesity (>120% to <140% of the 95th percentile) or a BMI ≥ 35 to ≤ 39, whichever lower
  - Class III obesity (>140% of the 95th percentile) or BMI ≥ 40, whichever lower.
- Class I obesity = 95th-99th percentile or Obesity
- Class II & III = > 99th percentile or Severe Obesity

### BMI z Score
- BMI z score should not be used to assess BMI changes among children with BMIs > 120% of the 95th percentile
- High BMI z scores are compressed into a narrow range, which can result in a clinically significant reduction in BMI being represented by a small reduction in BMI z score
- Changes in the % of the 95th percentile is the preferred metric to use in the management of children with severe obesity
- The same BMI z value maps to substantially different levels of %BMIp95 depending on age and sex

**Reference/s:** [22] [23] [24]

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Assessing the Child with a BMI above the 95th Percentile

### Definition
- BMI ≥ 120% of the 95th percentile (1.2 x 95th percentile)
  - or
- An absolute BMI ≥35 kg/m², whichever is lower based on age and sex

### As Compared To Adult
- The inclusion of an absolute BMI threshold (35 kg/m²) aligns the pediatric definition with class II obesity in adults, a high-risk category of obesity associated with increased morbidity and mortality

### Recommendations
- BMI 35 kg/m² is a higher threshold than BMI ≥120% of the 95th percentile among most children, but it is a somewhat lower threshold (and therefore expands the population that is categorized as severely obese) among boys ≈18 years and girls ≈16 years and older.

**Reference/s:** [22] [25]
Body Mass Index Charts for Children with Severe Obesity ages 2-20 yrs

Obesity as a Disease

obesitymedicine.org

Obesity as a Disease

Endocrine/Immune Response
- Adiposopathy
  - Impaired fasting glucose
  - Metabolic Syndrome
  - Hypertension
  - Menstrual Dysfunction (girls)
  - Early Puberty (girls)
  - Delayed Puberty (boys)
  - NAFLD
  - Dyslipidemia
  - Insulin Resistance
  - Type 2 DM
  - Increased uric acid, microalbuminuria
  - Gynecomastia
  - Cholecystitis

Physical Response
- Fat Mass Disease
  - Asthma
  - Immobility
  - Lipomastia
  - Tissue Compression (sleep apnea, GERD, HTN)
  - Tissue Friction (intertrigo)
  - Stress on weight-bearing joints
  - Slipped capital femoral epiphysis, Blount's disease, scoliosis, osteoarthritis

Psychological Response
- Quality of Life
  - Isolation from peers
  - Decrease in ability to participate in normal childhood activities
  - Subject to bullying
  - Lack of social/age appropriate relationships
  - Anxiety/depression
  - Binge eating disorder
  - Night eating disorder
  - Bulimia

Differential Diagnosis
### Childhood Obesity: Differential Diagnosis

#### Linear growth in pre-pubertal and pubertal children
- Consistent or accelerated linear growth
  - Consider endocrinopathy
  - Test for TSH, Free T4, dexamethasone suppression test, 24-hour urinary free cortisol if indicated
- Decreased linear growth
  - Consider exogenous obesity; nutritional origin
  - Consider precocious puberty if secondary sexual development at < 8 yrs. for girls (breast development) and < 9 yrs. for boys (enlarged testicular size)
  - Consider bone age

#### Developmental delay; suspect syndromal obesity
- Can be associated with decreased linear growth
- Evaluation dependent on presentation and family history
- Refer to genetics, consider genetic testing

#### Early onset obesity (before 5 years of age; ≥120% percent of the 95th percentile)
- Genetic testing recommended with clinical feature such as extreme hyperphagia and/or family history of extreme obesity
- Clinical history may include food seeking behavior such as searching for or stealing food, waking in night to eat, and eating food left behind by others (exclude neurological causes).
- Behaviors result from disruption of hypothalamic pathways involved in the regulation of energy balance.
- Consider multi-gene obesity panel

### Review of Systems

[obesitymedicine.org](https://www.obesitymedicine.org)
### Focused Review of Systems

<table>
<thead>
<tr>
<th>Symptoms</th>
<th>Related Co-Morbidity</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nervousness, school avoidance, social inhibitions</td>
<td>Depression, anxiety, bullying</td>
</tr>
<tr>
<td>Fatigue, Muscle aches</td>
<td>Vitamin D deficiency</td>
</tr>
<tr>
<td>Polyuria, polydipsia, fatigue, nocturia</td>
<td>Type 2 Diabetes (T2DM)</td>
</tr>
<tr>
<td>Headaches, facial numbness</td>
<td>Idiopathic Intracranial Hypertension (Pseudotumor cerebri)</td>
</tr>
<tr>
<td>Skin pigmentation, skin tags</td>
<td>Insulin resistance (IR)</td>
</tr>
<tr>
<td>Daytime somnolence, loud snoring, witnessed apnea, attention deficit</td>
<td>Obstructive sleep apnea (OSA)</td>
</tr>
<tr>
<td>Abdominal pain, indigestion</td>
<td>Gastroesophageal reflux disease (GERD), gall bladder disease, constipation</td>
</tr>
<tr>
<td>Hip or knee pain</td>
<td>Slipped capital femoral epiphysis (SCFE), early osteoarthritis</td>
</tr>
<tr>
<td>In-toeing, leg bowing, mild knee pain</td>
<td>Blount’s disease</td>
</tr>
<tr>
<td>Hirsutism, acne, irregular menses</td>
<td>Polycystic Ovarian Syndrome (PCOS)</td>
</tr>
</tbody>
</table>

### Diagnostic Work Up
Diagnostic Work-Up: Taking the History in Infants 0-12 months

<table>
<thead>
<tr>
<th>Family History/Prenatal Factors</th>
<th>Feeding</th>
<th>Miscellaneous</th>
</tr>
</thead>
<tbody>
<tr>
<td>Maternal/Paternal obesity</td>
<td>Duration of exclusive breast feeding</td>
<td>Amount of screen time</td>
</tr>
<tr>
<td>Gestational DM/Weight gain</td>
<td>Timing of early introduction of complementary foods</td>
<td>Juice</td>
</tr>
<tr>
<td>Siblings with obesity</td>
<td>Early introduction of cereal (&lt;6 months of age)</td>
<td>Sugar Sweetened Beverages</td>
</tr>
<tr>
<td>Birth Weight (small or large for gestational age)</td>
<td></td>
<td>Sleep duration</td>
</tr>
<tr>
<td>Family Hx of Early CVD</td>
<td></td>
<td>Multiple courses of antibiotics</td>
</tr>
</tbody>
</table>

Diagnostic Work-Up: Taking the History in Children with Obesity Ages 1-18 years

<table>
<thead>
<tr>
<th>Toddler (Age 1-4 years)</th>
<th>Early Childhood (Age 5-9 years)</th>
<th>Puberty (Age 10-14 years)</th>
<th>Adolescent (Age 15-18 years)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Exercise</td>
<td>Active Play</td>
<td>Vigorous Exercise for 60 min or more every day</td>
<td></td>
</tr>
<tr>
<td>Feeding</td>
<td>Food as reward or punishment Diversity in diet</td>
<td>Family Meals Eating out Fast food/Sugar Sweetened Beverages</td>
<td>Modified meals or pack lunch Continue to challenge vegetables and fruits</td>
</tr>
<tr>
<td>Miscellaneous</td>
<td>Screen time Parents as role models</td>
<td>Bullying</td>
<td>Sedentary Time Sleep Duration Snoring</td>
</tr>
</tbody>
</table>

References: [29] [30] [31] [32]
## Diagnostic Work Up: Labs and Studies

<table>
<thead>
<tr>
<th>Infancy (0-24 months)</th>
<th>Toddler (Age 2-4 years)</th>
<th>Early Childhood (Age 5-9 years)</th>
<th>Puberty (Age 10-14 years)</th>
<th>Adolescent (Age 15-18 years)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Weight&gt;Length</strong></td>
<td><strong>BMI ≥ 95th percentile</strong>&lt;br&gt;Or&lt;br&gt;<strong>BMI ≥ 85th percentile with 2 or more risk factors (24-48 months)</strong></td>
<td><strong>BMI ≥ 95th percentile</strong>&lt;br&gt;Or&lt;br&gt;<strong>BMI ≥ 85th percentile with 2 or more risk factors</strong></td>
<td><strong>BMI ≥ 95th percentile</strong>&lt;br&gt;Or&lt;br&gt;<strong>BMI ≥ 85th percentile with 2 or more risk factors</strong></td>
<td><strong>BMI ≥ 95th percentile</strong>&lt;br&gt;Or&lt;br&gt;<strong>BMI ≥ 85th percentile with 2 or more risk factors</strong></td>
</tr>
</tbody>
</table>
| - Fasting Blood Glucose and/or HbA1c<br>- Fasting Lipid Panel/Non fasting if fasting not feasible<br>- ALT, AST, consider GGT<br>- Consider 25 OH Vitamin D<br>- BP annually if ≥ 3 years | - Consider Sleep Study<br>- Consider Liver Ultrasound<br>- Consider Uric Acid<br>- Consider fasting serum insulin | - Consider Urine Microalbumin/Creatinine ratio<br>- Consider C-peptide, hs-CRP

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## Physical Exam

Visit [obesitymedicine.org](http://obesitymedicine.org) for more information.
Physical Exam- Common Clinical Findings

Acanthosis nigricans

Abdominal striae

Gynecomastia

Hirsutism

Common Physical and Radiologic Findings

**Tonsillar Hypertrophy**

**Steatosis and Increased Abdominal Visceral Fat**

- Increased echogenicity
- VF 5.43 cm

Blount’s Disease

Pictures taken with consent of patients

Special Populations
Special Populations: Children with Obesity and Turner Syndrome (TS): glucose metabolism

**Background**
- Abnormal glucose metabolism is seen in 70% of girls with TS
- Impaired glucose tolerance, hyperinsulinemia, reduced insulin sensitivity
- There is an increased prevalence of diabetes in girls with TS (T1 and T2)
- The abnormal glucose metabolism may result from deletion of genes related in insulin signal transduction and beta cell function that are located on the X chromosome

**Health Impact**
- Girls with TS have increased waist circumference and higher adipose content both contributing to obesity
- Development of obesity and cardiovascular disease in childhood strongly predicts the development of T2DM in adolescence
- Girls with TS have restricted vertical growth but continue to grow horizontally leading to total and visceral fat mass being elevated while lean body mass is decreased
- Girls with TS generally have lower VO2 max and decreased activity comparatively

**Screening Tool/Recommendations**
- Link for Turner Syndrome BMI curve based on European data:
**Special Populations: Children with Obesity and Down Syndrome (DS)**

### Background
- Children with DS typically have short stature, small head circumference and normal to high relative weight for length and BMI.
- Obesity is seen in most children with DS.
- Children with DS have shorter limbs and poor coordination leading to less activity and a propensity to gain weight.

### Tracking BMI in DS
- BMI charts have been developed based on relatively small populations.
- AAP recommends that clinicians should use BMI guidelines from the CDC for normally developing children to classify BMI status although they recommend using DS specific curves to track height and weight.

### Screening Tool/Recommendations
- Link for Down Syndrome height and weight curves.
- [https://doi.org/10.1016/j.jped.2016.04.005](https://doi.org/10.1016/j.jped.2016.04.005)

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**Special Populations: BMI Charts Down Syndrome**

<table>
<thead>
<tr>
<th>Age (Years)</th>
<th>BMI (kg/m²)</th>
</tr>
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<tbody>
<tr>
<td>2</td>
<td>3</td>
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Down syndrome (BMI) Boys: 2 to 18 years

Down syndrome (BMI) Girls: 2 to 18 years

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Pediatric Obesity Algorithm® ©2020-2022 Obesity Medicine Association. References: [39] [40] [41]
### Diagnosis
- Achondroplasia is the most common dwarfing syndrome
- 1 in 26,000-28,000 births
- Mutation in the FGFR3 gene results in continuous activation of the FGFR3 protein with inhibits bone growth

### BMI Differences as Compared to Normally Developed Children
- BMI is substantially higher in children with achondroplasia than in normally developing peers
- Cut off values for the 95th percentile are different due to the difference in body proportions
- Use achondroplasia specific curves to evaluate obesity, not the actual BMI measurement
- Pattern of change of BMI is different
- Increase in height is particularly compromised during infancy and puberty as compared to normally developing children
- No pubertal growth spurt
- No "J" shaped curve in children with achondroplasia; no adiposity rebound

### Complications
- Cardiovascular disease
- Obstructive sleep apnea
- Difficulty with mobility
- Link to BMI chart: DOI: 10.1093/ajcn/88.2.364

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### Special Populations: BMI Charts for Children with Achondroplasia

#### Boys

<table>
<thead>
<tr>
<th>AGE (y)</th>
<th>5th</th>
<th>50th</th>
<th>95th</th>
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<tbody>
<tr>
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#### Girls

<table>
<thead>
<tr>
<th>AGE (y)</th>
<th>5th</th>
<th>50th</th>
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<td>16</td>
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</tr>
</tbody>
</table>
## Nutritional Recommendations

### General Intake Guidelines (Normal Weight): 0 to 12 months

<table>
<thead>
<tr>
<th></th>
<th>Birth- 4 months</th>
<th>4-6 months</th>
<th>6-8 months</th>
<th>8-10 months</th>
<th>10-12 months</th>
</tr>
</thead>
<tbody>
<tr>
<td>Breast milk and/or fortified infant formula</td>
<td>8 to 12 feedings</td>
<td>4 to 6 feedings</td>
<td>3 to 5 feedings</td>
<td>3 to 4 feedings</td>
<td>3 to 4 feedings</td>
</tr>
<tr>
<td></td>
<td>*2 to 6oz per feeding (18-32oz per day)</td>
<td>*4 to 6oz per feeding (27-45oz per day)</td>
<td>*6 to 8oz per feeding (24-32oz per day)</td>
<td>*7 to 8oz per feeding (24-32oz per day)</td>
<td>*24-32oz per day</td>
</tr>
<tr>
<td>Cereal, breads, starches</td>
<td>None</td>
<td>None</td>
<td>2-3 servings of iron-fortified baby cereal (serving= 1 to 2 Tbsp)</td>
<td>2-3 servings of iron-fortified baby cereal (serving= 1 to 2 Tbsp)</td>
<td>4 servings of iron-fortified bread or other soft starches or baby cereal (serving= 1 to 2 Tbsp)</td>
</tr>
<tr>
<td></td>
<td>None</td>
<td>None</td>
<td>Offer plain, cooked, mashed, or strained baby foods vegetables and fruits. Avoid combination foods. No juice.</td>
<td>2-3 servings (1 to 2 Tbsp) of soft, cut-up, and mashed vegetables and fruits daily. No juice.</td>
<td>4 servings (2 to 3 Tbsp) daily of fruits and vegetables. No juice.</td>
</tr>
<tr>
<td>Fruits and Vegetables</td>
<td>None</td>
<td>None</td>
<td>Offer plain, cooked, mashed, or strained baby foods vegetables and fruits. Avoid combination foods. No juice.</td>
<td>2-3 servings (1 to 2 Tbsp) of soft, cut-up, and mashed vegetables and fruits daily. No juice.</td>
<td>4 servings (2 to 3 Tbsp) daily of fruits and vegetables. No juice.</td>
</tr>
<tr>
<td>Meats and Other Protein Sources</td>
<td>None</td>
<td>None</td>
<td>Begin to offer plain-cooked meats. Avoid combination dinners.</td>
<td>Begin to offer well-cooked, soft, finely chopped meats.</td>
<td>1 to 2oz daily of soft, finely cut or chopped meat or other protein foods</td>
</tr>
</tbody>
</table>

While there is no comprehensive research indication which complimentary foods is best to introduce first, focus should be on first foods that are higher in iron and zinc such as pureed meats and fortified-iron rich foods.
### General Intake Guidelines (Normal Weight): 1 to 4 years

<table>
<thead>
<tr>
<th></th>
<th>12-23 months</th>
<th>2-3 years</th>
<th>3-4 years</th>
</tr>
</thead>
<tbody>
<tr>
<td>Milk and Milk Products</td>
<td>2 cups/day (whole milk or milk products)</td>
<td>2 to 2.5 cups/day</td>
<td>2.5-3 cups/day</td>
</tr>
<tr>
<td>Serving:</td>
<td>1 cup of milk or cheese, 1/3 oz of natural cheese, 1/3 cup shredded cheese</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Meat and Other Protein Foods</td>
<td>1 1/2 oz/day</td>
<td>2 oz/day</td>
<td>2-3 oz/day</td>
</tr>
<tr>
<td>Serving:</td>
<td>(1oz equivalent) = 1oz beef, poultry, fish, 1/4 cup cooked beans, 1 egg, 1Tbsp peanut butter*, 1/2 oz of nuts*</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Breads, Cereal, and Starches</td>
<td>2 oz/day</td>
<td>2 oz/day</td>
<td>2-3 oz/day</td>
</tr>
<tr>
<td>Fruits</td>
<td>1 cup/day</td>
<td>1 cup/day</td>
<td>1-1 1/2 cups/day</td>
</tr>
<tr>
<td>Vegetables (non-starchy vegetables to include sources of vitamin C and A)</td>
<td>3/4 cup/day</td>
<td>1 cup/day</td>
<td>1-1 1/2 cups/day</td>
</tr>
<tr>
<td>Fats and Oil</td>
<td>Do not limit*</td>
<td>3 tsp/day</td>
<td>3-4 tsp/day</td>
</tr>
<tr>
<td>Miscellaneous</td>
<td>desserts, sweets, soft drinks, candy, jams, jelly</td>
<td>none</td>
<td>none</td>
</tr>
</tbody>
</table>

*peanut butter and nuts may be a choking hazard under the age of three

**General Intake Guidelines (Normal Weight): 5-18 years**

<table>
<thead>
<tr>
<th></th>
<th>5-9 years</th>
<th>10-14 years</th>
<th>15-18 years</th>
</tr>
</thead>
<tbody>
<tr>
<td>Milk and Milk Products</td>
<td>2.5-3 cups/day</td>
<td>3 cups/day</td>
<td>3 cups/day</td>
</tr>
<tr>
<td>Serving:</td>
<td>1 cup of milk or cheese, 1/3 oz of natural cheese, 1/3 cup shredded cheese; encourage low-fat dairy sources</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Meat and Other Protein Foods</td>
<td>4-5 oz/day</td>
<td>5 oz/day</td>
<td>5-6 oz/day</td>
</tr>
<tr>
<td>Serving:</td>
<td>(1oz equivalent) = 1oz beef, poultry, fish, 1/4 cup cooked beans, 1 egg, 1Tbsp peanut butter*, 1/2 oz of nuts</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Breads, Cereal, and Starches</td>
<td>5-6 oz/day</td>
<td>5-6 oz/day</td>
<td>6-7 oz/day</td>
</tr>
<tr>
<td>Fruits</td>
<td>1 1/2 cups/day</td>
<td>1 1/2 cups/day</td>
<td>1 1/2 cups</td>
</tr>
<tr>
<td>Vegetables (non-starchy vegetables to include sources of vitamin C and A: broccoli, bell pepper, tomatoes, spinach, green beans, squash)</td>
<td>1 1/2 - 2 cups/day</td>
<td>2-3 cups/day</td>
<td>3+ cups/day</td>
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<tr>
<td>Serving:</td>
<td>1 cup of fruit or 1/2 cup dried fruit; NO JUICE</td>
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<tr>
<td>Fats and Oil</td>
<td>4-5 tsp/day</td>
<td>5 tsp/day</td>
<td>5-6 tsp/day</td>
</tr>
<tr>
<td>Miscellaneous</td>
<td>desserts, sweets, soft drinks, candy, jams, jelly</td>
<td>none</td>
<td>none</td>
</tr>
</tbody>
</table>

*Low-fat products are not recommended under the age of 2

Reference/s: [30] [31]
# Food Insecurity

## Background
- 21% of children meet USDA definition of FI
- 7.5 million American families lack consistent access to adequate, nutritious food.
- Households with children: 2X>FI
- Poverty, underemployment not only factors
- Greater risk: children in immigrant families, families headed by single women, less education, large families, parental separation or divorce.
- 16% of low-income households do not receive federal support
- Poverty associated with obesity.
- FI disproportionately threatens certain populations at high risk for obesity.

## Health Impact
- Depression, anxiety, toxic stress
- 4-36 months: high risk for developmental problems.
- <;/=36 months: poorer overall health/more hospitalizations
- More likely to be iron deficient
- Pre-Adolescent boys: lower bone density
- Lower cognitive indicators, dysregulated behavior, emotional distress
- Adolescents: higher risk for dysthymia/suicidal ideation.
- Health effects of FI may persist beyond early life into adulthood. Including: DM, hyperlipidemia, and CV disease.
- Environmental realities: presence of full grocery, increased fast food. Intermittent access to adequate food – results in unhealthy eating patterns and increased stress.
- Food Assistance Programs (NSLP/WIC/SNAP) participation shows reduction in FI (Healthy People2020)

## Screening Tool/Recommendations
- **Screening Tool**
  - Hunger Vital Sign:
    - 1. Within the past 12 months, we worried whether our food would run out before we got money to buy more. (Yes or No)
    - 2. Within the past 12 months, the food we bought just didn’t last and we didn’t have money to get more. (Yes or No)
- **Recommendations**
  - 1. Screen for FI with 2 question validated tool
  - 2. Be familiar with community, state, and federal resources
  - 3. Be aware of nutritional content of these resources
  - 4. Be aware of factors that increase vulnerability for FI
  - 5. Advocate

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For a list of resources see Appendix B

SNAP = Supplemental Nutrition Assistance Program;
WIC = Special Supplemental Nutrition Program for Women, Infants, and Children

Reference/s: [36] [46] [47] [48] [49] [50] [51] [52] [53] [54] [55] [56] [57] [58] [59] [60] [61] [62] [63]
### Nutritional Therapy: Comparison of Common Recommendations

<table>
<thead>
<tr>
<th>Portion Control or Balanced</th>
<th>CHO Restricted or Reduced</th>
<th>Low Glycemic Index</th>
<th>Low Fat</th>
<th>Elimination</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Tolerance is high</td>
<td>• Adherence to diet is approximately 50%</td>
<td>• Tolerance is high</td>
<td>• Tolerance is high</td>
<td>• Used mostly in fast food or cafeteria type settings</td>
</tr>
<tr>
<td>• Useful in toddlers and young children</td>
<td>• Weight loss is moderate to good</td>
<td>• Small amount of weight loss</td>
<td>• Limited trials: in 3 RCT small reduction in BMI, total and LDL Cholesterol</td>
<td>• No clear evidence for effectiveness</td>
</tr>
<tr>
<td>• Small amount of weight loss</td>
<td>• Lowers fasting insulin and triglyceride levels</td>
<td>• Decreases postprandial glucose response, slows insulin secretion, and delays gastric emptying leading to longer satiety</td>
<td>• No weight loss or minimal</td>
<td>• Weight loss is small to moderate</td>
</tr>
<tr>
<td></td>
<td>• Amount of protein is not associated with effect on muscle sparing</td>
<td>• Favorable for high fasting insulin level</td>
<td>• No difference between low fat &amp; low glycemic in reduction of steatosis</td>
<td>• Tolerance is high</td>
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<tr>
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<td></td>
<td></td>
<td>• Not same as elimination diets for food allergies/intolerance</td>
</tr>
</tbody>
</table>

References: [55] [65] [66] [67] [68] [69] [70] [71] [72] [73] [74] [75] [76]
**Ketogenic Diet (KD)**
- Used in children for epilepsy for few RCTs in obesity
- Ketosis ratio: \(0.9F + 0.46P)/(1.0C + 0.58P + 0.1F)\) where \(F, P, C\) are grams of fat, protein, carbohydrates (CHO)
- Current popular version is 20-50 grams of CHO/day
- Fat content 70-80%
- Protein 1.2-1.5 g/kg/d to maintain ketosis
- Levels of 20 g/d CHO require eliminating fiber rich starchy vegetables, most fruit, legumes and whole grains

**Children may gain weight if they increase their meat consumption significantly, thereby increasing saturated fat**

**KD may impair glucose tolerance compared to diets containing >20 g of CHO**

**KD may achieve improved metabolic control quickly but may be more difficult for a child to stay on long term**

**Intermittent Fasting or Time Restricted Feeding**
- 5 days on, 2 days off, alternate days fasting and ad lib eating, eating for 6 hours and fasting for 18 hours
- Some studies show consumption is greater on fasting days than planned and less on non-fasting days
- Most studies show poor adherence
- Not enough research in children to recommend
- May lead to disordered eating
- Not a great option for those with binge eating disorder or depression

**Plant Based**
- Seen as a population-wide risk factor strategy to reduce CV disease
- Excludes products of animal origin, e.g. meat, eggs and fish
- Promoted as more sustainable for the planet
- Lower energy density than an omnivore diet if high in vegetables, fruit, legumes and nuts
- No significant difference in weight status in children in a small 4 week study
- More studies needed.

**Type 2 Diabetes**
- Conventional management: 30 grams CHO/meal, 45-60% CHO
- If baseline high insulin secretion, better response to low CHO/nonrestricted calorie diet
- Unsaturated vs saturated fat
- Source of CHO important, if CHO restricted, metabolic control improved
- Frequent monitoring important

**Hypertension**
- DASH diet: 2300 mg Na/day or less, 1500 mg/day for African Americans
- Adherence to diet worse as children age
- Of the 9 nutrient goals (fat, sat fat, protein, cholesterol, fiber, calcium, magnesium, potassium and sodium) most meet only 2
- Lower blood pressure correlates with higher adherence
- Anxiety, depression, anger, lack of sleep or physical activity all associated with poor response to diet

**Metabolic Syndrome**
- Framingham Offspring Cohort
- Whole grain intake was inversely associated with prevalence
- Glycemic Index was positively associated with prevalence
- The source and quality of dietary carbohydrates may differentially optimize insulin action
- Fasting insulin concentrations are lower among individuals reporting higher dietary fiber or whole grain intake

**Familial Hypercholesterolemia (FH)**
- Heterozygous FH and Homozygous FH: start dietary therapy as soon as the diagnosis is made
- <7% saturated fat
- <200 mg/day cholesterol
- NHLBI Child 2 diet
- Most children with obesity have LDL<140 mg/dl, if higher, suspect Heterozygous FH
**Nutritional Therapy: Specific Complications Associated with Obesity**

### NAFLD
- **A CHO rich diet provides the hepatic free fatty acids which cause fatty deposition in the liver**
- **Weight loss should be no more rapid than 1 kg/week**
- **Rapid weight loss has been associated with disease progression in some patients with NAFLD**
- **Fructose**
  - Causes lipogenesis, TG synthesis and steatosis
- **Fiber**
  - Soluble fiber-slow gastric emptying, reduces total cholesterol levels
  - Insoluble fiber-promote satiety, reduces LDL levels
- **Protein**
  - Low protein intake can induce steatosis
  - High protein low carbohydrate diet can improve liver enzymes
- **Fat**
  - Fat triggers lipolysis in adipocytes and increases FFAs
  - High fat diet increases liver weight, ALT, TG, IL-6, TNF-α
  - Saturated Fats
    - Induce endoplasmic reticulum stress and hepatocyte injury
    - Induce hypothalamic inflammation
    - Intake of more than 10% of total energy supports insulin resistance
  - MUFA
    - Decreases LDL, total cholesterol and triglycerides and reduces hepatic fat content
  - PUFA
    - Decreases total cholesterol, LDL and hepatic enzymes
    - Omega-3s decrease steatosis and improve ALT if used over long periods (>6 mos) of time
- **Trans Fat**
  - Unclear role
- **Vitamin E**
  - Controversial: may be effective against steatohepatitis in NAFLD but ineffective once fibrosis is established
- **Vitamin D**
  - Regulates genes distributed in the liver, some of which are involved in glucose and fat metabolism
  - Deficiency exacerbates NAFLD and insulin resistance
- **Polyphenols (Resveratrol, curcumin, green tea, etc.)**
  - Reduces liver enzymes and steatosis in adults, no studies in children

### NAFLD
- **Fat**
  - Fat triggers lipolysis in adipocytes and increases FFAs
  - High fat diet increases liver weight, ALT, TG, IL-6, TNF-α
  - Saturated Fats
    - Induce endoplasmic reticulum stress and hepatocyte injury
    - Induce hypothalamic inflammation
    - Intake of more than 10% of total energy supports insulin resistance
  - MUFA
    - Decreases LDL, total cholesterol and triglycerides and reduces hepatic fat content
  - PUFA
    - Decreases total cholesterol, LDL and hepatic enzymes
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  - Trans Fat
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### NAFLD
- **Vitamin E**
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- **Vitamin D**
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  - Deficiency exacerbates NAFLD and insulin resistance
- **Polyphenols (Resveratrol, curcumin, green tea, etc.)**
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**Attention Deficit Syndromes**
- **No specific diet has been shown to improve symptoms of ADHD**
- **ADHD associated with disordered eating**
- **Youth with ADHD have a worse diet than those without regardless of medication/no medication status**
- **Weight trajectory in ADHD medicated youth:**
  - If ADHD diagnosed before the age of 11, BMI decreases and growth decelerates for the first 3 years
  - After the fifth grade or 10-11 years of age, BMI accelerates in ADHD medicated youth as compared to non medicated ADHD youth and youth without a diagnosis of ADHD

**Attention Deficit Syndromes**
- **As a group has more difficulty with mundane and repetitive tasks such as following a dietary plan**
- **May say they are “bored” with trying to lose weight**
- **Characterized by seeking new and intense stimuli**
- **Complain of low energy**
- **Frequently have many comorbid conditions (depression, emotional irritability)**
- **Medication associated with improvement**
- **Targets their reward deficiency**
- **Improves executive functioning**

**Physical Disabilities (Nonambulatory)**
- **Allow the child to have some control over the diet plan**
- **Particularly adolescents**
- **Use REE to calculate caloric needs for a sedentary individual and adjust for level of activity**
- **Get kids involved with other kids with like disabilities as much as possible**
- **Kinetic Kids or Programs specifically adapted for wheelchairs and other assistive devices**
- **Support groups**

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References: [86] [87] [88]
## Nonnutritive Sweeteners (NNS)

### Background
- Be cautious! NNS are 180-20,000 times sweeter than sucrose
- Most evidence shows that those who use NNS do not lose weight
- Evidence suggests that if NNS are used in combination with food, insulin and GLP-1 levels actually are higher
- Sweetness without calories can result in a disturbance in appetite regulation and a higher preference for sweet taste
- However, some studies in adults report success if used in a weight management program in the context of intentional weight loss
- Most studies suggest unfavorable metabolic and health outcomes

### Health Impact
- NNS have been approved by the FDA as “generally regarded as safe,” meaning that data is insufficient to ensure the long term safety
- No associations between NNS and cancer, ADHD or ADD, birth defects, diabetes or lupus
- Over 50% of parents in one study stated that they seek products that have reduced sugar but did not know they contain NNS
- Studies point to a preference for sweet vs salty or savory foods after NNS

### Common Nonnutritive Sweeteners
- Sucralose
- Aspartame
- Acesulfame potassium
- Neotame
- Advantame
- Saccharin
- Stevia

### Summary of Dietary Interventions
- Current evidence suggests that improved weight status can be achieved in children and adolescents with overweight or obesity irrespective of the macronutrient distribution of a reduced-energy diet.

- Tailoring the macronutrient content to target specific cardio-metabolic risk factors, such as a low-carbohydrate diet to treat insulin resistance, may be possible.
### General Dietary Guidelines: 0 to 24 months

<table>
<thead>
<tr>
<th>Weight/length percentile appropriate</th>
<th>Weight/length percentile crossing lines</th>
<th>Weight/length percentile exceeding 85th for age</th>
<th>Weight/length percentile exceeding 95th for age</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Exclusive breastfeeding for as long as possible, complementary foods at 6 months</td>
<td>• Exclusive breastfeeding for as long as possible</td>
<td>• Exclusive breastfeeding for as long as possible</td>
<td>• Exclusive breastfeeding for as long as possible</td>
</tr>
<tr>
<td>• If formula feeding, review dietary history, if no complementary foods, intake 27-47 oz/day, if complementary foods (after 6 months of age) intake 24-32oz/day</td>
<td>• If formula feeding, reduce intake to lower limits, 24 oz/day, feedings should be every 4-5 hours for ≤ 6 months, 3 times per day for &gt; 6 months. Review appropriate intake of complementary foods after 6 months of age, consider limiting cereal</td>
<td>• If formula feeding, reduce intake to lower limits, 24 oz/day, 3 times per day, minimal intake of complementary food after 6 months of age, consider excluding cereal</td>
<td>• If formula feeding, reduce intake to lower limits, 24 oz/day, 3 times per day, minimal intake of complementary food after 6 months of age, consider excluding cereal</td>
</tr>
<tr>
<td>• No media</td>
<td>• No media</td>
<td>• No media</td>
<td>• No media</td>
</tr>
</tbody>
</table>

### Management of the Infant with Obesity: 0 to 24 months

- No screen time
- No TV in bedroom
- Allow infant to feed themselves
- Do not force/finish foods when infant indicating refusal
- 12-18 Hours of sleep

**Intake**

- Exclusive breastfeeding for 6-12 months
- Appropriate formula feeding ingestion for age
- Delay complementary foods until 6 months
- No juice/sugar sweetened beverages
- No fast food
- No desserts

**Activity**

- Keep active in playpen/floor
- Encourage direct interaction with parents as much as possible
- No media

**Behavior and Sleep**
Management of the Toddler with Obesity: 2 to 4 years

- Routine sleep pattern
- No TV in bedroom
- 11-14 hours of sleep
- All meals at the table/highchair
- Parents as role models
- Food not used as reward
- Parents should not be over controlling
- Family Based Therapy

Intake
- Three meals plus snack(s)
- 3 servings of protein (1-3oz)/day
- 2-2.5 cups dairy/day
- 3 servings non-starchy vegetables (3/4 cup to 1 ½ cups)/day
- Fruit 1 cup /day
- Dessert only on special occasion
- No sugar sweetened beverages
- No fast food
- Age appropriate portion sizes
- Praise for trying new foods

Activity
- Active play almost constantly
- Minimal sedentary time
- No screen time < 2 years, < 1 hour/day 2-4 years

Behavior and Sleep
- Routine sleep pattern
- No TV in bedroom
- 11-14 hours of sleep
- All meals at the table/highchair
- Parents as role models
- Food not used as reward
- Parents should not be over controlling
- Family Based Therapy

General Dietary Guidelines: 2-4 years

<table>
<thead>
<tr>
<th>BMI &lt; 85th percentile</th>
<th>BMI 85th-95th percentile</th>
<th>BMI 95th-120th percentile</th>
<th>BMI &gt; 120th percentile</th>
</tr>
</thead>
<tbody>
<tr>
<td>1-1.5 cups each of fruits and vegetables/day</td>
<td>1-1.5 cups each of fruits and vegetables/day</td>
<td>Restricted CHO/LGI*</td>
<td>Restricted CHO*LGI**</td>
</tr>
<tr>
<td>&lt;2 hours screen time/day if 2-4 years</td>
<td>1-2 hours of screen time/day if 2-4 years</td>
<td>&lt;1 hour of screen time/day</td>
<td>Screen time 50% of active time up to 1 hour/day</td>
</tr>
<tr>
<td>Free play for as many hours as possible/day</td>
<td>Free play for as many hours as possible/day</td>
<td>Reduce sedentary activity</td>
<td>Reduce sedentary activity</td>
</tr>
<tr>
<td>No sugar sweetened beverages</td>
<td>No sugar sweetened beverages</td>
<td>Free play for as many hours as possible/day</td>
<td>Free play for as many hours as possible/day</td>
</tr>
<tr>
<td></td>
<td></td>
<td>No sugar sweetened beverages</td>
<td>No sugar sweetened beverages</td>
</tr>
</tbody>
</table>

**Low glycemic index
*Carbohydrate
Management of the Young Child with Obesity: 5 to 9 years

**Pharmacology**
- Minimize obesogenic medications especially SGAs*
- Treat asthma with controller meds to minimize systemic steroid use
- Consider ACE inhibitor for persistent hypertension

**Intake**
- Three meals; 1-2 snacks
- 3 servings of protein/day
- 2-3 servings of dairy/day
- 4-5 servings non-starchy vegetables
- Dessert only on special occasion
- No sugar sweetened beverages
- No fast food
- Age appropriate portion sizes
- Praise for trying new foods
- Consider low glycemic index/reduced carbohydrate diet

**Activity**
- Moderate - vigorous activity for 60 minutes or greater each day; Can be organized or not

**Behavior and Sleep**
- Screen time <1-2 hours
- Routine sleep pattern
- No TV in bedroom
- 11-14 hours of sleep
- All meals at the table
- Parents as role models
- Parenting style should not be overly controlling
- Sleep study if severe obesity and/or symptoms
- Tonsillectomy and adenoidectomy if indicated

*Second generation antipsychotics

---

General Dietary Guidelines: Childhood through Adolescence

**BMI < 85th percentile**
- 1.5-2 cups of fruits and 3+ cups vegetables /day
- < 2 hours screen time/day
- At least 60 min of age appropriate activity/day
- No sugar sweetened beverages

**BMI 85th-95th percentile**
- 1.5-2 cups each of fruits and 3+ cups vegetables/day
- < 1-2 hours of screen time/day
- At least 60 min of age appropriate activity/day
- No sugar sweetened beverages

**BMI 95-120th percentile**
- Restricted CHO/LGI
- < 1 hour of screen time/day
- Reduce sedentary activity
- At least 60 min of age appropriate activity /day
- No sugar sweetened beverages

**BMI >120th percentile**
- Restricted CHO/LGI
- Screen time 50% of active time up to 1 hour/day
- Reduce sedentary activity
- At least 60 min of age appropriate activity /day
- No sugar sweetened beverages

*Carbohydrate/Low glycemic index
Management of the Pubertal Child with Obesity: 10-14 years

- Orlistat (Xenical) FDA approved for > age 12
- Minimize obesogenic medications especially SGAs
- Treat asthma with controller meds to minimize systemic steroid use
- Consider ACE inhibitor for persistent hypertension
- Metformin FDA approved for T2DM > age 10

- 3 meals; 1-2 nutritious snacks
- 3 servings of protein/day
- 3 servings of dairy/day
- 4-5 servings of non-starchy vegetables
- Dessert only on special occasion
- No sugar sweetened beverages
- No fast food
- Age-appropriate portion sizes
- Allow child to leave food on plate

- Screen time less than 1-2 hours/day
- 10-12 hours of sleep
- Routine sleep pattern
- No TV in bedroom
- Parenting style should not be overly controlling
- Peer groups become increasing important
- All meals at table with family and encourage socialization
- Recommend meal and exercising tracking

Pharmacology

Intake

Activity

Behavior and Sleep

Management of the Adolescent with Obesity: 15-18 years

- Orlistat (Xenical) ≥ age 12, Phentermine approved for ≥ age 16
- Minimize obesogenic medications especially SGAs*
- Treat asthma with controller meds to minimize systemic steroid use
- Consider ACE inhibitor for persistent hypertension
- Metformin FDA approved for T2DM ≥ age 10

- 3 meals; nutritious snacks
- 3 servings of protein/day
- 3 servings of dairy/day
- 4-5 servings of non-starchy vegetables
- Dessert only on special occasion
- No sugar sweetened beverages
- No fast food
- Age-appropriate portion sizes
- Allow adolescent to leave food on plate

- Screen time less than one hour/day
- 10-12 hours of sleep
- Routine sleep pattern
- No TV in bedroom
- Parenting style should not be overly controlling
- Friends and relationships are important
- Recommend meal/exercising tracking or monitoring

Vigorous activity for 60-90 minutes or more daily. Can be organized or not
- Monitor for changes in decreased activity level
- Decrease non academic sedentary time as much as possible

Pharmacology

Intake

Activity

Behavior and Sleep

*Second generation antipsychotics

References: [29] [30] [31] [32]
## Activity Recommendations General Guidelines

### Infants (0-12 months)
- Interaction with caregivers dedicated to exploring movement and the environment
- Setting that encourages and stimulates movement experiences and active play for short periods of time several times a day
- Promote skill development in movement
- Environment should meet or exceed recommended safety standards for performing large-muscle activities
- Structured and unstructured physical activity

### Toddlers (12-36 months)
- **30 minutes** of structured physical activity each day.
- At least 60 minutes per day of unstructured physical activity
- Opportunity to develop movement skills that will serve as the building blocks for future motor skillfulness and physical activity
- Access to indoor and outdoor areas for performing large-muscle activities
- Caretakers should promote movement skills by providing activity and physical movement experiences

### Young Children (3-5 yrs)
- **60 minutes** of structured physical activity each day.
- At least 60 minutes of unstructured physical activity
- Opportunity to develop fundamental motor skills that will serve as the building blocks for future motor skillfulness and physical activity
- Access to indoor and outdoor areas for performing large-muscle activities
- Caregivers should promote movement skills by providing opportunities for structured and unstructured physical activity

### Older Children (5-12 yrs)
- **60 minutes, and up to several hours**, of age-appropriate physical activity on all, or most days.
- Should include moderate and vigorous physical activity with the majority of the time spent in activity that is intermittent
- Several bouts of physical activity lasting 15 minutes or more each day.
- Should participate each day in a variety of age-appropriate physical activities designed to achieve optimal health, wellness, fitness, and performance benefits.
- Extended periods (two hours or more) of inactivity are discouraged, especially during the daytime hours.
### Activity Recommendations for Various Ages (Normal Weight)

<table>
<thead>
<tr>
<th>Activity Type</th>
<th>9-13 Years</th>
<th>14-18 Years</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Aerobic/Endurance</strong></td>
<td>Vigorous intensity: Running Dancing Swimming Bicycle riding</td>
<td>Running Bicycle riding Sports: soccer or swimming</td>
</tr>
<tr>
<td><strong>Bone-Building</strong></td>
<td>Basketball Tennis Running</td>
<td>Running Jumping</td>
</tr>
<tr>
<td><strong>Muscle Strengthening</strong></td>
<td>Push-ups Use of resistance bands</td>
<td>Use of free-weights of 15-20 pounds with high repetitions</td>
</tr>
<tr>
<td><strong>Active Play</strong></td>
<td>Football Basketball Ice Hockey Volleyball Individual sports: Tennis Track &amp; Field Running Swimming Dance</td>
<td>Yoga Dance Running Walking Cycling Household chores Competitive or noncompetitive sport</td>
</tr>
</tbody>
</table>

**Co-morbidities**

---

Hypertension (HTN)

**Prevalence/Diagnosis**

- **Prevalence**
  - 3.8-24.8% of adolescents with obesity have HTN
  - 2 fold increase BMI 95-98th
  - 4 fold increase BMI >99th

- **Diagnosis**
  - Three separate measurements at least one week apart
  - < 13 years
    - Elevated BP = > 90th and < 95th percentile or
    - 120/80 mm Hg to < 95th percentile (whichever is lower)
  - Stage 1 HTN: > 95th percentile to < 95th percentile + 12 mm Hg or
    - 130/80 to 139/89 mm Hg (whichever is lower)
  - Stage 2 HTN: >95th percentile + 12 mm Hg, or ≥ 140/90 mm Hg (whichver is lower)

- < 13 years
  - Obesity associated with increased risk for
    - Masked HTN (Clinic BP <95th percentile but
    - ambulatory BP >95th percentile)
    - Circadian variability: up to 50% do not experience the expected nocturnal BP dip

- > 13 years
  - Elevated BP = syst 120-129 mm Hg, dia < 80 mm Hg
  - Stage 1 Hypertension = syst 130-139 mm Hg, dia 80-89 mm Hg
  - Stage 2 Hypertension = ≥ 140/90 mm Hg

- Obesity associated with increased risk for
  - Masked HTN (Clinic BP <95th percentile but
  - ambulatory BP >95th percentile)
  - Circadian variability: up to 50% do not experience the expected nocturnal BP dip

**Evaluation**

- Ambulatory blood pressure monitoring recommended due to prevalence of white coat HTN and masked HTN
- Screen for glucose intolerance and dyslipidemia
- Sleep history
- Further studies not necessary unless history of chronic kidney disease or other renal disorders
- If unresponsive to lifestyle intervention, consider renal US, electrolytes, CBC, creatinine, renin and aldosterone

**Treatment**

- Primary treatment is weight loss: Diet and lifestyle interventions before medication
- Low Na (<1500 mg/day) or DASH diet
- For Stage 1 HTN lifestyle intervention for 6-12 months
- Physical activity beneficial: moderate to vigorous, 30-60 min 5 days or more a week
- Restrict only Stage 2 from high static competition until BP is controlled
- Pharmacotherapy if Stage 1 not controlled after 12 months or symptomatic or Stage 2 in combination with lifestyle intervention
- Pharmacotherapy
  - Begin with single agent at the low end of the dosing range
  - ACE inhibitor, Angiotensin II Receptor Blocker, Ca Channel Blocker or thiazide diuretic
  - Increase dose every 2-4 weeks until BP is normalized
  - Thiazide diuretic is usually the second agent

---

**2017 Clinical Practice Guideline (CPG) for Screening and Management of HTN and Children with Obesity**

**Issues**

- New normative data was based on normal weight children, thus the cut off values for the categories of "elevated or >90th percentile, Stage 1 or >95th percentile, and Stage 2 or >99th percentile + 12 mm Hg are lower than other CPGs
- Problem: The proportion of children with obesity and HTN is now higher based on the lowering of the normative values

**Associations between HTN and Children with Obesity**

- Associations between children with obesity and HTN include
  - Graded increase in rate of HTN with increasing waist circumference
  - Lack of circadian variability of blood pressure
  - Up to 50% do not experience the expected nocturnal dip
  - Development of HTN as an adult
  - HTN in children with obesity is frequently accompanied by dyslipidemia and disordered glucose metabolism, all of which contribute to greater increases in CV risk
  - Severe OSA is commonly associated with HTN in children with obesity
  - In children with T2DM and obesity, progression from normo-tension to prehypertension to hypertension is strongly associated with central obesity

**General Guidelines**

- Diet and lifestyle interventions are the cornerstone, but 7% of children with elevated BP progress to HTN
- Children with obesity and HTN can participate in competitive sports once target levels for BP are reached (normative values)
- Children with obesity and Stage 2 HTN should be restricted from high static sports (wrestling, weight lifting, boxing)
- Acute, severe HTN requires immediate evaluation
- Ambulatory blood pressure monitoring can be an objective method to evaluate treatment
## Intracranial Hypertension (IIH)

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>Evaluation</th>
<th>Treatment</th>
<th>Clinical Finding</th>
</tr>
</thead>
</table>
| - IIH is also known as pseudotumor cerebri (PTC), benign intracranial hypertension (BIH), and pseudotumor cerebri syndrome.  
- IIH can lead to permanent visual impairment or blindness.  
- A strong association between childhood obesity and increased risk of pediatric IIH.  
- The incidence of IIH in youth with obesity is lower in prepubertal children than in adolescents. | - Idiopathic intracranial hypertension (IIH) is defined as elevated intracranial pressure without clinical, radiological or laboratory evidence of a cause.  
- Full neurological examination usually with imaging and spinal tap  
- Full eye examination for papilledema or loss of visual field | - Preserving vision and controlling symptoms are the goals of therapy  
- Variable response to all therapies.  
- Ventricular/lumbar peritoneal shunts may lower intracranial pressure, preserve vision, and relieve other symptoms.  
- Some case reports of success with weight loss after RYGB.  
- Loss of as little as 6% of body weight has been noted to reduce intracranial pressure. | - One study in 606 adults with obesity:  
- incidence of 2.8% with abnormalities on non-mydriatic fundus photographs  
- 0.6% incidence of asymptomatic optic disc edema  
- Headaches, visual loss, including blind spots, poor peripheral (side) vision, double vision, and short temporary episodes of blindness. |

Reference/s: [102] [103] [104] [105] [106] [107] [108] [109]  

## Sleep Hygiene Instructions

<table>
<thead>
<tr>
<th>Definition</th>
<th>Things to Follow</th>
<th>Things to Avoid</th>
</tr>
</thead>
</table>
| - Sleep hygiene is a variety of different practices and habits that are necessary to have good nighttime sleep quality and full daytime alertness. | - Keep bedtimes and wake times consistent every day of the week. Late weekend nights or sleeping-in can throw off a sleep schedule for days.  
- Bedtime should follow a predictable sequence of events, such as brushing teeth and reading a story.  
- Having physical exercise early in the day can help with sleep time.  
- Worry time should not be at bedtime. Children with this problem can try having a "worry time" scheduled earlier to think about and discuss their worries with a parent.  
- Security objects at bedtime are often helpful for children who need a transition to feel safe.  
- When checking on a child at night, checks should be "brief and boring." | - High stimulation activities just before bed, such as watching TV, or playing video games  
- Spending lots of non-sleep time in bed. This also includes if a child is awake in bed tossing and turning: get out of bed to do a low stimulation activity (e.g., reading), then return to bed later.  
- Intake of caffeine (sodas, chocolate, tea, coffee) in the afternoons/evenings.  
- For the best night’s sleep, most people should avoid strenuous workouts close to bedtime. |
### Guidelines for Sleep Duration

<table>
<thead>
<tr>
<th>Age Group</th>
<th>National Sleep Foundation (hours)</th>
<th>Not Recommended (hours)*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Newborn</td>
<td>14-17</td>
<td>&lt; 11 or &gt; 19</td>
</tr>
<tr>
<td>Infants</td>
<td>12-15</td>
<td>&lt; 10 or &gt; 18</td>
</tr>
<tr>
<td>Toddler</td>
<td>11-14</td>
<td>&lt; 9 or &gt; 16</td>
</tr>
<tr>
<td>Preschool</td>
<td>10-13</td>
<td>&lt; 8 or &gt; 14</td>
</tr>
<tr>
<td>School-Aged</td>
<td>9-11</td>
<td>&lt; 7 or &gt; 12</td>
</tr>
<tr>
<td>Teenager</td>
<td>8-10</td>
<td>&lt; 7 or &gt; 11</td>
</tr>
<tr>
<td>Young Adult</td>
<td>7-9</td>
<td>&lt; 7 or &gt; 9</td>
</tr>
<tr>
<td>Adult</td>
<td>7-9</td>
<td>&lt; 7 or &gt; 9</td>
</tr>
<tr>
<td>Older Adult (+ 65)</td>
<td>7-8</td>
<td>&lt; 7 or &gt; 8</td>
</tr>
</tbody>
</table>


### Sleep Disorder Syndromes

<table>
<thead>
<tr>
<th>Condition</th>
<th>Diagnosis</th>
<th>Evaluation</th>
<th>Treatment</th>
<th>Clinical Findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>Primary Snoring</td>
<td>• Prevalence: 3-16%</td>
<td>• Sleep study if severe to rule out apnea or hypopnea, hypercapnia, hypoxemia, high arousal index, disruption of normal sleep</td>
<td>• Sleep hygiene counseling</td>
<td>• Decrease in mean verbal IQ scores, global IQ scores, selective attention scores, sustained attention scores, memory index</td>
</tr>
<tr>
<td></td>
<td>• Snoring without apnea or hypopnea, hypercapnia, hypoxemia, high arousal index, disruption of normal sleep</td>
<td></td>
<td></td>
<td>• Direct correlation between number of mild desaturations (3%), arousals &amp; severity of neurocognitive deficits</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>• Characterized as benign; linked to neuropsychological impairments.</td>
</tr>
<tr>
<td>Restless leg syndrome (RLS)</td>
<td>• Insomnia with “creepy or crawling” feeling in the arms and legs at night</td>
<td>• Diagnosis of exclusion</td>
<td>• Sleep hygiene counseling</td>
<td>• Autosomal dominant, sensorimotor disorder</td>
</tr>
<tr>
<td></td>
<td>• Insomnia accompanied by an urge to move the limbs</td>
<td>• Low iron levels are suggestive</td>
<td>• Dopamine agonists</td>
<td>• 2/3 of children may have low levels of serum ferritin; iron is a cofactor in synthesis of dopamine.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• EMG and possible muscle biopsy to rule out other neuro-muscular disease</td>
<td>• Oral iron</td>
<td>• Restless legs syndrome and attention deficit disorder are associated.</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>• Clonazepam</td>
<td>• Interferes with going to sleep and staying asleep</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>• Gabapentin</td>
<td>• May be accompanied by daytime fatigue, inattentiveness, or frank sleepiness.</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>• Dopamine deficiency implicated in the pathogenesis</td>
</tr>
</tbody>
</table>

Reference/s: [112] [113] [114] [115] [116] [117] [118] [119] [120] [121]
# Sleep Disorder Syndromes

## Delayed Sleep Phase Syndrome
- Habitually unable to fall asleep before 2-3 am
- Neurological sleep disorder in which a person's sleep/wake cycle is delayed with respect to the external day/night cycle
- Normal sleep if sleeps freely but very sleepy if conforming to conventional sleep-wake schedules.

### History of circadian sleep pattern
- Perform sleep logs and wrist actigraphy
- Evaluate for secondary effects of chronically poor sleep

### Treatment
- "Bright light" therapy: provide 2700-10 000 lux of bright light via a light box for 20-30 minutes immediately after waking leading to a gradual phase advancement (shifting back) of the sleep onset time at night.
- 0.5-1 mg melatonin about 5.5 hours before sleep onset
- Stimulant drugs to counter residual daytime sleepiness

## Obesity Hypoventilation Syndrome
- Awake chronic hypercapnia (PaCO2 >45 mm Hg) + Obesity
- Excessive work of breathing and increased CO2 production
- Abnormal central ventilatory drive and obesity
- Other disorders should be ruled out. Also known as Pickwickian Syndrome

### Evaluation for concurrent OSA (90% chance)
- Sleep study: O2 Sats, CO2 levels, and apnea episodes
- Cardiac and pulmonary work up if symptomatic

### Treatment
- Weight loss
- Respiratory support as indicated, including CPAP
- May have central respiratory control system abnormality with decreased responsiveness to CO2 rebreathing, hypoxia, or both.
- Inspiratory muscle strength and resting tidal volumes decreased
- Leptin deficiency/resistance may contribute to OHS by reducing ventilatory responsiveness leading to CO2 retention.
- Pulmonary HTN more common & severe than in OSA.

## Obstructive Sleep Apnea
- Upper airway becomes blocked repeatedly during sleep, reducing or completely stopping airflow
- OSA may be a cause of obesity and not a consequence alone

### Diagnosis
- Snoring or disrupted sleeping
- Daytime sleepiness
- Hyperactivity
- Audible pauses in breathing
- Nocturia

### Treatment
- Tonsillectomy and adenoiectomy (T&A) if indicated
- Repeat sleep study > 6-8 weeks post op
- Weight loss (other recommended treatment should be used pending weight loss)
- Routine sleep pattern CPAP

### History
- Snoring or disrupted sleeping
- Daytime sleepiness
- Hyperactivity
- Audible pauses in breathing
- Nocturia

---

**Reference/s:** [112] [113] [114] [115] [117] [118] [119] [120] [122] [123] [124] [126] [127] [128] [129] [130] [131] [132] [133] [134]
**Prediabetes**

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>Evaluation</th>
<th>Treatment</th>
<th>Clinical Findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>• HbA1c &gt; 5.7% but &lt; 6.5% x 2 measurements</td>
<td>• Fasting blood glucose</td>
<td>• Restricted carbohydrate diet</td>
<td>• Reversible intermediate phase of altered glucose metabolism in progression from normal glucose tolerance to T2DM</td>
</tr>
<tr>
<td>• FBG &gt; 100 mg/dl but &lt; 126 mg/dl on repeat measurements (impaired fasting glucose)</td>
<td>• Consider fasting insulin, may be falsely low if disease severe or mildly elevated during pubertal growth spurt</td>
<td>• Adolescent studies have shown increase in insulin sensitivity with intensive diet and exercise induced weight loss</td>
<td></td>
</tr>
<tr>
<td>• 2 hour oral glucose tolerance test (OGTT) for blood glucose &gt;140 mg/dl but &lt; 200 mg/dl (impaired glucose tolerance)</td>
<td>• 2 hour OGTT</td>
<td>• Consider Metformin for HbA1c &gt; 5.8% when compliant with diet</td>
<td>• High risk ethnic groups include African Americans, Mexican Americans, Native Hawaiians, American Indians, Pacific Islanders and Asian Americans</td>
</tr>
</tbody>
</table>

**Type 2 Diabetes Mellitus**

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>Evaluation</th>
<th>Treatment</th>
<th>Management</th>
</tr>
</thead>
<tbody>
<tr>
<td>• HbA1c &gt; 6.5% OR • FBG &gt; 126 mg/dl (fasting is defined as no caloric intake for at least 8 hours) OR • 2 hour oral glucose tolerance test (OGTT) for blood glucose &gt;200 mg/dl (The test should be performed as described by the WHO, using a glucose load containing the equivalent of 1.75 mg/kg (max 75 g) anhydrous glucose dissolved in water OR • Classic symptoms of hyperglycemia and a random glucose of &gt;200 mg/dl</td>
<td>• Measure HbA1c every 3 months</td>
<td>• Comprehensive lifestyle management aiming to achieve a 7-10% decrease in excess weight</td>
<td>• Treat BP &gt; than the 95th percentile with ACE inhibitor if no response to lifestyle management after 6 months</td>
</tr>
<tr>
<td></td>
<td>• Individualize home self-monitoring</td>
<td>• If HbA1c &lt; 8.5%, metformin is the initial pharmacologic choice</td>
<td></td>
</tr>
<tr>
<td></td>
<td>• Comprehensive diabetes self-management education and support</td>
<td>• If FBG &gt; 250 mg/dl or HbA1c &gt; 8.5%, treat with basal insulin while metformin is being initiated and titrated</td>
<td></td>
</tr>
<tr>
<td></td>
<td>• Assess social context</td>
<td>• In 2019, the use of GLP-1 agonist liraglutide was approved to treat T2DM in children &gt;10 years.</td>
<td></td>
</tr>
<tr>
<td></td>
<td>• Address mental/behavioral health/disordered eating</td>
<td>• If ketotic or ketoacidosis, use subcutaneous or IV insulin to correct the hyperglycemia</td>
<td></td>
</tr>
<tr>
<td></td>
<td>• Consider weight when recommending medication</td>
<td>• If presenting with severe hyperglycemia (FBG &gt; 600 mg/dl) assess for HHNK (hyperosmolar hyperglycemic nonketotic coma)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>• Provide preconception counseling for females starting at puberty</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>• Measure pancreatic autoantibodies: GAD65 and IA2 antibodies (insulin autoantibody if no exposure to exogenous insulin).</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>• Measure urine albumin/creatinine ratio (UACR)</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>• Measure ALT/AST at baseline</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
### Type 2 Diabetes Mellitus in Youth: Specific Issues

<table>
<thead>
<tr>
<th>Background</th>
<th>Evaluation</th>
<th>Comorbidities</th>
<th>Comorbidities</th>
</tr>
</thead>
</table>
| • Dramatic increase in youth onset T2DM in the late 1990s | • Nonmodifiable risk factors include strong family history of T2DM in first or second degree relatives, offspring of pregnancy complicated by GDM, minority race/ethnicity, and the physiologic insulin resistance of puberty. | • Hypertension: present in 1/3-2/3rds within 1-4 years of onset | • Depression: higher than in T1DM, associated with poor adherence to diet and medication  
Refer for mental health care |
| • Prevalence has been shown to increase with age, tripling from 10–14 years to 15–18 years of age | • As compared to adults, insulin sensitivity in adolescents is 50% lower, even when adjusted for BMI  
• Adolescents hyper secrete insulin at levels 2-3 times that in adults  
• Treatments which are effective in adults: insulin and/or metformin, are not successful in maintaining beta cell function at 15 months post baseline | • Treat upon diagnosis with ACE inhibitor | • NAFLD: baseline AST/ALT and referral to gastroenterologist if indicated  
• OSA: polysomnogram if suspected, ECHO if present, treatment if indicated  
• Polycystic ovary syndrome (PCOS): evaluate in female adolescents, including laboratory studies when indicated  
• Dyslipidemia: Lipid testing to be performed once initial glycemic control achieved and annually |
| • Progression to a HbA1c >8% occurs in 46.6% if treated with metformin plus lifestyle intervention | • HbA1c>6.3% after 3 months of metformin predicts loss of glycemic control | • Dyslipidemia: present in 60-75%  
• Target LDL is <100 mg/dl | |
| • Pancreatic beta cell function declines at 20-35% per year | • 2 overall forms: one that is easily controlled and one that is rapidly progressive | • Retinopathy: present in 9% by first visit  
• Initial and every 2-3 years dilated comprehensive eye exam | |
| • HbA1c>6.3% after 3 months of metformin predicts loss of glycemic control | • Treat hypertension with ACE inhibitor  
• Microalbuminuria: begin screening at diagnosis, repeat annually  
• ACE inhibitor if persistent | • Microalbuminuria: begin screening at diagnosis, repeat annually | |
| • 2 overall forms: one that is easily controlled and one that is rapidly progressive | | | |

### Dyslipidemia

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>Evaluation</th>
<th>Treatment</th>
<th>Pharmacology</th>
</tr>
</thead>
</table>
| • Although any dyslipidemia can co-exist with obesity, obesity is directly associated with: TG >100 mg/dl if <10 yrs, > 130 mg/dl if >10 yrs or HDL <50 mg/dl if female, <40 mg/dl if male | • Fasting lipid profile every 3-6 months if abnormal  
• May monitor with non fasting lipid profile if more feasible  
• Family history of premature cardiovascular disease (May help differentiate dyslipidemia of obesity from heterozygous familial hypercholesterolemia)  
• Consider genetic testing for persistent elevations of LDL >190 mg/dl, TG > 500 mg/dl | • Weight loss: Aggressive diet and lifestyle intervention  
• Restricted CHO/LGI/Elimination diet  
• Decreased saturated fat diet for LDL >130 mg/dl  
• HDL may increase with exercise, especially vigorous exercise | • The dyslipidemia of obesity is not usually treated with medication but the following applies to other dyslipidemias which may co-exist with the dyslipidemia of obesity  
• Statin for Familial Hypercholesterolemia (FH) or LDL > 190mg/dl and failure to respond to weight loss/phytosterols/ increased fiber  
• Phytosterols 2 grams per day to achieve an LDL < 130 mg/dl  
• Increased fiber to 12 grams per day to achieve an LDL < 130 mg/dl  
• Omega-3 to 1-4 grams per day to achieve TG < 100 mg/dl |
| • If pattern of dyslipidemia is different than high TG/low HDL, pursue work up and treatment per NHLBI guidelines and consider referral to Lipidologist | | | |

References: [146] [147] [148] [149] [150] [151]
**Early Cardiovascular Disease**

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>Evaluation</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Elevated BMI: thickening of the carotid intima is associated with increased BMI</td>
<td>• CV damage already apparent in childhood (autopsies)</td>
<td>• Intensive lifestyle modifications</td>
</tr>
<tr>
<td>• If Metabolic Syndrome (MetS) is used as a predictor, individual components are more reliable than presence or absence of MetS</td>
<td>• Elevated systolic and/or diastolic blood pressure; &gt; 140/90 mm Hg associated with early CV disease</td>
<td>• Exercise programs (aerobic and resistance training)</td>
</tr>
<tr>
<td>• Male sex is a risk predictor for CV disease at all weight categories</td>
<td>• Increased severity of obesity is correlated with increased CV risk as measured by components of the metabolic syndrome up until BMI of 140% of the 95th percentile when risk plateaus</td>
<td>• Regular exercise over 6 mos restored endothelial function and improved cIMT in children with obesity</td>
</tr>
<tr>
<td>• MetS identifies children between 8-14 years at increased risk for T2DM and early subclinical atherosclerosis: risk is 15 fold</td>
<td>• Carotid intima-media thickness (cIMT) 0.07 mm greater in age/height matched children with obesity vs normal weight</td>
<td>• Improved CRF associated with reduction in CVD mortality</td>
</tr>
<tr>
<td>• Traditional biomarkers in adults such as LDLc may not be as reliable in children</td>
<td>• Maximal or submaximal exercise testing is a strong predictor of CVD</td>
<td>• Although treatment with statins and hypertensive medications in adults is associated with decreased incidence of MI and stroke and predicted by declines in acceleration of cIMT, no intervention studies with medication have been done in children</td>
</tr>
<tr>
<td>• Increased LA, LV, LV mass, epicardial fat associated with early CVD</td>
<td>• Measurement of left ventricular mass using echocardiography is an independent risk factor of CV morbidity and mortality</td>
<td>• Bariatric surgery in adolescents reverses the metabolic complications and cardiac structural and functional changes but there are, as of yet, no reports of the long term effects on CV health</td>
</tr>
<tr>
<td>• Prepubertal children with obesity have a vascular adaptation state with higher heart rates, greater resting and reactive arterial blood flow and larger brachial artery diameter than normal weight children but by late adolescence develop increased arterial stiffness suggesting that adaptation fails due to the more severe or longer duration of obesity and/or onset of puberty</td>
<td>• Increase in left ventricular mass associated with increased BMI in childhood</td>
<td></td>
</tr>
</tbody>
</table>

**Metabolic Syndrome**

<table>
<thead>
<tr>
<th>Diagnostic Criteria: Modified NCEP-ATP III*</th>
<th>Diagnostic Criteria: International Diabetes Federation</th>
<th>Evaluation</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Age 12-19 years</td>
<td>• Central obesity + 2 risk factors</td>
<td>• Fasting plasma glucose &gt;100mg/dL</td>
<td>• Children with lifestyle intervention (physical activity, nutrition education, and behavior therapy) found to have significant decrease of MetS prevalence and improvement of blood pressure, waist circumference, and 2 hour glucose values on OGTT compared to children with obesity without intervention.</td>
</tr>
<tr>
<td>• 2003 Cook criteria with modification of fasting blood glucose (FBS) criterion to 2003 ADA criterion of ≥100 mg/dl as abnormal.</td>
<td>• Age 10-16 years</td>
<td>• Fasting lipid panel: triglycerides and HDL</td>
<td>• Degree of weight loss (BMI SDS reduction &gt;0.5) associated with improvement of prevalence of all MetS components.</td>
</tr>
<tr>
<td>• The presence of any 3 of the following:</td>
<td>• WC ≥ 90th percentile for age and sex</td>
<td>• Waist circumference (WC)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>• FBS ≥100mg/dl</td>
<td>• Systolic (SBP) and diastolic blood pressure (DBP)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>• WC ≥ 90th percentile for ethnicity, age, and sex</td>
<td>• Birth History: Metabolic syndrome (MetS) has been associated with birth weight, maternal obesity, and gestational diabetes mellitus</td>
<td></td>
</tr>
<tr>
<td></td>
<td>• BP (mmHg) ≥ 90th percentile for age, height, and sex</td>
<td>- Children LGA at birth and exposed to intrauterine environment of diabetes or maternal obesity at increased risk.</td>
<td></td>
</tr>
<tr>
<td>*NCEP-ATP III: National Cholesterol Education Program Adult Treatment Panel III</td>
<td></td>
<td>**Asians and Central and South American: ≥ 90 cm in males</td>
<td></td>
</tr>
</tbody>
</table>

References: [154] [155] [156] [157] [158] [159] [160] [161] [162] [163] [164] [165] [166] [167] [168]

### PCOS/Menstrual Irregularity

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>Menstrual Irregularity</th>
<th>PCOS</th>
<th>Evaluation</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Excessive weight gain can be associated with menstrual irregularity</td>
<td>• Oligomenorrhea/amenorrhea and clinical or biochemical hyperandrogenism with frequent presence of: obesity, glucose intolerance, dyslipidemia, and OSA</td>
<td>• Prolactin, estradiol, consider LH/FSH</td>
<td>• Symptomatic and individualized</td>
<td></td>
</tr>
<tr>
<td>• Irregular menses: Less than 21 days or &gt; 45 days interval, treat for &gt; 3 month intervals or less than 9 cycles in 12 months at gynecological age &gt; 18 months and HCG negative</td>
<td>• PCOS can present in lean adolescents and those with obesity</td>
<td>• T4/TSH</td>
<td>• Oral contraceptive pills (OCPs) is first line treatment for most</td>
<td></td>
</tr>
<tr>
<td>• Not every adolescent with obesity and menstrual irregularity has PCOS</td>
<td>• Not every adolescent with obesity and menstrual irregularity has PCOS</td>
<td>• Free testosterone, total testosterone, DHEA-S, sex hormone binding globulin</td>
<td>• Progestin monotherapy is an alternative if OCPs are contraindicated</td>
<td></td>
</tr>
<tr>
<td>• Hirsutism (may or may not be clinically evident)</td>
<td>• Hirsutism (may or may not be clinically evident)</td>
<td>• Early morning 17-OH progesterone</td>
<td>• Lifestyle modification and dietary control</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Provera challenge if oligomenorrhea</td>
<td>• Beneficial effects of exercise in adolescents have been found for a range of metabolic, anthropometric, and cardiorespiratory fitness related outcomes</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Consider pelvic US</td>
<td>• Weight loss shown to improve insulin sensitivity and reduce cardiovascular risk.</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Consider 2 hour OGTT</td>
<td>• Consider Metformin: most effective in combination with weight loss</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>• Metformin clearly indicated for abnormal glucose tolerance</td>
<td></td>
</tr>
</tbody>
</table>

### Orthopedic Conditions

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>Evaluation</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Blount’s Disease</td>
<td>• Early walking (before the age of 12 months) in a child with severe obesity</td>
<td>• AP and Lateral views of the tibia</td>
</tr>
<tr>
<td></td>
<td>• Dome-shaped metaphysis, open growth plate and disruption of the continuity between the lateral borders of the epiphysis and metaphysis, with inferomedial translation of the proximal tibial epiphysis</td>
<td></td>
</tr>
<tr>
<td>Slipped Capital Femoral Epiphysis</td>
<td>• Hip pain or limp: consider referred pain to groin, knee, limb</td>
<td>• AP and Lateral views of the hips</td>
</tr>
<tr>
<td></td>
<td>• M:F = 1.5:1</td>
<td>• Ultrasound</td>
</tr>
<tr>
<td></td>
<td>• Age of onset Males = 12.7-13.5 years</td>
<td>• Degree of severity depends on avascular necrosis and/or instability</td>
</tr>
<tr>
<td></td>
<td>• Age of onset Females = 11.2-12 years</td>
<td></td>
</tr>
<tr>
<td></td>
<td>• Severe obesity at 5 to 6 years old: 5.9 times greater risk of SCFE (95% CI 3.9–9.0) compared with those with a normal BMI; Severe obesity at 11 to 12 years had 17.0 times the risk of SCFE (95% CI 5.9–49.0).</td>
<td></td>
</tr>
<tr>
<td>Scoliosis</td>
<td>• Physical findings may be obscured by obesity</td>
<td>• Shoulder height asymmetry or use of a scoliometer in addition to the traditional Adam Forward Bend Test</td>
</tr>
<tr>
<td></td>
<td>• Increased curve magnitude at presentation</td>
<td></td>
</tr>
</tbody>
</table>

Reference/s: [168] [169] [170] [171] [172] [173] [174] [175] [176] [177] [178] [179] [180] [181]
Nonalcoholic Fatty Liver Disease (NAFLD)

### Diagnosis
- Screen: severe obesity or family history NAFLD
- ALT > 2X normal (NAFLD)
- ALT > 80 Nonalcoholic steatohepatitis (NASH)
- Diagnosis of exclusion: R/O genetic/metabolic, medications, dietary, infections
- ALT: currently best screening test
- Elevated AST & GGT with normal ALT suggests diagnosis other than NAFLD
- Follow-up screening for NAFLD when initial is normal: repeat q 2-3 years if risk factors are same; sooner if clinical risk factors increase

### Evaluation
- Imaging Technology:
  - Liver Ultrasound
  - MRI and MR spectroscopy
  - CT Scan
  - Transient Elastography (Fibroscan)
  - MR Elastography
  - Liver Biopsy: Gold Standard
- Persistently elevated ALT: further evaluation/other causes of chronic hepatitis.

### Treatment
- Intensive Life Style Treatment
- Improved diet and exercise
- Remove SSBs
- Medications:
  - No currently available medications or supplements are recommended
  - Liraglutide currently used in adults, studies in pediatrics needed
- Consider:
  - Vitamin E
  - Probiotics
- Emerging therapies
- If NAFLD, screen for DM, HTN, dyslipidemia
- Liver transplant in advanced cases
- Metabolic & bariatric surgery may be beneficial, further study needed

### Clinical Findings
- Clinical Signs:
  - Often asymptomatic
  - Acanthosis nigricans
  - Hepatomegaly

- Epidemiology:
  - Most common chronic liver disease in US children
  - 40+% children with obesity
  - Male > Female
  - Hispanic > Caucasian & Asian > Blacks

- At Risk Population:
  - overweight and obesity
  - OSA, insulin resistance, prediabetic, diabetic, dyslipidemia, central adiposity
  - panhypopituitarism
  - Family history of NAFLD and/or NASH

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### Vitamin D Deficiency

#### Diagnosis
- Deficiency defined by Institute of Medicine and Endocrine Society clinical practice guidelines as serum 25-hydroxyvitamin D [25(OH)D] < 20 ng/mL

#### Significance
- Fat soluble vitamin essential for skeletal health in growing children
- Important role for bone health through the absorption of calcium from small intestine
- Available in diet and through synthesis from sunlight
- Low serum vitamin D status in US adolescents has been found to be strongly associated with hyperglycemia, hypertension, and metabolic syndrome with relationship independent of adiposity
- A recent meta-analysis of patients with T2DM provided level 1 evidence that vitamin D supplementation may reduce chronic low-grade inflammation
  - Lower levels of CRP, TNF-alpha and ESR, and increased leptin concentrations found in vitamin D supplemented groups

#### Treatment
- Children aged 1–18 years:
  - 1) 2000 IU/d of vitamin D2 or vitamin D3 for at least 6 weeks or
  - 2) 50,000 IU of vitamin D2 or D3 once a week for at least 6 weeks to achieve a blood level of 25(OH)D above 20 ng/ml, followed by maintenance therapy of 600-1000 IU/d

#### Special considerations
- Children with obesity, malabsorptive syndromes, or on medications affecting vitamin D metabolism (i.e. anticonvulsants, glucocorticoids, antifungals, and antiretrovirals) may require 2 to 3 times the dose of vitamin D to achieve the same serum 25(OH)D levels as children without these conditions
Behavioral Health Problems and Children with Obesity

Common Behavioral Health Problems Associated with Obesity in Children

- Attention Deficit Hyperactivity Syndrome
- Depression
- Emotional Eating
- Anxiety
### Attention Deficit Hyperactivity Disorder and Obesity in Children

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>Associations</th>
<th>Evaluation</th>
<th>Treatment/Medication</th>
</tr>
</thead>
</table>
| • Theoretical hypotheses  
  • Impulsivity and inattention: risk factors for weight gain and ADHD  
  • Reward deficiency hypothesis  
  • Insufficiency of dopamine  
  • In children < 10 yrs, ADHD associated with lower not higher BMI  
  • Strongest association is between ADHD and adolescent females but overall effect is very small  
  • Preadolescent boys are the biggest group diagnosed with ADHD and in that group the association with obesity is very small | • Difficulty in planning as a consequence of inattentiveness often expressed by skipping breakfast and lunch.  
  • Binge eating is one of the manifestations of impulsivity  
  • Patients with ADHD can suffer from short sleep duration due to insomnia leading to delayed onset of melatonin  
  • ADHD is highly associated with other psychiatric disorders including seasonal depression, anxiety.  
  • Short sleep duration, binge eating, and skipping meals are all associated with obesity. | • ADHD or inattention presents at ages 7-8  
  • Assess level of physical activity  
  • Inattention is associated with reduced levels of physical activity in childhood and associated with obesity in adolescence  
  • Assess for co-existing conduct disorder, an additional risk factor | • Physical activity is a mediating factor  
  • Social play may alleviate ADHD symptoms in children  
  • Address academic performance as ongoing poor performance is associated with obesity in adolescence  
  • There is association of un-medicated ADHD with higher BMIs during childhood compared with those without ADHD or ADHD treated with stimulants.  
  • Starting stimulants at younger age and longer duration associated with slower BMI growth earlier in childhood but a more rapid rebound to higher BMIs later |
| Reference/s: [211] [212] [213] [214] [215] [216] [217] [218] [219] [220] [221] [222] |

### Depression and Obesity in Children

<table>
<thead>
<tr>
<th>Association</th>
<th>Evaluation</th>
<th>Treatment</th>
</tr>
</thead>
</table>
| • Children with OB/OW significantly more likely to have depression  
  • More severe depression in groups with the most obesity  
  • Increase in use of social media associated with an increase in body dissatisfaction  
  • Peer victimization, bullying and teasing increase with obesity status and are strongly associated with depression  
  • Depression strongly associated with physical inactivity and binge eating | • Girls more likely to suffer from depression than boys  
  • Diagnosis of depression and antidepressant usage are independently associated with BMI trajectory over time  
  • Antidepressants more than twice as likely to be prescribed to low SES children  
  • Bidirectional association between depression and obesity | • Combination of obesity and depression (as well as other mood disorders) is strongly associated with an increased risk for early CVD (5 fold)  
  • Depression should be considered as big a risk factor for early CVD as obesity especially in combination with components of the metabolic syndrome  
  • Metabolic & bariatric surgery: initial improvement in depression with weight loss but return of symptoms with weight regain |
| Reference/s: [223] [224] [225] [226] [227] [228] |
### Anxiety

<table>
<thead>
<tr>
<th>Evaluation</th>
<th>Diagnosis</th>
<th>Clinical Findings</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Screening tools</td>
<td>• SCARED</td>
<td>• Differential Diagnosis</td>
<td>• Routine screening for anxiety in children and adolescents presenting for obesity management</td>
</tr>
<tr>
<td>• QOL measures—not always specific for anxiety</td>
<td>• Generalized anxiety disorder</td>
<td>• Prevalence</td>
<td>Counseling:</td>
</tr>
<tr>
<td>If positive: Referral to mental health professional who may use</td>
<td>• Social anxiety disorder</td>
<td>• General population: 20-25% of youth</td>
<td>• CBT</td>
</tr>
<tr>
<td>• SAFA Scale Score: has subscales for age ranges</td>
<td>• Specific phobias</td>
<td>• Odds of having severe obesity vs obesity were 5x higher for those with anxiety</td>
<td>• Mindfulness Based Psychotherapies</td>
</tr>
<tr>
<td>• The Hospital Depression and Anxiety Index-self-assessment scale</td>
<td>• Obesity in children is strongly associated with depression and anxiety even after other risk factors are taken into account, e.g. socioeconomic status, parental depression, other neuropsychiatric disorders</td>
<td>• Anxiety with increased BMI &gt;F than in M</td>
<td>• Psychodynamic Psychotherapy</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Social anxiety: strong association with obesity in elementary aged pts with severe obesity &gt; obesity &gt; adolescent population</td>
<td>Psychotherapy</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• May be a barrier to seeking obesity treatment</td>
<td>• Fluoxetine</td>
</tr>
</tbody>
</table>

Reference/s: [228] [229] [230] [231] [232] [233] [234] [235] [236]

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### Emotional Eating

<table>
<thead>
<tr>
<th>Etiology</th>
<th>Diagnosis/Evaluation</th>
<th>Clinical Findings</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Hx of high dietary restraint</td>
<td>Diagnosis</td>
<td>• F&gt;M but low prevalence in children</td>
<td>• Should not focus on calorie-restricted diet but on emotion regulation skills</td>
</tr>
<tr>
<td>• Poor interoceptive (feelings of hunger/satiety) awareness</td>
<td>Eating in response to negative emotions or stress</td>
<td>• Usually emerges in adolescence</td>
<td>• Dialectical behavior therapy with modules on mindfulness, emotion regulation and distress tolerance</td>
</tr>
<tr>
<td>• Alexithymia or emotion dysregulation</td>
<td>• Prior dieters at higher risk</td>
<td>• Associated with depression</td>
<td></td>
</tr>
<tr>
<td>• Reversed hypothalamic pituitary adrenal (HPA) stress axis (normal response to stress is loss of appetite)</td>
<td>• Associated with PTSD, adult and childhood trauma exposure or emotional abuse</td>
<td>• Genetic loading combined with parental psychological control</td>
<td></td>
</tr>
<tr>
<td>• May be the outcome of inadequate parenting or depressive feelings in interaction with genetic susceptibility</td>
<td>• Emotional eaters shift negative emotions narrowly to food cues</td>
<td></td>
<td></td>
</tr>
<tr>
<td>• May be a mediator between depression and obesity</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Eating in response to negative emotions or stress**

**Prior dieters at higher risk**

**Associated with PTSD, adult and childhood trauma exposure or emotional abuse**

**Emotional eaters shift negative emotions narrowly to food cues**

**DEBQ (Dutch Eating Behavior Questionnaire)**

Reference/s: [228] [237]

## Eating Disorders

### Binge Eating

**Prevalence:**
- 30% patients with obesity
- 0.7-4% general population
- 1.5 X more likely female population

**Average age of onset:**
- Late adolescents- early 20s

**Pathophysiology:**
Pathological overeating related to:
- Dysfunction of dopamine (DA) and norepinephrine systems.
- Food stimulation results in abnormal DA responses in pts with obesity.
- Methylphenidate-mediated inhibition of DA transport results in greater increases in DA levels within caudate in patients with obesity and BED compared to obesity/non BED population.

**Clinical Findings:**
- Recurrent episodes of eating an abnormally large amount of food
- Recurrent inappropriate compensatory behavior in order to prevent weight gain.
- Binge episodes cause distress
- Consists of feeling out of control while eating larger amounts of food than most people would eat in 2 hours
- Self-evaluation is influenced by body share and weight
- Two Subtypes:
  1. Purging
  2. Non-purging

**Treatment**
- Cognitive Behavioral Therapy
- Interpersonal psychotherapy
- Dialectical Behavior Therapy
- Pharmacotherapy

**Agents that help with BED and Weight Loss:**
- Topiramate (not FDA approved)
- Vyvanse
  - Not FDA-approved for isolated BED in children
  - Approved for treatment of ADHD (ages 6-17)
  - Inhibits reuptake of DA and norepinephrine
  - Elicits release of monoamine neurotransmitters likely altering pathologic BE.
- Agents that help with BED symptoms without weight loss:
  - Tricyclic Agents
  - SSRIs
  - SSNRIs

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### Night Eating Syndrome

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### Bulimia Nervosa

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### Sleep Related Eating Disorder
### Bulimia Nervosa

<table>
<thead>
<tr>
<th>Evaluation</th>
<th>Diagnosis</th>
<th>Treatment</th>
<th>Challenges</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Self report by patient</td>
<td>• Recurrent episodes of eating an objectively large amount of food with an associated loss of control</td>
<td>• Medical complications: electrolyte imbalance, esophageal tears, gastric disruption, infertility, dental decay</td>
<td>• Since patients hide symptoms, diagnosis is difficult</td>
</tr>
</tbody>
</table>
| • Patient is ashamed and tries to keep diagnosis a secret   | • Inappropriate compensatory behavior (self-induced vomiting, misuse of laxatives or diuretics, fasting, or excessive exercise) | • Psychological treatment:  
  • Cognitive behavioral therapy  
  • Integrative cognitive-effective therapy | • Patients often conceal their inappropriate compensatory behavior even after treatment |
| • Adolescents are susceptible population   | • Overvaluation of shape and weight | • Medication: only one medication trial (N=13) of fluoxetine in conjunction with CBT in adolescents with obesity and overweight showing limited efficacy | • High index of suspicion when adolescents present with mood or anxiety disorders |
| • Prevalence: 1-1.5% for strict criteria, much higher for subthreshold presentations   | • Minimum frequency of once per week over 3 months | • Studies of psychotherapeutic approaches have been inconclusive | • High level of comorbidity |
| • Median age is 12.4 years   | • Subthreshold presentations common | | • Can occur at ANY weight |
| • 41% report purging   | | | |
| • Strong association with mood (50%) and anxiety (66%) disorders   | | | |
| • 53% with suicidal ideation | | | |

### Night Eating Syndrome

<table>
<thead>
<tr>
<th>NES vs BED vs SRED</th>
<th>Prevalence/Evaluation</th>
<th>Clinical Findings/Diagnosis</th>
<th>Treatment</th>
</tr>
</thead>
</table>
| **NES vs BED**     | • Although 7-24% NES pts also meet BED, still a different diagnosis  
  • In BED pts eat large amounts in one sitting, in NES pts eat smaller amounts throughout the night  
  • NES also associated with nocturnal anxiety, not so with BED | • 1st described in 1955: delay in the circadian intake of food with 25% or more of intake in evening or night  
  • Wake up 2/week plus 2 qualifiers:  
    • Morning anorexia  
    • Strong urge to eat between dinner and bed and/or night  
    • Sleep onset/insomnia 4 nights/week  
    • Belief that one must eat to get to sleep  
    • Mood frequently depressed and/or mood worse in evening  
    • Awareness and recall of eating  
    • Present for 3 months | • Anti-Depressants: SSRIs  
  • Cognitive Behavioral Therapy  
  • Melatonin  
  • Relaxation Techniques |
| **NES vs SRED**     | • Timing of nocturnal eating: SRED occurs during slow wave sleep, no disturbance prior to falling asleep, NES occurs between dinner onward and during wakeful periods, Frequently NES patients have insomnia  
  • State of consciousness during nocturnal eating: SRED patients have partial or complete amnesia for eating episodes, NES patients can recall eating, episodes occur during wakefulness  
  • Rate of comorbid sleep disorders present in those with nocturnal eating: 80% of SRED patients have associated comorbid sleep disorders (RLS, PLMS, somnambulism)  
  • Food Type: SRED patients frequently consume non food items, can injure themselves during eating/cooking, NES patients eat food, not at risk of injury | • Possible genetic link: PER1 gene—possibly involved in controlling your body clock  
  • Defect in this gene could cause NES  
  • Diagnosis of exclusion  
  • Substance abuse  
  • Other medical disorders  
  • Medication  
  • Other psychiatric disorders  
  • Sleep Study | |
| **NES vs SRED**     | | | |
Sleep Related Eating Disorder (SRED)

<table>
<thead>
<tr>
<th>Evaluation</th>
<th>Diagnosis</th>
<th>Clinical Findings</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sleep study:</td>
<td>• Rule out underlying causes of SRED, medications, concurrent psychiatric dxs.</td>
<td>-SRED is a parasomnia — abnormal activity or behavior that occurs while falling asleep, sleeping or waking up.</td>
<td>Discontinue medications that can be triggers.</td>
</tr>
<tr>
<td>• 50-70% somnambulism</td>
<td>• Review sleep-related questions: sleep hygiene</td>
<td>-Episodes of SRED occur in the first half of the night and include:</td>
<td>-Stop or change medications that may be contributing</td>
</tr>
<tr>
<td>• 25% PLMS</td>
<td>• Complete questionnaire to determine sleep-wake pattern / level of daytime sleepiness.</td>
<td>• Frequent episodes, generally nightly, of eating and drinking in an out-of-control manner</td>
<td>-Treat other sleep disorders.</td>
</tr>
<tr>
<td>• 10-14% OSA</td>
<td>Information from your sleep partner, parent or other household members may be helpful.</td>
<td>• Impaired consciousness while preparing and eating food</td>
<td>-Treat other associated sleep disorders such as sleepwalking, restless legs syndrome or obstructive sleep apnea.</td>
</tr>
<tr>
<td>• 5-6% where nocturnal eating was observed — occurred during NREM sleep</td>
<td></td>
<td>• Little or no memory of these actions the next morning</td>
<td>Safety strategies.</td>
</tr>
<tr>
<td>Medications that may be associated with SRED</td>
<td>Differential Diagnosis:</td>
<td>• Eating high-carbohydrate and high-fat foods or odd combinations of food</td>
<td>• Safety and gently coax back to bed without using restraint or awakening. Strategies may also include changes to your sleep routine.</td>
</tr>
<tr>
<td>• Zolpidem, triazolam, amitriptyline, olanzapine, risperidone</td>
<td>• Nocturnal Eating Disorder</td>
<td>• Possibly experiencing injuries or engaging in dangerous food preparation activities</td>
<td>Medications:</td>
</tr>
<tr>
<td>Assess for associations with:</td>
<td>• Kleine-Levin Syndrome</td>
<td>• Not being easily awakened or redirected during the episode</td>
<td>• Pramipexole</td>
</tr>
<tr>
<td>• OSA, sleepwalking, narcolepsy and restless legs syndrome</td>
<td>• Dissociative disorder</td>
<td>• Experiencing a negative impact on your health from the nighttime eating</td>
<td>• Topiramate</td>
</tr>
<tr>
<td>• Hypnotic sleep medications, i.e. zolpidem, antidepressants or antipsychotics</td>
<td>• Bulimia nervosa w/ nocturnal eating</td>
<td></td>
<td>• Fluoxetine</td>
</tr>
<tr>
<td>• A daytime eating disorder, i.e. BN/AN</td>
<td>• Binge Eating Disorder</td>
<td>Causes:</td>
<td>Lifestyle and home remedies</td>
</tr>
<tr>
<td>• Stress, anxiety or depression</td>
<td>Prevalence: 5% general population 9-17% in patients with other eating disorders</td>
<td>• Exact mechanism unknown</td>
<td>• Keep sleep area and kitchen safe</td>
</tr>
<tr>
<td>A first-degree relative — a parent, child or sibling — with sleep-related eating disorder or sleepwalking</td>
<td>• Age of Onset: late teens early 20s</td>
<td>• Often occurs in people with history of sleepwalking, and stress</td>
<td>• Consider storing foods eaten during a SRED episode outside the kitchen or placing locks on cabinets and the fridge.</td>
</tr>
<tr>
<td>-Experiencing sleep deprivation</td>
<td>• Gender: 65% female, &gt;40% also have overweight</td>
<td></td>
<td>• Develop regular sleep and wake times.</td>
</tr>
</tbody>
</table>

**Reference/s:** [116] [118] [119] [120] [121] [252] [256] [262] [263] [264]

Social Consequences Affecting Children with Obesity

socialconsequences.org


obesitymedicine.org

## Psychosocial Consequences of Living with Obesity as a Child or Adolescent

### Adverse Childhood Experiences
- The Adverse Childhood Experience (ACE) Questionnaire is a 10-item self-report measure developed for the ACE study to identify childhood experiences of abuse and neglect.
- Adverse childhood experiences (ACEs)
  - Potentially traumatic events:
    - physical, emotional, or sexual abuse
    - parental divorce
    - incarceration of a parent or guardian
    - May have negative, lasting effects on health and self esteem.

### Bullying

### Weight Stigma

### Quality of Life

## Adverse Childhood Experiences (ACE)

<table>
<thead>
<tr>
<th>Evaluation</th>
<th>Diagnosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>The Adverse Childhood Experience (ACE) Questionnaire is a 10-item self-report measure developed for the ACE study to identify childhood experiences of abuse and neglect.</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Adverse childhood experiences (ACEs)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Potentially traumatic events:</td>
</tr>
<tr>
<td>physical, emotional, or sexual abuse</td>
</tr>
<tr>
<td>parental divorce</td>
</tr>
<tr>
<td>incarceration of a parent or guardian</td>
</tr>
<tr>
<td>May have negative, lasting effects on health and self esteem.</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Clinical Findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>ACEs create dangerous levels of stress.</td>
</tr>
<tr>
<td>ACEs are associated with negative behavioral and health outcomes, such as obesity, alcoholism, and depression</td>
</tr>
<tr>
<td>Specific ACEs predictive of obesity in childhood and adolescence include:</td>
</tr>
<tr>
<td>Death of parent</td>
</tr>
<tr>
<td>family economic hardship</td>
</tr>
<tr>
<td>Sexual abuse</td>
</tr>
<tr>
<td>Witnessing domestic violence</td>
</tr>
<tr>
<td>Physical abuse</td>
</tr>
<tr>
<td>Weight gain may be protective mechanism used by ACE survivors against further abuse</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Implement approaches to prevent and recognize Adverse Childhood Experiences (ACEs) and mitigate their impact through building resilience.</td>
</tr>
<tr>
<td>Proactively support safe, stable and nurturing family relationships (SSNRs), teach resilience, and intervene early to promote healing of the trauma and stress associated with disruptions in SSNRs</td>
</tr>
<tr>
<td>Utilize connection between ACEs, social issues, and mental/physical health to inform social programs and health policies that support prevention and recovery from ACE events.</td>
</tr>
</tbody>
</table>

**Reference/s:** [275] [276] [277] [278] [279] [280] [281] [282] [283] [284] [285] [286]
## Bullying

<table>
<thead>
<tr>
<th>Definition</th>
<th>Epidemiology</th>
<th>Consequences</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Involves intentional, and largely unprovoked, efforts to harm another&lt;br&gt;• Can be physical or verbal, and direct or indirect&lt;br&gt;• Involves repeated negative actions by one or more against another&lt;br&gt;• Involves an imbalance of physical or psychological power</td>
<td>• Being overweight or obese is one of the most common reasons that children and adolescents are teased or bullied at school&lt;br&gt;• Verbal teasing was the most frequent type of victimization reported by adolescents with obesity, followed by relational aggression, cyberbullying and physical aggression&lt;br&gt;• Boys with obesity were significantly more likely to have dual role of bully-victim, which has the highest psychosocial implications among the bullying roles, whereas girls with obesity were mainly victims</td>
<td>• Weight-based teasing in children with obesity may contribute to negative emotional consequences, academic failure, and peer rejection&lt;br&gt;• Risk of depression/low self-esteem in children with obesity is substantial even after controlling for other factors including duration of obesity&lt;br&gt;• Psychological sequelae are a consequence of victimization &amp; not simply of weight status</td>
<td>• Talk to patients/don’t blame child&lt;br&gt;• Don’t encourage children to fight back (physically)&lt;br&gt;• Telling the child to ignore the bullying may cause escalation.&lt;br&gt;• Bystanders may be affected in that they don’t often know how to respond&lt;br&gt;Consult mental health if the child has:&lt;br&gt;• severe depression, anxiety, or suicidality&lt;br&gt;• particular difficulty in discussing the bullying&lt;br&gt;• severe impairment in daily activities</td>
</tr>
</tbody>
</table>

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## Weight Stigma & Victimization

<table>
<thead>
<tr>
<th>Forms</th>
<th>Settings</th>
<th>Health Consequences</th>
<th>Recommendations</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Bullying&lt;br&gt;• Teasing&lt;br&gt;• Victimization&lt;br&gt;• Relational&lt;br&gt;• Verbal Abuse&lt;br&gt;• Physical Abuse&lt;br&gt;• Cyberbullying&lt;br&gt;• Prevalence: &gt;70 %</td>
<td>• School&lt;br&gt;• Peers&lt;br&gt;• Educators&lt;br&gt;• Home&lt;br&gt;• Parents&lt;br&gt;• Family members&lt;br&gt;• Media&lt;br&gt;• Healthcare Providers&lt;br&gt;• All disciplines</td>
<td>• Emotional&lt;br&gt;• Physical&lt;br&gt;• Psychological&lt;br&gt;• Social&lt;br&gt;• Academic&lt;br&gt;• Unhealthy eating behaviors&lt;br&gt;• Decreased activity&lt;br&gt;• Increased weight</td>
<td>• Positive role modeling&lt;br&gt;• Appropriate language (person first)&lt;br&gt;• Clinical management of comorbidities&lt;br&gt;• Counseling&lt;br&gt;• Behavioral health screening&lt;br&gt;• Coping with stigma stress&lt;br&gt;• Healthy behaviors&lt;br&gt;• Advocacy</td>
</tr>
</tbody>
</table>

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Reference/s: [287] [288] [289] [290] [291] [292] [293] Pediatric Obesity Algorithm®. ©2020-2022 Obesity Medicine Association.
Health Related Quality of Life (HRQOL)

**Assessment**

- Impact of Weight Quality of Life-Kids (IWQOL-Kids®) Tool
  - 4 Domains: physical comfort, body-esteem, social life, family relations
- Pediatric Quality of Life Inventory (PedsQL™) Tool
  - Domains: Physical, Emotional, Social, School
- Parent-proxy tools are available

**Health Consequences**

- Psychosocial
  - Depression
  - Low self-esteem
  - Altered eating patterns
  - Sleep disturbances
  - Increased cardiometabolic abnormalities may decrease QOL scores
  - Inverse relationship between lower QOL scores and increased BMI

**Recommendations**

- Interventions: screen and treat mood and social problems: address family/friend support for healthy eating.
- Improvements may be due to treatment program itself and not exclusively to reductions in BMI; however, improved HRQOL seen with sustained weight loss
- BMI may not directly relate to improved QOL but may be associated through other factors, including child social problems.
- Perform comprehensive biopsychosocial assessment when providing treatment to youth with obesity and their families.

References: [309] [310] [311] [312] [313] [314] [315] [316] [317] [318] [319] [320] [321] [322] [323]
# Genetic Syndromes Associated with Childhood Obesity

<table>
<thead>
<tr>
<th>Syndrome</th>
<th>Associated Features</th>
</tr>
</thead>
<tbody>
<tr>
<td>Prader-Willi Syndrome</td>
<td>Hypotonia at birth, difficult feeding followed hyperphagia, hypogonadism, Intellectual Disabilities (ID), deletion of q11-13 region</td>
</tr>
<tr>
<td>Bardet-Biedl Syndrome</td>
<td>Retinitis pigmentosa, polydactyly, ID, hypogonadism, renal abnormalities</td>
</tr>
<tr>
<td>Fragile X Syndrome</td>
<td>Macro-orchidism, ID, prominent jaw, large ears</td>
</tr>
<tr>
<td>Albright's Hereditary Osteodystrophy</td>
<td>Short stature, skeletal defects, resistance to several hormones</td>
</tr>
<tr>
<td>Alstrom Syndrome</td>
<td>Photophobia, nystagmus, deafness, blindness, diabetes</td>
</tr>
<tr>
<td>Congenital Leptin Deficiency</td>
<td>Hypogonadism, intense hyperphagia, absence of growth spurt, T-immune dysfunction, frequent infections</td>
</tr>
<tr>
<td>POMC Deficiency</td>
<td>Hyperphagia and early-onset obesity; adrenal crisis due to ACTH deficiency, hypoglycopenia</td>
</tr>
<tr>
<td>MC4R Deficiency</td>
<td>Increased linear growth and final height, severe hyperinsulinemia</td>
</tr>
<tr>
<td>Cohen Syndrome</td>
<td>Chromosome 8q22 mutation. Obesity (central only- thin arms and legs), ID, head/face defects (small jaw, shortened philtrum, high raised palate). Some children have seizures and deafness.</td>
</tr>
<tr>
<td>Beckwith-Wiedemann Syndrome</td>
<td>Chromosome 11p15.5 mutation. Macroglossia, hepatosplenomegaly, hypoglycemia in 30-50% of babies, predisposition to tumor development</td>
</tr>
</tbody>
</table>

*This list is not comprehensive*

**References:** [139] [264] [267] [268] [269] [270] **Pediatric Obesity Algorithm®. ©2020-2022 Obesity Medicine Association.**

## Prader-Willi Syndrome

### Presentation
- Small and unusually floppy at birth
- Almond shaped eyes, thin upper lips, thin facies
- Low muscle mass, small hands and feet
- Delayed motor development
- Intellectual and speech delay

### Behaviors
- Voracious and insatiable appetite, which often leads to obesity, usually starts around 2 years
- Severe behavioral problems

### Diagnosis
- Diagnosis: Paternal chromosome 15q partial deletion or unexpression
- 70% with paternal allele deletion, 25% with maternal disomy, and 5% with translocation or other structural abnormalities

### Complications
- Scoliosis
- Sleep Apnea
- Osteoporosis
- Infertility due to lack of development of secondary sexual characteristics

### Special Characteristics
- Hypotonia, poor suck, characteristic facial features
- Bitemporal narrowing of the head, almond shaped eyes, elongated face, and thin upper lip
- May have relatively normal weight until 2-5 years of age, then rapid weight gain
- Ketotic at levels of carbohydrate intake that are higher than normally expected
- Hunger control

### Treatment
- Calorie control and behavioral management
- Growth hormone therapy and replacement of sex hormones at puberty

**References:** [139] [264] [267] [268] [269] [270] **Pediatric Obesity Algorithm®. ©2020-2022 Obesity Medicine Association.**
### Additional Genetic Disorders Associated with Childhood Obesity

#### Bardet-Biedl Syndrome
- Autosomal recessive ciliopathic genetic disorder
- Obesity, visual problems (including retinitis pigmentosa, loss of vision), polydactyly, hypogonadism
- Often mental retardation and kidney failure
- Test for Bardet Beidl syndrome: BBS gene mutation.

#### Cohen Syndrome
- Chromosome 8q22 mutation
- Obesity (central only- thin arms and legs), ID, head/face defects (small jaw, shortened area between the nose and upper lip, high raised palate)
- Some children have seizures and deafness

#### POMC Deficiency
- Autosomal recessive
- Hyperphagia and early-onset obesity; adrenal crisis due to ACTH deficiency; hypopigmentation

#### Beckwith-Wiedemann Syndrome
- Fetal overgrowth syndrome
- Chromosome 11p15.5
- Macroglossia, hepatosplenomegaly and/or nephromegaly
- Growth parameters typically with height and weight above the 95th percentile
- Hypoglycemia in 30-50% of babies
- Predisposition of tumor development: surveillance includes abdominal ultrasounds and AFP levels

#### Alstrom Syndrome
- Very rare- ALMS 1 mutation
- Childhood obesity, blindness and hearing loss
- Photophobia, nystagmus, deafness
- Can have endocrine issues such as type II diabetes, high insulin, and high triglycerides

#### Fragile X Syndrome
- Autosomal recessive
- Hyperphagia and early-onset obesity; adrenal crisis due to ACTH deficiency; hypopigmentation
**Additional Genetic Disorders Associated with Childhood Obesity**

**MC4R Deficiency**
- MC4R mutations are the most common monogenic form of obesity.
- 2-5% of severe obesity in pediatric and adult populations
- Increased linear growth and final height, increased fat mass, severe hyperinsulinemia

**Albright’s Hereditary Osteodystrophy**
- Short stature, skeletal defects, shortened 4th and 5th metacarpals
- Impaired olfaction
- Developmental delay
- Resistance to several hormones

**Congenital Leptin Deficiency**
- Autosomal recessive
- Homozygous mutations of the leptin gene
- Hypogonadotropic hypogonadism, intense hyperphagia and impaired satiety, absence of growth spurt.
- T-Immune dysfunction, frequent bacterial infections
- Clinical response to recombinant leptin administration with major effect on appetite

**Rare Genetic Variants of Obesity**

**Genetic Factors**
- Genetic factors are estimated to account for 40-70% of the obesity predisposition of an individual
- Genetic causes underlying obesity remain largely unknown
- There are rare genetic variants affecting several genes such as LEP, LEPR, MC4R, PCSK1, and POMC found in non-syndromic patients
- Estimated that 7% of patients with severe pediatric obesity may have rare abnormalities of chromosomes and/or highly penetrant genetic mutations

**Genome Wide Association Studies (GWAS)**
- GWAS have found strongly associated genomic variants with a number of loci reported from GWAS analyses of adult BMI and obesity also having a role in obesity in pediatric populations.
- Genome wide approach has revealed genes that underlie the pathogenesis of childhood obesity.
- Understanding genetics of childhood obesity is vital in the prevention and treatment of cases in pediatrics

**GWAS Approach**
- GWAS approach provides a more comprehensive strategy to locate causal genes which are obesity related.
- Challenges arise from lack of availability of resources and limited GWAS-related reports specific for childhood obesity
- Most studies uncovering loci in the adult context.
Advanced Treatment for Pediatric Obesity

- Pharmacotherapy
- Emerging Bariatric Technologies
- Metabolic and Bariatric Surgery (MBS)
- Post Metabolic & Bariatric Surgery + Pharmacology
Adjustment of Weight Promoting Medication

- Weight gain secondary to medications is an important modifiable risk factor in the development of obesity.
- Several medications are associated with weight gain, and include certain antidiabetic, antipsychotic, antidepressant, antihypertensive, anti-epileptic, and antihistamine agents, in addition to glucocorticoids and hormonal contraceptives.
- It is important for providers to be familiar with alternative medications within medication classes which are weight neutral or have weight loss effects in the management of medication induced weight gain.*
  - For patients with obesity and T2DM requiring insulin, it is recommended to prescribe at least one weight loss promoting medication, such as metformin, pramlintide, or a GLP-1 agonist to decrease the weight gain associated with insulin administration.
- If no alternative medications are available, weight gain can be prevented or decreased by choosing the lowest medication dose to produce clinical efficacy for the shortest duration necessary during treatment.

*Please see Medication Related Weight Gain section for further details.
## Pharmacotherapy

### Orlistat
- FDA approved in children > 12 years
- Weight loss is small
- Side effects preclude usage in most patients.
- May cause oily stools

### Metformin
- FDA approved in children with T2DM > 10 years
- Provides very modest reduction in BMI when combined with lifestyle interventions over short time
- May have a role in prediabetes and/or strong family hx of T2DM
- Dose in adolescents, if tolerated, should be at the maximum recommended dose of 1,000 mg twice daily to take advantage of the effect of decreasing appetite
- May prolong duration of time before onset of T2DM
- May cause gastrointestinal upset especially in first few weeks, can be managed with dose titration

### Topiramate
- Not FDA approved in children for weight loss
- Has been used for seizure control in children for years
- May control cravings
- Can cause paresthesias of extremities, cognitive disruption (confusion, difficulty concentrating)
- Warnings: avoid in patients with acute myopia and secondary angle closure glaucoma, consider discontinuing for visual field defects, watch for oligohidrosis and hyperthermia, metabolic acidosis, suicidal behavior, and ideation. May cause cognitive slowing. Do not use in combination with a ketogenic diet. Do not use during pregnancy; associated with cleft lip and/or palate and small for gestational age.

### Phentermine
- FDA approved in children > 16 years for weight loss
- Has been used in adolescents
- Weight loss is small to moderate.
- May cause anxiety, tremors, slightly increased blood pressure
- Warnings and Precautions: Should not be used in combination with fenfluramine or dexfenfluramine. Contraindicated in patients with serious regurgitant cardiac valvular disease. Use cautiously in patients with HTN, monitor for an increase in BP. Phentermine is categorized as "Pregnancy Category X" meaning that no animal studies have been conducted. No studies have been done in nursing mothers. Use cautiously in patients with severe renal impairment. If GFR is < 30 mL/min, limit dose to 15 mg daily. Avoid use if GFR < 15 mL/min.

### Exenatide
- FDA status: not approved for pediatric use
- MOA: slows gastric motility, suppresses appetite
- Reduction of BMI (4%) and body weight in adolescents with severe obesity in small RCT of 10 mcg BID. Slight rebound of BMI after down titration of dose, then recovery when dose increased again
- Preliminary evidence suggests that higher doses for prolonged periods of time may be necessary for weight loss

### Bupropion
- FDA status: not approved for pediatric use
- MOA: aminoketone class of antidepressants, unrelated to SSRIs. Moderates the levels and activity of neurotransmitters norepinephrine and dopamine, exactly how it works to treat depression uncertain.
- Black Box warning for worsening of depression in pts 18-24 yrs. of age.
- May increase risk of seizures – particularly in pts with epilepsy, eating disorder such as bulimia or anorexia nervosa.
- Indications:
  - Depression
  - Seasonal Affective Disorder
  - Smoking Cessation (Zyban)
  - Wellbutrin and Weight Loss
  - Unlike other antidepressants it is not associated with weight gain
- Not FDA approved for weight loss

### Lisdexamfetamine
- FDA approved for ADHD in children in 2007, not for weight loss
- FDA approved for BED in adults in 2015 (50-75 mg/day), not for weight loss
- MOA: LDX hydrolyzes to release the active drug, d-amphetamine which inhibits reuptake of dopamine and/or NE and enhances release of DA, NE, and serotonin. LDX may decrease pathological overeating and therefore tx BED.
- SE: decreased appetite, insomnia, HA, GI upset, irritability, monitor for CV changes (increased BP/pulse)
- Contraindications: MAOI use
- Scheduled II controlled drug;
- Prescribing rules apply as with other stimulants
Pharmacology

Naltrexone/Bupropion
- FDA approved for weight loss with BMI > 30 or BMI >27 with at least one weight-related condition.
- MOA: reuptake inhibitor of Norepinephrine and dopamine (Wellbutrin) and opioid antagonist (naltrexone)
- 4.8% weight loss in trial over placebo – study duration 1 yr.
- Advantages: greater weight loss, food addiction, long term data
- Common Side effects: Nausea, constipation, headache, vomiting and dizziness
- Concerns: Worsening depression and/or suicidal ideation in youth
- Contraindications: uncontrolled HTN, Seizure disorders, anorexia or bulimia, drug or alcohol withdrawal, MAOI usage

Phentermine/Topiramate
- FDA status: not approved for pediatric use
- Combination of phentermine and topiramate
- Black box warning for worsening of depression in pts 18-24 yrs: www.fda.gov/bbs/topics/AN
- Contraindications: pregnancy, glaucoma, hyperthyroidism, monoamine oxidase inhibitors within 14 days, hypersensitivity or idiosyncrasy to sympathomimetic amines
- Adverse reactions: paraesthesia, dizziness, dysgeusia, insomnia, constipation, dry mouth, suicidal ideation, possible seizures if medication discontinued abruptly
- Warnings: fetal toxicity, metabolic acidosis
- Avoid: pregnancy, alcohol. Activities requiring concentration until establishing effect (if any) on these activities

Liraglutide
- Not FDA approved for weight loss in adolescents, approved at 3.0 mg for adults
- In RCT with adolescents with obesity (BMI 35 kg/m2), treatment group more than twice as likely to reduce BMI by 5%, but adverse effects high in treatment group at 10% leading to discontinuation
- Treatment emergent adverse events: GI disorders (abdominal pain), hypoglycemia
- In small study of children with obesity with prediabetes, significant reduction in FBG and 2 hour postprandial glucose noted
- FDA approved for use in Type 2 diabetes with the use of Metformin and with or without the use of basal insulin but weight loss not as great, and not significant, for adolescents with obesity and T2D as in adults

Emerging Bariatric Technologies

Categories
- Placement varies: endoscopy, laparoscopy, patient swallows
- Restrictive
  - Intragastric balloons
  - Patient swallows or Endoscopically placed
- 3 FDA approved adults in US
- Malabsorptive endoscopic procedures
- Luminal sleeve (not FDA approved; in trial)
- Endoscopic gastroplasty techniques
- Intragastric gastroplasty (multi-institutional trials)
- Gastric electrical stimulation
- Vagal nerve stimulation
- Aspiration (FDA approved adults)

Advantages & Challenges
- Temporary
- Removable
- Adjustable
- May bridge to more invasive treatment performed at safer weight/condition
- Recommended that these technologies are used in conjunction with multi-disciplinary weight management program
- Widespread use has been limited by reproducibility, long-term durability, and lack of insurance coverage

Adolescent Specific Info
- None of the emerging bariatric technologies and devices FDA approved in adults are currently approved for adolescents less than 18 years of age.
- Building on FDA safety & efficacy trials in adults, consider bariatric technologies, endoscopic surgical procedures, and medical devices for prospective clinical trials in adolescents.
- Outside US, 4 preliminary studies (small subject numbers) describe trials of intragastric balloon in adolescents within a multi-disciplinary weight management program. Demonstrate safety & effectiveness consistent with adult data warranting adolescent trials in US. (Fittipaldi-Fernandez et al, 2017; De Peppo et al, 2017; Nobili et al, 2015; Reece et al, 2017.)
**Adolescent Metabolic & Bariatric Surgery**

**Indications**

- **Indications** for adolescents mirror the NIH recommendations for adults:
  - BMI ≥ 40 kg/m²
  - BMI ≥ 35 kg/m² with significant current comorbidities including severe OSA, T2D, hypertension.

  In adolescents, use of percentile BMI values are used along with existing severe medical conditions and/or debilitating quality of life.

- MBS considered for youth with severe obesity: BMI ≥ 120% of the 95th percentile.

**Outcomes**  
**(5 year Teen Labs Study)**

- MBS Outcomes (5 year Teen Labs Study)
  - 96% follow up at 5 years
  - Mean percent weight loss=26%
  - 68% normalized blood pressure
  - 81% normalized triglycerides
  - 86% with T2DM in remission
  - 48% low ferritin levels

- Mental Health Outcomes (5 year AMOS Study)
  - At 5 years: Self-esteem improved, mood down slightly
  - Mental health problems persist after surgery necessitating ongoing MH treatment

**Recommendations**

- Adolescents undergoing MBS need to be followed in weight management clinic pre and postoperatively, preferably American College of Surgeons MBSAQIP recommendations including sensitivity training.
- Vertical sleeve gastrectomy (VSG) most common operation with adolescents; both Roux-en-Y Gastric Bypass and VSG similar risk/benefit
- Screen for micronutrient deficiencies at baseline and ongoing after MBS.
- Obesity is a chronic disease requiring multimodal therapies and treatment by a multidisciplinary weight management team which can provide surgical, pharmacologic, behavioral, nutritional interventions and activity recommendations.
- Counsel females about increased fertility with weight loss
- All adolescents counseled on risk/benefit and provided informed consent along with family/guardian
- For more information, review the 2018 ASMBS Pediatric Metabolic and Bariatric Surgery Guidelines. [353]

**Post Metabolic & Bariatric Surgery + Pharmacology**

**Treatment Principles**

- Recognize that obesity is a chronic disease that requires multimodal therapies delivered by a multidisciplinary team that can provide surgical, pharmacologic, behavioral, nutritional, and activity interventions.
- Pharmacologic therapies as adjuncts to surgical therapies may provide improved outcomes long term in the pediatric population; more studies are needed.
- Since weight regain and inadequate weight loss are common in patients who undergo bariatric surgery, there is a need for a range of therapeutic options to treat this patient population.

**What to Expect Post MBS**

- Higher activity levels associated with improvements in CVD-related lipid measures and weight loss.
- Appropriate portion size and less eating while bored associated with long-term weight-loss maintenance
- Higher QOL was strongly associated with maintenance of weight loss among adolescents
- Adolescent males have higher overall QOL than adolescent females, particularly in the self-esteem domain suggesting females may require more intensive social support post-surgery

References: [332] [333] [334] [335] [336] [337] [338] [339] [340] [341]
Ongoing assessment & adjustment in response to the chronic disease of obesity

Principles

- Obesity is a chronic disease characterized by relapsing episodes of weight gain as the body struggles to retain a former set point of weight.
- Obesity is a heterogeneous disease characterized by person-specific weight loss response to obesity treatments including lifestyle modification, pharmacotherapy, endoscopic devices, metabolic and bariatric surgery, and combination therapies.
- Treatment of pediatric obesity, particularly severe obesity, requires a chronic care management by interdisciplinary teams of health care providers and multi-pronged therapies.
- The chronic care model should guide obesity care.
- As with other chronic diseases of childhood, a plan of transition to adult obesity specialists should be part of every pediatric care plan.

Reference/s: [139] [264] [268] [269] [369] [370] [371] [372] [373] [374]

Medication Related Weight Gain
## Review of Medications: ADHD, Anti-Seizure, Migraine, Diabetic Medications and Others

<table>
<thead>
<tr>
<th>Medications</th>
<th>Significant Weight Gain</th>
<th>Small to Neutral Weight Gain</th>
<th>Weight Loss (neutral to mild)</th>
</tr>
</thead>
<tbody>
<tr>
<td>ADHD</td>
<td></td>
<td>Guanfacine</td>
<td>Atomoxetine</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Lisdexamfetamine</td>
<td>Amphetamine</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Methylphenidate</td>
<td></td>
</tr>
<tr>
<td>Anti-Seizure</td>
<td>Valproate</td>
<td>Pregabalin</td>
<td>Carbamazepine</td>
</tr>
<tr>
<td></td>
<td>Vigabatrin</td>
<td>Gabapentin</td>
<td>Oxocarbazepine</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Lamotrigine</td>
<td>Levetiracetam</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Phenytoin</td>
<td></td>
</tr>
<tr>
<td>Migraine</td>
<td>Amitriptyline</td>
<td>Gabapentin</td>
<td>Timolol</td>
</tr>
<tr>
<td></td>
<td>Divalproex</td>
<td>Metoprolol</td>
<td>Levetiracetam</td>
</tr>
<tr>
<td></td>
<td>Flunarizine</td>
<td>Propranolol</td>
<td></td>
</tr>
<tr>
<td>Diabetic</td>
<td></td>
<td>Insulin and analogs</td>
<td></td>
</tr>
<tr>
<td>Medications</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Other</td>
<td>Glucocorticoids</td>
<td>Benzodiazepines</td>
<td></td>
</tr>
<tr>
<td>Medications</td>
<td>Gleevec</td>
<td>Statins</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Depo Provera</td>
<td>Antihistamines (Cyproheptadine)</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Carvedilol</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Oral Contraceptive Pills</td>
<td></td>
</tr>
</tbody>
</table>

### Antiepileptic Drugs (AEDs)

#### Incidence and Associations
- In the last 2 decades, > 11 new AEDs have been introduced to the market.
- Several AEDs are associated with weight gain such as gabapentin, pregabalin, valproic acid (VPA) and vigabatrin and to some extent carbamazepine (CBZ).
- Others are weight neutral such as lamotrigine (LTG), levetiracetam, and phenytoin or associated with slight weight loss as, e.g., felbamate.

#### Risk Factors for Weight Gain
- Weight gain occurring in 40% of children on VPAs.
- 38% of VPA-treated gained >10% of their BW compared with 8% treated with lamotrigine.
- Hyperinsulinemia /hyperleptinemia common with VPA, marked in epileptic children who gained weight.
- Alterations not seen with CBZ or LTG.
- MOA: ↑ insulin and insulin/glucose with VPA possibly stimulating appetite.

#### Special Considerations
- Topiramate and zonisamide are the 2 AEDs causing weight loss.
- Topiramate significantly reduced adiponectin, leptin/adiponectin, and markedly increase the serum level of adiponectin, increases energy metabolism, resulting in weight loss since adiponectin play a significant role in metabolic regulations.

#### Treatment of Complications
- Weight gain with the AEDs is not only a cosmetic problem but also a risk for obesity-related vascular disorders.
- As AEDs may influence weight, providers have to properly select and characterize the suitable AED as an initial step or modify the existing AED if it compromises patient’s health.

Reference/s: [336] [340] [375] [376] [377] [378] [379] [380] [381] [382] [383] [384] [385] [386] [387] [388] [389] [390] [391] [392] [393] [394] [395] [396] [397] [398] [399] [400] [401] [402] [403]
### Migraine

<table>
<thead>
<tr>
<th>Incidence and Associations</th>
<th>Risk Factors for Weight Gain</th>
<th>Special Consideration</th>
<th>Treatment of Complications</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Positive association with headache and obesity among children</td>
<td>• There are few pediatric data on the insulin regimens used, but they all seem to have equal efficacy.</td>
<td>• Topiramate is, generally, a well-tolerated migraine therapy in children that is weight negative.</td>
<td>• Recognizing and avoiding trigger factors of migraines as well as a suitable therapy strategy that is not weight positive may give the child with migraines the chance for a good quality of life.</td>
</tr>
<tr>
<td>• Migraine is ↑ in those with obesity. Risk ↑ with ↑ obesity status.</td>
<td>• Insulin and insulin analogues (insulin Glargine) cause weight gain in adolescents.</td>
<td>• Treatment starts at 15 mg/day and it is increased during an 8 week-period to 2 to 3 mg/kg daily (Max dose is 200 mg/day).</td>
<td>• Psychological and physical triggers include: stress, anxiety, worry, depression, fatigue, fever, illness, poor sleep habits, irregular meals, fasting, hypoglycemia, and dehydration.</td>
</tr>
<tr>
<td>• Overlap between migraine pathophysiology, central and peripheral pathways regulating feeding.</td>
<td>• Sulfonyleureas: not commonly used in pediatric patients and cause weight gain.</td>
<td>• Side effects: gastroenteritis, concentration difficulty, somnolence or even cognitive impairment.</td>
<td>• An important part of the prophylactic treatment in pediatric migraine is the psychological support.</td>
</tr>
<tr>
<td>• Specifically, neurotransmitters like serotonin, peptides such as orexin, and adipocytokines (adiponectin and leptin) may have roles in both feeding and migraine.</td>
<td></td>
<td>• Due to the low dose required for treatment of migraine, Topiramate can be a good choice for the pediatric population due to its efficacy and the low frequency of side effects.</td>
<td></td>
</tr>
</tbody>
</table>

### Drug Class/Drug Weight Change

- **Anti-depressants**:
  - Amitriptyline ↑↑↑
  - Nortriptyline ↑
  - Desipramine ↓
  - Venlafaxine, Duloxetine ↔
- **Calcium channel blockers**:
  - Verapamil ↔
  - Flunarizine ↑
- **β-Blockers**:
  - Propranolol ↑
  - Nadolol ↔
  - Metoprolol ↑
- **Serotonin antagonists**:
  - Cyproheptadine ↑↑↑

### Diabetes Medications

<table>
<thead>
<tr>
<th>Incidence and Associations</th>
<th>Risk Factors for Weight Gain</th>
<th>Special Consideration</th>
<th>Special Consideration</th>
</tr>
</thead>
<tbody>
<tr>
<td>• SEARCH for Diabetes in Youth study (2002-12): rate of new diagnosed cases of type 2 diabetes ↑ at 4.8% each year.</td>
<td>• There are few pediatric data on the insulin regimens used, but they all seem to have equal efficacy.</td>
<td>• Metformin: effectiveness proven for adolescents.</td>
<td>• GLP-1 receptor agonists: liraglutide, exenatide, lixisenatide, dulaglutide): few studies have been carried out in the pediatric population but several trials are currently underway.</td>
</tr>
<tr>
<td>• Since T2D in youth only recognized recently, natural history is lacking. There are only few studies examining treatments beyond metformin and insulin.</td>
<td>• Insulin and insulin analogues (insulin Glargine) cause weight gain in adolescents.</td>
<td>• Metformin decreases hepatic glucose output/ enhances primarily hepatic and muscle insulin sensitivity without a direct effect on β-cell function.</td>
<td>• The TODAY study preferred early combination therapy in pediatric patients with type 2 diabetes, rather than wait for the failure of metformin monotherapy.</td>
</tr>
<tr>
<td>• Although only metformin and insulin are currently licensed for use for under age 18 endocrinologists use oral agents for children with type 2 diabetes mellitus.</td>
<td>• Sulfonyleureas: not commonly used in pediatric patients and cause weight gain.</td>
<td>• Metformin has the advantage of weight reduction, decrease in lipids without the risk of hypoglycaemia.</td>
<td></td>
</tr>
<tr>
<td>• Thiazolidinediones (TZD): Rosiglitazone and pioglitazone are the only remaining drugs from TZD family in clinical use, and rosiglitazone was used in the TODAY study.</td>
<td></td>
<td>• Thiazolidinediones (TZD): Rosiglitazone and pioglitazone are the only remaining drugs from TZD family in clinical use, and rosiglitazone was used in the TODAY study.</td>
<td></td>
</tr>
</tbody>
</table>

Reference/s: [404] [405] [406] [407] [408] [409] [410] [411]
Review of Medications: Psychiatric

<table>
<thead>
<tr>
<th>Class</th>
<th>Significant Weight Gain</th>
<th>Small to Neutral Weight Gain</th>
<th>Weight Loss</th>
</tr>
</thead>
<tbody>
<tr>
<td>Antipsychotics</td>
<td>Clozapine, Olanzapine, Chlorpromazine, Quetiapine, Risperidone</td>
<td>Antipirazole, Haloperidol, Ziprasidone</td>
<td></td>
</tr>
</tbody>
</table>

Special considerations: "Youth may be particularly sensitive to weight gain, especially with olanzapine, as well as extrapyramidal side effects and metabolic changes." Many of the medications listed here have only been well studied in adults.

Antidepressants

<table>
<thead>
<tr>
<th>Class</th>
<th>Significant Weight Gain</th>
<th>Small to Neutral Weight Gain</th>
<th>Weight Loss</th>
</tr>
</thead>
<tbody>
<tr>
<td>Antidepressants</td>
<td>Paroxetine, Amitriptyline, Olanzapine, Citalopram, Nortriptyline, Lithium, Desipramine, Imipramine, Duloxetine, Escitalopram, Doxepin, Mirtazapine, Venlafaxine, Fluvoxamine, Sertraline, Trazodone, Fluoxetine</td>
<td></td>
<td>Bupropion*</td>
</tr>
</tbody>
</table>

Mood Stabilizers

<table>
<thead>
<tr>
<th>Class</th>
<th>Significant Weight Gain</th>
<th>Small to Neutral Weight Gain</th>
<th>Weight Loss</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mood Stabilizers</td>
<td>Valproate, Lithium</td>
<td></td>
<td>Topiramate</td>
</tr>
</tbody>
</table>

Anxiolytics

<table>
<thead>
<tr>
<th>Class</th>
<th>Significant Weight Gain</th>
<th>Small to Neutral Weight Gain</th>
<th>Weight Loss</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anxiolytics</td>
<td>Lorazepam, Diazepam, Oxazepam</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Black box warning

References: [375] [424] [425] [426] [427] [428] [429] [430] [431]
### Antidepressants and Mood Stabilizers

<table>
<thead>
<tr>
<th>Valproic Acid</th>
<th>Lithium</th>
<th>SSRI</th>
<th>SNRI</th>
</tr>
</thead>
</table>
| • Weight gain seen during first 3 months of treatment  
  • F>M  
  • Pathogenic mechanism unknown  
  • Insulin resistance  
  • Increased hunger due to low glucose levels  
  • No direct effect on lipids  
| • Risk of weight gain is high  
  • F>M  
  • No direct effect on lipids  
| • Fluoxetine, paroxetine, sertraline  
  • Most commonly used medication for treatment of depression in adolescents  
  • Small increase (0.007) in zBMI over a 14 month period of treatment as compared to no medication  
  • F>M  
  • No direct effect on lipids  
| • Mirtazapine and Trazadone  
  • F>M  
  • Unfavorable effect on triglycerides and LDL |

### Mood Stabilizers (MS)

<table>
<thead>
<tr>
<th>Incidence and Associations</th>
<th>Risk Factors for Weight Gain</th>
<th>Special Consideration</th>
<th>Treatment of Complications</th>
</tr>
</thead>
</table>
| • Mood stabilizers medications are medications that are off-label for the treatment of bipolar disorder in children and adolescents.  
  • Mood stabilizers can be divided into traditional agents lithium, valproate, and carbamazepine and newer agents, including the anticonvulsants lamotrigine, oxcarbazepine, topiramate, and gabapentin.  
  • Newer medications are often used as second-line choices behind atypical antipsychotics for overall efficacy.  
| • Data regarding body composition and fasting metabolic effects of mood stabilizers in pediatric bipolar disorder are sparse.  
  • Combining antipsychotics with mood stabilizers seems to lead to greater weight gain than treatment with one or two mood stabilizers.  
| • There is a resurgence of interest in lithium treatment of bipolar disorders due to its unique anti-suicidal and neuro-protective effects.  
  • Lithium may be less effective than risperidone for treating chronic mixed/manic symptoms in young children but comparable to anticonvulsants. However, in comparison, risperidone was associated with higher weight gain and prolactin levels than lithium.  
  • Some reports indicated that topiramate may be useful as an add-on therapy to induce weight loss in patients who have experienced psychotropic-induced weight gain.  
  • Amantadine appears to stabilize weight gain related to psychotropics. It also may help decrease BMI with continued amantadine usage but controlled trials in lacking to date. |
Appendices
- Added as reference for American Board of Obesity Medicine review

Appendix A: Staged Treatment Approach
Identification and Management

### First Steps
- Initial identification of children with overweight or obesity in primary care office
- First approach is counseling on consuming 5 or more fruits and vegetables/day, < 2 hours screen time, 1 hour of play or exercise, and no SSBs
- Family based counseling
- Motivational Interviewing

### Second Steps
- If child not losing weight, child is usually referred to weight management clinic or, if no clinic available, to see a dietitian and behavioral health (PhD/SW)

### Staged Treatment
- Recommendations for staged management were first published in 2007 in Pediatrics: AAP Expert Committee Recommendations

---

Staged Treatment Approach (Barlow)

<table>
<thead>
<tr>
<th>Prevention Plus</th>
<th>Structured Weight Management</th>
<th>Comprehensive Multidisciplinary Intervention</th>
<th>Tertiary Care Intervention</th>
</tr>
</thead>
<tbody>
<tr>
<td>• BMI &lt; 85th percentile or &lt; 95th percentile with no health risk factors</td>
<td>• BMI &gt; 95th percentile or ≥ 85th percentile with health risk factors</td>
<td>• Structured intervention at more frequent intervals (weekly for 8-12 weeks) by team experienced with care of children affected by obesity</td>
<td>• Tertiary care center with designed protocol</td>
</tr>
<tr>
<td>• Basic Healthy Behaviors</td>
<td>• Monthly visits working on behavior change and MI</td>
<td>• Family involvement, supervised activity</td>
<td>• May include meal replacements, weight loss medications</td>
</tr>
<tr>
<td></td>
<td>• Dietician evaluation</td>
<td>• Negative energy balance through diet and exercise</td>
<td>• May include weight loss surgery</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• May include medication management, meal replacements</td>
<td></td>
</tr>
</tbody>
</table>
## Edmonton Obesity Staging System for Pediatrics

<table>
<thead>
<tr>
<th>Stage 0</th>
<th>Stage 1</th>
<th>Stage 2</th>
<th>Stage 3</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Metabolic:</strong> No metabolic abnormalities</td>
<td><strong>Metabolic:</strong> Mild metabolic abnormalities (i.e. IGT, pre-hypertension, mild lipid abnormalities, mild fatty infiltration of liver/elevation in transaminases)</td>
<td><strong>Metabolic:</strong> Moderate metabolic complications requiring pharmacotherapy (i.e. Type 2 Diabetes, Hypertension, lipid abnormalities, PCOS, moderate to severe fatty infiltration of liver)</td>
<td><strong>Metabolic:</strong> Uncontrolled metabolic complications (i.e. T2DM + complications/not meeting glycemic targets), uncontrolled hypertension, FSGS, markedly elevated liver enzymes and/or liver dysfunction, symptomatic gall stones, marked lipid abnormalities)</td>
</tr>
<tr>
<td><strong>Mechanical:</strong> No functional limitations</td>
<td><strong>Mechanical:</strong> Mild bio-mechanical complications (i.e. OSA not requiring PAP therapy, mild MSK pain not interfering with ADL, GERD)</td>
<td><strong>Mechanical:</strong> Moderate bio-mechanical complications (i.e. OSA requiring PAP therapy, GERD, MSK pain limiting activity, moderate limitations in ADLs)</td>
<td><strong>Mechanical:</strong> OSA requiring PAP therapy and suppl. oxygen, limited mobility, shortness of breath sitting/sleeping</td>
</tr>
<tr>
<td><strong>Mental:</strong> No psychopathology</td>
<td><strong>Mental:</strong> Mild psychopathology, ADHD, LD, mild body image pre-occupation, occasional emotional/binge eating, bullying, mild developments delay</td>
<td><strong>Mental:</strong> Moderate mental health issues (i.e. major depression, anxiety, frequent binging, significant body image disturbance, moderate developmental delay)</td>
<td><strong>Mental:</strong> Uncontrolled psychopathology, school refusal, daily binge eating, severe body image disturbance</td>
</tr>
<tr>
<td><strong>Milieu:</strong> No parental, familial, or social environment concerns</td>
<td><strong>Milieu:</strong> Minor problems in relationships, minor limitations in caregivers ability to support child’s needs</td>
<td><strong>Milieu:</strong> Moderate problems in relationships, significant bullying at home or at school, significant limitations in caregivers ability to support child’s needs</td>
<td><strong>Milieu:</strong> Severe problems in relationships, caregivers unable to support child’s needs (may include exposure to family violence), dangerous environment (home, neighborhood or school)</td>
</tr>
</tbody>
</table>

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**Appendix B: Tools**

[obesitymedicine.org](http://obesitymedicine.org)
Resources

- **Food Insecurity**
  - Summer Food Service Program: [http://www.fns.usda.gov/sfsp/summerfood-service-program](http://www.fns.usda.gov/sfsp/summerfood-service-program)

Sleep Disorders, Binge Eating Disorder, Sleep Related Eating Disorder

**Sleep Disorders**

- [http://www.pennstatehershey.org/c/document_library/get_file?uuid=0c0ceca4-5963-4d8c-b297-6189d1a92ae2&groupId=307082](http://www.pennstatehershey.org/c/document_library/get_file?uuid=0c0ceca4-5963-4d8c-b297-6189d1a92ae2&groupId=307082)

**BED Screening Tools**

- [https://psychology-tools.com/binge-eating-scale/](https://psychology-tools.com/binge-eating-scale/)

**Sleep Related Eating Disorder**

- [https://sleepfoundation.org/sleep-disorders-problems](https://sleepfoundation.org/sleep-disorders-problems)
ACE

Centers for Disease Control and Prevention, Violence Prevention Program, ACEs Study
  - http://www.cdc.gov/violenceprevention/acestudy/about.html

Robert Wood Johnson Foundation, The Truth about ACEs

ACEs Connection

Bullying/Weight Stigma and Victimization

  • Bullying
    – Stop Bullying Now
      • www.stopbullyingnow.hrsa.gov/adults/default.aspx
    – Centers for Disease Control
      • www.cdc.gov/ncipc/dvp/electronic_aggression.htm

  • Weight Stigma and Victimization
    – Rudd Center for Food Policy & Obesity: www.uconnruddcenter.org
      • Resources regarding weight stigma, how to incorporate into a practice, media, literature
    – AAP Institute for Healthy Childhood Weight: Change Talk
      • www.go.kognito.com/changetalk
    – Obesity Action Coalition
      • www.obesityaction.org
References:

References:


19. http://www.cdc.gov/mmwr/preview/mmwrhtml/mm5909a1.htm?s_cid=mm5909a1_w


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References:


References:

References:

90. Casperson SL, Johnson L, Roemmich JN. The relative reinforcing value of sweet versus savory snack foods after consumption of sugar- or non-nutritive sweetened beverages. Appetite. 2017; 112:143–149.
References:


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145. Pediatric Obesity—Assessment, Treatment, and Prevention: An Endocrine Society Clinical Practice Guideline Dennis M. Styne,1 Silva A. Arslanain,2 Ellen L. Connor,3 lsmaa Sadaf Farooqi,4 M. Hassan Murad,5 Janet H. Silverstein,6 and Jack A. Yanovski7 – should already be cited in algorithm but could not find in references.
References:


References:


189. Papandreou D, Karavelis M, Karabouta Z, Andreou E. Obese children with metabolic syndrome have 3 times higher risk to have nonalcoholic fatty liver disease compared with those without metabolic syndrome. Int J Endocrinol. 2017;2017:2671692.


References:


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Disclosures

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