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Childhood Obesity

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Definition

- Excess of fat
- Usually assessed by the relationship between weight and height (an estimate of body fat, sufficiently accurate for clinical purposes)
- Body mass index (BMI): widely accepted standard measure of overweight and obesity for children two years of age and older.
- BMI provides a guideline for weight in relation to height and is equal to the body weight (in kilograms) divided by the height (in meters) squared



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Definition

* Other indices:

- Weight-for-height (which is particularly useful for the child younger than two years)
- Measures of regional fat distribution (eg, waist circumference and waist-to-hip ratio)
- The growth standards developed by the World Health Organization (WHO)

Definition

- The norms for BMI in children vary with age and sex
- In 2000, the National Center for Health Statistics (NCHS) and the Centers for Disease Control and Prevention (CDC) published BMI reference standards for children between the ages of 2 and 20 years
- As children approach adulthood, the thresholds for defining overweight and obesity (85th and 95th percentiles for BMI) are approximately 25 and 30 kg/m2, respectively



Body mass index-for-age percentiles, females 2 to 20 years



Body mass index-forage percentiles, males 2 to 20 years

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

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

Overweight – BMI between >85th and 95th percentile for age and sex.

Obesity –

- Class I BMI ≥95th percentile for age and sex or BMI ≥30 (whichever is lower).
- Class II BMI ≥120 percent of the 95th percentile values or a BMI ≥35 kg/m2 (whichever is lower).
- Class III obesity BMI ≥140 percent of the 95th percentile values or a BMI ≥40 kg/m2.





Prevalence

United States:

- Approximately **one-third** of children and adolescents are either overweight or obese.
- The population is distributed into higher weight categories with advancing age.
- The prevalence of overweight and obesity are 15 and 5 percent, respectively, by definition (the BMI percentiles).
- A child's risk of having obesity is two- to three-fold higher if they have one birth parent with obesity, and up to 15-fold if they have two birth parents with obesity.
- Obesity is also more prevalent among populations with lower income, education levels or those living in rural environments.

Prevalence

Other countries

- High in most resource-abundant settings worldwide.
- However, studies using comparable statistics show that rates are particularly high (greater than 30 percent) in most countries in North and South America, Great Britain, Greece, Italy, Malta, Portugal, and Spain.
- Worldwide, overweight and obesity in children have generally increased across a wide range of resource-abundant and resource-limited countries during the past 50 years
- The rate at which childhood obesity is increasing in middle- and low-income countries is 30 percent higher than the rate of increase in high-income countries.





Coronavirus disease 2019 (COVID-19)-related changes

Increases in childhood obesity in several countries.

As an example, in a large health care system in California: the greatest change was among children 5 to 11 years old, among whom the prevalence of obesity rose from 19 to 26 percent.





Persistence into adulthood

Many but not all obese children will become obese adults.

Risk factors for persistence of childhood obesity

- Age (excessive weight gain during the preschool years, a strong predictor)
- BMI during childhood

The risk for adult obesity increases with age and childhood BMI.

- parental obesity
- severity of obesity
- Large for gestational age at birth
- Sex?

Environmental factors

Almost all obesity in children is strongly influenced by: Environmental factors, A sedentary lifestyle A caloric intake that is greater than needs

> Although only part of obesity risk but are important targets for treatment because they are potentially modifiable

- Increasing trends in glycemic index of foods
- **sugar-containing beverages:** (including fruit juice): **an important contributor** sugar-sweetened beverages sugar-sweetened beverages supplied an average of 270 kcal/day (10 to 15 percent of total caloric intake)
- portion sizes for prepared foods
- fast food service
- diminishing family presence at meals
- decreasing structured physical activity
- increasing use of computer-, electronic-, and/or digital-oriented play activity
- school meal nutrition content
- elements of the built environment (eg, availability of sidewalks and playgrounds)

Recreational screen media use

Television

the best-established environmental influence

The amount of time spent watching television or the presence of a television in a child's bedroom

Mechanisms

- Displacement of physical activity
- Depression of metabolic rate
- Adverse effects on diet quality (purported mechanism is that food consumption may not be appetite-driven in this setting, eg, prompted by food commercials)
- Effects of television on sleep

Video games

- The effect is small
- Through increased consumption of sugar-sweetened beverages and irregular bedtimes
- ✤ Weaker for electronic games than for television

Exergames

- the intensity and participant enjoyment of the game
- activities replaced by the gaming
- motivating support systems



Sleep

Shortened sleep duration or irregular sleep increased food intake, weight gain, and decreased leptin levels

- An association with insulin resistance and cardiometabolic risk factors, independent of its association with obesity.
- Obesity and sleep-disordered breathing, sleep fragmentation and intermittent hypoxemia were associated with decreased insulin sensitivity, independent of adiposity
- Shorter sleep duration was associated with markers of cardiometabolic risk factors, including abdominal obesity, increased blood pressure, and abnormal lipid profile.

Mechanism:

- Alterations in serum leptin and ghrelin levels (both in the regulation of appetite)
- Sleep deprivation is also associated with increased neural reward processing, which may lead to increased food intake in susceptible individuals.
- Short sleep duration, reduced physical activity levels and/or provide a longer opportunity to ingest food.

Gut microbiome

 Association between obesity and exposure to antibiotics (or maternal infection) during gestation or infancy

Viruses

- Obesity can be triggered or exacerbated by exposure to a virus
- Adenovirus 36 increases body fat in several animal models

Toxins

- Environmental endocrine-disrupting chemicals, such as the pesticide dichlorodiphenyltrichloroethane (DDT) or bisphenol A (BPA)
- An association between urinary BPA concentrations and obesity or obesityrelated diseases, including diabetes and cardiovascular disease

Medications

- Psychoactive drugs (particularly olanzapine and risperidone)
- ✤ Antiseizure medication
- Glucocorticoids

Genetic Factors

Polygenic factors

- Strong evidence for interaction of genetic factors with environmental factors
- Most of the genetic polymorphisms responsible have not yet been isolated

Monogenic obesity

- Single-gene defects are rare
- Several of these affect the melanocortin pathway in the central nervous system (MC4R)
- Mutations causing deficiencies in leptin or its receptor are rare

Genetic Factors

Syndromic obesity

- A variety of specific syndromes in which obesity is a primary manifestation (like Prader-willi, Beckwith- Wiedemann)
- Rare causes of obesity(less than one percent)
- A component of several relatively common genetic syndromes, including Down syndrome.
- Typically early-onset obesity and characteristic findings on physical examination (dysmorphic features, short stature, developmental delay, intellectual disability, retinal changes, or deafness)
- Prader-Willi is the most common

Endocrine disorders

- Less than 1 percent of children and adolescents with obesity
- * Associated with overweight or mild obesity rather than severe obesity
- Most have short stature and/or hypogonadism

The primary considerations:

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

Hypothalamic obesity

- Rapidly progressive severe obesity
- Difficult to treat
- Most often arises after surgical treatment for craniopharyngioma and usually is associated with panhypopituitarism.
- Similar patterns may be caused by trauma, tumor, or an inflammatory disease affecting the hypothalamus.

ROHHAD and ROHHADNET

- ✤ A rare cause of hypothalamic obesity
- A syndrome consisting of rapid-onset obesity, hypothalamic dysfunction,
 hypoventilation, and autonomic dysregulation
- ROHHADNET if with neuroendocrine tumors
- May present in infancy or early childhood with central hypoventilation and a variety of abnormalities in the hypothalamic-pituitary axis, with autonomic dysregulation, including episodes of hyperthermia or hypothermia



To identify treatable causes and comorbidities.



Clinical evaluations

- focused history
- **Physical examination**
- Laboratory
- Radiologic studies for selected patients

HISTORY

1. Weight history

Pattern of weight gain (on serial growth measurements (if available) or history)

Gradual onset

Typical for the most common forms of obesity (genetic predisposition combined with excess caloric intake or other environmental contributors).

Abrupt onset of weight gain

- Prompt investigation of a major psychosocial trigger such as a loss or change in the family or new symptoms of anxiety or depression
- Medication-induced weight gain
- Neuroendocrine causes of obesity (eg, Cushing disease, hypothalamic tumor, or, rarely, ROHHADNET syndrome

Severe early-onset

age

- A strong genetic component
- Some forms of syndromic or monogenic obesity have onset before two years of age,
- While others (especially Prader-Willi syndrome) tend to have growth failure during infancy followed by rapid weight gain and development of obesity after two years of

2. Diet

HISTORY

Caregiver(s) involved in feeding

meal planning, shopping, and preparing and being present at meals

Eating patterns

Timing, content, and location of meals and snacks.

The possibility of disordered eating is raised by :

Recurrent episodes of consuming large amounts of food with a sense of loss of control (eating disorder) Inappropriate compensatory behavior to prevent weight gain (self-induced vomiting or other purging, fasting, and/or excessive exercise) associated with binge eating (suspect bulimia nervosa) Children who eat less frequent meals (eg, those who skip meals) are more likely to have obesity than those who eat more frequently

Food frequency

Common sources of excess calories include:

Sugar-containing beverages, including soda, juice, sugar-containing "sports" drinks, and flavored milks Snack foods (chips, sweets)

Food preferences and dislikes

Restaurants and prepared foods

HISTORY

3. Activity 4. Sleep

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- Sleep habits (typical sleep duration, sleep quality, and sleep schedule)
- Symptoms of sleep disorders
- Obesity is associated with an increased risk for **obstructive sleep apnea**
- Any child who snores habitually (eg, ≥3 nights per week), has loud snoring, or has pauses in breathing during sleep should be further evaluated.
- Obstructive sleep apnea also may cause **nocturnal enuresis or daytime symptoms** including inattention, learning problems, and hyperactivity, with or without sleepiness.

Recommended sleep times for children

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Age group	Recommended sleep time		
Infants 4 to 12 months	12 to 16 hours (including naps)		
Toddlers 1 to 2 years	11 to 14 hours (including naps)		
Children 3 to 5 years	10 to 13 hours (including naps)		
Children 6 to 12 years	9 to 12 hours		
Teens 13 to 18 years	8 to 10 hours		

For optimal health, daytime functioning, and development, the above sleep times are recommended on a regular basis. These consensus recommendations were made by the American Academy of Sleep Medicine $\begin{bmatrix} 1 \\ 2 \end{bmatrix}$ and endorsed by the American Academy of Pediatrics $\begin{bmatrix} 2 \end{bmatrix}$.

5. Medical history

- All medications
- Any known comorbidities of obesity

Table 65.2 Obesity-Associat	ed Comorbidities			
DISEASE	POSSIBLE SYMPTOMS	LABORATORY CRITERIA		
CARDIOVASCULAR Dyslipidemia Hypertension	HDL <40, LDL >130, total cholesterol >200 mg/dL SBP >95% for sex, age, height	Fasting total cholesterol, HDL, LDL, triglycerides Serial testing, urinalysis, electrolytes, blood urea nitrogen, creatinine		
ENDOCRINE				
Type 2 diabetes mellitus	Acanthosis nigricans, 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 Nonalcoholic fatty liver disease (NAFLD)	Abdominal pain, vomiting, jaundice Hepatomegaly, abdominal pain, dependent edema, 1 transaminases Can progress to fibrosis, cirrhosis	Ultrasound AST, ALT, ultrasound, CT, or MRI		
NEUROLOGIC				
Pseudotumor cerebri Migraines	Headaches, vision changes, papilledema Hemicrania, headaches	Cerebrospinal fluid opening pressure, CT, MRI None		
ORTHOPEDIC				
Blount disease (tibia vara) Musculoskeletal problems	Severe bowing of tibia, knee pain, limp Back pain, joint pain, frequent strains or sprains,	Knee radiographs 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 Inventor Peds QL, Eating Disorder Inventory 2, subjective rating of stress and depression, Behavior Assessment System for Children, Pediatric Symptom Checklist		
PULMONARY				
Asthma Shortness of breath, wheezing, coughi exercise intolerance		Pulmonary function tests, peak flow		
Obstructive sleep apnea	Snoring, apnea, restless sleep, behavioral problems	Polysomnography, hypoxia, electrolytes (respiratory acidosis with metabolic alkalosis)		

HISTORY

6. Family history

Obesity

- In first-degree relatives (parents and siblings)
- In particular, obesity in one or both parents is an important predictor for whether a child's obesity will persist into adulthood(both genetic and environmental components).

Comorbidities

- Common comorbidities of obesity, such as cardiovascular disease, hypertension, diabetes, liver or gallbladder disease, and respiratory problems (severe asthma or sleep apnea) in first- and second-degree relatives.
- Maternal gestational diabetes is associated with adverse cardiometabolic outcomes in the offspring

Family dynamics

HISTORY

7. Psychosocial history

* Anxiety and depression

Disrupt eating patterns Promote obesity Interfere with weight management interventions.

✤ Events

Possible triggering events such as a loss of a loved one (death or relocation), divorce, or changes in primary caregiver(s).

* School function and social issues

Smoking or vaping
Cigarette smoking increases long-term cardiovascular risk

Social determinants of health

Poverty, food insecurity, access to sources of healthy food and recreation, and transportation

Evaluation for comorbidities and possible causes of the obesity

1. General examination

dysmorphic features

assess the fat distribution:

 A markedly Cushingoid fat distribution (concentrated in the interscapular area, face, neck, and trunk) suggests the possibility of Cushing syndrome
 Abdominal obesity (also called central, visceral, android, or male-type obesity) is associated with certain comorbidities, including metabolic syndrome, polycystic ovary syndrome, and insulin resistance.

2. Blood pressure

Stature

- Hyperinsulinemia: accelerates skeletal maturation, earlier epiphyseal fusion, variable effects on adult
- Endocrine and genetic causes: often short stature. As an example, decreased height velocity is an early sign in Cushing disease.

Examination of the head, eyes, ears, nose, and throat (HEENT)

- Enlarged tonsils suggest increased risk for obstructive sleep apnea.
- Blurred optic disc margins may indicate pseudotumor cerebri, an unexplained but not uncommon association with obesity.
- Nystagmus or visual complaints raise the possibility of a hypothalamicpituitary lesion.
- Clumps of pigment in the peripheral retina may indicate retinitis pigmentosa, which occurs in Bardet-Biedl syndrome.

Skin and hair

- Acanthosis nigricans: may signify insulin resistance with or without type 2 diabetes
- Hirsutism and acne are common features of polycystic ovary syndrome and Cushing syndrome.
- Striae distensae (eg, abdomen, breasts, thighs): usually the result of rapid weight gain, manifestations of Cushing syndrome.
- Dry, coarse, or brittle hair may be present in hypothyroidism.
- Red hair (in individuals with light skin pigmentation), hyperphagia, and early onset of obesity: proopiomelanocortin deficiency, low levels of adrenocorticotropic hormone with associated adrenal insufficiency.



Abdomen

- Abdominal tenderness, particularly in the right upper quadrant: sign of gallbladder disease or nonalcoholic fatty liver disease (NAFLD).
- Hepatomegaly: a clue to NAFLD

Extrimities

- slipped capital femoral epiphysis.
- ✤Genu varum (bow legs) or valgus (knock-knees).
- Nonpitting edema: hypothyroidism
- Postaxial polydactyly (an extra digit next to the fifth digit): Bardet-Biedl syndrome
- small hands and feet: Prader-Willi syndrome
- Pes planus (flat feet) :common in children with obesity and frequently give rise to pain during exercise

Genitourinary

- Endocrine or genetic causes : hypogonadism
- Undescended testicles, small penis, and scrotal hypoplasia : Prader-Willi syndrome.
- Small testes: Prader-Willi or Bardet-Biedl syndrome
- Delayed or absent puberty: hypothalamic-pituitary tumors, Prader-Willi syndrome, Bardet-Biedl syndrome, leptin deficiency, or leptin receptor deficiency
- Precocious puberty occasionally is a presenting symptom of a hypothalamic-pituitary lesion or dysfunction, including in ROHHAD(NET) syndrome

Development

Most of the syndromic causes: cognitive or developmental delay

Laboratory

Routine blood tests

Dyslipidemia

 A fasting lipid profile (total cholesterol, triglycerides, and high- and low-density lipoprotein cholesterols)

Type 2 diabetes mellitus

- Laboratory testing for diabetes is suggested for children with overweight or obesity and additional risk factors
- Screening: fasting plasma glucose or hemoglobin A1c.

Fatty liver disease

- ✤ a serum alanine aminotransferase (ALT) level.
- between 9 and 11 years of age.
- If normal, repeat at least every two to three years
- Sustained elevations of ALT (eg, >2 times the upper limit of normal [ULN] for six months) warrant further evaluation.

Laboratory

Tests for selected patients

Tests for causes of obesity

Hypothyroidism or Cushing syndrome

Routine laboratory screening: **not** recommended Suggestive signs or symptoms, particularly evidence of growth attenuation (decreased height velocity)

Syndromic obesity

Severe early-onset obesity Screening for genetic obesity syndromes may also be appropriate for children with severe early-onset obesity (younger than five years of age)

Laboratory

Tests for selected patients

Tests for comorbidities

Vitamin D deficiency

not routine screening for vitamin D deficiency targeted screening may be appropriate for children with very low dairy/vitamin D intake or those with genu varum/valgus malformations

Hypertension

For children with hypertension, additional laboratory testing may be warranted (eg, serum electrolytes, blood urea nitrogen, creatinine, and urinalysis).

Polycystic ovary syndrome

in females with symptoms suggesting polycystic ovary syndrome, ie, an irregular menstrual pattern, hirsutism or treatment-resistant acne, or acanthosis nigricans with central obesity.

imaging

by findings on the history and physical examination

Plain radiographs of the lower extremities:

Slipped capital femoral epiphysis (hip or knee pain, limited range of motion, abnormal gait) or Blount disease (bowed tibia).

Abdominal ultrasonography:

- In children with findings consistent with gallstones (eg, abdominal pain, abnormal transaminases).
- Abdominal ultrasonography is **not recommended** as a routine screening test for NAFLD in children with obesity
- But with persistently elevated serum aminotransferases, ultrasound or other radiographic evaluation may be indicated as part of a full evaluation for suspected NAFLD.





Management

Referrals

- Severe obesity (body mass index [BMI] ≥120 percent of the 95th percentile or BMI ≥35 kg/m2, whichever is lower), especially if any comorbidities are present or if BMI≥40 kg/m2.
- Refractory obesity (progressive increase in BMI percentiles despite structured interventions in the primary care setting).
- Severe obesity in a child younger than two years. These children warrant special evaluation for the underlying cause of the obesity.

intensive dietary and behavioral therapy and may offer pharmacologic and/or surgical therapy.

Intervention

- * Target behaviors
- Self-monitoring
- ✤ Goal setting
- Stimulus control
- Promotion of self-efficacy and self-management skills

Because obesity is multifactorial, not all children and adolescents will respond to the same approach

Intervention

Table 65.5	Recommended Caloric Intake Designated by Age and Gender						
LIFE-STAGE G	ROUP	AGE (YR)	RELATIVELY SEDENTARY LEVEL OF ACTIVITY (KCAL)	MODERATE LEVEL OF ACTIVITY (KCAL)	ACTIVE (KCAL)		
Child		2-3	1,000	1,000-1,400	1,000-1,400		
Female		4-8	1,200	1,400-1,600	1,400-1,800		
		9-13	1,600	1,600-2,000	1,800-2,200		
		14-18	1,800	2,000	2,400		
Male		4-8	1,400	1,400-1,600	1,600-2,000		
		9-13	1,800	1,800-2,200	2,000-2,600		
		14-18	2,200	2,400-2,800	2,800-3,200		

Adapted from U.S. Department of Agriculture. Dietary guidelines for Americans, 2005. http://www.health.gov/DIETARYGUIDELINES/dga2005/document/html/chapter2.htm.

Management

Pharmacotherapy

Various classes of drugs:

Decrease energy intake Act centrally as anorexiants Affect the availability of nutrients through intestinal or renal tubular reabsorption Affect metabolism.

Orlistat :The only U.S. Food and Drug Administration (FDA)–approved medication in children <16 yr old Decreases absorption of fat, resulting in modest weight loss. Complications include flatulence, oily stools, and spotting.

		AVAILABLE FOR CHRONIC USE		MEAN PERCENTAGE WEIGHT LOSS			
MEDICATION	ACTION	USA	EU	PLACEBO	DRUG	ADVANTAGES	DISADVANTAGES
Phentermine, 15- 30 mg PO	Sympathomimetic	For short- term use	No	Not stated in label	Not stated in label	Inexpensive	Side effect profile; no long-term data*
Orlistat, 120mg PO tid before meals	Pancreatic lipase inhibitor	Yes	Yes	-2.6%†	-6.1%†	Not absorbed; long-term data*	Modest weight loss; side effect profile
Lorcaserin, 10mg 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*	Expensive; modest weight loss
Phentermine/ topiramate ER, 7.5mg/46mg or 15mg/92mg PO indicated as rescue (requires titration)	Sympathomimetic anticonvulsant (GABA receptor modulation, carbonic anhydrase inhibition, glutamate antagonism)	Yes	No	-1.2%	–7.8% (mid- dose) –9.8% (full dose)	Robust weight loss; long-term data*	Expensive; teratogen
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*	Moderately expensive; side effect profile
Liraglutide, 3.0mg injection (requires titration)	GLP-1 receptor agonist	Yes	Yes	-3%	–7.4% (full dose)	Side effect profile; long-term data*	Expensive; injectable
Semaglutide, 2.4 mg once weekly injection	GLP-1 receptor agonist	Yes	Yes	-1%	-10 to -15%	As above	As above

Information is from US product labels, except where noted. The data supporting these tables are derived from the prescribing information labeling approved by the US Food and

Management

Weight loss surgery

For adolescents with severe obesity, and especially those with major comorbidities (type 2 diabetes, severe sleep apnea, and/or steatohepatitis),

Specialty referral for comorbidities

Mental health specialists

Indications for referral include:

Clinically significant depression or anxiety. Weight loss therapy may be ineffective without concurrent psychological care. Suspected eating disorder

Table 65.8 Proposed Suggestions for Preventing Obesity

PREGNANCY

Normalize body mass index (BMI) before pregnancy. Do not smoke.

Maintain moderate exercise as tolerated.

In women with gestational diabetes, provide meticulous glucose control.

Monitor gestational weight gain within Institute of Medicine (IOM) recommendations.

POSTPARTUM AND INFANCY

Breastfeeding: exclusive for 4-6 mo; continue with other foods for 12 mo.

Postpone introduction of baby foods to 4-6 mo and juices to 12 mo.

FAMILIES

Eat meals as a family in a fixed place and time.

Do not skip meals, especially breakfast.

Do not allow television during meals.

Use small plates and keep serving dishes away from the table.

Avoid unnecessary sweet or fatty foods and sugar-sweetened drinks.

Remove televisions from children's bedrooms; restrict times for TV viewing and video games.

Do not use food as a reward.

SCHOOLS

Eliminate candy and cookie sales as fundraisers.

Review the contents of vending machines and replace with healthier choices; eliminate sodas.

Avoid financial support for sports teams from beverage and food industries.

Install water fountains and hydration stations.

Educate teachers, especially physical education and science faculty, about basic nutrition and the benefits of physical activity (PA).

Educate children from preschool through high school on appropriate diet and lifestyle.

Mandate minimum standards for physical education, including 60 min of strenuous exercise 5 times weekly.

Encourage "the walking school bus": groups of children walking to school with adult supervision.

COMMUNITIES

Increase family-friendly exercise and safe play facilities for children of all ages.

Develop more mixed residential-commercial developments for walkable and bicyclable communities.

Discourage the use of elevators and moving walkways.

Provide information on how to shop and prepare healthier versions of culture-specific foods.

HEALTHCARE PROVIDERS

Explain the biologic and genetic contributions to obesity.

Give age-appropriate expectations for body weight in children.

Work toward classifying obesity as a disease to promote recognition, reimbursement for care, and willingness and ability to provide treatment.

INDUSTRY

Mandate age-appropriate nutrition labeling for products aimed at children (e.g., "red light/green light" foods, with portion sizes).

Encourage marketing of interactive video games in which children must exercise to play.

Use celebrity advertising directed at children for healthful foods to promote breakfast and regular meals.

Reduce portion size (drinks and meals).

GOVERNMENT AND REGULATORY AGENCIES

Classify childhood obesity as a legitimate disease.

- Find novel ways to fund healthy lifestyle programs (e.g., with revenues from food and drink taxes).
- Subsidize government-sponsored programs to promote the consumption of fresh fruits and vegetables.

Provide financial incentives to the industry to develop more healthful products and to educate the consumer on product content.

Provide financial incentives to schools that initiate innovative PA and nutrition programs.

Allow tax deductions for the cost of weight loss and exercise programs. Provide urban planners with funding to establish bicycle, jogging, and walking paths.

Ban advertising of fast foods, non-nutritious foods, and sugarsweetened beverages directed at preschool children, and restrict advertising to school-aged children.

Ban toys as gifts to children for purchasing fast foods.

Adapted from Speiser PW, Rudolf MCJ, Anhalt H, et al. Consensus statement: Childhood obesity. J Clin Endocrinol Metab. 2005;90:1871-1887.

