

PEDIATRICS®

OFFICIAL JOURNAL OF THE AMERICAN ACADEMY OF PEDIATRICS

Assessment of Child and Adolescent Overweight and Obesity

Nancy F. Krebs, John H. Himes, Dawn Jacobson, Theresa A. Nicklas, Patricia Guilday and Dennis Styne

Pediatrics 2007;120;S193-S228

DOI: 10.1542/peds.2007-2329D

The online version of this article, along with updated information and services, is located on the World Wide Web at:

http://www.pediatrics.org/cgi/content/full/120/Supplement_4/S193

PEDIATRICS is the official journal of the American Academy of Pediatrics. A monthly publication, it has been published continuously since 1948. PEDIATRICS is owned, published, and trademarked by the American Academy of Pediatrics, 141 Northwest Point Boulevard, Elk Grove Village, Illinois, 60007. Copyright © 2007 by the American Academy of Pediatrics. All rights reserved. Print ISSN: 0031-4005. Online ISSN: 1098-4275.

American Academy of Pediatrics

DEDICATED TO THE HEALTH OF ALL CHILDREN™



Assessment of Child and Adolescent Overweight and Obesity

Nancy F. Krebs, MD, MS^a, John H. Himes, PhD, MPH^b, Dawn Jacobson, MD, MPH^c, Theresa A. Nicklas, DrPH^d, Patricia Guilday, RN^e, Dennis Styne, MD^f

^aDepartment of Pediatrics, University of Colorado School of Medicine, Denver, Colorado; ^bDivision of Epidemiology and Community Health, University of Minnesota School of Public Health, Minneapolis, Minnesota; ^cOffice of Disease Prevention and Health Promotion, Department of Health and Human Services, Rockville, Maryland; ^dDepartment of Pediatrics, Baylor College of Medicine, Houston, Texas; ^ePS duPont Elementary School, Wilmington, Delaware; ^fDepartment of Pediatrics, University of California, Davis, Sacramento, California

The authors have indicated they have no financial relationships relevant to this article to disclose.

ABSTRACT

Accurate appropriate assessment of overweight and obesity in children and adolescents is a critical aspect of contemporary medical care. However, physicians and other health care professionals may find this a somewhat thorny field to enter. The BMI has become the standard as a reliable indicator of overweight and obesity. The BMI is incomplete, however, without consideration of the complex behavioral factors that influence obesity. Because of limited time and resources, clinicians need to have quick, evidence-based interventions that can help patients and their families recognize the importance of reducing overweight and obesity and take action. In an era of fast food, computers, and DVDs, it is not easy to persuade patients to modify their diets and to become more physically active. Because research concerning effective assessment of childhood obesity contains many gaps, this report is intended to provide a comprehensive approach to assessment and to present the evidence available to support key aspects of assessment. The discussion and recommendations are based on >300 studies published since 1995, which examined an array of assessment tools. With this information, clinicians should find themselves better equipped to face the challenges of assessing childhood overweight and obesity accurately.

www.pediatrics.org/cgi/doi/10.1542/peds.2007-2329D

doi:10.1542/peds.2007-2329D

Key Words

obesity, clinical assessment, BMI, diet, physical activity

Abbreviations

IOTF—International Obesity Task Force
CDC—Centers for Disease Control and Prevention

PPV—positive predictive value
DGAC—Dietary Guidelines Advisory Committee

HDL—high-density lipoprotein

LDL—low-density lipoprotein

T2DM—type 2 diabetes mellitus

CVD—cardiovascular disease

WAVE—weight, activity, variety, and excess

Accepted for publication Aug 31, 2007

Address correspondence to Nancy F. Krebs, MD, MS, Department of Pediatrics, University of Colorado School of Medicine, 4200 E. Ninth Ave, Box C225, Denver, CO 80262. E-mail: nancy.krebs@uchsc.edu

PEDIATRICS (ISSN Numbers: Print, 0031-4005; Online, 1098-4275). Copyright © 2007 by the American Academy of Pediatrics

OBESSE AND OBESITY are terms commonly used in the clinic as well as on the street corner, often with a wide range of meanings. For medical purposes, obesity refers to excess body fat; however, the exact meaning of excess has not been defined. Obesity most often is regarded as an excess percentage of body weight that is fat, but no widely accepted diagnostic definitions or cutoff points are available for children. For an understanding of developmental patterns, mean body fat percentages (derived from bioelectrical impedance analyses) are available for US children >12 years of age,¹ and percentile curves have been published for British children 5 to 18 years of age.²

MEASUREMENTS OF OVERWEIGHT AND OBESITY

Defining Obesity

In the absence of established criteria that define childhood obesity on the basis of whole-body fatness and its relationship to health outcomes, cutoff points based on distributions of anthropometric measurements (eg, weight and BMI) generally are used. To define obesity in US children, the percentile distributions relative to gender and age in the Centers for Disease Control and Prevention (CDC) 2000 growth charts³ are now the preferred reference. The CDC 2000 growth charts were not developed as health-related standards of how healthy children should grow. Rather, the growth charts present percentiles as points of reference, primarily based on national surveys of US children. Although the most-recent growth charts were published in 2000, they include selected data from 1963 to 1995, which makes them statistically nonrepresentative of the US population in 2000.³ Nevertheless, the CDC 2000 growth charts were developed carefully and provide the best reference data available for the growth of US children.

Several expert and advisory groups have recommended BMI as the preferred measure for evaluating obesity among children and adolescents 2 to 19 years of age.⁴⁻⁷ BMI expresses the weight-for-height relationship as a ratio, that is, weight (in kilograms)/[height (in meters)]². Experts recommend BMI because it can be obtained easily, it is correlated strongly with body fat percentage (especially at extreme BMI levels), it is associated only weakly with height, and it identifies the fattest individuals correctly, with acceptable accuracy at the upper end of the distribution (eg, ≥ 85 th or ≥ 95 th percentile for age and gender).

In 1994, the Expert Committee on Clinical Guidelines for Overweight in Adolescent Preventive Services recommended that children whose BMI exceeds 30 kg/m² or is ≥ 95 th percentile for age and gender (whichever is smaller) should be considered overweight.⁵ The BMI limit of 30 kg/m² was recommended because, at the oldest ages of adolescence for which the 95th percentile values exceed 30 kg/m² (>17 years), elevated BMI is associated with early

adulthood patterns of risk for obesity-related disease and death,^{8,9} as well as to provide continuity with recommendations for adults. The expert committee considered adolescents whose BMI was ≥ 85 th percentile but <95th percentile to be "at risk of overweight." The committee deliberately avoided the term obese, because of the inference regarding adiposity and body composition and the inability of height and weight data, even as BMI, to measure total body fat specifically. These definitions are considered standard in describing the weight status of children and adolescents 2 to 18 years of age.^{4,10}

In 2005, the Institute of Medicine consciously departed from the previously described terminology and elected to define children with BMI of ≥ 95 th percentile for age and gender as obese, rather than overweight.⁶ The Institute of Medicine report conveyed the seriousness, urgency, and medical nature of childhood obesity, as well as the need to take action. The current expert committee endorses the position of the Institute of Medicine report and recommends that individuals 2 to 18 years of age with BMI of >30 kg/m² or ≥ 95 th percentile for age and gender (whichever is smaller) should be considered obese (see summary report). We think that the nature of the current epidemic and the need for medical professionals and others to address the problem actively justifies this change.¹¹ Moreover, we recommend that individuals with BMI of ≥ 85 th percentile but <95th percentile or 30 kg/m² (whichever is smaller) now be considered overweight and that this term replace the term "at risk of overweight." The Institute of Medicine report published in 2005 was silent regarding this category of BMI.

The expert committee concluded that the scientific data linking elevated BMI to risk factors and morbidity,^{12,13} as well as the difficulty of changing early trajectories of weight gain, support the change in terminology. The terms overweight and obese also may be easier than "at risk of overweight" for parents to understand. This new terminology will allow US medical practice to parallel the recommendations of the International Obesity Task Force (IOTF)¹⁴ and to align with the *International Classification of Diseases, Ninth Edition*, diagnosis codes. Finally, these changes in descriptions of weight status for children and adolescents will provide continuity with the recommended adult cutoff points of BMI of ≥ 25 kg/m² and ≥ 30 kg/m² for overweight and obesity, respectively.¹⁵ We are aware that the BMI categories for adults that are linked to minimal subsequent mortality rates are not without controversy.¹⁶ However, the threshold levels of 25 and 30 kg/m² for adult BMI are still recommended by national and international organizations.^{7,15}

For children ≤ 2 years of age, the weight-for-recumbent length percentiles from the CDC 2000 growth charts are appropriate for evaluating weight relative to linear growth, but the term obese generally should not be applied to children this young. Weight-for-length

percentiles of ≥ 95 th identify these children as overweight.³

Little evidence is available regarding the most effective way to evaluate the severity of obesity for children with BMI of >97 th percentile (the highest level on the CDC growth charts). Inge et al¹⁷ recommended that bariatric surgery for adolescents should be restricted to those with BMI of ≥ 40 and significant comorbidities that may be improved with surgery. In research settings, age-specific z scores or SD scores are used for extreme values of anthropometric measures. These scores describe the number of SD units above or below the median for the individual value. For example, in a normally distributed population, the 99th percentile is equivalent to a z score of ~ 3.0 . Unfortunately, a computer program is needed to calculate BMI z scores, and many clinicians are unfamiliar with their use and interpretation. A BMI z score calculator is available on the Internet (www.kidsnutrition.org/bodycomp/bmiz2.html). Limited available data suggest that BMI-for-age values of ≥ 99 th percentile are associated strongly with the presence of comorbidities, excess adiposity, and persistence of obesity into adulthood.¹⁸ This severity of obesity may well warrant more-aggressive therapeutic interventions. Although they are not available currently on growth charts, more-routine availability of 99th percentile BMI cutoff points would likely be valuable for tailoring optimal treatment approaches.

Anthropometric Methods and Determination of BMI

Weight, height (sometimes referred to as stature), and recumbent length of children are measured routinely in most clinics. Nevertheless, the importance of careful accurate measurements should be emphasized to clinic staff members. Staff members should take particular care when BMI is calculated, compared to reference data, and made the basis for important decisions regarding the child's health. Detailed protocols are available for measuring recumbent length, height, and weight in a manner comparable to that for reference data.¹⁹

BMI may be calculated directly as weight (in kilograms)/[height (in meters)]² or determined from published tables or nomograms.^{5,20,21} Many BMI tables, nomograms, and calculator programs are available online (eg, www.cdc.gov/nccdphp/dnpa/bmi/calc-bmi.htm or <http://nhlbisupport.com/bmi/bmicalc.htm>). The National Heart, Lung, and Blood Institute provides a free program for calculating BMI on hand-held devices (http://hp2010.nhlbi.nih.gov/bmi_palm.htm). If BMI is calculated from height and weight measured in inches and pounds, then the formula is $BMI = [\text{weight (in pounds)} / [\text{height (in inches)}]^2] \times 703$. Some BMI tables and charts are designed for adults and either may not accommodate the smaller heights and weights appropriate for children or may not provide age/gender-specific percentiles.

Development of BMI References and Implications

The developmental pattern for BMI differs somewhat from the more-familiar patterns for height and weight (Fig 1). The normal pattern is for BMI to decrease from ~ 2 years of age until 5 or 6 years of age and to increase thereafter. This early decrease in BMI reflects a corresponding decrease in subcutaneous fat and the percentage of body fat.²² The resulting V-shaped pattern in early childhood has been termed the "adiposity rebound."²³ It coincides with the period between the ages of 4 and 7 years when BMI reaches its nadir and then begins to increase through the remainder of childhood and into young adulthood. Early adiposity rebound has been cited as a risk factor for the development and persistence of later obesity.²⁴ More-recent analyses suggest that this primarily is a reflection of rapid weight gain during infancy and early childhood and that it identifies young children with high BMI percentiles and/or children who are crossing percentiles upward.^{24,25} Rapid weight gain in infancy, including during the first week,²⁶ the first 4 months,²⁷ and the first year,²⁸ has been found to predict later obesity. In one prospective cohort, increased weight gain during the first 3 years of life was associated independently with higher BMI, fat mass, and waist circumference at 17 years of age.²⁹ For clinical purposes, the utility of assessing adiposity rebound is limited, because it is difficult to determine for an individual child and it is, by definition, a retrospective determination. Identification of the age of adiposity rebound as a strategy for clinicians to identify children at risk of overweight or obesity is unlikely to contribute more than plotting of weight and length for age and determination of BMI percentiles for young children.

Another distinctive feature of the BMI developmental curve is that it lacks the marked increase in growth velocity during the adolescent spurt that is characteristic of height and weight growth curves. Although BMI increases during the adolescent spurt, the slope with age is dampened by the nature of the BMI ratio and the difference in timing of the growth spurts of height and weight.²² Because the upper percentiles of BMI increase so dramatically with age, the BMI levels used to identify overweight and obese children are usually presented according to age. BMI percentiles also must be gender-specific, because of the systematic physiologic differences between boys and girls. Finally, as is evident in the percentiles presented in Fig 1, the statistical distribution of BMI at any age is asymmetric or skewed toward the higher values.

An attractive aspect of BMI is that it correlates closely with total body fat^{30,31} and other risk factors for obesity-related morbidity in adults.^{32,33} Such correlations are based on the joint associations of the entire distributions of BMI and related outcomes. Interpretation of assessments of overweight in children using only BMI for age and gender should include the realization that some

NAME _____
RECORD # _____

This sort of categorical identification often is evaluated by using the same sensitivity/specificity approach that is used to evaluate medical screening procedures.³⁶ Sensitivity (or the true-positive rate) in this case represents the proportion of children who are considered the fattest with a standard method for assessment of total body fat (eg, dual-energy X-ray absorptiometry) and who also are identified correctly with the BMI criteria. The complement of sensitivity is specificity, or the proportion of children who are considered not the fattest with the standard method and who are identified correctly as not overweight or obese with the BMI criteria. Finally, the positive predictive value (PPV) is the pro-

portion of children who are identified as overweight or obese with BMI criteria who are truly the fattest children identified with the standard method. The PPV is important for clinical applications because its complement ($1 - \text{PPV}$) is an estimate of the proportion of children who may be identified incorrectly as overweight or obese when BMI is used. Such children may be labeled, treated, or referred inappropriately.³⁷

The sensitivities of the 85th BMI percentiles on the CDC 2000 growth charts in identifying correctly the fattest children range from 75% to 93% in several studies, and the corresponding specificities range from 67% to 96%.^{30,38,39} The accompanying PPVs (presented or calculated from sensitivity, specificity, and prevalence values) range from 61% to 98%. The sensitivities of the 95th BMI percentiles on the CDC charts in identifying correctly the fattest children range from 54% to 100%, and the corresponding specificities range from 96% to 99%.^{38,39} PPVs for the ≥ 95 th BMI percentile criterion range from 56% to 99%.

Some of the aforementioned estimates of sensitivity, specificity, and PPV are difficult to compare directly across studies, because of the differing samples and standard criteria used. Nevertheless, several general conclusions can be drawn from these and similar studies. Most important is that the BMI criteria, although imperfect, perform reasonably well in identifying correctly children who have the highest percentages of body fat. As the BMI criteria become more restrictive (ie, ≥ 95 th percentile versus ≥ 85 th percentile), the sensitivities in identifying the fattest children decrease and the specificities increase. Finally, the specificities and PPVs are almost always higher than the corresponding sensitivities. This means that there should be relatively few children diagnosed incorrectly as overweight or obese by using BMI.

The CDC 2000 BMI-for-age percentiles are recommended for US children from all racial/ethnic backgrounds. Some evidence exists that, in general, black children tend to have relatively less body fat and Mexican American children tend to have relatively more body fat, compared with white children with the same BMI.⁴⁰ Also, South Asian adolescents living in England have higher percentages of body fat than do their peers of European heritage with the same BMI.⁴¹ Because the racial/ethnic differences in body fat/BMI relationships have not been described fully for children, however, the same BMI reference values are currently recommended for assessment of all children. Any racial/ethnic differences in health risks assessed by using the CDC BMI reference values should be small.

The validity of using high BMI to identify children with the highest total body fat levels seems to be approximately the same for healthy children and children receiving growth hormone, children with inflammatory bowel disease, and children treated previously for malignancy.⁴² Differences between boys and girls in the

sensitivities and specificities of BMI for identifying the fattest children are inconsistent and probably not important clinically.^{30,39,43} Similarly, available data show no consistent age patterns in BMI sensitivities and specificities between the ages of 6 and 18 years.

Pediatric obesity is associated with increased risks of concomitant psychological or psychiatric problems, cardiovascular risk factors, chronic inflammation, type 2 diabetes mellitus (T2DM), and asthma.^{33,44} In an important study, Katzmarzyk et al⁴⁵ assessed the validity of BMI and waist circumference criteria for overweight and obesity for identifying correctly youths 5 to 18 years of age who had ≥ 3 of 6 risk factors (low high-density lipoprotein [HDL] cholesterol levels, high low-density lipoprotein [LDL] cholesterol levels, high triglyceride levels, high plasma glucose levels, high plasma insulin levels, or high blood pressure). The overall sensitivities and specificities for BMI of ≥ 85 th percentile were 69% and 76%, respectively, and those for BMI of ≥ 95 th percentile were 49% and 90%, respectively. These sensitivities and specificities are quite low, and the corresponding PPVs calculated from the authors' data are 36% and 50%, respectively, for BMI of ≥ 85 th and ≥ 95 th percentiles. Therefore, even among children with ≥ 3 risk factors, the least-restrictive and therefore most-sensitive BMI cutoff point (BMI of ≥ 85 th percentile) still identified correctly only approximately two thirds. Moreover, of all children with BMI of ≥ 85 th percentile who were considered overweight, approximately two thirds did not have ≥ 3 risk factors. The authors concluded that waist circumference added substantially to BMI alone for assessment of cardiovascular disease (CVD) risk.⁴⁵ If these results can be replicated in other samples, they argue strongly that BMI criteria by themselves are insufficient to identify children who are most likely to have clusters of risk factors and that additional screening and assessment criteria should be applied to estimate risks.

Implications for Overweight and Obese Children in Adulthood

Systematic reviews confirm the persistence of obesity from childhood into adulthood.⁴⁶ Predictably, the higher the BMI is in childhood, the greater the probability is of obesity in adulthood. Guo et al⁴⁷ analyzed lifelong data from the Fels Longitudinal Study and estimated the probabilities of having a BMI of $\geq 30 \text{ kg/m}^2$ at 35 years of age. For girls with BMI of 95th percentile during childhood, the probabilities of being obese as an adult were 20% to 39.9% from 3 to 5 years of age, 40% to 59.9% from 6 to 11 years of age, and $\geq 60\%$ from 12 to 20 years of age. For boys with BMI of 95th percentile during childhood, the probabilities of being obese as an adult were $<20\%$ from 3 to 4 years of age, 20% to 39.9% from 5 to 11.5 years of age, 40% to 59.9% from 11.5 to 16 years of age, and $\geq 60\%$ from 17 to 20 years of age.

For children with BMI of 85th percentile during childhood, the probabilities of adult obesity were lower. For girls, the probabilities of being obese as an adult were

<20% from 3 to 4 years of age, 20% to 39.9% from 5 to 17 years of age, and 40% to 59.9% from 18 to 20 years of age. For boys with BMI of 85th percentile during childhood, the probabilities of being obese as an adult were <20% from 3 to 16 years of age, 20% to 39.9% at 17 years of age, and 40% to 59.9% from 18 to 20 years of age. On the basis of these data, the odds ratios for being obese (BMI of ≥ 30 kg/m²) at 35 years of age were 19.3 for boys and 15.7 for girls if BMI at 18 years of age was >72nd percentile (the most discriminating level). Clearly, if individuals end their adolescence with moderately elevated BMI, then the likelihood of obesity as an adult is high.

Overweight and obesity in childhood and adolescence have been associated with adverse socioeconomic outcomes, increased health risks and morbidities, and increased mortality rates in adulthood.^{32,33,46} Must et al⁴⁸ studied children in Boston (13–18 years of age) who were evaluated initially between 1922 and 1935 and were assessed in 1988. Compared with those with BMI of 25th to 50th percentile in adolescence, those with BMI of >75th percentile in adolescence had increased heart disease, atherosclerosis, T2DM, colorectal cancer (men), gout (men), hip fracture (women), arthritis (women), and all-cause mortality (men) rates.

Alternative Reference Data and Measures of Fatness

IOTF Standards

In 2000, reference BMI categories based on 6 pooled international data sets were developed for children 2 to 18 years of age.¹⁴ These reference curves have become known as the IOTF standards. They assume that the most-appropriate cutoff points for overweight and obesity in children are those corresponding to the locations of BMI of 25 kg/m² and 30 kg/m², respectively, in the BMI distribution for adults, points that are recognized internationally as defining overweight and obesity.⁴⁹ Particularly outside the United States, the IOTF standards have been widely used to classify overweight and obesity in children. The IOTF charts provide only overweight and obesity categories and not a full array of percentile levels. Therefore, they are not recommended for monitoring the BMI progress of individual children. Sensitivities and specificities of IOTF cutoff points in identifying the fattest children and predicting adult morbidity are similar to those of the CDC 2000 BMI charts.^{39,50} The IOTF reference values are not recommended for routine clinical use.

In various research settings and in the scientific literature, measures other than BMI that are related to childhood fatness and obesity are used frequently. These were investigated, and consideration was given to their appropriateness for routine clinical use in the assessment of pediatric overweight and obesity, as well as whether

each provides important information beyond that available from BMI.

Skinfold Thickness

Skinfolds are double, compressed thicknesses of subcutaneous fat and skin that are measured with standardized calipers at selected sites (eg, triceps, subscapular, and suprailiac sites).⁵¹ Skinfold measurements have a long history in studies of nutrition and body composition. They are considered attractive research tools because measurements are noninvasive and specific to subcutaneous fat.⁵² Previous expert committees considering childhood obesity recommended that skinfold measurements be included in in-depth medical assessments, to distinguish those who are overweight from those who are overfat.^{5,53}

Without question, skinfold thicknesses are predictive of total body fat in children and adolescents.^{54,55} Moreover, when skinfold measurements are included in regression models, they provide unique information beyond height and weight in accounting for variations in risk indicators, including blood lipid levels, lipoprotein levels, blood pressure, plasma glucose levels, plasma insulin levels, insulin resistance, and inflammation.^{13,56–59}

When categories of skinfold thicknesses or ratios based on percentile cutoff points are used to identify the fattest individuals or those with metabolic syndrome, the skinfold measurements perform as well as BMI or waist circumference values.^{60–63} Nevertheless, there is little evidence that, once height and weight (or BMI) are known, skinfold thickness categories increase the accuracy of identifying those with the most total body fat or other risk factors.

Therefore, the expert committee does not recommend the routine clinical use of skinfold thickness measurements in the assessment of childhood obesity. The basis for this conclusion includes the lack of readily available reference data on skinfold thicknesses for US children, the considerable potential for measurement errors without rigorous training and regular experience,^{51,55} and the lack of optimal criteria as a basis for intervention.

Waist Circumference

Waist circumference has attracted much recent attention as an indicator of fatness and health risks in children and adults. The interest in waist circumference stems from research linking accumulated visceral adipose tissue to increased health risks and metabolic disorders in children and adults.^{45,64,65}

Compared with BMI, waist circumference in children provides a better estimate of visceral adipose tissue measured with MRI at the level of the fourth lumbar vertebra (65% vs 56% of variance), whereas BMI is better at estimating subcutaneous adipose tissue (89% vs 84% of variance).⁶⁶ In multivariate regression models, waist cir-

cumference is significantly more efficient than BMI in predicting insulin resistance, blood pressure, serum cholesterol levels, and triglyceride levels.^{67–69} Consequently, measurements of waist circumference provide unique predictive information regarding health risks, especially for adolescents.

The overall ability of waist circumference percentile cutoff points to identify the fattest boys (as assessed with areas under the receiver operating curves), however, was no greater than that of triceps skinfold or BMI percentiles.⁶³ Also, Moreno et al⁶¹ found no overall differences in the ability of BMI, waist circumference, and triceps/subscapular skinfold ratio cutoff points to identify correctly Spanish children with the metabolic syndrome.⁴⁵

Translation of the available information on waist circumference into meaningful clinical application for the assessment of overweight and obesity in children is difficult. No data are available to identify waist circumference cutoff points that are appropriate for identifying children with the most visceral fat or the greatest risk for cardiovascular or metabolic problems, having been identified as overweight or obese through BMI. Consequently, it is not known exactly which waist circumference percentile clinicians should use and what clinical actions that value would indicate. Nevertheless, clinicians, especially those in subspecialty referral settings, may add waist circumference to the tools they use to assess risk. If they do, clinicians should use a high, age-specific, percentile cutoff point, such as the 90th or 95th percentile, to evaluate risk.

Waist circumference may prove useful in the future, but the expert committee withheld recommending it for routine clinical use at the present time because of incomplete information and the lack of specific guidelines for clinical application. Waist circumference percentiles are now available for US children⁷⁰ and for other populations.^{71–73} One possible approach may be to calculate the best waist circumference cutoff points for identifying at-risk children within BMI categories, as has been proposed for adults.⁷⁴

ASSESSMENT COMPONENTS OF THE MEDICAL HISTORY

Importance

The medical history is critical for 3 purposes, namely, identification of modifiable lifestyle behaviors (eg, dietary and physical activity practices), assessment of current and future risks for medical comorbidities, and assessment of the patient's and/or family's readiness to make behavioral changes. Although obesity is a condition with medical consequences, the treatment inevitably involves behavior changes, which pose exceptionally difficult challenges for successful treatment implementation, compared with many other medical conditions.

Health Behavior Changes

The history portion of assessment of childhood obesity should be directed, in part, toward identifying modifiable behaviors. Physicians and other health care professionals are more likely to provide successful treatment if they work with patients to target behaviors for change, rather than working from a “top-down” approach. Several approaches are available to negotiate lifestyle behavior changes that can improve health. The principles described below are intended for use with overweight or obese patients, but they apply to any circumstance in which health behavior changes are desired.

Self-efficacy is the personal belief that one can attain or accomplish successfully what one sets out to do. Because patients and families are more likely to do what they perceive to be both pleasant and feasible, providers should assess which activities patients enjoy and think they are capable of performing. Interventions and recommendations should be tailored accordingly.^{75–79}

“Readiness to change” is a behavioral approach that assesses an individual's readiness to adopt a particular behavior (otherwise known as the transtheoretical model).^{80,81} This approach stresses the interest in and motivation for thinking about, starting, or maintaining a behavior and allows for tailored messages and interventions based on 5 stages of change, as follows: stage 1, precontemplation; the patient is not yet considering the change; stage 2, contemplation; the patient is evaluating reasons for and against the change; stage 3, preparation; the patient is planning for the change; stage 4, action; the patient has made the change (<6 months); stage 5, maintenance; the patient has maintained the change (>6 months).

Individuals may not go through each step sequentially, and they may not spend the same amount of time in each stage. Behavior is seen as a dynamic process and not an “all-or-none” phenomenon. Assessing a person's stage of change acknowledges the patient's attitudes, respects his or her perspective, and is a vital step in ensuring that the behavioral intervention is delivered in a manner that is most beneficial for the patient and/or family.^{76,78,82,83} For example, recommending that a family change its food choices when the parent is not aware or convinced of the child's weight being problematic (precontemplative) may not be as successful as first identifying the issue and discussing the rationale for concern.

Rollnick et al⁸⁴ incorporated the principles described above into an approach called motivational interviewing. They defined motivational interviewing as a “client-centered counseling style for eliciting behavior change by helping clients explore and resolve ambivalence.” For a brief clinical assessment, they suggest asking 2 questions to gauge a patient's motivation to change an unhealthy behavior, that is, (1) how important (on scale of 1–10) the change in behavior is to the patient and (2) how confident the patient feels in his or her ability to

TABLE 1 Rapid Dietary Assessment Measures

Measure	Internet Address
WAVE (adult version)	http://biomed.brown.edu/nutrition
WAVE (child version)	http://biomed.brown.edu/nutrition
REAP	http://biomed.brown.edu/nutrition
REAP physician key	http://biomed.brown.edu/nutrition
Rate Your Plate	http://biomed.brown.edu/nutrition
MyPyramid.gov	www.mypyramid.gov
Healthy Eating Index	www.cnpp.usda.gov/Publications/HEI

REAP indicates Rapid Eating and Activity Assessment.

make the change.⁸⁴ These 2 concepts help direct the focus of the interaction between the clinician and the patient. If a patient does not identify a condition (eg, a child's high BMI) as important, then the discussion may target health-related risks. If the patient or family member recognizes the problem and its importance but is not confident in making a change, then the discussion may usefully target strategies for change, as well as barriers that may interfere with the change. This approach allows health care professionals to collaborate with patients to promote change by using a brief, patient-centered assessment that can be adapted easily to the clinic setting.

A related approach put forth as a general clinical prevention tool is the 5As, that is, ask/assess, advise, agree, assist, and arrange follow-up care.⁸⁵ The exact wording of the 5As varies slightly among different publications, but the intent and process remain the same. These steps reinforce the concept that health care professionals need to assess behavior patterns and health belief structures to agree on a plan of action or intervention that is most appropriate for each patient.

Preliminary data on successful behavior changes using these approaches in health care settings show mixed results, and these approaches have been applied most often in the adult population^{86–91} for tobacco, alcohol, and drug use/addiction. Several studies that applied these methods to nutrition and physical activity assessment showed successful short-term results but less-convincing long-term results.⁹² Ongoing projects are examining the feasibility of these behavior change strategies in primary care settings, including pediatric practices.⁹³

Dietary Assessment

Assessment Methods

Many complex dietary factors are associated with obesity, and age, gender, and genetic predisposition are likely to influence their effects. Although individual nutrients have been linked to obesity,⁹⁴ few attempts have been made to identify eating patterns that may lead to obesity. Scientists have reached a consensus that obesity results from an imbalance in the energy balance equation; energy intake exceeds energy expenditure. There-

fore, assessment should address both sides of the equation (diet and physical activity) in efforts to prevent or to treat obesity. Assessment of energy intake is challenging even under the most-controlled research conditions, and typically assessment includes a combination of assessment methods. Traditional dietary assessment methods include 24-hour recalls, food records, and food frequency questionnaires.⁹⁵ In a 24-hour recall, the interviewer asks an individual what he or she ate and drank in the past 24 hours. Ideally, this is repeated several times, to obtain a view of the individual's usual dietary intake. To complete food records, patients write down, for several days, the foods, amounts, recipes, and preparation methods for everything they consume. A food frequency questionnaire asks patients how often they consume specific foods and beverages and the sizes of their usual portions.

All of the methods described above have advantages and disadvantages in research settings, but they are impractical for use in most clinical settings. These interventions are time-consuming, expensive, and difficult for health care professionals to administer in the office. Furthermore, the value of estimating energy intake per se is limited because it is virtually impossible to assess energy expenditure accurately and precisely and therefore to determine energy balance. A few rapid assessment methods are available for practitioners to evaluate their patients' eating behaviors and physical activity, as well as to deliver effective nutrition counseling (Table 1). The weight, activity, variety (in diet), and excess (WAVE) tool allows a quick assessment of the patient's weight status, activity and inactivity patterns, variety of foods, and potential excessive consumption of selected foods. The evidence base for the WAVE tool and other potential assessment tools is presented in Table 2.

Targets for Behavior Change

Diverse eating patterns confound our understanding of the relationship between nutrient intake and chronic diseases, including obesity.¹⁰⁴ Factors that are named frequently as contributors to relative excess energy intake include restaurant food, sweetened beverages, 100% fruit juice, large portion sizes, and the frequency of meals and snacks. A body of research has addressed each of these dietary components as it relates to energy intake and to overweight. These eating patterns seem to be related more consistently to increased total energy intake than to actual weight status.

An important consideration in the interpretation of the results of this research is that the percentage variance in the eating pattern/overweight models was extremely small,^{105,106} which suggests that weight status likely stems from a combination of interrelated eating patterns, rather than a single eating pattern. In addition, the effects of these interrelated patterns on weight status may be cumulative, and they may vary according to gender, ethnicity, and genetic factors. Limitations of the

TABLE 2 Evidence for Dietary Assessment Tools

Authors and Year	Study Population	Study Design	Control Variables	Measures	Association	Quality
Soroudi et al, ⁹⁶ 2004	111 first-year medical students (mean age: 24 y)	Completed as part of basic science course	NA	WAVE screener (adult version)	NA	Feasibility: acceptable; acceptability: acceptable; reliability: NA; validity: NA
Segal-Isaacson et al, ⁹⁷ 2004	110 first-year medical students (mean age: 24.2 y); 53% male, 44% female, 65% white, 21% Asian, 8% Hispanic, and 6% black		NA	Rapid Eating and Activity Assessment (short version) and semiquantitative Block food frequency questionnaire	$r = -0.20$ to 0.51 ($P = .685-.0001$)	Feasibility: acceptable; acceptability: acceptable; reliability: NA; validity: NA
Prochaska et al, ⁹⁸ 2001	Study 1: 278 middle/high school students; study 2: 62 middle/high school students	2 middle schools; 2 high schools	NA	Assessment of fat intake and 3-d food records	ICC > 0.60 ; $r = 0.36$ ($P < .01$)	Feasibility: acceptable; acceptability: acceptable; reliability: good; validity: good
Block et al, ⁹⁹ 2000	200 adults	Employees of company	NA	Fruit, vegetable, fiber screener, Block food frequency questionnaire, and Healthy Eating Index	$r > 0.60$	Feasibility: acceptable; acceptability: acceptable; reliability: NA; validity: good
Gans et al, ¹⁰⁰ 1993	102 adults (age: 18–64 y)	Cross-sectional biennial household interview surveys in Pawtucket, Rhode Island, and comparison city	NA	Rate Your Plate and Willett questionnaire	$r = 0.28$ to -0.48 ($P = .004-.0001$)	Feasibility: acceptable; acceptability: acceptable; reliability: NA; validity: good
Prochaska and Sallis, ¹⁰¹ 2004	138 middle school students (mean age: 12.1 y); 65% female, 28% white, 23% Asian, 7% black, 5% Hispanic, 23% multiracial, and 13% other	1 school	NA	Fruit and vegetable screening measure and 3-d food records	ICC = 0.47 (1 mo); 1CC = 0.80 (same day); $\kappa = 44\%$ (1 mo); $\kappa = 59\%$ (same day); $r = .23$ ($P = .008$)	Feasibility: acceptable; acceptability: acceptable; reliability: good; validity: good
Weinstein et al, ¹⁰² 2004	16 467 adults (age: ≥ 17 y)	National Health and Nutrition Examination Survey	Age, race/ethnicity, gender, census region, poverty income ratio, pregnancy, BMI, energy intake, alcohol intake, smoking, vitamin or mineral, and energy intake	Healthy Eating Index, dietary intakes, and blood nutrient levels	Dietary intakes: $r = -0.03$ to 0.29 ; blood nutrients: $r = -0.005$ to 0.30 ($P = .05-.0001$)	Feasibility: NA; acceptability: NA; reliability: NA; validity: good
Kennedy et al, ¹⁰³ 1995	7463 (age: ≥ 2 y)	Continuing Survey of Food Intake by Individuals	NA	Healthy Eating Index and 3-d dietary intake data	Dietary intakes: $r = 0.06-0.42$	Feasibility: NA; acceptability: NA; reliability: NA; validity: acceptable

NA indicates not applicable; ICC indicates intraclass correlation.

literature include the predominance of cross-sectional studies, rather than prospective longitudinal studies, small sample sizes, and limited study populations (particularly a dearth of studies with children). Therefore, results often are inconsistent, and the findings of many studies have not been replicated. Despite these limitations, studies that identify eating patterns that may contribute to excessive energy intake, and that propose targets for behavior changes are useful for clinicians who are helping their patients prevent excessive weight gain.

Restaurant Food Consumption

Consumption of foods away from home increased considerably in children^{107,108} and adults¹⁰⁹ between 1977 and 1996. The proportion of foods that children consumed from restaurants and fast food outlets increased by nearly 300% during that 19-year period.¹⁰⁸ Fast food consumption was reported by 42% of children and 37% of adults,¹¹⁰ although investigators noted that there is no uniform definition of fast food and definitions varied among studies. The percentage of energy obtained from food prepared away from home also increased during that period, from 18% to 32%.¹¹¹ Portion sizes in restaurants increased from 1970 to 1999,^{112,113} with the result that soft drinks contained an additional 206 kJ, hamburgers 407 kJ, and French fries 286 kJ.

Portion sizes influence energy intake. Diliberti et al¹¹⁴ found that customers who purchased a larger portion of the entree served at a fast food outlet increased their intake of the entree by 43% and that of the entire meal by 25%, resulting in greater energy intake. Some studies showed that children^{110,115} and adolescents^{116–118} who consumed fast food more frequently had higher energy intakes and poorer diet quality, compared with those who did not. Interestingly, overweight adolescents were less likely than their leaner counterparts to compensate for the increased energy in the food by adjusting energy intake throughout the day.¹¹⁹

Studies have reported that the frequency of eating fast food is associated with BMI and body fatness in children¹²⁰ and adults.^{121–126} In a longitudinal study of 101 girls 8 to 12 years of age, the frequency of eating quick-service food at baseline was associated positively with changes in BMI *z* scores at 11- and 19-year follow-up evaluations.¹²⁰ In young adult women, increases in frequency of fast food restaurant use were associated with increases in body weight over 3 years in a randomized, prospective, intervention trial on weight gain.¹²² In the Coronary Artery Risk Development in Young Adults study of 3031 young adults, Pereira et al¹²⁶ reported that changes in fast food frequency were associated with changes in body weight but the changes varied according to racial group. Data from another study suggested that older children who consumed fried foods away from home more frequently over a 1-year period were heavier and had greater total energy intake, compared

with children with low frequency of fried food consumption away from home.¹²⁷ In contrast, French et al¹¹⁶ reported that the frequency of fast food consumption by adolescents was not associated with overweight status. Although not entirely consistent, the data suggest that fast food consumption may be related to BMI. For individuals and families that eat regularly at restaurants or fast food establishments, reducing the frequency of these meals may be a strategy to decrease total energy intake.

Sweetened Beverage Consumption

Experts have raised concerns about high intakes of sweetened beverages and their possible association with the increasing prevalence of overweight and obesity among children.^{128,129} Over the past 4 decades, national data on individuals ≥ 2 years of age showed an increase in sweetened beverage consumption for all age groups.^{112,130} Soft drink consumption accounts for one third of added sugar intake in the US diet.¹³¹ In one study of fourth-grade and fifth-grade children, sweetened beverages constituted 51% of the average daily intake of beverages consumed.¹³² This large intake of sweetened beverages could contribute to increased energy intake, tilting the energy balance toward excessive weight gain.

Most cross-sectional studies have shown a positive relationship between greater intake of added sugars and total energy intake.^{128,129,133–137} Energy intake has been reported to be related positively to consumption of sweetened beverages by children and adolescents.¹³³ In another report, children who drank the most sweetened beverages consumed ~ 1390 kJ more per day than did those who did not drink sweetened beverages.¹³² The Bogalusa Heart Study examined energy intake among 10-year-old children from 1973 to 1994. Findings from the study showed that children who did not consume sweetened beverages did not have increased energy intake. However, energy intake did increase among children who consumed small to moderate to large amounts of sweetened beverages.¹³⁷ Of interest, mean BMI increased in all categories of sweetened beverage consumption, including children who did not consume sweetened beverages.

Although several studies showed an association between sweetened beverage consumption and risk of obesity,^{128,129,134,139–142} other studies found no association,^{105,143–148} and a few indicated a negative relationship.^{149–151} In a pilot intervention study, Ebbeling et al¹⁴⁰ showed that reducing sweetened beverage consumption reduced body weight in adolescents in the upper baseline BMI tertile. In their comprehensive review of studies that examined the relationship between sweetened beverages and adiposity, Bachman et al¹⁵¹ concluded that the association between sweetened beverages and overweight is unclear and that the evidence is inconsistent. In another review, however, the authors came to a completely different conclusion.¹⁵³

The strongest current evidence supports a positive

association between sweetened beverage consumption and energy intake. These conclusions were similar to those made by the 2005 Dietary Guidelines Advisory Committee (DGAC).¹⁵⁴ Decreasing sweetened beverage consumption may be one strategy to decrease total energy intake. More intervention studies are needed, particularly in children, for better understanding of the relationship between sweetened beverage consumption and weight gain. However, it may be concluded intuitively that, if an individual consumes excessive sweetened beverages, then the resulting increase in energy intake may lead to weight gain.

Fruit Juice Consumption

Recently, 100% fruit juice has received much attention as a potential culprit in the prevalence of obesity among young children. In 2001, the Committee on Nutrition of the American Academy of Pediatrics concluded that 100% fruit juice had no beneficial effect over whole fruit for infants >6 months of age and children.¹⁵⁵ For a number of reasons, the recommendations included limiting 100% fruit juice to 4 to 6 oz/day for children 1 to 6 years of age and 8 to 12 oz for older children. The 2005 DGAC recommended that no more than one third of the total fruit group intake recommended come from fruit juice.¹⁵⁴

Limited data are available to assess the relationship between 100% fruit juice consumption and body weight in children. Two separate studies by Dennison et al^{156,157} showed that consumption of 100% fruit juice (>12 fl oz/day)¹⁵⁶ and apple juice only¹⁵⁷ was associated positively with BMI in samples of children 2 to 5 years of age. Tanasescu et al¹⁵⁸ found that fruit juice and possibly fruit drinks were associated with overweight in 29 obese Puerto Rican children 7 to 10 years of age. In contrast, 6 longitudinal and cross-sectional studies reported either a negative or neutral association between 100% fruit juice intake and weight status in children.^{105,142,148,159–163} Overall, the current evidence shows only a weak association between 100% fruit juice consumption and excessive weight gain.

Portion Sizes

A number of short-term feeding studies, 1 longitudinal study, and 3 observational studies showed that portion sizes influence energy intake. Adults served large portion sizes consumed more food and more total energy^{130,164–167} than did individuals who were served smaller portion sizes; there was no evidence of meal-to-meal compensation for higher intakes.

Several well-controlled, laboratory-based studies showed that providing older children and adults with larger food portions could lead to significant increases in food and energy intakes, independent of the energy density of the food.^{164,165,167} This effect was demonstrated for snacks,¹⁶⁸ delicatessen-style sandwiches,¹⁶⁹ and entrees.^{130,164,166,170} The responses to the variations in por-

tion sizes were not influenced by gender or BMI.^{130,166,169} The energy density of food can have an effect on energy intake when portion sizes are varied.^{164,167} Therefore, increases in portion sizes and energy density may lead to independent and additive increases in energy intake.¹⁶⁵

The responses of young children to portion sizes seemed to be similar to those of adults; presentation of larger portion sizes resulted in increased energy intake.^{170,171} One study found that larger portion sizes resulted in greater energy intakes for children 5 years of age but not for children 3 years of age.¹⁷¹ Another study by the same group found that, when children 3 to 5 years of age were presented with a large portion size of an entree, they consumed 25% more of that entree and their energy intake increased 15% for the whole meal, compared with children who were presented with an age-appropriate portion size.¹⁷⁰ That study also reported that the children consumed 25% less of the entree when they were allowed to serve themselves than when the entree was served to them on individual plates.

Two cross-sectional studies of preschool-aged children, using national data, examined the relationships between portion sizes, energy intake, and body weight. Portion size alone accounted for 17% to 19% of the variance in energy intake, whereas body weight predicted only 4%.¹⁷² Body weight was related positively to energy intake and portion size but not the number of different foods or the number of eating occasions.¹⁷³

More studies with infants and children are needed to understand how larger portion sizes at a single feeding or meal affect total energy intake over a 24-hour period. There are no longitudinal studies with children showing an association between increased portion size and BMI. Data suggest, however, that reducing food portion sizes may be an effective strategy for decreasing energy intake, especially for energy-dense foods. For clinicians, however, determining the appropriateness of portion sizes presented to and consumed by a child is difficult, as is making specific recommendations for age-appropriate portion sizes.

Energy-Dense Foods

Energy density refers to the amount of energy in a given weight of food and depends on the content of fat, carbohydrate, protein, and water. Water has the greatest impact on energy density, because it adds weight without energy. The high-energy content of fat also influences the energy density of food. Fiber can decrease the energy density of foods.

In several feeding studies, ad libitum consumption of foods that were high in energy density resulted in significantly greater total energy intake, compared with foods that were low in energy density.^{174–177} Delayed satiety may be the reason why some individuals consumed large amounts of energy-dense foods.¹⁷⁶

A number of other laboratory studies indicated that energy density was associated with a reduction in energy intake.^{165,178–180} For example, eating low-density foods such as salad or soup as the first course of a meal reduced total energy intake, compared with eating a meal that consisted entirely of foods high in energy density.^{165,181} Rolls et al¹⁷⁶ showed that adding air to test meals that had similar macronutrient compositions and energy contents reduced energy intake significantly, which suggests that the mass and volume of a meal are important. For foods that are low in energy density, satisfying portions should be encouraged, because they provide little energy and produce satiety.

Low-fat diets have been associated with lower energy intake,¹⁵ possibly because of a reduction in energy density. Laboratory studies showed that fat content, independent of energy density, had little influence on energy intake.^{165,171,174} Because lower-fat diets generally have lower energy density, reducing the intake of total fat may be one strategy for reducing energy intake.

What is the relationship between energy-dense foods and weight? Two clinical trials tested the influence of variations in energy density on body weight. In one study, adults who incorporated 2 servings of soup (which is low in energy density) into a calorie-restricted diet lost significantly more weight than did those who incorporated a similar number of calories as energy-dense snacks.¹⁸² In another study, investigators examined how 2 strategies to reduce energy density in the diet affected body weight during a 1-year period.¹⁸³ One group was counseled to reduce fat intake and to limit portions. The other group was counseled to increase intake of water-rich foods and to choose reduced-fat foods. Both groups succeeded in lowering the energy density of their diets, and they lost significant amounts of weight and kept the weight off over the year. These studies provide promising results, but more long-term intervention studies are needed to understand whether diets with reduced energy density prevent weight gain, particularly in children. One cross-sectional study with children 10 years of age found that consumption of energy-dense foods was a predictor of being overweight.¹⁰⁵ However, those results were not confirmed by others.^{184,185}

On the basis of the current studies, insufficient evidence exists to determine the contribution of energy-dense foods to weight gain; no studies of children are available. However, consuming energy-dense foods may contribute to excessive energy intake. The 2005 DGAC came to a similar conclusion in its report.¹⁵⁴ Encouraging consumption of foods low in energy density, including those with high fiber and/or water contents and those with modest fat content, may be a useful strategy for individuals who are trying to lose weight or to maintain their current weight.¹⁸⁶ Unfortunately, there is no standard calculation method for determination of energy

density in foods.¹⁸⁷ No published studies, particularly involving children, have examined the impact of consuming high-energy density foods on diet quality and intake of fat-soluble vitamins, essential fatty acids, and amino acids. One adult study showed that low energy density of diets was associated with high diet quality.¹⁸⁸ Unfortunately, there were some concerns about the study¹⁸⁹ and the definition of the energy density categories. More studies are needed in this area of research, particularly involving children.

Fruit and Vegetable Consumption

The specific relationships between fruit and vegetable consumption, energy balance, and obesity prevention represent an emerging area of research. Fruits and vegetables are high in fiber and water content, and they may play a role in promoting satiety and decreasing total energy intake by displacing energy-dense foods. Despite long-standing recommendations to eat several servings of fruits and vegetables each day, intake among US children remains low.¹⁹⁰

Findings from observational studies are equivocal,¹⁹¹ with some studies showing an inverse association^{125,192–205} and others showing no relationship^{158,184,206–216} between fruit and vegetable consumption and a measure of body adiposity. The studies showing an inverse association, however, have not been consistent with respect to gender, ethnicity, age group, and type of fruit or vegetable. Two studies reported that fruit consumption was associated inversely with weight status in children,^{105,198} but a relationship with vegetable intake was not apparent. Several observational studies did not control for potential confounders (physical activity and, in some cases, dietary energy intake). The percentage of the variance in children's BMI explained by fruit and fruit juice consumption was <3%.¹⁰⁵

The lack of association between vegetable consumption and weight status may be specific to the type of vegetable consumed. Some vegetables typically are consumed with fat added during preparation, such as fried potatoes. This may explain why the study by Lin and Morrison¹⁹⁸ found a positive association between intake of potatoes and weight status among adults. Clearly, more studies are needed to better understand these inconsistencies in the findings across studies.

Numerous interventions have been designed to promote increased consumption of fruits and vegetables, but very few studied weight status or change in BMI as an outcome variable. Some interventions included multiple components, making the identification of an independent effect of fruit and vegetable consumption in the prevention of overweight or weight gain difficult.

A number of adult trials examined the effects of increased fruit and vegetable consumption on weight; those studies were reviewed by Rolls et al.²¹⁷ The 2005 DGAC concluded that data from those studies showed

that, without advice to lose weight, increased fruit and vegetable consumption by itself did not lead to weight loss.¹⁵⁴

Intervention studies in children that examined fruit and vegetable consumption targeted mainly changes in intake and not effects on body weight. Encouragement to eat more fruits and vegetables often has been one of several messages aimed at modifying energy balance.^{218,219} However, efforts to increase knowledge and to improve attitudes toward fruit and vegetable consumption have had modest effects on actual consumption.^{220,221}

In one randomized trial examining weight loss in children, Epstein et al²²¹ reported that a message that targeted specifically increasing fruit and vegetable consumption resulted in greater weight loss than did an intervention message that focused on reducing high-fat and high-sugar food intakes. More studies with children are needed to understand the independent effect of increased fruit and vegetable consumption in randomized, controlled trials on prevention of weight gain. Encouraging greater fruit and vegetable consumption is a sound message in general, and limited evidence suggests that it may be a useful strategy in efforts to achieve and to sustain weight loss.

Breakfast Consumption

Several studies showed that skipping breakfast decreased the nutritional quality of the diets of children^{107,222–224} and adults.^{225–227} The average total energy intake was significantly lower for children who did not consume breakfast, and they did not make up the differences in energy intake at other meals.²²² The energy content of school breakfasts has increased in the past 15 years.²²⁸

A few cross-sectional and longitudinal studies and one randomized, clinical trial have examined the association between breakfast consumption and BMI. A number of cross-sectional studies have shown a positive association between overweight and skipping breakfast among children^{216,229–232} and adults.²³³ However, other studies, particularly one with children,¹⁰⁷ found no association.

Two longitudinal studies, one each with children and adults, have been reported. The first was conducted with >14 000 children 9 to 14 years of age.²³⁴ After a 1-year follow-up period, overweight children who never ate breakfast had a greater decline in BMI than did overweight children who ate breakfast. Normal-weight children who never ate breakfast, however, had weight gains comparable to those of normal-weight children who ate breakfast. The adult study found that skipping breakfast was associated with an increased prevalence of obesity.²³³ In a randomized, clinical trial,²³⁵ adults who ate no breakfast at baseline and who were assigned randomly to eat 3 meals per day lost slightly more

weight by 12 weeks, compared with those who were assigned randomly to consume no breakfast and to eat 2 meals per day. However, of breakfast eaters at baseline, those who were assigned randomly to eat only 2 meals per day lost more weight than did those who continued to eat breakfast. The authors suggested that the effects might have been influenced by subjects having to make the most-substantial changes to their usual routine.²³⁵ Clearly, more studies are needed, because current evidence related to the effect of breakfast consumption and the content of the meal is inconclusive. Children should not be encouraged to skip breakfast. More importantly, skipping breakfast may result in poorer nutritional quality of the diet and may have adverse effects on performance in school.^{236–238}

Meal Frequency and Snacking

Previous studies demonstrated an inverse association between meal frequency and the prevalence of obesity in children^{239,240} and adults.^{241,242} Four studies examined this association. Three found no association between the number of eating episodes and overweight in children 10 years of age.^{105,243} However, a cross-sectional study with 4370 German children 5 to 6 years of age found that the prevalence of obesity decreased according to the reported number of meals consumed each day.²⁴⁰ The prevalence of obesity was 4.2% among children who consumed ≤ 3 meals per day, compared with 1.7% among those who consumed ≥ 5 meals. Although some studies suggested that a “nibbling” or “grazing” meal pattern may be associated with leanness, those studies were vulnerable to methodologic errors that might have generated spurious relationships because of dietary underreporting and posthoc alterations in eating patterns in response to weight gain. Moreover, the association between increased eating frequency and lower body weight status might have been influenced by increased physical activity and a reduction in the mean energy consumed per eating episode. More longitudinal studies are needed to better understand the association, if any, between meal frequency and overweight in children.

On the basis of national data, the prevalence of snacking has increased for individuals 2 to 18 years of age,^{244,245} although the average size of snacks and energy per snack have remained relatively constant.²⁴⁴ There has been a shift from meals to snacks in the past 20 years.²²⁸ In contrast, one study showed that snacking decreased among children 10 years of age from 1973 to 1994 in Bogalusa, Louisiana,²⁴³ although the prevalence of obesity increased. These conflicting findings may reflect differences in age groups studied, regions of the country, methodologic changes over time, or the definition of what constitutes a snack or a snacking occasion. In adults, increased snacking resulted in increased energy intake but was not associated with BMI.²⁴⁶ Other studies showed that obese adults were more frequent snack-

ers^{247,248} and total energy intake was higher for snackers²⁴⁸ than for reference adults. Two cross-sectional studies of children 10 years of age showed no association between snacking and overweight status.^{105,243} More longitudinal studies are needed to better understand the associations between snacking, total energy intake, and overweight in both children and adults. The data on meal frequency and snacking are inconclusive and therefore do not represent a priority area of inquiry for all patients.

Summary

A number of studies have been conducted with adults, but far fewer with children, that address the associations between specific eating patterns and weight status. Results are inconsistent, largely because of methodologic limitations and small sample sizes. More well-designed, longitudinal studies and randomized, controlled trials are needed before any definitive statements can be made regarding which eating patterns are associated most strongly with overweight and how age, gender, ethnicity, and geographic location affect these associations. Evidence supports an association between at least some of the eating patterns discussed in this report and increased energy intake for some individuals, and these patterns represent behaviors that can be targeted for change.

Overall Recommendations for Dietary Assessment

The assessment of dietary patterns among children and adolescents should address the following: (1) assessment of self-efficacy and readiness to change, (2) qualitative assessment of dietary patterns, and (3) working in conjunction with patients and families to identify dietary practices that are targets for change. The assessment writing group recommends the following. (1) Qualitative assessment of dietary patterns should be performed for all pediatric patients at each clinic visit, at a minimum, for anticipatory guidance. (2) Assessment should address dietary practices for which evidence supports a positive association with energy intake and behaviors for some individuals and that represent behaviors that can be targeted for change. By decreasing energy intake without increasing energy intake throughout the day or from other foods, changes in these behaviors may prevent excessive weight gain. These behaviors include the frequency of eating outside the home at restaurants or fast food establishments, excessive consumption of sweetened beverages, and consumption of excessive portion sizes for age.

The assessment writing group also suggests assessment of additional dietary practices that have a weaker evidence base for association with energy intake but may be important for some individuals and that represent behaviors that can be targeted for change. The writing group suggests consideration of (1) excessive consumption of 100% fruit juice, (2) breakfast consumption (frequency

and quality), (3) excessive consumption of foods that are high in energy density, (4) low consumption of fruits and vegetables, and (5) meal frequency and snacking patterns (including quality). The child version of the WAVE assessment tool (Table 1), which provides a means for quick assessment of both diet and activity, may be useful to clinicians in primary care settings.

Physical Activity Assessment

Levels of Physical Activity

Physical activity is an important component of health and well-being for people of all ages. Children who are physically active may gain immediate and long-term positive effects, such as improved mental health status and self-esteem; increased physical fitness, which enhances performance of daily activities; promotion of bone formation; weight maintenance; and prevention of cardiovascular risk factors.^{76,249} In addition, physical activity patterns established during childhood may continue into adulthood, establishing healthier choices over the entire lifespan.^{250,251} Health benefits for physically active adults include lower risks of coronary artery disease, T2DM, hypertension, hyperlipidemia, osteoporosis, certain cancers, and depressive symptoms.^{76,252,253}

Despite these benefits, results from the 2003 Youth Risk Behavior Surveillance Study and the 2002 Youth Media Campaign Longitudinal Survey showed that many children and adolescents do not meet recommended physical activity levels.^{254,255} Nationwide, 62.6% of students in the ninth through 12th grades met the recommendations for vigorous physical activity (≥ 20 minutes on ≥ 3 of the past 7 days), and 24.7% of students nationwide met recommendations for moderate physical activity (≥ 30 minutes on ≥ 5 of the past 7 days). Overall, $\sim 33\%$ of this group of students reported some but insufficient levels of physical activity, and 11.5% reported no moderate or vigorous physical activity.²⁵⁵ In addition, 38.2% reported watching >3 hours of television per day, on average. Twenty-three percent of younger children (9–12 years of age) had not engaged in any free-time physical activity outside of school in the past 7 days, and 61.5% had not participated in organized physical activity during nonschool hours.²⁵⁴ Higher levels of physical activity were reported by boys than by girls and by non-Hispanic white youths than by other racial and ethnic groups. Levels of physical activity also decline as children get older. It is estimated that physical activity levels decrease by 1.8% to 2.7% per year for boys 10 to 17 years of age and by 2.6% to 7.4% per year for girls 10 to 17 years of age.⁷⁷

Diet and physical activity are inextricably linked. Overweight and obesity result when daily energy intake is greater than daily energy expenditure over time. This concept of energy balance is crucial for successful assessment, prevention, and management of overweight and

obesity in childhood and adolescence. Energy intake is a relatively easy concept, because it includes all foods and beverages consumed during the day. Energy expenditure is more complex, because it is a combination of resting metabolic rate, the thermic effects of food, and the variety of activities the individual performs during the day.^{253,256} Therefore, measurement of physical activity is not equivalent to measurement of total energy expenditure; rather, physical activity is one (albeit the most variable and modifiable) element of total energy expenditure. For children and adolescents, a certain amount of positive energy balance is necessary for proper growth and development. The overall energy balance should tip in favor of slightly greater energy intake, relative to expenditure, although the percentage of total energy required for growth is small after infancy.²⁵⁷

Clarification of several terms is necessary to understand what is being measured when physical activity is being discussed. Physical activity is defined as any bodily movement produced by the contraction of skeletal muscles that increases energy expenditure above the basal level.²⁵⁸ Physical activity thus encompasses movement resulting from free play, structured activities such as sports, and general activities of daily living. Exercise is planned, structured, and repetitive bodily movement performed specifically to improve or to maintain physical fitness.²⁵⁸ Children and adolescents often participate in planned activities during physical education classes or in structured sports activities; however, the goal is not necessarily physical fitness. Physical fitness is a set of attributes that people have or achieve, such as cardiorespiratory fitness, muscular strength, flexibility, endurance, and body composition.^{258,259} This report focuses on the assessment of physical activity for the purpose of preventing or managing overweight and obesity in childhood and adolescence. Total energy expenditure, exercise, and fitness are beyond the scope of this report.

Assessment Methods

Appropriate assessment of physical activity patterns requires valid (accurate) and reliable (reproducible) instruments. Researchers have developed several approaches for measuring physical activity in children and adolescents, and most are reasonably reliable, with low to moderate validity.²⁵³ Briefly, these include questionnaires (self-report or interviewer-administered), direct observation, and electronic or mechanical monitoring (with a pedometer, accelerometer, or heart rate monitor). Methods such as double-labeled water testing and calorimetry assess total energy expenditure and resting metabolic rate, respectively, and can be used to estimate physical activity. Each method has strengths and weaknesses, which are described elsewhere.^{252,253,256,260,261} This report discusses the methods most adaptable to the clinic setting, that is, brief questionnaires and accelerometers or pedometers.

Questionnaires

The most common method for measuring physical activity is a self-report survey or checklist of the frequency, intensity, and duration of specific activities within a defined period (eg, past 24 hours, 1 week, or 1 month). Recurring problems with any self-report survey include recall bias and the documented tendency to overestimate activity levels, compared with observation, movement monitoring, or estimations from total energy expenditure. Depending on the goals of the questionnaire, this limitation may be tolerable. It is also a challenge to determine the lower age limit at which children can recall accurately what they did, as well as the intensity and duration of their physical activity. In general, children <10 years of age are considered too young to give reliable answers to physical activity questions.^{252,262} Parents should be used for proxy responses; however, they do not always capture accurately the physical activity levels for their children, either at home or in other settings.²⁶³

An additional challenge is the sporadic unstructured nature of physical activity among children, especially those <10 years of age. Unlike adolescents and adults, who can sustain 10 to 60 minutes or more of physical activity, young children typically have multiple frequent bursts of activity followed by periods of rest. Questions that aim to assess 30 minutes of moderate-intensity activity or 20 minutes of vigorous-intensity activity are not realistic for children; alternate assessment questions would be more appropriate. For example, proxy measures such as time spent outside or involvement in community sports programs have been shown to be predictive of physical activity in children.^{261,264}

A review of the literature reveals that very few questionnaires have been developed and validated for pediatric age groups. Most focus on adolescents, are quite lengthy, and have not been assessed for use in the clinic setting. Examples include written, verbal, and computer-based questionnaires. More-detailed information about these research questionnaires can be found elsewhere.^{252,253,265} A few questionnaires with the potential for clinic use have been designed and are discussed in Table 3.

Accelerometers and Pedometers

Most accelerometers measure quantity, duration, and intensity in the vertical plane. Newer products measure movement in 3 planes. Accelerometers are relatively easy to use, but they are more expensive than pedometers, and some require frequent downloading of information into a computer. Resource limitations and inconvenience will likely preclude their routine use in clinical settings for assessment of baseline physical activity levels.

Pedometers are easier to use and measure physical activity as steps walked, distance walked, or energy expended. Several studies have shown the reliability (correlation range: 0.51–0.92) and validity (correlation

TABLE 3 Evidence for Physical Activity and Sedentary Behavior Assessment Tools

Authors and Year	Study Design Measures	Results and Conclusions
Burdette et al, ²⁶⁴ 2004	Cross-sectional study; (1) outdoor time checklist (average daily score) (2 questions using parental report for preschool-aged children), (2) outdoor time recall questions (average daily minutes), (2) questions using parental report for preschool-aged children), and (3) television or videotape viewing time (average daily minutes) (2 questions using parental report for preschool-aged children)	Acceptability: NA; feasibility: tools like this approximate physical activity in preschool-aged children and may be easy to use in clinic setting; reliability: checklist (tool 1) compared with recall (tool 2), $r = 0.57$ ($P < .001$); validity (comparison with accelerometer): checklist (tool 1), $r = 0.33$ ($P < .001$); recall (tool 2), $r = 0.20$ ($P = .003$); minutes of television/videotapes (tool 3), $r = -0.16$ ($P = .2$)
Jimmy and Martin, ⁹² 2005	Randomized, controlled trial; (1) Revised Physical Activity Readiness Questionnaire (waiting room written assessment identifying contraindications for physical activity), (2) stages of change (waiting room assessment with 2 written questions identifying inactive adolescents and adults and their intention to become more active), (3) acceptability (in-person interviews with providers and patients), and (4) modified 7-d physical activity recall questionnaire (telephone assessment 7 wk and 14 mo after intervention)	Acceptability: providers: generally acceptable for patients ≥ 15 y of age (needed 2–10 min to review written answers to questions, discuss issue, and recommend counseling); patients: perceived as good and useful; office support staff members and counselors: NA; feasibility: general clinic workflow was reported as feasible; reliability: NA; validity: NA
Koo and Rohan, ²⁶³ 1999	Retrospective cohort study; (1) perspiration score (1 question assessing times of physical activity that caused heavy perspiration per week in past year), (2) stairs score (1 question assessing flights of stairs per day over past year), (3) Godin-Shephard score (1 question assessing weekly average of ≥ 15 min of strenuous, moderate, or mild exercise in past year), and (4) specific activity score (11 questions assessing the average time per week in 11 sports activities over past year)	Acceptability: NA; feasibility: authors suggest tools 1, 3, and 4 may be simple and practical physical activity measures for 7–15-y-old youths; reliability (reproducibility of repeat testing with same measure 11 mo apart): perspiration score (tool 1), $r = 0.44$; stairs score (tool 2), $r = 0.59$; Godin-Shephard score (tool 3), $r = 0.48$; specific activity score (tool 4), $r = 0.53$; validity: NA
Ortega-Sanchez et al, ²⁶⁶ 2004	Randomized, controlled trial; (1) 15 physical activity assessment questions verbally asked by physician during medical visit, allowing for calculation of frequency (days per week), duration (minutes per week), and intensity (mild, moderate, or vigorous) of school physical education, organized sports, and leisure time physical activity; categories of physical activity are (a) active (met MPA or VPA recommendations at baseline), (b) partially active (active but does not meet physical activity recommendations), and (c) inactive; and (2) same questions administered at clinic visit or in telephone assessment at 6 and 12 mo	Acceptability: NA; feasibility: NA; reliability: NA; validity (comparison of resting heart rate between patients in active and inactive physical activity categories): significant t score for 12–21-y-old boys; not significant for 12–21-y-old girls
Patrick et al, ²⁶⁷ 2001	Randomized, control trial; (1) brief self-report of physical activity that measures days per week of MPA and VPA consistent with various behavior change methods, using previously validated questions (performed interactively on computer in waiting room); (2) assessment of disordered eating; (3) same questions in telephone assessment at 4 mo; (4) acceptability (patient and parent satisfaction at 1 wk and at 4 mo), and (5) feasibility (no measure)	Acceptability: generally high satisfaction (range: 3.31–3.84 of 5); feasibility: general conclusion that approach seems feasible for 11–18-y-old youths if computers can be made available; reliability (reproducibility of repeat testing of same measure 1 wk apart): days per week of ≥ 20 min of VPA, $r = 0.67$ ($P = .02$); days per week of ≥ 30 min of MPA, $r = 0.55$ ($P = .16$); validity (comparison with accelerometer): days per week of ≥ 20 min of VPA, $r = 0.31$ ($P = .02$); days per week of ≥ 30 min of MPA, $r = 0.20$; ($P = 0.16$)
Prochaska et al, ⁹⁸ 2001	Cross-sectional/convenience sample; (1) study 1: total of 9 measures using Youth Risk Behavior Surveillance modified physical activity assessment questions, including (a) days in past week, days in typical week, and composite measure of ≥ 20 min of VPA, (b) days in past week, days in typical week, and composite measure of ≥ 30 min of MPA, and (c) days in past week, days in typical week, and composite measure of ≥ 60 min of MPA; (2) study 2 (subset of subjects from study 1 who also wore accelerometers), including (a) same 9 measures from study 1 and (b) average minutes of MPA and VPA per day on accelerometer; (3) study 3: (a) days per week, days in typical week, and composite measure of ≥ 60 min of MPA and VPA combined and (b) average minutes of MPA and VPA per day on accelerometer	Acceptability: NA; feasibility: authors recommend composite measure of days per week of accumulated 60 min of MPA and VPA from study 3 for clinical assessment of physical activity among adolescents in middle or high school (brief, easy to score, most reliable, and greatest validity); reliability (reproducibility of repeat testing of same measure): study 1: VPA, $r = 0.66$ – 0.76 ; MPA, 30 min, $r = 0.55$ – 0.71 ; MPA, 60 min, $r = 0.65$ – 0.79 ; study 3: $r = 0.76$ (range of 0.53 for 1-mo retest to 0.88 for same-day retest); validity (comparison with accelerometer): study 2: VPA, $r = 0.31$ – 0.37 ($P < .05$); MPA, 30 min, $r = 0.20$ – 0.26 ($P > .05$); MPA, 60 min, $r = 0.37$ – 0.46 ($P < .01$); study 3: $r = 0.40$ ($P < .001$)
Souroudi et al, ⁹⁶ 2004	Cross-sectional/convenience sample; quick WAVE screener developed to be used in primary care settings (1 page, 17 total items, 3 items to assess sedentary behavior and physical activity; one version for children and one version for adolescents). Adapted from Youth Risk Behavior Surveillance and Paffenberger physical activity questionnaire; dialogue guide available, based on behavior change theories such as motivational interviewing and stages of change	Acceptability: providers: patients generally were comfortable taking the screener assessment; feasibility: potentially feasible (takes 5–10 min during clinic visit); reliability: NA; validity: NA

MPA indicates moderate physical activity; VPA, vigorous physical activity; NA, not applicable.

range: 0.49–0.93) of pedometer use for children and adolescents.^{260,268–272} Two studies found that, on average, children 8 to 10 years of age²⁷² take between 12 000 and 16 000 steps per day.^{271,273} Jago et al²⁶⁹ determined that taking 4000 steps in 30 minutes and taking 8000 steps in 60 minutes (fast walking) meet current US physical activity recommendations.

Pedometers could be used at home to assess baseline physical activity levels for children and adolescents, and specific activities could be recorded in conjunction with times the monitor is worn. For example, a clinic visit with BMI screening may prompt a physical activity assessment and counseling on the basis of overweight or obese status. The patient can be instructed to wear a pedometer daily for 1 week, to record specific physical activities in a diary, and to determine a baseline average number of daily steps (with the assistance of a parent, if necessary). Results can be used as a proxy for overall physical activity and compared with documentation in the activity diary. Discussion at a follow-up visit with a designated clinic staff member can determine necessary modifications and can address any barriers to increasing physical activity levels. No studies have assessed the feasibility, reliability, and validity of using pedometers for baseline assessment in this way. For a list of available pedometers, see the report by Bjornson²⁶⁸ (and www.pedometers.com).

Behavior Changes

Approaches such as readiness to change, motivational interviewing, and the 5As (see above) emphasize the assessment of psychological readiness, so that providers can more effectively help their patients increase physical activity levels. Pediatric health care professionals can assess briefly the self-efficacy and readiness to change of patients and their families by asking the following 2 questions. (1) How important is it to become more physically active? (2) How confident do you feel in your ability to become more physically active?²⁸⁴ If time allows, health care professionals should assess specific activities their patients enjoy and consider within their capabilities and should tailor interventions and recommendations accordingly.^{75–79} Practitioners also can determine the readiness to change of patients and their families. This approach may allow greater acceptance of the plan of action/intervention by the patient and perhaps increase motivation and compliance.

Environment and Social Support

Patients of any age rarely, if ever, act or respond independently of their social and physical environments. Children and adolescents are influenced by home, school, and after-school environments, as well as by family and peer dynamics.^{76–79,82,83} Practitioners should assess barriers and facilitators in these settings, to determine the best way to increase physical activity levels of children and adolescents. Table 4 provides a list of items

TABLE 4 Social and Environmental Barriers to and Facilitators of Physical Activity in Children and Adolescents

Home
Television in bedroom ^{82,274}
Family physical activity routines ^{77,78,82,83,275}
Willingness of family members to be active with patient ⁷⁵
Encouragement from parents ^{76,79}
Options and access for free play and organized sports ^{76,77,79,82}
School
Physical education classes and recess ⁸²
Affordability/socioeconomic status ^{76,77,82}
Safety ^{76,77,82}
Discretionary activity (eg, walking or biking to school, taking stairs, and running errands) ⁸²

identified in the literature as important to address. Although practitioners may not have the time or ability to change everything on the list, knowing which unique barriers and facilitators for physical activity exist should allow more effective messages and interventions to be tailored for each patient.

Current Levels of Physical Activity and Sedentary Behavior

Physical activity research has centered on measuring the type, frequency, intensity, and duration of physical activity. The consensus of recommendations from a variety of government and professional organizations is that children and adolescents should accumulate 60 minutes of at least moderate physical activity on a daily basis.^{4,6,154,276–281} This total duration does not have to be consecutive, and briefer bouts can be added up for a total of 60 minutes.

Age is an important consideration for assessment of physical activity in children and adolescents. Adolescents have physical activity patterns similar to those of adults, which can be assessed by using the moderate/vigorous physical activity framework that is widely used with the adult population. Self-reporting is reasonable with this age group, with the acknowledgment that self-report options likely overestimate the amount of physical activity performed. Children <10 years of age should not be relied on for self-reporting of physical activity; parental responses should be used instead. Questions should be centered on time in organized sports programs or outdoor unstructured play, to account for the sporadic and unsustained nature of physical activity among children.^{261,262,264,282}

Research also has stressed the importance of balancing sedentary behaviors, such as television/DVD/videotape watching and computer games (“screen time”), and less-active hobbies with physical activity.^{253,283} Time spent in sedentary behaviors, especially television viewing, should be reduced, and preliminary research results suggest limiting screen time to <2 hours/day.^{4,277–279,284–286} Television viewing is the only sedentary behavior that has been associated with an increase in BMI,²⁸⁴ and 2 studies have shown that

altering this behavior can affect weight gain.^{286,287} Interestingly, only weak associations between television viewing time and decreased levels of physical activity have been documented.^{285,288,289} Epstein et al²⁹⁰ reported that children do value various sedentary behaviors and may work to substitute physical activity with sedentary behavior. More research is needed to assess accurately the balance of sedentary behavior with physical activity and the associated outcomes.

To provide the most-effective weight maintenance and management interventions for children and adolescents, practitioners need to assess baseline levels of physical activity and sedentary behaviors and to determine whether each patient is likely to be meeting recommended levels. It may be prudent to assess parents' baseline levels briefly, to illuminate family physical activity patterns and routines during leisure time at home and on weekends. Fulkerson et al⁷⁵ found that parents being physically active with their children may be more important than simply giving verbal encouragement. Practitioners should ask about and record physical activity patterns at each visit, to determine patterns over time. Table 5 provides a list of commonly cited categories that practitioners could ask about routinely and document for their pediatric patients. These categories are suggested on the basis of both the research literature and the ability to target these behaviors for change.

Available Tools for Measuring Physical Activity Among Children and Adolescents in Clinical Settings

The nature of the clinician-patient interaction is based on sharing information verbally. The patient and/or family, with direction from the physician, communicates pertinent health, wellness, and illness information. The clinician incorporates that information with physical examination findings to determine health status and to establish other required steps, such as prevention interventions and laboratory or radiologic tests. Therefore, use of a brief, 5- to 10-minute, age-appropriate, self-report assessment and intervention tool seems the most logical and practical approach. Ideally, this tool would assess the 2 components of energy balance (dietary intake and physical activity) at the same time.

This report is not intended to develop a reliable and validated assessment tool to be used in the clinic setting. Rather, it draws attention to those tools that are most ready to use now and it highlights gaps in the research that, when filled, could lead to improved assessment of physical activity and sedentary behavior in children and adolescents.

In the clinic setting, comprehensive assessment usually is not the goal; the challenge is to develop a brief assessment tool that captures the usual amount of physical activity and sedentary behaviors the patient performs over time. A systematic review of the literature shows that few clinic-based questionnaires have been developed and validated for children and adolescents.

TABLE 5 Physical Activity and Sedentary Behaviors to Explore With Patients

Hours of television watched and/or screen time daily and/or weekly (<2 h/d) ^{75,76,82,83,256,285}
Type, frequency, duration, and intensity of physical activity daily and/or weekly ^{75,76,256}
Time spent in organized physical activity, unstructured activity/play/ time outside, and routine activity (eg, walking to school) ^{82,83,261}
Time spent in sedentary activity ^{76,83}

Six have been documented in the literature,^{92,96,98,263,266,267} and all except one⁹⁶ are directed toward adolescent assessment. One additional research study tool for pre-school-aged children may be adaptable to the clinic²⁶⁴ (Table 3).

Four criteria were used to determine the quality of the physical assessment tool used in these 7 studies, that is, acceptability, feasibility, reliability, and validity. For the purpose of these recommendations, these terms were defined as follows: (1) acceptability is the degree to which providers and patients are comfortable with the duration, wording, and other intangibles of the questionnaire; (2) feasibility is the degree to which implementation of the questionnaire is affordable and fits easily into the office environment and workflow; (3) reliability is the ability of the questionnaire to produce the same results when administered at different times or by different practitioners to the same patient (ie, reproducibility); and (4) validity is the ability of the questionnaire to measure the correct frequency, duration, and intensity of physical activity for each patient (ie, accuracy).

As is evident in Table 3, none of the studies was designed to assess all 4 criteria, and no single tool stands out as the most effective for use in the clinic setting. The WAVE tool for older children and adolescents, also described in the dietary assessment section, is feasible and acceptable from the provider perspective, and it focuses on the concept of energy balance in its assessment of weight, physical activity, variety of diet, and excess. Unfortunately, there are no reliability and validity studies to support its immediate use in the clinic setting. The Patient-Centered Assessment and Counseling for Exercise brief questionnaire for adolescents is a feasible, reliable, valid, brief physical activity questionnaire that has yet to be applied in the clinic setting. The longer Patient-Centered Assessment and Counseling for Exercise computer-based physical activity and nutrition questionnaire is acceptable, reliable, and valid for use in clinics that have computers available.²⁶⁷ However, its feasibility and acceptability for all patients and practices need to be examined more closely. The tool described by Burdette et al²⁶⁴ is the only brief and valid approach to be used for children <10 years of age; however, it has not been applied in the clinic setting.

Overall Recommendations for Physical Activity Assessment

Four general categories to be addressed in the assessment of physical activity among children and adolescents have been identified, as follows: (1) self-efficacy and readiness to change, (2) environment and social support, (3) level of physical activity, and (4) level of sedentary behavior. The assessment writing group recommends the following. (1) Assessment of physical activity levels should be performed for all pediatric patients at least at each well-child visit for anticipatory guidance, to determine whether they are meeting recommendations of 60 minutes of at least moderate physical activity per day. (2) Assessment of sedentary behaviors such as watching television and/or DVDs, playing video games, and using the computer should be performed at each well-child visit, in comparison with a suggested baseline of <2 hours/day.

The assessment writing group also suggests the following. (1) Assessment of social and environmental barriers and facilitators for physical activity should be performed at each visit at which physical activity levels and sedentary behaviors are assessed. Results should be used to discuss and to develop reasonable prevention and treatment interventions for patients not meeting recommended levels of physical activity. (2) Assessment of readiness to change and motivation to change should be performed at each visit at which physical activity and sedentary behaviors are assessed. The 2 questions (importance and confidence) established by Rollnick et al⁸⁴ represent a brief way to perform such an assessment. (3) Until additional research delineates a standardized approach, providers should use one of the tools in Table 2, rather than developing their own physical assessment tools. The WAVE tool shows the most promise, because it is based on reliable validated questionnaires; however, it has yet to be evaluated in the clinic setting.

Medications

Some classes of medications are particularly associated with weight gain, including conventional and atypical antipsychotic agents, selective serotonin reuptake inhibitors, tricyclic antidepressants, anticonvulsants/mood stabilizers, conventional mood stabilizers, prednisone, and oral contraceptives. The mechanisms of weight gain vary among the drug classes, and the responses vary among individuals. Clinicians should thus recognize the potential for a variety of medications to act as confounding factors in excessive weight gain and in efforts to lose weight.

Family History

Risk Assessment

Clinical risk assessment is important to help gauge the likelihood of adverse medical consequences (current and future) from a child's weight status. Although persistence of overweight status generally is less likely for

younger children, the risk of persistence for an individual child is influenced strongly by parental weight status. Similarly, an overweight child's risk for comorbidities is influenced by genetic factors and may influence the intensity of interventions (eg, those aiming for prevention of additional weight gain, compared with those aiming for actual weight loss). The family history is an important aspect of risk assessment, along with other clinical and especially anthropometric and biochemical data. The 3 conditions recommended for family history evaluation (among first- and second-degree relatives) for all children are obesity, T2DM, and CVD (including hyperlipidemia and hypertension).

Susceptibility to Obesity

For the vast majority of individuals, weight status is attributable to interactions of multiple genetic and environmental factors, resulting ultimately in positive energy balance. As discussed by Barsh et al,²⁹¹ susceptibility to obesity is determined largely by genetic factors but the environment determines the phenotypic expression. A corollary is that, for different genotypes, the impact of environmental factors differs. Testing for specific genotypes currently is quite limited but, as more-refined testing becomes available, such assessments may help guide therapy.

From a practical standpoint, the clinician is faced with assessing an individual child's risk of persistence of overweight. Relevant in this context is a retrospective analysis of a large group of records for newborns monitored through 21 years of age, along with their parents' medical charts, in a health maintenance organization.²⁹² The weight status of a child's parents was associated strongly with the child's risk of persistence of overweight. At all ages, the risk of adult obesity was greater if one or both parents were obese. This applied especially to both obese and nonobese children <10 years of age. In that study, the "obese" categorization applied to BMI of ~85th percentile for age, whereas the "very obese" categorization applied to BMI of ~95th percentile. The very obese children with at least one obese parent were at highest risk for adult obesity. In early childhood (1–3 years of age), parental weight status was found to be a stronger predictor than the child's actual weight status. After 3 years of age, both child and parental weight status predicted adult obesity; as the child aged (≥ 6 years of age), his or her own weight status became the more important predictor. For children who were obese at any age, parental obesity was more important than either severity or duration of the child's obesity.²⁹²

Results of a cross-sectional study of school-aged children in Italy supported the strong impact of parental weight status. Maternal and paternal BMI influenced the child's BMI independently and significantly, even when multiple other factors, including lifestyle, parental education, and parental history of diabetes and/or hyperten-

sion, were considered.²⁹³ A prospective longitudinal study of growth from birth to 6 years of age in children born to either lean or obese mothers confirmed the influence of parental weight on the weight of young children. By 6 years of age, nearly one third of the children born to obese women had BMI of >85th percentile for age, compared with <3% of those born to lean women (odds ratio: 15.7). There were no differences in growth between the low-risk and high-risk groups during the first 2 years of life, whereas weight and lean body mass were higher in the high-risk group by 4 years of age and fat mass was significantly greater by 6 years of age. The limitations of that study, which were partially offset by the longitudinal design, included the relatively small sample size (~35 subjects per group), inclusion of only white subjects, and limited information (beyond income) on educational and behavioral characteristics of the parents or the subjects' environment.²⁹⁴

Genetic influences have been estimated to explain up to 70% of interindividual differences in BMI.²⁹⁵ Furthermore, heritability is estimated to account for 30% to 40% of such interrelated factors as adipose tissue distribution, physical activity, energy expenditure, eating behaviors, hunger and satiety, food preferences, lipoprotein lipase activity, lipid synthesis, and lipolysis.^{296,297} Ravussin and Bogardus²⁹⁸ estimated that ≥40% of the variability in BMI is related to genetic factors involved in the regulation of food intake and/or volitional activity. These findings do not explain the cause of an individual's weight status but highlight the complexity of potential influences.

Despite the recognized important genetic role in obesity, the multigenic nature of the condition is also abundantly clear. Single-gene disorders that result in severe obesity (eg, Prader-Willi, Bardet-Biedl, Alstrom, and Cohen syndromes) are relatively rare. As the study of obesity through molecular genetic methods expands inevitably, greater insight into these monogenic forms of obesity is likely to be forthcoming and, following from that, better understanding for assessment and treatment of larger subgroups of the obese population may result. Several reviews of this topic are available.^{291,296,299}

T2DM

The genetic component of T2DM is quite strong, and a positive family history has been found to be an independent predictor or risk factor for insulin resistance in children of several ethnic/racial backgrounds. The prevalence of T2DM is especially high in children of non-European ancestry, including Hispanic, black, and North American and Pima Indian children.³⁰⁰ The prevalence of insulin resistance also varies considerably among different racial/ethnic groups. White European children had a much lower prevalence than did those of South Asian ancestry, with the latter having a risk ratio of 13.7, compared with white children. A positive family history explained 29% to 88% of the variability.³⁰¹ Hemoglobin

A1c data for individuals 5 to 24 years of age from National Health and Nutrition Examination Survey III identified 3 factors associated with higher hemoglobin A1c levels in young adolescents of all ethnic/racial groups: positive family history of T2DM, overweight status, and lower socioeconomic status.³⁰² In a series examining insulin resistance and metabolic syndrome in US Latino children 8 to 13 years of age, ~90% of the children who had a positive family history of T2DM and who were overweight had ≥1 feature of metabolic syndrome.³⁰³ In a T2DM surveillance study involving Japanese children, 56.5% of the diabetic children had a positive family history in either first- or second-degree relatives.³⁰⁴

CVD (Hyperlipidemia and Hypertension)

High BMI has been associated with a positive family history of CVD and with markers of CVD.^{305,306} In a series of patients from referral clinics, approximately one third of obese children, both with and without hypertension, were found to have a positive family history of CVD (defined as CVD, myocardial infarction, stroke, or recognized CVD risk factors, including obesity, hypertension, and diabetes).³⁰⁵ Conversely, a positive family history of CVD alone was shown in a number of studies to be a poor predictor of hyperlipidemia.^{306,307} Risk of T2DM, identified by family history and clinical screening, also is associated with risk of CVD, with both conditions reflecting effects of insulin resistance.³⁰⁸ Inquiries should address a history of early cardiac arrest or stroke in first-degree relatives.

Both obesity and family history of hypertension seem to be independent risk factors for hypertension in children. On the basis of a retrospective analysis of medical charts from a clinical population referred for treatment of primary or secondary hypertension, Robinson et al³⁰⁹ found that BMI was greater in those with primary hypertension, compared with secondary hypertension. Family history of hypertension was associated with higher child BMI. Family history of hypertension also was associated significantly with primary hypertension independent of obesity in the child.³⁰⁹ In a case-control heritability analysis with a similar group of patients, 49% of patients with primary hypertension had parents with primary hypertension, whereas only 24% of patients with secondary hypertension had parents with hypertension. The heritability of primary hypertension was calculated to be 0.80, indicating that 80% of the variance in liability of primary hypertension is attributable to additive genetic factors.³¹⁰ In an analysis of CVD risk factors among pediatric patients with hypertension, family history of hypertension was found for 61% and 72% of hypertensive children with and without obesity, respectively.³⁰⁵ A small study of normotensive adolescents with positive family history for hypertension reported proximal renal tubular dysfunction independent of BMI. In the same series, obese adolescents had higher

aldosterone levels, regardless of family history of hypertension, which was interpreted to indicate increased distal tubule sodium reabsorption in the obese subjects (ie, a different renal function profile, compared with the nonobese subjects).³¹¹ These results were interpreted as indicating independent effects of family history and obesity.

In summary, family history of obesity, especially in the parents, yields substantial risk for a child's propensity for overweight. This relationship is strongest for children <6 years of age. Therefore, when a young child is found to have a high BMI, consideration of parental weight status provides an important determinant of risk for the persistence of overweight. Positive family history is an independent risk factor for insulin resistance in children of several ethnic/racial backgrounds, especially those of non-European ancestry, including Hispanic, black, and North American Indian children.

Overall Recommendation for Family History Assessment

It is strongly recommended that clinicians obtain a focused family history regarding obesity, T2DM, and CVD (particularly hypertension) in first-degree (parents) and second-degree (grandparents) relatives, to assess the risks of current or future comorbidities associated with a child's overweight status.

Review of Systems for Weight-Related Problems

Approach to Assessment

Assessment of symptoms associated with recognized comorbidities is an important aspect of evaluating the risks associated with a child's degree of overweight. The severity of overweight does not predict strictly the presence of associated health conditions, many of which are influenced by genetic predisposition and environmental factors. In addition, families may not recognize some of the symptoms, such as sleep disturbances, that are related to weight status. Therefore, such symptoms may not be acknowledged unless the clinician specifically asks about them. Table 6 provides a summary reference, and the following text elaborates on some of the most common conditions that the medical history may help to identify. Of note, insulin resistance and T2DM are relatively asymptomatic conditions, and diagnosis depends more on laboratory testing than on the review of systems.

Sleep Disorders

Disordered sleep may be one of the many contributors to excessive weight during childhood.³¹² Overweight individuals are at risk for more symptoms of sleep-disordered breathing, later sleep onset, shorter sleep time, and more-disrupted sleep, compared with those with normal weight.³¹² One of the categories of sleep-disordered breathing is obstructive sleep apnea syndrome, which is a disabling condition characterized by excessive daytime sleepiness, disruptive snoring, repeated episodes of upper

airway obstruction during sleep, and nocturnal hypoxemia.³¹³ Excessive weight is a risk factor for obstructive sleep apnea; between 13% and 33% of overweight children have obstructive sleep apnea, which is several times the prevalence in lean children.^{314–316} Differences in academic performance and depressive symptoms were in part attributable to short sleep times and daytime sleepiness.³¹²

Longitudinal studies have documented that shorter sleep times predict the later emergence of overweight.³¹⁷ Sleep deprivation hampers attention, impulse control, and higher-level problem-solving,³¹⁸ providing an indirect route through which dietary choices may be undermined. Sleepiness may contribute to more sedentary behaviors, although activity levels have not been found to mediate statistically the link between short sleep times and overweight.³¹⁹ A cross-sectional study indicated that obese adolescents experienced less sleep than did nonobese adolescents ($P < .01$), and daytime physical activity diminished by 3% for every 1-hour increase in sleep disturbance.³²⁰

Sleep debt may affect human hormonal mechanisms that affect metabolic and endocrine functions, including glucose metabolism and the release of serotonin³²¹ and other neuropeptides that affect eating behavior.³²² Serotonin has been implicated in both within-meal and post-meal satiety, with regard to the signals arising from food intake. Carbohydrate craving, which may be driven by the need for increased serotonin levels and subsequent feelings of well-being, has been implicated in obesity.³²³ Typically, serotonin levels are replenished during sleep.

Menstrual Irregularities

Many women with polycystic ovary syndrome are overweight or obese, but obesity itself is not considered to be etiologic in the development of the syndrome. Excess adiposity, however, can exacerbate associated reproductive and metabolic disorders. The syndrome can be diagnosed when other medical conditions that cause irregular menstrual cycles and androgen excess have been excluded and when ≥ 2 of the following are present: oligoovulation or anovulation (usually manifested as oligomenorrhea or amenorrhea), elevated levels of circulating androgens (hyperandrogenemia) or clinical manifestations of androgen excess (hyperandrogenism), and polycystic ovaries, as defined with ultrasonography.³²⁴

Abdominal Pain

Vague recurrent abdominal pain may be an indicator of nonalcoholic fatty liver disease,³²⁵ the prevalence of which has been estimated to range from 10% to 20% in obese children and adolescents.^{326,327} Depending on the presence and character of other symptoms, abdominal pain can also be a clue to the presence of gastroesophageal reflux, gallstones, or constipation, all of which are not uncommon in obese children.

TABLE 6 Review of Systems for Weight-Related Problems

Symptoms	Explanation	Potential Consequences/Comments
Sleep problems		
Loud snoring or apnea (prolonged intervals without respiratory effort)	Obstructive sleep apnea	Poor sleep efficiency, poor attention, poor academic performance, pulmonary hypertension, right ventricular hypertrophy, or enuresis
Shorter sleep time, later onset of sleep, daytime sleepiness, or restlessness	Disordered sleep	Depression, poor attention, poor academic performance, food cravings, or difficulty responding to satiety cues
Respiratory problems		
Shortness of breath, exercise intolerance, wheezing, or cough	Asthma	Progression of disease, resistance to treatment, exacerbation of excessive weight gain, or exacerbation of asthma with weight gain
Gastrointestinal problems		
Vague recurrent abdominal pain	Nonalcoholic fatty liver disease	Fatty deposits in liver; small percentage progresses to steatohepatitis, cirrhosis, and future hepatocarcinoma
Heartburn, dysphagia, regurgitation, or chest or epigastric pain	Gastroesophageal reflux	Increased abdominal pressure or esophagitis
Abdominal pain and/or distention, flatulence, fecal soiling/encopresis, anorexia, or enuresis	Constipation	Disordered eating pattern, physical inactivity, or decreased social interaction
Right upper quadrant or epigastric pain or vomiting and colicky pain	Gall bladder disease, with or without gallstones	Cholecystectomy (most patients with gallstones are asymptomatic)
Endocrine disorders		
Polyuria and polydipsia	T2DM	Lack of symptoms is normal for T2DM; unexpected weight loss may occur and may not indicate compliance with treatment of obesity
Menstrual irregularities		
Oligomenorrhea (<9 menses per y) or dysfunctional uterine bleeding (anovulation)	Polycystic ovary syndrome	Insulin resistance, metabolic syndrome, T2DM, infertility, or worsening obesity with worsening of aforementioned conditions
Orthopedic problems		
Hip pain, groin pain, thigh pain, painful gait, or waddling gait	Slipped capital femoral epiphysis	Permanent hip deformity and dysfunction, decreased physical activity, or worsening obesity
Knee pain	Slipped capital femoral epiphysis or Blount disease	Decreased physical function, decreased physical activity, or worsening obesity
Foot pain	Increased weight-bearing	Decreased physical activity or worsening obesity
Mental health		
Psychiatric conditions		
Flat affect or sad mood, loss of interest/pleasure, or worries/fears	Depression or anxiety	Worsening obesity, suicide, or eating disorder
Psychosocial conditions		
Body dissatisfaction, school avoidance, problems with social interactions, poor self-esteem, or neglect	Depression or anxiety	Worsening obesity
History/ongoing sexual abuse	Depression or anxiety	Worsening obesity
Hyperphagia or binge eating, eating "out of control," or bulimia	Disordered eating	Worsening obesity; medications may cause/exacerbate obesity
Genitourinary problems		
Nocturia or nocturnal enuresis	Disordered sleep	See above
Skin conditions		
Rash or irritations acne	Intertrigo attributable to increased skin-to-skin contact with persistent moisture	More serious skin infections and abscesses

Potential for Harm Resulting From Screening

The potential for harm resulting from screening was examined because of the impact it may have on the willingness of practitioners to address overweight and obesity with their pediatric patients. There is no direct evidence of harm resulting from screening for childhood overweight and obesity.⁴⁶ One study³²⁸ provided preliminary evidence that genetic susceptibility testing for obe-

sity in undergraduate students may motivate healthier dietary behaviors. However, data also suggested that individuals may engage in less-healthy behaviors after receiving results that indicate an average risk for obesity.³²⁸

Numerous studies have demonstrated the social and psychological consequences of obesity. Theoretically, these could be triggered and/or amplified with screen-

ing. The potential adverse effects related specifically to obesity screening include labeling and social stigmatization,^{195,328–330} low self-esteem,^{331–333} depressive feelings,^{334,335} negative body image,³³⁶ disordered eating or self-managed dieting,^{336–338} and negative effects resulting from parental concerns and attitudes.³³⁹ Available evidence, however, does not indicate that screening per se causes these potential harms. A recent evidence-based review by the US Preventive Services Task Force concluded that the evidence was insufficient to make conclusions about harms resulting from screening.³⁴⁰

CLINICAL DATA: PHYSICAL EXAMINATION AND LABORATORY TESTING

Physical Examination

Physical examination reflects the customary pediatric examination with a few extra foci. A summary of physical examination signs related to obesity or its comorbidities is provided in Table 7.

Anthropometry

Anthropometric features should be assessed. Height is to be measured in bare or stocking feet with a stadiometer, not a platform scale with a moveable rule on top. Children >2 years of age who are unable to stand erect but who are able to lie supine and fully extended should have recumbent length measured; 1.0 cm should be subtracted to approximate erect height³⁴¹ before BMI is calculated. Measurement should be made in centimeters rather than inches, which encourages rounding errors (see www.cdc.gov/nchs). Weight is to be measured with a calibrated balance-beam scale in light clothing and bare feet or, if possible, in a gown (if it is to be worn during the upcoming physical examination). Expression in kilograms is preferred. BMI should be calculated as $BMI = \text{weight (in kilograms)} / [\text{height (in meters)}]^2$ or calculated with the use of an automated calculator (www.cdc.gov/nccdphp/dnpa/bmi/calc-bmi.htm, or other sites noted above). Circular slide-rule BMI calculators are available and can be used to calculate BMI. These calculators do not indicate the BMI percentile for age.

Height, weight, and BMI for age are plotted on standard growth charts (available at www.cdc.gov/growthcharts) or from various industry sources. BMI should be calculated and plotted once per year for all children and adolescents.⁴ The growth velocity may also be determined and compared with standard charts; if growth velocity decreases, then the likelihood of endocrine disease increases, because non-endocrine disease-related obesity usually is associated with tall stature. Growth velocity charts can be obtained from growth hormone manufacturers. As a general rule, no child should grow <5 cm/y after 4 years of age and before puberty, although actual growth rates vary with age. The velocity

TABLE 7 Physical Examination in Primary Care Settings

System or Condition Assessed	Assessment
Anthropometric features	Calculation of BMI (weight in kilograms and height in centimeters)
Vital signs	Pulse and blood pressure (use correct cuff size; often must be checked manually because of "white coat hypertension")
General	Body fat distribution and affect
Skin	Acanthosis nigricans, keratosis pilaris, skin tags, intertrigo, excessive acne, hirsutism, or violaceous striae of Cushing syndrome
Eyes	Papilledema
Throat	Tonsillar size and abnormal breathing
Neck	Goiter
Chest	Auscultation for rhythm and sounds (heart) and rhonchi, rales, and wheezes (lungs)
Abdomen	Palpation for liver size, right upper quadrant tenderness, and epigastric tenderness
Secondary sexual characteristics	Premature/abnormal appearance of pubic hair, breast development, testicular enlargement, acne or comedones, axillary odor, appearance of micropallus because penis is buried in fat, or gynecomastia
Extremities	Abnormal gait, hip or knee tenderness, limited range of motion in hip (slipped capital femoral epiphyses), Blount disease, joint and foot pain, small hands and feet, polydactyly, lower back pain or limited motion, deep tendon reflexes, or edema
Prader-Willi syndrome	Short stature, acromicria, characteristic facies, hypotonia, and developmental delay
POMC mutation	Red hair, pale skin, low blood pressure or rapid pulse, and corticotropin deficiency/adrenal insufficiency
Albright hereditary osteodystrophy	Developmental delay, short stature, and short fourth and fifth metacarpals
Laurence-Moon or Bardet-Biedl syndrome	Short stature, developmental delay, retinitis pigmentosa, and polydactyly
MC4R mutation	Tall stature and rapid growth
Down syndrome	Typical phenotypic features
Fragile X syndrome	Macroorchia and developmental delay

and age of onset of increased weight velocity are of importance, because early inexorable weight gain is more consistent with a monogenic form of obesity, as noted above.

Waist circumference is now more-frequently invoked as an indicator of comorbidities of obesity, but it does not necessarily add more to the evaluation, compared with BMI, as reviewed above.⁷⁰ Measurement is made horizontally at the level just above the right ileum (reference data are available at www.cdc.gov/growthcharts).

Vital Signs

Pulse should be measured in the standard pediatric manner. At <4 years of age, the heart rate is counted by listening to the heart at approximately the fourth intercostal space, at the midclavicular line, by using the bell device of a pediatric stethoscope. The heart rate should be recorded after the child rests for 4 minutes. At >4 years of age, the radial pulse is measured and compared with age-specific standards. Increased pulse rates in the resting state could be consistent with low fitness levels, whereas decreased pulse rates could be consistent with hypothyroidism.^{342,343} Blood pressure should be measured with a cuff large enough that 80% of the arm is covered by the bladder of the cuff. Very large cuffs are needed for obese youths. Blood pressure is interpreted according to age, gender, and height; reference tables are available.³⁴⁴ Because of "white coat hypertension" (which is defined as blood pressure in the 95th percentile in the physician's office or clinic but normal values during daily life, as assessed with a 24-hour monitor), repeated measurement, after the subject rests for 10 to 15 minutes, often is necessary. Initial volatility of blood pressure is present for 10% to 15% of children and adolescents.³⁴⁵ Automatic measuring devices often are inaccurate, and careful manual evaluation may be indicated. Ambulatory 24-hour monitoring may be necessary if repeated measurements in a standard office situation are not less than 95th percentile. Masked hypertension also may be determined with this method.

Head, Eyes, Ears, Nose, Throat

Optic disks should be observed specifically for the papilledema or decreased venous pulsations of pseudotumor cerebri, particularly if there is a significant history of headache, particularly in the prepubertal population.³⁴⁶ The neck should be examined for goiter. Most acquired hypothyroidism is autoimmune in origin and is associated with a goiter. Hypothyroidism is not a cause of extreme obesity, however, especially in the absence of growth failure. The pharynx should be examined for enlarged tonsils, with observation for obstructed breathing.

Skin

The skin should be examined for acanthosis nigricans, in which hyperpigmented, hyperkeratotic, velvety plaques are found on the dorsal surface of the neck, in the axillae, in body folds, and over joints. The association of acanthosis nigricans with insulin resistance is weaker than thought previously and may be found more often in dark-skinned individuals than in white individuals. Acanthosis nigricans may be a valid indicator of insulin resistance and decreased plasma HDL cholesterol levels in Mexican American adolescents.³⁰⁸ Keratosis pilaris, or skin tags, are a strong sign of insulin resistance.³⁴⁷ Intertrigo and furunculosis may develop independently in

skin folds and increase pigmentation. The deep purple striae of Cushing syndrome, the "buffalo hump" at the back of the neck, and the ruddy complexion and round face of the condition should be assessed, as should xanthelasmas of dyslipidemias.

Cardiopulmonary

The heart should be auscultated for irregular rhythms or sounds and the lungs for pulmonary edema if heart failure is considered. Wheezes of asthma, often associated with or intensified by obesity, should be evaluated. Heart and lung sounds may be difficult to hear.

Abdomen

The abdomen should be examined for organomegaly, especially hepatomegaly of nonalcoholic liver disease.³⁴³ The abdomen may be difficult to palpate because of abdominal girth and excess adiposity.

Secondary Sexual Development

Signs of secondary sexual development should be assessed, including early appearance of pubic hair (<7 years for white girls, <6 years for black girls, or <9 years for boys is presently considered early), early onset of comedones, acne, or axillary odor and hair.³⁴⁸ Obesity often is associated with premature pubarche, which in turn may be an early marker for later polycystic ovary syndrome in girls.³⁴⁹ Early enlargement of the penis in boys (9 years) should be assessed; alternatively, the penis may be partially hidden by fat, which gives it the appearance of being too small when it is normal in size. Early appearance of breast tissue in girls (<7 years for white girls and <6 years for black girls) should be evaluated. This may be a difficult examination, because of adipose tissue that often covers glandular tissue. If the areolae are more pigmented or erectile, however, then there is likely an estrogen effect. Gynecomastia in boys may be false, because of adipose tissue causing the appearance of development or conversion of precursors to estrogen in the local adipose tissue. Hirsutism involving the body or face in girls or excessive acne should be noted as an indication of polycystic ovary disease.³⁴⁹

Extremities

The lower extremities should be evaluated for limitations of motion or pain, including the hips (slipped capital femoral epiphyses), knees (Blount disease), and ankles. Slipped capital femoral epiphyses are indicated by a waddling gait or limited hip motion. Radiograph evaluation may be diagnostic for orthopedic conditions. The lower back should be evaluated through physical examination, as well as history of low back pain. If there is a history of severe trauma to the central nervous system or previous central nervous system surgery near the hypothalamus, then physical evaluation for neurologic signs

TABLE 8 Laboratory Assessments to be Considered in Primary Care Settings

BMI	Tests
>85th–94th percentile, with no risk factors	Fasting lipid levels
>85th–94th percentile, with risk factors (eg, family history of obesity-related diseases, elevated blood pressure, elevated lipid levels, or tobacco use)	Fasting lipid levels, AST and ALT levels, and fasting glucose levels
≥95th percentile	Fasting lipid levels, AST and ALT levels, and fasting glucose levels

AST indicates aspartate aminotransferase; ALT, alanine aminotransferase.

is more important, because of the possibility of hypothalamic damage and increased appetite.³⁵⁰

Signs of Syndromes

Signs of syndromes should be evaluated. Prader-Willi syndrome manifests as short stature, small hands and feet, almond-shaped eyes, round face, hypogonadism, and developmental delay.³⁵¹ *POMC* mutation manifests as red hair and pale skin and is associated with adrenal insufficiency attributable to corticotropin deficiency.³⁵² Pseudohypoparathyroidism, when manifesting as Albright hereditary osteodystrophy, is associated with round face, short fourth and fifth metacarpals, and developmental delay, and it may present with hypocalcemic syndromes.³⁵³ Individuals with Laurence-Moon or Bardet-Biedl syndromes have retinitis pigmentosa, polydactyly with short stature, elevated BMI, and developmental delay.³⁵⁴ *MC4R4* mutation is associated with tall stature and rapid growth, with rapid bone age advancement.³⁵⁵ Mentation may be decreased in other syndromes associated with obesity, such as Down syndrome, Prader-Willi syndrome, and fragile X syndrome (with macroorchia on examination), among others. Hypotonia is also found. Many other syndromes are associated with obesity, and suspicion suggests that the patient be referred to a geneticist.

Laboratory Assessments

For laboratory and radiographic evaluations of childhood obesity, the degree of investigation depends on the BMI, physical and historical findings, and the presence of risk factors. Clinicians should also consider the likely impact on treatment strategies of the results obtained. If results are unlikely to alter treatment, then the value of the testing may be limited. Assessments recommended for primary care professionals (Table 8) and specialists, such as pediatric endocrinologists, geneticists, or pediatric gastroenterologists (Table 9), are indicated. Risk factors, as used below, include family history of obesity-related diseases, including hypertension, early cardiovascular deaths, and strokes, elevated blood pressure (in the patient), hyperlipidemia, and tobacco use.

TABLE 9 Laboratory Assessments to Be Considered by Subspecialists

If cardiac disease is suspected	Electrocardiography, assessing length of QTc interval and cardiac rhythm, and echocardiography; consider measurement of lipoprotein(a)
If blood pressure is elevated	24-h ambulatory blood pressure monitoring
If nonalcoholic fatty liver disease is suspected	Ultrasonography of liver and α_1 -antitrypsin, ceruloplasmin, antinuclear antibody, and hepatitis antibody measurements; liver biopsy if recommended by pediatric gastroenterologist
If goiter is present or hypothyroidism is suspected	Serum free thyroxine measurement or total thyroxine measurement with resin triiodothyronine uptake, serum thyroid-stimulating hormone measurement, and anti[en]thyroid peroxidase and antithyroglobulin antibody measurements
If diabetes is suspected	Glucose tolerance test (measuring insulin levels as well as glucose over 3 h) and urinary microalbumin (first morning void) or microalbumin/creatinine ratio measurement
If sleep apnea is suspected	Polysomnography, oxygen saturation measurement, and carbon dioxide measurement for carbon dioxide retention
If orthopedic disease is suspected	Radiographs of hip, knee, and foot
If Cushing syndrome is suspected	24-h urinary free cortisol measurement or salivary cortisol measurement at bedtime or midnight
If Albright hereditary osteodystrophy is suspected	Serum calcium and phosphate measurements
If hirsutism and oligomenorrhea is present	Plasma 17-hydroxyprogesterone (basal or corticotropin-stimulated), plasma DHEAS (basal or corticotropin-stimulated), androstenedione, testosterone and free testosterone, and sensitive (third-generation) LH and FSH measurements
If precocious puberty is suspected	Sensitive (third-generation) LH and FSH, sensitive testosterone (for boys) or estradiol (for girls), and DHEAS measurements
If specific syndromes are suspected	<i>MC4R</i> evaluation, fluorescent in situ hybridization for Prader-Willi syndrome, or fragile X evaluation (high-resolution chromosomal analysis)

The results of these tests require detailed interpretation. LH indicates luteinizing hormone; FSH, follicle-stimulating hormone; DHEAS, dehydroepiandrosterone sulfate.

For BMI for age of 85th to 94th percentile with no risk factors, a fasting lipid profile should be obtained.⁵³ The American Heart Association and the American Academy of Pediatrics recommend screening at 2 years of age if there is a family history of lipid abnormalities or if risk factors are present in the absence of a positive family history.²⁸²

For BMI for age of 85th to 94th percentile with risk

factors in the history or physical examination, serum chemistry determinations should be performed, in addition, at the time of the fasting lipid profile, including aspartate aminotransferase and alanine aminotransferase measurements for assessment of possible nonalcoholic fatty liver disease. If transaminase levels are normal, then measurements may be repeated every 2 years for obese children after 10 years of age. α_1 -Antitrypsin, ceruloplasmin, antinuclear antibody, or hepatitis antibodies indicate other reasons for elevated liver enzyme levels. Ultrasonography of the liver is more sensitive in detecting nonalcoholic fatty liver disease but does not predict fibrosis. Liver biopsy is the standard method and provides more sensitivity if suggested and performed by a pediatric gastroenterologist. Glucose levels should be measured to determine diabetes mellitus (fasting level: >126 mg/dL; casual level: >200 mg/dL) or impaired glucose tolerance (fasting level: >100 mg/dL; casual level: >140 mg/dL). The American Academy of Pediatrics and the American Diabetes Association recommend empirically that, beginning at 10 years of age or the onset of puberty and every 2 years thereafter, overweight individuals with ≥ 2 risk factors for diabetes (eg, family history, high-risk ethnic/racial group, or signs associated with insulin resistance syndrome) should be tested for T2DM, with fasting plasma glucose measurement as the primary screening test. Fasting plasma insulin measurements are not generally recommended, because of lack of standardization of results and reflection of any medical condition in addition to obesity that predisposes patients to insulin resistance.

For BMI of >95 th percentile, all of the tests listed for the preceding category are recommended, even in the absence of risk factors.⁵³ Urinary microalbumin levels in first morning void or the microalbumin/creatinine ratio can be used to screen for focal segmental glomerulosclerosis, which has been described for obese children.³⁵⁶ Abnormal results are a urinary albumin excretion rate of >20 μ g/minute or a urinary albumin/creatinine ratio of >30 .

The National Cholesterol Education Program guidelines for the metabolic syndrome in adults have been adapted for adolescents and include triglyceride levels of ≥ 110 mg/dL, HDL cholesterol levels of ≤ 40 mg/dL, waist circumference of ≥ 90 th percentile (from National Health and Nutrition Examination Survey III), and blood pressure of ≥ 90 th percentile.³⁵⁷ Although there are no readily available clinical tests for LDL particle size and density, the presence of small, dense, LDL particles in adults is reported in the metabolic syndrome. For children, elevated triglyceride levels and decreased HDL cholesterol levels may serve as proxies for the presence of small, dense, LDL particles.³⁵⁸ Lipoprotein(a) measurements in adults are related to cardiac disease, and levels track from infancy. Obesity increases lipoprotein(a) levels, and it has been suggested that children

with a family history of cardiac disease have lipoprotein(a) levels measured.^{305,359}

If there are appropriate historical features, then specialty tests are indicated. If there is history suggesting sleep apnea (snoring, interrupted breathing while asleep, secondary enuresis, daytime sleepiness, and falling school performance), polysomnography is the standard method for diagnosis.³⁶⁰ Polysomnography may miss cases requiring treatment, however, which indicates the importance of clinical evaluation.³⁶¹ Electrocardiography can be used to search for prolongation of the QTc interval, ventricular arrhythmias, or right ventricular hypertrophy; echocardiography can be performed on the basis of pediatric cardiology consultation. Oxygen saturation can be measured to search for hypoxia, and carbon dioxide values can be measured to search for carbon dioxide retention. If blood pressure is elevated without explanation, then 24-hour ambulatory blood pressure monitoring may be an appropriate first step to rule out white coat volatile hypertension before extensive laboratory evaluation for other causes.

If orthopedic disease is suspected, then appropriate extremity films should be obtained (hip for slipped capital femoral epiphyses, knee for Blount disease, and foot for localized foot pain). Orthopedic consultation may be helpful. If there is a goiter, poor growth, and slow pulse, then free thyroxine and sensitive thyrotropin determinations are indicated (thyroid function tests have low yield in obesity without suggestive findings; hypothyroidism should not cause this extent of obesity, although some coarseness of features may occur).

If Cushing syndrome is suspected, then overnight, dexamethasone-suppressed, early morning, salivary cortisol measurements should be used for screening. Cortisol would not be suppressed, and the subtleties of diagnosis would require a pediatric endocrine consultation.

If Albright hereditary osteodystrophy associated with pseudohypoparathyroidism is suspected, then serum calcium, phosphorus, and parathyroid hormone levels should be measured. Calcium levels would be low, and phosphorous levels would be high.

If hirsutism and excessive acne are seen in a girl with irregular menses (if she is old enough), then the following should be measured in a laboratory with pediatric standards and sensitive methods^{362–365}: (1) serum 17-hydroxyprogesterone levels; basal levels would be high (if the index of suspicion is high, then a corticotropin-stimulated test is indicated); (2) dehydroepiandrosterone sulfate levels; basal levels would be high (if the index of suspicion is high, then a corticotropin-stimulated test is indicated); (3) androstenedione levels; (4) testosterone and free testosterone levels; and (5) third-generation, follicle-stimulating hormone and luteinizing hormone levels. If true precocious puberty is suspected, then the following should be measured³⁶⁶: (1) third-generation, follicle-stimulating hormone and luteinizing

hormone levels and (2) testosterone (boys) or estradiol (girls) levels and dehydroepiandrosterone sulfate levels (both boys and girls).

Genetic tests are not available for all syndromes and mutations but are indicated in the presence of specific findings. *MCR4* mutation could be measured with continuous and rapid weight gain since birth (test available at Athena Diagnostics, Worcester, MA) but the cost is approximately \$1000 and the test cannot yet be recommended widely. Follicle-stimulating hormone testing should be performed if Prader-Willi syndrome is suspected. Fragile X evaluation should be performed if a boy has macroorchidism and developmental delay (see the Appendix in the summary report for the complete expert committee recommendations on the assessment, prevention, and treatment of childhood overweight and obesity).

Summary

This document provides a comprehensive review of the thorough assessment of an overweight or obese child. Although much of the content will be beyond the scope of the primary care setting, the goal is to provide the evidence base and practical considerations for categorization of weight status, identification of targets for behavior change, and assessment of medical risk. Further, the document can serve as a reference for selected, more in-depth evaluations.

The complexity and magnitude of the current epidemic of child and adolescent overweight and obesity likely preclude clinicians from being the sole, or even the major, agents of treatment. Nevertheless, the access to children and their health information, the authority and respect that physicians and other clinicians earn from families, and the potential to apply their knowledge to the very real medical aspects of obesity and its associated conditions, make an imperative that all clinicians be familiar with at least a rudimentary assessment of the overweight or obese child. Furthermore, the well child visit offers a unique opportunity to track a child's growth and to routinely assess for risk from lifestyle practices, family history, or other conditions. Every clinician who provides care to children can use the recommendations in this document, from the simplest screening procedures to more comprehensive evaluation, to guide preventive and therapeutic interventions.

REFERENCES

- Chumlea WC, Guo SS, Kuczmarski RJ, et al. Body composition estimates from NHANES III bioelectrical impedance data. *Int J Obes Relat Metab Disord*. 2002;26:1596–1609
- McCarthy HD, Cole TJ, Fry T, Jebb SA, Prentice AM. Body fat reference curves for children. *Int J Obes (Lond)*. 2006;30:598–602
- Kuczmarski RJ, Ogden CL, Grummer-Strawn LM, et al. CDC growth charts: United States. *Adv Data*. 2000;(314):1–27
- Krebs NF, Jacobson MS, American Academy of Pediatrics. Prevention of pediatric overweight and obesity. *Pediatrics*. 2003;112:424–430
- Himes JH, Dietz WH. Guidelines for overweight in adolescent preventive services: recommendations from an expert committee: the Expert Committee on Clinical Guidelines for Overweight in Adolescent Preventive Services. *Am J Clin Nutr*. 1994;59:307–316
- Koplan JP, Liverman CT, Kraak VI, eds. *Preventing Childhood Obesity: Health in the Balance*. Washington, DC: National Academies Press; 2005
- World Health Organization. *Obesity: Preventing and Managing the Global Epidemic: Report of a WHO Consultation*. Geneva, Switzerland: World Health Organization; 2000
- Hoffmans MD, Kromhout D, de Lezenne Coulander C. The impact of body mass index of 78 612 18-year old Dutch men on 32-year mortality from all causes. *J Clin Epidemiol*. 1988;41:749–756
- Johnson AL, Cornoni JC, Cassel JC, Tyroler HA, Heyden S, Hames CG. Influence of race, sex and weight on blood pressure behavior in young adults. *Am J Cardiol*. 1975;35:523–530
- Hedley AA, Ogden CL, Johnson CL, Carroll MD, Curtin LR, Flegal KM. Prevalence of overweight and obesity among US children, adolescents, and adults, 1999–2002. *JAMA*. 2004;291:2847–2850
- Ogden CL, Carroll MD, Curtin LR, McDowell MA, Tabak CJ, Flegal KM. Prevalence of overweight and obesity in the United States, 1999–2004. *JAMA*. 2006;295:1549–1555
- Dietz WH, Robinson TN. Use of the body mass index (BMI) as a measure of overweight in children and adolescents. *J Pediatr*. 1998;132:191–193
- Freedman DS, Dietz WH, Srinivasan SR, Berenson GS. The relation of overweight to cardiovascular risk factors among children and adolescents: the Bogalusa Heart Study. *Pediatrics*. 1999;103:1175–1182
- Cole TJ, Bellizzi MC, Flegal KM, Dietz WH. Establishing a standard definition for child overweight and obesity worldwide: international survey. *BMJ*. 2000;320:1240–1243
- National Institutes of Health. Clinical guidelines on the identification, evaluation, and treatment of overweight and obesity in adults: the evidence report. *Obes Res*. 1998;6(suppl 2):51S–209S
- Flegal KM, Graubard BI, Williamson DF, Gail MH. Excess deaths associated with underweight, overweight, and obesity. *JAMA*. 2005;293:1861–1867
- Inge TH, Krebs NF, Garcia VF, et al. Bariatric surgery for severely overweight adolescents: concerns and recommendations. *Pediatrics*. 2004;114:217–223
- Freedman DS, Mei Z, Srinivasan SR, Berenson GS, Dietz WH. Cardiovascular risk factors and excess adiposity among overweight children and adolescents: the Bogalusa Heart Study. *J Pediatr*. 2007;150:12–17
- Lohman TG, Roche AF, Martorell R. *Anthropometric Standardization Reference Manual*. Champaign, IL: Human Kinetics Books; 1988
- Bray GA, Bouchard C, James WPT. Definitions and proposed current classifications of obesity. In: Bray GA, Bouchard C, James WPT, eds. *Handbook of Obesity*. New York, NY: Marcel Dekker; 1998:31–40
- Thomas AE, McKay DA, Cutlip MB. A nomograph method for assessing body weight. *Am J Clin Nutr*. 1976;29:302–304
- Malina RM, Bouchard C, Bar-Or O. *Growth, Maturation, and Physical Activity*. 2nd ed. Champaign, IL: Human Kinetics Books; 2004
- Rolland-Cachera MF, Deheeger M, Bellisle F, Sempe M, Guil-loud-Bataille M, Patois E. Adiposity rebound in children: a simple indicator for predicting obesity. *Am J Clin Nutr*. 1984;39:129–135

24. Dietz WH. Periods of risk in childhood for the development of adult obesity: what do we need to learn? *J Nutr*. 1997;127:1884S–1886S
25. Cole TJ. Children grow and horses race: is the adiposity rebound a critical period for later obesity? *BMC Pediatr*. 2004;4:6
26. Stettler N, Stallings VA, Troxel AB, et al. Weight gain in the first week of life and overweight in adulthood: a cohort study of European American subjects fed infant formula. *Circulation*. 2005;111:1897–1903
27. Stettler N, Kumanyika SK, Katz SH, Zemel BS, Stallings VA. Rapid weight gain during infancy and obesity in young adulthood in a cohort of African Americans. *Am J Clin Nutr*. 2003;77:1374–1378
28. Reilly JJ, Armstrong J, Dorosty AR, et al. Early life risk factors for obesity in childhood: cohort study. *BMJ*. 2005;330:1357
29. Ekelund U, Ong K, Linné Y, et al. Upward weight percentile crossing in infancy and early childhood independently predicts fat mass in young adults: the Stockholm Weight Development Study (SWEDES). *Am J Clin Nutr*. 2006;83:324–330
30. Mei Z, Grummer-Strawn LM, Pietrobelli A, Goulding A, Goran MI, Dietz WH. Validity of body mass index compared with other body-composition screening indexes for the assessment of body fatness in children and adolescents. *Am J Clin Nutr*. 2002;75:978–985
31. Pietrobelli A, Faith MS, Allison DB, Gallagher D, Chiumello G, Heymsfield SB. Body mass index as a measure of adiposity among children and adolescents: a validation study. *J Pediatr*. 1998;132:204–210
32. Must A, Strauss RS. Risks and consequences of childhood and adolescent obesity. *Int J Obes Relat Metab Disord*. 1999;23(suppl 2):S2–S11
33. Reilly JJ, Methven E, McDowell ZC, et al. Health consequences of obesity. *Arch Dis Child*. 2003;88:748–752
34. Demerath EW, Schubert CM, Maynard LM, et al. Do changes in body mass index percentile reflect changes in body composition in children? Data from the Fels Longitudinal Study. *Pediatrics*. 2006;117(3). Available at: www.pediatrics.org/cgi/content/full/117/3/e487
35. Maynard LM, Wisemandle W, Roche AF, Chumlea WC, Guo SS, Siervogel RM. Childhood body composition in relation to body mass index. *Pediatrics*. 2001;107:344–350
36. Kraemer HC. *Evaluating Medical Tests: Objective and Quantitative Guidelines*. Newbury Park, CA: Sage Publications; 1992
37. Himes JH. Anthropometric indicators of obesity: epidemiologic and public health aspects of their development and use. In: Pena M, Bacallao J, eds. *Obesity and Poverty: A New Public Health Challenge*. Washington, DC: Pan American Health Organization; 2000:95–100
38. Field AE, Laird N, Steinberg E, Fallon E, Semega-Janneh M, Yanovski JA. Which metric of relative weight best captures body fatness in children? *Obes Res*. 2003;11:1345–1352
39. Zimmermann MB, Gubeli C, Puntener C, Molinari L. Detection of overweight and obesity in a national sample of 6–12-year-old Swiss children: accuracy and validity of reference values for body mass index from the US Centers for Disease Control and Prevention and the International Obesity Task Force. *Am J Clin Nutr*. 2004;79:838–843
40. Ellis KJ, Abrams SA, Wong WW. Monitoring childhood obesity: assessment of the weight/height index. *Am J Epidemiol*. 1999;150:939–946
41. Ehtisham S, Crabtree N, Clark P, Shaw N, Barrett T. Ethnic differences in insulin resistance and body composition in United Kingdom adolescents. *J Clin Endocrinol Metab*. 2005;90:3963–3969
42. Warner JT, Cowan FJ, Dunstan FD, Gregory JW. The validity of body mass index for the assessment of adiposity in children with disease states. *Ann Hum Biol*. 1997;24:209–215
43. Lazarus R, Baur L, Webb K, Blyth F. Body mass index in screening for adiposity in children and adolescents: systematic evaluation using receiver operating characteristic curves. *Am J Clin Nutr*. 1996;63:500–506
44. Daniels SR, Arnett DK, Eckel RH, et al. Overweight in children and adolescents: pathophysiology, consequences, prevention, and treatment. *Circulation*. 2005;111:1999–2012
45. Katzmarzyk PT, Srinivasan SR, Chen W, Malina RM, Boucharde C, Berenson GS. Body mass index, waist circumference, and clustering of cardiovascular disease risk factors in a biracial sample of children and adolescents. *Pediatrics*. 2004;114(2). Available at: www.pediatrics.org/cgi/content/full/114/2/e198
46. Whitlock EP, Williams SB, Gold R, Smith PR, Shipman SA. Screening and interventions for childhood overweight: a summary of evidence for the US Preventive Services Task Force. *Pediatrics*. 2005;116(1). Available at: www.pediatrics.org/cgi/content/full/116/1/e125
47. Guo SS, Wu W, Chumlea WC, Roche AF. Predicting overweight and obesity in adulthood from body mass index values in childhood and adolescence. *Am J Clin Nutr*. 2002;76:653–658
48. Must A, Jacques PF, Dallal GE, Bajema CJ, Dietz WH. Long-term morbidity and mortality of overweight adolescents: a follow-up of the Harvard Growth Study of 1922 to 1935. *N Engl J Med*. 1992;327:1350–1355
49. World Health Organization Expert Committee on Physical Status. *Physical Status: The Use and Interpretation of Anthropometry: Report of a WHO Expert Committee, Vol 854*. Geneva, Switzerland: World Health Organization; 1995
50. Janssen I, Katzmarzyk PT, Srinivasan SR, et al. Utility of childhood BMI in the prediction of adulthood disease: comparison of national and international references. *Obes Res*. 2005;13:1106–1115
51. Harrison GG, Buskirk ER, Carter JEL, et al. Skinfold thicknesses and measurement technique. In: Lohman TG, Roche AF, Martorell R, eds. *Anthropometric Standardization Reference Manual*. Champaign, IL: Human Kinetics Books; 1988:55–69
52. Himes JH. Subcutaneous fat thickness as an indicator of nutritional status. In: Greene LS, Johnston FE, eds. *Social and Biological Predictors of Nutritional Status, Physical Growth and Neurological Development*. New York, NY: Academic Press; 1980:9–32
53. Barlow SE, Dietz WH. Obesity evaluation and treatment: expert committee recommendations: the Maternal and Child Health Bureau, Health Resources and Services Administration and the Department of Health and Human Services. *Pediatrics*. 1998;102(3). Available at: www.pediatrics.org/cgi/content/full/102/3/e29
54. Lohman TG. Anthropometric assessment of fat-free body mass. In: Himes JH, ed. *Anthropometric Assessment of Nutritional Status*. New York, NY: Wiley-Liss; 1991:173–183
55. Roche AF. Anthropometry and ultrasound. In: Roche AF, Heymsfield SB, Lohman TG, eds. *Human Body Composition*. Champaign, IL: Human Kinetics Books; 1996:167–189
56. Chu NF, Rimm EB, Wang DJ, Liou HS, Shieh SM. Relationship between anthropometric variables and lipid levels among school children: the Taipei Children Heart Study. *Int J Obes Relat Metab Disord*. 1998;22:66–72
57. Hansen SE, Hasselstrøm H, Grønfelt V, Froberg K, Andersen LB. Cardiovascular disease risk factors in 6–7-year-old Danish children: the Copenhagen School Child Intervention Study. *Prev Med*. 2005;40:740–746
58. Moran A, Jacobs DR, Steinberger J, et al. Insulin resistance during puberty: results from clamp studies in 357 children. *Diabetes*. 1999;48:2039–2044
59. Vikram NK, Misra A, Pandey RM, Dwivedi M, Luthra K.

- Adiponectin, insulin resistance, and C-reactive protein in postpubertal Asian Indian adolescents. *Metabolism*. 2004;53:1336–1341
60. Bedogni G, Iughetti L, Ferrari M, et al. Sensitivity and specificity of body mass index and skinfold thicknesses in detecting excess adiposity in children aged 8–12 years. *Ann Hum Biol*. 2003;30:132–139
 61. Moreno LA, Pineda I, Rodriguez G, Fleta J, Sarria A, Bueno M. Waist circumference for the screening of the metabolic syndrome in children. *Acta Paediatr*. 2002;91:1307–1312
 62. Sardinha LB, Going SB, Teixeira PJ, Lohman TG. Receiver operating characteristic analysis of body mass index, triceps skinfold thickness, and arm girth for obesity screening in children and adolescents. *Am J Clin Nutr*. 1999;70:1090–1095
 63. Sarria A, Moreno LA, Garcia-Llop LA, Fleta J, Morellón MP, Bueno M. Body mass index, triceps skinfold and waist circumference in screening for adiposity in male children and adolescents. *Acta Paediatr*. 2001;90:387–392
 64. Gower BA, Nagy TR, Goran MI. Visceral fat, insulin sensitivity, and lipids in prepubertal children. *Diabetes*. 1999;48:1515–1521
 65. Larsson B, Svärdsudd K, Welin L, Wilhelmsen L, Björntorp P, Tibblin G. Abdominal adipose tissue distribution, obesity, and risk of cardiovascular disease and death: 13 year follow up of participants in the study of men born in 1913. *Br Med J (Clin Res Ed)*. 1984;288:1401–1404
 66. Brambilla P, Bedogni G, Moreno LA, et al. Crossvalidation of anthropometry against magnetic resonance imaging for the assessment of visceral and subcutaneous adipose tissue in children. *Int J Obes (Lond)*. 2006;30:23–30
 67. Lee S, Bacha F, Gungor N, Arslanian SA. Waist circumference is an independent predictor of insulin resistance in black and white youths. *J Pediatr*. 2006;148:188–194
 68. Maffei C, Pietrobello A, Grezzani A, Provera S, Tato L. Waist circumference and cardiovascular risk factors in prepubertal children. *Obes Res*. 2001;9:179–187
 69. Savva SC, Tornaritis M, Savva ME, et al. Waist circumference and waist-to-height ratio are better predictors of cardiovascular disease risk factors in children than body mass index. *Int J Obes Relat Metab Disord*. 2000;24:1453–1458
 70. Fernández JR, Redden DT, Pietrobello A, Allison DB. Waist circumference percentiles in nationally representative samples of African-American, European-American, and Mexican-American children and adolescents. *J Pediatr*. 2004;145:439–444
 71. Eisenmann JC. Waist circumference percentiles for 7- to 15-year-old Australian children. *Acta Paediatr*. 2005;94:1182–1185
 72. Fredriks AM, van Buuren S, Fekkes M, Verloove-Vanhorick SP, Wit JM. Are age references for waist circumference, hip circumference and waist-hip ratio in Dutch children useful in clinical practice? *Eur J Pediatr*. 2005;164:216–222
 73. McCarthy HD, Jarrett KV, Crawley HF. The development of waist circumference percentiles in British children aged 5.0–16.9 y. *Eur J Clin Nutr*. 2001;55:902–907
 74. Arder CI, Janssen I, Ross R, Katzmarzyk PT. Development of health-related waist circumference thresholds within BMI categories. *Obes Res*. 2004;12:1094–1103
 75. Fulkerson JA, French SA, Story M, Hannan PJ, Neumark-Sztainer D, Himes JH. Weight-bearing physical activity among girls and mothers: relationships to girls' weight status. *Obes Res*. 2004;12:258–266
 76. Fulton JE, Garg M, Galuska DA, Rattay KT, Caspersen CJ. Public health and clinical recommendations for physical activity and physical fitness: special focus on overweight youth. *Sports Med*. 2004;34:581–599
 77. Kohl HW, Hobbs KE. Development of physical activity behaviors among children and adolescents. *Pediatrics*. 1998;101:549–554
 78. McClelland JW, Keenan DP, Lewis J, et al. Review of evaluation tools used to assess the impact of nutrition education on dietary intake and quality, weight management practices, and physical activity of low-income audiences. *J Nutr Educ*. 2001;33(suppl 1):S35–S48
 79. Taylor WC, Sallis JF, Dowda M, Freedson PS, Eason K, Pate RR. Activity patterns and correlates among youth: differences by weight status. *Pediatr Exerc Sci*. 2002;14:418–431
 80. Prochaska JO, DiClemente CC. Transtheoretical therapy: toward a more integrative model change. *Psychotherapy Theory, Research, Practice, Training*. 1982;20:161–173
 81. Prochaska JO, DiClemente CC, Norcross JC. In search of how people change: applications to addictive behaviors. *Am Psychol*. 1992;47:1102–1114
 82. Dietz WH, Robinson TN. Clinical practice: overweight children and adolescents. *N Engl J Med*. 2005;352:2100–2109
 83. Jonides L, Buschbacher V, Barlow SE. Management of child and adolescent obesity: psychological, emotional, and behavioral assessment. *Pediatrics*. 2002;110:215–221
 84. Rollnick S, Mason P, Butler C. *Health Behavior Change: A Guide for Practitioners*. London, United Kingdom: Churchill Livingstone; 1999
 85. Whitlock EP, Orleans CT, Pender N, Allan J. Evaluating primary care behavioral counseling interventions: an evidence-based approach. *Am J Prev Med*. 2002;22:267–284
 86. Burke BL, Arkowitz H, Menchola M. The efficacy of motivational interviewing: a meta-analysis of controlled clinical trials. *J Consult Clin Psychol*. 2003;71:843–861
 87. Emmons KM, Rollnick S. Motivational interviewing in health care settings: opportunities and limitations. *Am J Prev Med*. 2001;20:68–74
 88. Erickson SJ, Gerstle M, Feldstein SW. Brief interventions and motivational interviewing with children, adolescents, and their parents in pediatric health care settings: a review. *Arch Pediatr Adolesc Med*. 2005;159:1173–1180
 89. Rubak S, Sandbaek A, Lauritzen T, Christensen B. Motivational interviewing: a systematic review and meta-analysis. *Br J Gen Pract*. 2005;55:305–312
 90. Scales R, Miller JH. Motivational techniques for improving compliance with an exercise program: skills for primary care clinicians. *Curr Sports Med Rep*. 2003;2:166–172
 91. Sindelar HA, Abrantes AM, Hart C, Lewander W, Spirito A. Motivational interviewing in pediatric practice. *Curr Probl Pediatr Adolesc Health Care*. 2004;34:322–339
 92. Jimmy G, Martin BW. Implementation and effectiveness of a primary care based physical activity counselling scheme. *Patient Educ Couns*. 2005;56:323–331
 93. Cifuentes M, Fernald DH, Green LA, et al. Prescription for health: changing primary care practice to foster healthy behaviors. *Ann Fam Med*. 2005;3(suppl 2):S4–S11
 94. Nicklas TA, Baranowski T, Cullen KW, Berenson G. Eating patterns, dietary quality and obesity. *J Am Coll Nutr*. 2001;20:599–608
 95. Thompson FE, Byers T. Dietary assessment resource manual. *J Nutr*. 1994;124(suppl):2245S–2317S
 96. Soroudi N, Wylie-Rosett J, Mogul D. Quick WAVE screener: a tool to address weight, activity, variety, and excess. *Diabetes Educ*. 2004;30:616–628
 97. Segal-Isaacson CJ, Wylie-Rosett J, Gans KM. Validation of a short dietary assessment questionnaire: the Rapid Eating and Activity Assessment for Participants short version (REAP-S). *Diabetes Educ*. 2004;30:774–778
 98. Prochaska JJ, Sallis JF, Long B. A physical activity screening measure for use with adolescents in primary care. *Arch Pediatr Adolesc Med*. 2001;155:554–559

99. Block G, Gillespie C, Rosenbaum EH, Jenson C. A rapid food screener to assess fat and fruit and vegetable intake. *Am J Prev Med*. 2000;18:284–288
100. Gans KM, Jack B, Lasater TM, Lefebvre RC, McQuade W, Carleton RA. Changing physicians' attitudes, knowledge, and self-efficacy regarding cholesterol screening and management. *Am J Prev Med*. 1993;9:101–106
101. Prochaska JJ, Sallis JF. Reliability and validity of a fruit and vegetable screening measure for adolescents. *J Adolesc Health*. 2004;34:163–165
102. Weinstein SJ, Vogt TM, Gerrior SA. Healthy Eating Index scores are associated with blood nutrient concentrations in the Third National Health and Nutrition Examination Survey. *J Am Diet Assoc*. 2004;104:576–584
103. Kennedy ET, Ohls J, Carlson S, Fleming K. The Healthy Eating Index: design and applications. *J Am Diet Assoc*. 1995;95:1103–1108
104. Randall E, Marshall J, Graham S, Brasure J. Frequency of food use data and the multidimensionality of diet. *J Am Diet Assoc*. 1989;89:1070–1075
105. Nicklas TA, Yang SJ, Baranowski T, Zakeri I, Berenson G. Eating patterns and obesity in children: the Bogalusa Heart Study. *Am J Prev Med*. 2003;25:9–16
106. Storey ML, Forshee RA, Weaver AR, Sansalone WR. Demographic and lifestyle factors associated with body mass index among children and adolescents. *Int J Food Sci Nutr*. 2003;54:491–503
107. Nicklas TA, O'Neil C, Myers L. The importance of breakfast consumption to nutrition of children, adolescents, and young adults. *Nutr Today*. 2004;39:30–39
108. St-Onge MP, Keller KL, Heymsfield SB. Changes in childhood food consumption patterns: a cause for concern in light of increasing body weights. *Am J Clin Nutr*. 2003;78:1068–1073
109. Lin BH, Guthrie J, Frazão E. Nutrient contribution of food away from home. In: Frazão E, ed. *American's Eating Habits: Changes and Consequences*. Washington, DC: US Department of Agriculture; 1999:213–242
110. Paeratakul S, Ferdinand DP, Champagne CM, Ryan DH, Bray GA. Fast-food consumption among US adults and children: dietary and nutrient intake profile. *J Am Diet Assoc*. 2003;103:1332–1338
111. Guthrie JF, Lin BH, Frazão E. Role of food prepared away from home in the American diet, 1977–78 versus 1994–96: changes and consequences. *J Nutr Educ Behav*. 2002;34:140–150
112. Nielsen SJ, Popkin BM. Patterns and trends in food portion sizes, 1977–1998. *JAMA*. 2003;289:450–453
113. Young LR, Nestle M. The contribution of expanding portion sizes to the US obesity epidemic. *Am J Public Health*. 2002;92:246–249
114. Diliberti N, Bordi PL, Conklin MT, Roe LS, Rolls BJ. Increased portion size leads to increased energy intake in a restaurant meal. *Obes Res*. 2004;12:562–568
115. Bowman SA, Gortmaker SL, Ebbeling CB, Pereira MA, Ludwig DS. Effects of fast-food consumption on energy intake and diet quality among children in a national household survey. *Pediatrics*. 2004;113:112–118
116. French SA, Story M, Neumark-Sztainer D, Fulkerson JA, Hannan P. Fast food restaurant use among adolescents: associations with nutrient intake, food choices and behavioral and psychosocial variables. *Int J Obes Relat Metab Disord*. 2001;25:1823–1833
117. Nielsen SJ, Siega-Riz AM, Popkin BM. Trends in food locations and sources among adolescents and young adults. *Prev Med*. 2002;35:107–113
118. Schmidt M, Affenito SG, Striegel-Moore R, et al. Fast-food intake and diet quality in black and white girls: the National Heart, Lung, and Blood Institute Growth and Health Study. *Arch Pediatr Adolesc Med*. 2005;159:626–631
119. Ebbeling CB, Sinclair KB, Pereira MA, Garcia-Lago E, Feldman HA, Ludwig DS. Compensation for energy intake from fast food among overweight and lean adolescents. *JAMA*. 2004;291:2828–2833
120. Thompson OM, Ballew C, Resnicow K, et al. Food purchased away from home as a predictor of change in BMI z-score among girls. *Int J Obes Relat Metab Disord*. 2004;28:282–289
121. Bowman SA, Vinyard BT. Fast food consumption of US adults: impact on energy and nutrient intakes and overweight status. *J Am Coll Nutr*. 2004;23:163–168
122. French SA, Harnack L, Jeffery RW. Fast food restaurant use among women in the Pound of Prevention study: dietary, behavioral and demographic correlates. *Int J Obes Relat Metab Disord*. 2000;24:1353–1359
123. Jeffery RW, French SA. Epidemic obesity in the United States: are fast foods and television viewing contributing? *Am J Public Health*. 1998;88:277–280
124. Kant AK, Graubard BI. Eating out in America, 1987–2000: trends and nutritional correlates. *Prev Med*. 2004;38:243–249
125. McCrory MA, Fuss PJ, Hays NP, Vinken AG, Greenberg AS, Roberts SB. Overeating in America: association between restaurant food consumption and body fatness in healthy adult men and women ages 19 to 80. *Obes Res*. 1999;7:564–571
126. Pereira MA, Kartashov AI, Ebbeling CB, et al. Fast-food habits, weight gain, and insulin resistance (the CARDIA study): 15-year prospective analysis. *Lancet*. 2005;365:36–42
127. Taveras EM, Berkey CS, Rifas-Shiman SL, et al. Association of consumption of fried food away from home with body mass index and diet quality in older children and adolescents. *Pediatrics*. 2005;116(4). Available at: www.pediatrics.org/cgi/content/full/116/4/e518
128. Ludwig DS, Peterson KE, Gortmaker SL. Relation between consumption of sugar-sweetened drinks and childhood obesity: a prospective, observational analysis. *Lancet*. 2001;357:505–508
129. Tordoff MG, Alleva AM. Effect of drinking soda sweetened with aspartame or high-fructose corn syrup on food intake and body weight. *Am J Clin Nutr*. 1990;51:963–969
130. Rolls BJ, Roe LS, Meengs JS. Larger portion sizes lead to a sustained increase in energy intake over 2 days. *J Am Diet Assoc*. 2006;106:543–549
131. Guthrie JF, Morton JF. Food sources of added sweeteners in the diets of Americans. *J Am Diet Assoc*. 2000;100:43–51
132. Cullen KW, Ash DM, Warneke C, de Moor C. Intake of soft drinks, fruit-flavored beverages, and fruits and vegetables by children in grades 4 through 6. *Am J Public Health*. 2002;92:1475–1478
133. Harnack L, Stang J, Story M. Soft drink consumption among US children and adolescents: nutritional consequences. *J Am Diet Assoc*. 1999;99:436–441
134. Mrdjenovic G, Levitsky DA. Nutritional and energetic consequences of sweetened drink consumption in 6- to 13-year-old children. *J Pediatr*. 2003;142:604–610
135. Nielsen SJ, Popkin BM. Changes in beverage intake between 1977 and 2001. *Am J Prev Med*. 2004;27:205–210
136. Raben A, Vasilaras TH, Møller AC, Astrup A. Sucrose compared with artificial sweeteners: different effects on ad libitum food intake and body weight after 10 wk of supplementation in overweight subjects. *Am J Clin Nutr*. 2002;76:721–729
137. Troiano RP, Flegal KM. Overweight children and adolescents: description, epidemiology, and demographics. *Pediatrics*. 1998;101:497–504
138. Rajeshwari R, Yang SJ, Nicklas TA, Berenson GS. Secular trends in children's sweetened-beverage consumption

- (1973–1994): the Bogalusa Heart Study. *J Am Diet Assoc.* 2005; 105:208–214
139. Berkey CS, Rockett HR, Field AE, Gillman MW, Colditz GA. Sugar-added beverages and adolescent weight change. *Obes Res.* 2004;12:778–788
 140. Ebbeling CB, Feldman HA, Osganian SK, Chomitz VR, Ellenbogen SJ, Ludwig DS. Effects of decreasing sugar-sweetened beverage consumption on body weight in adolescents: a randomized, controlled pilot study. *Pediatrics.* 2006;117:673–680
 141. James J, Thomas P, Cavan D, Kerr D. Preventing childhood obesity by reducing consumption of carbonated drinks: cluster randomised controlled trial. *BMJ.* 2004;328:1237
 142. Tam CS, Garnett SP, Cowell CT, Campbell K, Cabrera G, Baur LA. Soft drink consumption and excess weight gain in Australian school students: results from the Nepean study. *Int J Obes (Lond).* 2006;30:1091–1093
 143. Blum JW, Jacobsen DJ, Donnelly JE. Beverage consumption patterns in elementary school aged children across a two-year period. *J Am Coll Nutr.* 2005;24:93–98
 144. Forshee RA, Storey ML. The role of added sugars in the diet quality of children and adolescents. *J Am Coll Nutr.* 2001;20:32–43
 145. Newby PK, Peterson KE, Berkey CS, Leppert J, Willett WC, Colditz GA. Beverage consumption is not associated with changes in weight and body mass index among low-income preschool children in North Dakota. *J Am Diet Assoc.* 2004; 104:1086–1094
 146. Øverby NC, Lillegaard IT, Johansson L, Andersen LF. High intake of added sugar among Norwegian children and adolescents. *Public Health Nutr.* 2004;7:285–293
 147. Phillips SM, Bandini LG, Naumova EN, et al. Energy-dense snack food intake in adolescence: longitudinal relationship to weight and fatness. *Obes Res.* 2004;12:461–472
 148. O'Connor TM, Yang SJ, Nicklas TA. Beverage intake among preschool children and its effects on weight status. *Pediatrics.* 2006;118:1010–1018
 149. Bolton-Smith C, Woodward M. Dietary composition and fat to sugar ratios in relation to obesity. *Int J Obes Relat Metab Disord.* 1994;18:820–828
 150. Gibson SA. Consumption and sources of sugar in the diets of British schoolchildren: are high-sugar diets nutritionally inferior? *J Hum Nutr Diet.* 1993;6:355–371
 151. Lewis CJ, Park YK, Dexter PB, Yetley EA. Nutrient intakes and body weights of persons consuming high and moderate levels of added sugars. *J Am Diet Assoc.* 1992;92:708–713
 152. Bachman CM, Baranowski T, Nicklas TA. Is there an association between sweetened beverages and adiposity? *Nutr Rev.* 2006;64:153–174
 153. Malik VS, Schulze MB, Hu FB. Intake of sugar-sweetened beverages and weight gain: a systematic review. *Am J Clin Nutr.* 2006;84:274–288
 154. US Department of Health and Human Services, US Department of Agriculture. *Dietary Guidelines for Americans, 2005.* 6th ed. Washington, DC: US Government Printing Office; 2005
 155. American Academy of Pediatrics, Committee on Nutrition. The use and misuse of fruit juice in pediatrics. *Pediatrics.* 2001;107:1210–1213
 156. Dennison BA, Rockwell HL, Baker SL. Excess fruit juice consumption by preschool-aged children is associated with short stature and obesity. *Pediatrics.* 1997;99:15–22
 157. Dennison BA, Rockwell HL, Nichols MJ, Jenkins P. Children's growth parameters vary by type of fruit juice consumed. *J Am Coll Nutr.* 1999;18:346–352
 158. Tanasescu M, Ferris AM, Himmelgreen DA, Rodriguez N, Pérez-Escamilla R. Biobehavioral factors are associated with obesity in Puerto Rican children. *J Nutr.* 2000;130:1734–1742
 159. Alexy U, Sichert-Hellert W, Kersting M, Manz F, Schöch G. Fruit juice consumption and the prevalence of obesity and short stature in German preschool children: results of the DONALD Study: Dortmund Nutritional and Anthropometrical Longitudinally Designed. *J Pediatr Gastroenterol Nutr.* 1999; 29:343–349
 160. Forshee RA, Storey ML. Total beverage consumption and beverage choices among children and adolescents. *Int J Food Sci Nutr.* 2003;54:297–307
 161. Kloeblen-Tarver AS. Fruit juice consumption not related to growth among preschool-aged children enrolled in the WIC program. *J Am Diet Assoc.* 2001;101:996
 162. Skinner JD, Carruth BR. A longitudinal study of children's juice intake and growth: the juice controversy revisited. *J Am Diet Assoc.* 2001;101:432–437
 163. Skinner JD, Carruth BR, Moran J, Houck K, Coletta F. Fruit juice intake is not related to children's growth. *Pediatrics.* 1999;103:58–64
 164. Kral TV, Roe LS, Rolls BJ. Combined effects of energy density and portion size on energy intake in women. *Am J Clin Nutr.* 2004;79:962–968
 165. Kral TV, Rolls BJ. Energy density and portion size: their independent and combined effects on energy intake. *Physiol Behav.* 2004;82:131–138
 166. Rolls BJ, Morris EL, Roe LS. Portion size of food affects energy intake in normal-weight and overweight men and women. *Am J Clin Nutr.* 2002;76:1207–1213
 167. Rolls BJ, Roe LS, Meengs JS. Reductions in portion size and energy density of foods are additive and lead to sustained decreases in energy intake. *Am J Clin Nutr.* 2006;83:11–17
 168. Rolls BJ, Roe LS, Kral TV, Meengs JS, Wall DE. Increasing the portion size of a packaged snack increases energy intake in men and women. *Appetite.* 2004;42:63–69
 169. Rolls BJ, Roe LS, Meengs JS, Wall DE. Increasing the portion size of a sandwich increases energy intake. *J Am Diet Assoc.* 2004;104:367–372
 170. Orlet Fisher J, Rolls BJ, Birch LL. Children's bite size and intake of an entrée are greater with large portions than with age-appropriate or self-selected portions. *Am J Clin Nutr.* 2003;77:1164–1170
 171. Rolls BJ, Engell D, Birch LL. Serving portion size influences 5-year-old but not 3-year-old children's food intakes. *J Am Diet Assoc.* 2000;100:232–234
 172. McConahy KL, Smiciklas-Wright H, Mitchell DC, Picciano MF. Portion size of common foods predicts energy intake among preschool-aged children. *J Am Diet Assoc.* 2004;104: 975–979
 173. McConahy KL, Smiciklas-Wright H, Birch LL, Mitchell DC, Picciano MF. Food portions are positively related to energy intake and body weight in early childhood. *J Pediatr.* 2002; 140:340–347
 174. Bell EA, Castellanos VH, Pelkman CL, Thorwart ML, Rolls BJ. Energy density of foods affects energy intake in normal-weight women. *Am J Clin Nutr.* 1998;67:412–420
 175. Bell EA, Rolls BJ. Energy density of foods affects energy intake across multiple levels of fat content in lean and obese women. *Am J Clin Nutr.* 2001;73:1010–1018
 176. Rolls BJ, Bell EA, Castellanos VH, Chow M, Pelkman CL, Thorwart ML. Energy density but not fat content of foods affected energy intake in lean and obese women. *Am J Clin Nutr.* 1999;69:863–871
 177. Stubbs RJ, Johnstone AM, O'Reilly LM, Barton K, Reid C. The effect of covertly manipulating the energy density of mixed diets on ad libitum food intake in "pseudo free-living" humans. *Int J Obes Relat Metab Disord.* 1998;22:980–987
 178. Poppitt SD, Prentice AM. Energy density and its role in the control of food intake: evidence from metabolic and community studies. *Appetite.* 1996;26:153–174

179. Rolls BJ, Bell EA. Dietary approaches to the treatment of obesity. *Med Clin North Am*. 2000;84:401–418
180. Yao M, Roberts SB. Dietary energy density and weight regulation. *Nutr Rev*. 2001;59:247–258
181. Rolls BJ, Roe LS, Meengs JS. Salad and satiety: energy density and portion size of a first-course salad affect energy intake at lunch. *J Am Diet Assoc*. 2004;104:1570–1576
182. Rolls BJ, Roe LS, Beach AM, Kris-Etherton PM. Daily consumption of a low-energy-dense food enhances long-term weight loss. *Obes Res*. 2004;12:A55
183. Ello-Martin JA, Roe LS, Rolls BJ. A diet reduced in energy density results in greater weight loss than a diet reduced in fat. *Obes Res*. 2004;12:A23
184. Bandini LG, Vu D, Must A, Cyr H, Goldberg A, Dietz WH. Comparison of high-calorie, low-nutrient-dense food consumption among obese and non-obese adolescents. *Obes Res*. 1999;7:438–443
185. Kant AK. Reported consumption of low-nutrient-density foods by American children and adolescents: nutritional and health correlates, NHANES III, 1988 to 1994. *Arch Pediatr Adolesc Med*. 2003;157:789–796
186. Rolls BJ, Drewnowski A, Ledikwe JH. Changing the energy density of the diet as a strategy for weight management. *J Am Diet Assoc*. 2005;105(suppl 1):S98–S103
187. Ledikwe JH, Blanck HM, Khan LK, et al. Dietary energy density determined by eight calculation methods in a nationally representative United States population. *J Nutr*. 2005;135:273–278
188. Ledikwe JH, Blanck HM, Khan LK, et al. Low-energy-density diets are associated with high diet quality in adults in the United States. *J Am Diet Assoc*. 2006;106:1172–1180
189. Newby PK. Examining energy density: comments on diet quality, dietary advice, and the cost of healthful eating. *J Am Diet Assoc*. 2006;106:1166–1169
190. Krebs-Smith SM, Cook A, Subar AF, Cleveland L, Friday J, Kahle LL. Fruit and vegetable intakes of children and adolescents in the United States. *Arch Pediatr Adolesc Med*. 1996;150:81–86
191. Tohill BC, Seymour J, Serdula M, Kettel-Khan L, Rolls BJ. What epidemiologic studies tell us about the relationship between fruit and vegetable consumption and body weight. *Nutr Rev*. 2004;62:365–374
192. Appleby PN, Thorogood M, Mann JI, Key TJ. Low body mass index in non-meat eaters: the possible roles of animal fat, dietary fibre and alcohol. *Int J Obes Relat Metab Disord*. 1998;22:454–460
193. Bazzano LA, He J, Ogden LG, et al. Fruit and vegetable intake and risk of cardiovascular disease in US adults: the first National Health and Nutrition Examination Survey Epidemiologic Follow-up Study. *Am J Clin Nutr*. 2002;76:93–99
194. Flood A, Velie EM, Chatterjee N, et al. Fruit and vegetable intakes and the risk of colorectal cancer in the Breast Cancer Detection Demonstration Project follow-up cohort. *Am J Clin Nutr*. 2002;75:936–943
195. Kann L, Kinchen SA, Williams BI, et al. Youth risk behavior surveillance: United States, 1999. *MMWR CDC Surveill Summ*. 2000;49:1–32
196. Kennedy E, Bowman S. Assessment of the effect of fat-modified foods on diet quality in adults, 19 to 50 years, using data from the Continuing Survey of Food Intake by Individuals. *J Am Diet Assoc*. 2001;101:455–460
197. Lahti-Koski M, Pietinen P, Heliövaara M, Vartiainen E. Associations of body mass index and obesity with physical activity, food choices, alcohol intake, and smoking in the 1982–1997 FINRISK Studies. *Am J Clin Nutr*. 2002;75:809–817
198. Lin BH, Morrison RM. Higher fruit consumption linked with lower body mass index. *Food Rev*. 2002;25:28–32
199. Maskarinec G, Novotny R, Tasaki K. Dietary patterns are associated with body mass index in multiethnic women. *J Nutr*. 2000;130:3068–3072
200. Neumark-Sztainer D, Story M, Resnick MD, Blum RW. Correlates of inadequate fruit and vegetable consumption among adolescents. *Prev Med*. 1996;25:497–505
201. Ortega RM, Redondo MR, Zamora MJ, López-Sobaler AM, Andrés P. Eating behavior and energy and nutrient intake in overweight/obese and normal-weight Spanish elderly. *Ann Nutr Metab*. 1995;39:371–378
202. Ortega RM, Requejo AM, Andrés P, López-Sobaler AM, Redondo R, González-Fernández M. Relationship between diet composition and body mass index in a group of Spanish adolescents. *Br J Nutr*. 1995;74:765–773
203. Pérez CE. Fruit and vegetable consumption. *Health Rep*. 2002;13:23–31
204. Rockett HR, Berkey CS, Field AE, Colditz GA. Cross-sectional measurement of nutrient intake among adolescents in 1996. *Prev Med*. 2001;33:27–37
205. Wardle J, Sanderson S, Guthrie CA, Rapoport L, Plomin R. Parental feeding style and the inter-generational transmission of obesity risk. *Obes Res*. 2002;10:453–462
206. Boutelle K, Neumark-Sztainer D, Story M, Resnick M. Weight control behaviors among obese, overweight, and nonoverweight adolescents. *J Pediatr Psychol*. 2002;27:531–540
207. Hanley AJ, Harris SB, Gittelsohn J, Wolever TM, Saksvig B, Zinman B. Overweight among children and adolescents in a Native Canadian community: prevalence and associated factors. *Am J Clin Nutr*. 2000;71:693–700
208. Heitmann BL, Harris JR, Lissner L, Pedersen NL. Genetic effects on weight change and food intake in Swedish adult twins. *Am J Clin Nutr*. 1999;69:597–602
209. Liu S, Manson JE, Lee IM, et al. Fruit and vegetable intake and risk of cardiovascular disease: the Women's Health Study. *Am J Clin Nutr*. 2000;72:922–928
210. Macdiarmid JI, Vail A, Cade JE, Blundell JE. The sugar-fat relationship revisited: differences in consumption between men and women of varying BMI. *Int J Obes Relat Metab Disord*. 1998;22:1053–1061
211. Parker DR, Gonzalez S, Derby CA, Gans KM, Lasater TM, Carleton RA. Dietary factors in relation to weight change among men and women from two southeastern New England communities. *Int J Obes Relat Metab Disord*. 1997;21:103–109
212. Rissanen TH, Voutilainen S, Virtanen JK, et al. Low intake of fruits, berries and vegetables is associated with excess mortality in men: the Kuopio Ischaemic Heart Disease Risk Factor (KIHD) Study. *J Nutr*. 2003;133:199–204
213. Schulz M, Kroke A, Liese AD, Hoffmann K, Bergmann MM, Boeing H. Food groups as predictors for short-term weight changes in men and women of the EPIC-Potsdam cohort. *J Nutr*. 2002;132:1335–1340
214. Serdula MK, Byers T, Mokdad AH, Simoes E, Mendlein JM, Coates RJ. The association between fruit and vegetable intake and chronic disease risk factors. *Epidemiology*. 1996;7:161–165
215. Terry P, Giovannucci E, Michels KB, et al. Fruit, vegetables, dietary fiber, and risk of colorectal cancer. *J Natl Cancer Inst*. 2001;93:525–533
216. Wolfe WS, Campbell CC, Frongillo EA, Haas JD, Melnik TA. Overweight schoolchildren in New York State: prevalence and characteristics. *Am J Public Health*. 1994;84:807–813
217. Rolls BJ, Ello-Martin JA, Tohill BC. What can intervention studies tell us about the relationship between fruit and vegetable consumption and weight management? *Nutr Rev*. 2004;62:1–17
218. Gortmaker SL, Cheung LW, Peterson KE, et al. Impact of a school-based interdisciplinary intervention on diet and physical activity among urban primary school children: Eat Well

- and Keep Moving. *Arch Pediatr Adolesc Med.* 1999;153:975–983
219. Boynton-Jarrett R, Thomas TN, Peterson KE, Wiecha J, Sobol AM, Gortmaker SL. Impact of television viewing patterns on fruit and vegetable consumption among adolescents. *Pediatrics.* 2003;112:1321–1326
 220. Luepker RV, Perry CL, McKinlay SM, et al. Outcomes of a field trial to improve children's dietary patterns and physical activity: the Child and Adolescent Trial for Cardiovascular Health. *JAMA.* 1996;275:768–776
 221. Epstein LH, Gordy CC, Raynor HA, Beddome M, Kilanowski CK, Paluch R. Increasing and decreasing fat and sugar intake in families at risk for childhood obesity. *Obes Res.* 2001;9:171–178
 222. Nicklas TA, Bao W, Webber LS, Berenson GS. Breakfast consumption affects adequacy of total daily intake in children. *J Am Diet Assoc.* 1993;93:886–891
 223. Sampson AE, Dixit S, Meyers AF, Houser R. The nutritional impact of breakfast consumption on the diets of inner-city African-American elementary school children. *J Natl Med Assoc.* 1995;87:195–202
 224. Siega-Riz AM, Popkin BM, Carson T. Trends in breakfast consumption for children in the United States from 1965–1991. *Am J Clin Nutr.* 1998;67:748S–756S
 225. Cho S, Dietrich M, Brown CJ, Clark CA, Block G. The effect of breakfast type on total daily energy intake and body mass index: results from the Third National Health and Nutrition Examination Survey (NHANES III). *J Am Coll Nutr.* 2003;22:296–302
 226. Morgan KJ, Zabik ME, Stampely GL. The role of breakfast in diet adequacy of the US adult population. *J Am Coll Nutr.* 1986;5:551–563
 227. Nicklas TA, Myers L, Reger C, Beech B, Berenson GS. Impact of breakfast consumption on nutritional adequacy of the diets of young adults in Bogalusa, Louisiana: ethnic and gender contrasts. *J Am Diet Assoc.* 1998;98:1432–1438
 228. Nielsen SJ, Siega-Riz AM, Popkin BM. Trends in energy intake in U.S. between 1977 and 1996: similar shifts seen across age groups. *Obes Res.* 2002;10:370–378
 229. Gibson SA, O'Sullivan KR. Breakfast cereal consumption patterns and nutrient intakes of British schoolchildren. *J R Soc Health.* 1995;115:366–370
 230. Ortega RM, Requejo AM, López-Sobaler AM, et al. Difference in the breakfast habits of overweight/obese and normal weight schoolchildren. *Int J Vitam Nutr Res.* 1998;68:125–132
 231. Pastore DR, Fisher M, Friedman SB. Abnormalities in weight status, eating attitudes, and eating behaviors among urban high school students: correlations with self-esteem and anxiety. *J Adolesc Health.* 1996;18:312–319
 232. Summerbell CD, Moody RC, Shanks J, Stock MJ, Geissler C. Relationship between feeding pattern and body mass index in 220 free-living people in four age groups. *Eur J Clin Nutr.* 1996;50:513–519
 233. Ma Y, Bertone ER, Stanek EJ, et al. Association between eating patterns and obesity in a free-living US adult population. *Am J Epidemiol.* 2003;158:85–92
 234. Berkey CS, Rockett HR, Gillman MW, Field AE, Colditz GA. Longitudinal study of skipping breakfast and weight change in adolescents. *Int J Obes Relat Metab Disord.* 2003;27:1258–1266
 235. Schlundt DG, Hill JO, Sbrocco T, Pope-Cordle J, Sharp T. The role of breakfast in the treatment of obesity: a randomized clinical trial. *Am J Clin Nutr.* 1992;55:645–651
 236. Murphy JM, Pagano ME, Nachmani J, Sperling P, Kane S, Kleinman RE. The relationship of school breakfast to psychosocial and academic functioning: cross-sectional and longitudinal observations in an inner-city school sample. *Arch Pediatr Adolesc Med.* 1998;152:899–907
 237. Pollitt E, Mathews R. Breakfast and cognition: an integrative summary. *Am J Clin Nutr.* 1998;67:804S–813S
 238. Wyatt HR, Grunwald GK, Mosca CL, Klem ML, Wing RR, Hill JO. Long-term weight loss and breakfast in subjects in the National Weight Control Registry. *Obes Res.* 2002;10:78–82
 239. Fábry P, Hejda S, Cerný K, Osancová K, Pechar J. Effect of meal frequency in schoolchildren: changes in weight-height proportion and skinfold thickness. *Am J Clin Nutr.* 1966;18:358–361
 240. Toschke AM, Küchenhoff H, Koletzko B, von Kries R. Meal frequency and childhood obesity. *Obes Res.* 2005;13:1932–1938
 241. Fábry P, Hejl Z, Fodor J, Braun T, Zvolankova K. The frequency of meals: its relation to overweight, hypercholesterolaemia, and decreased glucose-tolerance. *Lancet.* 1964;2:614–615
 242. Fábry P, Tepperman J. Meal frequency: a possible factor in human pathology. *Am J Clin Nutr.* 1970;23:1059–1068
 243. Nicklas TA, Morales M, Linares A, et al. Children's meal patterns have changed over a 21-year period: the Bogalusa Heart Study. *J Am Diet Assoc.* 2004;104:753–761
 244. Jahns L, Siega-Riz AM, Popkin BM. The increasing prevalence of snacking among US children from 1977 to 1996. *J Pediatr.* 2001;138:493–498
 245. Zizza C, Siega-Riz AM, Popkin BM. Significant increase in young adults' snacking between 1977–1978 and 1994–1996 represents a cause for concern! *Prev Med.* 2001;32:303–310
 246. Hampl JS, Heaton CL, Taylor CA. Snacking patterns influence energy and nutrient intakes but not body mass index. *J Hum Nutr Diet.* 2003;16:3–11
 247. Basdevant A, Craplet C, Guy-Grand B. Snacking patterns in obese French women. *Appetite.* 1993;21:17–23
 248. Bertéus Forslund H, Torgerson JS, Sjöström L, Lindroos AK. Snacking frequency in relation to energy intake and food choices in obese men and women compared to a reference population. *Int J Obes (Lond).* 2005;29:711–719
 249. Baranowski T, Mendlein J, Resnicow K, Frank E, Weber-Cullen KW, Baranowski J. Physical activity and nutrition in children and youth: an overview of obesity prevention. *Prev Med.* 2000;31:S1–S10
 250. Kelder SH, Perry CL, Klepp KI, Lytle LL. Longitudinal tracking of adolescent smoking, physical activity, and food choice behaviors. *Am J Public Health.* 1994;84:1121–1126
 251. Malina RM. Tracking of physical activity and physical fitness across the lifespan. *Res Q Exerc Sport.* 1996;67(suppl):S48–S57
 252. Rice MH, Howell CC. Measurement of physical activity, exercise, and physical fitness in children: issues and concerns. *J Pediatr Nurs.* 2000;15:148–156
 253. Kohl HW, Fulton JE, Caspersen CJ. Assessment of physical activity among children and adolescents: a review and synthesis. *Prev Med.* 2000;31:S54–S76
 254. Duke J, Huhman M, Heitzler C. Physical activity levels among children aged 9–13 years: United States, 2002. *MMWR Morb Mortal Wkly Rep.* 2003;52:785–788
 255. Grunbaum JA, Kann L, Kinchen S, et al. Youth risk behavior surveillance: United States, 2003. *MMWR Surveill Summ.* 2004;53:1–96
 256. Goran MI. Measurement issues related to studies of childhood obesity: assessment of body composition, body fat distribution, physical activity, and food intake. *Pediatrics.* 1998;101:505–518
 257. National Institute of Diabetes and Digestive and Kidney Diseases. *The Science of Energy Balance, Calorie Intake and Physical Activity: A Teacher's Guide.* 2005. Bethesda, MD: National Institutes of Health; 2005. NIH publication 05–5169

258. Caspersen CJ, Powell KE, Christenson GM. Physical activity, exercise, and physical fitness: definitions and distinctions for health-related research. *Public Health Rep.* 1985;100:126–131
259. Dubbert PM. Exercise in behavioral medicine. *J Consult Clin Psychol.* 1992;60:613–618
260. Sirard JR, Pate RR. Physical activity assessment in children and adolescents. *Sports Med.* 2001;31:439–454
261. Welk GJ, Corbin CB, Dale D. Measurement issues in the assessment of physical activity in children. *Res Q Exerc Sport.* 2000;71(suppl):S59–S73
262. Noland M, Danner F, DeWalt K, McFadden M, Kotchen JM. The measurement of physical activity in young children. *Res Q Exerc Sport.* 1990;61:146–153
263. Koo MM, Rohan TE. Comparison of four habitual physical activity questionnaires in girls aged 7–15 yr. *Med Sci Sports Exerc.* 1999;31:421–427
264. Burdette HL, Whitaker RC, Daniels SR. Parental report of outdoor playtime as a measure of physical activity in pre-school-aged children. *Arch Pediatr Adolesc Med.* 2004;158:353–357
265. Sallis JF, Saelens BE. Assessment of physical activity by self-report: status, limitations, and future directions. *Res Q Exerc Sport.* 2000;71(suppl):S1–S14
266. Ortega-Sanchez R, Jimenez-Mena C, Cordoba-Garcia R, Muñoz-Lopez J, Garcia-Machado ML, Vilaseca-Canals J. The effect of office-based physician's advice on adolescent exercise behavior. *Prev Med.* 2004;38:219–226
267. Patrick K, Sallis JF, Prochaska JJ, et al. A multicomponent program for nutrition and physical activity change in primary care: PACE+ for adolescents. *Arch Pediatr Adolesc Med.* 2001;155:940–946
268. Bjornson KF. Physical activity monitoring in children and youths. *Pediatr Phys Ther.* 2005;17:37–45
269. Jago R, Watson K, Baranowski T, et al. Pedometer reliability, validity and daily activity targets among 10- to 15-year-old boys. *J Sports Sci.* 2006;24:241–251
270. McDonald CM, Widman L, Abresch RT, Walsh SA, Walsh DD. Utility of a step activity monitor for the measurement of daily ambulatory activity in children. *Arch Phys Med Rehabil.* 2005;86:793–801
271. Ramírez-Marrero FA, Smith BA, Sherman WM, Kirby TE. Comparison of methods to estimate physical activity and energy expenditure in African American children. *Int J Sports Med.* 2005;26:363–371
272. Tudor-Locke C, Williams JE, Reis JP, Pluto D. Utility of pedometers for assessing physical activity: construct validity. *Sports Med.* 2004;34:281–291
273. Tudor-Locke CE, Myers AM. Methodological considerations for researchers and practitioners using pedometers to measure physical (ambulatory) activity. *Res Q Exerc Sport.* 2001;72:1–12
274. Dennison BA, Erb TA, Jenkins PL. Television viewing and television in bedroom associated with overweight risk among low-income preschool children. *Pediatrics.* 2002;109:1028–1035
275. Kalakanis LE, Goldfield GS, Paluch RA, Epstein LH. Parental activity as a determinant of activity level and patterns of activity in obese children. *Res Q Exerc Sport.* 2001;72:202–209
276. Byers T, Nestle M, McTiernan A, et al. American Cancer Society guidelines on nutrition and physical activity for cancer prevention: reducing the risk of cancer with healthy food choices and physical activity. *CA Cancer J Clin.* 2002;52:92–119
277. US Department of Health and Human Services. *The Surgeon General's Call to Action to Prevent and Decrease Overweight and Obesity.* Rockville, MD: US Department of Health and Human Services; 2001
278. Williams CL, Hayman LL, Daniels SR, et al. Cardiovascular health in childhood: a statement for health professionals from the Committee on Atherosclerosis, Hypertension, and Obesity in the Young (AHOY) of the Council on Cardiovascular Disease in the Young, American Heart Association. *Circulation.* 2002;106:143–160
279. Strong WB, Malina RM, Blimkie CJ, et al. Evidence based physical activity for school-age youth. *J Pediatr.* 2005;146:732–737
280. National Association for Sport and Physical Education. *Physical Activity for Children: A Statement of Guidelines for Children Ages 5 to 12.* 2nd ed. Reston, VA: National Association for Sport and Physical Education; 2004
281. Cavill N, Biddle S, Sallis JF. Health enhancing physical activity for young people: statement of the United Kingdom Expert Consensus Conference. *Pediatr Exerc Sci.* 2001;13:12–25
282. American Academy of Pediatrics, Committee on Practice and Ambulatory Medicine. Recommendations for preventative pediatric health care. *Pediatrics.* 2000;105:645–646
283. Epstein LH, Paluch RA, Kalakanis LE, Goldfield GS, Cerny FJ, Roemmich JN. How much activity do youth get? A quantitative review of heart-rate measured activity. *Pediatrics.* 2001;108(3). Available at: www.pediatrics.org/cgi/content/full/108/3/e44
284. Anderson RE, Crespo CJ, Bartlett SJ, Cheskin LJ, Pratt M. Relationship of physical activity and television watching with body weight and level of fatness among children: results from the Third National Health and Nutrition Examination Survey. *JAMA.* 1998;279:938–942
285. Rideout VJ, Vandewater VJ, Wartella EA. *Zero to Six: Electronic Media in the Lives of Infants, Toddlers, and Preschoolers.* Menlo Park, CA: Henry J. Kaiser Family Foundation; 2003
286. Robinson TN. Reducing children's television viewing to prevent obesity: a randomized controlled trial. *JAMA.* 1999;282:1561–1567
287. Gortmaker SL, Peterson K, Wiecha J, et al. Reducing obesity via a school-based interdisciplinary intervention among youth: Planet Health. *Arch Pediatr Adolesc Med.* 1999;153:409–418
288. DuRant RH, Baranowski T, Johnson M, Thompson WO. The relationship among television watching, physical activity, and body composition of young children. *Pediatrics.* 1994;94:449–455
289. Feldman DE, Barnett T, Shrier I, Rossignol M, Abenhaim L. Is physical activity differentially associated with different types of sedentary pursuits? *Arch Pediatr Adolesc Med.* 2003;157:797–802
290. Epstein LH, Roemmich JN, Stein RI, Paluch RA, Kilanowski CK. The challenge of identifying behavioral alternatives to food: clinic and field studies. *Ann Behav Med.* 2005;30:201–209
291. Barsh GS, Farooqi IS, O'Rahilly S. Genetics of body-weight regulation. *Nature.* 2000;404:644–651
292. Whitaker RC, Wright JA, Pepe MS, Seidel KD, Dietz WH. Predicting obesity in young adulthood from childhood and parental obesity. *N Engl J Med.* 1997;337:869–873
293. Giampietro O, Virgone E, Carneglia L, Griesi E, Calvi D, Matteucci E. Anthropometric indices of school children and familial risk factors. *Prev Med.* 2002;35:492–498
294. Berkowitz RI, Stallings VA, Maislin G, Stunkard AJ. Growth of children at high risk of obesity during the first 6 y of life: implications for prevention. *Am J Clin Nutr.* 2005;81:140–146
295. Maes HH, Neale MC, Eaves LJ. Genetic and environmental factors in relative body weight and human adiposity. *Behav Genet.* 1997;27:325–351
296. Hebebrand J, Sommerlad C, Geller F, Görg T, Hinney A. The

- genetics of obesity: practical implications. *Int J Obes Relat Metab Disord.* 2001;25(suppl 1):S10–S18
297. Rosenbaum M, Leibel RL, Hirsch J. Obesity. *N Engl J Med.* 1997;337:396–407
 298. Ravussin E, Bogardus C. Energy balance and weight regulation: genetics versus environment. *Br J Nutr.* 2000;83(suppl 1):S17–S20
 299. Pérusse L, Chagnon YC, Weisnagel SJ, et al. The human obesity gene map: the 2000 update. *Obes Res.* 2001;9:135–169
 300. Fagot-Campagna A, Pettitt DJ, Engelgau MM, et al. Type 2 diabetes among North American children and adolescents: an epidemiologic review and a public health perspective. *J Pediatr.* 2000;136:664–672
 301. Malecka-Tendera E, Erhardt E, Molnar D. Type 2 diabetes mellitus in European children and adolescents. *Acta Paediatr.* 2005;94:543–546
 302. Saaddine JB, Fagot-Campagna A, Rolka D, et al. Distribution of HbA_{1c} levels for children and young adults in the U.S.: Third National Health and Nutrition Examination Survey. *Diabetes Care.* 2002;25:1326–1330
 303. Cruz ML, Weigensberg MJ, Huang TT, Ball G, Shaibi GQ, Goran MI. The metabolic syndrome in overweight Hispanic youth and the role of insulin sensitivity. *J Clin Endocrinol Metab.* 2004;89:108–113
 304. Urakami T, Kubota S, Nitadori Y, Harada K, Owada M, Kitagawa T. Annual incidence and clinical characteristics of type 2 diabetes in children as detected by urine glucose screening in the Tokyo metropolitan area. *Diabetes Care.* 2005;28:1876–1881
 305. Glowinska B, Urban M, Koput A. Cardiovascular risk factors in children with obesity, hypertension and diabetes: lipoprotein(a) levels and body mass index correlate with family history of cardiovascular disease. *Eur J Pediatr.* 2002;161:511–518
 306. Muratova VN, Islam SS, Demerath EW, Minor VE, Neal WA. Cholesterol screening among children and their parents. *Prev Med.* 2001;33:1–6
 307. Dennison BA, Kikuchi DA, Srinivasan SR, Webber LS, Berenson GS. Parental history of cardiovascular disease as an indication for screening for lipoprotein abnormalities in children. *J Pediatr.* 1989;115:186–194
 308. Urrutia-Rojas X, Menchaca J, Wadley W, et al. Cardiovascular risk factors in Mexican-American children at risk for type 2 diabetes mellitus (T2DM). *J Adolesc Health.* 2004;34:290–299
 309. Robinson RF, Batisky DL, Hayes JR, Nahata MC, Mahan JD. Body mass index in primary and secondary pediatric hypertension. *Pediatr Nephrol.* 2004;19:1379–1384
 310. Robinson RF, Batisky DL, Hayes JR, Nahata MC, Mahan JD. Significance of heritability in primary and secondary pediatric hypertension. *Am J Hypertens.* 2005;18:917–921
 311. Simsolo RB, Romo MM, Rabinovich L, Bonanno M, Grunfeld B. Family history of essential hypertension versus obesity as risk factors for hypertension in adolescents. *Am J Hypertens.* 1999;12:260–263
 312. Beebe DW, Lewin D, Zeller M, et al. Sleep in overweight adolescents: shorter sleep, poorer sleep quality, sleepiness, and sleep-disordered breathing. *J Pediatr Psychol.* 2007;32:69–79
 313. Marik PE. Leptin, obesity, and obstructive sleep apnea. *Chest.* 2000;118:569–571
 314. Chay OM, Goh A, Abisheganaden J, et al. Obstructive sleep apnea syndrome in obese Singapore children. *Pediatr Pulmonol.* 2000;29:284–290
 315. Marcus CL, Curtis S, Koerner CB, Joffe A, Serwint JR, Loughlin GM. Evaluation of pulmonary function and polysomnography in obese children and adolescents. *Pediatr Pulmonol.* 1996;21:176–183
 316. Wing YK, Hui SH, Pak WM, et al. A controlled study of sleep related disordered breathing in obese children. *Arch Dis Child.* 2003;88:1043–1047
 317. Atras WS, Hammer LD, McNicholas F, Kraemer HC. Risk factors for childhood overweight: a prospective study from birth to 9.5 years. *J Pediatr.* 2004;145:20–25
 318. Durmer JS, Dinges DF. Neurocognitive consequences of sleep deprivation. *Semin Neurol.* 2005;25:117–129
 319. von Kries R, Toschke AM, Wurmser H, Sauerwald T, Koletzko B. Reduced risk for overweight and obesity in 5- and 6-year-old children by duration of sleep: a cross-sectional study. *Int J Obes Relat Metab Disord.* 2002;26:710–716
 320. Gupta NK, Mueller WH, Chan W, Meininger JC. Is obesity associated with poor sleep quality in adolescents? *Am J Hum Biol.* 2002;14:762–768
 321. Rosmond R, Björntorp P. The role of antidepressants in the treatment of abdominal obesity. *Med Hypotheses.* 2000;54:990–994
 322. Sutcliffe JG, de Lecea L. The hypocretins: excitatory neuromodulatory peptides for multiple homeostatic systems, including sleep and feeding. *J Neurosci Res.* 2000;62:161–168
 323. Wurtman RJ, Wurtman JJ. Brain serotonin, carbohydrate-craving, obesity and depression. *Adv Exp Med Biol.* 1996;398:35–41
 324. Ehrmann DA. Polycystic ovary syndrome. *N Engl J Med.* 2005;352:1223–1236
 325. Marion AW, Baker AJ, Dhawan A. Fatty liver disease in children. *Arch Dis Child.* 2004;89:648–652
 326. Strauss RS, Barlow SE, Dietz WH. Prevalence of abnormal serum aminotransferase values in overweight and obese adolescents. *J Pediatr.* 2000;136:727–733
 327. Lenders CM, McElrath TF, Scholl TO. Nutrition in adolescent pregnancy. *Curr Opin Pediatr.* 2000;12:291–296
 328. Puhl RM, Brownell KD. Psychosocial origins of obesity stigma: toward changing a powerful and pervasive bias. *Obes Rev.* 2003;4:213–227
 329. Kraig KA, Keel PK. Weight-based stigmatization in children. *Int J Obes Relat Metab Disord.* 2001;25:1661–1666
 330. Latner JD, Stunkard AJ. Getting worse: the stigmatization of obese children. *Obes Res.* 2003;11:452–456
 331. Davison KK, Birch LL. Weight status, parent reaction, and self-concept in five-year-old girls. *Pediatrics.* 2001;107:46–53
 332. Strauss RS. Childhood obesity and self-esteem. *Pediatrics.* 2000;105(1). Available at: www.pediatrics.org/cgi/content/full/105/1/e15
 333. French SA, Story M, Perry CL. Self-esteem and obesity in children and adolescents: a literature review. *Obes Res.* 1995;3:479–490
 334. Erickson SJ, Robinson TN, Haydel KF, Killen JD. Are overweight children unhappy? Body mass index, depressive symptoms, and overweight concerns in elementary school children. *Arch Pediatr Adolesc Med.* 2000;154:931–935
 335. Fabricatore AN, Wadden TA. Psychological aspects of obesity. *Clin Dermatol.* 2004;22:332–337
 336. Greenberg I, Perna F, Kaplan M, Sullivan MA. Behavioral and psychological factors in the assessment and treatment of obesity surgery patients. *Obes Res.* 2005;13:244–249
 337. Hsu LK, Mulliken B, McDonagh B, et al. Binge eating disorder in extreme obesity. *Int J Obes Relat Metab Disord.* 2002;26:1398–1403
 338. Williamson DA, Martin CK. Binge eating disorder: a review of the literature after publication of DSM-IV. *Eat Weight Disord.* 1999;4:103–114
 339. Pierce JW, Wardle J. Cause and effect beliefs and self-esteem of overweight children. *J Child Psychol Psychiatry.* 1997;38:645–650
 340. US Preventive Services Task Force. Screening and interven-

- tions for overweight in children and adolescents: recommendation statement. *Pediatrics*. 2005;116:205–209
341. Roche AF, Davila GH. Differences between recumbent length and stature within individuals. *Growth*. 1974;38:313–320
 342. Nguyen TT, Keil MF, Russell DL, et al. Relation of acanthosis nigricans to hyperinsulinemia and insulin sensitivity in overweight African American and white children. *J Pediatr*. 2001;138:474–480
 343. Riley MR, Bass NM, Rosenthal P, Merriman RB. Underdiagnosis of pediatric obesity and underscreening for fatty liver disease and metabolic syndrome by pediatricians and pediatric subspecialists. *J Pediatr*. 2005;147:839–842
 344. National High Blood Pressure Education Program Working Group on High Blood Pressure in Children and Adolescents. The fourth report on the diagnosis, evaluation, and treatment of high blood pressure in children and adolescents. *Pediatrics*. 2004;114(suppl):555–576
 345. Stergiou GS, Yiannes NJ, Rarra VC, Alamara CV. White-coat hypertension and masked hypertension in children. *Blood Press Monit*. 2005;10:297–300
 346. Kesler A, Fattal-Valevski A. Idiopathic intracranial hypertension in the pediatric population. *J Child Neurol*. 2002;17:745–748
 347. Jabbour SA. Cutaneous manifestations of endocrine disorders: a guide for dermatologists. *Am J Clin Dermatol*. 2003;4:315–331
 348. Styne DM. Puberty, obesity and ethnicity. *Trends Endocrinol Metab*. 2004;15:472–478
 349. Ibáñez L, de Zegher F, Potau N. Anovulation after precocious pubarche: early markers and time course in adolescence. *J Clin Endocrinol Metab*. 1999;84:2691–2695
 350. Lustig RH, Mietus-Snyder ML, Bacchetti P, Lazar AA, Velasquez-Mieyer PA, Christensen ML. Insulin dynamics predict body mass index and z-score response to insulin suppression or sensitization pharmacotherapy in obese children. *J Pediatr*. 2006;148:23–29
 351. Gunay-Aygun M, Schwartz S, Heeger S, O'Riordan MA, Cassidy SB. The changing purpose of Prader-Willi syndrome clinical diagnostic criteria and proposed revised criteria. *Pediatrics*. 2001;108(5). Available at: www.pediatrics.org/cgi/content/full/108/5/e92
 352. Krude H, Biebermann H, Luck W, Horn R, Brabant G, Grüters A. Severe early-onset obesity, adrenal insufficiency and red hair pigmentation caused by *POMC* mutations in humans. *Nat Genet*. 1998;19:155–157
 353. De Sanctis L, Romagnolo D, Olivero M, et al. Molecular analysis of the *GNAS1* gene for the correct diagnosis of Albright hereditary osteodystrophy and pseudohypoparathyroidism. *Pediatr Res*. 2003;53:749–755
 354. Moore SJ, Green JS, Fan Y, et al. Clinical and genetic epidemiology of Bardet-Biedl syndrome in Newfoundland: a 22-year prospective, population-based, cohort study. *Am J Med Genet A*. 2005;132:352–360
 355. Vaisse C, Clement K, Durand E, Herberg S, Guy-Grand B, Froguel P. Melanocortin-4 receptor mutations are a frequent and heterogeneous cause of morbid obesity. *J Clin Invest*. 2000;106:253–262
 356. Adelman RD, Restaino IG, Alon US, Blowey DL. Proteinuria and focal segmental glomerulosclerosis in severely obese adolescents. *J Pediatr*. 2001;138:481–485
 357. Cook S, Weitzman M, Auinger P, Nguyen M, Dietz WH. Prevalence of a metabolic syndrome phenotype in adolescents: findings from the third National Health and Nutrition Examination Survey, 1988–1994. *Arch Pediatr Adolesc Med*. 2003;157:821–827
 358. Kojima M, Kanno H, Yamazaki Y, Koyama S, Kanazawa S, Arisaka O. Association of low-density lipoprotein particle size distribution and cardiovascular risk factors in children. *Acta Paediatr*. 2005;94:281–286
 359. Wang XL, Wang J. Lipoprotein(a) in children and adolescence. *Pediatr Endocrinol Rev*. 2003;1:109–119
 360. American Academy of Pediatrics, Section on Pediatric Pulmonology, Subcommittee on Obstructive Sleep Apnea Syndrome. Clinical practice guideline: diagnosis and management of childhood obstructive sleep apnea syndrome. *Pediatrics*. 2002;109:704–712
 361. Ray RM, Bower CM. Pediatric obstructive sleep apnea: the year in review. *Curr Opin Otolaryngol Head Neck Surg*. 2005;13:360–365
 362. Dunaif A, Thomas A. Current concepts in the polycystic ovary syndrome. *Annu Rev Med*. 2001;52:401–419
 363. Ehrmann DA, Liljenquist DR, Kasza K, et al. Prevalence and predictors of the metabolic syndrome in women with polycystic ovary syndrome. *J Clin Endocrinol Metab*. 2006;91:48–53
 364. New MI. Diagnosis and management of congenital adrenal hyperplasia. *Annu Rev Med*. 1998;49:311–328
 365. New MI, Wilson RC. Steroid disorders in children: congenital adrenal hyperplasia and apparent mineralocorticoid excess. *Proc Natl Acad Sci USA*. 1999;96:12790–12797
 366. Grumbach MM, Styne DM. Puberty: ontogeny, neuroendocrinology, physiology, and disorders. In: Larsen PR, Kronenberg HM, Melmed S, Polonsky KS, eds. *Williams Textbook of Endocrinology*. 10th ed. Philadelphia, PA: WB Saunders; 2003: 1115–1286

Assessment of Child and Adolescent Overweight and Obesity

Nancy F. Krebs, John H. Himes, Dawn Jacobson, Theresa A. Nicklas, Patricia Guilday and Dennis Styne

Pediatrics 2007;120;S193-S228

DOI: 10.1542/peds.2007-2329D

Updated Information & Services

including high-resolution figures, can be found at:
http://www.pediatrics.org/cgi/content/full/120/Supplement_4/S193

References

This article cites 338 articles, 121 of which you can access for free at:
http://www.pediatrics.org/cgi/content/full/120/Supplement_4/S193#BIBL

Post-Publication Peer Reviews (P³Rs)

One P³R has been posted to this article:
http://www.pediatrics.org/cgi/eletters/120/Supplement_4/S193

Subspecialty Collections

This article, along with others on similar topics, appears in the following collection(s):
Nutrition & Metabolism
http://www.pediatrics.org/cgi/collection/nutrition_and_metabolism

Permissions & Licensing

Information about reproducing this article in parts (figures, tables) or in its entirety can be found online at:
<http://www.pediatrics.org/misc/Permissions.shtml>

Reprints

Information about ordering reprints can be found online:
<http://www.pediatrics.org/misc/reprints.shtml>

American Academy of Pediatrics

DEDICATED TO THE HEALTH OF ALL CHILDREN™

