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SUPPLEMENT ARTICLE

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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.

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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

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OBESE 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 impedence 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 charts[3] 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, ≥85th or ≥95th 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/m^2 or is ≥95th percentile for age and gender (whichever is smaller) should be considered overweight.[5] The BMI limit of 30 kg/m^2 was recommended because, at the oldest ages of adolescence for which the 95th percentile values exceed 30 kg/m^2 (>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 ≥85th 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 ≥95th 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/m^2 or ≥95th 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 ≥85th percentile but <95th percentile or 30 kg/m^2 (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/m^2 and ≥30 kg/m^2 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/m^2 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 ≥95th 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 >97th percentile (the highest level on the CDC growth charts). Inge et al[17] 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 ≥99th 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 be 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.nhlbihin.net/bmi_palm.htm). If BMI is calculated from height and weight measured in inches and pounds, then the formula is BMI = [weight (in pounds)/[height (in inches)]^2] × 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

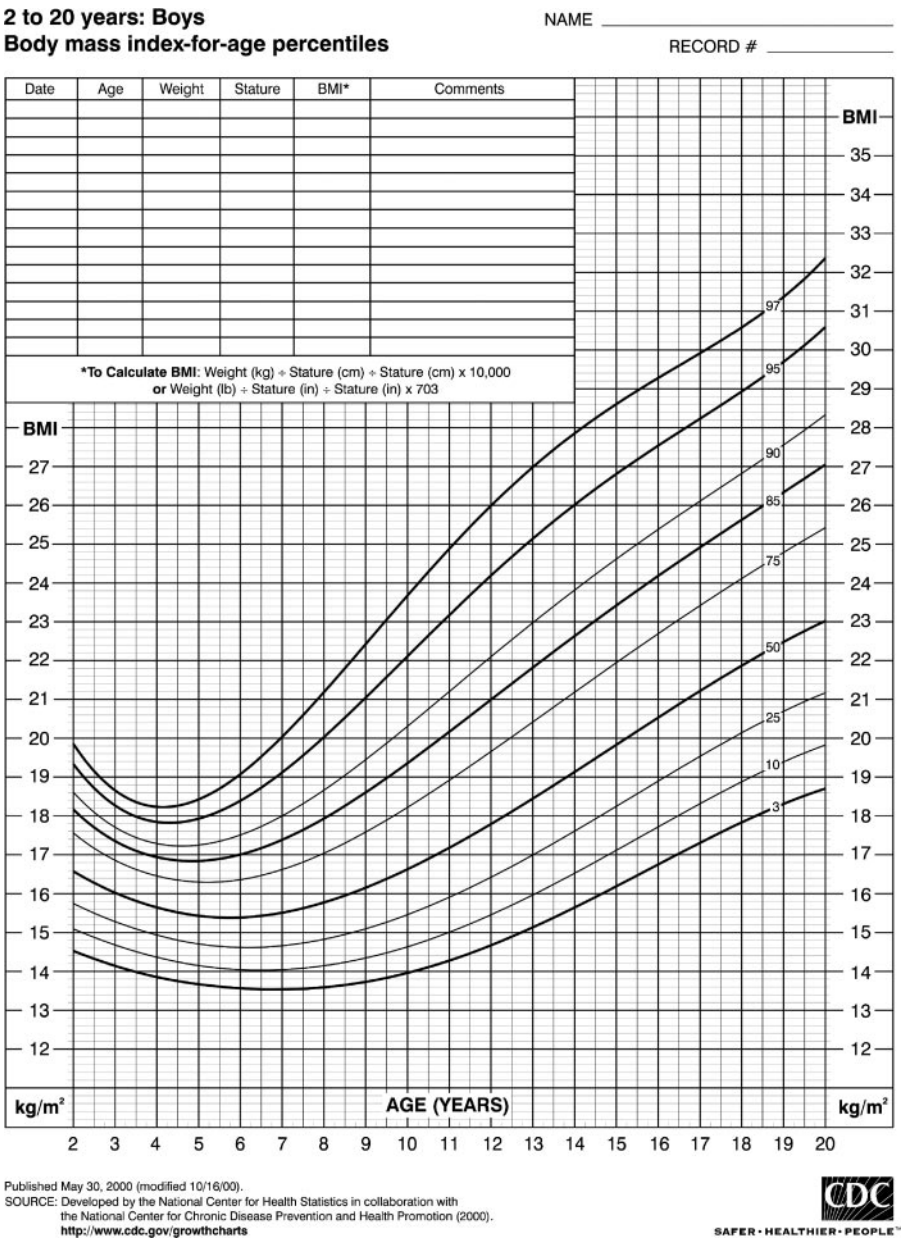

FIGURE 1
BMI percentiles for age for boys 2 to 20 years of age. (Reproduced with permission from Kuczmarski RJ, Ogden CL, Grummer-Strawn LM, et al. *Adv Data.* 2000;(314):1–27.)

children may have relatively high weights primarily because of high lean mass, rather than high body fat levels. This is most common among male adolescents, for whom gains in BMI during adolescence may have a large component of lean mass.[34,35] At ≥95th percentile, however, almost all of those who are identified as obese on the basis of BMI have high weights because they have high total body fat levels. In clinical practice, the important question is whether the criteria for overweight and obese that are based only on the upper portion of the BMI distribution (ie, ≥85th percentile or ≥95th percentile) identify correctly the fattest children and those at greatest health risk.

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 − 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 ≥95th 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, ≥95th percentile versus ≥85th 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 al[45] 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 ≥85th percentile were 69% and 76%, respectively, and those for BMI of ≥95th 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 ≥85th and ≥95th percentiles. Therefore, even among children with ≥3 risk factors, the least-restrictive and therefore most-sensitive BMI cutoff point (BMI of ≥85th percentile) still identified correctly only approximately two thirds. Moreover, of all children with BMI of ≥85th 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 al[47] analyzed lifelong data from the Fels Longitudinal Study and estimated the probabilities of having a BMI of ≥30 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 ≥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 ≥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 al[48] 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 al[61] 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 children[70] 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 al[84] 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. Therefore, 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 adults[109] 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 al[114] 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 children[120] 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 al[126] 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 al[116] 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 al[140] 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 al[151] 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 only[157] was associated positively with BMI in samples of children 2 to 5 years of age. Tanasescu et al[158] 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 portion 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 al[176] 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 study[189] 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 Morrison[198] 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 al[221] 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 snackers[248] 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, ~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 age[272] take between 12 000 and 16 000 steps per day.[271,273] Jago et al[269] 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 Bjornson[268] (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?[84] 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 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.

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]

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 al[290] 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 al[75] 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.

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]

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. Six have been documented in the literature,[92,96,98,263,266,267] and all except one[96] are directed toward adolescent assessment. One additional research study tool for preschool-aged children may be adaptable to the clinic[264] (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 al[264] 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 al[84] 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 Bogardus[298] 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, Alstorm, 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 al[309] 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 recommend 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 serotonin[321] and other neuropeptides that affect eating behavior.[322] Serotonin has been implicated in both within-meal and postmeal 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 polydypsia	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 study[328] provided preliminary evidence that genetic susceptibility testing for obesity 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.

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 violacious 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 microphallus 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, acromicia, 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 pigmentosum, and polydactyly
MC4R mutation	Tall stature and rapid growth
Down syndrome	Typical phenotypic features
Fragile X syndrome	Macroorchia and developmental delay

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 height[341] 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 = weight (in kilograms)/[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 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 skinfolds 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] *MC_4R4* 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, ceruloplasm, 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 triidothyronine 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	*MCR4* 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, ceruloplasm, 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 >95th 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 μg/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 ≥90th percentile (from National Health and Nutrition Examination Survey III), and blood pressure of ≥90th 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 measured[366]: (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.

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