Chapter 47

Overweight and Obesity

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Obesity is an important pediatric public health problem associated with risk of complications in childhood and increased morbidity and mortality throughout adult life.

EPIDEMIOLOGY

Obesity is a global public health problem, sparing only dramatically poor regions with chronic food scarcity such as sub-Saharan Africa and Haiti. In 2008, according to the World Health Organization, more than 1.4 billion persons ≥20 yr old were overweight or obese.

In the United States, 36% of adults are obese, and an additional 35% of adults are overweight. In children, the prevalence of obesity increased 300% over approximately 40 yr. The National Health and Nutrition Examination Survey, 2009-2010, found 32% of children, 2-19 yr old to be overweight or obese, and 17% in the obese range. Children's risk varies significantly by race/ethnicity. In 2009-2010, 24% of non-Hispanic Black, 21% of Hispanic, and >20% of American Indian/Alaskan Native children and adolescents were obese compared to 14% of white children. Across all racial groups, higher maternal education confers protection against childhood obesity.

Parental obesity correlates with a higher risk for obesity in their children. Prenatal factors including high preconceptual weight, gestational weight gain, high birth weight, and maternal smoking are associated with increased risk for later obesity. Paradoxically, intrauterine growth restriction with early infant catch-up growth is associated with the development of central adiposity and adult-onset cardiovascular risk. Breastfeeding is only modestly protective for obesity. Infants with high levels of negative reactivity (temperament) are at risk for obesity. Better self-regulation is protective.

BODY MASS INDEX

Obesity or increased adiposity is defined using the body mass index (BMI), which is an excellent proxy for more direct measurement of body fat. BMI = weight in kg/(height in meters)^2. Adults with a BMI ≥30 meet the criterion for obesity, and those with a BMI 25-30 fall in the overweight range. During childhood, levels of body fat change beginning with high adiposity during infancy. Body fat levels decrease for approximately 5.5 yr until the period called *adiposity rebound*, when body fat is typically at the lowest level. Adiposity then increases until early adulthood (Fig. 47-1). Consequently, obesity and overweight are defined using BMI percentiles; children >2 yr old with a BMI ≥95th percentile meet the criterion for obesity, and those with a BMI between the 85th and 95th percentiles fall in the overweight range.

ETIOLOGY

Humans have the capacity to store energy in adipose tissue, allowing improved survival in times of famine. Furthermore, humans innately prefer sweet and salty foods and reject bitter flavors. Many vegetables are bitter. These preferences probably reflect evolutionary adaptations to avoid consuming toxic plants. Nonetheless, repeated exposure to healthy foods promotes their acceptance and liking, especially in early life. Simplistically, obesity results from an imbalance of caloric intake and energy expenditure. Even incremental but sustained caloric excess results in excess adiposity. Individual adiposity is the result of a complex interplay among genetically determined body habitus, appetite, nutritional intake, physical activity, and energy expenditure. Environmental factors determine levels of available food, preferences for types of foods, levels of physical activity, and preferences for types of activities.

Environmental Changes

Over the last 4 decades, the food environment has changed dramatically. Changes in the food industry relate in part to social changes, as extended families have become more dispersed. Fewer families routinely prepare meals. Foods are increasingly prepared by a food industry, with high levels of calories, simple carbohydrates, and fat. The price of many foods has declined relative to the family budget. These changes, in combination with marketing pressure, have resulted in larger portion sizes and increased snacking between meals. The increased consumption of high-carbohydrate beverages, including sodas, sport drinks, fruit punch, and juice, adds to these factors.

One-third of U.S. children consume fast food daily. A typical fast food meal can contain 2000 kcal and 84 g of fat. Many children consume 4 servings of high-carbohydrate beverages per day, resulting in an additional 560 kcal of low nutritional value. Sweetened beverages have been linked to increased risk for obesity because children who drink high amounts of sugar do not consume less food. The dramatic increase in the use of high-fructose corn syrup to sweeten beverages and prepared foods is another important environmental change, leading to availability of inexpensive calories.

Since World War II, levels of physical activity in children and adults have declined. Changes in the built environment have resulted in more reliance on cars and decreased walking. Work is increasingly sedentary, and many sectors of society do not engage in physical activity during leisure time. For children, budgetary constraints and pressure for academic performance have led to less time devoted to physical education in schools. Perception of poor neighborhood safety is another factor that can lead to lower levels of physical activity when children are required to stay indoors. The advent of television, computers, and video games has resulted in opportunities for sedentary activities that do not burn calories.

Changes in another health behavior, sleep, might also contribute. Over the last 4 decades, children and adults have decreased the amount of time spent sleeping. Reasons for these changes may relate to increased time at work, increased time watching television, and a generally faster pace of life. Chronic partial sleep loss can increase risk for weight gain and obesity, with the impact possibly greater in children than in adults. In studies of young, healthy, lean men, short sleep duration was associated with decreased leptin levels and increased ghrelin levels, along with increased hunger and appetite. Sleep debt also results in decreased glucose tolerance and insulin sensitivity related to alterations in glucocorticoids and sympathetic activity. Some effects of sleep debt might relate to orexins, peptides synthesized in the lateral hypothalamus that can increase feeding, arousal, sympathetic activity, and/or neuropeptide Y activity.

Genetics

Genetic determinants also have a role in individual susceptibility to obesity (Table 47-1). Findings from genome-wide association studies explain a very small portion of interindividual variability in obesity. One important example, the *FTO* gene at 16q12, is associated with adiposity in childhood, probably explained by increased energy intake (Table 47-1). Monogenic forms of obesity have also been identified, including MC4R deficiency, associated with early-onset obesity and food-seeking behavior. In addition, there are genetic conditions associated with obesity, such as Prader-Willi syndrome, which results from absence of paternally expressed imprinted genes in the 15q11.2–q13 region. Prader-Willi syndrome is characterized by insatiable appetite and food seeking. Epigenetic environmental modification of genes may have a role in the development of obesity, especially during fetal and early life.

Endocrine and Neural Physiology

Monitoring of "stored fuels" and short-term control of food intake (appetite and satiety) occurs through neuroendocrine feedback loops linking adipose tissue, the gastrointestinal tract, and the central

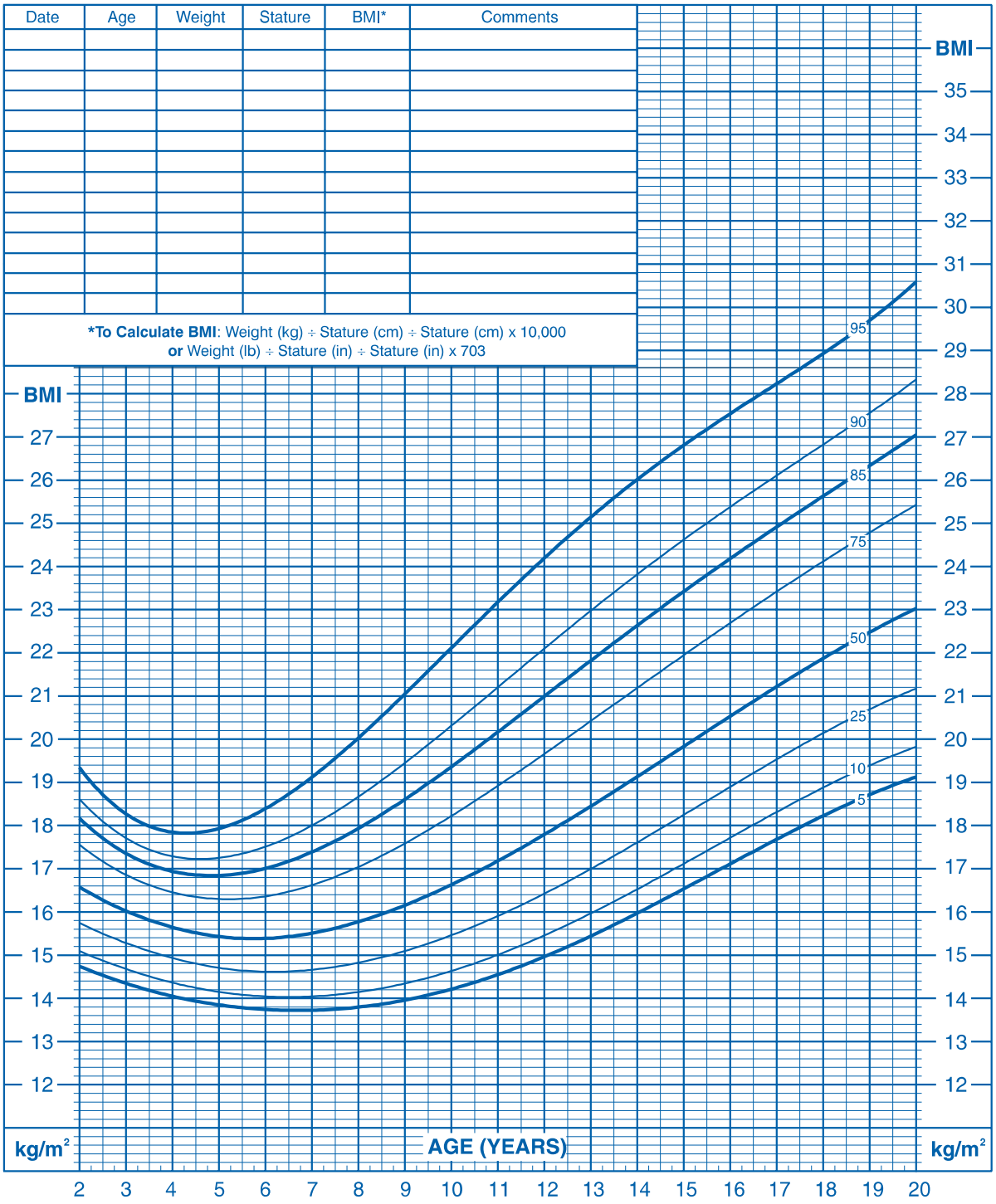
2 to 20 years: Boys
Body mass index-for-age percentiles

NAME _____

RECORD # _____

Date	Age	Weight	Stature	BMI*	Comments

***To Calculate BMI**: Weight (kg) ÷ Stature (cm) ÷ Stature (cm) x 10,000
or Weight (lb) ÷ Stature (in) ÷ Stature (in) x 703



Published May 30, 2000 (modified 10/16/00).

SOURCE: Developed by the National Center for Health Statistics in collaboration with the National Center for Chronic Disease Prevention and Health Promotion (2000).
http://www.cdc.gov/growthcharts

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A

Figure 47-1 Body mass index (BMI)-for-age profiles for boys and men *(A)* and girls and women *(B)*. Developed by the National Center for Health Statistics in collaboration with the National Center for Chronic Disease Prevention and Health Promotion (2000). See www.cdc.gov/growthcharts

2 to 20 years: Girls
Body mass index-for-age percentiles

NAME _____

RECORD # _____

Date	Age	Weight	Stature	BMI*	Comments

***To Calculate BMI**: Weight (kg) ÷ Stature (cm) ÷ Stature (cm) x 10,000
or Weight (lb) ÷ Stature (in) ÷ Stature (in) x 703

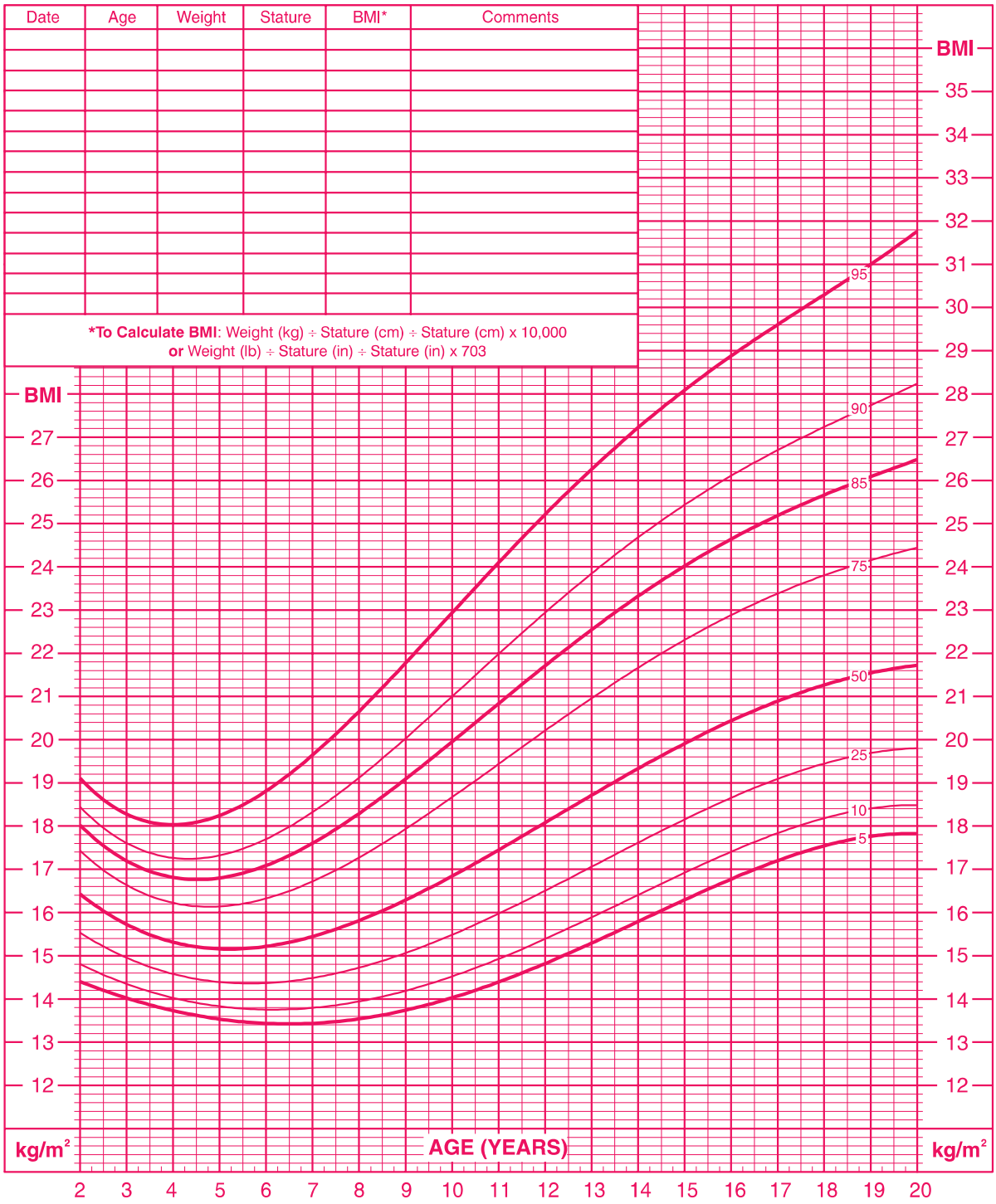

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Figure 47-1, cont'd

Table 47-1 Endocrine and Genetic Causes of Obesity

DISEASE	SYMPTOMS	LABORATORY
ENDOCRINE		
Cushing syndrome	Central obesity, hirsutism, moon face, hypertension	Dexamethasone suppression test
GH deficiency	Short stature, slow linear growth	Evoked GH response, IGF-1
Hyperinsulinism	Nesidioblastosis, pancreatic adenoma, hypoglycemia, Mauriac syndrome	Insulin level
Hypothyroidism	Short stature, weight gain, fatigue, constipation, cold intolerance, myxedema	TSH, FT_4
Pseudohypoparathyroidism	Short metacarpals, subcutaneous calcifications, dysmorphic facies, mental retardation, short stature, hypocalcemia, hyperphosphatemia	Urine cAMP after synthetic PTH infusion
GENETIC		
Alstrom syndrome	Cognitive impairment, retinitis pigmentosa, diabetes mellitus, hearing loss, hypogonadism, retinal degeneration	*ALMS1* gene
Bardet-Biedl syndrome	Retinitis pigmentosa, renal abnormalities, polydactyly, hypogonadism	*BBS1* gene
Biemond syndrome	Cognitive impairment, iris coloboma, hypogonadism, polydactyly	
Carpenter syndrome	Polydactyly, syndactyly, cranial synostosis, mental retardation	Mutations in the *RAB23* gene, located on chromosome 6 in humans
Cohen syndrome	Mid-childhood-onset obesity, short stature, prominent maxillary incisors, hypotonia, mental retardation, microcephaly, decreased visual activity	Mutations in the *VPS13B* gene (often called the *COH1* gene) at locus 8q22
Deletion 9q34	Early-onset obesity, mental retardation, brachycephaly, synophrys, prognathism, behavior and sleep disturbances	Deletion 9q34
Down syndrome	Short stature, dysmorphic facies, mental retardation	Trisomy 21
ENPP1 gene mutations	Insulin resistance, childhood obesity	Gene mutation on chromosome 6q
Fröhlich syndrome	Hypothalamic tumor	
FTO gene polymorphism	Dysregulation of orexigenic hormone acyl-ghrelin, poor postprandial appetite suppression	Homozygous for *FTO* AA allele
Leptin or leptin receptor gene deficiency	Early-onset severe obesity, infertility (hypogonadotropic hypogonadism)	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
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 of chromosome 15 or loss of paternally expressed genes
Proopiomelanocortin deficiency	Obesity, red hair, adrenal insufficiency, hyperproinsulinemia	Loss-of-function mutations of the *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, growth hormone deficiency, pain insensitivity, hypothermia, precocious puberty, neural crest tumors	Unknown genes May be a paraneoplastic disorder
Turner syndrome	Ovarian dysgenesis, lymphedema, web neck, short stature, cognitive impairment	XO chromosome

cAMP, cyclic adenosine monophosphate; FT_4, free thyroxine; GH, growth hormone; IGF, insulin-like growth factor; PTH, parathyroid hormone; TSH, thyroid-stimulating hormone.

NHÂN CUNG HẠ ĐỐI

béo => nhiều mỡ => giảm lượng Adiponectin => vì vậy ngta thấy giảm Adiponectin thường kết hợp với giảm nhạy cảm insulin và tăng biến cố tim mạch
đói => tăng lượng Adiponectic

nervous system (Fig. 47-2). Gastrointestinal hormones, including cholecystokinin, glucagon-like peptide-1, peptide YY, and vagal neuronal feedback promote satiety. Ghrelin stimulates appetite. Adipose tissue provides feedback regarding energy storage levels to the brain through hormonal release of adiponectin and leptin. These hormones act on the arcuate nucleus in the hypothalamus and on the solitary tract nucleus in the brainstem and, in turn, activate distinct neuronal networks. Adipocytes secrete adiponectin into the blood, with reduced levels in response to obesity and increased levels in response to fasting. Reduced adiponectin levels are associated with lower insulin sensitivity and adverse cardiovascular outcomes. Leptin is directly involved in satiety, as low leptin levels stimulate food intake and high leptin levels inhibit hunger in animal models and in healthy human volunteers. Adiposity correlates to serum leptin levels among children and adults, with the direction of effect remaining unclear.

Numerous neuropeptides in the brain, including peptide YY, agouti-related peptide, and orexin, appear to affect appetite stimulation, whereas melanocortins and α-melanocortin–stimulating hormone are involved in satiety. The neuroendocrine control of appetite and weight involves a negative-feedback system, balanced between short-term control of appetite and long-term control of adiposity (including leptin). Peptide YY reduces food intake via the vagal–brainstem–hypothalamic pathway. Developmental changes in peptide YY are evident as infants have higher levels of peptide YY than school-age children and adults. Obese children have lower fasting levels of peptide YY compared to adults. Weight loss may restore levels of peptide YY in children even though this does not happen in adults. In addition, patients homozygous for the *FTO* obesity risk allele demonstrate poor regulation of the orexigenic hormone acyl-ghrelin and have poor postprandial appetite suppression.

- leptin: kích thích trung tâm no và ức chế trung tâm đói

- adiponectin: kich thích gan, cơ sử dụng glucose và tăng nhạy cảm insulin

- GLP1: kích thích trung tâm no kích thích tụy tiết insulin và ức chế tiết glucagon

- ghrelin kích thích trung tâm đói ở hạ đồi

- IL6: làm mô mỡ,, gan, cơ kháng insulin

- peptite YY (PYY): ức chế trung tâm no

- insulin: ức chế trung tâm no

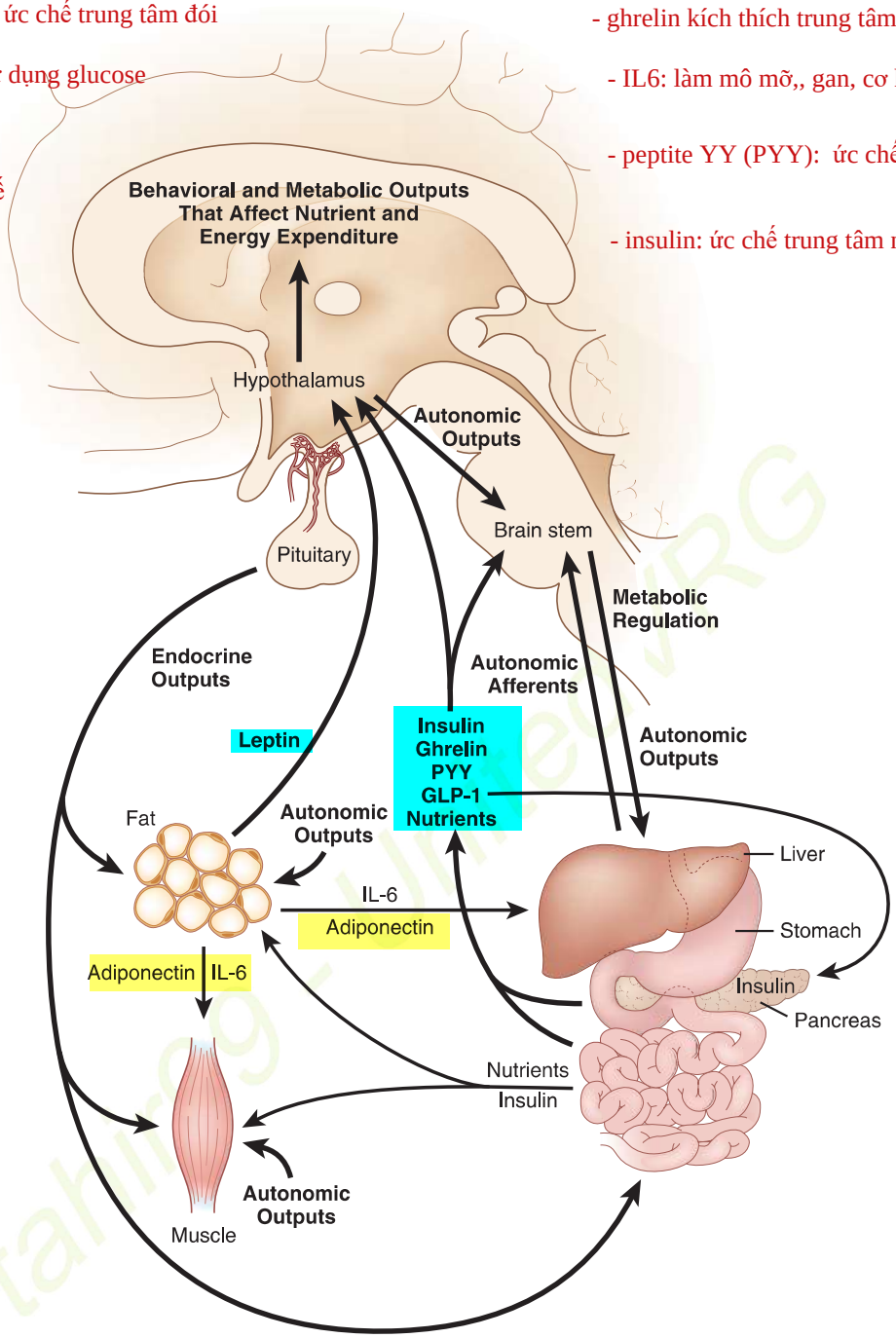

Figure 47-2 Regulation of energy homeostasis by the brain–adipose tissue–intestinal axis. Leptin stimulates hypothalamic anorexigenic and inhibits orexigenic neurons. Adiponectin stimulates hepatic, and muscle glucose utilization and increases insulin sensitivity, while interleukin-6 (IL-6) contributes to adipose tissue, muscle and hepatic insulin resistance. Peptide YY (PYY) inhibits orexigenic and glucagon-like peptide 1 (GLP-1) stimulates anorexigenic hypothalamic neurons. GLP-1 also augments glucose stimulated pancreatic insulin secretion and suppresses glucagon secretion. Insulin stimulates adipose tissue and muscle glucose uptake, enhances lipogenesis, suppresses hepatic glucose production, and has an inhibitory effect on the hypothalamic anorexigenic system. Ghrelin stimulates the orexigenic hypothalamic pathways. *(Modified from Melmed S, Polonsky KS, Larsen PR, Kronenberg HM:* Williams Textbook of Endocrinology, *ed 12, Philadelphia, 2011, Saunders.* Fig. 35-1.*)*

COMORBIDITIES

Complications of pediatric obesity occur during childhood and adolescence and persist into adulthood. An important reason to prevent and treat pediatric obesity is the increased risk for morbidity and mortality later in life. The Harvard Growth Study found that boys who were overweight during adolescence were twice as likely to die from cardiovascular disease as those who had normal weight. More immediate comorbidities include type 2 diabetes, hypertension, hyperlipidemia, and nonalcoholic fatty liver disease (Table 47-2). Insulin resistance increases with increasing adiposity and independently affects lipid metabolism and cardiovascular health. The metabolic syndrome (central obesity, hypertension, glucose intolerance, and hyperlipidemia) increases risk for cardiovascular morbidity and mortality. Nonalcoholic fatty liver disease (NAFLD) occurs in 10-25% of obese

bệnh tim mạch,ĐTĐ type 2, THA, tăng mỡ máu, gan nhiễm mỡ k do rượu

- hội chứng chuyển hóa: béo trung tâm, THA, bất dung nạp glucose, tăng mỡ máu

Table 47-2 Obesity-Associated Comorbidities

DISEASE	POSSIBLE SYMPTOMS	LABORATORY CRITERIA
CARDIOVASCULAR		
Dyslipidemia	HDL <40, LDL >130, total cholesterol >200	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 x-rays
Musculoskeletal problems	Back pain, joint pain, frequent strains or sprains, limp, hip pain, groin pain, leg bowing	X-rays
Slipped capital femoral epiphysis	Hip pain, knee pain, limp, decreased mobility of hip	Hip x-rays
PSYCHOLOGICAL		
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)

ALT, alanine aminotransferase; AST, aspartate aminotransferase; CT, computed tomography; FSH, follicle-stimulating hormone; HDL, high-density lipoprotein; LDL, low-density lipoprotein; LH, luteinizing hormone; MRI, magnetic resonance imaging; Peds QL, Pediatric Quality of Life Inventory; SBP, systolic blood pressure.

- Adiponectin (lad một peptide có hoạt tính kháng viêm) => giảm trong những người béo phì

adolescents. NAFLD is now the most common chronic liver disease in U.S. children and adolescents. It can present with advanced fibrosis or nonalcoholic steatohepatitis and may result in cirrhosis and hepatocellular carcinoma. Insulin resistance is commonly associated. Furthermore, NAFLD is independently associated with increased risk of cardiovascular disease.

Obesity may also be associated with chronic inflammation. Adiponectin, a peptide with antiinflammatory properties, occurs in reduced levels in obese patients as compared to insulin-sensitive, lean persons. Low adiponectin levels correlate with elevated levels of free fatty acids and plasma triglycerides as well as a high BMI, and high adiponectin levels correlate with peripheral insulin sensitivity. Adipocytes secrete peptides and cytokines into the circulation, and proinflammatory peptides such interleukin (IL)-6 and tumor necrosis factor-α (TNF-α) occur in higher levels in obese patients. Specifically, IL-6 stimulates production of C-reactive protein in the liver. C-reactive protein is a marker of inflammation and might link obesity, coronary disease, and subclinical inflammation.

Some complications of obesity are mechanical, including obstructive sleep apnea and orthopedic complications. Orthopedic complications include Blount disease and slipped femoral capital epiphysis (see Chapters 677, 678.4).

Mental health problems can coexist with obesity, with the possibility of bidirectional effects. These associations are modified by gender, ethnicity, and socioeconomic status. Self-esteem may be lower in obese adolescent girls compared to nonobese peers. Some studies have found an association between obesity and adolescent depression. There is considerable interest in the cooccurrence of eating disorders and obesity.

IDENTIFICATION

Overweight and obese children are often identified as part of routine medical care, and the child and family may be unaware that the child has increased adiposity. They may be unhappy with the medical provider for raising this issue and respond with denial or apparent lack of concern. It is often necessary to begin by helping the family understand the importance of healthy weight for current and future health, especially because intervention requires considerable effort by the child and the family. Forging a good therapeutic relationship is important, because obesity intervention requires a chronic disease management approach. Successful resolution of this problem necessitates considerable family and child effort over an extended period in order to change eating and activity behaviors.

EVALUATION

The evaluation of the overweight or obese child begins with examination of the growth chart for weight, height, and BMI trajectories; consideration of possible medical causes of obesity; and detailed

exploration of family eating, nutritional, and activity patterns. A complete pediatric history is used to uncover comorbid disorders. The family history focuses on the adiposity of other family members and the family history of obesity-associated disorders. The physical examination adds data that can lead to important diagnoses. Laboratory testing is guided by the need to identify comorbid conditions.

Examination of the growth chart reveals the severity, duration, and timing of obesity onset. Children who are overweight (BMI in the 85th-95th percentile) are less likely to have developed comorbid conditions than those who are obese (BMI ≥95th percentile). Those with a BMI ≥99th percentile are even more likely to have coexisting medical problems. Once obesity severity is determined, the BMI trajectory is examined to elucidate when the child became obese. Several periods during childhood are considered sensitive periods or times of increased risk for developing obesity, including infancy, adiposity rebound (when body fat is lowest at approximately age 5.5 yr), and adolescence. An abrupt change in BMI might signal the onset of a medical problem or a period of family or personal stress for the child. Examination of the weight trajectory can further expand understanding of how the problem developed. A young child might exhibit high weight and high height because linear growth can increase early in childhood if a child consumes excess energy. At some point, the weight percentile exceeds the height percentile and the child's BMI climbs into the obese range. Another example is a child whose weight rapidly increases when she reduces her activity level and consumes more meals away from home. Examination of the height trajectory can reveal endocrine problems, which often occur with slowing of linear growth.

Consideration of possible medical causes of obesity is essential, even though endocrine and genetic causes are rare (see Table 47-1). Growth hormone deficiency, hypothyroidism, and Cushing syndrome are examples of endocrine disorders that can lead to obesity. In general, these disorders manifest with slow linear growth. Because children who consume excessive amounts of calories tend to experience accelerated linear growth, short stature warrants further evaluation. Genetic disorders associated with obesity can have coexisting dysmorphic features, cognitive impairment, vision and hearing abnormalities, or short stature. In some children with congenital disorders such as myelodysplasia or muscular dystrophy, lower levels of physical activity can lead to secondary obesity. Some medications can cause excessive appetite and hyperphagia, resulting in obesity. Atypical antipsychotic medications often have this dramatic side effect. Rapid weight gain in a child or adolescent taking one of these medications might require a discontinuation of that medication. Poor linear growth and rapid changes in weight gain are indications for evaluation of possible medical causes.

Exploration of family eating and nutritional and activity patterns begins with a description of regular meal and snack times and family habits for walking, bicycle riding, active recreation, television, computer, and video game time. It is useful to request a 24-hr dietary recall with special attention to intake of fruits, vegetables, and water, as well as high-calorie foods and high-carbohydrate beverages. When possible, evaluation by a nutritionist is extremely helpful. This information will form the basis for incremental changes in eating behavior, caloric intake, and physical activity during the intervention.

Initial assessment of the overweight or obese child includes a complete review of bodily systems focusing on the possibility of comorbid conditions (see Table 47-2). Developmental delay and visual and hearing impairment can be associated with genetic disorders. Difficulty sleeping, snoring, or daytime sleepiness suggests the possibility of sleep apnea. Abdominal pain might suggest NAFLD. Symptoms of polyuria, nocturia, or polydipsia may be the result of type 2 diabetes. Hip or knee pain can be caused by secondary orthopedic problems, including Blount disease and slipped capital femoral epiphysis. Irregular menses may be associated with polycystic ovary syndrome. Acanthosis nigricans can suggest insulin resistance and type 2 diabetes (Fig. 47-3).

The family history begins with identifying other obese family members. Parental obesity is an important risk for child obesity. If all family members are obese, focusing the intervention on the entire family is reasonable. The child may be at increased risk for developing type 2 diabetes if a family history exists. Patients of African-American, Hispanic, or Native American heritage are also at increased risk for developing type 2 diabetes. Identification of a family history of hypertension, cardiovascular disease, or metabolic syndrome indicates increased risk for developing these obesity-associated conditions. If one helps the family to understand that childhood obesity increases risk for developing these chronic diseases, this educational intervention might serve as motivation to improve their nutrition and physical activity.

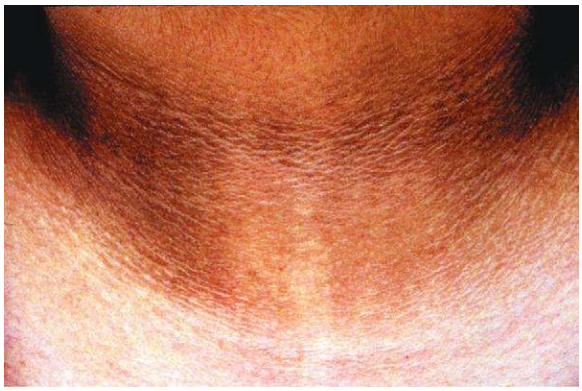

Figure 47-3 Acanthosis nigricans. *(From Gahagan S: Child and adolescent obesity,* Curr Probl Pediatr Adolesc Health Care *34:6–43, 2004.)*

Physical examination should be thorough, focusing on possible comorbid conditions (see Table 47-2). Careful screening for hypertension using an appropriately sized blood pressure cuff is important. Systematic examination of the skin can reveal acanthosis nigricans, suggesting insulin resistance, or hirsutism, suggesting polycystic ovary syndrome. Tanner staging can reveal premature adrenarche secondary to advanced sexual maturation in overweight and obese girls.

Laboratory testing for fasting plasma glucose, triglycerides, low-density lipoprotein and high-density lipoprotein cholesterol, and liver function tests are recommended as part of the initial evaluation for newly identified pediatric obesity (Table 47-3). Overweight children (BMI 85th-95th percentile) who have a family history of diabetes mellitus or signs of insulin resistance should also be evaluated with a fasting plasma glucose test. Other laboratory testing should be guided by history or physical examination findings.

INTERVENTION

There is evidence that some interventions result in modest but significant and sustained improvement in body mass. Based on behavior change theories, treatment includes specifying target behaviors, self-monitoring, goal setting, stimulus control, and promotion of self-efficacy and self-management skills. Behavior changes associated with improving BMI include drinking lower quantities of sugar-sweetened beverages, consuming higher-quality diets, increasing exercise, watching less TV, and self-weighing. Most successful interventions have been family based and take into account the child's developmental age. "Parent-only" treatment can be as effective as "parent–child" treatment. Because obesity is multifactorial, not all children and adolescents will respond to the same approach. For example, "loss-of-control" eating, associated with weight gain and obesity, predicts poor outcome in response to family-based treatment. Furthermore, clinical-treatment programs are expensive and not widely available. Therefore there is interest in novel approaches including Internet-based treatments and guided self-help.

It is important to begin with clear recommendations about appropriate caloric intake for the obese child (Table 47-4). Working with a dietitian is very helpful. Meals should be based on fruits, vegetables, whole grains, lean meat, fish, and poultry. Prepared foods should be chosen for their nutritional value, with attention to calories and fat. Foods that provide excessive calories and low nutritional value should be reserved for infrequent treats.

Weight-reduction diets in adults generally do not lead to sustained weight loss. Therefore, the focus should be on changes that can be maintained for life. Attention to eating patterns is helpful. Families should be encouraged to plan family meals, including breakfast. It is almost impossible for a child to make changes in nutritional intake and eating patterns if other family members do not make the same changes. Dietary needs also change developmentally, as adolescents require greatly increased calories during their growth spurts, and adults who lead inactive lives need fewer calories than active and growing children.

Table 47-3 Normal Laboratory Values for Recommended Tests

LABORATORY TEST	NORMAL VALUE
Glucose	<110 mg/dL
Insulin	<15 mU/L
Hemoglobin A_{1c}	<5.7%
AST (age 2-8 yr)	<58 U/L
AST (age 9-15 yr)	<46 U/L
AST (age 15-18 yr)	<35 U/L
ALT	<35 U/L
Total cholesterol	<170 mg/dL
LDL	<110 mg/dL
HDL	>45 mg/dL
Triglycerides (age 0-9 yr)	<75 mg/dL
Triglycerides (age 10-19 yr)	<90 mg/dL

AST, aspartate aminotransferase; ALT, alanine aminotransferase; LDL, low-density lipoprotein; HDL, high-density lipoprotein.

From Children's Hospital of Wisconsin: The NEW (nutrition, exercise and weight management) kids program *(PDF file).* http://www.chw.org/display/displayFile.asp?docid=33672&filename=/Groups/NEWKids/NewKidsReferral.PDF.

Psychological strategies are helpful. The "traffic light" diet groups foods into those that can be consumed without any limitations (green), in moderation (yellow), or reserved for infrequent treats (red) (Table 47-5). The concrete categories are very helpful to children and families. This approach can be adapted to any ethnic group or regional cuisine. Motivational interviewing begins with assessing how ready the patient is to make important behavioral changes. The professional then engages the patient in developing a strategy to take the next step toward the ultimate goal of healthy nutritional intake. This method allows the professional to take the role of a coach, helping the child and family reach their goals. Other behavioral approaches include family rules about where food may be consumed; for example, "not in the bedroom."

Increasing physical activity without decreasing caloric intake is unlikely to result in weight loss. Nonetheless, it can increase aerobic fitness and decrease percent body fat even without weight loss. Therefore, increasing physical activity can decrease risk for cardiovascular disease, improve well-being, and contribute to weight loss. Increased physical activity can be accomplished by walking to school, engaging in physical activity during leisure time with family and friends, or enrolling in organized sports. Children are more likely to be active if their parents are active. Just as family meals are recommended, family physical activity is recommended.

Active pursuits can replace more sedentary activities. The American Academy of Pediatrics recommends that screen time be restricted to no more than 2 hr/day for children >2 yr old and that children <2 yr old not watch television. Television watching is often associated with eating, and many highly caloric food products are marketed directly to children during child-oriented television programs.

Pediatric providers should assist families to develop goals to change nutritional intake and physical activity. They can also provide the child and family with needed information. The family should not expect immediate lowering of BMI percentile related to behavioral changes but can instead count on a gradual decrease in the rate of BMI percentile increase until it stabilizes, followed by a gradual decrease in BMI percentile. Referral to multidisciplinary, comprehensive pediatric weight-management programs is ideal for obese children whenever possible.

There is no effective pharmacotherapy resulting in reversal of excess adiposity in children and adolescents. Available medications result in

Table 47-4 Recommended Caloric Intake Designated by Age and Gender

LIFE-STAGE GROUP	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.

Table 47-5 Traffic Light Diet Plan

FEATURE	GREEN LIGHT FOODS	YELLOW LIGHT FOODS	RED LIGHT FOODS
Quality	Low-calorie, high-fiber, low-fat, nutrient-dense	Nutrient-dense, but higher in calories and fat	High in calories, sugar, and fat
Types of food	Fruits, vegetables	Lean meats, dairy, starches, grains	Fatty meats, sugar, sugar-sweetened beverages, fried foods
Quantity	Unlimited	Limited	Infrequent or avoided

modest weight loss or BMI improvement even when combined with behavioral interventions. Various classes of drugs are of interest, including those that decrease energy intake or act centrally as anorexiants, those that affect the availability of nutrients through intestinal or renal tubular reabsorption, and those that affect metabolism. The only U.S. Food and Drug Administration (FDA)-approved medication for obesity in children <16 yr old is orlistat, which decreases absorption of fat, resulting in modest weight loss. Complications include flatulence, oily stools, and spotting. This agent offers little benefit to severely obese adolescents. Because there are multiple redundant neural mechanisms that act to protect body weight, promoting weight loss is extremely difficult. For this reason, there is considerable interest in combining therapies that simultaneously target multiple weight-regulating pathways. One example, approved for adults, combines phentermine, a noradrenergic agent, with topiramate, a γ-aminobutyric acid (GABA)-ergic medication. This combination resulted in a mean 10.2-kg weight loss compared to 1.4 kg in the placebo group. Side effects are common and include dry mouth, constipation, paresthesias, insomnia, and cognitive dysfunction. Another promising example is the combination of amylin (decreases food intake and slows gastric emptying) with leptin (which has no anorexigenic effects when given alone). This combination requires injection and is in clinical trials in adults. Another FDA approved (for adults) drug is lorcaserin, a selective serotonin 2C receptor agonist. Establishing long-term safety and tolerability in children is a challenge as medications of interest have central nervous system effects or interfere with absorption of nutrients; teratologic effects must be considered for use in adolescent girls.

In some cases, it is reasonable to refer adolescents for evaluation for bariatric surgery. The American Pediatric Surgical Association Guidelines recommends that surgery be considered only in children with complete or near-complete skeletal maturity, a BMI ≥40, and a medical complication resulting from obesity, *after* they have failed 6 mo of a multidisciplinary weight management program. Surgical approaches include the Roux-en-Y and the adjustable gastric band. In obese adults, bariatric surgery reduces the risk of developing type 2 diabetes mellitus. In obese adult patients with existing type 2 diabetes, bariatric surgery improves the control of diabetes.

PREVENTION

Prevention of child and adolescent obesity is essential for public health in the United States and most other countries (Table 47-6 and 47-7). Efforts by pediatric providers can supplement national- and community-level public health programs. The National Institutes of Health and Centers for Disease Control and Prevention recommend a variety of initiatives to combat the current obesigenic environment, including promotion of breastfeeding, access to fruits and vegetables, walkable communities, and 60 min/day of activity for children. The U.S. Department of Agriculture sponsors programs promoting 5.5 cups of fruits and vegetables per day. Incentives for the food industry to promote consumption of healthier foods should be considered. Marketing of unhealthy foods to children has begun to be regulated. We expect to see changes in federal food programs including commodity foods, the Women, Infant, and Children Supplemental Food Program, and school-lunch programs to meet the needs of today's children.

Pediatric prevention efforts begin with careful monitoring of weight and BMI percentiles at healthcare maintenance visits. Attention to changes in BMI percentiles can alert the pediatric provider to increasing adiposity before the child becomes overweight or obese. All families should be counseled about healthy nutrition for their children because the current prevalence of overweight and obesity in adults is 65%. Therefore, approximately two-thirds of all children can be considered at risk for becoming overweight or obese at some time in their lives. Those who have an obese parent are at increased risk. Prevention efforts begin with promotion of exclusive breastfeeding for 6 mo and total breastfeeding for 12 mo. Introduction of infant foods at 6 mo should focus on cereals, fruits, and vegetables. Lean meats, poultry, and fish may be introduced later in the 1st year of life. Parents should be specifically counseled to avoid introducing highly sugared beverages and foods in the 1st year of life. Instead, they should expose their infants and young children to a rich variety of fruits, vegetables, grains, lean meats, poultry, and fish to facilitate acceptance of a diverse and healthy diet. Parenting matters, and authoritative parents are more likely to have children with a healthy weight than those who are authoritarian or permissive. Families who eat regularly scheduled meals together are less likely to have overweight or obese children. Child health professionals are able to address a child's nutritional status and to provide expertise in child growth and development.

Child health professionals can also promote physical activity during regular healthcare maintenance visits. Parents who spend some of their leisure time in physical activity promote healthy weight in their children. Beginning in infancy, parents should be cognizant of their child's developmental capability and need for physical activity. Because television, computer, and video game time can replace health-promoting physical activity, physicians should counsel parents to limit screen time for their children. Snacking during television watching should be discouraged. Parents can help their children to understand that television commercials intend to sell a product. Children can learn that their parents will help them by responsibly choosing healthy foods.

As obesity is determined by complex multifactorial conditions, prevention will take efforts at multiple levels of social organization. One example, EPODE (Ensemble Prévenons l'Obésité Des Enfants), is a multilevel prevention strategy, which began in France and has been adopted by more than 500 communities in 6 countries. The goal is for local environments, daycare centers, schools, recreational settings and families to adopt practices that promote healthy lifestyles for children from birth to 12 yr old. This initiative relies on 4 necessary components: political commitment to change, resources to support social marketing and changes, support services, evidence-based practices. All EPODE sites include monitoring and evaluation. Similar efforts are taking place in the United States. An example of a U.S. community effort is Shape Up Somerville, a citywide campaign to increase daily physical activity and healthy eating in Somerville, MA, which has been ongoing since 2002. This systems intervention focuses on school health curricula, healthier food in schools and restaurants, safe routes to school, walkable and bikeable streets and worksite wellness. Communitywide programs are important because neighborhood environmental factors (poverty) have been associated with obesity in its residents. Although these efforts have resulted in lower weight gain in older children and adolescents, there is considerable interest in focusing earlier in the life cycle. Beginning obesity prevention during pregnancy and engaging health systems, early childhood programs, and community systems to support healthier life cycles is an approach with tremendous promise.

Bibliography is available at Expert Consult.

Table 47-6 Proposed Suggestions for Preventing Obesity

PREGNANCY
Normalize body mass index before pregnancy.
Do not smoke.
Maintain moderate exercise as tolerated.
In gestational diabetics, provide meticulous glucose control.
Gestational weight gain within the Institute of Medicine (IOM) recommendations.
POSTPARTUM AND INFANCY
Breastfeeding: exclusive for 4-6 mo, continue with other foods for 12 mo.
Postpone the 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.
No 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 television 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.
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 in order 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 industry to develop more healthful products and to educate the consumer on product content.
Provide financial incentives to schools that initiate innovative physical activity 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, nonnutritious foods, and sugar-sweetened beverages directed at preschool children, and restrict advertising to school-age 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 *90:1871–1887, 2005.*

Table 47-7 Anticipatory Guidance: Establishing Healthy Eating Habits in Children

Do not punish a child during mealtimes with regard to eating. The emotional atmosphere of a meal is very important. Interactions during meals should be pleasant and happy.
Do not use foods as rewards.
Parents, siblings, and peers should model healthy eating, tasting new foods, and eating a well-balanced meal.
Children should be exposed to a wide range of foods, tastes, and textures.
New foods should be offered multiple times. Repeated exposure leads to acceptance and liking.
Forcing a child to eat a certain food will decrease the child's preference for that food. Children's wariness of new foods is normal and should be expected. Offering a variety of foods with low-energy density helps children balance energy intake.
Parents should control what foods are in the home. Restricting access to foods in the home will increase rather than decrease a child's desire for that food.
Children tend to be more aware of satiety than adults, so allow children to respond to satiety, and stop eating. Do not force children to "clean their plate."

Adapted from Benton D: Role of parents in the determination of food preferences of children and the development of obesity, Int J Obes Relat Metab Disord *28:858–869, 2004. Copyright 2004. Reprinted by permission from Macmillan Publishers Ltd.*