به نام خدا

تازه های تشخیصی و درمانی چاقی در کودکان

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Body mass index (BMI)

Calculation of body mass index:

English formula for BMI: 703 × weight in pounds ÷ (height in inches)2

Metric formula for BMI: Weight in kilograms ÷ (height in meters)2

Weight categories
between 2 and 20 years of age

●Underweight – BMI <5th percentile for age and sex

●Normal weight – BMI between the 5th and <85th percentile for age and sex

•Overweight – BMI between the 85th and 95th percentile for age and sex

•Obesity – Classified by severity, using the following thresholds:

-Class I: BMI ≥95th percentile for age and sex or BMI ≥30

-Class II: BMI ≥120 percent of the 95th percentile values or BMI ≥35

-Class III: BMI ≥140 percent of the 95th percentile values or BMI ≥40 kg/m2
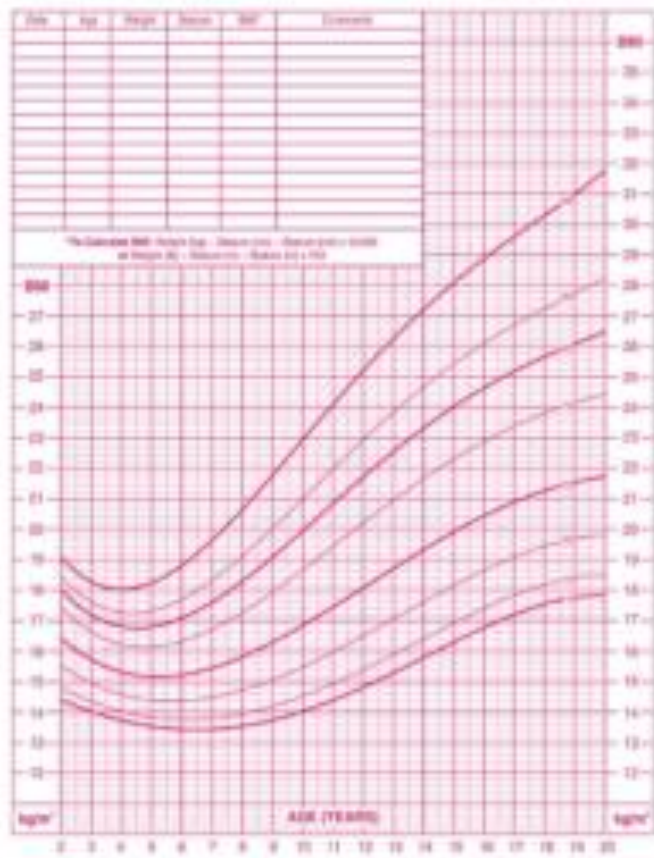
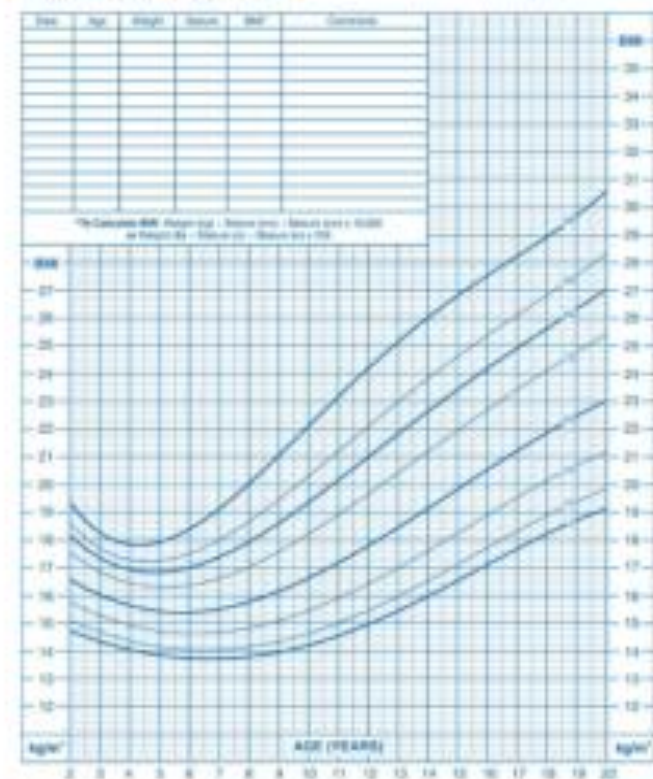A, Body mass index (BMI)-for-age profiles for boys and men. (Developed by the National Center for Health Statistics in collaboration with the National Center for Chronic Disease Prevention and Health Promotion.)

B, BMI-for-age profiles for girls and women.
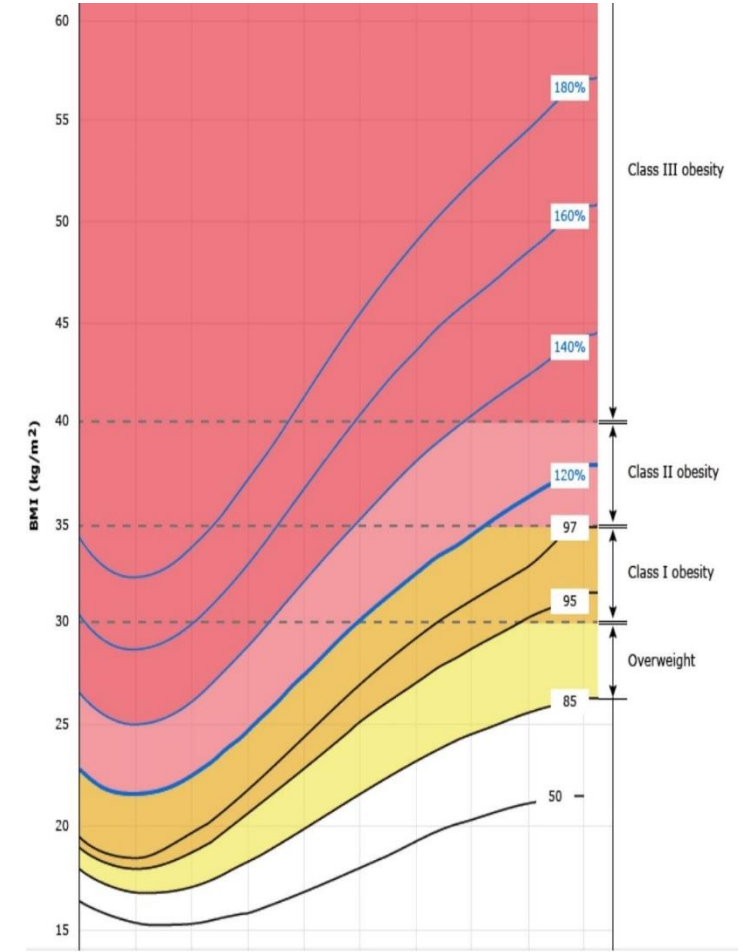
Epidemiology

- Overweight (body mass index [BMI] ≥85th to 95th percentile)

-12.3 percent of preschool-aged children (2 to 5 years)

-15.4 percent of school-aged children (6 to 11 years)

-19.4 percent of adolescents (12 to 19 years)

•Class I obesity (BMI ≥95th percentile to <120 percent of the 95th percentile and <35 kg/m2)

-11.7 percent of preschool-aged children

-12.3 percent of school-aged children

-8.9 percent of adolescents

•Class II or III obesity (BMI that is either ≥120 percent of the 95th percentile or ≥35 kg/m2)

-2 percent of preschool-aged children

-6.4 percent of school-aged children

-10.1 percent of adolescent females and 13.2 percent of adolescent males

The prevalence of childhood overweight and obesity also increased in many other resource-abundant countries worldwide

Etiology of obesity

1) Environmental factors:

- Increasing trends in glycemic index of foods

- sugar-containing beverages

- portion sizes for prepared foods

- fast food

- diminishing family presence at meals

- decreasing structured physical activity

- increasing use of computer , electronic , digital-oriented play activity

- school meal nutrition content

- elements of the built environment (availability of sidewalks and playgrounds)

2) Sleep :

Mounting evidence suggests an association between shortened sleep duration or irregular sleep schedules and obesity

3) Gut microbiome :

probiotics , antibiotics

4) Toxins :

such as the pesticide dichlorodiphenyltrichloroethane (DDT) or bisphenol A

5) Viruses :

A few studies suggest the possibility that obesity can be triggered or exacerbated by exposure to a virus. Adenovirus 36 increases body fat…….

6) Syndromic obesity :

A variety of specific syndromes in which obesity is a primary manifestation have been identified Sach as praderwilli

7) Medications :

- Prednisone and other glucocorticoids
- Thioridazine
- Olanzapine
- Clozapine
- Quetiapine
- Risperidone
- Lithium
- Amitriptyline and other tricyclic antidepressants
- Paroxetine
- Valproate
- Carbamazepine
- Gabapentin
- Cyproheptadine
- Propranolol and other β-blockers

8) Genetic factors :

Monogenic obesity : Single-gene defects in which obesity is the primary manifestation are even more rare

Polygenic factors : There is strong evidence that genetic factors play a permissive role and interact with environmental factors to produce obesity

KSR2 deficiency	Mild hyperphagia and reduced basal metabolic rate, insulin resistance often with acanthosis nigricans, irregular menses, early development of type 2 diabetes mellitus	*KSR2* gene
Leptin or leptin receptor gene deficiency	Early-onset severe obesity, infertility (hypogonadotropic hypogonadism), hyperphagia, infections	Leptin
Melanocortin 4 receptor gene mutation	Early-onset severe obesity, increased linear growth, hyperphagia, hyperinsulinemia Most common known genetic cause of obesity Homozygous worse than heterozygous	*MC4R* mutation
PCSK1 deficiency	Small bowel enteropathy, hypoglycemia, hypothyroidism, ACTH deficiency, diabetes insipidus	*PCSK1* gene
Prader–Willi syndrome	Neonatal hypotonia, slow infant growth, small hands and feet, mental retardation, hypogonadism, hyperphagia leading to severe obesity, paradoxically elevated ghrelin	Partial deletion c chromosome 15 loss of paternally expressed genes
Proopiomelanocortin (POMC) deficiency	Obesity, red hair, adrenal insufficiency due to ACTH deficiency, hyperproinsulinemia, hyperphagia, pale skin, cholestatic jaundice	Loss-of-function mutations of *POMC* gene
Rapid-onset obesity with hypothalamic dysfunction, hypoventilation, and autonomic dysregulation (ROHHAD)	Often confused with congenital central hypoventilation syndrome (CCHS); presentation ≥1.5 yr with weight gain, hyperphagia, hypoventilation, cardiac arrest, central diabetes insipidus, hypothyroidism, GH deficiency, pain insensitivity, hypothermia, precocious puberty, neural crest tumors	Unknown genes May be a paraneoplast disorder
SH2B1 deficiency	Hyperphagia, disproportionate hyperinsulinemia, early speech and language delay that often resolves, behavioral problems including aggression	*SH2B1* gene
SIM1 deficiency	Hyperphagia with autonomic dysfunction (characterized by low systolic blood pressure), speech and language delay, neurobehavioral abnormalities including autistic-type behaviors	*SIM1* gene
TUB deficiency	Retinal dystrophy, deafness	*TUB* gene
Turner syndrome	Ovarian dysgenesis, lymphedema, web neck, short stature, cognitive impairment	XO chromosome

9) Endocrine disorders :

- Cortisol excess (the use of corticosteroid medication, Cushing syndrome)
- Hypothyroidism
- Growth hormone deficiency
- Pseudohypoparathyroidism type 1a (Albright hereditary osteodystrophy)

10) Hypothalamic obesity :

A rare cause of hypothalamic obesity is a syndrome consisting of rapid-onset obesity, hypothalamic dysfunction, hypoventilation, and autonomic dysregulation (ROHHAD), which may be associated with neuroendocrine tumors, leading to the expanded acronym, ROHHADNET.

11) Metabolic programming :

There is increasing evidence that environmental and nutritional influences during critical periods in development can have permanent effects on an individual's predisposition to obesity and metabolic disease. The precise mediators and mechanisms for these effects have not been established but are the subject of ongoing investigations

12) Gestation :

Maternal body weight and nutritional factors during gestation are probably an important determinant of metabolic programming

Obesity-Associated Comorbidities

DISEASE	POSSIBLE SYMPTOMS	LABORATORY CRITERIA
CARDIOVASCULAR		
Dyslipidemia	HDL <40, LDL >130, total cholesterol >200 mg/dL	Fasting total cholesterol, HDL, LDL, triglycerides
Hypertension	SBP >95% for sex, age, height	Serial testing, urinalysis, electrolytes, blood urea nitrogen, creatinine
ENDOCRINE		
Type 2 diabetes mellitus	Acanthosis nigrans, polyuria, polydipsia	Fasting blood glucose >110, hemoglobin A_{1c}, insulin level, C-peptide, oral glucose tolerance test
Metabolic syndrome	Central adiposity, insulin resistance, dyslipidemia, hypertension, glucose intolerance	Fasting glucose, LDL and HDL cholesterol
Polycystic ovary syndrome	Irregular menses, hirsutism, acne, insulin resistance, hyperandrogenemia	Pelvic ultrasound, free testosterone, LH, FSH
GASTROINTESTINAL		
Gallbladder disease	Abdominal pain, vomiting, jaundice	Ultrasound
Nonalcoholic fatty liver disease (NAFLD)	Hepatomegaly, abdominal pain, dependent edema, ↑ transaminases Can progress to fibrosis, cirrhosis	AST, ALT, ultrasound, CT, or MRI
NEUROLOGIC		
Pseudotumor cerebri	Headaches, vision changes, papilledema	Cerebrospinal fluid opening pressure, CT, MRI
Migraines	Hemicrania, headaches	None
ORTHOPEDIC		
Blount disease (tibia vara)	Severe bowing of tibia, knee pain, limp	Knee radiographs
Musculoskeletal problems	Back pain, joint pain, frequent strains or sprains, limp, hip pain, groin pain, leg bowing	Radiographs
Slipped capital femoral epiphysis	Hip pain, knee pain, limp, decreased mobility of hip	Hip radiographs
PSYCHOLOGIC		
Behavioral complications	Anxiety, depression, low self-esteem, disordered eating, signs of depression, worsening school performance, social isolation, problems with bullying or being bullied	Child Behavior Checklist, Children's Depression Inventory, Peds QL, Eating Disorder Inventory 2, subjective ratings of stress and depression, Behavior Assessment System for Children, Pediatric Symptom Checklist
PULMONARY		
Asthma	Shortness of breath, wheezing, coughing, exercise intolerance	Pulmonary function tests, peak flow
Obstructive sleep apnea	Snoring, apnea, restless sleep, behavioral problems	Polysomnography, hypoxia, electrolytes (respiratory acidosis with metabolic alkalosis)

Laboratory testing

Fasting plasma glucose

HbA1C

Triglycerides

Total Cholesterol

low-density lipoprotein and high-density lipoprotein cholesterol

liver function tests

Insulin level

TSH , FT4

Other laboratory testing should be guiled by history or physical examination findings

Management

Estimated Energy Requirement (EER)

Infants and young children

Estimated Energy Requirement (kcal/day) = Total Energy Expenditure + Energy Deposition

0-3 months	EER = (89 × weight [kg] −100) + 175
4-6 months	EER = (89 × weight [kg] −100) + 56
7-12 months	EER = (89 × weight [kg] −100) + 22
13-35 months	EER = (89 × weight [kg] −100) + 20

Estimated Energy Requirement (EER)

Children and Adolescents 3-18 years

Estimated Energy Requirement (kcal/day) = Total Energy Expenditure + Energy Deposition

Boys 3-8 years	EER = 88.5 − (61.9 × age [y]) + PA × { (26.7 × weight [kg]) + (903 × height [m]) } + 20
9-18 years	EER = 88.5 − (61.9 × age [y]) + PA × { (26.7 × weight [kg]) + (903 × height [m]) } + 25
Girls 3-8 years	EER = 135.3 − (30.8 × age [y]) + PA × { (10.0 × weight [kg]) + (934 × height [m]) } + 20
9-18 years	EER = 135.3 − (30.8 × age [y]) + PA × { (10.0 × weight [kg]) + (934 × height [m]) } + 25

Acceptable Macronutrient Distribution Ranges (AMDR)

Males & Females [34]	Total Carbohydrate Percent of Energy	Total Protein Percent of Energy	Total Fat Percent of Energy
1-3 years	45 – 65 %	5 – 20 %	30 – 40 %
4-18 years	45 – 65 %	10 – 30 %	25 – 35 %
19 years and over	45 – 65 %	10 – 35 %	20 – 35 %

Age (years)	BMI 85-95th No risks	BMI 85-95th With risks	More than 95th percentile
2-5	Maintain weight velocity	Decrease weight velocity or weight maintenance	Weight maintenance or gradual weight loss up to 0.5 kg per month in case of severe obesity
6-11	Maintain weight velocity	Decrease weight velocity or weight maintenance	Weight loss not exceed an average of 0.5 kg per week
12-18	Maintain weight velocity, and after linear growth is complete, we can decrease weight around 0.5 kg per week	Maintain weight velocity, and after linear growth is complete, we can decrease weight around 0.5 kg per week	Weight loss not exceed an average of 1 kg per week

Stage 1

- **Goal:** Prevention of weight gain OR weight maintenance with growth that results in a decreasing BMI with age

➢Dietary: limit faulty behaviors

➢Allow child to self-regulate meals (avoid restrictions)

➢Increase physical activity

➢Follow-up:

- Weekly to assess weight

- If no change in 3 months, move to stage 2 based on client/family readiness to change.

Junk Food v's Healthy Food

Fats, Oils and Sweets

Milk

Meat, Meat Substitutes and Other Proteins

Vegetables

Fruits

Breads, Grains and Other Starches

THE NEW Physical Activity Pyramid For Kids

Flexibility Exercises

Muscle Fitness Exercises

Vigorous Sports and Recreation

Vigorous Aerobics

Moderate Physical Activity

Avoid Inactivity!
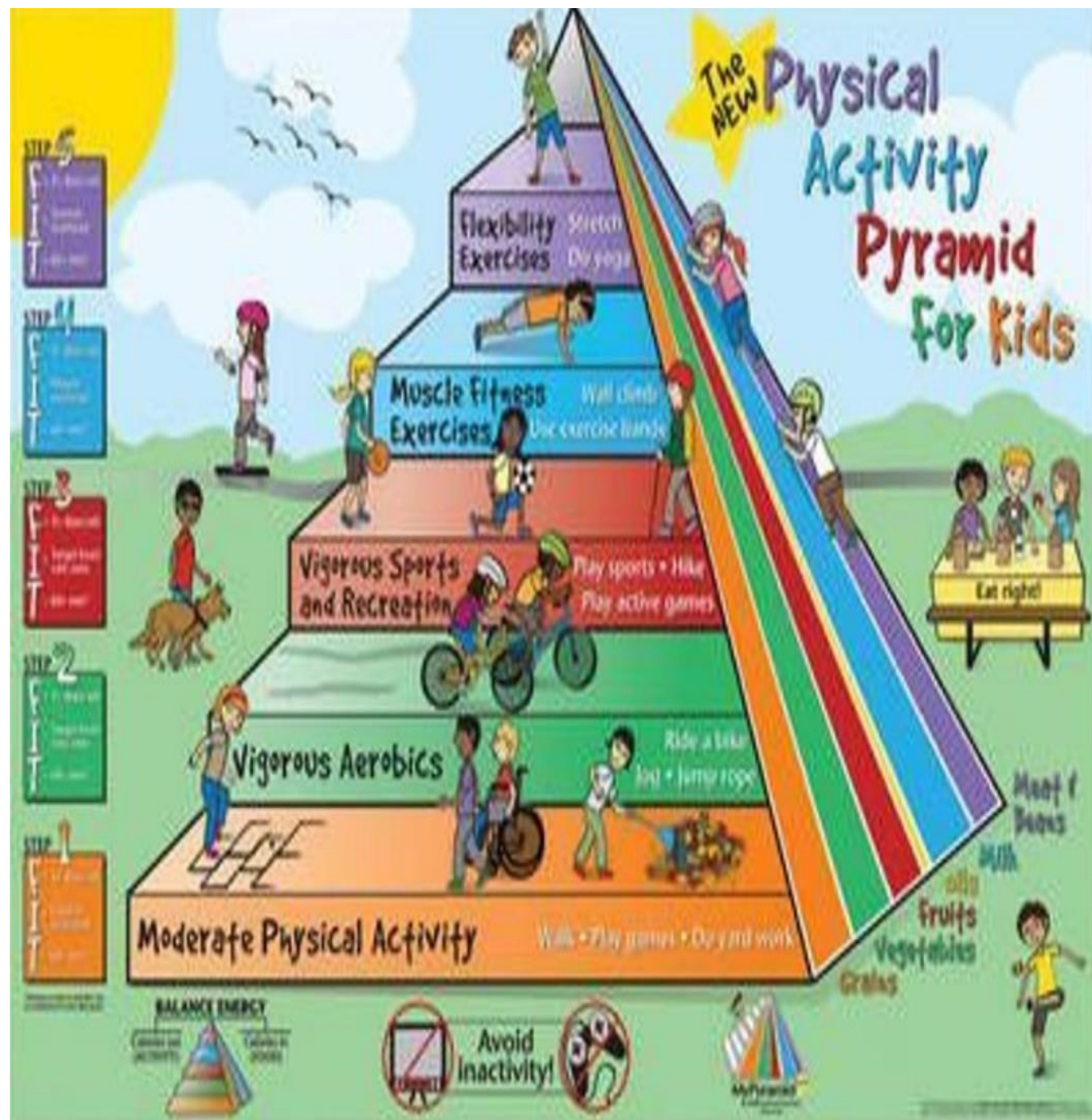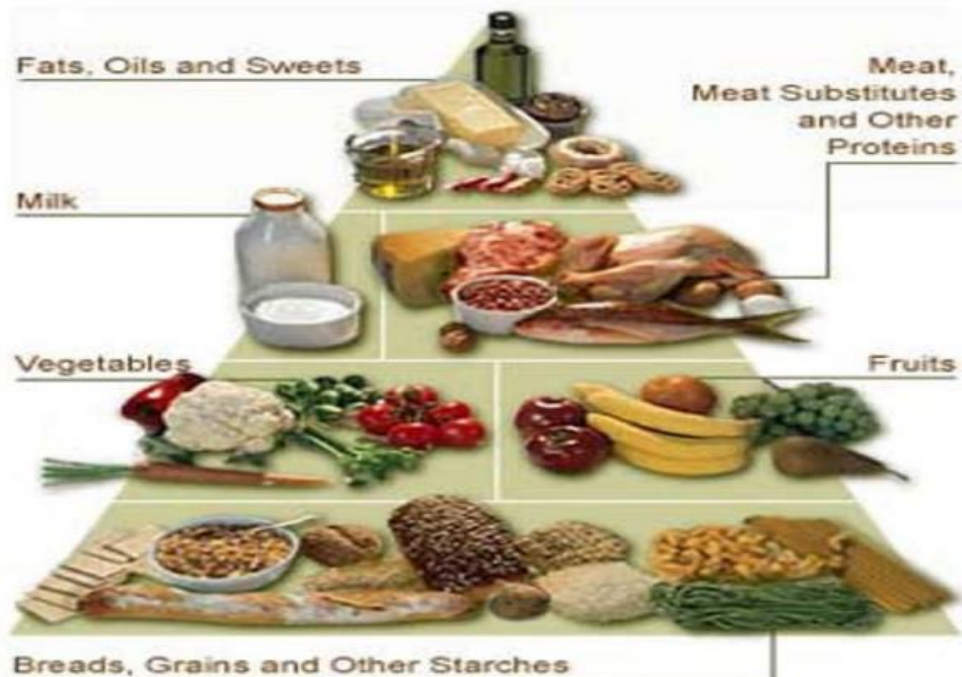
Stage 2

- **Goal: Weight loss that results in decreasing BMI as weight loss should not exceed 1 lb per month (age 2-11) or an average of 1 lbs per week (older youth)**

➤All components of stage 1

➤Individualized meal plan and snacks

➤keep records of the child meal plan and activity (food diary)

➤Supervised active play for at least one hour a day ◦ Limit sedentary activities to no more than 1 hour

➤**Follow-up:**

- As Stage 1 +If no improvement in 3 months, move to stage3

Stage 3

- **Goal:** gradual weight loss until BMI < 85th percentile

➤ Dietary and physical activity recommendation as stage 1 & 2

➤ Multidisciplinary team approach: structured behavior modification (food and activity monitoring, short term meal and activity goals; family involvement)

➤ **Follow-up:**

- As Stage 1 +

- Move to stage 4 if BMI remains > 95th percentile with significant comorbidities and HAVE NOT been successful with stage 1-3

Stage 4

- **Referral to pediatric weight management center :**
  - **Meal replacements**
  - **Severe caloric restriction**

- **Drug therapy**

- **Surgery (bariatric):**

- **Age 16 and older and BMI > 40 or BMI > 35 with obesity related comorbidities**

Medications for Weight Management With Mechanism of Action, Availability, and Dosing

MEDICATION	MECHANISM OF ACTION	AVAILABLE FOR CHRONIC USE		MEAN PERCENTAGE WEIGHT LOSS		ADVANTAGES	DISADV
		USA	European Union	Placebo	Drug		
Phentermine, 15-30 mg PO	Sympathomimetic	For short-term use	No	Not stated in label	Not stated in label	Inexpensive	Side effe no long-t
Orlistat, 120 mg PO tid before meals	Pancreatic lipase inhibitor	Yes	Yes	−2.6% †	−6.1% †	Not absorbed; long-term data*	Modest v side effe
Lorcaserin, 10 mg PO bid	5-HT$_{2c}$ serotonin agonist with little affinity for other serotonergic receptors	Yes	No	−2.5%	−5.8%	Mild side effects; long-term data*	Expensiv weight lc
Phentermine/topiramate ER, 7.5 mg/46 mg or	Sympathomimetic anticonvulsant	Yes	No	−1.2%	−7.8% (mid-	Robust weight loss; long-term	Expensiv
15 mg/92 mg PO indicated as rescue (requires titration)	(GABA receptor modulation, carbonic anhydrase inhibition, glutamate antagonism)				dose) −9.8% (full dose)	data*	
Naltrexone SR/bupropion SR, 32 mg/360 mg PO (requires titration)	Opioid receptor antagonist; dopamine and noradrenaline reuptake inhibitor	Yes	Yes	−1.3%	−5.4%	Reduces food craving; long-term data*	Moderate expensiv effect prc
Liraglutide, 3.0 mg injection (requires titration)	GLP-1 receptor agonist	Yes	Yes	−3%	−7.4% (full dose)	Side effect profile; long-term data*	Expensiv injectablc

Metformin

Bariatric Surgery

suggest BS in adolescents with:

BMI>35 kg/m2 but <40 kg/m2 with major comorbidity,(class 2) such as :

type 2 diabetes

moderate to extreme sleep apnea

pseudotumor cerebri

debilitating orthopedic problems

NASH with advanced fibrosis

or

BMI >40 kg/m2(class 3)

Clinically significant reduction in BMI among adolescents with Class 2 and Class 3 severe obesities is rarely achieved with lifestyle therapy alone.

The most common types of MBS in adolescents include Roux-en-Y gastric bypass (RYGB) and vertical sleeve gastrectomy (VSG).

VSG now considered the gold standard for bariatric surgery .