Prevention and Treatment of Pediatric Obesity:
An Endocrine Society Clinical Practice Guideline Based on Expert Opinion
Commercial Reprint Information

For information on reprint requests of more than 101 and commercial reprints contact:

Menna Burgess
Reprint Sales Specialist
Cadmus Professional Communications
Phone: 410.819.3960
Fax: 410.684.2789
Email: reprints2@cadmus.com

Single Reprint Information

For information on reprints of 100 and fewer, complete the guideline order form and return using one of the following methods:

Mail: The Endocrine Society
c/o Bank of America
P.O. Box 630721
Baltimore, MD 21263-0736
Fax: 301.941.0257
Email: Societyservices@endo-society.org

Questions & Correspondences

The Endocrine Society
Attn: Government & Public Affairs Department
8401 Connecticut Avenue, Suite 920
Chevy Chase, MD 20815
Phone: 301.941.0200
Email: govt-prof@endo-society.org
Web: www.endo-society.org

For more information on The Endocrine Society's Clinical Practice Guidelines or to download the complete version of this guideline, visit http://www.endo-society.org/guidelines/index.cfm.

© The Endocrine Society, 2008
Prevention and Treatment of Pediatric Obesity:
An Endocrine Society Clinical Practice Guideline
Based on Expert Opinion
# Table of Contents

<table>
<thead>
<tr>
<th>Section</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>Summary of Recommendations</td>
<td>4</td>
</tr>
<tr>
<td>Method of Development of Evidence-Based Guidelines</td>
<td>6</td>
</tr>
<tr>
<td>The Problem with Obesity</td>
<td>6</td>
</tr>
<tr>
<td>Diagnosis of Overweight and Obesity</td>
<td>9</td>
</tr>
<tr>
<td>Treatment of Obesity</td>
<td>14</td>
</tr>
<tr>
<td>Prevention of Obesity</td>
<td>24</td>
</tr>
<tr>
<td>Societal Barriers to Implementation</td>
<td>26</td>
</tr>
<tr>
<td>References</td>
<td>28</td>
</tr>
<tr>
<td>Order Form</td>
<td>39</td>
</tr>
<tr>
<td>Reprint Information, Questions &amp; Correspondences</td>
<td>Inside Back Cover</td>
</tr>
</tbody>
</table>
**Objective:** Our objective was to formulate practice guidelines for the treatment and prevention of pediatric obesity.

**Participants:** The Task Force was composed of a chair, selected by the Clinical Guidelines Subcommittee (CGS) of The Endocrine Society, eight additional experts, one methodologist, and a medical writer. The Task Force received no corporate funding or remuneration.

**Evidence:** Systematic reviews of available evidence were used to formulate the key treatment and prevention recommendations. We used the Grading of Recommendations, Assessment, Development, and Evaluation (GRADE) system to describe both the quality of evidence and the strength of recommendations. We used 'recommend' for strong recommendations and 'suggest' for weak recommendations.

**Consensus Process:** Consensus was guided by systematic reviews of evidence and discussions during one group meeting, several conference calls, and e-mail communications. The drafts prepared by the task force with the help of a medical writer were reviewed successively by The Endocrine Society’s CGS, Clinical Affairs Core Committee (CACC), the Lawson Wilkins Pediatric Endocrine Society’s Obesity Task Force, and Executive Committee. The version approved by the CGS and CACC was placed on The Endocrine Society’s Web site for comments by members. At each stage of review, the Task Force received written comments and incorporated needed changes.

**Conclusions:** We recommend defining overweight as body mass index (BMI) in at least the 85th percentile but less than the 95th percentile and obesity as BMI in at least the 95th percentile against routine endocrine studies unless the height velocity is attenuated or inappropriate for the family background or stage of puberty; referring patients to a geneticist if there is evidence of a genetic syndrome; evaluating for obesity-associated co-morbidities in children with BMI in at least the 85th percentile; and prescribing and supporting intensive lifestyle (dietary, physical activity, and behavioral) modification as the prerequisite for any treatment.

We suggest that pharmacotherapy (in combination with lifestyle modification) be considered in: 1) obese children only after failure of a formal program of intensive lifestyle modification; and 2) overweight children only if severe co-morbidities persist despite intensive lifestyle modification, particularly in children with a strong family history of type 2 diabetes or premature cardiovascular disease. Pharmacotherapy should be provided only by clinicians who are experienced in the use of antiobesity agents and aware of the potential for adverse reactions. We suggest bariatric surgery for adolescents with BMI above 50 kg/m², or BMI above 40 kg/m² with severe co-morbidities in whom lifestyle modifications and/or pharmacotherapy have failed. Candidates for surgery and their families must be psychologically stable and capable of adhering to lifestyle modifications. Access to experienced surgeons and sophisticated multidisciplinary teams who assess the benefits and risks of surgery is obligatory.

We emphasize the prevention of obesity by recommending breast-feeding of infants for at least 6 months and advocating that schools provide for 60 min of moderate to vigorous daily exercise in all grades. We suggest that clinicians educate children and parents through anticipatory guidance about healthy dietary and activity habits, and we advocate for restricting the availability of unhealthy food choices in schools, policies to ban advertising unhealthy food choices to children, and community redesign to maximize opportunities for safe walking and bike riding to school, athletic activities, and neighborhood shopping.

*(J Clin Endocrinol Metab 93: 4576–4599, 2008)*

**Abbreviations:** ALT, Alanine aminotransferase; BMI, body mass index; HDL, high-density lipoprotein; HOMA, homeostasis model assessment; IR, insulin resistance; LAGB, laparoscopic adjustable gastric banding; LDL, low-density lipoprotein; NAFLD, nonalcoholic fatty liver disease; PCOS, polycystic ovary disease; RYGB, Roux-en-Y gastric bypass; T2DM, type 2 diabetes mellitus.
SUMMARY OF RECOMMENDATIONS

1.0. THE PROBLEM WITH OBESITY

The objective of interventions in overweight and obese children and adolescents is the prevention or amelioration of obesity-related co-morbidities, e.g., glucose intolerance and T2DM, metabolic syndrome, dyslipidemia, and hypertension.

2.0. DIAGNOSIS OF OVERWEIGHT AND OBESITY

2.1. We recommend the use of the BMI (calculated as weight in kilograms divided by height in meters squared), with CDC derived normative percentiles, as the preferred method for the diagnosis of the overweight or obese child (48) (11 ΦΟΟΟ).

2.2. We recommend that a child be diagnosed as overweight if the BMI is in at least the 85th percentile but less than the 95th percentile for age and sex, and as obese if the BMI is in at least the 95th percentile for age and sex (11 ΦΟΟΟ).

2.3. We recommend against a routine laboratory evaluation for endocrine causes of obesity in obese children or early to mid-pubertal obese adolescents unless the child’s height velocity, assessed in relation to stage of puberty and family background, is attenuated (11 ΦΟΟΟ).

2.4.a. We recommend referral to a geneticist for children whose obesity has a syndromic etiology, especially in the presence of neurodevelopmental abnormalities (11 ΦΟΟΟ).

2.4.b. We suggest that parents of children who have inexorably gained weight from early infancy and have risen above the 97th percentile for weight by 3 yr of age be informed of the availability of MC4R genetic testing. However, the test is positive in only 2–4% of such patients who are above the 97th percentile for weight (71) and currently will not alter treatment (21 ΦΟΟΟ).

2.5. We recommend that children with a BMI in at least the 85th percentile be evaluated for associated co-morbidities and complications (see Table 1 and Fig. 1) (11 ΦΟΟΟ).

3.0. TREATMENT OF OBESITY

3.1. Lifestyle recommendations

3.1.0. We recommend that clinicians prescribe and support intensive lifestyle (dietary, physical activity, and behavioral) modification for the entire family and the patient in an age-appropriate manner and as the prerequisite for all overweight and obesity treatments for children and adolescents (11 ΦΟΟΟ).

3.1.1. Dietary recommendations

3.1.1.a. We recommend that clinicians prescribe and support healthy eating habits such as:
- Avoiding the consumption of calorie-dense, nutrient-poor foods (e.g. sweetened beverages, sports drinks, fruit drinks and juices, most “fast food,” and calorie-dense snacks) (11 ΦΟΟΟ).

3.1.1.b. We suggest that clinicians prescribe and support:
- Reducing saturated dietary fat intake for children older than 2 yr of age (21 ΦΟΟΟ).
- Increasing the intake of dietary fiber, fruits, and vegetables (21 ΦΟΟΟ).
- Eating timely, regular meals, particularly breakfast, and avoiding constant “grazing” during the day, especially after school (21 ΦΟΟΟ).

3.1.2. Physical activity recommendations

3.1.2.1. We recommend that clinicians prescribe and support 60 min of daily moderate to vigorous physical activity (11 ΦΟΟΟ).

3.1.2.2. We suggest that clinicians prescribe and support a decrease in time spent in sedentary activities, such as watching television, playing video games, or using computers for recreation. Screen time should be limited to 1–2 h per day, according to the American Academy of Pediatrics (182) (21 ΦΟΟΟ).

3.1.3. Psychosocial recommendations

3.1.3.a. We suggest that clinicians educate parents about the need for healthy rearing patterns related to diet and activity. Examples include parental modeling of healthy habits, avoidance of overly strict dieting, setting limits of acceptable behaviors, and avoidance of using food as a reward or punishment (21 ΦΟΟΟ).
3.1.3.b. We suggest that clinicians probe for and diagnose unhealthy intrafamily communication patterns and support rearing patterns that seek to enhance the child’s self-esteem (21 ΦΟΟΟ).

3.2. Pharmacotherapy recommendations

3.2.a. We suggest that pharmacotherapy (in combination with lifestyle modification) be considered if a formal program of intensive lifestyle modification has failed to limit weight gain or to mollify co-morbidities in obese children. Overweight children should not be treated with pharmacotherapeutic agents unless significant, severe co-morbidities persist despite intensive lifestyle modification. In these children, a strong family history of T2DM or cardiovascular risk factors strengthens the case for pharmacotherapy (21 ΦΟΟΟ).

3.2.b. We suggest that pharmacotherapy be offered only by clinicians who are experienced in the use of anti-obesity agents and are aware of the potential for adverse reactions (21 ΦΟΟΟ).

3.3. Bariatric surgery recommendations

3.3.a. We suggest that bariatric surgery be considered only under the following conditions:
1. The child has attained Tanner 4 or 5 pubertal development and final or near-final adult height.
2. The child has a BMI greater than 50 kg/m² or has BMI above 40 kg/m² and significant, severe co-morbidities.
3. Severe obesity and co-morbidities persist despite a formal program of lifestyle modification, with or without a trial of pharmacotherapy.
4. Psychological evaluation confirms the stability and competence of the family unit.
5. There is access to an experienced surgeon in a medical center employing a team capable of long-term follow-up of the metabolic and psychosocial needs of the patient and family, and the institution is either participating in a study of the outcome of bariatric surgery or sharing data.
6. The patient demonstrates the ability to adhere to the principles of healthy dietary and activity habits (21 ΦΟΟΟ).

3.3.b. We recommend against bariatric surgery for preadolescent children, for pregnant or breast-feeding adolescents, and for those planning to become pregnant within 2 yr of surgery; for any patient who has not mastered the principles of healthy dietary and activity habits; for any patient with an unresolved eating disorder, untreated psychiatric disorder, or Prader-Willi syndrome (11 ΦΟΟΟ).

4.0. PREVENTION OF OBESITY

4.1.a. We recommend breast-feeding for a minimum of 6 months (11 ΦΟΟΟ).

4.1.b. We suggest that clinicians promote and participate in efforts to educate children and parents by means of ongoing anticipatory guidance about healthy dietary and activity habits and, further, that clinicians encourage school systems to provide adequate health education courses promoting healthy eating habits (21 ΦΟΟΟ).

4.1.c. We suggest that clinicians promote and participate in efforts to educate the community about healthy dietary and activity habits (21 ΦΟΟΟ).

5.0. SOCIETAL BARRIERS TO IMPLEMENTATION

We suggest that clinicians advocate:

5.1. For regulatory policies designed to decrease the exposure of children and adolescents to the promotion of unhealthy food choices in the community (e.g. by media advertisements targeting children and adolescents) (21 ΦΟΟΟ).

5.2. That school districts ensure that only nutritionally sound food and drinks are available to children in the school environment, including the school cafeteria and alternative sources of food such as vending machines (21 ΦΟΟΟ).

5.3. For parental participation in the design of school-based dietary or physical activity programs and that schools educate parents about the rationale for these programs to ensure their understanding and cooperation (21 ΦΟΟΟ).

5.4. That community master planners design, redesign, and organize communities to maximize opportunities for safe walking or cycling to school, recreational activity and athletic events, and neighborhood shopping as a means to encourage greater physical activity (21 ΦΟΟΟ).

5.5. That clinicians advocate that policymakers provide incentives to ensure that retailers can offer affordable, high-quality fresh fruits and vegetables to all (21 ΦΟΟΟ).
METHOD OF DEVELOPMENT OF EVIDENCE-BASED GUIDELINES

The Clinical Guidelines Subcommittee of The Endocrine Society identified pediatric obesity as a priority area requiring practice guidelines and appointed a Task Force to formulate evidence-based recommendations.

Accordingly, the purpose of these guidelines is to summarize information concerning:

- The seriousness of pediatric obesity and overweight.
- The diagnostic criteria.
- The available treatments and when to apply them.
- The available measures to prevent overweight and obesity.

The Task Force elected to use the Grading of Recommendations Assessment, Development, and Evaluation (GRADE) method proposed by an international group with expertise in development and implementation of evidence-based guidelines (1). The Task Force used systematic reviews of available evidence to inform its key recommendations, and consistent language and graphical descriptions of both the strength of recommendations and the quality of evidence. The strength of a recommendation is indicated by the number 1 (strong recommendation, associated with the phrase “we recommend”) or 2 (weak recommendation, associated with the phrase “we suggest”). The quality of the evidence is indicated by cross-filled circles, such that ◼ denotes very low quality, ◼ ◼ low quality, ◼ ◼ ◼ moderate quality, and ◼ ◼ ◼ ◼ high-quality evidence. A detailed description of this grading scheme has been published elsewhere (2).

Each recommendation is followed by a description of the evidence, the values that panelists considered in making the recommendation, and in some instances remarks, a section in which panelists offer either technical comments or caveats. These remarks usually reflect unsystematic observations and should be considered suggestions.

MAKING STRONG RECOMMENDATIONS WITH LOW-QUALITY EVIDENCE

For many issues for which the evidence base is of low or very low quality, the Task Force, nonetheless, elected to make strong recommendations. As noted by Guyatt et al. (3), “The strength of any recommendation depends on the following two factors: the tradeoff between the benefits and risks and burdens; and the quality of the evidence regarding treatment effect…. A category 1 (strong) recommendation can be made when “the tradeoff is clear enough that most patients, despite differences in values, would make the same choice…. A category 2 (weak) recommendation is made when “the tradeoff is less clear, and individual patient values will likely lead to different choices…” (3).

We concur with the statement of Snow et al. (4): “Clinical practice guidelines are guides only and may not apply to all patients and all clinical situations. Thus, they are not intended to override clinicians’ judgment”. Endocrine Society clinical guidelines are valid for 3 yr, after which time they are revised.

1.0. THE PROBLEM WITH OBESITY

The objective of interventions in overweight and obese patients is the prevention or amelioration of obesity-related co-morbidities, e.g. glucose intolerance and type 2 diabetes mellitus (T2DM), metabolic syndrome, dyslipidemia, and hypertension.

The prevalence of obesity (BMI in the 95th percentile or above) increased almost 4-fold for 6- to 11-yr-old children and 3-fold for 12 to 19 yr olds between the surveys of 1963–1970 and 1999–2000 (5). For infants (0–23 months), the increase was from 7.2% in 1976–1980 to 11.6%; for 2 to 5 yr olds, the increase was from 5 to 13.9% during this time (6). Although rates vary among different ethnic groups, the overall prevalence of childhood obesity is 17.1% (7). The standards for weight and BMI percentiles are derived from the data of National Health and Nutrition Examination Survey (NHANES) II (1976–1980) and National Health...
Examination Survey (NHES) I (1963–1965) and II (1966–1970), when children were leaner.

The prevalence of overweight (BMI ≥ 85th but <95th percentile) in the same period increased by 2.5-fold to 37.2% in boys and girls older than 6 yr of age. Exact rates vary according to ethnicity (6).

The prevalence of pediatric obesity varies according to ethnicity, with especially high rates among Mexican-Americans and African-Americans. The role of poverty, although tending to be associated with a higher prevalence of obesity, is inconsistent across ethnicity and gender (8).

1.1. Association with adult disease

This increased prevalence is particularly important because childhood overweight and obesity are predictive of adult overweight and obesity. After adjusting for parental obesity, the odds ratio for a BMI greater than 27.8 kg/m² for men and greater than 27.3 kg/m² for women aged 21–29 yr increased from 1.3 when children aged 1–2 yr had a BMI greater than the 85th percentile to an odds ratio of 17.5 when children aged 15–17 yr had a BMI greater than the 85th percentile (9). The results are similar when looking at overweight adults at age 35 yr compared with childhood BMIs at the 75th, 85th, and 95th percentiles (10, 11). A BMI in the 85th percentile or above during the preschool and elementary school years is associated with a significantly increased risk of overweight in adolescents (12).

A major concern regarding the increased prevalence of obesity is its association with cardiovascular risk factors. Autopsy studies show the presence of not only fatty streaks but also fibrous plaques in the aorta and coronary arteries of obese teenagers (13, 14). The prevalence of cardiovascular risk factors [hypertriglyceridemia, high low-density lipoprotein (LDL) cholesterol, low high-density lipoprotein (HDL) cholesterol, hyperinsulinemia, and hypertension] increases with the rise in BMI. For children and adolescents with a BMI between the 85th and 94th percentiles, 19% had two or more cardiovascular risk factors and 5% had three or more. When the BMI was in the 95th percentile or above, 39% had two or more risk factors and 18% had three or more. When the BMI was in the 99th percentile or above, 59% had two or more cardiovascular risk factors and 33% had three or more (15).

The presence of cardiovascular risk factors during childhood can lead to an increased incidence of fatal and nonfatal cardiac events in adulthood (16). In this study, a cohort of 276,835 children aged 7–13 yr was followed into adult ages of 25–60 yr; 10,235 men and 4,318 women suffered either a fatal or a nonfatal cardiovascular event. The risk of a cardiovascular event was associated with the BMI of boys aged 7–13 and girls aged 10–13 yr. The relative risk of an event was apparent even with an increase in BMI z-score of just 1 U. For this Danish population of children, a BMI z-score of zero was equivalent to the 44th percentile on the Centers for Disease Control and Prevention (CDC) BMI charts, and a z-score of 1 was equivalent to the 88th percentile. For boys whose BMI z-score was +1, the relative risk of an adult cardiovascular event increased from 1.06 at 7 yr of age to 1.17 at 13 yr of age. For girls, the relative risk increased from the nonsignificant relative risk of 1.02 at 7 yr to a significant 1.12 at 13 yr. This study illustrates the deleterious effects of even a moderate increase in body weight during childhood and pinpoints the ages between 7 and 13 yr as one of the critical periods for intervention and prevention of overweight and obesity (16).

1.2. Pediatric obesity-associated co-morbidities

Concomitant with the greater prevalence of childhood obesity, the prevalence of T2DM has increased in children and adolescents (17). T2DM now accounts for 20% of diabetes in children aged 10–19 yr (18).

Overweight and obese individuals are at increased risk for dyslipidemia, most commonly a low HDL cholesterol. Lipid abnormalities were found in 12–17% of overweight and obese children (19).

The overall prevalence of the metabolic syndrome (a constellation of cardiovascular risk factors, comprising abdominal obesity and two or more of the following: elevated triglycerides, low HDL cholesterol, high blood pressure, increased plasma...
glucose) (20–22) among 12 to 19 yr olds in the United States is about 4.2% (23). The risk of the metabolic syndrome is nearly 50% in severely obese (BMI ≥ 40.6 kg/m²) adolescents (24).

Acanthosis nigricans is associated with both T2DM and insulin resistance (IR), but the strongest correlation is with obesity (25). The prevalence of acanthosis nigricans is greater in African-Americans (51%) than in Caucasians (8%) (25). Skin tags are also associated with T2DM (26).

Obesity, BMI in at least the 95th percentile, is associated with hyperandrogenemia and hyperinsulinism in pre- to mid-pubertal girls. Free testosterone concentrations in such girls are higher than in girls with a BMI below the 85th percentile (27). Taken together, these abnormalities place the obese adolescent girl at risk for polycystic ovary disease (PCOS), which, in turn, is exacerbated by obesity.

Systolic blood pressure correlates with BMI, skinfold thickness, and waist-to-hip ratio in children and adolescents (28). Normal values for blood pressure are standardized to the child's height percentile (29). For children with BMI in the 85th–94th percentile, a blood pressure greater than 95th percentile ranged from 6.6% at 2–5 yr to 13.3% at 16–19 yr for males; for females, the range was 4.4% at 2–5 yr to 16.3% at 16–19 yr of age (30). The relative risk for hypertension in obese children (BMI ≥ 95th percentile) was 3.26 after three consecutive blood pressure screenings (31).

There is a 10-fold increase in obesity-related glomerulosclerosis among adults (32). Morbidly obese adolescents have also been diagnosed with proteinuria and focal segmental glomerulosclerosis (FSGS), which may progress to end-stage renal disease or remit with weight loss (33).

Obese children are up to six times more likely than lean children to have obstructive sleep apnea (34). Obstructive sleep apnea is independently related to the development of hypertension, cardiovascular diseases, behavioral disorders, poor school performance in children, and poor quality of life in adults (35).

The prevalence of nonalcoholic fatty liver disease (NAFLD) is unclear and depends on the detection method. Between 10 and 25% of obese children have elevated transaminases, primarily alanine aminotransferase (ALT); as the degree of obesity increases, so does the prevalence of an elevated ALT. Abdominal sonography can detect a fatty liver, which is also associated with a greater BMI, in 52% of obese children. In an autopsy study, the incidence of fatty liver was 38% in obese children 2–19 yr of age (36). Morbidly obese children are particularly prone to fatty liver. Obese Hispanic children have a higher incidence of NAFLD. Weight reduction is an effective treatment for NAFLD (37). Although a benign clinical course is typical of NAFLD, it may be associated with increasing fibrosis and, rarely, progression to cirrhosis (38).

Obesity is a major risk factor for gallstones, which were found in 2% of obese children with a BMI greater than 30 kg/m² compared with an incidence of only 0.6% in nonobese children (39). Additional risk factors for gallstone development are the metabolic syndrome, hyperinsulinemia, and rapid, significant weight loss (40).

Excess weight is associated with slipped capital femoral epiphysis, genu valga, tibia vara (Blount disease), flat kneecap pressure/pain, flat foot, spondylolysthesis (low back pain), scoliosis, and osteoarthritis (41).

The prevalence of pseudotumor cerebri increases 15-fold with increasing BMI. The risk of intracranial hypertension is not related to the degree of obesity and is increased even in individuals who are just 10% above ideal body weight (41).

Psychosocial problems may become an issue for many overweight and obese children and adolescents (42).

1.3. Remarks

Although not a comorbidity per se, an earlier onset of pubarche and thelarche is associated with an elevated BMI (43, 44). Whether it is also associated with an earlier onset of menarche is controversial, a question that has been extensively reviewed (45).
There is, however, increasing evidence that premature pubarche is linked to the elements of the metabolic syndrome and to polycystic ovarian disease (46, 47). These children and adolescents require close observation as they mature into early adulthood.

### 2.0. DIAGNOSIS OF OVERWEIGHT AND OBESITY

#### 2.1. Recommendation

*We recommend the use of BMI [calculated as weight in kilograms divided by height in meters squared], with CDC-derived normative percentiles, as the preferred method for the diagnosis of the overweight or obese child (48) (11).*

#### 2.1. Evidence

Although various techniques are available to measure body fat, many are impractical for clinical use. BMI was adopted as the international standard clinical measure of adiposity (49).

An increased BMI is related to morbidity and mortality in adults, even if there is imprecision as to the relationship of BMI values to body fat content. Following its widespread use in adults, BMI is now accepted as the standard in children (8). However, the use of BMI in children is more complex than in adults. The BMI standard percentile distribution changes with an individual's age, sex, and, in some populations, ethnicity—thus limiting the utility of the international standard BMI charts for age (50).

#### 2.1. Values and preferences

The Task Force placed a high value on the identification of children and adolescents with high BMI to enable targeting recommended clinical interventions to those individuals and placed a relatively lower value on avoiding the potential psychological and socioeconomic consequences (e.g. labeling and medicalization) of such practice.

#### 2.1. Remarks

Although not ideal, the BMI is the internationally recognized standard for the definition of overweight and obesity, and the CDC standard curves are the most readily available standards for American children. The Task Force elected not to recommend another standard without higher quality evidence.

Stunted populations (51) may have an increased BMI without increased body fat. Taller individuals may have a high BMI even if their body proportions are equivalent to those of shorter individuals (52). In individuals who are not unusually short or tall, a high BMI is likely to predict abnormally high body fat content. However, a normal BMI does not always exclude the presence of increased body fat or increased risk of obesity-associated co-morbidities (see 2.2. Remarks). Fat tissue and BMI increase naturally with pubertal progression in girls (53), so that matching the value to chronological age may be misleading if the child progresses through puberty outside the average age range.

Much research has focused on using the *waist circumference* or *waist-to-height* ratio as a marker of obesity (54–58), as well as an additional marker for IR (23, 54, 59–61). This approach has the advantage of taking into account body fat distribution and the greater cardiovascular risk associated with visceral fat (62). Waist circumference standards for American children of various ethnic groups are available (63). Waist circumference should be recorded because future research may prove this measure to reflect fatness or morbidity better.

BMI (weight in kilograms divided by height in meters squared) is plotted according to the standardized CDC growth charts for the United States (48). Waist circumference is measured at the level of the iliac crest and interpreted according to age, sex, and racial standards (63).

#### 2.2. Recommendation

*We recommend that a child be diagnosed as overweight if the BMI is at least in the 85th percentile but less than the 95th percentile and obese if the BMI is at least in the 95th percentile for age and sex (11).*
2.2. Evidence

Adults experience increased morbidity and mortality when the BMI rises over 30 kg/m², which is about the 95th percentile of BMI at 19 1/2 yr of age (64). Clinicians caring for children can use curves generated by tracing the progression of BMI values for age back from the various percentiles at this older age to determine, in an age-adjusted manner, what BMI is equivalent to 25 or 30 kg/m² for an adult. Although these cutoffs are not derived from pediatric data, some experts consider them to be relevant indicators of health and morbidity (65, 66), but others do not (67).

2.2. Remarks

The BMI may be particularly imprecise in children younger than 4 yr of age (see 2.1. Remarks). Thus, clinicians may consider resorting to the available weight and height percentile charts. An increase in weight percentile that is out of proportion to the increase in height percentile should be a warning sign.

Data indicate that individuals of Asian and Native American ethnicity have a greater susceptibility to obesity-associated co-morbidities at a lower BMI than other ethnicities. An adult BMI of at least 23 kg/m² was associated with an increased risk of obesity-associated co-morbidities, and a BMI of at least 27.5 kg/m² represented high risk (68, 69). There are also differences in the BMI and waist circumference cut-points for Mexican-Americans, African-Americans, and whites in relation to the likelihood of having a cardiovascular disease risk factor (70). These discrepancies may also apply to children of different ethnic groups, although no long-term data are available as yet. Currently, there are insufficient data to recommend modifying the definition of either overweight or obesity in Asian- or Native-American children or adolescents.

2.3. Recommendation

We recommend against a routine laboratory evaluation for endocrine causes of obesity in obese children or early to mid-pubertal obese adolescents unless the child’s height velocity, assessed in relation to stage of puberty and family background, is attenuated (11 \( \Theta \Theta \Theta \Theta \)).

2.3. Evidence

GH deficiency and other forms of hypopituitarism, hypothyroidism, Cushing disease or syndrome, or pseudohypoparathyroidism are associated with increased BMI, but stature and height velocity are decreased, unless there is pubertal acceleration in height velocity. By contrast, stature and height velocity are usually increased with exogenous obesity. Testing for endocrine disorders is unlikely to be useful unless the patient is short in relation to the family’s background or is showing a deceleration in height velocity (71).

There are two uncommon circumstances in which the above rule may seem to be invalid. First is an adrenal tumor with concomitant hypersecretion of androgens and cortisol, but a careful physical examination should reveal signs of virilization. Second is the condition termed “growth without growth hormone” (72). It is rare in patients with idiopathic isolated GH deficiency or idiopathic combined pituitary hormone deficiencies. It can occur in patients whose panhypopituitarism follows removal of a craniopharyngioma or other suprasellar tumor or who have some other central nervous system abnormality. In 2003, five additional patients with this condition and panhypopituitarism were reported (73). These particular patients were not obese and presented with such features of pituitary deficiency as small penis and delayed puberty.

Hypothalamic obesity may be a related condition because it is associated with central nervous system insult with hypothalamic damage, which may lead to decreased energy expenditure and excessive caloric intake. It is not amenable to most lifestyle alterations (74) (see 3.2. Evidence for discussion of octreotide.)

2.3. Remarks

Deceleration in height velocity can be demonstrated either by using a height velocity curve normalized for age and/or stage of puberty or by observing that the patient is crossing major height percentile lines downward on the standardized height attainment charts (48). Because these height attainment charts are standardized for age and not for stage of puberty, they can be misleading in children with delayed or
precocious puberty. Height attainment and velocity charts that have the growth patterns not only of average-maturing children but also of early maturing and late-maturing children are available (75). Early- and late-maturing children represented on these curves are within the normal variation for the onset of puberty. Children demonstrating either true precocious or true delayed puberty deserve a full evaluation.

2.4. Recommendations

2.4.a. We recommend referral to a geneticist for children whose obesity has a syndromic etiology, especially in the presence of neurodevelopmental abnormalities (11).

2.4.b. We suggest that parents of children who have inexorably gained weight from early infancy and have risen above the 97th percentile for weight by 3 yr of age be informed of the availability of MC4R genetic testing. However, the test is positive in only 2%–4% of such patients who are above the 97th percentile for weight (71) and currently will not alter treatment (21).

2.4. Evidence

The more common genetic syndromes associated with obesity are also associated with neurodevelopmental abnormalities (Online Mendelian Inheritance in Man, available at: http://www.ncbi.nlm.nih.gov/omim/). Children with the various forms of Turner syndrome or SHOX deletions (76), however, are most often developmentally normal and may have few, if any, stigmata. Although it is attractive to postulate that human obesity is attributable to a single mutation or group of mutations in candidate genes with prominent roles in the appetite and fuel metabolic pathways, it is likely that most obesity is caused by the accumulation of polymorphisms in multiple genes with relatively small effects on weight gain or quantitative trait loci (77, 78). This concept is supported by a genome-wide scan in participants in the Framingham Heart Study demonstrating that a common polymorphism in a cholesterol-regulating gene is associated with obesity (79).

2.4. Values and preferences

In making the recommendation for referral to a geneticist, the Task Force placed a higher value on the identification of genetic disorders that will provide an explanation of the individual’s overweight problem, offer prognostic implications, and spare the family from feelings of guilt that often occur when traditional weight loss interventions are unsuccessful. We also placed a lower value on avoiding the downsides of genetic consultation, namely cost, labeling, and false-positive results of testing, when this referral is carefully limited to individuals with a syndromic etiology of obesity.

2.5. Recommendation

We recommend that children with a BMI in at least the 85th percentile be evaluated for associated co-morbidities and complications (Table 1 and Fig. 1, pp 12–13).

2.5. Evidence

In Section 1.1, we discussed the evidence for the increased prevalence of co-morbidities that are associated with a BMI in at least the 85th percentile. Of most concern are the cardiovascular risk factors (hypertriglyceridemia, increased LDL cholesterol, decreased HDL cholesterol, increased fasting blood glucose, and hypertension) predictive of future cardiovascular disease or T2DM or both. Thus, a large enough number of overweight children with a cardiovascular risk factor exist to warrant screening them, as well as obese children. Measurement of ALT is of more importance as the BMI increases into the obese range (discussed in Section 1.2).)

2.5. Values and preferences

We placed a relatively high value on the identification of weight-related complications and co-morbidities because this simultaneously focuses our limited treatment resources on those who, as a group, could accrue the greatest potential benefits of treatment and allows the identification and management of common and potentially important conditions. This recommendation places a lower value on avoiding medicalization and its costs and consequences.
## 2.5. Remarks

The medical history should assess the risk factors for the development of pediatric obesity including maternal diabetes, small for gestational age, large for gestational age, parental obesity (maternal is more important than paternal), early adiposity rebound (9, 82, 83), maternal weight gain during pregnancy, breast-feeding duration, weight of siblings and more distant relatives, possible consanguinity, as well as all other aspects of a standard pediatric history.

The medical history should also assess the presence of snoring and other manifestations of sleep apnea; polyuria, polydipsia, or weight loss (diabetes); and, in pubertal girls, acne, hirsutism (including the recent use of hair removal techniques that would mask the degree of hirsutism at the time of the examination), and onset and frequency of menses (PCOS). Note the use of antipsychotics associated with weight gain such as clozapine, risperidone, olanzapine, and quetiapine.

Although the various techniques assessing dietary intake are unreliable and subject to error (84), it is still important to estimate the type and quantity of beverage intake, frequency of dining out and where, and frequency and type of snacks, among other dietary issues. The efficacy of food frequency and activity questionnaires is discussed in a 2007 review (85). Activity history includes the duration, the frequency, and an estimate of the degree of difficulty of exercise performed during school and athome days, including participation in sports teams or other activities, walking to school and stores, etc. Estimates of screen time (i.e. time in front of a computer, playing video games, or viewing TV) per day may be useful. An environmental history includes safety of parks and neighborhoods and availability of playground equipment, gyms, and pools. All of this information is needed to develop an action plan.

Physical findings should include: 1) waist circumference at the level of the iliac crest and interpreted

### TABLE 1. Screening Tests for the More Common Obesity Co-Morbidities

<table>
<thead>
<tr>
<th>Co-morbidity</th>
<th>Case Detection Tests (Abnormal Values*)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Pre-diabetes</strong></td>
<td></td>
</tr>
<tr>
<td>1) Impaired fasting plasma glucose (Verify fasting status)</td>
<td>Fasting plasma glucose (&gt;100 mg/dL).</td>
</tr>
<tr>
<td>2) Impaired glucose tolerance (if OGTT is used)</td>
<td>2 hr glucose &gt;140 but &lt;200</td>
</tr>
<tr>
<td><strong>Diabetes mellitus</strong></td>
<td></td>
</tr>
<tr>
<td>Fasting plasma glucose &gt;126 mg/dL, or random value &gt;200 mg/dL (if OGTT used, 2 hr glucose &gt;200)</td>
<td>If asymptomatic, must have repeat abnormal values on another occasion</td>
</tr>
<tr>
<td><strong>Dyslipidemia</strong></td>
<td></td>
</tr>
<tr>
<td>Fasting (12-14 hours) lipids—</td>
<td></td>
</tr>
<tr>
<td>Triglycerides &gt;110 mg/dL (75th percentile); ≥160 mg/dL (90th percentile)</td>
<td></td>
</tr>
<tr>
<td>LDL cholesterol ≥110 mg/dL (75th percentile); ≥130 mg/dL (90th percentile)</td>
<td></td>
</tr>
<tr>
<td>Total cholesterol ≥180 mg/dL (75th percentile); ≥200 mg/dL (90th percentile)</td>
<td></td>
</tr>
<tr>
<td>HDL cholesterol ≤35 mg/dL (10th percentile); ≤40 mg/dL (25th percentile)</td>
<td>(80)**</td>
</tr>
<tr>
<td><strong>Hypertension</strong></td>
<td>Blood pressure &gt;90th percentile (standardized according to sex, age, and height percentile) (29)</td>
</tr>
<tr>
<td><strong>NAFLD</strong></td>
<td>Alanine aminotransferase (ALT) &gt;2 SD above the mean for the laboratory</td>
</tr>
</tbody>
</table>

OGTT, oral glucose tolerance test.

* To convert mg/dL to mmol/L, multiply by 0.0555 for glucose, 0.0259 for cholesterol, and 0.0113 for triglycerides.

** A proposed refinement of these abnormal lipid levels has the potential advantage of linking adolescents’ lipid levels to those of adults (81).
according to age, sex, and racial standards (63); 2) blood pressure, using height percentile normalized blood pressure tables to interpret the findings (29); 3) acanthosis nigricans and skin tags; 4) severe acne and hirsutism in pubertal age girls; 5) tenderness and range of motion of the knee, leg, or foot; and 6) peripheral edema. Physical findings associated with syndromic obesity should be identified, particularly if there is a neurodevelopmental abnormality.

Suggested screening tests are listed in Table 1. Other tests may be pertinent when other findings suggest sleep apnea (a sleep study, electrocardiogram, echocardiogram) or PCOS (serum testosterone, SHBG, dehydroepiandrosterone sulfate, 17-hydroxyprogesterone, and third generation LH and FSH). Testosterone assays in the range found in women and children may be imprecise (86, 87).

The fasting insulin is an optional test to quantify IR by calculation of homeostasis model assessment (HOMA)-IR or by noting that the fasting insulin is more than 2 SD above the mean for the laboratory doing the test. There is growing evidence for the association of IR with the development of T2DM in children (88, 89). Most of these children and adolescents are obese (90). Identification of children with IR has been proposed as a strategy for identifying high-risk children for targeted diabetes prevention. The gold standard for measuring IR,
the hyperinsulinemic-euglycemic clamp, is labor intensive and expensive and, therefore, not recommended for clinical use. A surrogate marker of IR is the HOMA-IR index, based on the measurement of fasting insulin and glucose with higher levels representing greater degrees of IR [HOMA-IR = (fasting insulin in µU/ml x fasting glucose in mmol/liter) divided by 22.5] (91). In nondiabetic children, HOMA-IR correlates as high as 0.91 with clamp measures (92, 93). Based on a study of 1,802 adolescents aged 12–19 yr from the 1999–2002 NHANES, a HOMA-IR greater than 4.39 was recommended as evidence for IR (90). On the basis of that criterion, roughly 46–52% of the adolescents with a BMI in at least the 95th percentile were insulin resistant; roughly 11–16% of those with a BMI in at least the 85th percentile but below the 95th percentile were insulin resistant; and only 4% of those with a BMI below the 85th percentile were insulin resistant (90). In a study of Mexican children aged 10–14 yr, very similar results were obtained, with 3, 12, and 47% of the children insulin resistant at BMIs below the 85th percentile, at least the 85th but below the 95th percentile, and at least the 95th percentile, respectively (94). In a study of Australian children aged 6–13 yr (95), increasing BMI z-score was continuously associated with complications of overweight in children. Even in the healthy weight range, there was a continuous linear or curvilinear association between increasing BMI z-score and comorbid risk factors. But there was a subset of children in whom there was no evidence of hyperinsulinism, even at BMI z-scores of +1 to +3. Conversely, some children had evidence for hyperinsulinism although their BMI z-scores ranged from −2 to 0.

The use of any index of IR is complicated by concerns about the lack of standardized measures for the quantification of insulin (96), by the need to be assured that the blood sample is obtained in a truly fasting state, and by the increase in IR from stage 1 to stage 3 of puberty and then a decrease in IR from stage 3 until adulthood (97).

Assessment of HOMA-IR in overweight obese children and adolescents may represent an important strategy for improving the efficacy of treatment for weight loss and chronic disease prevention (98–100). Furthermore, measurement of fasting serum insulin or of HOMA-IR is not necessary to establish a need for weight control or weight loss, especially because the expense of the test limits its potential for broad use in IR screening.

3.0. TREATMENT OF OBESITY

3.1. LIFESTYLE: GENERAL CONSIDERATIONS

3.1.0. Recommendation

We recommend that clinicians prescribe and support intensive lifestyle (dietary, physical activity, and behavioral) modification to the entire family and to the patient, in an age-appropriate manner, and as the prerequisite for all overweight and obesity treatments for children and adolescents (11, 1100).

3.1.0. Evidence

Successful weight management, through lifestyle interventions, confers important intermediate-term health benefits to adults such as reducing the incidence of T2DM (101) and improving cardiovascular fitness (102). Weight management programs in overweight children have also improved body composition and metabolic parameters (103, 104).

Unfortunately, it is commonly thought that lifestyle modification is not efficacious. Weight loss may not occur or, despite initial success, weight regain often begins after the active phase of the program has ended (105, 106).

A factor contributing to weight regain may be lack of a continued exercise program. The odds for weight regain are 2-fold greater in those patients who are sedentary (105). The importance of exercise in maintaining much of a person’s weight loss was one of the conclusions of a meta-analysis of long-term (3- to 5-yr) weight maintenance studies (27.2% weight loss retention in the low exercise group and 53.8% weight loss retention in the high exercise group) (107). This meta-analysis also found
that a weight loss of more than 20 kg in adults was associated with a greater degree of weight loss retention as compared with adults losing less than 10 kg. Additional factors associated with weight regain include Mexican-American ethnicity vs. non-Hispanic whites (odds ratio, 2.0) and losing more than 20% of body weight vs. less than 15% (odds ratio, 2.8) (105). Changes in hormone production and action may also play important roles (98). In socio-economically disadvantaged families, weight control may not be an important priority when balanced against other problems they face.

Although the long-term outlook may appear bleak, some studies report long-term success in a significant subgroup of patients. The results of population surveys indicate that 25% of adults who had lost more than 10% of their body weight maintained their weight losses for more than 5 yr (108). In another population-based study of individuals who had completed a commercial weight loss program, 18.8% maintained a weight loss of 10% or greater for 5 yr, whereas 42.6% maintained a weight loss of 5% or greater for 5 yr; 19.4% maintained their weights within 5 lb of their original goals (109). In both papers, the authors opined that patients who seek out a health care setting have already tried and failed to lose weight through other means and may represent a more refractory population.

Other factors, besides high levels of physical activity, associated with successful weight maintenance included continued reduced caloric intake, reduced fat intake, and reduced fast food consumption (110). Certain psychological traits also are associated with successful weight loss maintenance (111, 112). The success of lifestyle modification, at least in a reasonable portion of adults, has prompted its endorsement in guidelines from the U.S. Preventive Services Task Force (USPSTF). That group “recommends that clinicians screen all adult patients for obesity and offer intensive counseling and behavioral interventions to promote sustained weight loss...” (113). The USPSTF emphasizes the concept of intensive counseling, defined as at least one “person-to-person (individual or group) session per month for at least the first 3 months of the intervention” (113).

Emphasis should be on providing an intensive lifestyle modification program. This was most clearly shown by the results of a T2DM prevention trial in which one comparison was between the “standard lifestyle modification”—in which patients received the traditional written instructions about diet and exercise as well as an annual session discussing the importance of a healthy lifestyle—and an intensive plan covering diet, exercise, and behavior modification for the first 24 wk followed by monthly follow-up sessions. The initial goal was a 7% weight loss. The average weight loss in the standard lifestyle modification group was 0.1 kg and in the intensive modification group was 5.6 kg (P < 0.001). Significant weight loss was maintained over the 4 yr of follow-up. The cumulative incidence of diabetes after 3 yr was 28.9% in the standard modification group and only 14.4% in the intensive modification group (114).

Although good-quality pediatric and adolescent data are scarce (115), there is sufficient evidence that intensive lifestyle modification programs, as in adults, can be an effective tool for pediatric weight control (104, 116). Furthermore, implementation of a formal maintenance program after the treatment phase is completed can be of added importance in maintaining achieved weight loss (116). This fits into a concept of obesity as a chronic disease (117).

A meta-analysis of randomized pediatric trials, commissioned by this Task Force, of combined lifestyle interventions (diet and exercise) for treating obesity showed a modest but significant effect on obesity (equivalent to a decrease in BMI of 1.5 kg/m²; P < 0.00001) when these interventions targeted family involvement. There was a decrease in BMI of 0.4 kg/m² when parents were not specifically included and the effect on weight loss was not significant (P = 0.13). Although the indirect comparison of effects across studies indicates that treatment is significantly more effective when it targets the family (P value for the interaction was 0.04), the direct comparisons (within the same trial) were inconsistent across trials and showed no significant difference (118). These results suggest involving the family when delivering combined lifestyle interventions. Another meta-analysis that
looked specifically at the effects of family-behavioral therapy produced parallel results (119).

An additional meta-analysis of randomized controlled trials of lifestyle interventions, but without an analysis of family involvement, also found moderate positive effects from the interventions when compared with no treatment, wait-list, or information-only controls. These effects persisted for an average follow-up period of 15 months (120). Although there was overlap with the meta-analysis we commissioned, each study contained reports not covered by the other.

An evidence-based position statement of the American Dietetic Association (ADA) supports the utility of family-based lifestyle interventions in children and of similar multi-component programs for adolescents (121). These recommendations are consistent with the conclusions of an evidence-based review of pharmacological interventions for childhood obesity that highlighted the importance of concomitant intensive lifestyle interventions—dietary, exercise, and family counseling (98)—as well as by a combined CDC and American Medical Association expert committee (122).

3.1.0. Values and preferences

In making this recommendation (and other recommendations associated with specific lifestyle choices), the Task Force placed a relatively higher value on promoting lifestyle choices with their potential wide-reaching benefits and safety, and it placed a relatively lower value on avoiding the costs of implementation of lifestyle interventions with their potentially limited impact.

Although physicians generally strive to cure the great majority of their patients and may view a long-term success rate of 25% with despair, we should not retreat into a state of therapeutic nihilism. We are at a stage where we must treat overweight and obese patients, accept that perhaps only 25% may respond, but refine our techniques so that lifestyle modification will be effective in an increasing percentage of patients.

3.1.0. Remarks

Weight loss should be encouraged in patients with severe obesity and significant co-morbidities. In this regard, a decrease in BMI of 1.5 kg/m², as reported in the meta-analysis commissioned by the Task Force, may seem trivial, but if it is maintained over a longer term, overweight or minimally obese growing children and adolescents without co-morbidities may benefit by simply maintaining weight; BMI will decline as linear growth proceeds, and lifestyle modification may reduce fat mass, increase lean body mass, and improve cardiovascular fitness (123). In the more severely obese or in physically mature patients, moderate weight loss of only 7% was associated with a decrease in the incidence of T2DM (114). This may be a more realistic goal for the severely obese (see 3.1.0. Evidence). Well-designed, randomized controlled studies of large numbers of patients, employing intensive lifestyle intervention and follow-up maintenance programs, are clearly needed to develop improved techniques. Some of the factors that should be addressed in such studies are described (115).

Successful lifestyle intervention and preventive measures are labor intensive, and even more so if there is a post-treatment maintenance program. The time spent in delivering intensive lifestyle interventions—direct contact with the patient and family (we cannot overemphasize the importance of family involvement) at least once a month for the first 3 months and comprising dietary and nutritional education, a physical activity prescription, and behavioral therapy—is poorly reimbursed, and thus there may be a disincentive to provide these services (Ref. 124, pp 221–227).

3.1.1 Dietary recommendations

3.1.1.a. We recommend that clinicians prescribe and support healthy eating habits such as:
- Avoiding the consumption of calorie-dense, nutrient-poor foods (e.g. sweetened beverages, sports drinks, fruit drinks and juices, most “fast food,” and calorie-dense snacks) (114)

3.1.1.b. We suggest that clinicians prescribe and support:
• Controlling caloric intake through portion control in accordance with the Guidelines of the American Academy of Pediatrics (http://pediatrics.aappublications.org/cgi/reprint/117/2/544) (21). Reducing saturated dietary fat intake for children older than 2 yr of age (21).
• Increasing the intake of dietary fiber, fruits, and vegetables (21).
• Eating timely, regular meals, particularly breakfast, and avoiding constant “grazing” during the day, especially after school (21).

3.1.1. Evidence
Excessive intake of low-nutrient, calorie-dense, high-fat food, and sugar-sweetened beverages is a risk factor for obesity (125–127). Since 1965 teens have doubled their consumption of sugared soft drinks and fruit-flavored beverages. The average adolescent boy now consumes 50 ounces of such beverages per day (128). Reducing consumption of sugared beverages (e.g., soda, juices and fruit drinks, and sports drinks) (129, 130) may be an effective way to reduce ingested calories (131, 132). School-based interventions can reduce soda consumption and reduce weight in students at the highest BMI percentiles (133, 134).

Increased portion size parallels the increase in obesity (135, 136). Texas elementary schools are imparting portion control education using educational materials available from the Texas Department of Agriculture (http://www.squaremeals.org/).

Decreasing caloric intake by consuming more fruits and vegetables instead of dietary fat can decrease the risk of developing obesity and T2DM (134, 137). Most children do not meet the dietary recommendations needed to maintain a healthy weight and eat less than half the recommended five fruits and vegetables a day (138, 139). Inadequate consumption of dietary fiber may contribute to excessive weight gain (140).

Fast food accounted for only 2% of children’s total caloric intake in 1977–1978, but the proportion increased to 10% by 1994–1996 (141). Consumption of food prepared away from home increased from 18% of total calories in 1977–1978 to 32% in 1994–1996 (142). Overweight children are more likely to report eating fast food and to attend a school with vending machines selling chips and candy (143). Vending machine foods are sold separate from the National School Lunch Program and are currently exempt from the Dietary Guidelines; these food sales represent an increasing percentage of total school food sales (144).

Overweight children and adolescents are now more likely to skip breakfast and consume a few large meals per day (145, 146) than their leaner counterparts who are more likely to consume smaller, more frequent meals (147–149). Eating breakfast reduces snacking throughout the remainder of the day (150). One fourth to one third of the energy intake of adolescents is from snacks (151). Because snacks tend to be higher in calorie density than meals (152), frequent snacking is associated with high intake of fat, sugar, and calories (153, 154) and with overweight among children (155). Intervention studies to confirm a relationship between meal and snack patterns and overweight are needed.

3.1.1. Values and preferences
See description of values and preferences in Section 3.1.0.

3.1.1. Remarks
Many different diets have been proposed for weight loss. Currently there is debate about whether a low-fat (usually 30% of calories as fat) or a low-carbohydrate diet is more efficacious. A meta-analysis of randomized controlled studies in adults showed that the low-carbohydrate diet resulted in moderately greater weight loss by 6 months but not after 12 months (156). At the present time, there is insufficient pediatric evidence to warrant recommending any one hypocaloric diet over another. But caution should be exercised when using unbalanced hypocaloric diets that may be deficient in essential vitamins and minerals.

3.1.2. Physical activity recommendations
3.1.2.1. We recommend that clinicians prescribe and support 60 min of daily moderate to vigorous physical activity (1).
3.1.2.2. We suggest that clinicians prescribe and support a decrease in time spent in sedentary activities, such as watching television, playing video games, or using computers for recreation. Screen time should be limited to 1–2 h per day, according to the American Academy of Pediatrics (182) (Ref. 2).

3.1.2.1. Evidence

In the absence of caloric restriction, moderate exercise does not generally cause weight loss. However, in combination with decreased caloric intake, exercise can achieve significant weight loss. Studies performed in the school setting have shown the beneficial effects of exercise in children and youth (157–161). The beneficial effects of both aerobic exercise and resistance training can be short-lived, and exercise must be sustained. Time spent in daily vigorous exercise in excess of 60 min per day provides additional reduction in cardiovascular risk factors (162).

Physical fitness, even without weight loss, may offer some health benefits. Improvement in cardiovascular fitness was associated, in young adults, with improvement in cardiovascular disease risk factors over a 7-yr period (163).

School-based interventions have focused on reducing obesity rates (164–174). The Cardiovascular Health in Children (CHIC-I) study was able to improve physiological outcomes by decreasing body fat and cholesterol concentrations (168). The CHIC-II study was effective in reducing body fat and blood pressure in middle school youth (171). One of the main reasons the short-term CHIC interventions were successful in affecting physiological variables may be the increased time spent in actual moderate to vigorous physical activity in school (20 min in elementary schools and 30 min in middle schools). Both school design and adult supervision for physical activity have been shown to affect the amount of physical activity that 6th to 8th graders engage in during their free time (175). School systems are beginning to sponsor after-school lifetime fitness programs (176). Such pilot programs appear successful in controlling the rate of weight gain (177).

A meta-analysis of physical activity interventions, commissioned by this Task Force, found a moderate treatment effect when the outcome measure reflected body composition (e.g. fat percentage) ($P = 0.00001$) and no effect when the outcome was BMI; the difference in results was highly significant ($P = 0.002$). This would support the concept that exercise may affect cardiovascular risk factors by improving insulin sensitivity and adiposity, and by increasing lean body mass without affecting total body weight. However, there was no significant difference in responses in the six trials reporting both measures (118). This lack of a significant difference in responses may be due to there being no true difference, to chance [imprecision, i.e. there is a difference but the studies or the effects were not large enough to be detected (a type II error)], or to bias. This probably reflects reporting bias in which the significant outcome measure was more likely to be reported than an insignificant finding, such that when studies reported both measures the effect was similar.

3.1.2.1. Values and preferences

See description of values and preferences at Section 3.1.0.

3.1.2.1. Remarks

Although current recommendations state that schoolchildren (who spend about half their waking hours in school) should receive a minimum of 30 min of moderately vigorous physical activity each school day, only 8% of elementary schools and roughly 6% of junior and senior high schools provide physical education for the entire school year for all students and for all grades (Ref. 124, pp 237–284). Furthermore, only 10–30% of that time is spent in moderate to vigorous exercise. The limited support for physical education in the schools is unfortunate because exercise has been linked not only to cardiovascular benefits in children (178) but also to improvements in cognitive function and concentration (179, 180). School units on health and hygiene, in which children are taught about nutrition and good health habits, have atrophied. An average of only 9h is now spent on health education.
during the school year, although pilot studies have demonstrated the efficacy of curricula designed to reduce television-viewing time (Ref. 124, pp 237–318).

Moderate to vigorous exercise is defined as causing “some increase in breathing and heart rate usually associated (in a healthy person) with brisk walking, dancing, swimming, or cycling on flat terrain.” In exercise physiology terms, the energy expended is at least 3 (METS) metabolic equivalents (181).

As discussed under 3.1.0. Values and preferences and 3.1.0. Remarks, studies using either weight loss or BMI as an endpoint may miss the positive effects of exercise, such as shifting body composition from fat to muscle. The net weight loss under such circumstances may be nil.

3.1.2.2. Evidence

Activities involving TV, videotapes, video games, and computers average 5 1/2 h per day (183, 184). An intervention to reduce TV viewing and meals eaten in front of the TV was successful in a comparatively high-socioeconomic-status population of ethnically homogeneous schoolchildren (185). Results of the meta-analysis, commissioned by the Task Force, of the three randomized trials of interventions focused on reducing sedentary activity were imprecise (i.e., consistent with both favorable and unfavorable impact on obesity outcomes) (118).

3.1.2.2. Values and preferences

See description of values and preferences at Section 3.1.0.

3.1.3 Psychosocial recommendations

3.1.3.a. We suggest that clinicians educate parents about the need for healthy rearing patterns related to diet and activity. Examples include parental modeling of healthy habits, avoidance of overly strict dieting, setting limits of acceptable behaviors, and avoidance of using food as a reward or punishment (21 ☐☒☒☒ ).

3.1.3.b. We suggest that clinicians probe for and diagnose unhealthy intrafamily communication patterns and support rearing patterns that seek to enhance the child’s self-esteem (21 ☐☒☒☒ ).

3.1.3. Evidence

The importance of involving the whole family, and not just the child, in treatment interventions is discussed in Section 3.1.0.

The role of parent-child interactions and parenting style in the development of unhealthy lifestyle habits is a subject of investigation (186, 187). An additional factor to overcome before any intervention may be the parents’ inability to recognize that their child is overweight, particularly for the preschool child (188).

3.1.3. Remarks

It is important to remember that clinician interactions with the family and all educational materials should be culturally sensitive and in the language best understood by the family.

3.2. PHARMACOTHERAPY RECOMMENDATIONS

3.2.a. We suggest that pharmacotherapy (in combination with lifestyle modification) be considered if a formal program of intensive lifestyle modification has failed to limit weight gain or to mollify co-morbidities in obese children. Overweight children should not be treated with pharmacotherapeutic agents unless significant, severe co-morbidities persist despite intensive lifestyle modification. In these children, a strong family history of T2DM or cardiovascular risk factors strengthens the case for pharmacotherapy (21 ☐☒☒☒ ).

3.2.b. We suggest that pharmacotherapy be offered only by clinicians who are experienced in the use of antiobesity agents and are aware of the potential for adverse reactions (21 ☐☒☒☒ ).

3.2. Evidence

The utility of pharmacotherapy in adolescents has been reviewed (98, 189), and the use of medication to treat severe obesity can be an additional treatment modality (176, 190–192). Several limitations preclude physicians from early implementation of drug therapies. These include: 1) the lack of U.S. Food and Drug Administration (FDA) approval for use in preadolescents and younger adolescents; 2) reduced efficacy over time, with a plateau after 6 months of
treatment due to reduced energy expenditure offsetting the decrease in energy intake—an effect also noted with hypocaloric diets (193); 3) the existence of a limited number of well-controlled studies of the safety and efficacy of pharmacological intervention in obese children; and 4) the need to weigh the relative risk of severe adverse events in children against the long-term potential for obesity-related morbidity and mortality. Despite these concerns, the negative health impact of childhood obesity may justify long-term medication, but only in combination with lifestyle modification (98, 176, 190–192).

Three pharmacotherapeutic agents—sibutramine, a nonselective reuptake inhibitor appetite suppressant that is most potent for serotonin and norepinephrine, but also blocks dopamine reuptake (194, 195); orlistat, which specifically inhibits intestinal lipase and can reduce fat and cholesterol absorption by approximately 30% (196); and metformin (not FDA approved for the treatment of obesity)—are most commonly used at present. Although metformin reduces hepatic glucose production and plasma insulin, inhibits lipogenesis, increases peripheral insulin sensitivity, and may reduce appetite by increasing levels of glucagon-like peptide (98), its mechanism of action on weight is unresolved. Only sibutramine (for children > 16 yr of age) and orlistat (for children 12 yr of age) are FDA approved for the treatment of obesity in adolescents (Table 2).

The meta-analysis commissioned by the Task Force (118) showed that sibutramine demonstrated the most effect, with a decrease in BMI of 2.4 kg/m² after 6 months. This effect was statistically significant, but patients receiving sibutramine had greater increase in blood pressure and pulse rate than placebotreated patients. Orlistat was associated with a significant fall in BMI of 0.7 kg/m², but treatment was associated with increased rates of gastrointestinal side effects including abdominal discomfort, pain, and steatorrhea. Side effects are usually mild to moderate and generally decrease in frequency with continued treatment; this decrease may result from patients learning to consume less dietary fat to avoid these side effects. Orlistat must be taken with each meal, thus reducing its utility in children who often are in school during lunchtime.

**Additional medications used but not FDA-approved for the treatment of obesity**

Metformin decreased BMI slightly but significantly in each of the three studies analyzed, but the overall effect did not reach statistical significance in the meta-analysis (118). This outcome may, in part, reflect differences in study design, because one of the studies (201) did not include dietary restrictions. Metformin may be useful in combating the weight gain observed in children taking atypical psychotropic medications, e.g. clozapine, olanzapine, risperidone, quetiapine, aripiprazole, and valproate (209). However, cessation of metformin therapy may lead to a rebound hyperinsulinemia and rapid weight gain, whether or not the offending psychotropic medication is continued. Although not approved by the FDA for this indication, metformin has been successfully used to treat PCOS with and without concomitant obesity and insulin insensitivity (210, 211). Metformin is approved for the treatment of T2DM in children at least 10 yr of age.

Although not FDA-approved for the treatment of obesity, the FDA-approved product labeling for growth hormone (GH) presents data indicating that GH treatment of children with Prader-Willi syndrome decreases body fat percentage and increases lean body mass (212). GH seemed to be of particular benefit when started before 18 months of age (213). A summary of the benefits and risks of GH treatment of Prader-Willi syndrome was published in 2008 (208). Despite these encouraging preliminary results in Prader-Willi syndrome, a review of the effects of GH treatment in adult obesity failed to reveal any consistent beneficial effects and described the difficulties in assessing body composition (214).

**Octreotide** acts on the voltage-gated calcium channel of the β-cell coupled to the somatostatin receptor (215, 216) and through G- inhibition limits the opening of this calcium channel, decreasing the magnitude of insulin response to glucose (217). An examination of BMI responses to octreotide in a multivariate analysis in children with hypothalamic obesity demonstrated that insulin hypersecretion with concomitant retention of insulin sensitivity before therapy predicted success (99). In
Mutations of the leptin gene in humans recapitulate the phenotype of the ob/ob leptin-deficient mouse (219). Few such patients (no more than 10 as of 2008) have been described. These patients manifest hyperphagia from birth, with obesity documentable as

hyperinsulin-secretin obese adults, treatment with octreotide long-acting repeatable (LAR) for 6 months resulted in significant weight loss as compared with controls. Greater weight loss correlated with a greater degree of insulin hypersecretion (218).
early as 6 months of age. The excess insulin, due to vagal overactivity and IR, may cross-react with the IGF-I receptor to increase height velocity and bone age. Serum leptin is undetectable. The diagnosis is made by unmeasurable serum leptin levels. Leptin therapy in these patients results in extraordinary loss of weight and fat mass (205, 220), along with reduction in hyperphagia, resolution of obesity, induction of puberty, and improvement in T cell responsiveness (206).

Topiramate is a novel anticonvulsant used in children and adults that blocks voltage-dependent sodium channels, enhances the activity of the GABA receptor, and antagonizes a glutamate receptor other than the N-methyl-D-aspartate receptor. Topiramate may induce insulin sensitivity in liver and muscle and directly in adipocytes (221). In a study of adults (207), almost 33% of the subjects dropped out because of adverse events (Table 2). Anorexia and weight loss occur early in 10 to 40% of children treated with topiramate for seizures, an effect that in some cases has led to discontinuing the medication (222). There are no studies of topiramate in childhood obesity. Its use as an anti-obesity agent should be limited because it promotes drowsiness and interferes with cognition, and it should not be used outside of a clinical research study.

Table 2 summarizes the dosage, efficacy, adverse effects, contraindications, and monitoring needs of some of the medications used for the treatment of obesity.

### 3.2. Values and Preferences

The suggestion to limit use of pharmacotherapy in children and adolescents reflects our preference for managing pediatric obesity as a serious lifestyle condition with important lifelong consequences and our placing a lower value on achieving short term success, a higher value on avoiding drug side effects and costs that accumulate over time, and a higher value on achieving healthy weight through the incorporation of healthy behaviors.

### 3.2. Remarks

The assessment of drug efficacy presented here is founded only on the ability of medications to reduce BMI or BMI z-score. It must be emphasized that “antiobesity” drugs may have differential effects on BMI and obesity-associated co-morbidities (98). For example, certain medications (e.g. sibutramine, orlistat) may be more effective for weight loss than for treatment of impaired glucose tolerance, whereas other medications (e.g. metformin) have more potent effects on insulin production and glucose tolerance than on body weight per se. Drug selection should be tailored to the individual patient, with strong attention paid to the family history. The primary objective is to prevent co-morbidities in the obese (BMI ≥ 95th percentile) patient. Most importantly, the benefits of any drug used to treat childhood obesity should clearly outweigh its risks.

In general, children with a BMI below the 95th percentile should not be treated with antiobesity drugs. Pharmacotherapy for overweight children (BMI ≥ 85th but < 95th percentile) should be reserved for those with significant, severe co-morbidities who have not responded to lifestyle modification. Although, as mentioned in 2.2. Remarks, data suggest that adult Asians (and Native-Americans) develop obesity-associated co-morbidities at a lower BMI than do Europeans (68, 69), similar data are not available for children and adolescents, and so we cannot recommend the use of pharmacotherapy at a BMI range differing from the above recommendations.

The use of pharmacotherapeutic agents not yet approved for the treatment of pediatric obesity should be restricted to participation in large, well-controlled clinical studies.

### 3.3. BARIATRIC SURGERY RECOMMENDATIONS

#### 3.3.a. We suggest that bariatric surgery be considered only under the following conditions:

1. The child has attained Tanner 4 or 5 pubertal development and final or near-final adult height.
2. The child has a BMI greater than 50 kg/m² or has BMI above 40 kg/m² and significant, severe co-morbidities.
3. Severe obesity and co-morbidities persist despite a formal program of lifestyle modification, with or
without a trial of pharmacotherapy.

4. Psychological evaluation confirms the stability and competence of the family unit.

5. There is access to an experienced surgeon in a medical center employing a team capable of long-term follow-up of the metabolic and psychosocial needs of the patient and family, and the institution is either participating in a study of the outcome of bariatric surgery or sharing data.

6. The patient demonstrates the ability to adhere to the principles of healthy dietary and activity habits.

3.3.b. We recommend against bariatric surgery for preadolescent children, for pregnant or breast-feeding adolescents, and for those planning to become pregnant within 2 yr of surgery; for any patient who has not mastered the principles of healthy dietary and activity habits; for any patient with an unresolved eating disorder, untreated psychiatric disorder, or Prader-Willi syndrome.

3.3. Evidence

Bariatric procedures for weight loss can be divided into malabsorptive, restrictive, and combination procedures. Purely malabsorptive procedures aim to decrease the functional length or efficiency of the intestinal mucosa through anatomic rearrangement of the intestine. These procedures include the jejunooileal bypass and the biliopancreatic diversion with duo
denal switch. Because of the high morbidity and mortality associated with these procedures, they cannot be recommended for use in children.

Laparoscopic adjustable gastric banding (LAGB) is a wholly restrictive procedure. It uses a prosthetic band to encircle and compartmentalize the proximal stomach into a small pouch and a large remnant. The Roux-en-Y gastric bypass (RYGB) is a combination procedure. It is a modification of gastric bypass and has become the most commonly performed bariatric surgical procedure. It involves dividing the stomach to create a small (15- to 30-ml) stomach pouch into which a segment of jejunum approximately 15 to 60 cm inferior to the ligament of Treitz is inserted, whereas the proximal portion of the jejunum that drains the bypassed lower stomach and duodenum is re-anastomosed 75 to 150 cm inferior to the gastrojejunostomy. This procedure combines the restrictive nature of gastrectomy with the consequences of dumping physiology with a negative conditioning response when high-calorie liquid meals are ingested. Although the LAGB procedure is considered safer than RYGB, the FDA has not yet approved LAGB for use in adolescents.

Bariatric surgery is an effective weight loss treatment for adults with a BMI above 40 kg/m², and there are sufficient data to calculate complication risks for adults. Similar data for adolescents are scarce. A health technology assessment for the Washington State Health Care Authority reviewed the data from 17 studies comprising 553 morbidly obese adolescent patients. A clinically significant weight loss was defined as a loss of 7% of body weight. This amount corresponded to a decrease in BMI of 4 U for patients undergoing RYGB (period of follow-up ranged from 1 to 6.3 yr) and a decrease of 3.5 U for patients undergoing LAGB (period of follow-up ranged from 1.7 to 3.3 yr). The conclusions reached in this assessment, mostly based on weak evidence, were that 1) both procedures resulted in clinically significant weight loss; 2) LAGB resolved the comorbid conditions of diabetes and hypertension, whereas RYGB resolved hypertension (there were insufficient data to rate the resolution of other comorbidities); 3) the safety profile (moderate evidence) for LAGB after a follow-up period of 1 to 85 months revealed no operative or postoperative deaths; 26 of 328 patients required reoperation to correct complications (band slippage, intragastric migration, and port/tubing problems); 4) the safety profile (moderate evidence) for RYGB after a follow-up period of 2 wk to 6 yr revealed a combination of mild (slight malnutrition) and severe (pulmonary embolism, severe malnutrition, postoperative bleeding, and gastrointestinal obstruction) complications.

We agree with the expert panels that suggest bariatric surgery for adolescents with obesity-related comorbid conditions that threaten the adolescent’s health—a BMI above 40 kg/m² and a severe comorbidity or a BMI above 50 kg/m² and less severe comorbidity. These cut-points are the ones generally accepted for adolescents. Others have
suggested that we should consider using a BMI in at least the 99th percentile—equivalent to an adult BMI of 35–40 kg/m²—with severe co-morbidities as a cut-point (228). There are insufficient data concerning the complication rates using the current cut-points to warrant suggesting any changes (224, 225, 229).

Gastric pacing studies are being done in adults (230), but no studies in children have been published.

3.3. Values and preferences

Our suggestion for limited use of bariatric surgery places a relatively higher value on avoiding anatomical and functional changes in developing children, on avoiding unforeseen complications associated with lifelong exposure to these changes, and on avoiding the costs and perioperative complications of these procedures. It places a relatively lower value on the weight loss and amelioration of obesity-related complications associated with bariatric surgery.

3.3. Remarks

**Requirements for patients.** It must be clear to the patient and the family that bariatric surgery is an adjunct to a sincere commitment to alteration of lifestyle and behavior rather than a cure. All obese children must first demonstrate their ability to adhere to a family-based dietary and lifestyle modification program.

**Requirements for preoperative care.** Bariatric surgery in adolescents should be performed in regional pediatric academic centers with programs equipped to handle the data acquisition, long-term follow-up, and multidisciplinary issues of these difficult patients (226). A multidisciplinary team with medical (including endocrine, gastrointestinal, cardiovascular, pulmonary, and otolaryngological expertise), surgical, nutritional, and psychological expertise should carefully select adolescents who are well informed and motivated as potential candidates for bariatric surgery and should provide preoperative care and counseling. Patients and families must be well informed as to the risks and complications of bariatric surgery.

**Requirements for postoperative care.** Postoperative attention to the principles of growth, development, and compliance is essential to avoid adverse physical, cognitive, and psychosocial outcomes after bariatric surgery (226). Adolescents undergoing bariatric surgery require lifelong medical and nutritional surveillance postoperatively (231), especially to ensure adequate vitamin and mineral intake, as well as extensive counseling. Patients lacking such help tend to regain their weight over time (232).

Finally, more data are required to assess the effects of and complications from bariatric surgery in adolescents, and clinicians are encouraged to enroll their patients into a national database. The National Institutes of Health has funded a multi-center study of bariatric surgery in adolescents (http://clinicaltrials.gov/ct2/show/NCT00474318). Criteria for patient enrollment include adolescents who have reached physical and psychological maturity, whose BMI is at least 40 kg/m² with significant obesity-related comorbidity, and who were unable to lose weight after 6 months of supervised participation in two separate behavioral or medical weight loss programs.

## 4.0. PREVENTION OF OBESITY

### 4.1. RECOMMENDATIONS FOR PREVENTING OBESITY

4.1.a. We recommend breast-feeding for a minimum of 6 months (1 ||| 1 ).

4.1.b. We suggest that clinicians promote and participate in efforts to educate children and parents by means of ongoing anticipatory guidance about healthy dietary and activity habits and, further, that clinicians encourage school systems to provide adequate health education courses promoting healthy eating habits (21 ||| 1 ).

4.1.c. We suggest that clinicians promote and participate in efforts to educate the community about healthy dietary and activity habits (21 ||| 1 ).

### 4.1. Evidence

The prime objective should be to prevent obesity...
before it happens, because once it develops, treatment is difficult (233–235). A meta-analysis commissioned by the Task Force (236) of randomized trials of lifestyle interventions to prevent pediatric obesity found significant but modest effects of these interventions on increasing physical activity ($P = 0.004$), decreasing sedentary behavior ($P < 0.00001$) with a significantly greater effect when directed toward children in contrast to adolescents ($P = 0.02$), and reducing unhealthy dietary habits ($P < 0.00001$). These beneficial effects did not translate into important changes in BMI (236). But, as discussed under 3.1.0. Remarks, weight maintenance in a growing child may be as effective as weight loss in an adult.

Breast-feeding in infancy is associated with a decreased incidence of overweight and obesity in childhood (237–241). Infants exclusively breast-fed for 3 to 5 months are 35% less likely to be obese when they enter school (242). A study of sibling pairs supports this finding (243). A meta-analysis demonstrated an inverse relationship between the duration of breast-feeding and the risk of becoming overweight with a plateau after 9 months of breast-feeding. The odds ratio for becoming overweight declines to 0.81 after 3 months of breast-feeding, to 0.76 after 6 months, and to 0.67 after 9 months of breast-feeding, after which it plateaus (244).

A review of 39 published school-based intervention studies designed to prevent childhood obesity showed that 40% of the 33,852 participating children had a positive effect on weight control, and the remainder had a neutral effect. None of the studies showed a negative effect (245). Exercise can play an important role for adolescents in the context of prevention of young adult obesity (246).

For most children and families, lifestyle patterns related to eating and exercise are established early, affecting children not only when they are young but also throughout life. To avoid the harmful health consequences of less-than-optimal lifestyle choices, it is incumbent upon health care providers to convey to their patients and their families healthy guidelines, explaining, in a culturally sensitive and language-appropriate manner, the caloric needs and essential nutrient requirements of young children, as well as the importance of physical activity. This is of particular importance when we consider the increased efficacy of prevention trials when directed toward children rather than adolescents (236).

Although healthy infants can differ considerably from one another in their caloric intake, appetite is the most efficient way to determine what an infant needs. Most infants instinctively know how much food they need and will not undereat or overeat unless pressured (247). Babies should be fed when hungry but should not be forced to finish all that they are served. Although low-fat and low-cholesterol diets are in vogue, they are not recommended for children under 2 yr of age.

4.1. Values and preferences

In making these recommendations, the Task Force placed a relatively high value on promoting these lifestyle preventive activities with their potential wide-reaching benefits (beyond weight control) and potential safety.

4.1. Remarks

Key players in the prevention and treatment of pediatric obesity are primary health care professionals (124, 233). Although anticipatory guidance is an important facet of pediatric care and it appears capable of having a salutatory effect on excessive weight gain (248), less than half of the recommended guidance is received (249). In some studies only 19% of primary care physicians were aware of the AAP recommendations on obesity and only 3% complied with all of the recommendations (250). The situation was little better in an academic medical center where charts of obese children documented the obesity only 53% of the time (251). Whereas a diet was prescribed for 71% of the obese children, increased physical activity was prescribed 33% of the time and reduced television time only 5% of the time; the recommended laboratory tests were obtained only 13% of the time. Although obesity was recognized in 76% of the obese adolescents, it was recognized only 31% of the time in preschoolers (251). This latter finding was particularly disappointing because prevention of overweight and obesity should be undertaken while
the condition is still mild and more readily treated by weight maintenance rather than weight loss. If a toddler or child is crossing BMI percentiles upwards (or weight-for-height percentiles), the primary care provider should begin to intervene by recommending reduced-fat milk and by restricting the intake of calorie-containing beverages such as juice as an initial step. If progress is not seen, then additional steps need to be undertaken (234, 235).

5.0. SOCIETAL BARRIERS TO IMPLEMENTATION

5.1. Recommendation
We suggest that clinicians advocate for regulatory policies designed to decrease the exposure of children and adolescents to the promotion of unhealthy food choices in the community (e.g. by media advertisements targeting children and adolescents) (214).

5.1. Evidence
Through advertising, especially on television, the food industry exerts an enormous influence on children that can negate the influence of parents and teachers. Although television can provide valuable educational programs, its advertisements for sweetened drinks, fast food restaurants, and high-calorie snacks can instill poor eating and leisure-time habits in children. In 2002 the food industry spent $10 to $12 billion on advertising directed toward children (Ref. 124, pp 153–192). In a survey of television programs aimed at children aged 2–11 and 12–17 yr, investigators found that 97.8 and 89.4%, respectively, of the advertisements were for food products consisting predominantly of nutrient-poor, high-sugar ingredients (252). A 30-sec ad can affect a child’s food choices (253). A 2006 Institute of Medicine report cited these negative factors as influencing children and adolescents to adopt unhealthy lifestyle choices (254).

5.2. Recommendation
We suggest that clinicians advocate that school districts ensure that only nutritionally sound food and drinks are available to children in the school environment, including the school cafeteria and alternative sources of food such as vending machines (214).

5.2. Evidence
The average child who participates in the school lunch program consumes one third of the daily recommended caloric intake in school and three fifths if breakfast is also taken in school (Ref. 124, pp 237–284). Of the 58 million schoolchildren in this country, about 28 million take part in the lunch program and 8 million in the breakfast program. Although federal standards require that these meals meet certain nutritional standards, the total fat composition of lunches exceeded the federal nutrition target of no more than 30% of total calories (Ref. 124, pp 237–284). As children move through the school system, fewer students participate in the school lunch program, and they consume a nutritionally poorer lunch obtained through alternative food sources. The problem largely lies in the availability of alternative food sources (termed “competitive foods”) in the school via snack bars, vending machines, or school-sponsored fund-raising sales. School districts have been tackling the issue by banning the sale of nutritionally poor food in their cafeterias, snack bars, and vending machines at school-sponsored events. These efforts have met resistance on the part of children, their parents (255), and school administrators who feared the loss of an important revenue source if children were to diminish their purchases of “competitive foods.” But in a 5-month post-ban study, schools did not suffer any loss in revenues from changed food and beverage sales (256). In several community studies, the fat content of meals decreased for those students exposed to a ban on junk food sales (Ref. 124, pp 237–284).

5.3. Recommendation
We suggest that clinicians advocate parental participation in the design of school-based dietary or physical activity programs and that schools educate parents about the rationale for these programs to ensure their understanding and cooperation (214).
5.3. Evidence

Even if schools institute health promotion programs and restrict the availability of unhealthy foods on site, these efforts may be contravened by cultural concepts and parental actions. Children may not be receptive to new, albeit healthy, foods, and persistence is required on the part of school authorities and parents (255). Adults, as well as children, may resist efforts to restrict unhealthy foods in schools. For example, when pastries and candies for at-school celebrations and birthdays were banned in one Texas school district, parents objected, and the Texas state legislature passed a measure prohibiting such a ban (257).

It is important that parents, as the primary caretakers, be educated about proper nutrition because the likelihood of the family’s consuming the recommended amount of fruits and vegetables increases not only with family income and general educational level, but also with the amount of nutrition education received (258).

5.4. Recommendation

We suggest that clinicians advocate that community master planners design, redesign, and organize communities to maximize opportunities for safe walking or cycling to school, recreational activity and athletic events, and neighborhood shopping as means to encourage greater physical activity (21 /desktop/).

5.4. Evidence

The physical and social organization of communities plays a role in the prevention and treatment of childhood obesity. Today, only 25% of children walk or bike to school as compared with 48% in 1969 (Ref. 124, pp 193–221).

Communities need to “provide places where children can play outside, particularly within their residential neighborhoods, and where they can safely walk, bike, or travel by other self-propelled means to destinations such as the park, playground, or school” (Ref. 124, pp 193–221). Some communities are now tackling this task as they adopt street layouts that suppress vehicular speed.

Parental concern about neighborhood crime and, therefore, their children’s safety may cause children to remain at home after school and to engage in sedentary rather than physical activities. Although the data on this particular correlation are inconsistent (Ref. 124, pp 193–221), studies show a correlation between parents’ perceptions of neighborhood safety and childhood obesity (259, 260).

Some localities have made an effort to promote the “Safe Routes to School” (SR25) initiative to increase the percentage of students walking or riding bicycles to school. Comprehensive SR25 programs have been successful where tried (261, 262).

5.5. Recommendation

We suggest that clinicians advocate that policymakers provide incentives to ensure that retailers can offer affordable, high-quality fresh fruits and vegetables to all (21  desktop/).

5.5. Evidence

For the poor, food expense represents an important item in a family’s budget (Ref. 124, pp 193–221). At least one study showed that a diet rich in fresh fruits, vegetables, whole grains, and protein costs more than a diet based on refined grains, added sugars, and fats (263). But a diet richer in fruits and vegetables can reduce caloric intake and lead to weight control (264). There are fewer conveniently located supermarkets in poorer neighborhoods but many “convenience stores” selling calorie-dense foods. Urban stores tend to stock fewer of the healthier foods and have less of a variety of foods (265). Even if supermarket availability were to improve, prices are still lower in the poorer neighborhoods for the higher fat, calorie-dense foods (266). Some policies (including the federal government farm subsidy program) have encouraged production of high-fructose corn syrup and other commodities used extensively in processed foods (267). Even when restaurants advertise and make healthy food items available, consumer habits may continue to prefer burgers, fries, and other high-calorie, high-fat items (268, 269).
References


70. Zlive L, Dan S, Phillip M 2003 Growth without growth hormone: growth pattern and final height of five patients with idiopathic combined pituitary hormone deficiency. Clin Endocrinol (Oxf) 59:82–88


82. Cole TJ 2004 Children grow and horses race: is the adiposity rebound a critical period for later obesity? BMC Pediatr 4:6
100. Weiss R 2006 Insulin sensitivity and secretion: swaying the pendulum. J Pediatr 148:3–4
32


110. Phelan S, Wyatt HR, Hill JO, Wing RR 2006 Are the eating and exercise habits of successful weight losers changing? Obesity (Silver Spring) 14:710–716


117. Rhodes ET, Ludwig DS 2007 Childhood obesity as a chronic disease: keeping the weight off. JAMA 298:1695–1696


130. Levine AA 1997 Excessive fruit juice consumption: how can something that causes failure to thrive be associated with obesity? J Pediatr 96:187–193


181. Pate RR, Davis MG, Robinson TN, Stone EJ, McKenzie TL, Young JC 2006 Promoting physical activity in children and youth: a leadership role for schools: a scientific statement from the American Heart Association Council on Nutrition, Physical Activity, and Metabolism (Physical Activity Committee) in collaboration with the Councils on Cardiovascular Disease in the Young and Cardiovascular Nursing, Circulation 114:1214–1224


185. Robinson TN 1999 Reducing children’s television viewing to prevent obesity: a randomized controlled trial. JAMA 282:1561–1567


243. Nelson MC, Gordon-Larsen P, Adair LS 2005 Are adolescents who were breast-fed less likely to be overweight? Analyses of sibling pairs to reduce confounding. Epidemiology 16:247–253


257. Mui Y 2005 At many elementary schools, the party’s over. Washington Post, October 30, 2005; C01


269. Pressler MW 2005 Hold the health, serve that burger. Washington Post, August 18, 2005; A01
Acknowledgments

The members of the Task Force thank Dr. Robert Vigersky and the Clinical Guidelines Subcommittee, the Clinical Affairs Core Committee, the Executive Committee of The Endocrine Society, and the Obesity Task Force of the Lawson Wilkins Pediatric Endocrine Society for review of the earlier versions of this guideline and for their suggestions, which were incorporated into the final document. We also thank the members of The Endocrine Society and the Lawson Wilkins Pediatric Endocrine Society who contributed additional comments. We particularly thank Dr. Patricia A. Stephens, the medical writer for this guideline, for checking the references and formatting the manuscript. We also thank the support staff of The Endocrine Society, without whose assistance this guideline could not have been produced.

Financial Disclosure of Task Force

Gilbert P. August, M.D. (chair)—Significant Financial Interests: none declared; Governance: none declared; Consultation or Advisement: none declared; Grant or Other Research Support: none declared. Sonia Caprio, M.D.—Significant Financial Interests: none declared; Governance: none declared; Consultation or Advisement: none declared; Grant or Other Research Support: none declared. Ilene Fennoy, M.D., MPH—Significant Financial Interests: none declared; Governance: none declared; Consultation or Advisement: Novo Nordisk Turner’s Syndrome Advisory Committee (2005); Grant or Other Research Support: Novo Nordisk Answer Program, Serono—Cod Click Adolescent Transition Study, Unimed—Androgel in Male Hypogonadal Puberty Study. Michael Freemark, M.D.—Significant Financial Interests: none declared; Governance: none declared; Consultation or Advisement: none declared; Grant or Other Research Support: none declared. Francine R. Kaufman, M.D.—Significant Financial Interests: none declared; Governance: none declared; Consultation or Advisement: Medtronic, Lifescan, Omnipod, Mannkind, Novo Nordisk; Grant or Other Research Support: GSK, Medtronic. Robert H. Lustig, M.D.—Significant Financial Interests: none declared; Governance: none declared; Consultation or Advisement: Novo Nordisk Pharmaceuticals; Grant or Other Research Support: EndoVx, Inc., Novartis Pharmaceuticals Corp. Janet H. Silverstein, M.D.—Significant Financial Interests: none declared; Governance: none declared; Consultation or Advisement: Advisory board member for the Genentech Center for Clinical Research in Endocrinology; Grant or Other Research Support: none declared. Phyllis W. Speiser, M.D.—Significant Financial Interests: none declared; Governance: none declared; Consultation or Advisement: none declared; Grant or Other Research Support: Speakers Bureau—Lilly, Pfizer, and Investigator—Lilly, Genetech, Novo Nordisk, Pfizer, Serono. Dennis M. Styne, M.D.—Significant Financial Interests: none declared; Governance: none declared; Consultation or Advisement: Healthy Living Academy; Grant or Other Research Support: none declared. *Victor M. Montori, M.D.—Financial or Business/Organizational Interests: none declared; Significant Financial or Leadership Position: none declared; Consultation/Advisement: KER Unit (Mayo Clinic).

*Evidence-based reviews for this guideline were prepared under contract with The Endocrine Society.
# The Endocrine Society Guideline Order Form

(Single reprint request for orders of 100 and less)

<table>
<thead>
<tr>
<th>PRODUCTS</th>
<th>QTY</th>
<th>PRICE (USD)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Androgen Therapy in Women</td>
<td></td>
<td>$15.00</td>
</tr>
<tr>
<td>Case Detection, Diagnosis, and Treatment of Patients with Primary</td>
<td></td>
<td>$15.00</td>
</tr>
<tr>
<td>Aldosteronism</td>
<td></td>
<td>$15.00</td>
</tr>
<tr>
<td>The Diagnosis of Cushing’s Syndrome</td>
<td></td>
<td>$15.00</td>
</tr>
<tr>
<td>Evaluation &amp; Treatment of Adult Growth Hormone Deficiency</td>
<td></td>
<td>$15.00</td>
</tr>
<tr>
<td>Evaluation &amp; Treatment of Hirsutism in Premenopausal Women</td>
<td></td>
<td>$15.00</td>
</tr>
<tr>
<td>Management of Thyroid Dysfunction during Pregnancy and Postpartum</td>
<td></td>
<td>$15.00</td>
</tr>
<tr>
<td>Prevention and Treatment of Pediatric Obesity</td>
<td></td>
<td>$15.00</td>
</tr>
<tr>
<td>Testosterone Therapy in Adult Men with Androgen Deficiency Syndromes</td>
<td></td>
<td>$15.00</td>
</tr>
<tr>
<td>Primary Prevention of Cardiovascular Disease and Type 2 Diabetes</td>
<td></td>
<td>$15.00</td>
</tr>
<tr>
<td>Evaluation &amp; Treatment of Adult Growth Hormone Deficiency</td>
<td></td>
<td>$15.00</td>
</tr>
<tr>
<td>Executive Summary (MMTD07)—Guideline (MTSD07)—Executive Summary</td>
<td></td>
<td>$10.00</td>
</tr>
<tr>
<td>Prevention and Treatment of Pediatric Obesity</td>
<td></td>
<td>$20.00</td>
</tr>
<tr>
<td>Testosterone Therapy in Adult Men with Androgen Deficiency Syndromes</td>
<td></td>
<td>$20.00</td>
</tr>
<tr>
<td>Primary Prevention of Cardiovascular Disease and Type 2 Diabetes</td>
<td></td>
<td>$20.00</td>
</tr>
<tr>
<td>Miscellaneous</td>
<td></td>
<td>$20.00</td>
</tr>
</tbody>
</table>

Subtotal: All prices include sales tax $80.00

**Payment Information:**

- [ ] Check
- [ ] MasterCard
- [ ] Visa

Card Number: __________________________________________________________
Expiration Date: ______________________________________________________
Billing Address: _______________________________________________________
Signature: _____________________________________________________________

Are you a member of The Endocrine Society? [ ] Yes [ ] No
If you are a member and you know your member ID, please provide: __________

Prefix: __________________________ First Name (Given): ____________________
Middle: _________________________ Last (Surname): _________________________

Institution/Company: __________________________ Dept/Div: ________________
Street/PO: __________________________
City: __________________________ State/Province: __________________________
Telephone: ______________________ Fax: __________________________ Email: __________

Degree(s) that you would like listed after your name: ______________________
Professional Title: __________________________ Date of Birth: ______________
Gender: [ ] Male [ ] Female

Which of the following best describes your primary professional role? (Please mark only one)

- [ ] Administrator
- [ ] Basic Researcher
- [ ] Clinical Practitioner
- [ ] Clinical Researcher
- [ ] Industry/Corporate Professional
- [ ] Nurse/Healthcare Professional
- [ ] Retired
- [ ] Teacher/Educator
- [ ] Fellow (Clinical)
- [ ] Fellow (Postdoctoral/Research)
- [ ] Student
- [ ] Other __________________________

Race or Ethnic Affiliation (voluntary)

- [ ] African American, Black
- [ ] Asian
- [ ] Hispanic
- [ ] Native American, Eskimo, Aleut
- [ ] Pacific Islander
- [ ] White, Caucasian
- [ ] Other __________________________
What goes into our Clinical Guidelines is a story worth telling

The extensive process that goes into creating The Endocrine Society’s Clinical Guidelines not only provides validation and assurance, but also raises the standard for the development of guidelines everywhere.

The guidelines are developed using a multi-step process that reflects the standards of excellence embraced by The Endocrine Society.

Endocrine Society Clinical Guidelines Also Available:

- Evaluation and Treatment of Adult Growth Hormone Deficiency
- Testosterone Therapy in Adult Men with Androgen Deficiency Syndromes
- Androgen Therapy in Women
- Management of Thyroid Dysfunction during Pregnancy and Postpartum
- Evaluation and Treatment of Hirsutism in Premenopausal Women
- The Diagnosis of Cushing’s Syndrome
- Case Detection, Diagnosis, and Treatment of Patients with Primary Aldosteronism
- Primary Prevention of Cardiovascular Disease and Type 2 Diabetes in Patients at Metabolic Risk

Endocrine Society Clinical Guidelines Coming Soon:

- Evaluation and Management of Adult Hypoglycemic Disorders
- Endocrine Treatment of Transsexual Persons
- Managing Patients Post-Bariatric Surgery
- Vitamin D & Bone
- Continuous Glucose Monitoring
- Congenital Adrenal Hyperplasia
- Pituitary Incidentallyoma
- Hypertriglyceridemia
- Hyperprolactinemia
- Intensive Insulin Therapy of Hospital Patients
- Management of Anabolic Drugs for Osteoporosis in Men

To purchase the available guidelines visit:

To view patient guides (companion pieces to the clinical guidelines), visit The Hormone Foundation’s Web site at www.hormone.org/public/patientguides.cfm.
Authors: Gilbert P. August, Sonia Caprio, Ilene Fennoy, Michael Freemark, Francine R. Kaufman, Robert H. Lustig, Janet H. Silverstein, Phyllis W. Speiser, Dennis M. Styne, and Victor M. Montori

Co-sponsoring Association: Lawson Wilkins Pediatric Endocrine Society

Affiliations: Professor Emeritus of Pediatrics, George Washington University School of Medicine (G.P.A.), Washington, D.C.; Yale University School of Medicine (S.C.), New Haven, Connecticut; Columbia University (I.F.), New York, New York; Duke University Medical Center (M.F.), Durham, North Carolina; Children’s Hospital of Los Angeles (F.R.K.), Los Angeles, California; University of California San Francisco (R.H.L.), San Francisco, California; University of Florida (J.H.S.), Gainesville, Florida; Schneider Children’s Hospital (P.W.S.), New Hyde Park, New York; University of California-Davis Medical Center (D.M.S.), Sacramento, California; and Mayo Clinic (V.M.M.), Rochester, Minnesota

Disclaimer Statement: Clinical Practice Guidelines are developed to be of assistance to endocrinologists by providing guidance and recommendations for particular areas of practice. The Guidelines should not be considered inclusive of all proper approaches or methods, or exclusive of others. The Guidelines cannot guarantee any specific outcome, nor do they establish a standard of care. The Guidelines are not intended to dictate the treatment of a particular patient. Treatment decisions must be made based on the independent judgment of health care providers and each patient’s individual circumstances.

The Endocrine Society makes no warranty, express or implied, regarding the Guidelines and specifically excludes any warranties of merchantability and fitness for a particular use or purpose. The Society shall not be liable for direct, indirect, special, incidental, or consequential damages related to the use of the information contained herein.

Evidence-based reviews for this guideline were prepared under contract with The Endocrine Society.


© The Endocrine Society, 2008
Prevention and Treatment of Pediatric Obesity:
An Endocrine Society Clinical Practice Guideline Based on Expert Opinion