Screening and Interventions for Childhood Overweight: A Summary of Evidence for the US Preventive Services Task Force

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ABSTRACT. Background. Childhood and adolescent overweight and obesity are related to health risks, medical conditions, and increased risk of adult obesity, with its attendant effects on morbidity and mortality rates. The prevalence of childhood overweight and obesity has more than doubled in the past 25 years.

Purpose. This evidence synthesis examines the evidence for the benefits and harms of screening and early treatment of overweight among children and adolescents in clinical settings.

Methods. We developed an analytic framework and 7 key questions representing the logical evidence connecting screening and weight control interventions with changes in overweight and behavioral, physiologic, and health outcomes in childhood or adulthood. We searched the Cochrane Library from 1996 to April 2004. We searched Medline, PsycINFO, DARE, and CINAHL from 1966 to April 2004. One reviewer abstracted relevant information from each included article into standardized evidence tables, and a second reviewer checked key elements. Two reviewers quality-graded each article with US Preventive Services Task Force criteria.

Results. Although BMI is a measure of relative weight rather than adiposity, it is recommended widely for use among children and adolescents to determine overweight and is the currently preferred measure. The risk of adult overweight from childhood overweight provides the best available evidence to judge the clinical validity of BMI as an overweight criterion for children and adolescents. BMI measures in childhood track to adulthood moderately or very well, with stronger tracking seen for children with ≥1 obese parent and children who are more overweight or older. The probability of adult obesity (BMI of ≥30 kg/m2) is ≥50% among children >13 years of age whose BMI percentiles meet or exceed the 95th percentile for age and gender. BMI-based overweight categorization for individuals, particularly for racial/ethnic minorities with differences in body composition, may have limited validity because BMI measures cannot differentiate between increased weight for height attributable to relatively greater fat-free mass (muscle, bone, and fluids) and that attributable to greater fat. No trials of screening programs to identify and to treat childhood overweight have been reported. Limited research is available on effective, generalizable interventions for overweight children and adolescents that can be conducted in primary care settings or through primary care referrals.

Conclusions. BMI measurements of overweight among older adolescents identify those at increased risk of developing adult obesity. Interventions to treat overweight adolescents in clinical settings have not been shown to have clinically significant benefits, and they are not widely available. Screening to categorize overweight among children under age 12 or 13 who are not clearly overweight may not provide reliable risk categorization for adult obesity. Screening in this age group is compromised by the fact that there is little generalizable evidence for primary care interventions. Because existing trials report modest short- to medium-term improvements (~10–20% decrease in percentage of overweight, or weight control interventions with a small effect size), we recommend future trials to evaluate interventions for overweight among children younger than age 12 or 13 and among older adolescents who can be treated in primary care settings or through primary care referrals.

O besity/overweight has been declared an epidemic1-3 and a “public health crisis” among children worldwide4 due to an alarming increase in its prevalence. Overweight among children (defined by experts as a BMI of ≥95th percentile for age and gender)5,6 ≥2 years of age has at least doubled in the past 25 years (Fig 1). The age- and gender-specific mean BMIs and the proportion of children with BMIs of ≥95th percentile increased markedly among children from the mid-1970s to the 1990s, with almost all of this increase occurring among children in the upper half of the BMI distribution.7 Therefore, ~50% of children appear to have “obesity susceptibility genes” on which environmental changes have acted in the past 25 years.8

Because increases in mean BMI have occurred primarily because of increases in the upper half of the BMI distribution,7,9 weight-related health consequences will become increasingly common among children. The health consequences of childhood overweight and obesity include pulmonary, orthopedic, gastroenterologic, neurologic, and endocrine

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Abbreviations. USPSTF, US Preventive Services Task Force; NHANES, National Health and Nutrition Examination Survey; CDC, Centers for Disease Control and Prevention.
conditions and cardiovascular risk factors.\textsuperscript{4,10–15} Tables 1 and 2 contain the limited prevalence data for key morbidities and risk factors available from recent summaries. Rarely, severe childhood obesity is associated with immediate morbidity resulting from conditions such as slipped capital femoral epiphysis,\textsuperscript{16} whereas steatohepatitis and sleep apnea are more common.\textsuperscript{17–21} Medical conditions new to this age group, such as type 2 diabetes mellitus,\textsuperscript{22} represent “adult” morbidities that are now seen more frequently among overweight adolescents.\textsuperscript{23} Most medical complications, however, do not become clinically apparent for decades.\textsuperscript{10}

Overweight is associated with a higher prevalence of intermediate metabolic consequences and risk factors, such as insulin resistance, elevated blood lipid levels, increased blood pressure, and impaired glucose tolerance.\textsuperscript{24–29} Perhaps the most significant short-term morbidities for overweight/obese children are psychosocial and include social marginalization, decreased self-esteem, and decreased quality of life.\textsuperscript{30–33} Risk factors for developing childhood overweight include increased parental adiposity, low parental education, social deprivation, and perhaps infant feeding patterns, early or more rapid puberty, extreme birth weights, gestational diabetes, and various social and environmental factors, such as childhood diet and time spent in sedentary behaviors.\textsuperscript{4}

The US Preventive Services Task Force (USPSTF) makes recommendations about clinical preventive services to assist primary care clinicians using an explicit, evidence-based approach. In 1996, the USPSTF recommended periodic height and weight measurements for all patients (B recommendation).\textsuperscript{34} Comparing height and weight measurements against appropriate age and gender normative values to determine additional evaluation, intervention, or referral was recommended, with BMI (>85th percentile) for adolescents and weight and height (or length, as appropriate) plotted on growth charts or compared with average weight tables for age, gender, and height for younger children. Previously, the USPSTF has not made separate recommendations about screening criteria or specific interventions for overweight or obesity in childhood populations. To assist the USPSTF in making its recommendation, the Oregon Evidence-Based Practice Center undertook a systematic review and summary of the strength of the evidence concerning screening and interventions for overweight in childhood populations. We combined the findings of prior fair- or good-quality\textsuperscript{35} systematic evidence reviews with fair- to good-quality studies not covered in these reviews or published subsequently.

METHODS

Terminology

Because BMI is the primary clinical measure and is a measure of relative weight, we adopted the use of the term “overweight” for children, as opposed to obesity.\textsuperscript{7} Considering the limitations of BMI in defining adiposity and concerns about labeling (stigma or concern resulting from being labeled obese), overweight is more accurate than obesity when the designation is based on a BMI value alone. Using accepted conventions, we use “overweight” to describe those with \(\geq 95\text{th} \) percentile BMI for age and gender and “at risk for overweight” to describe those in the 85th to 95th percentile for age and gender.\textsuperscript{5,6}

Key Questions and Analytic Framework

We developed an analytic framework (Fig 2) and 7 key questions, with USPSTF methods, to guide our literature search.\textsuperscript{35} The first key question examined direct evidence that screening to identify and to treat overweight among children and adolescents improves age-appropriate behavioral, anthropometric, or physiologic measures. Because we found no evidence addressing this key question, we searched for indirect evidence for key questions 2 through 6, to estimate the benefits and harms of overweight screening and interventions. Key question 2 concerned appropriate standards for overweight among children and adolescents, the overweight prevalence based on appropriate standards, and validity of clinical screening tests for predicting poorer health outcomes and obesity in adulthood. Key question 3 examined adverse effects of screening for overweight. Key questions 4 and 5 examined the efficacy of behavioral counseling, pharmacotherapeutic, and surgical interventions for improving age-appropriate anthro-
<table>
<thead>
<tr>
<th>Health Condition</th>
<th>Population Source</th>
<th>Age/Race Ethnicity/Gender</th>
<th>Level of Overweight</th>
<th>Prevalence, %</th>
<th>Reference Cited in Source Bibliography</th>
</tr>
</thead>
<tbody>
<tr>
<td>Diabetes mellitus type 2</td>
<td>Community (N = 142)</td>
<td>Navajo Native American, 12–19 y</td>
<td>1.4</td>
<td></td>
<td>Lobstein et al14 (2004)</td>
</tr>
<tr>
<td></td>
<td>NHANES III (N = 2867)</td>
<td>Non-Hispanic white, non-Hispanic black, Mexican American adolescents, 12–19 y</td>
<td>0.04 (includes type 1 diabetes mellitus)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Diabetes mellitus type 2</td>
<td>Obesity clinic (N = 55)</td>
<td>Multiethnic boys and girls, 4–10 y</td>
<td>BMI &gt;95th percentile for age and gender</td>
<td>0</td>
<td>Sinha et al14 (2002)</td>
</tr>
<tr>
<td></td>
<td>Obesity clinic (N = 112)</td>
<td>Multiethnic boys and girls, 11–18 y</td>
<td>BMI &gt;95th percentile</td>
<td>4 (all were non-Hispanic black or Hispanic)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Sleep-associated abnormal breathing tests</td>
<td>Obese children referred for history of sleep disordered breathing (N = 32)</td>
<td>196 ± 45% mean IBW</td>
<td>40–90</td>
<td>Silvestri et al17 (1993)</td>
</tr>
<tr>
<td></td>
<td>≥1 abnormal polysomnography test</td>
<td>Obesity clinic (N = 222)</td>
<td>“Severe obesity,” &gt;150% IBW; mean: 208% IBW</td>
<td>6.8 (calc),</td>
<td>Mallory et al18 (1989)</td>
</tr>
<tr>
<td></td>
<td>Severe abnormal tests</td>
<td>Children and adolescents, 3–20 y</td>
<td>BMI &lt;85th percentile</td>
<td>1 (calc)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>(severe sleep apnea)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>≥1 abnormal polysomnography test</td>
<td>Obesity clinic patients with history of sleep disordered breathing (N = 41)</td>
<td>32</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Severe abnormal tests</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Nonalcoholic fatty liver disease</td>
<td>Steatosis by ultrasonography</td>
<td>General pediatric (N = 810)</td>
<td>Population sample</td>
<td>3</td>
<td>Tominaga et al19 (1995)</td>
</tr>
<tr>
<td></td>
<td>Steatosis by ultrasonography</td>
<td>Japanese school boys and girls, 4–12 y</td>
<td>Obese, &gt;120% IBW</td>
<td>53</td>
<td>Franzese et al20 (1997)</td>
</tr>
<tr>
<td></td>
<td>Steatosis and elevated transaminases (presumptive NASH)</td>
<td>Obesity clinic (N = 72)</td>
<td>Obese, &gt;120% IBW</td>
<td></td>
<td>Of these, 32% had elevated transaminases (calc)</td>
</tr>
<tr>
<td></td>
<td>Steatosis by ultrasonography</td>
<td>Italian boys and girls, 4.5–15.9 y</td>
<td>Obese, &gt;120% IBW</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Slipped capital femoral epiphysis</td>
<td></td>
<td></td>
<td>24</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>3.4 cases per 100,000 children (50–70% are “obese”)</td>
<td>Kelsey16 (1973)</td>
</tr>
</tbody>
</table>

Childhood morbidities discussed in reviews without reported prevalence were binge-eating disorders and low self-esteem. IBW indicates ideal body weight; NASH, nonalcoholic steatohepatitis; calc, calculated.

* The highest-risk population in the world.
<table>
<thead>
<tr>
<th>Risk Factor</th>
<th>Population Source</th>
<th>Age/Race Ethnicity/Gender</th>
<th>Level of Overweight (Percentile of BMI for Age and Gender if Given)</th>
<th>Prevalence, %</th>
<th>Reference Cited in Source Bibliography</th>
</tr>
</thead>
<tbody>
<tr>
<td>Impaired glucose tolerance</td>
<td>Obesity clinic (N = 55)</td>
<td>Multiethnic boys and girls, 4-10 y</td>
<td>&gt;95th</td>
<td>25</td>
<td>Sinha et al(^{24}) (2002)</td>
</tr>
<tr>
<td>Hyperinsulinemia</td>
<td>Obesity clinic (N = 112)</td>
<td>Multiethnic boys and girls, 11-18 y</td>
<td>&gt;95th</td>
<td>21</td>
<td>Sinha et al(^{24}) (2002)</td>
</tr>
<tr>
<td>Hyperinsulinemia Insulin levels &gt;95th percentile</td>
<td>Bogalusa Heart Study</td>
<td>Black and white boys and girls, 5-10 y</td>
<td>&lt;95th</td>
<td>4</td>
<td>Freedman et al(^{25}) (2002)</td>
</tr>
<tr>
<td>Metabolic syndrome Hypertension, hypertriglyceridemia, low HDL cholesterol, hyperinsulinemia</td>
<td>NHANES</td>
<td>Adolescent boys and girls, 12-19 y</td>
<td>&lt;85th</td>
<td>0.1</td>
<td>Cook et al(^{26}) (2003)</td>
</tr>
<tr>
<td>Metabolic syndrome Hypertension, hypertriglyceridemia, low HDL cholesterol, hyperinsulinemia</td>
<td>Obese sample (N = 439)</td>
<td>41% white, 31% black, 27% Hispanic, 4-20 y</td>
<td>z score: 2-2.5</td>
<td>38.7</td>
<td>Weiss et al(^{27}) (2004)</td>
</tr>
<tr>
<td>Hypertension</td>
<td>Population based</td>
<td>Multiethnic boys and girls, 5-11 y</td>
<td>Obese</td>
<td>Up to 30</td>
<td>Figueroa-Colon et al(^{29}) (1997)</td>
</tr>
<tr>
<td>Hypertension</td>
<td>Muscatine Heart Study (N=6600)</td>
<td>5-18 y</td>
<td>Community distribution</td>
<td>1 (60% of these had relative weight &gt;120%)</td>
<td>Dietz(^{29}) (1998)</td>
</tr>
<tr>
<td>Increased SBP measures &gt;95th percentile</td>
<td>Bogalusa Heart Study</td>
<td>Black and white boys and girls, 5-10 y</td>
<td>&lt;95</td>
<td>2-7</td>
<td>Freedman et al(^{25}) (2002)</td>
</tr>
<tr>
<td>Increased DBP &gt;95th percentile</td>
<td>Bogalusa Heart Study</td>
<td>Black and white boys and girls, 5-10 y</td>
<td>95th-97th</td>
<td>9</td>
<td>Freedman et al(^{25}) (2002)</td>
</tr>
<tr>
<td>Dislipidemia LDL cholesterol &gt;130 mg/dL</td>
<td>Bogalusa Heart Study (N = 3599)</td>
<td>Black and white boys and girls, 5-10 y</td>
<td>&lt;85th</td>
<td>8-10 across all percentiles</td>
<td>Freedman et al(^{25}) (2002)</td>
</tr>
<tr>
<td>Dislipidemia HDL cholesterol &lt;35 mg/dL</td>
<td>Bogalusa Heart Study (N = 3599)</td>
<td>Black and white boys and girls, 5-10 y</td>
<td>&lt;85th</td>
<td>8</td>
<td>Freedman et al(^{25}) (2002)</td>
</tr>
<tr>
<td>Dislipidemia TG levels &gt;130 mg/dL</td>
<td>Bogalusa Heart Study (N = 3599)</td>
<td>Black and white boys and girls, 5-10 y</td>
<td>&lt;85th</td>
<td>2-6</td>
<td>Freedman et al(^{25}) (2002)</td>
</tr>
</tbody>
</table>

Risk factors discussed in reviews without reported prevalence were menstrual disorders, polycystic ovarian syndrome, early maturation (girls), late maturation (boys). HDL indicates high-density lipoprotein; LDL, low-density lipoprotein; TG, triglyceride; SBP, systolic blood pressure; DBP, diastolic blood pressure; GTT, \(\gamma\)-glutamyltransferase.
pometric, physiologic, and health outcomes, and key question 6 addressed intervention-associated harms. The relationship between intervention-associated improvements in intermediate health measures and decreased morbidity in childhood or adulthood (key question 7) was examined only in the presence of adequate evidence for intervention efficacy (key questions 4 and 5). We did not examine key question 7 because of limited and inconsistent evidence for key questions 4 and 5. Review methods are summarized below and detailed elsewhere.36

Literature Search Strategy

We developed literature search strategies and terms for each key question and conducted 4 separate literature searches (for key questions 1 and 2, for key questions 4 and 5, for key question 3, and for key question 6) in Medline, PsycINFO, CINAHL, and the Cochrane Library, to update the literature from previous good-quality systematic reviews (key questions 4, 5, and 6) or to examine comprehensively literature from 1966 to the present (key questions 1, 2, and 3). Literature searches were supplemented extensively with source material from experts in the field, bibliographies of included trials, and other reviews. We also conducted limited hand-searching of pediatric obesity-focused editions of selected journals. A single investigator reviewed abstracts. A second investigator reviewed all excluded abstracts for all key questions, except key question 2 because of this search’s large yield, we conducted blinded dual reviews for a random subset (27%), with acceptable agreement (97.5%) between reviewers. Inter-reviewer discrepancies were resolved through consensus.

Article Review and Data Abstraction

With prespecified inclusion criteria,36 we reviewed 2162 abstracts and 353 complete articles for key questions 1 and 2, 949 abstracts and 196 complete articles for key questions 4 and 5, and 1176 abstracts and 36 complete articles for key questions 3 and 6. We included 0 articles for key question 1, 41 articles for key question 2, 0 articles for key question 3, 22 articles for key questions 4 and 5, and 4 articles for key question 6. Two investigators quality-rated all included articles and those excluded for quality reasons, using the USPSTF criteria.35

One primary reviewer abstracted relevant information from included studies into standardized evidence tables.36 To be within the USPSTF scope, interventions needed to be conducted in primary care or be feasible for primary care conduct or referral (defined elsewhere),36 and they were categorized as pharmaceutical, surgical, or behavioral counseling interventions. Abstracted behavioral counseling intervention details included setting, type of professional delivering the intervention, parent/family participation, intervention components, number and type of contacts, and intervention duration.37 Comprehensive behavioral treatments were those using a combination of behavioral modification (e.g., self-monitoring, stimulus control, or cognitive-behavioral techniques), dietary modification (e.g., Traffic Light Diet,38 reduced glycemic load, or reduced-fat or reduced-energy diets), and physical activity components (broadly specified as aerobic, callisthenic, lifestyle, or decreased sedentary behaviors).37

Studies needed to report weight outcomes, preferably as BMI or BMI percentile changes, to be included. We also recorded all reported behavioral, physiologic, and health outcomes specified on our analytic framework (Fig 2).

Literature Synthesis

There were insufficient homogeneous studies for any key question to allow quantitative synthesis. To better illustrate the study participants’ degree of overweight and the treatment impact of clinical interventions on overweight, we converted baseline measures and outcomes to BMI percentiles and plotted the results on the Centers for Disease Control and Prevention (CDC) growth charts. Treatment effects that were typical of interventions in this age group (10–20% reductions in percent overweight after 1 year) were modeled and plotted for 8-, 10-, and 12-year-old girls. We plotted reported mean BMI treatment effects at ≥6 months for 6 trials with adolescents included in our review (1 adolescent trial did not report BMI or percent overweight outcomes). These methods are described in more detail elsewhere.36 With the USPSTF
approach, we summarized the overall quality of the evidence for each key question.

RESULTS

Key Question 1: Is There Direct Evidence That Screening (and Intervention) for Overweight Among Children/Adolescents Improves Age-Appropriate Behavioral or Physiologic Measures or Health Outcomes?

Our searches found no studies addressing this key question, and neither did examination of all individual trials included in previous systematic evidence reviews.

Key Question 2

Key Question 2a: What Are Appropriate Standards for Overweight Among Children/Adolescents and What Is the Prevalence of Overweight Based on These Standards?

Eight nationally representative, health examination surveys that included children have been conducted in the United States since 1963. These surveys have gathered a variety of anthropometric measures for a range of ages (2 months to 18 years), providing growth references and trend analyses of changes within the population over time. To provide useful trend analyses, measures must be valid, must be gathered consistently in surveys, and must use a single source for comparison. Because of these limitations, almost all data on prevalence and trends among US children are based on BMI measures calculated from standardized height and weight information.

BMI measurements for an individual, or for determination of population prevalence, must be compared with a reference population to determine the age- and gender-specific percentile ranking. Although multiple reference data sets to determine childhood BMI percentiles are available, where possible we used the CDC 2000 gender-specific BMI growth charts (for ages 2–19 years). Prevalence estimates and trend information were taken primarily from the National Health and Nutrition Examination Survey (NHANES) program conducted from 1971 to 2000, which provides the most comprehensive data available on boys and girls of age 6 months through 19 years, with recent over-sampling of black and Mexican American children. These prevalence estimates use the CDC 2000 gender-specific BMI growth charts as their reference data set to assign BMI percentiles.

Prevalence

With the BMI ≥95th percentile, overweight prevalence in 1999–2002 was 10% among 2- to 5-year-old children and 16% among ≥6-year-old children (Fig 3). For children 2 to 5 years of age, the prevalences were similar for all racial/ethnic subgroups and both genders but were lower than those for older children in the same racial/ethnic subgroups. Among children 6 to 11 years of age, differences were seen between racial/ethnic subgroups, with significantly more Mexican American (21.8%) and non-Hispanic black (19.8%) children being categorized as overweight, compared with non-Hispanic white children (13.5%) (P < .05). Gender-specific differences were also apparent, with the highest prevalence of overweight among 6- to 11-year-old children being noted for Mexican American boys (26.5%); the prevalence was significantly higher than those for non-Hispanic black boys (17%), non-Hispanic white boys (14%), and Mexican American girls (17.1%) and was similar to that for non-Hispanic black girls (22.8%). Among youths 12 to 19 years of age, significantly more non-Hispanic black (21.1%) and Mexican American (22.5%) youths had overweight BMI measurements than did non-Hispanic white youths (13.7%) (P < .05), with no differences between male and female youths.

Key Question 2b: What Clinical Screening Tests for Overweight in Childhood Are Reliable and Valid in Predicting Obesity in Adulthood?

We found 19 fair- or good-quality, longitudinal, cohort studies (in 20 publications) that reported on BMI and other weight status measurements in childhood and adulthood. BMI measurements in childhood and adulthood correlated with each other as well as, or better than, other overweight measures, such as Ponderal Index or skinfold measurements, in childhood and adulthood correlated. Table 3 illustrates that BMI tracking from childhood to adulthood varies according to age. BMI measures track reasonably well from childhood and adolescence (ages 6–18) into young adulthood (ages 20–37), as evidenced by longitudinal studies showing low/moderate (r = 0.2–0.4) or moderate/high (0.5–0.8) correlations between childhood and adult BMI measures. Increased tracking (r ≥ 0.6 or elevated odds of adult obesity) is seen for older children (after age 8), particularly with sexual maturity, for younger children (ages 6–12) who are more overweight (usually above the 95th or 98th percentile), and for children with an obese parent. Data on tracking for children before the age of 12 are not extensive. Gender differences in tracking are not consistent across ages or within age categories. Limited data are available comparing white and black children. Table 4 illustrates the probability of adult obesity (BMI of ≥30 kg/m²) at various BMI percentiles for children of various ages, taken from our larger report. A ≥50% probability of adult obesity is seen generally for children ≥13 years of age with BMI measures of ≥95th percentile. Combining younger and older children in these analyses may obscure the increased probability of adult obesity with older ages of childhood overweight.

Key Question 2c: What Clinical Screening Tests for Overweight in Childhood Are Reliable and Valid for Poor Health Outcomes in Adulthood?

Although many (n = 11) US studies examined the risks associated with childhood overweight and adult outcomes, including socioeconomic outcomes, mortality rates, and a range of adult cardiovascular risk factors and morbidities, studies rarely controlled for adult BMI, a critical confounder. In one study that did, the apparent
association between elevated BMI at age 10 and several elevated adult cardiovascular risk factors (total cholesterol level, low-density lipoprotein and high-density lipoprotein cholesterol levels, insulin level, and systolic and diastolic blood pressure) in the Bogalusa Heart Study was eliminated after controlling for adult BMI.57

Key Question 3: Does Screening Have Adverse Effects, Such as Labeling or Unhealthy Psychological or Behavioral Consequences?

We found no direct evidence on the harms of screening. Potential harms include labeling, induced self-managed dieting with its negative sequelae, poorer self-concept, poorer health habits, disordered eating, and negative impact from parental concerns.5,13,32,78–84

Key Question 4: Do Interventions (Behavioral Counseling, Pharmacotherapy, or Surgery) That Are Feasible to Conduct in Primary Care Settings or Available for Primary Care Referral Lead to Improved Intermediate Behavioral or Physiologic Measures, With or Without Weight-Related Measures?

Behavioral Counseling Interventions

The most extensive treatment literature for childhood overweight involves behavioral counseling interventions. Behavioral counseling interventions include behavioral modification, special diets, and/or activity components delivered to children and/or parents as individuals or in groups by primary care clinicians or related health care staff members, to help patients adopt, change, or maintain health behaviors affecting overweight and related outcomes.85
We considered all trials published since 1985 from Western industrialized nations (n = 22 from 23 publications) that addressed interventions feasible for primary care conduct or primary care referral (including one that combined comprehensive behavioral treatment with pharmacotherapy, which is described separately below) (Table 5).86–108 We limited our search to post-1985 trials, because of the dramatic increases in overweight among children that occurred during the 1980s and 1990s, suggesting a very different treatment environment.1,8,109 A previous good-quality, systematic review including 16 of these trials concluded that this behavioral counseling treatment literature is limited, with marginal-quality trials involving small samples of primarily white, school-aged children receiving short-term, noncomparable, nongeneralizable interventions.40 These trials typically tested intensive, often family-based,

### TABLE 3. Effects of Age and Race on the Correlation of Childhood With Young Adult BMI

<table>
<thead>
<tr>
<th>Reference</th>
<th>Population</th>
<th>Childhood Age, y</th>
<th>Probability Male</th>
<th>Probability Female</th>
</tr>
</thead>
<tbody>
<tr>
<td>Guo et al (1994)</td>
<td>100% white (n = 555)</td>
<td>3</td>
<td>0.18</td>
<td>0.22</td>
</tr>
<tr>
<td>Lauer and Clark (1989)</td>
<td>100% white (n = 109 observations)</td>
<td>7–8</td>
<td>0.57</td>
<td>0.45</td>
</tr>
<tr>
<td>Lauer and Clark (1989)</td>
<td>100% white (n = 603 observations)</td>
<td>9–10</td>
<td>0.63</td>
<td>0.61</td>
</tr>
<tr>
<td>Clarke and Lauer(1993)</td>
<td>100% white (n = 1286 observations)</td>
<td>9–10</td>
<td>0.61</td>
<td>0.59</td>
</tr>
<tr>
<td>Lauer and Clark (1989)</td>
<td>100% white (n = 1018 observations)</td>
<td>11–12</td>
<td>0.67</td>
<td>0.65</td>
</tr>
<tr>
<td>Guo et al (1994)</td>
<td>100% white (n = 555)</td>
<td>13</td>
<td>0.5</td>
<td>0.65</td>
</tr>
<tr>
<td>Lauer and Clark (1989)</td>
<td>100% white (n = 1041 observations)</td>
<td>13–14</td>
<td>0.64</td>
<td>0.68</td>
</tr>
<tr>
<td>Clarke and Lauer (1993)</td>
<td>100% white (n = 1104 observations)</td>
<td>13–14</td>
<td>0.7</td>
<td>0.7</td>
</tr>
<tr>
<td>Lauer and Clark (1989)</td>
<td>100% white (n = 615 observations)</td>
<td>17–18</td>
<td>0.74</td>
<td>0.73</td>
</tr>
<tr>
<td>Clarke and Lauer (1993)</td>
<td>100% white (n = 631 observations)</td>
<td>17–18</td>
<td>0.81</td>
<td>0.72</td>
</tr>
</tbody>
</table>

### TABLE 4. Probability of Adult Obesity (BMI of ≥30 kg/m²) Based on Childhood BMI Percentile Measures at Various Ages

<table>
<thead>
<tr>
<th>Study ID</th>
<th>Overweight Measure in Childhood, BMI Percentile</th>
<th>Child’s Age When Measured, y</th>
<th>Adult’s Age When Measured, y</th>
<th>Probability of Adult Overweight (Male and Female Combined)</th>
<th>Probability of Adult Overweight (Male)</th>
<th>Probability of Adult Overweight (Female)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gortmaker et al (1993)</td>
<td>&gt;95th</td>
<td>16–24</td>
<td>23–31</td>
<td>0.77*</td>
<td>0.66*</td>
<td></td>
</tr>
<tr>
<td>Freedman et al (2001)</td>
<td>&lt;50th</td>
<td>5–17</td>
<td>18–37</td>
<td>0.07</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Freedman et al (2001)</td>
<td>85th–94th</td>
<td>5–17</td>
<td>18–37</td>
<td>0.51</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Freedman et al (2001)</td>
<td>≥95th</td>
<td>5–17</td>
<td>18–37</td>
<td>0.77</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Guo et al (2002)</td>
<td>≥75th, 100% white, 52% female</td>
<td>3</td>
<td>35</td>
<td>0.1</td>
<td>0.14</td>
<td></td>
</tr>
<tr>
<td>Guo et al (2002)</td>
<td>≥85th</td>
<td>8</td>
<td>35</td>
<td>0.1</td>
<td>0.17</td>
<td></td>
</tr>
<tr>
<td>Guo et al (2002)</td>
<td>≥95th</td>
<td>13</td>
<td>35</td>
<td>0.2</td>
<td>0.24</td>
<td></td>
</tr>
<tr>
<td>Guo et al (2002)</td>
<td>≥75th, 100% white, 52% female</td>
<td>18</td>
<td>35</td>
<td>0.2</td>
<td>0.16</td>
<td></td>
</tr>
<tr>
<td>Guo et al (2002)</td>
<td>≥85th</td>
<td>13</td>
<td>35</td>
<td>0.2</td>
<td>0.23</td>
<td></td>
</tr>
<tr>
<td>Guo et al (2002)</td>
<td>≥95th</td>
<td>8</td>
<td>35</td>
<td>0.2</td>
<td>0.46</td>
<td></td>
</tr>
</tbody>
</table>

* In this study, adult overweight was defined as >95th percentile on NHANES.
### Table 5: Randomized Trials Addressing Overweight Among Children and Adults

<table>
<thead>
<tr>
<th>Study Reference</th>
<th>N Randomized, Country</th>
<th>Age, y (% Male, % Nonwhite)</th>
<th>Relative Overweight at Baseline</th>
<th>Intervention Characteristics*</th>
<th>Group</th>
<th>Units of Measure</th>
<th>Study Duration, mo</th>
<th>Outcome at Latest Follow-Up</th>
<th>P, Comparisons Between Groups†</th>
<th>Other Outcomes</th>
<th>USPSTF Quality Grade</th>
</tr>
</thead>
<tbody>
<tr>
<td>Berkowitz et al. (2003) 82 adolescents, USA</td>
<td>13–17 (% Male, % Nonwhite)</td>
<td>BMI: 37.8 kg/m² (38); BMI: z score: 2.4 (0.2)</td>
<td>BM, D, E (yes; yes; G) 6 mo (phase I), 19 (phase I), NR, NR</td>
<td>Sibutramine Change in BMI (% change from entry BMI)</td>
<td>6</td>
<td>−8.5%</td>
<td>.001</td>
<td>P, A</td>
<td>Good</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Duffy and Spence (1993)</td>
<td>29 children, Australia</td>
<td>7–13 (21, NR)</td>
<td>BM, D, E (yes; yes; NR) 8 wk, 8, 90 min, 720 min</td>
<td>BT placebo Cognitive self-management BT + relaxation placebo</td>
<td>6</td>
<td>−8.9%</td>
<td>NS</td>
<td>B</td>
<td>Fair/poor</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ebbeling et al. (2003) 16 adolescents, USA</td>
<td>13–21 (% Male, % Nonwhite)</td>
<td>BMI: 34.9 kg/m² (reduced glycemic group); 37.1 kg/m² (reduced fat diet group)</td>
<td>BM, D (no; no; NR) 12 mo, 14, NR, NR</td>
<td>Reduced-glycemic load diet Absolute change in BMI</td>
<td>12</td>
<td>−1.2 kg/m²</td>
<td>&lt;.05</td>
<td>B, P</td>
<td>Fair</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Epstein et al. (1985)</td>
<td>41 families, USA</td>
<td>8–12 (% Male, % Nonwhite)</td>
<td>BM, D, E (yes; yes; NR) 12 mo, 18, NR, NR</td>
<td>Lifestyle PA % Overweight change</td>
<td>24</td>
<td>−18.0%</td>
<td>&lt;.05 (lifestyle PA vs aerobic PA); &lt;.05 (lifestyle PA vs calisthenics PA)</td>
<td>B, P</td>
<td>Fair</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Epstein et al. (1985)</td>
<td>23 children, USA</td>
<td>8–12 (% Male, % Nonwhite)</td>
<td>BM, D, E (yes; yes; NR) 12 mo, NR, NR, NR</td>
<td>Aerobic PA Low-intensity calisthenics PA Diet + PA</td>
<td>12</td>
<td>−25.4%</td>
<td>NS</td>
<td>B, P</td>
<td>Fair</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Epstein et al. (1994)</td>
<td>44 families, USA</td>
<td>8–12 (% Male, % Nonwhite)</td>
<td>BM, D, E (yes; yes; unclear) 1 y, 32, NR, NR</td>
<td>Mastery criteria and contingent reinforcement group</td>
<td>24</td>
<td>−15.4%</td>
<td>NS</td>
<td>B</td>
<td>Fair</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Epstein et al. (1995)</td>
<td>61 families, USA</td>
<td>8–12 (% Male, % Nonwhite)</td>
<td>BM, D, E (yes; yes; I + G) 6 mo, 18, NR, NR</td>
<td>Combined Comparison group</td>
<td>12</td>
<td>−10.6%</td>
<td>&lt;.05 (combined vs increased PA)</td>
<td>B, P</td>
<td>Fair</td>
<td></td>
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</tr>
<tr>
<td>Epstein et al. (2000)</td>
<td>90 families, USA</td>
<td>8–12 (% Male, % Nonwhite)</td>
<td>BM, D, E (yes; yes; I + G) 6 mo, 20, 45–60 min, 900–1200 min</td>
<td>Decreased SB Increased PA</td>
<td>24</td>
<td>−14.3%</td>
<td>NS</td>
<td>B, P</td>
<td>Fair</td>
<td></td>
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</tr>
<tr>
<td>Epstein et al. (2000)</td>
<td>67 children, USA</td>
<td>BMI: 27.4 (3.2) kg/m²</td>
<td>BM, D, E (yes; yes; I + G) 6 mo, 18, 45–60 min, 810–1080 min</td>
<td>PS to parent and child</td>
<td>24</td>
<td>−0.5</td>
<td>&lt;.05 (PS to parent and child vs no PS); &lt;.05 (PS to parent and child vs PS to child only)</td>
<td>H, A</td>
<td>Fair</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Study Reference</td>
<td>N Randomized, Country</td>
<td>Age, y (% Male, % Nonwhite)</td>
<td>Relative Overweight at Baseline</td>
<td>Intervention Characteristics*</td>
<td>Group</td>
<td>Units of Measure</td>
<td>Study Duration, mo</td>
<td>Outcome at Latest Follow-Up Time</td>
<td>P. Comparisons Between Groups†</td>
<td>Other Outcomes</td>
<td>USPSTF Quality Grade</td>
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<tr>
<td>Epstein et al 96 (2001)</td>
<td>67 families, USA</td>
<td>8–12 (52, NR)</td>
<td>60.2% overweight (compared with 50th percentile BMI for age and gender); BMI: 27.4 kg/m² (3.6 kg/m²)</td>
<td>BM, D, E (yes; yes; I + G)</td>
<td>Increased PA</td>
<td>Change in absolute BMI (statistical comparisons, done on % overweight change)</td>
<td>6 mo, 20, 30 min, 600 min</td>
<td>Girls; −0.27 kg/m²; boys; −0.65 kg/m²</td>
<td>&lt;0.01 (interaction of group by gender); &lt;0.01 (boys in combined group vs girls in combined group); &lt;0.05 (boys in combined group vs girls in increased PA group)</td>
<td>None</td>
<td>Fair</td>
</tr>
<tr>
<td>Flodmark et al 97 (1993)</td>
<td>44 children, (plus 50 matched control subjects), Sweden</td>
<td>10–11 (48, NR)</td>
<td>24.7 kg/m² (family therapy group); 25.5 kg/m² (conventional treatment group); 25.1 kg/m² (control group)</td>
<td>D, E (no; yes; I)</td>
<td>Family therapy</td>
<td>Change in BMI</td>
<td>14–18 mo, 5 (+6 family therapy sessions), NR, NR</td>
<td>1.1 kg/m²; boys; 1.6 kg/m²</td>
<td>&lt;0.05 (family therapy vs untreated control subjects)</td>
<td>P</td>
<td>Fair</td>
</tr>
<tr>
<td>Golan et al 98 (1998)</td>
<td>60 children, Israel</td>
<td>6–11 (38, NR)</td>
<td>39.1% overweight (conventional group); 39.6% (parents agents of change group)</td>
<td>BM, D, E (yes; no*; G + I)</td>
<td>Conventional: children responsible for own weight loss</td>
<td>% Overweight change</td>
<td>1 y, 30, 60 min, 1800 min</td>
<td>8.1%</td>
<td>≤0.05 None</td>
<td>Fair</td>
<td></td>
</tr>
<tr>
<td>Graves et al 99 (1988)</td>
<td>40 children, USA</td>
<td>6–12 (NR, NR)</td>
<td>52.56% overweight</td>
<td>BM, D, E (yes; yes; G)</td>
<td>BT + parent PS</td>
<td>% Overweight change</td>
<td>8 wk, 8, 60 min, 480 min</td>
<td>−14.7%</td>
<td>None Fair/poor</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Israel et al 100 (1985)</td>
<td>33 children, USA</td>
<td>8–12 (30, NR)</td>
<td>45.88% overweight (parent training group), 53.13% (BT only), 56.02% (control subjects)</td>
<td>BM, D, E (yes; yes; G)</td>
<td>BT only</td>
<td>% Overweight change</td>
<td>12, 17, 2, 60-min sessions, &gt;900 min</td>
<td>−10.2% &lt;0.01</td>
<td>B Fair/poor</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Israel et al 101 (1994)</td>
<td>36 families, USA</td>
<td>8–13 (NR, NR)</td>
<td>48.1% overweight (enhanced child involvement group), 46.0% (standard treatment group)</td>
<td>BM, D, E (yes; yes; G)</td>
<td>Enhanced child involvement</td>
<td>% Overweight change</td>
<td>26 wk, 17, 90 min, 1530 min</td>
<td>4.8% NS</td>
<td>None Fair/poor</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Kang et al 102/ Gutin et al 103 (2002)</td>
<td>80 adolescents, USA</td>
<td>13–16 (33, 69)</td>
<td>40.7% body fat (white boys), 45.8% body fat (white girls), 43.9% body fat (black boys), 45.2% body fat (black girls)</td>
<td>BM, E (no; no; G)</td>
<td>LSE + high intensity PA</td>
<td>Change in % body fat</td>
<td>8 mo, 160, 60 min for LSE, variable for PA NR</td>
<td>−2.9% NS</td>
<td>B, P Fair/poor</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Study Reference</td>
<td>N Randomized, Country</td>
<td>Age, y ( % Male, % Nonwhite)</td>
<td>Relative Overweight at Baseline</td>
<td>Intervention Characteristics*</td>
<td>Group</td>
<td>Units of Measure</td>
<td>Study Duration, mo</td>
<td>Outcome at Latest Follow-Up Time</td>
<td>P, Comparisons Between Groups†</td>
<td>Other Outcomes</td>
<td>USPSTF Quality Grade</td>
</tr>
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</tr>
<tr>
<td>Mellin et al104 (1987)</td>
<td>66 adolescents, USA</td>
<td>12–18 (21, 22)</td>
<td>30–37% overweight BM, D, E (yes; yes; G)</td>
<td>SHAPEDOWN group (cognitive, behavioral, affective treatment)</td>
<td>14 wk, 16, 90 min, 1440 min</td>
<td>% Overweight change</td>
<td>15</td>
<td>−9.9% NR (between-group comparison; 15 mo vs baseline: &lt;.01, SHAPEDOWN; NS, control group)</td>
<td>B, H Fair</td>
<td>B, H</td>
<td>Fair</td>
</tr>
<tr>
<td>Saelens et al105 (2002)</td>
<td>44 adolescents, USA</td>
<td>12–16 (59, 30)</td>
<td>BMI: 30.7 (3.1) kg/m²</td>
<td>BM, D, E (yes; no; 1)</td>
<td>4 mo, 13, 10–20 min, &gt;200 min</td>
<td>Healthy habits intervention</td>
<td>7</td>
<td>−2.4%, 0.1 kg/m²</td>
<td>NS</td>
<td>B, A</td>
<td>Good</td>
</tr>
<tr>
<td>Senediak and Spence106 (1985)</td>
<td>45 children, USA</td>
<td>6–13 (~66, NR)</td>
<td>37.22% overweight BM, D, E (yes; yes; G)</td>
<td>Rapid-schedule BT</td>
<td>4 wk, 8, 90 min, 720 min</td>
<td>% Overweight change</td>
<td>6</td>
<td>−14.7% (rapid and gradual schedule BT groups combined vs nonspecific control subjects); NS (comparison of rapid vs gradual schedule BT groups)</td>
<td>B</td>
<td>Fair/poor</td>
<td></td>
</tr>
<tr>
<td>Wadden et al107 (1990)</td>
<td>47 girls, USA</td>
<td>12–16 (0, 100) [all black]</td>
<td>95.1 kg; BMI 35.6 kg/m²</td>
<td>BM, D, E (yes; yes; G)</td>
<td>10 mo, 22, 60 min (first 16 sessions), others NR, &gt;900 min</td>
<td>Change in weight</td>
<td>6</td>
<td>1.7 kg</td>
<td>3.0 kg (change in BMI)</td>
<td>NS</td>
<td>P, H</td>
</tr>
<tr>
<td>White108 (2003)/ Williamson, unpublished data</td>
<td>57 adolescents, USA</td>
<td>11–15 (0, 100)</td>
<td>BMI: 36.24 kg/m²; 98.3 BMI percentile</td>
<td>BM, D, E (yes; yes; I)</td>
<td>6 mo, 4 and weekly Web-site logins, NR, NR</td>
<td>Behavioral</td>
<td>6</td>
<td>−1.12%, −0.19 kg/m²</td>
<td>&lt;.05 (% body fat); &lt;.05 (change in BMI)</td>
<td>B</td>
<td>Good</td>
</tr>
</tbody>
</table>

NHS indicates National Health Service; NS, not significant; BM, behavior modification; D, special diet; E, exercise program; G, group; I, individual; other outcomes: B, behavioral; P, physiologic; H, childhood health outcomes; A, adverse effects; NR, not reported; LSE, lifestyle education; BT, behavioral therapy; TC, total cholesterol; SB, sedentary behavior; PA, physical activity; PS, problem solving.

* For most intensive intervention, which is listed first.
† If multiple comparisons, then presented only if $P < .05$.
‡ The actual number was not reported; the mean (SD) was 10.3 (1.1) years.
§ Intervention that uses a combination of behavior modification procedures, a special diet, and an exercise program.
interventions conducted in specialty obesity clinic settings to address overweight among school-aged children who were ~40% to 50% above ideal weight. Figure 4 models the short-term (1-year) results from these types of studies, translated to BMI percentiles.

Figure 5 demonstrates results from all behavioral counseling studies among adolescents\(^{86,88,104,105,107,108}\) that reported, or could be translated into, BMI percentiles. Most studies addressed extremely overweight patients, with short-term results showing modest to no change in BMI percentiles. Only 2 good-quality studies among adolescents were particularly relevant to primary care.\(^{105,108}\)

One short-term, primary care-conducted trial that used a computer-based approach to generate tailored plans for counseling obese (above the adult BMI cutoff of 30 kg/m\(^2\)) adolescents (12–16 years of age) showed small but significant improvements\(^{105}\) (Fig 5). An Internet-based, short-term trial targeting 57 overweight (mean BMI: 36.37 kg/m\(^2\)), non-Hispanic black, female youths (11–15 years of age) with ≥1 obese biological parent resulted in statistically significant differences in weight and BMI.\(^{108}\) Although both trials showed small but statistically significant benefits in BMI measures at 6 to 12 months, it is not clear that these BMI changes would have clinical benefits.

Considering other intermediate outcomes in addition to weight, more than one half \((n = 13)\) of fair- or good-quality trials\(^{86,88–94,97,99,104,105,108}\) reported intermediate behavioral \((n = 11)\) or physiologic \((n = 7)\) measures (Table 5). Two good-quality trials\(^{105,108}\)
reported behavioral changes but no physiologic outcomes. Although one indicated reduced total daily energy intake in the active treatment group, neither indicated changes in physical activity. One fair-quality study reported reductions in targeted dietary components (fat or glycemic load of diet), but not energy, whereas other fair-quality studies measured changes in eating behaviors, physical activity, and sedentary behaviors but did not provide a clear picture because of differences in subjects, interventions, and measures.

No good-quality trials of behavioral treatment reported intermediate physiologic outcomes, such as lipid or lipoprotein levels, glucose tolerance, or blood pressure, or physical fitness measures. Only one trial of at least fair quality reported intermediate physiologic measures. An intensive, 6-month, behavioral, weight control program comparing a reduced-glycemic load diet with a reduced-fat diet increased insulin resistance scores (measured with the homeostatic model) significantly less in the reduced-glycemic load diet group than the reduced-fat diet group (−0.4 ± 0.9 vs 2.6 ± 1.2, P = .03). Insulin resistance increases with sexual maturation, however, which was not assessed. These results are also limited by baseline differences between groups and lack of consideration of physical activity as a confounder.

Among the fair-quality studies that measured physical work capacity or physical fitness, most reported some improvement when physical activity or sedentary behaviors were addressed in the intervention.
Pharmacotherapy

One randomized, placebo-controlled trial of sibutramine within a comprehensive behavioral treatment program for adolescents showed superior weight change outcomes after 6 months (4.6-kg greater weight loss; 95% CI: 2.0–7.4 kg) in an intent-to-treat analysis (Fig 5). With continued use, weight loss at 6 months was maintained through 12 months. It is not clear whether the additional short-term weight change achieved with the addition of sibutramine to a comprehensive behavioral treatment program among adolescents would provide a net benefit, because changes in serum lipid levels, serum insulin levels, serum glucose levels, and homeostatic model of insulin sensitivity values did not differ between groups. Among all trial completers (63–76% of all participants) at 12 months, significant improvements from baseline were seen in high-density lipoprotein cholesterol levels, serum insulin levels, and homeostatic model of insulin sensitivity values. Blood pressure was not improved, and in some cases increased blood pressure was a reason for discontinuation. The rate of adverse effects and discontinuation was fairly high (12% discontinued and 28% reduced the medication) (see also key question 6). We found no evidence for metformin use for weight loss/disease prevention among normoglycemic obese adolescents with weight outcomes after <3 months, nor did we find acceptable evidence on alternative or complementary therapies.

Surgery

No acceptable quality evidence is available for adolescents, evaluating surgical approaches to overweight. There are no controlled treatment outcome data on bariatric surgery approaches among adolescents.

Key Question 5: Do Interventions Lead to Improved Adult Health Outcomes, Reduced Childhood Morbidity Rates, and/or Improved Psychosocial and Functional Childhood Outcomes?

Few (n = 3) studies reported health outcomes as defined in our analytic framework, and only 2 were rated at least fair quality (Table 5). In one fair-quality trial, depression scores measured with reliable and valid instruments showed improvement from baseline among treated adolescent girls but not control subjects, whereas reliably measured self-esteem scores improved from baseline in both groups. In a second fair-quality study, significantly fewer children 8 to 12 years of age, receiving comprehensive behavioral treatment, had elevated total behavior problem scores or elevated internalizing behavior problem scores at the 24-month follow-up assessment than at baseline.

Key Question 6: Do Interventions Have Adverse Effects, Such as Stigmatization, Binging or Purging Behaviors, Eating Disorders, Suppressed Growth, or Exercise-Induced Injuries?

Behavioral Counseling Interventions

Adverse effect reporting for behavioral counseling interventions was limited to 3 of 22 intervention trials. Potential eating problems or weight management behaviors were the only harms addressed in 2 trials. One good-quality trial reported no adverse effects on problematic eating (using validated measures for dietary restraint, eating disinhibition, problematic weight management behaviors, weight concern, and eating disorder psychopathologic features) after primary care-based comprehensive behavioral treatment for 37 of 44 adolescent trial completers. One fair-quality trial reported no effect on eating disorder symptoms, weight dissatisfaction, or purging/restricting behaviors among 47 children 8 to 12 years of age in a family-based comprehensive behavioral treatment program, using a reliable measure (Kids’ Eating Disorder Survey). Differences between boys (no effect) and girls (elevated total scores) were not significant but may be revealed in studies with larger sample sizes.

Pharmacotherapy

In the placebo-controlled phase of the sibutramine trial, 44% of patients (19 of 43 patients) in the active medication group reduced or discontinued the medication because of elevated blood pressure, pulse rate, or both, which were the main adverse events reported.

Surgery

We attempted to estimate the rate of harms from the uncontrolled cohort literature, but loss to follow-up (25%–60% at 4–24 months) and inadequate reporting prevented us from making reasonable estimates of surgery-associated harms.

Summary of Evidence Quality

Table 6 summarizes the overall quality of evidence, according to USPSTF criteria, for each key question addressed in this review (see Appendix). The overall evidence is poor for the direct effects of screening (and intervention) programs (key question 1), screening harms (key question 3), and bariatric surgery (key questions 4 and 5). The overall evidence is fair/poor for behavioral counseling interventions (key questions 4 and 5), because of small, noncomparable, short-term studies with limited generalizability that reported health or intermediate outcomes, such as cardiovascular risk factors, rarely. Trials are particularly inadequate for nonwhite subjects and children 2 to 5 years of age. Fair/poor evidence is available for behavioral counseling intervention harms because of very limited reporting (key question 6). Fair evidence supports childhood BMI as a risk factor for adult overweight, although data are limited for nonwhite subjects (key question 2b), and data addressing BMI as a risk factor for adult morbidities generally do not control for confounding by adult BMI (key question 2c). Good evidence is available for overweight prevalence based on BMI measures in all groups, except Native American and Asian groups (key question 2a).

CONCLUSIONS

Overweight has at least doubled among children and adolescents in the United States in the past 25 years, according to USPSTF criteria for each key question addressed in this review (see Appendix). The overall evidence is poor for the direct effects of screening (and intervention) programs (key question 1), screening harms (key question 3), and bariatric surgery (key questions 4 and 5). The overall evidence is fair/poor for behavioral counseling interventions (key questions 4 and 5), because of small, noncomparable, short-term studies with limited generalizability that reported health or intermediate outcomes, such as cardiovascular risk factors, rarely. Trials are particularly inadequate for nonwhite subjects and children 2 to 5 years of age. Fair/poor evidence is available for behavioral counseling intervention harms because of very limited reporting (key question 6). Fair evidence supports childhood BMI as a risk factor for adult overweight, although data are limited for nonwhite subjects (key question 2b), and data addressing BMI as a risk factor for adult morbidities generally do not control for confounding by adult BMI (key question 2c). Good evidence is available for overweight prevalence based on BMI measures in all groups, except Native American and Asian groups (key question 2a).
years and is particularly common among racial/ethnic minorities. This increase represents a major public health concern, with the potential for future health risks and growing burdens on the health care system. In terms of evidence, however, little has changed since a 1998 *Journal of Pediatrics* editorial concluded that, “In the case of obesity, the primary care physician is left in the uncomfortable (but familiar) position of needing to do something now for the patient and family seeking help, regardless of the uncertainty about the nature of the disease and the absence of a cure.”114 Given the nature of the problem, effective solutions will likely require substantial collaboration between the medical and public health communities.115 Greater understanding of how to expand the appropriate role of clinicians in community public health, such as through advocating necessary environmental and political changes, would be helpful.116,117

A major limitation for clinicians addressing overweight among children, most of whom are not morbidly overweight, is the uncertain criteria for determining clinically significant overweight. Although BMI is the best clinically available measure of overweight, uncertainty in its application to individual patients remains, because of limited knowledge of the current and future health effects of BMI and the possible limits in the applicability of current BMI cutoff points, particularly for minority race/ethnicity. Understanding normal variations in body composition with age, gender, race/ethnicity, sexual maturity, and other factors will be critical for defining accurately unhealthy excess fat or other components of overweight, and appropriate measurement methods. Similarly, as has been done elsewhere, examining the sensitivity and specificity of BMI percentile cutoff values for identifying overweight children, with large representative samples of US children of all ages and races/ethnicities, would increase our understanding of BMI as a screening tool.118

The risk for overweight children becoming overweight or obese adults has been judged as the best available criterion to judge the clinical validity of BMI in the pediatric age group.119 Adult BMI has been associated clearly with morbidity and mortality rates, particularly at higher BMI levels, although there is no single threshold for increased health risks.120 Adolescents in ≥95th percentile for age- and gender-specific BMI clearly have an increased probability of adult obesity, and early interventions could be very beneficial. Recent intervention research targeting this age group primarily addressed subjects who were very overweight, with some studies showing short-term (6–12-month) weight-related improvements. The treatment evidence in this age group could be strengthened with larger trials testing generalizable interventions that can demonstrate sustained effects on overweight status and weight-related outcomes. Many trials among adolescents have targeted minorities specifically107,108 or enrolled reasonable proportions in their studies86,102,105 and this should continue. Trials among mildly overweight adolescents, as well as those more severely affected, are needed. With limited to no evidence available, experts agree that surgical approaches should be considered only for adolescents with extreme morbid obesity, and pharmacologic approaches should be limited to a second-tier approach after failed behavioral counseling.111,112

In contrast, current data suggest that a substantial proportion of children under age 12 or 13, even with BMIs of ≥95th percentile, will not develop adult obesity. Children 8 to 12 years of age have been the most well studied for behavioral overweight treatment, but we still have very limited information about interventions that would be applicable to primary care. No current, randomized, controlled trial of clinical interventions of any type is available for children 2 to 5 years of age.

For all ages, there is very limited evidence for behavioral or other overweight treatment that is feasible for primary care delivery or referral. Few studies have taken place in primary care setting; most have been conducted in research or specialty obesity

### TABLE 6. Summary of Evidence Quality for Key Questions Addressing Childhood and Adolescent Overweight

<table>
<thead>
<tr>
<th>Key Question</th>
<th>Study Hierarchy</th>
<th>Overall USPSTF Quality</th>
</tr>
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<tbody>
<tr>
<td>1. Screening</td>
<td>II-2</td>
<td>Good but lacking for specific nonwhite racial/ethnic subgroups</td>
</tr>
<tr>
<td>2a. Prevalence</td>
<td>II-2</td>
<td>Fair; data for BMI as a risk factor for adult overweight from childhood overweight are the most valid but are very limited for nonwhite subjects; for data for BMI as a risk factor for adult morbidities generally do not control for confounding by adult BMI</td>
</tr>
<tr>
<td>2b and 2c. Screening tests as a risk factor</td>
<td>II-2</td>
<td>Poor; because of lack of screening studies, possible harms can only be inferred from other sources</td>
</tr>
<tr>
<td>3. Screening harms</td>
<td>I</td>
<td>Fair/poor; data are limited by very small samples, noncomparable interventions, and not using intent-to-treat analyses; little reporting of intermediate outcomes, including risk factor changes or changes in health outcomes; poor generalizability because of specialist interventions not widely available and addressing mostly 8–12 y; no data for 2–5 y; few trials include nonwhite subjects</td>
</tr>
<tr>
<td>4 and 5. BCI interventions</td>
<td>I</td>
<td>Fair; one good quality trial with adolescents</td>
</tr>
<tr>
<td>4 and 5. Pharmacotherapy with BCI</td>
<td>I</td>
<td>Poor</td>
</tr>
<tr>
<td>4 and 5. Surgery</td>
<td>I, II-2</td>
<td>Fair/poor; very limited reporting of harms for BCI interventions</td>
</tr>
</tbody>
</table>

BCI indicates behavioral counseling.
clinics with intensive, comprehensive, behavioral treatment. Experts have cautioned that behavioral therapy represents an expertise-driven approach to improving diet and physical activity with behavioral principles and is not simply an add-on to a diet and exercise plan. If larger studies confirm that behavioral skills and approaches are key to treatment success, then creation of referral clinics or involvement of clinic team members with behavioral medicine/psychology weight management expertise will be critical.

Experts recommend referring certain children to pediatric obesity treatment centers for expert management. These include children who are massively overweight (defined through clinical judgment) or who have a BMI exceeding the 95th percentile, with associated severe morbidities that require immediate weight loss. For asymptomatic children with a BMI of ≥95th percentile, experts recommend an in-depth medical assessment to detect treatable causes of obesity, risk factors, and comorbidities. For children whose BMI falls between the 85th and 95th percentiles for age and gender, they also recommend clinical evaluations for secondary effects of overweight, such as hypertension and hyperlipidemia. We did not find adequate evidence meeting our criteria to address the impact of BMI screening and/or treatment of overweight (or at risk for overweight) on any of these risks factors or morbidities.

Experts emphasize talking to families about energy balance behaviors that might help prevent obesity and would also promote other aspects of health and likely cause no harms. These behaviors include limiting television viewing, encouraging outdoor play, and limiting the consumption of sugar-sweetened soft drinks. For interested clinicians, pragmatic approaches for all children (particularly young children) that emphasize the “healthy lifestyle prescription” approach over targeting overweight identification seem appropriate, because we found limited evidence for secondary prevention or treatment. However, clinicians should be aware that others have found limited evidence for the effectiveness of primary prevention in clinical settings.

Given the current evidence, BMI measurements among older adolescents may provide an early reasonable indication of future adult health risks attributable to obesity. BMI measurements for younger children should be performed as a growth-monitoring tool that might indicate future risk for adult overweight and its attendant morbidities, with reduced emphasis on defining current overweight. Children, particularly those <13 years of age, without clinical weight-related morbidities would not necessarily be labeled overweight but might be considered at risk or at high risk, depending on the BMI level. Experts recommend regular longitudinal monitoring and careful documentation of BMI among children and adolescents. Such monitoring will likely prove even more valuable as our understanding grows about the predictive value of levels and patterns of growth and overweight status changes over time and about effective ways to address patterns that indicate overweight that affects current health or a high future risk of adult overweight.

In the absence of direct evidence of the effects of screening on improved weight and health outcomes among children and/or adults, we evaluated indirect evidence for screening and intervention. In the current literature, evidence linkages between screening and intervention are hampered by divergent definitions of overweight. It is important that a consistent definition of overweight be accepted, to encourage rapid progress in our understanding of how to address this critical problem.

Limited evidence on normal body composition among children and adolescents and lack of criterion standards for adiposity among children hampered our ability to determine the test characteristics (sensitivity and specificity) of clinically feasible screening tests. Valid, feasible, body composition measures for children are becoming established, which should allow examination of the sensitivity and specificity of BMI percentiles and overweight in US populations, as elsewhere. Similarly, clearly establishing current or future health consequences of elevated BMI (and other overweight measures) for boys and girls of all ages and racial/ethnic origins will enable future diagnostic research. By confining our review of childhood BMI and adult health consequences to longitudinal US studies, we gained some advantages from more similar overweight definitions, measurements, and reference standards but might have eliminated applicable data unnecessarily. Because the reviewed research was primarily among non-Hispanic white subjects, its applicability to minority groups, in which the prevalence of overweight is increasing particularly, may be limited.

We did not locate adequate longitudinal data relating childhood weight status to childhood health outcomes; therefore, we did not review it formally. Current literature is primarily cross-sectional, presents relative risks without absolute risks, or reports on the relationship of growth measures (or changes in the measures over time) to intermediate measures, such as blood pressure or lipid levels, rather than health outcomes.

Although we made an effort to review several areas of the literature comprehensively, some areas were not reviewed. We did not review any evidence for children <2 years of age, although this is an active area for research. We did not attempt to examine risk factors for childhood overweight, but others have done so. Similarly, research on changing children’s daily life habits that might also affect or prevent pediatric overweight, such as changing dietary intake, increasing physical activity, or limiting activities such as television viewing, that did not address weight effects directly was beyond the scope of this study.

There are critical research gaps in answering the most basic questions needed to enable clinicians to engage strategies to prevent current and future weight-related morbidities among children. Despite the fact that many of these gaps were pointed out 10 years ago, little subsequent research has addressed the most clinically relevant questions. In ad-
diction to the clinical research already underway to address childhood overweight prevention and treatment, we strongly urge the research community to prioritize research studies that would supply needed evidence to address the key questions formulated for this report, to guide pragmatic clinical and public health prevention strategies. Some of these studies could involve reporting from existing, good-quality, cross-sectional and longitudinal cohort studies, in addition to new studies and clinical trials. For a more complete list of research recommendations, readers can consult the full review.36

ACKNOWLEDGMENTS

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### APPENDIX 1. USPSTF Hierarchy of Research Design and Quality Rating Criteria

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<tr>
<th>Hierarchy of research design</th>
<th>Criteria</th>
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<tbody>
<tr>
<td>I</td>
<td>Properly conducted randomized, controlled trial</td>
</tr>
<tr>
<td>II-1</td>
<td>Well-designed controlled trial without randomization</td>
</tr>
<tr>
<td>II-2</td>
<td>Well-designed cohort or case-control analytic study</td>
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<tr>
<td>II-3</td>
<td>Multiple time series with or without the intervention; dramatic results from uncontrolled experiments</td>
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<tr>
<td>III</td>
<td>Opinions of respected authorities, based on clinical experience; descriptive studies or case reports; reports of expert committees</td>
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#### Design-specific criteria and quality category definitions

**Systematic reviews**
- Comprehensiveness of sources considered/search strategy used
- Standard appraisal of included studies
- Validity of conclusions
- Recency and relevance especially important

**Case-control studies**
- Accurate ascertainment of cases
- Nonbiased selection of case/control subjects with exclusion criteria applied equally to both
- Response rate
- Diagnostic testing procedures applied equally to each group
- Measurement of exposure accurate and applied equally to each group
- Appropriate attention to potential confounding variables

**Randomized, controlled trials and cohort studies**
- Initial assembly of comparable groups
  - For randomized, controlled trials: adequate randomization, including first concealment and whether potential confounders were distributed equally among groups
  - For cohort studies: consideration of potential confounders with either restriction or measurement for adjustment in the analysis; consideration of inception cohorts
- Maintenance of comparable groups (includes attrition, crossovers, adherence, contamination)
- Important differential loss to follow-up monitoring or overall high loss to follow-up monitoring
- Measurements: equal, reliable, and valid (includes masking of outcome assessment)
- Clear definition of the interventions
- All important outcomes considered

**Diagnostic accuracy studies**
- Screening test relevant, available for primacy care, adequately described
- Study uses credible reference standard, performed regardless of test results
- Reference standard interpreted independently of screening test
- Handles indeterminate result in a reasonable manner
- Spectrum of patients included in study
- Sample size
- Administration of reliable screening test
Screening and Interventions for Childhood Overweight: A Summary of Evidence for the US Preventive Services Task Force
Evelyn P. Whitlock, Selvi B. Williams, Rachel Gold, Paula R. Smith and Scott A. Shipman

*Pediatrics* 2005;116;e125
DOI: 10.1542/peds.2005-0242

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