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Screening and Interventions for Childhood Overweight: Evidence Synthesis

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Preface

The Agency for Healthcare Research and Quality (AHRQ) sponsors the development of Systematic Evidence Reviews (SERs) and Evidence Syntheses through its Evidence-based Practice Program. With guidance from the U.S. Preventive Services Task Force* (USPSTF) and input from Federal partners and primary care specialty societies, the Oregon Evidence-based Practice Center systematically reviews the evidence of the effectiveness of a wide range of clinical preventive services, including screening, counseling, and chemoprevention, in the primary care setting. The SERs and Evidence Syntheses—comprehensive reviews of the scientific evidence on the effectiveness of particular clinical preventive services—serve as the foundation for the recommendations of the USPSTF, which provide age- and risk-factor-specific recommendations for the delivery of these services in the primary care setting. Details of the process of identifying and evaluating relevant scientific evidence are described in the "Methods" section of each SER and Evidence Synthesis.

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We welcome written comments on this Evidence Synthesis. Comments may be sent to: Director, Center for Practice and Technology Assessment, Agency for Healthcare Research and Quality, 540 Gaither Road, Suite 3000, Rockville, MD 20850, or e-mail uspstf@ahrq.gov.

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*The USPSTF is an independent panel of experts in primary care and prevention first convened by the U.S. Public Health Service in 1984. The USPSTF systematically reviews the evidence on the effectiveness of providing clinical preventive services--including screening, counseling, and chemoprevention--in the primary care setting. AHRQ convened the current USPSTF in November 1998 to update existing Task Force recommendations and to address new topics.

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Structured Abstract

Background: Childhood and adolescent overweight and obesity are related to health risks, medical conditions, and an increased risk of adult obesity, with attendant impacts on morbidity and mortality. The prevalence of overweight has increased over the last 25 years among all American children and adolescents, but particularly among racial/ethnic minorities. The relatively greater increase in the upper body mass index (BMI) percentiles compared with the lower suggests that severity of overweight is increasing. Although obesity is the presumed condition impacting health, the use of the terms "at risk for overweight" and "overweight" are preferred when describing relative weight status based on age- and sex-specific BMI percentiles for children and adolescents, as these terms are more accurate and less pejorative.

Purpose: The focus of this evidence synthesis is to examine the evidence for the benefits and harms of screening and earlier treatment of overweight in children and adolescents in clinical settings.

Data Sources: We developed an analytic framework and seven key questions to represent the logical evidence connecting screening and weight control interventions with changes in overweight and behavioral, physiological, 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, using the Medical Subject Heading obesity and overweight and combining this term with predefined strategies to identify relevant English-language studies. We examined 2,162 abstracts related to screening, 312 related to screening harms, 949 related to treatment, and 864 related to treatment harms. We also contacted experts and checked bibliographies from review articles and selected trials. We found three recent, good-quality systematic reviews of interventions, one fair-to-good-quality systematic review relating screening measures to health outcomes, and a number of non-systematic, but comprehensive, review articles on screening, treatment, or other issues related to pediatric overweight. We relied on these as sources of relevant literature and, to a lesser extent, of synthesized information. When previous systematic reviews were incorporated in our results, we independently examined the individual studies to confirm or extend previous review findings. A bridge search between April 2004 and April 2005 did not identify any new intervention trials that would impact the findings of this report.

Study Selection: We included fair-to-good quality research (according to U.S. Preventive Services Task Force [USPSTF] criteria) in children and adolescents aged 2-18 years in the following categories: (1) the most current large, population-based, or nationally representative surveys of the prevalence of overweight and obesity to represent age- and, sex-specific prevalence for racial/ethnic subgroups (Mexican Americans, non-Hispanic blacks, Native Americans, Asians/Pacific Islanders, non-Hispanic whites); (2) prospective cohort studies conducted in the United States with clinically relevant childhood weight measures and adult health outcomes, including obesity; (3) randomized controlled trials (RCTs) or controlled clinical trials of screening; (4) RCTs of pharmacological agents or behavioral counseling interventions conducted in the United States or similarly industrialized countries, with at least six months' follow-up, reporting changes in overweight status with or without intermediate outcomes, health outcomes, or harms; (4) RCTs, controlled clinical trials, or controlled cohort studies of bariatric

surgeries; and (5) prospective cohort studies and controlled clinical intervention trials with at least three months' follow-up for possible harms of screening or intervention. To confirm inclusion/exclusion status, two reviewers examined all abstracts (or a random subset in the case of the screening search) and included articles.

Data Extraction: 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 using USPSTF criteria. Excluded articles were listed in tables.

Data Synthesis: No trials of screening programs to identify and treat overweight in children and adolescents have been reported. BMI (weight in kilograms [kg] divided by height in meters squared) is the preferred clinical measure for overweight. Although BMI is a measure of relative weight rather than of adiposity, it is widely recommended for use in children and adolescents to determine overweight, and correlates as well or better with measures of body fat in children and adolescents than other clinically feasible measures. Based on BMI criteria for overweight (BMI at or above the 95^{th} percentile for age and sex), 10% of two- to five-year-olds and 16% of those six and older are overweight, with significantly higher prevalence in minority racial/ethnic and sex-specific subgroups beginning at age six. Age- and sex-specific BMI percentiles for use as references for U.S. children and adolescents (CDC 2000 growth charts) have been created from nationally representative datasets that primarily included black and non-Hispanic white children. The validity of BMI-based overweight categorization in racial/ethnic minorities with differences in body composition may be limited, since BMI measures can not differentiate between increased weight for height due to relatively greater fat-free mass (muscle, bone, fluids) and increased weight due to greater fat in either individuals or populations.

BMI measures in childhood track to adulthood moderately or very well, with better tracking seen after age 12 to 13 (particularly when this age represents achieving sexual maturity), or in younger children (aged 6-12) with one or more obese parents or whose own BMI is above the 95^{th} percentile. The risk of adult obesity in those with childhood overweight (BMI $\geq 95^{th}$ percentile) provides the best available evidence by which to judge the clinical validity of BMI as an overweight criterion in children and adolescents. The probability of adult obesity in overweight adolescents is highest for 16-18 year-old white males (0.77-0.8) and white females (0.66-0.68), with little data on other race-ethnic groups in this age group available.

Limited research is available on effective, generalizable interventions for overweight children and adolescents that can be conducted in primary care or to which primary care can make referrals. Most research has investigated intensive behavioral counseling interventions conducted by specialists with repeated contacts over 6 to 12 months, many using family-based comprehensive behavioral treatments. The largest single body of research addresses children aged 8-12 years. No current research is reported in children aged two-five. The number of studies addressing adolescents is small, but increasing. Overall, current trials are limited due to small--often-selective--samples; non-comparable interventions between trials; short-term (6 to 24 months) follow-up; reporting of overweight outcomes only with minimal reporting of health outcomes; and failing to report intention-to-treat analyses.

Harms of screening and labeling children as overweight or obese theoretically include induced self-managed dieting with its sequelae, poorer self-concept, poorer health habits, disordered eating, or negative impacts of parental concern. Harms are not well reported in behavioral intervention trials. Limited good-quality evidence is available on pharmacological

approaches as an adjunct to comprehensive behavioral treatment in adolescents, and no reasonable-quality evidence is available on bariatric surgery outcomes in adolescents.

Conclusions: BMI measurement to detect overweight in older adolescents could identify those at increased risk of developing adult obesity, and its consequent morbidities. Promising interventions to address overweight adolescents in clinical settings are beginning to be reported but are not yet proven to have clinically significant benefits; nor are they widely available. Screening for the purposes of overweight categorization in children under age 12 to 13 who are not clearly overweight may not provide reliable risk categorization for adult obesity. Theoretical harms may occur from overweight labeling or from induced individual and parental concern. Screening approaches are further compromised by the fact that there is little generalizable evidence for interventions that can be conducted in primary care or are widely available for primary care referral. Despite this, the fact that many trials report short- to medium-term modest improvements (approximately 10%-20% decrease in percent overweight or a few units' change in BMI) suggests that overweight improvements in children and adolescents are possible.

Experts have identified pragmatic clinical recommendations for lifestyle changes that could be applied to all children and adolescents regardless of risk. While monitoring growth and development in children and adolescents through BMI documentation at visits is prudent, care should be taken not to unnecessarily label children and adolescents as overweight or at risk for overweight until more is known about BMI as a risk factor, and effective interventions are available.

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Chapter 1. Introduction

Scope And Purpose

This review examines the evidence for the benefits and harms of screening and earlier treatment of child and adolescent overweight in clinical settings. For simplicity, this review refers to children and adolescents collectively as children, unless sections pertain to a specific age group. This review's purpose is to summarize the current state of the evidence for primary care clinicians and identify key evidence gaps relating to clinical identification and treatment of childhood overweight. To facilitate the reading of this document, Appendix A lists all abbreviations used in the text, tables, figures, and appendixes.

Background

Burden of Illness

Obesity and overweight develop when there is a mismatch between energy intake and expenditure,[1,2] and are related to health risks and problems in children.[3] The genetic survival advantage for individuals whose bodies use calories more slowly has become a disadvantage in a society where abundant food and inactivity predominate.[4] Obesity and overweight are multi-factorial problems rooted in the interaction of the host (susceptibility due to genetics and learned behaviors), agent (energy imbalance), and environment (abundant food; reduced lifestyle activity; and economic, social, and cultural influences).[1] Obesity/overweight has been declared an epidemic[4-6] and a "public health crisis" among children in the United States and around the world[7] due to alarming upward trends in its prevalence. Overweight in children (defined by experts as a body mass index [BMI] $\geq$ 95th percentile for age and sex)[8,9] aged two and older has at least doubled in the last 25 years (Figure 1). The age- and sex-specific mean BMI and the proportion of children with BMI $\geq$ 95th percentile increased markedly in children from the mid-1970s through the 1990s, with almost all of this increase occurring in children in the upper half of the BMI distribution.[10] Thus, about 50% of children appear to have "obesity susceptibility genes" that have been acted upon by environmental changes in the last 25 years.[11]

Since increases in the mean BMI have occurred primarily due to increases in the upper half of the BMI distribution,[12] weight-related health consequences will become increasingly common in children. Health consequences of childhood overweight and obesity have been reviewed recently and include pulmonary, orthopedic, gastroenterological, neurological, and endocrine conditions, as well as cardiovascular risk factors. [7,13-18] 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 from conditions such as slipped capital femoral epiphysis,[19] while steatohepatitis and sleep apnea are somewhat more common.[20-24] Medical conditions new to this age group, such as Type 2 diabetes mellitus, [25] represent

"adult" morbidities that are now seen more frequently among overweight adolescents.[26] , In a multi-ethnic sample from an obesity clinic, for example, 4% of children and adolescents (BMIs above the 95th percentile for age and sex) had undiagnosed diabetes. All with undiagnosed diabetes were either Hispanic or black adolescents.[27] For most overweight/obese children, however, medical complications do not become clinically apparent for decades.[13]

Overweight is associated with a higher prevalence of intermediate metabolic consequences and risk factors, such as insulin resistance, elevated blood lipids, increased blood pressure, and impaired glucose tolerance.[27-32] In cohort studies, such as the Muscatine and Bogalusa Heart studies, these conditions are strongly correlated cross-sectionally with adiposity.[13] Among 2,430 adolescents aged 12 to 19 in the National Health and Nutrition Examination Survey (NHANES) III, the overall prevalence of metabolic syndrome (at least three of: elevated blood pressure, low HDL-cholesterol, high triglycerides, high fasting glucose, abdominal obesity) was 4.2%-6.1% of males and 2.1% of females (p=0.01).[29] Prevalence varied with BMI, occurring in 28.7% of overweight adolescents (BMI $\geq$ 95th percentile), 6.8% of "at risk for overweight" adolescents (BMI 85th to 95th percentile), and 0.1% of those with BMI < 85th percentile.

Perhaps the most significant short-term morbidities for overweight/obese children are psychosocial, including issues of social marginalization, self-esteem, and quality of life.[33-36] In a recent study, 10- to 11-year-old children rated same-sex obese children the least likeable, compared with children with various physical disabilities or normal healthy children.[37] This finding replicates research conducted over 40 years ago, and suggests that prejudice against obese children has not improved, and may have increased. Children referred for evaluation of severe overweight (mean BMI 34.7) are significantly more likely to report impaired quality of life (odds ratio [OR] 5.5, 95% confidence interval [CI] 3.4-8.7) than are healthy children, or those with cancer (OR 1.3, 95% CI 0.8-2.3).[38] While self-esteem is not necessarily affected in overweight children,[34] it may be reduced in overweight adolescents.[35] Adolescents with BMI > 95th percentile for sex and age are less likely to be nominated as one of their schoolmates' five best friends than normal weight adolescents, despite listing similar numbers of friends themselves.[36] Psychiatric conditions are not clearly increased in the general population of overweight children.[16]

Risk factors for developing childhood overweight have also been recently reviewed[7,39] and include parental fatness, low parental education, social deprivation, and, perhaps, infant feeding patterns, early or more rapid puberty, extremes of birth weight, gestational diabetes, and various social and environmental factors, such as childhood diet or time spent in sedentary behaviors. Racial/ethnic disparities may be largely explained by socioeconomic circumstances and parental education.[7]

Cost of Obesity and Overweight

The direct health costs of childhood overweight can only be estimated, particularly since their major impact is likely to be felt in the next generation of adults.[7] Adult obese patients are calculated to incur more health care costs than smokers or drinkers.[40] Obesity appears to reduce life expectancy, particularly when occurring in young adults,[41] and may soon exact the highest toll of all causes of preventable mortality.[42] One recent study estimated that hospital costs for obesity-related disorders in children and adolescents have more than tripled in the last two decades, based in part on the doubling of child hospital episodes for obesity-related asthma, diabetes, sleep apnea, and gall bladder disease and on lengthened hospital stays for obese

children.[43] Preventing current or future excess costs associated with obesity may be difficult if third party reimbursement for evaluation and treatment remains limited,[44] or if reimbursement hinges on the designation of overweight as a disease condition.

Condition Definition and Measurement

Defining obesity (excess body fat) in children is challenging. While gross obesity is obvious to all, differentiation of the mild case from the normal individual can be difficult, even for experts. Fatness forms a continuous spectrum from under-nutrition to "normal" fatness to gross obesity.[45] There remains no universally accepted definition for obesity in children that differentiates those with normal or healthy fat from those in whom fatness is unhealthy.[45] Epidemiological studies, obesity research, and clinical care of obese children have been hampered by this lack of clear and universally accepted diagnostic criteria.[46] "Overweight" and "obesity" are used in the literature based on a wide variety of definitions referenced to "normal" values from various reference datasets.[1] However, these values are not "norms," but rather references for comparison of growth patterns to those of a larger population. Thus, such references are not standards that reflect health, risk, or disease states, but rather they describe the distribution in the population studied.

Assuming that excess fat is the cause of adverse health consequences in children, ideal obesity measures should measure adiposity (total body fat expressed as a percent of total body weight) using valid and reliable body composition measures.[46] Body composition measurement in children is more complicated than in adults by virtue of developmental changes and normal variations in body composition between children due to sex, race/ethnicity, and other factors.[47-51] Clinically feasible, reliable, and acceptable measures of adiposity or body fat distribution are not currently available for children[52,53] (Appendix B). Instead, clinical measures of overweight based on height and weight are most commonly used (Appendix C).[46,54] BMI, measured as kilograms of weight divided by height in meters squared (kg/m^2), is widely recommended by experts as a simple and convenient measure of overweight for use during childhood,[55] particularly in adolescents.[6,9] As a proxy for obesity, however, BMI has some acknowledged limitations in accurately defining excess fat for all populations and individuals,[10] including the inability to distinguish between increased relative weight due to fat-free mass from that due to fat.[55,56] This has particular implications for non-white children due to differences in body composition, such as greater fat-free mass due to heavier bones or more muscle, and differences in growth patterns in children of different racial/ethnic groups.[50,57]

Nonetheless, BMI is currently the preferred measure of overweight in children and adolescents in the U.S. and around the world.[58,59] BMI has major advantages compared with other currently available clinical measures, including: 1) ease and reliability of measurement; 2) the most comprehensive base of normative data available for clinical measurement interpretation and for ongoing epidemiological surveillance; 3) correlating as well or better with direct measures of body fat than other competing clinical measures (e.g., triceps skinfold); 4) correlating as well or better with adult measures (tracking) than competing clinical measures (e.g., triceps skinfold, waist-to-hip ratio, Ponderal Index [weight/height cubed]); 5) sensitivity to behavioral or environmental changes (evidenced by recent population BMI increases); and 6) having the most comprehensive research base relating it to morbidity and mortality.

Typical BMI growth curves show increasing BMI levels up to about one year of age, followed by decreasing BMI levels to a nadir between ages three and seven (the "adiposity rebound" or more accurately, BMI rebound), followed by increasing BMI throughout childhood and adolescence.[60] The slope and shape of BMI curves are sex-specific and the percentile ranking of absolute BMI values varies by age.[61] Threshold BMI percentiles to define overweight have been proposed by expert consensus: BMI between the 85th and 95th percentiles for age and sex is considered at risk of overweight, while BMI at or above the 95th percentile is considered overweight (and by some authors, obese).[8,9]

A critical question is under what circumstances these BMI cutoffs should serve as a clinical overweight standard (a measure that embodies a target)[62] for individual children. An accurate determination in the individual child is needed if interventions based on BMI screening programs are to be considered.[63]

While a number of studies have found that BMI cutoffs at the upper end of the distribution are reasonably specific for classifying the fattest children,[64,65] this body of literature comprises studies using different BMI cutoff definitions and varying criterion measures of body fat, many of which are not directly comparable or are of questionable validity (Table B-1 in Appendix B).[53,63,66] There is very little evidence on the sensitivity and specificity of BMI as a screening tool for overweight or obesity, using valid reference standards composed of large U.S. samples representing boys and girls of all ages and races, with the broad range of body composition that would be seen in clinical practice. Thus, researchers have focused instead on the validity of BMI cutpoints as a pragmatic measure of risk for adult overweight and as an indicator of future morbidity or mortality.[15,59]

Prior USPSTF Recommendations

In 1996, the U.S. Preventive Services Task Force (USPSTF) recommended periodic measurement of height and weight for all patients (B recommendation).[67] Comparison of height and weight measures against appropriate age and sex norms to determine further evaluation, intervention, or referral was recommended using BMI (> 85th percentile) in adolescents, and using weight and height (or length as appropriate) plotted on growth charts or compared to average weight tables for age, sex, and height in younger children. The USPSTF has not previously 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 of the evidence concerning screening and interventions for overweight in childhood populations. We combined the findings of prior fair- or good-quality[68] systematic evidence reviews with fair- to good-quality studies not covered in these reviews or published subsequently.

Chapter 2. Methods

Terminology

Since BMI is the primary clinical measure and a measure of relative weight, we have adopted the use of the term "overweight" in children as opposed to obesity.[10] 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 designation in children is based on a BMI value alone. We adopt the term "overweight" to describe those with $\geq 95^{th}$ percentile BMI for age and sex and use "at risk for overweight" to describe those in the 85^{th} up to 95^{th} percentile for age and sex.[8,9]

Key Questions and Analytic Framework

Using the USPSTF's methods,[68] we developed an analytic framework (Figure 2) and seven key questions (KQs) to guide our literature search. The first KQ examined direct evidence that screening and intervention programs for overweight in children and adolescents improve age-appropriate behavioral, anthropometric, or physiologic measures. Because we found no evidence addressing this KQ, we searched for indirect evidence for KQs 2 through 6 to estimate the benefits and harms of screening and interventions for overweight. KQ 2 concerned appropriate standards for overweight in children and adolescents, the prevalence of overweight based on appropriate standards, and validity of clinical screening tests for overweight in predicting poorer health outcomes and obesity in adulthood. KQs 3 and 6 addressed adverse effects of screening and interventions for overweight, respectively. KQs 4 and 5 examined the efficacy of behavioral counseling, pharmacotherapeutic, and surgical interventions for improving age-appropriate anthropometric, physiologic, and health outcomes. The relationship between intervention-associated improvements in health measures and decreased morbidity in childhood or adulthood (KQ 7) was posited to be examined only in the presence of adequate evidence for intervention efficacy (KQs 4 and 5). We did not examine KQ 7 due to limited and inconsistent evidence for KQs 4 and 5.

Review methods are further detailed in the appendixes.

Literature Search Strategy

We developed literature search strategies and terms for each KQ (Appendix 4) and conducted four separate literature searches (for KQs 4, and 5; for KQs 1 and 2, for KQ 3; and for KQ 6) in Medline, PsycINFO, CINAHL, and the Cochrane library, to update the literature from previous good-quality systematic reviews (KQs 4, 5, and 6) through April 2004 or to comprehensively

examine literature from 1966 to through April 2004 (KQs 1, 2, and 3). Literature searches were focused for each KQ as described, but were reviewed with all KQs in mind. Literature searches were extensively supplemented with outside source material from experts in the field and from examining the bibliographies of systematic and non-systematic reviews (Appendix E) and included trials. We also conducted limited hand searching of pediatric obesity-focused editions of selected journals. A single investigator reviewed abstracts, with all excluded abstracts reviewed by a second investigator for all KQs except KQ 2. Due to a very large yield of abstracts in this search, we conducted blinded dual review for a random subset (27%), with acceptable agreement (97.5%) between reviewers. Inter-reviewer discrepancies during the dual review process were resolved by consensus.

To address the time lag between completion of the evidence synthesis and publication, we repeated our literature search for KQs 4-6 for April 2004-2005, since these KQ represented the most critical evidence gaps identified through the systematic review. We reviewed 333 abstracts for KQs 4 and 5 and 91 abstracts for KQ 6 were reviewed. A total of 15 articles were pulled for further review. While two studies met inclusion criteria for KQ 4, none met inclusion criteria for KQ 5 or 6. Neither study changed the report findings and both were included in our listing of pending studies.

Article Review and Data Abstraction

Using inclusion criteria developed for each key question as described in Appendix F, we reviewed 2,162 abstracts and 353 complete articles for KQ 1, 2,949 abstracts and 198 complete articles for KQs 4 and 5, and 1,176 abstracts and 36 complete articles for KQs 3 and 6. We included no articles for KQ 1, 41 articles for KQ 2, seven articles for KQ 3, 22 articles for KQs 4 and 5, and four articles for KQ 6. Listings of excluded articles are in Appendixes G and H. Two investigators quality rated all included articles and those excluded for quality reasons, using the USPSTF criteria (Appendix I).

For included studies, one primary reviewer abstracted relevant information into standardized evidence tables (Appendixes J, K, L, and M). To be within the USPSTF's scope, interventions needed to be primary care conducted or feasible (Appendix N), which were then 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.[69] Comprehensive behavioral treatments were those using a combination of behavioral modification (e.g., self-monitoring, stimulus control, cognitive-behavioral techniques), dietary modification (e.g., Traffic Light Diet,[70] reduced glycemic load, reduced fat or kilocalorie diets), and physical activity components (broadly specified as aerobic, calisthenic, lifestyle, or decreased sedentary behaviors) (Appendix O).

To be included, studies had to report weight outcomes, preferably as BMI or BMI percentile changes. We also recorded all reported behavioral, physiological, and health outcomes specified on our analytic framework (Figure 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 impact of current clinical interventions on overweight, we converted baseline measures and outcomes to BMI percentiles and plotted results on Centers for Disease Control and Prevention (CDC) growth charts. Treatment effects that were typical of interventions in 8- to 12-year-olds (10%-20% reduction in percent overweight after one year) were modeled and plotted for 8-,10-, and 12-year-old girls. We plotted mean BMI treatment effects at six months or longer for all trials in adolescents for which this was possible. Methods are further described in Appendix P. Using a best evidence approach, we constructed a screening "outcomes" table to examine the proportion of adolescents with BMI $\geq 95^{th}$ percentile who would develop adult morbidities due to excess adult weight, and the proportion of morbidities potentially prevented by our estimates of reasonable effect sizes for adolescent intervention (Appendix Q). Using the USPSTF approach, we summarized the overall quality of the evidence for each key question.

USPSTF Involvement

The authors worked intermittently with six liaisons from the USPSTF at key points throughout the review process to develop and refine the analytic framework and key questions and to present the evidence in a format that would be most useful to the USPSTF. 

Chapter 3. Results

Key Question 1. Is there direct evidence that screening for overweight in children/adolescents improves age-appropriate behavioral or physiologic measures, or health outcomes?

Our searches found no studies, nor did examination of all individual trials included in previous systematic evidence reviews.[65,71-74]

Key Question 2a. What are appropriate standards for overweight in children/adolescents and what is the prevalence of overweight based on these?

Eight nationally representative health examination surveys that included children have been conducted in the United States since 1963 (Appendix R).[54,75] These surveys have gathered a variety of anthropometric measures on children aged two months to 18 years that can be used to provide growth references (a tool for providing a common basis for purposes of comparison)[62] for children, as well as trend analyses of changes in the population over time. In order to provide useful trend analyses, measures must be valid, gathered consistently in surveys, and must use a single source for comparison. Due to one or more of these limitations, almost all data on prevalence and trends in U.S. children are based on BMI measures calculated from standardized weight and height information.[1]

BMI measurements must be compared to a reference population to determine their age- and sex-specific percentile ranking. While many reference datasets for childhood BMI are available, three that are commonly cited in current literature are: 1) NHANES I for children aged 6-19, which has been used widely in the United States and internationally; 2) the CDC's 2000 gender-specific BMI growth charts for children 2 through 19 years (based on National Health Examination Survey [NHES] II and III, NHANES I and II, and NHANES III for children under six years); and 3) the International Obesity Task Force (IOTF) standards for obesity derived from six different countries, including the U.S., for children aged 2 through 18 years to match the adult cutoffs of BMI of 25 (overweight) and 30 (obese) at age 18.[1] These three sets of BMI references give similar, but not identical, estimates of the prevalence of overweight in the U.S.[76] In this report, we focus on the current prevalence estimates and trend information available from the NHANES program, which uses the CDC's 2000 gender-specific BMI growth charts as their reference dataset. These are widely available to clinicians and provide curves smoothed to the nearest month in the data, rather than the nearest half-year or birthday, and are viewed to be generally preferable for use in the United States.[76] NHANES provides the most comprehensive data available on boys and girls aged 6 months through 19 years, and recently includes over-sampling of black and Mexican American children.

Prevalence

Using BMI $\geq$ 95th percentile, the prevalence of overweight in 1999-2002 was 10% in two- to five-year-olds and 16% in those six years and older[77] (Figure 3). For children two to five years of age, the prevalence was similar between all racial/ethnic subgroups and both sexes, but was lower than the prevalence in older children in the same racial/ethnic subgroups. Among children 6 to 11 years, differences were seen between racial/ethnic subgroups, with significantly more Mexican American (21.8%) and non-Hispanic black (19.8%) children categorized as overweight, compared with non-Hispanic whites (13.5%) ($p<.05$). Sex-specific differences were also seen, with the highest prevalence of overweight in 6- to 11-year-olds among Mexican American boys (26.5%), which was significantly higher than non-Hispanic black boys (17%), non-Hispanic white boys (14%), and Mexican American girls (17.1%), and similar to that of non-Hispanic black girls (22.8%). Among youth aged 12 to 19 years, significantly more non-Hispanic black (21.1%) and Mexican American (22.5%) children had overweight BMI measurements than non-Hispanic whites (13.7%) ($p<.05$), with no differences between males and females.

NHANES does not provide separate estimates for Native American children. In a population-based survey of 12,559 schoolchildren aged 5 to 17 years representing 18 tribes in the Midwest in 1995-1996, the overall age-adjusted prevalence of overweight was significantly greater in males (22.0%, 95% CI 21.0-23.0) than in females (18.0%, 95% CI 17.0-19.0).[78] In five-year-olds, the overweight prevalence was not significantly different in boys (16.1%) and girls (11.6%). Between ages 6 and 11 years, overweight prevalence remained similar between sexes, ranging from 12.0% to 24.6% in boys and 15.0% to 20.7% in girls. Beginning at age 12, males had consistently higher prevalence of overweight (20.9% to 25.9%) than females (15.9% to 22.5%).

Given the differences in reporting, it is difficult to directly compare prevalence estimates for Native Americans to other races/ethnicities. However, the overweight prevalence of Native American boys aged 6 to 11 is between that of non-Hispanic blacks (the second-highest prevalence) and the most prevalent group, Mexican Americans. Similarly, for girls 6 to 11, the prevalence of Native American overweight appears to rank between the second-highest (Mexican Americans) and the highest groups (non-Hispanic blacks). In adolescents, a similar ranking is seen in boys and girls, despite the younger age representation of Native American children (12 to 17 years) compared with other races (12 to 19 years). Thus, it appears that Native American children aged 6 to 17 rank as at least the second-highest group in prevalence of overweight among races.

The prevalence of overweight based on BMI changed little between 1960 and 1980 among children and adolescents in the United States.[1] Using the same reference population, sex- and age-specific subgroups aged 2-19 years showed an increase in overweight prevalence between the 1988-1994 and 1999-2000 surveys, which was similar to, or greater than, increases during the longer time period between earlier surveys (NHANES II [1976-1980] and NHANES III [1988-1994]).[75] In the 6-12 years before 2000, statistically significant increases in the prevalence of BMI measures above the 95th percentile threshold for overweight occurred among all 2- to 5-year-olds (3.1%), all 6- to 11-year-olds (4.0%), and all 12- to 19-year-olds (5.0%), with 12- to 19-year-old boys increasing 4.2% and girls increasing 5.8% ($p < .05$). When analyzed by race/ethnicity, only non-Hispanic black and Mexican American children exhibited statistically significant increases in prevalence between 1988 and 2000. In Mexican Americans, the prevalence increased 13% in boys, and the prevalence increased 10% in both non-Hispanic

black boys and girls in. Among all 12- to 19-year-olds, 11.2% met the adult definition of obesity (BMI of 30 or higher), with rates particularly high among non-Hispanic black females (20%) and Mexican American females (16%). Other data also demonstrate increased severity of excess weight among overweight children,[79] especially black and Mexican American children,[12] increasing the sense of urgency about childhood overweight.[80]

Important caveats apply to estimating the prevalence of overweight prevalence and trends among groups other than non-Hispanic whites. Unfortunately, representative national data are unavailable to reliably estimate the prevalence of overweight in children and adolescents of Asian/Pacific Islander descent. While recent surveys such as NHANES 1999-2000 have over-sampled Mexican Americans and non-Hispanic blacks,[75] comparable race/ethnicity information for these groups is limited to NHANES III and NHANES 1999-2000, with some supplementation by Hispanic HANES (1982-1984) (Appendix R). Commonly used growth references, such as the CDC's sex-specific BMI-for-age charts, which are based primarily on white and black samples from 1963 through 1980 (through 1994 for those under age 6),[61] may inaccurately measure the prevalence of overweight in Mexican American youth[54]or in Native Americans. Similarly, blacks are represented but do not make up the majority, and metabolic consequences in blacks, whites, and Hispanics at the same BMI z-score have been shown to differ.[30] These same issues could pertain to applying the available growth references to Native Americans. Given the known differences in body composition and growth and development between races,[47,57,81,82] and possible differences in the validity of BMI as a proxy for percent body fat in different races,[50] it will be important to clarify the health significance of BMI measurements at various ages among boys and girls of racial/ethnic subgroups.[49]

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 (Table J-1 in Appendix J). BMI measurements tracked as well as or better than other overweight measures, such as Ponderal Index or skinfold measures. We focus on the correlation between BMI measurements in childhood and adulthood (Table 3), and between overweight childhood BMI percentiles and the probability of adult obesity (BMI > 30) (Table 4) in selected fair- to good-quality prospective U.S. studies.[83-91] Single BMI measures track reasonably well from childhood and adolescence (aged 6 to 18) into young adulthood (aged 20 to 37), as evidenced by longitudinal studies showing low to moderate ($r = 0.2$-0.4) or moderate to high (0.5-0.8) correlations between childhood and adult BMI measures (Table 3). Increased tracking ($r \geq 0.6$) is seen in older children (after age eight, particularly when this age represents sexual maturity), in younger children (aged 6 to 12) who are more overweight (usually above the 95th or 98th percentile), and in children with one or more obese parent (also see Appendix J). Data on tracking for children before the age of 12 are not extensive. Sex differences in tracking are not consistent across ages or within age categories. Limited data are available comparing white and black children.

In terms of childhood overweight tracking to adult obesity, the probability is highest among white overweight males (0.77-0.8) and white overweight females (0.66-0.68) aged 16-18. When considered by age groups (Table 4), a 50% or greater probability of adult obesity (BMI > 30) is primarily reported in children over age 13 whose BMI measures are at or above the age- and sex-specific 95th percentile, with one exception. In the Bogalusa Heart Study (67% white and 32% black), children grouped across ages (5 to 17 years) with BMI levels at or above the 85th percentile had a 0.51-0.77 probability of adult obesity.

Key Question 2c. What clinical screening tests for overweight in childhood are reliable and valid in predicting poor health outcomes in adulthood?

Although a large number (n=11) of prospective or retrospective U.S. studies examined the risk associated with childhood overweight and adult outcomes--including socioeconomic outcomes, mortality, and a range of adult cardiovascular risk factors and morbidities--these studies rarely controlled for adult BMI, a critical potential confounder. In one that did, [89] the apparent association between elevated BMI at age 10 and elevated cardiovascular risk factors in adulthood was eliminated after controlling for adult BMI (also see Appendix K). While these data are useful in illustrating expected health consequences that may occur when childhood obesity persists into adulthood, it is not as useful in determining the level of health risk associated with childhood overweight measures that is independent of adult weight status.

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, or negative impacts from parental concerns.

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?

Potential interventions to improve weight status in children include behavioral counseling interventions, pharmacotherapy, and surgery. Experts agree that surgical approaches should be considered only in adolescents with extreme and morbid obesity, and that pharmacologic approaches should also be limited to a second-tier approach after failed behavioral counseling.[92] We did not limit our intervention studies to U.S. populations, but included interventions from other Western industrialized nations.

Behavioral Counseling Interventions

The most extensive treatment literature for childhood overweight involves behavioral counseling interventions. Behavioral counseling interventions are those activities delivered by primary care clinicians and related healthcare staff to assist patients in adopting, changing, or maintaining health behaviors proven to affect health outcomes and health status.[93] These interventions may occur, all or in part, during routine primary care and may involve both visit-based and outside intervention components, including referral to more intensive clinics in the community. Behavioral counseling interventions reviewed here included behavioral modification special diets, and/or activity components delivered to children and/or parents as individual or in groups.

We considered all trials published since 1985 (n = 22 from 23 publications) that addressed interventions that were feasible for primary care conduct or for primary care referral (including one that combined comprehensive behavioral treatment with pharmacotherapy, described separately below).[94-116]We limited our consideration to post-1985 trials given the dramatic increases in overweight in children that have occurred during the 1980s and 1990s.[5,11,80] A previous good-quality systematic review covering 16 of these trials concluded that this behavioral counseling treatment literature is limited, due to small sample sizes and marginal-quality trials testing primarily non-comparable interventions delivered in specialty obesity clinic treatment settings to significantly overweight school-aged children (40%-50% above ideal weight) with primarily short-term outcomes.[72] We found limited improvement from these conclusions by including six additional studies published in the interim (Table 5). These studies continued to be very small (16 to 82 participants), to primarily analyze treatment completers only, and to examine very different interventions over a relatively short period of time. Studies also tended to target those who were quite overweight (see Figure 4). Inclusion criteria and weight outcome measures tended to be BMI-related more than the earlier literature, likely reflecting the growing consensus about the use of BMI.

Over half (n=13) of fair- or good-quality trials[94,96-102,105,107,112,113,116] reported intermediate behavioral (n=11) or physiologic (n=7) measures in addition to weight outcomes (Table 5). These outcomes were more common in recent research, and five of the six recent studies reported one or more. Two good-quality trials[113,116] reported behavioral changes but no

physiological outcomes. While one of these trails[116] 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 kilocalories,[96] while other fair-quality studies[97-102,107,112] measuring changes in eating behaviors, physical activity, and sedentary behaviors did not provide a clear picture due to differences in subjects, interventions, and measures. No good-quality trials of behavioral treatment without pharmacologic adjuncts reported intermediate physiologic outcomes, such as lipids or lipoproteins, glucose tolerance, blood pressure, or physical fitness measures. Only one trial of at least fair quality reported key intermediate physiologic measures such as lipids or lipoproteins, glucose tolerance, or blood pressure. After an intensive six-month behavioral weight-control program comparing a reduced glycemic load (RGL) diet with a reduced fat (RF) diet, insulin resistance scores (measured by the homeostatic model) increased significantly less in the RGL group than the RF group (-0.4 +/- 0.9 vs. 2.6 +/- 1.2, p=0.03).[96] Insulin resistance, however, increases with sexual maturation, which was not assessed. The significance of these results is further limited given baseline differences between groups and lack of consideration of physical activity as a confounder. The other fair-quality studies measured other physiological outcomes such as physical work capacity or physical fitness and most reported some improvement when physical activity or sedentary behaviors were addressed in the intervention.[97,98,101,102]

Considering all trials covered in the earlier review and this one (Table 5), no current trials addressed preschool children (two to five years of age). The majority of trials addressed children aged 6 to 12or 13 years (n = 15), with a growing number of studies addressing adolescents aged 11 or 12 years and older (n = 7). Studies generally include boys and girls, with some over-representation of females. Few studies clearly included 10% or more non-whites (n = 7), and many did not report participants' race/ethnicity (n = 12). Two studies included 100% black female adolescents,[115,116] but only one met at least fair quality criteria.[116]

Comprehensive behavioral treatment programs have been the most studied intervention for overweight (Table 5). Fair- or good-quality studies have produced from 7% to 26% (generally 10%-20%) decreases in percent overweight (the most commonly reported outcome) from baseline after 12 to 24 months of intensive treatment. Much of this research has come from a single research group treating select patients aged 8-12 in a multidisciplinary obesity clinic setting specializing in behavioral therapy approaches to changing diet and activity behaviors.[70] Figure 4 models the BMI impacts of a 10%-20% reduction in overweight after 12 months in girls enrolled at ages 8-12. Methods for these calculations are described in Appendix P.

Long-term (after 5 to 10 years) weight outcomes from a set of these studies in 8-12 year olds in a multidisciplinary obesity clinic setting[117] generally maintained or improved weight-related treatment measures for the majority of patients. These studies are often cited,[118,119] but we excluded them from our review because most were prior to 1985, all involved intensive treatment-to-treatment comparisons without untreated controls, and we could not confirm the long-term results met our quality criteria (acceptable loss to follow-up of all those randomized, since analyses were not intention to treat).[68] Although this evidence offers hope of some success in treating childhood overweight for some subgroup of those treated, more generalizable and reliable evidence will be needed to accurately predict the probability of long-term treatment success in the broader population of overweight children and adolescents and to understand more about treatment responders.

Figure 5 demonstrates results from behavioral counseling studies in adolescents[96,112,113,115,116] with two good-quality studies particularly relevant to primary care.[113,116]

One short-term, primary care conducted trial used a computer-based approach to generate tailored plans for counseling of obese (above the adult BMI cutoff of 30) adolescents (aged 12 to 16) by trained and experienced pediatricians, supplemented with multiple follow-up telephone counseling calls from a qualified counselor.[113] Significant but small benefits were seen in BMI measures at seven months, primarily from stabilizing BMI (eliminating BMI increases) in those receiving the intensive intervention. While the magnitude of these benefits would be understated by this design, which compared two active treatments, changes were modest (Figure 5).

Similarly, a short-term trial that would be feasible for primary care involved an Internet- and e-mail-based family intervention targeting 57 overweight (mean BMI 36.37 kg/m^2) non-Hispanic black females aged 11 to 15 years with at least one obese biological parent. [116] Compared to a diet and physical activity education intervention, the comprehensive behavioral intervention resulted in a statistically significant difference in weight and BMI change from baseline between the two groups at six months, due largely to prevention of weight or BMI gain in the experimental group.

Although both trials showed small but statistically significant benefits in BMI measures at 6-12 months, it is not clear that these BMI changes would have clinical benefits or be sustained.

Pharmacotherapy

One randomized placebo-controlled trial of sibutramine within a comprehensive behavioral treatment program in adolescents showed superior weight change outcomes after six months (4.6 kg greater weight loss, 95% CI 2.0-7.4 kg) in an intent-to-treat analysis[94] (Figure 5). With continued use, weight loss at six months was maintained through 12 months. The rate of adverse effects and discontinuation, however, was fairly high (12% discontinued and 28% reduced the medication). It is not clear that the additional short-term weight change provided a net benefit, since changes in serum lipids, serum insulin, serum glucose, and HOMA (homeostatic model of insulin sensitivity) not differ between the groups. Among all trial completers (63%-76% of all participants) significant improvements from baseline were seen at 12 months in HDL cholesterol, serum insulin, and HOMA. Blood pressure was not improved, and in some cases, increased blood pressure was a reason for discontinuation. We found no evidence for the use of metformin for weight loss/disease prevention in normoglycemic obese adolescents with weight outcomes after more than three months, nor did we find acceptable evidence on alternative or complementary therapies.

Surgery

acceptable-quality evidence evaluated the effectiveness of surgical approaches to overweight in adolescents. There are no controlled treatment outcome data on bariatric surgery approaches in adolescents. Stringent NIH guidelines for surgery in the morbidly obese adolescent[120] specify strict qualification criteria and performance of surgery only in specialized centers with comprehensive weight-management programs. For mature, morbidly obese adolescents with comorbidities who meet these criteria, the evidence in adults[121] may be considered as a surrogate evidence source.

Key Question 5. Do interventions lead to improved adult health outcomes, reduced childhood morbidity, and/or improved psychosocial and functional childhood outcomes?

Behavioral Counseling Interventions

Few (n = 3) studies reported health outcomes as defined in our analytic framework,[103,112,115] and two of these were rated at least fair quality (Table 5). In one fair-quality trial, depression scores, measured using reliable and valid instruments, showed improvement from baseline in treated adolescent girls but not controls, while reliably measured self-esteem scores improved from baseline in both groups.[112] In a second fair-quality study, significantly fewer children aged 8-12 who received comprehensive behavioral treatment had elevated total behavior problem scores or elevated internalizing behavior problem scores at 24 months' follow-up than at baseline.[103]

Key Question 6. Do interventions have adverse effects, such as stigmatization, binging or purging behaviors, eating disorders, suppressed growth, or exercise-induced injuries?

Adverse effect reporting for behavioral counseling interventions was limited to 3 of 22 intervention trials.

Behavioral Counseling Interventions

Potential eating problems or weight management behaviors were the only harms addressed in two 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 concerns, and eating disorder psychopathology) after primary care-based comprehensive behavioral treatment in 37 of 44 adolescent trial completers.[113] One fair-quality trial reported no effect on eating disorder symptoms, weight dissatisfaction, or purging/restricting behaviors in 47 8- to 12-year-olds in a family-based comprehensive behavioral treatment program, using a reliable measure (Kids' Eating Disorder Survey) that has been validated in slightly older children.[103,122] 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,[94] 44% (19/43) of patients in the active medication group reduced or discontinued the medication due to elevated blood pressure, pulse rate, or both. These were the main adverse events reported.

Surgery

We attempted to estimate the rate of harms from the uncontrolled cohort literature, but found loss to follow-up (25-60% at 4-24 months)[123-125], and inadequate reporting prevented us from making reasonable estimates of rates of surgery-associated harms.

Summary of Evidence Quality

Table 6 summarizes the overall quality of evidence according to USPSTF criteria[68] for each of the key questions addressed in this review. The overall evidence is poor for the direct effects of screening programs (KQ 1), screening harms (KQ 3), and bariatric surgery (KQs 4 and 5). The overall evidence is fair-to-poor for behavioral counseling interventions (KQs 4 and 5) due to small, non-comparable short-term studies with limited generalizability that rarely report health or intermediate outcomes, such as cardiovascular risk factors. Trials are particularly inadequate for non-whites and children aged 2-5. Fair-to-poor evidence is available for behavioral counseling intervention harms due to very limited reporting (KQ 6). Fair evidence supports childhood BMI as a risk factor for adult overweight, although data are again limited in non-whites (KQ 2b), and data addressing BMI as a risk factor for adult morbidities generally do not control for confounding by adult BMI (KQ 2c). Good evidence is available on the prevalence of overweight based on BMI measures in all groups except Native Americans and Asians (KQ 2a).

Chapter 4. Discussion

Conclusions

Overweight has at least doubled in children and adolescents in the U.S. over the last 25 years. There is no doubt that this increase represents a major public health concern with the potential for future health risks and growing burdens on the healthcare system. Clinicians who provide health care to children and adolescents may be in an excellent position to address this. In terms of evidence, however, little has changed since a 1998 editorial in the *Journal of Pediatrics* 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."[126] Given the nature of the problem, effective solutions will likely require substantial collaboration between the medical and the public health communities. Further understanding of ways to expand the appropriate role of the clinician in community public health, such as through advocating necessary environmental and political changes, would be helpful.[127,128]

A major limitation to clinicians' addressing overweight in the broad population of children in medical practice, 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. This uncertainty is due to limited knowledge of the current and future health impacts of increased BMI in children, and possible limits in the applicability of current BMI cutpoints for overweight to some individual children and adolescents, particularly those of minority race/ethnicity. As stated well by others, "In clinical practice, the variations found in body fat mass and non-fat mass for a given body weight may make any judgment based on weight (adjusted for height and/or for age) unreliable as an estimate of an individual's actual body fat. At higher levels, BMI and the BMI cutoffs may be helpful in informing clinical judgment, but at levels near the norm additional criteria may be needed."[7] Understanding normal variations in body composition with age, sex, race/ethnicity, sexual maturity, and other factors will be critical to accurately defining 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 cutoffs for identifying overweight children using large, representative samples of U.S. children of all ages and races/ethnicities would increase our understanding of BMI as a screening tool.[64]

The risk for overweight children to become overweight or obese adults has been judged as the best available criterion to judge the clinical validity of BMI in the pediatric age group.[59] Adult BMI has been clearly associated with morbidity and mortality, particularly at higher BMI levels,[129] though there is no single threshold for increased health risks.[130] Adolescents who are at or above the 95th percentile for age- and sex-specific BMI clearly have an increased probability of adult obesity, and early interventions may be potentially beneficial. Recent interventions targeting this age group primarily addresses those who are very overweight, with some studies showing short-term (6-12 month) weight-related improvements. The treatment evidence in this age group could be strengthened by larger trials testing generalizable

interventions that can demonstrate sustained effects on overweight status, weight-related health outcomes, risk factors. Many trials in adolescents have specifically targeted minorities [115,116] or enrolled reasonable proportions in their studies ,[94,110,113]and this should continue. Trials among adolescents who are mildly overweight, and those more severely affected, are needed.

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

For all ages, there is very limited evidence for overweight treatment that is feasible for primary care delivery or referral. Generalizability from the existing evidence is a major concern. Few studies have taken place in primary care or seem primary care feasible –most have been conducted in research or specialty obesity clinics using non-medical personnel, and are not widely available for primary care referral. Indeed, experts have cautioned that behavioral therapy is not simply the third component in a triad of intervention elements (along with a diet and exercise plan), but represents an expertise-driven approach to improving diet and physical activity using behavioral principles. These principles include patient-specific means of specifying the behavior to be changed, breaking it down into smallest behavioral units, helping the patient to make changes, and monitoring and reinforcing change.[131] If larger studies confirm that the behavioral skills and approaches tested thus far are key to success, it will be critical to find a way to create referral clinics or involve clinical team members with expertise in behavioral medicine/psychology in implementing weight control programs.[131]

Experts recommend referring certain children to pediatric obesity treatment centers for expert management. These include children who are massively overweight (defined through clinical judgment)[8] or who have BMI exceeding the 95th percentile with one or more associated severe morbidities that require immediate weight loss. In asymptomatic children with a BMI $\geq$ 95th percentile, these experts have also recommended an in-depth medical assessment to detect treatable causes of obesity, risk factors, and comorbidities, and to determine need for treatment. For children whose BMI falls between the 85th and 95th percentiles for age and sex, 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 risk factors or morbidities. It was beyond the scope of our review to systematically evaluate other types of evidence to support these expert recommendations; however, this is an important area for future systematic review.

Experts emphasize talking to families about energy balance behaviors that prevent obesity, promote other aspects of health, and are likely to cause no harms.[132] These behaviors include limiting television viewing, encouraging outdoor play, and limiting the consumption of sugar-sweetened soft drinks. For the interested clinician, pragmatic approaches for all children (particularly young children) emphasizing the "healthy lifestyle prescription" approach over targeting overweight identification seem appropriate since we found limited evidence for secondary prevention or treatment. Similarly, others have found limited evidence for the effectiveness of primary prevention in clinical settings.[133]

Given the current evidence, perhaps BMI measurement in children should be performed as a growth-monitoring tool that may indicate future risk for adult overweight and its attendant morbidities, rather than as a screen for determining current overweight. Children, particularly those under the age of 13, 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 in children and adolescents.[134] Such monitoring will likely prove valuable as our understanding about the predictive value of growth levels and patterns, and overweight status, change over time, and effective ways to address patterns that indicate overweight.

Limitations of the Literature

In the absence of direct evidence screening's impact on improved weight and health outcomes in children and/or adults, we have evaluated and linked indirect evidence for screening and intervention. In the current literature, evidence linkages between screening and intervention are hampered by divergent definitions of overweight. In the epidemiological research addressing childhood overweight as a risk factor, more recent reports focus on BMI percentiles or z-scores (which can be translated approximately into percentiles), and many use the current CDC growth charts as references. In the treatment literature, few studies use BMI percentiles as weight entry criteria, probably due to the older age of the literature. More recent studies use BMI-based criteria and are more explicit about their references for its use. 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 in children and adolescents, and lack of criterion standards for adiposity in children, hampered our ability to determine the test characteristics (sensitivity and specificity) of clinically feasible screening tests. The state of the science is changing,[135] with evidence comparing diagnostic tests with valid body composition measures beginning to emerge from other countries. This evidence should become more available in U.S. populations in the future. Similarly, weight-related criterion standards with clearly established current or future health consequences for children of both sexes and all ages and races/ethnicities will enable diagnostic test research addressing BMI and other measures. We limited our review of the relationships between childhood weight status (primarily BMI) and adult health consequences / adult obesity to longitudinal U.S. studies. While this may have given us an advantage in minimizing the differences due to overweight definitions, reference standards, and country and population measurement differences,[136] such an approach may have unnecessarily eliminated our review of applicable data. Much of this research was based on non-Hispanic whites, which limits applicability to minorities in whom the prevalence of overweight is increasing.

We did not locate adequate longitudinal data relating childhood weight status to childhood health outcomes, and thus 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 them over time) to intermediate measures, such as blood pressure or lipids, rather than health outcomes.

The literature or pediatric obesity is growing due to enormous public health and scientific interest, which makes it challenging to ensure comprehensiveness. It is possible that some relevant literature was missed or excluded through our methods. Reviewing this literature is complicated by the rapid evolution in appropriate methods to define, diagnose, monitor, and intervene with childhood and adolescent overweight and obesity for different ages, sexes, and racial/ethnic and cultural groups. Although we made an effort to comprehensively review several areas of the literature, some areas were not reviewed. We did not review any evidence on children under the age of two, although this is an active area for research. We did not attempt to examine risk factors for childhood overweight, but note that others have recently done so.[39] Similarly, research about approaches to changing daily life habits, dietary intake, and physical activity may help address and prevent pediatric overweight, including intriguing evaluations of television and overweight.

Future Research

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 in children. Despite the fact that many of these gaps were identified over 10 years ago,[137] little subsequent research has addressed the most clinically relevant questions. In addition to the clinical research already underway to address childhood overweight prevention and treatment (Table 7), we strongly urge the research community to prioritize research studies that will supply needed evidence to address the key questions formulated for this report in order to inform pragmatic clinical, as well as public health, prevention strategies. Some of these may result from reporting from existing good-quality cross-sectional and longitudinal cohort studies in addition to new studies and clinical trials.

In order to address the direct and indirect impact of clinical screening programs on health outcomes, an established definition of overweight level(s) based on documented health effects is urgently needed. Common and agreed-upon definitions for overweight and obesity within the U.S., if not internationally, must be established to facilitate the integration of epidemiological and clinical research and the speed with which we can learn. Clinicians will also need to understand how risks and morbidities vary with overweight levels, age, and other factors in order to support subsequent screening in those identified as overweight. Key needed research includes:

a. Diagnostic accuracy testing for BMI (sensitivity and specificity) compared to a valid gold standard in detecting clinically significant overweight in children and adolescents.

b. Refine single and repeated BMI measures over time, in conjunction with other pragmatic clinical measures, for use in children. This may include creating growth references for children eight and older to include maturational status, confirming that current growth references adequately apply to minority children known or suspected to have different growth trajectories and body compositions (black, Asian, Native American, and Mexican American children), and developing and validating approaches to supplement modestly elevated BMI measurements with added medical measurements or risk factor status. The

latter might include parental obesity, physical examination, laboratory tests, and/or anthropometric measures, such as waist-hip ratio, triceps skinfold, or clinical adiposity measures such as bioelectrical impedance assay, once they are sufficiently validated.

c. Continued investigation of growth trajectories and susceptible periods for overweight development and their role as predictors of future morbidity and adult overweight and obesity. True "growth curves" for BMI and other measures should be created from longitudinal data and related to current and subsequent morbidities, as growth patterns may be more relevant than measurement at a single time point. Reporting of tracking between childhood BMI (single or repeated BMI measures, or growth patterns) and adult weight status should demonstrate similarities and differences due to age, sex, and race/ethnicity. Prognostic studies of childhood overweight and adult morbidities should control for adult weight status.

d. Better understanding of overweight-associated risks in children and adolescents and how they vary by age, gender, and racial/ethnic subgroups in order to develop realistic goals and strategies to improve short-term and long-term health in an environment conducive to high levels of overweight. Research is needed to establish the prevalence of, and prospective risk for, medical and psychosocial risk factors and morbidities by various weight-related measures (such as BMI percentiles) for race-, age-, and sex-specific groups using large, racially and ethnically representative samples of children and adolescents. Clinical measurement strategies should account for differences in body composition due to race/ethnicity, maturational status, athletic development, and linear growth,[50] particularly when these affect weight-related health consequences.

e. Clear evidence-based approaches to medical and psychological screening in children identified as overweight at various ages, including research on the benefits of screening for and managing cardiovascular and diabetes disease risk factors in overweight adolescents.

f. Clinical research on the harms and acceptability of overweight screening.

Intervention programs to reduce weight-related risks and morbidities in children and adolescents identified through screening programs are required to justify screening. These must be feasible for primary care delivery or referral and should address children at all overweight levels. Key needed research includes:

a. Experimental and quasi-experimental studies of child and adolescent obesity treatment on BMI and childhood and adult psychosocial and health outcomes,[31] with a priority on adolescents.

b. Clinical or observational research on changes in morbidity among children and adolescents who lose weight and maintain this loss or those that regain it in adulthood.[31]

c. Clinical research on the long-term maintenance of healthy weight changes in children and adolescents.

d. Research on practical approaches to overweight treatment that can be implemented in pediatrician and family practice offices using novel adaptations and partnerships in the health care system and the community.[127]

e. Randomized clinical trials on clinical approaches by pediatricians and family practitioners to overweight prevention in children and adolescents, and their interaction with population approaches, such as school- and family-based approaches.

f. Clinical research on the harms and acceptability of overweight treatments.

References

1. Ogden CL, Carroll MD, Flegal KM. Epidemiologic trends in overweight and obesity. *Endocrinology & Metabolism Clinics of North America* 2003; 32(4):741-760, vii.

2. Troiano RP, Flegal KM, Kuczmarski RJ, Campbell SM, Johnson CL. Overweight prevalence and trends for children and adolescents. The National Health and Nutrition Examination Surveys, 1963 to 1991. *Archives of Pediatrics & Adolescent Medicine* 1995; 149(10):1085-1091.

3. Morgan CM, Tanofsky-Kraff M, Wilfley DE, Yanovski JA. Childhood obesity. *Child & Adolescent Psychiatric Clinics of North America* 2002; 11(2):257-278.

4. Sokol RJ. The chronic disease of childhood obesity: the sleeping giant has awakened. *J Pediatr* 2000; 136(6):711-713.

5. Flegal KM. The obesity epidemic in children and adults: current evidence and research issues. *Med Sci Sports Exerc* 1999; 31(11 Suppl):S509-S514.

6. Kohn M, Booth M. The worldwide epidemic of obesity in adolescents. *Adolescent Medicine State of the Art Reviews* 2003; 14(1):1-9.

7. Lobstein T, Baur L, Uauy R. Obesity in children and young people: a crisis in public health. *Obes Rev* 2004; 5 Suppl 1:4-85.

8. Barlow SE, Dietz WH. Obesity evaluation and treatment: Expert Committee recommendations. The Maternal and Child Health Bureau, Health Resources and Services Administration and the Department of Health and Human Services. *Pediatrics* 1998; 102(3):E29.

9. Himes JH, Dietz WH. Guidelines for overweight in adolescent preventive services: recommendations from an expert committee. The Expert Committee on Clinical Guidelines for Overweight in Adolescent Preventive Services. *Am J Clin Nutr* 1994; 59(2):307-316.

10. Troiano RP, Flegal KM. Overweight children and adolescents: description, epidemiology, and demographics. *Pediatrics* 1998; 101(3 Pt 2):497-504.

11. Koplan JP, Dietz WH. Caloric imbalance and public health policy. *JAMA* 1999; 282(16):1579-1581.

12. Dwyer JT, Stone EJ, Yang M, Webber LS, Must A, Feldman HA et al. Prevalence of marked overweight and obesity in a multiethnic pediatric population: findings from the Child and Adolescent Trial for Cardiovascular Health (CATCH) study. *Journal of the American Dietetic Association* 2000; 100(10):1149-1156.

13. Must A, Strauss RS. Risks and consequences of childhood and adolescent obesity. *Int J Obes Relat Metab Disord* 1999; 23 Suppl 2:S2-S11.

14. Must A. Morbidity and mortality associated with elevated body weight in children and adolescents. *Am J Clin Nutr* 1996; 63(3 Suppl):445S-447S.

15. Reilly JJ, Methven E, McDowell ZC, Hacking B, Alexander D, Stewart L et al. Health consequences of obesity. *Arch Dis Child* 2003; 88(9):748-752.

16. Zametkin AJ, Zoon CK, Klein HW, Munson S. Psychiatric aspects of child and adolescent obesity: a review of the past 10 years. *J Am Acad Child Adolesc Psychiatry* 2004; 43(2):134-150.

17. Must A, Anderson SE. Effects of obesity on morbidity in children and adolescents. *Nutr Clin Care* 2003; 6(1):4-12.

18. Rodriguez MA, Winkleby MA, Ahn D, Sundquist J, Kraemer HC. Identification of population subgroups of children and adolescents with high asthma prevalence: findings from the Third National Health and Nutrition Examination Survey. *Archives of Pediatrics & Adolescent Medicine* 2002; 156(3):269-275.

19. Kelsey JL. Epidemiology of slipped capital femoral epiphysis: a review of the literature. *Pediatrics* 1973; 51(6):1042-1050.

20. Silvestri JM, Weese-Mayer DE, Bass MT, Kenny AS, Hauptman SA, Pearsall SM. Polysomnography in obese children with a history of sleep-associated breathing disorders. *Pediatr Pulmonol* 1993; 16(2):124-129.

21. Mallory GB, Jr., Fiser DH, Jackson R. Sleep-associated breathing disorders in morbidly obese children and adolescents. *J Pediatr* 1989; 115(6):892-897.

22. Tominaga K, Kurata JH, Chen YK, Fujimoto E, Miyagawa S, Abe I et al. Prevalence of fatty liver

in Japanese children and relationship to obesity. An epidemiological ultrasonographic survey. *Dig Dis Sci* 1995; 40(9):2002-2009.

23. Franzese A, Vajro P, Argenziano A, Puzziello A, Iannucci MP, Saviano MC et al. Liver involvement in obese children. Ultrasonography and liver enzyme levels at diagnosis and during follow-up in an Italian population. *Dig Dis Sci* 1997; 42(7):1428-1432.

24. Chan DF, Li AM, Chu WC, Chan MH, Wong EM, Liu EK et al. Hepatic steatosis in obese Chinese children. *Int J Obes Relat Metab Disord* 2004; 28(10):1257-1263.

25. Rosenbloom AL, Joe JR, Young RS, Winter WE. Emerging epidemic of type 2 diabetes in youth. *Diabetes Care* 1999; 22(2):345-354.

26. Dabelea D, Hanson RL, Bennett PH, Roumain J, Knowler WC, Pettitt DJ. Increasing prevalence of Type II diabetes in American Indian children. *Diabetologia* 1998; 41(8):904-910.

27. Sinha R, Fisch G, Teague B, Tamborlane WV, Banyas B, Allen K et al. Prevalence of impaired glucose tolerance among children and adolescents with marked obesity. *New England Journal of Medicine* 2002; 346(11):802-810.

28. Freedman DS, Srinivasan SR, Berensen GS. Risk of cardiovascular complication. In: Burniat W, Cole T, Lissau I, Poskitt EME, editors. *Child and Adolescent Obesity Causes and Consequences, Prevention and Management*. Cambridge: Cambridge University Press, 2002: 221-239.

29. Cook S, Weitzman M, Auinger P, Nguyen M, Dietz WH. Prevalence of a metabolic syndrome phenotype in adolescents: findings from the third National Health and Nutrition Examination Survey, 1988-1994. *Archives of Pediatrics & Adolescent Medicine* 2003; 157(8):821-827.

30. Weiss R, Dziura J, Burgert TS, Tamborlane WV, Taksali SE, Yeckel CW et al. Obesity and the metabolic syndrome in children and adolescents. *N Engl J Med* 2004; 350(23):2362-2374.

31. Dietz WH. Health consequences of obesity in youth: childhood predictors of adult disease. *Pediatrics* 1998; 101(3 Pt 2):518-525.

32. Figueroa-Colon R, Franklin FA, Lee JY, Aldridge R, Alexander L. Prevalence of obesity with increased blood pressure in elementary school-aged children. *South Med J* 1997; 90(8):806-813.

33. Dietz WH. Childhood weight affects adult morbidity and mortality. *Journal of Nutrition* 1998; 128(2 Suppl):411S-414S.

34. French SA, Story M, Perry CL. Self-esteem and obesity in children and adolescents: a literature review. *Obesity Research* 1995; 3(5):479-490.

35. Strauss RS. Childhood obesity and self-esteem. *Pediatrics* 2000; 105(1):e15.

36. Strauss RS, Pollack HA. Social marginalization of overweight children. *Arch Pediatr Adolesc Med* 2003; 157(8):746-752.

37. Latner JD, Stunkard AJ. Getting worse: the stigmatization of obese children. *Obes Res* 2003; 11(3):452-456.

38. Schwimmer JB, Burwinkle TM, Varni JW. Health-related quality of life of severely obese children and adolescents. *JAMA* 2003; 289(14):1813-1819.

39. Parsons TJ, Power C, Logan S, Summerbell CD. Childhood predictors of adult obesity: a systematic review. *Int J Obes Relat Metab Disord* 1999; 23 Suppl 8:S1-S107.

40. Sturm R. The effects of obesity, smoking, and drinking on medical problems and costs. Obesity outranks both smoking and drinking in its deleterious effects on health and health costs. *Health Aff (Millwood)* 2002; 21(2):245-253.

41. Fontaine KR, Redden DT, Wang C, Westfall AO, Allison DB. Years of life lost due to obesity. *JAMA* 2003; 289(2):187-193.

42. Mokdad AH, Marks JS, Stroup DF, Gerberding JL. Actual causes of death in the United States, 2000. *JAMA* 2004; 291(10):1238-1245.

43. Wang G, Dietz WH. Economic burden of obesity in youths aged 6 to 17 years: 1979-1999. *Pediatrics* 2002; 109(5):E81.

44. Tershakovec AM, Watson MH, Wenner WJ, Jr., Marx AL. Insurance reimbursement for the treatment of obesity in children. *Journal of Pediatrics* 1999; 134(5):573-578.

45. Poskitt EM. Defining childhood obesity: the relative body mass index (BMI). European Childhood Obesity group. *Acta Paediatr* 1995; 84(8):961-963.

46. Oken E, Lightdale JR. Updates in pediatric nutrition. *Curr Opin Pediatr* 2000; 12(3):282-290.

47. Lohman T. *Advances in Body Composition Assessment*. 3 ed. Champaign, IL: Human Kinetics Publishers, 1992.

48. Lohman TG. Applicability of body composition techniques and constants for children and youths. *Exerc Sport Sci Rev* 1986; 14:325-357.

49. Goran MI. Measurement issues related to studies of childhood obesity: assessment of body composition, body fat distribution, physical activity, and food intake. *Pediatrics* 1998; 101(3 Pt 2):505-518.

50. Daniels SR, Khoury PR, Morrison JA. The utility of body mass index as a measure of body fatness in children and adolescents: differences by race and gender. *Pediatrics* 1997; 99(6):804-807.

51. Maynard LM, Wisemandle W, Roche AF, Chumlea WC, Guo SS, Siervogel RM. Childhood body composition in relation to body mass index. *Pediatrics* 2001; 107(2):344-350.

52. Ellis D. Growth and renal function after steroid-free tacrolimus-based immunosuppression in children with renal transplants. *Pediatric Nephrology* 2000; 14(7):689-694.

53. Wells JC, Fuller NJ, Dewit O, Fewtrell MS, Elia M, Cole TJ. Four-component model of body composition in children: density and hydration of fat-free mass and comparison with simpler models. *Am J Clin Nutr* 1999; 69(5):904-912.

54. Kuczmarski RJ. Trends in body composition for infants and children in the U.S. *Crit Rev Food Sci Nutr* 1993; 33(4-5):375-387.

55. Dietz WH, Bellizzi MC. Introduction: the use of body mass index to assess obesity in children. *Am J Clin Nutr* 1999; 70(1):123S-125S.

56. Garn SM, Leonard WR, Hawthorne VM. Three limitations of the body mass index. *Am J Clin Nutr* 1986; 44(6):996-997.

57. Kimm SY, Barton BA, Obarzanek E, McMahon RP, Sabry ZI, Waclawiw MA et al. Racial divergence in adiposity during adolescence: The NHLBI Growth and Health Study. *Pediatrics* 2001; 107(3):E34.

58. Cole TJ, Bellizzi MC, Flegal KM, Dietz WH. Establishing a standard definition for child overweight and obesity worldwide: international survey. *BMJ* 2000; 320(7244):1240-1243.

59. Dietz WH, Robinson TN. Use of the body mass index (BMI) as a measure of overweight in children and adolescents. *J Pediatr* 1998; 132(2):191-193.

60. Cole TJ. Children grow and horses race: is the adiposity rebound a critical period for later obesity? *BMC Pediatr* 2004; 4(1):6.

61. Kuczmarski RJ, Ogden CL, Guo SS, Grummer-Strawn LM, Flegal KM, Mei Z et al. 2000 CDC Growth Charts for the United States: methods and development. *Vital Health Stat 11* 2002;(246):1-190.

62. Use and interpretation of anthropometric indicators of nutritional status. WHO Working Group. *Bull World Health Organ* 1986; 64(6):929-941.

63. Ellis KJ. Measuring body fatness in children and young adults: comparison of bioelectric impedance analysis, total body electrical conductivity, and dual-energy X-ray absorptiometry. *Int J Obes Relat Metab Disord* 1996; 20(9):866-873.

64. Reilly JJ, Dorosty AR, Emmett PM. Identification of the obese child: adequacy of the body mass index for clinical practice and epidemiology. *Int J Obes Relat Metab Disord* 2000; 24(12):1623-1627.

65. Reilly JJ, Wilson ML, Summerbell CD, Wilson DC. Obesity: diagnosis, prevention, and treatment; evidence based answers to common questions. *Arch Dis Child* 2002; 86(6):392-394.

66. Himes JH. Agreement among anthropometric indicators identifying the fattest adolescents. *Int J Obes Relat Metab Disord* 1999; 23 Suppl 2:S18-S21.

67. U.S. Preventive Services Task Force. *Guide to clinical preventive services*. 2nd ed. Washington, DC: Office of Disease Prevention and Health Promotion, 1996.

68. Harris RP, Helfand M, Woolf SH, Lohr KN, Mulrow CD, Teutsch SM et al. Current methods of the US Preventive Services Task Force: a review of the process. *Am J Prev Med* 2001; 20(3 Suppl):21-35.

69. Haddock CK, Shadish WR, Klesges RC, Stein RJ. Treatments for childhood and adolescent obesity. *Ann Behav Med* 1994; 16(3):235-244.

70. Epstein LH, Roemmich JN, Raynor HA. Behavioral therapy in the treatment of pediatric obesity. *Pediatr Clin North Am* 2001; 48(4):981-993.

71. Glenny AM, O'Meara S, Melville A, Sheldon TA, Wilson C. The treatment and prevention of obesity: a systematic review of the literature. *Int J Obes Relat Metab Disord* 1997; 21(9):715-737.

72. Summerbell CD, Ashton V, Campbell KJ, Edmunds L, Kelly S, Waters E. Interventions for treating obesity in children. *Cochrane Database of Systematic Reviews* 2003;(3):CD001872.

73. Reilly JJ, McDowell ZC. Physical activity interventions in the prevention and treatment of paediatric obesity: systematic review and critical appraisal. *Proceedings of the Nutrition Society* 2003; 62(3):611-619.

74. McLean N, Griffin S, Toney K, Hardeman W. Family involvement in weight control, weight maintenance and weight-loss interventions: a systematic review of randomised trials. *International Journal of Obesity & Related Metabolic Disorders* 2003; 27(9):987-1005.

75. Ogden CL, Flegal KM, Carroll MD, Johnson CL. Prevalence and trends in overweight among US children and adolescents, 1999-2000. *JAMA* 2002; 288(14):1728-1732.

76. Flegal KM, Ogden CL, Wei R, Kuczmarski RL, Johnson CL. Prevalence of overweight in US children: comparison of US growth charts from the Centers for Disease Control and Prevention with other reference values for body mass index. *American Journal of Clinical Nutrition* 2001; 73(6):1086-1093.

77. Hedley AA, Ogden CL, Johnson CL, Carroll MD, Curtin LR, Flegal KM. Prevalence of overweight and obesity among US children, adolescents, and adults, 1999-2002. *JAMA* 2004; 291(23):2847-2850.

78. Zephier E, Himes JH, Story M. Prevalence of overweight and obesity in American Indian School children and adolescents in the Aberdeen area: a population study. *Int J Obes Relat Metab Disord* 1999; 23 Suppl 2:S28-S30.

79. Flegal KM, Carroll MD, Kuczmarski RJ, Johnson CL. Overweight and obesity in the United States: prevalence and trends, 1960-1994. *Int J Obes Relat Metab Disord* 1998; 22(1):39-47.

80. Strauss RS, Pollack HA. Epidemic increase in childhood overweight, 1986-1998. *JAMA* 2001; 286(22):2845-2848.

81. Ellis KJ. Human body composition: in vivo methods. *Physiol Rev* 2000; 80(2):649-680.

82. Dai S, Labarthe DR, Grunbaum JA, Harrist RB, Mueller WH. Longitudinal analysis of changes in indices of obesity from age 8 years to age 18 years. Project HeartBeat! *American Journal of Epidemiology* 2002; 156(8):720-729.

83. Lauer RM, Lee J, Clarke WR. Predicting adult cholesterol levels from measurements in childhood and adolescence: the Muscatine Study. *Bulletin of the New York Academy of Medicine* 1989; 65(10):1127-1142.

84. Guo SS, Roche AF, Chumlea WC, Gardner JD, Siervogel RM. The predictive value of childhood body mass index values for overweight at age 35 y. *American Journal of Clinical Nutrition* 1994; 59(4):810-819.

85. Clarke WR, Lauer RM. Does childhood obesity track into adulthood? *Crit Rev Food Sci Nutr* 1993; 33(4-5):423-430.

86. Hulman S, Kushner H, Katz S, Falkner B. Can cardiovascular risk be predicted by newborn, childhood, and adolescent body size? An examination of longitudinal data in urban African Americans. *Journal of Pediatrics* 1998; 132(1):90-97.

87. Wattigney WA, Webber LS, Srinivasan SR, Berenson GS. The emergence of clinically abnormal levels of cardiovascular disease risk factor variables among young adults: the Bogalusa Heart Study. *Preventive Medicine* 1995; 24(6):617-626.

88. Gortmaker SL, Must A, Perrin JM, Sobol AM, Dietz WH. Social and economic consequences of overweight in adolescence and young adulthood. *New England Journal of Medicine* 1993; 329(14):1008-1012.

89. Freedman DS, Khan LK, Dietz WH, Srinivasan SR, Berenson GS. Relationship of childhood obesity to coronary heart disease risk factors in adulthood: the Bogalusa Heart Study. *Pediatrics* 2001; 108(3):712-718.

90. Guo SS, Wu W, Chumlea WC, Roche AF. Predicting overweight and obesity in adulthood from body mass index values in childhood and adolescence. *American Journal of Clinical Nutrition* 2002; 76(3):653-658.

91. Freedman DS, Khan LK, Serdula MK, Dietz WH, Srinivasan SR, Berenson GS. Inter-relationships among childhood BMI, childhood height, and adult obesity: the Bogalusa Heart Study. *International Journal of Obesity & Related Metabolic Disorders: Journal of the International Association for the Study of Obesity* 2004; 28(1):10-16.

92. Daniels S. Pharmacological treatment of obesity in paediatric patients. *Paediatric Drugs* 2001; 3(6):405-410.

93. Whitlock EP, Orleans CT, Pender N, Allan J. Evaluating primary care behavioral counseling interventions: an evidence-based approach. *Am J Prev Med* 2002; 22(4):267-284.

94. Berkowitz RI, Wadden TA, Tershakovec AM, Cronquist JL. Behavior therapy and sibutramine for the treatment of adolescent obesity: a randomized controlled trial. *JAMA* 2003; 289(14):1805-1812.

95. Duffy G, Spence SH. The effectiveness of cognitive self-management as an adjunct to a behavioural intervention for childhood obesity: a research note. *J Child Psychol Psychiatry* 1993; 34(6):1043-1050.

96. Ebbeling CB, Leidig MM, Sinclair KB, Hangen JP, Ludwig DS. A reduced-glycemic load diet in the treatment of adolescent obesity. *Archives of Pediatrics & Adolescent Medicine* 2003; 157(8):773-779.

97. Epstein LH, Wing RR, Koeske R, Valoski A. A comparison of lifestyle exercise, aerobic exercise, and calisthenics on weight loss in obese children. *Behavior Therapy* 1985; 16(4):345-356.

98. Epstein LH, Wing RR, Penner BC, Kress MJ. Effect of diet and controlled exercise on weight loss in obese children. *J Pediatr* 1985; 107(3):358-361.

99. Epstein LH, Wing R, Woodall K, Penner BC, Kress MJ, Koeske R. Effects of family-based behavioral treatment on obese 5-to-8-year-old children. *Behavior Therapy* 1985; 16(2):205-212.

100. Epstein LH, McKenzie SJ, Valoski A, Klein KR, Wing RR. Effects of mastery criteria and contingent reinforcement for family-based child weight control. *Addictive Behaviors* 1994; 19(2):135-145.

101. Epstein LH, Valoski AM, Vara LS, McCurley J, Wisniewski L, Kalarchian MA et al. Effects of decreasing sedentary behavior and increasing activity on weight change in obese children 3814. *Health Psychology* 1995; 14(2):109-115.

102. Epstein LH, Paluch RA, Gordy CC, Dorn J. Decreasing sedentary behaviors in treating pediatric obesity. *Arch Pediatr Adolesc Med* 2000; 154(3):220-226.

103. Epstein LH, Paluch RA, Gordy CC, Saelens BE, Ernst MM. Problem solving in the treatment of childhood obesity. *Journal of Consulting & Clinical Psychology* 2000; 68(4):717-721.

104. Epstein LH, Paluch RA, Raynor HA. Sex differences in obese children and siblings in family-based obesity treatment. *Obesity Research* 2001; 9(12):746-753.

105. Flodmark CE, Ohlsson T, Ryden O, Sveger T. Prevention of progression to severe obesity in a group of obese schoolchildren treated with family therapy. *Pediatrics* 1993; 91(5):880-884.

106. Golan M, Fainaru M, Weizman A. Role of behaviour modification in the treatment of childhood obesity with the parents as the exclusive agents of change. *Int J Obes Relat Metab Disord* 1998; 22(12):1217-1224.

107. Graves T, Meyers AW, Clark L. An evaluation of parental problem-solving training in the behavioral treatment of childhood obesity. *J Consult Clin Psychol* 1988; 56(2):246-250.

108. Israel AC, Stolmaker L, Andrian CA. The effects of training parents in general child management skills on a behavioral weight loss program for children. *Behavior Therapy* 1985; 16:169-180.

109. Israel AC, Guile CA, Baker JE, Silverman WK. An evaluation of enhanced self-regulation training in the treatment of childhood obesity. *Journal of Pediatric Psychology* 1994; 19(6):737-749.

110. Gutin B, Barbeau P, Owens S, Lemmon CR, Bauman M, Allison J et al. Effects of exercise intensity on cardiovascular fitness, total body composition, and visceral adiposity of obese adolescents. *American Journal of Clinical Nutrition* 2002; 75(5):818-826.

111. Kang HS, Gutin B, Barbeau P, Owens S, Lemmon CR, Allison J et al. Physical training improves insulin resistance syndrome markers in obese adolescents. *Medicine & Science in Sports & Exercise* 2002; 34(12):1920-1927.

112. Mellin LM, Slinkard LA, Irwin CE, Jr. Adolescent obesity intervention: validation of the SHAPEDOWN program 3987. *Journal of the American Dietetic Association* 1987; 87(3):333-338.

113. Saelens BE, Sallis JF, Wilfley DE, Patrick K, Cella JA, Buchta R. Behavioral weight control for overweight adolescents initiated in primary care. *Obesity Research* 2002; 10(1):22-32.

114. Senediak C, Spence SH. Rapid versus gradual scheduling of therapeutic contact in a family based behavioural weight control programme for children. *Behavioural Psychotherapy* 1985; 13(4):265-287.

115. Wadden TA, Stunkard AJ, Rich L, Rubin CJ, Sweidel G, McKinney S. Obesity in black

adolescent girls: a controlled clinical trial of treatment by diet, behavior modification, and parental support 3928. *Pediatrics* 1990; 85(3):345-352.

116. White MA. *Mediators of weight loss in an Internet-based intervention for African-American adolescent girls*. Louisiana State University, 2003.

117. Epstein LH, Valoski A, Wing RR, McCurley J. Ten-year outcomes of behavioral family-based treatment for childhood obesity. *Health Psychol* 1994; 13(5):373-383.

118. Robinson TN. Behavioural treatment of childhood and adolescent obesity. *International Journal of Obesity & Related Metabolic Disorders* 1999; 23:Suppl-7.

119. Jeffery RW, Drewnowski A, Epstein LH, Stunkard AJ, Wilson GT, Wing RR et al. Long-term maintenance of weight loss: current status. *Health Psychol* 2000; 19(1 Suppl):5-16.

120. Yanovski JA. Intensive therapies for pediatric obesity. *Pediatric Clinics of North America* 2001; 48(4):1041-1053.

121. McTigue KM., Harris R, Hemphill MB, Bunton AJ, Lux LJ, Sutton S et al. *Screening and interventions for overweight and obesity in adults*. Systemic Evidence Review No. 21 (prepared by the Research Triangle Institute, University of North Carolina, Evidence-based Practice Center under Contract No. 290-97-0011). Rockville, MD: Agency for Healthcare Research and Quality, 2003.

122. Epstein LH, Paluch RA, Saelens BE, Ernst MM, Wilfley DE. Changes in eating disorder symptoms with pediatric obesity treatment. *Journal of Pediatrics* 2001; 139(1):58-65.

123. Inge TH, Garcia V, Daniels S, Langford L, Kirk S, Roehrig H et al. A multidisciplinary approach to the adolescent bariatric surgical patient. *Journal of Pediatric Surgery* 2004; 39(3):442-447; discussion 446-447.

124. Stanford A, Glascock JM, Eid GM, Kane T, Ford HR, Ikramuddin S et al. Laparoscopic Roux-en-Y gastric bypass in morbidly obese adolescents. *Journal of Pediatric Surgery* 2003; 38(3):430-433.

125. Strauss RS, Bradley LJ, Brolin RE. Gastric bypass surgery in adolescents with morbid obesity. *J Pediatr* 2001; 138(4):499-504.

126. Charney E. Childhood obesity: the measurable and the meaningful. *J Pediatr* 1998; 132(2):193-195.

127. Stettler N. Comment: the global epidemic of childhood obesity: is there a role for the paediatrician? *Obes Rev* 2004; 5(Suppl 1):1-3.

128. Gruen RL, Pearson SD, Brennan TA. Physician-citizens--public roles and professional obligations. *JAMA* 2004; 291(1):94-98.

129. Flegal KM, Graubard BI, Williamson DF, Gail MH. Excess deaths associated with underweight, overweight, and obesity. *JAMA* 2005; 293(15):1861-1867.

130. Pi-Sunyer FX. Medical hazards of obesity. *Ann Intern Med* 1993; 119(7 Pt 2):655-660.

131. Stunkard A. Diet, exercise and behavior therapy: A cautionary tale. *Obes Res* 1996; 4:293-294.

132. Whitaker RC. Obesity prevention in pediatric primary care: four behaviors to target. *Arch Pediatr Adolesc Med* 2003; 157(8):725-727.

133. Campbell K, Waters E, O'Meara S, Kelly S, Summerbell C. Interventions for preventing obesity in children. *Cochrane Database of Systematic Reviews* 2003;(1).

134. Krempf M, Louvet JP, Allanic H, Miloradovich T, Joubert JM, Attali JR. Weight reduction and long-term maintenance after 18 months treatment with orlistat for obesity. *International Journal of Obesity & Related Metabolic Disorders* 2003; 27(5):591-597.

135. Sopher AB, Thornton JC, Wang J, Pierson RN, Jr., Heymsfield SB, Horlick M. Measurement of percentage of body fat in 411 children and adolescents: a comparison of dual-energy X-ray absorptiometry with a four-compartment model. *Pediatrics* 2004; 113(5):1285-1290.

136. Power C, Lake JK, Cole TJ. Measurement and long-term health risks of child and adolescent fatness. *Int J Obes Relat Metab Disord* 1997; 21(7):507-526.

137. Robinson TN. Defining obesity in children and adolescents: clinical approaches. *Critical Reviews in Food Science & Nutrition* 1993; 33(4-5):313-320.

Figure 1. Overweight Trends in Children and Adolescents[75]

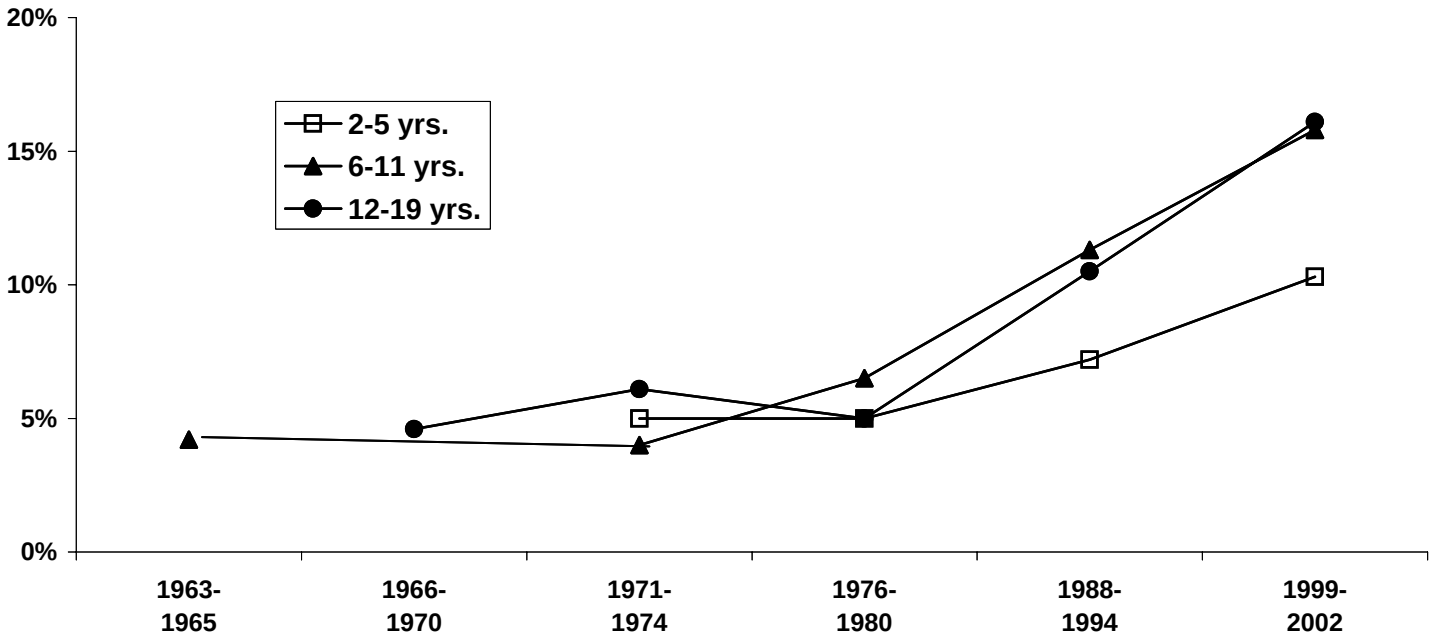
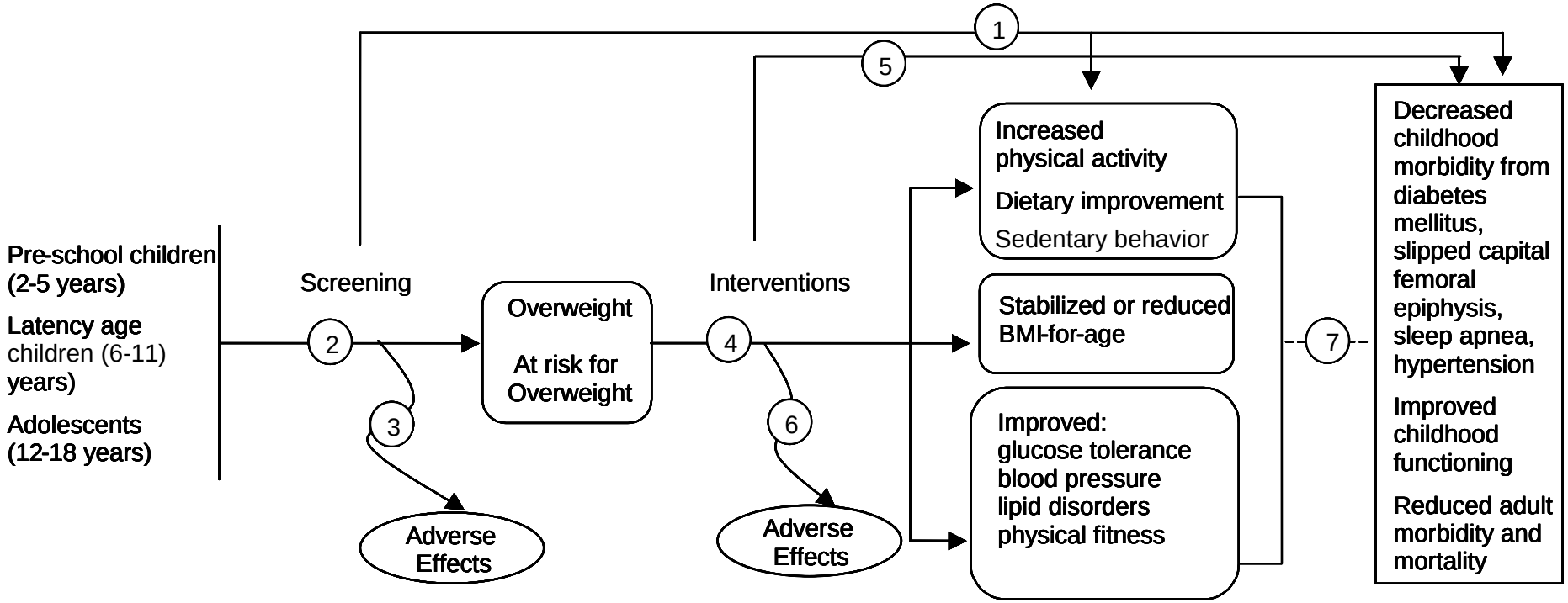


Figure 2. Screening and interventions for overweight in childhood: Analytic framework and Key Questions



Key Questions

Arrow 1: Is there direct evidence that screening (and intervention) for overweight in childhood improves age-appropriate behavioral or physiologic measures, or health outcomes?

Arrow 2: a. What are appropriate standards for overweight in childhood and what is prevalence of overweight based on these?
b. What clinical screening tests for overweight in childhood are reliable and valid in predicting obesity in adulthood?
c. What clinical screening tests for overweight in childhood are reliable and valid in predicting poor health outcomes in adulthood?

Arrow 3: What are the adverse effects of screening, including labeling? Is screening acceptable to patients?

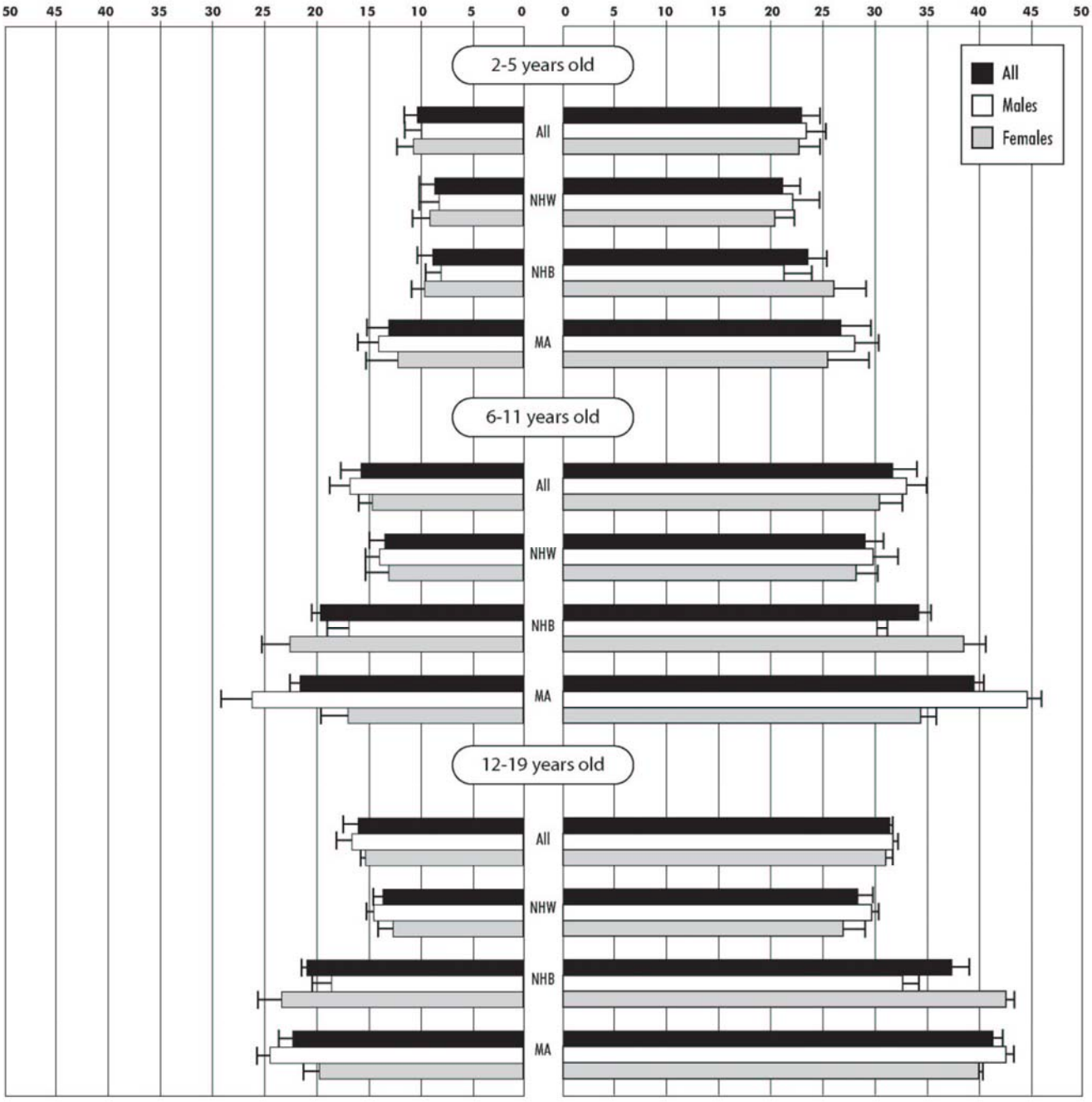
Arrow 4: Do weight control interventions (behavioral counseling, pharmacotherapy, surgery) lead to improved intermediate outcomes, including behavioral, physiologic or weight-related measures?
a. What are common behavioral and health system elements of efficacious interventions?
b. Are there differences in efficacy between patient subgroups?

Arrow 5: Do weight control interventions lead to improved health outcomes, including decreased morbidity, and/or improved functioning (school attendance, self-esteem and other psychosocial indicators)?

Arrow 6: What are the adverse effects of interventions? Are interventions acceptable to patients?

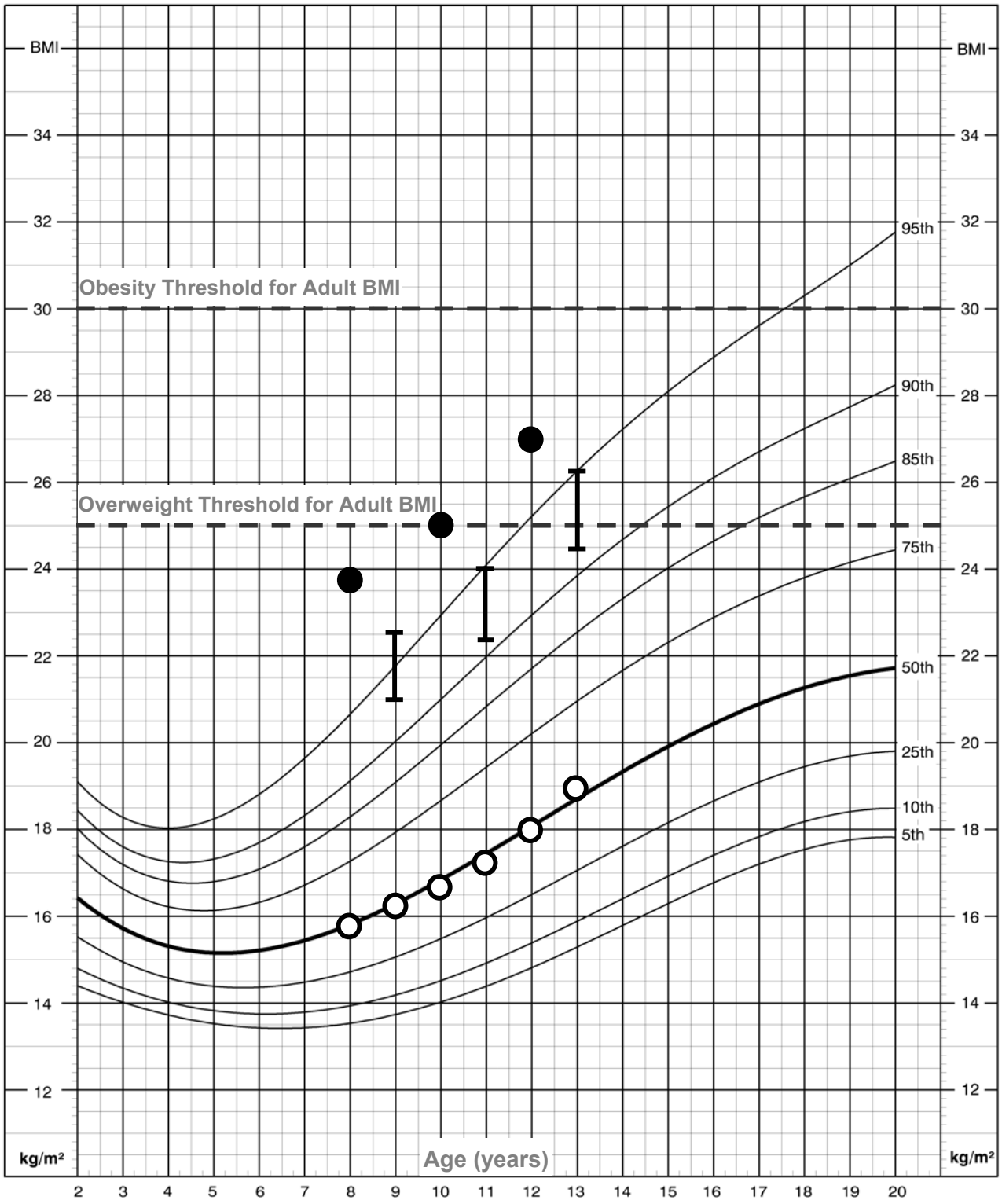
Arrow 7: Are improvements in intermediate outcomes associated with improved health outcomes? (Only evaluated if there is no direct evidence for KQ1 or KQ5 and if there is sufficient evidence for KQ4)

Figure 3. Prevalence of Overweight 1999-2002 % with BMI equal to or greater than 95% with standard error bars[76] Prevalence of Overweight or at Risk for Overweight 1999-2002 % with BMI equal to or greater than 85% with standard error bars[76]



NHW = Non-Hispanic white; NHB = Non-Hispanic black; MA = Mexican American

Figure 4. Effects of behavioral weight loss treatment on BMI for children ages 8 to 13: Modeled results using CDC Growth Charts: United States. Body mass index-for-age percentiles: Girls, 2 to 20 years.



Published May 30, 2000.
SOURCE: Developed by the National Center for Health Statistics in collaboration with the National Center for Chronic Disease Prevention and Health Promotion (2000).

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Ages 8-12 yrs Modeled Results:

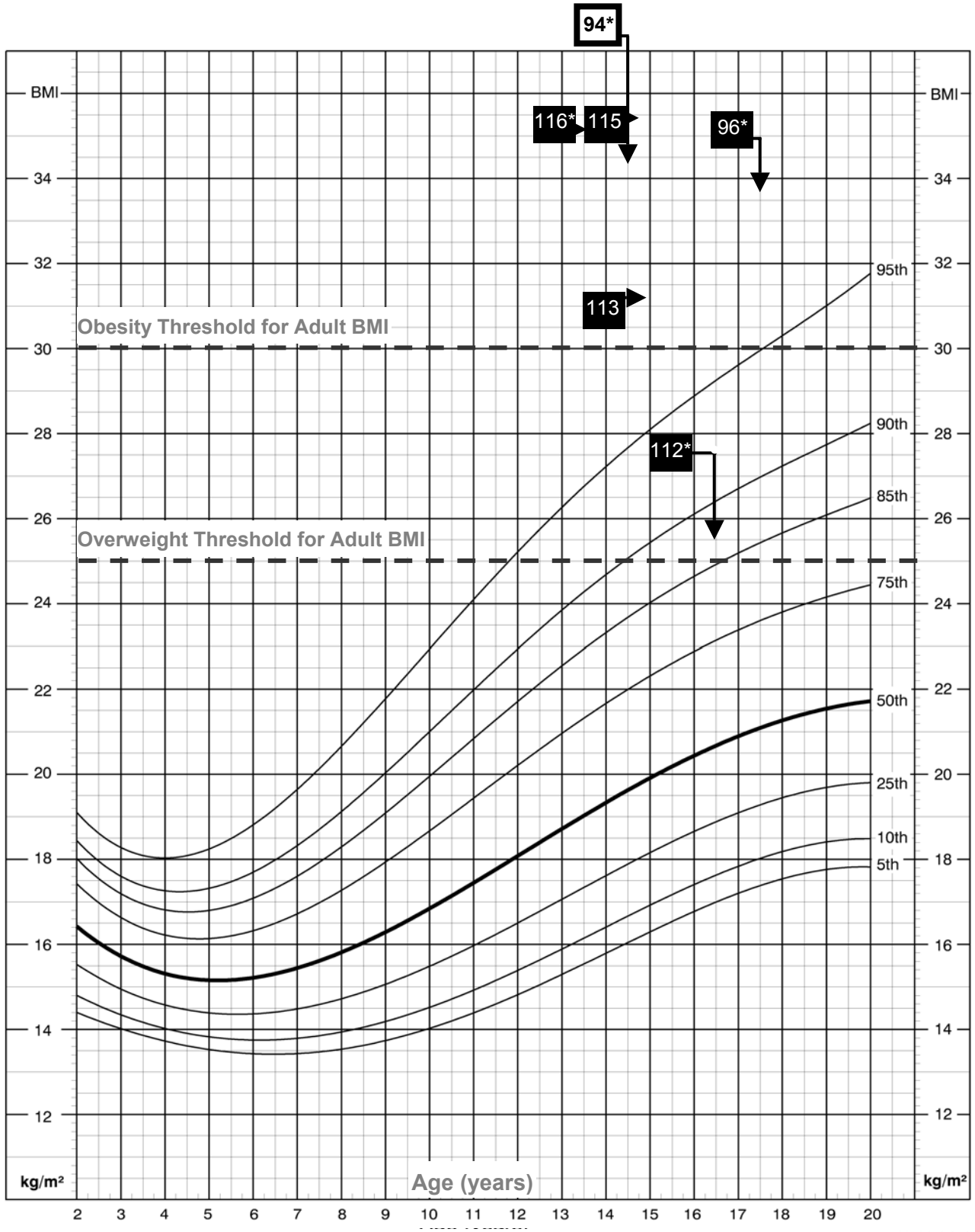
10% reduction

20% reduction

BMI if 50% overweight

BMI if ideal weight

Figure 5. Effects of behavioral weight loss treatment on BMI for adolescents using CDC Growth Charts: United States. Body mass index-for-age percentiles: Girls, 2 to 20 years.



Published May 30, 2000.
SOURCE: Developed by the National Center for Health Statistics in collaboration with the National Center for Chronic Disease Prevention and Health Promotion (2000).

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Adolescents Trial Results:	Mean BMI entry For BCI study	Mean change in BMI	Mean BMI entry for BCI + drug study

Study numbers correspond to the Bibliography *** Results statistically significant (p<0.05)**

Table 1. Overweight and Obesity-Associated Health Conditions in Children and Adolescents

Health Condition	Population Source (Number)	Age/Race-Ethnicity/Gender	Level of Overweight	Prevalence %	Reference Cited in Source Bibliography
Diabetes Mellitus (DM)					
Type II	Community N = 2066	Pima NA* -ages 15-19 yrs girls boys -ages 10-14 yrs girls boys		 5.31 3.78 2.88 1.4	Dabelea et al 1998[26]
	Community N = 142	Navajo NA -ages 12-19		1.41	Lobstein et al 2004[7]
	NHANES III	NHW, NHB, MA adolescents -aged 12-19		.04 (includes Type I DM as well)	
Type II	Obesity clinic N = 55	Multi-ethnic boys and girls ages 4-10 years	BMI > for age & sex (95%ile)	0	Sinha et al 2002[27]
	Obesity clinic N = 112	Multi-ethnic boys and girls ages 11-18 yrs	BMI >95%ile BMI <85%ile BMI 85-97%ile	4 (all were NHB or H) 0 0	
Asthma					
Parental report of doctor diagnosed asthma in child	NHANES III Subset of children with family history of parental asthma N = 625	Ages 10 –16 years, multiethnic girls and boys	BMI ≥ 85 BMI < 85	31 14.5	Rodriguez et al 2002[18]
Sleep apnea (SA)					
Sleep-associated abnormal breathing tests	Obese children referred for history of sleep-disordered breathing N = 32	Ages 2.7 – 13.8 years, multi-ethnic boys and girls	Mean IBW = 196 ± 45%	40-90	Silvestri et al 1993[20]

Table 1. Overweight and Obesity-Associated Health Conditions in Children and Adolescents

Health Condition	Population Source (Number)	Age/Race-Ethnicity/Gender	Level of Overweight	Prevalence %	Reference Cited in Source Bibliography
1 or more abnormal polysomnography test	Obesity clinic N = 222	Children and adolescents ages 3 – 20 years	"Severe obesity" - >150% IBW, mean = 208% IBW	6.8 (calc)	Mallory et al 1989[21]
Severely abnormal tests (severe SA)				1 (calc)	
1 or more abnormal polysomnography test	Obesity clinic patients with history of sleep-disordered breathing N = 41			32	
Severely abnormal test (severe SA)				5	
Nonalcoholic fatty liver disease					
Steatosis by US	General pediatric N = 810	Japanese school boys and girls ages 4 – 12 years	Population sample	3	Tominaga et al 1995[22]
Steatosis by US	Obesity clinic N = 72	Italian boys and girls ages 4.5-15.9 years	Obese > 120% IBW	53	Franzese et al 1997[23]
Steatosis and elevated transaminases (presumptive NASH)				Of these, 32% had elevated transaminases (calc)	
Steatosis by US	Obesity clinic N = 84	Chinese children, mean age 12	BMI > 95th percentile for age and sex	77	Chan et al 2004[24]
Steatosis and elevated transaminases (presumptive NASH)				24	
Slipped capital femoral epiphysis				3.4 per 100,000 children (50-70% are "obese")	Kelsey 1973[19]

*The highest risk population in the world (Dabelea D., Pettitt D, Jones KL, Arslanian S. Type 2 diabetes mellitus in minority children and adolescents. *Endocrinology and Metabolism Clinics* of North America 1999; 28(4): 709-729, viii.
Calc: calculated number

Childhood morbidities discussed in reviews without reporting prevalence: Binge-eating disorders, low self-esteem.

Table 2. Overweight and Obesity-Associated Risk Factors in Children and Adolescents

Risk Factor	Population Source	Age/Race-Ethnicity/Gender	Level of Overweight (percentile of BMI for age and sex if given)	Prevalence %	Reference Cited in Source Bibliography
Impaired glucose tolerance					
Impaired glucose tolerance	Obesity clinic N = 55	Multi-ethnic boys and girls ages 4-10 years	> 95	25	Sinha et al 2002[27]
	Obesity clinic N = 112	Multi-ethnic boys and girls ages 11-18 yrs	> 95	21	Sinha et al 2002[27]
Hyperinsulinemia					
(Insulin levels above the 95%ile)	Bogalusa Heart Study	Black and white boys and girls aged 5-10 years	< 95 95-97 > 97	≤ 4 10 27	Freedman et al 2002[28]
Metabolic syndrome					
Hypertension, hypertriglyceridemia, low HDL cholesterol, hyperinsulinemia)	NHANES	Adolescent boys and girls aged 12-19 years	< 85 84-95 ≥ 95	0.1 6.8 28.7	Cook et al 2003[29]
With at least three of: SBP or DBP > 95%ile, triglycerides > 95%ile, 2 hr. GTT > 140 mg/dl, BMI z-score ≥ 2.0 (97%ile), HDL cholesterol < 5%ile	Obese sample N = 439	41% white, 31% black, 27% Hispanic. Ages 4-20 years	-z-score 2-2.5 -z-score>2.5	38.7 49.7 overall (39 in blacks)	Weiss et al 2004[30]
Hypertension					
Hypertension	Population based	Multi-ethnic boys and girls, ages 5-11	Obese	Up to 30	Figueroa-Colon et al 1997[32]
Hypertension	Muscatine Heart Study (>6600)	5-18 years	Community distribution	1 (60% of these had relative wt >120%)	Dietz 1998[31]
Increased SBP measures >95%ile	Bogalusa Heart Study	Black and white boys and girls aged 5-10 years	<95 95-97 >97	2-7 12 22	Freedman et al 2002[28]
Increased DBP >95%ile	Bogalusa Heart Study	Black and white boys and girls aged 5-10 years	<95 95-97 >97	2-7 9 14	Freedman et al 2002[28]

Table 2. Overweight and Obesity-Associated Risk Factors in Children and Adolescents

Risk Factor	Population Source	Age/Race-Ethnicity/Gender	Level of Overweight	Prevalence %	Reference Cited in Source Bibliography
Dyslipidemia					
LDL cholesterol > 130 mg/dl	Bogalusa Heart Study N = 3599	Black and white boys and girls ages 5-10 years	< 85 85-94 95-97 > 97	8-10 across all percentiles 18 12 23	Freedman et al 2002[28]
HDL cholesterol < 35 mg/dl	Bogalusa Heart Study N = 3599	Black and white boys and girls ages 5-10 years	< 85 85-94 95-97 > 97	5-8 & non-linear 8 7 18	Freedman et al 2002[28]
TG levels > 130 mg/dl	Bogalusa Heart Study N = 3599	Black and white boys and girls ages 5-10 years	< 85 85-94 95-97 > 97	2-6 10 10 21	Freedman et al 2002[28]

Risk factors discussed in reviews without reporting prevalence: Menstrual disorders, polycystic ovarian syndrome, early maturation (girls), late maturation (boys)

Table 3. Effect of Age and Race on the Correlation of Childhood with Young Adult Body Mass Index (BMI)

Effect of Age on the Correlation of Childhood with Young Adult BMI

Reference	Population	Childhood Age, Years	Males	Females
Guo et al 1994[84]	100% white (n=555)	3	.18	.22
Lauer et al1989[83]	100% white (n=109 observations)	7 to 8	.57	.45
Lauer et al 1989[83]	100% white (n=603 observations)	9 to 10	.63	.61
Clarke and Lauer 1993[85]	100% white (n=1,286 observations)	9 to 10	.61	.59
Lauer et al 1989[83]	100% white (n=1,018 observations)	11 to 12	.67	.65
Guo et al 1994[84]	100% white (n=555)	13	.5	.65
Lauer et al 1989[83]	100% white (n=1,041 observations)	13 to 14	.64	.68
Clarke and Lauer 1993[85]	100% white (n=1,104 observations)	13 to 14	.7	.7
Lauer et al 1989[83]	100% white (n=615 observations)	17 to 18	.74	.73
Clarke and Lauer 1993[85]	100% white (n=631 observations)	17 to 18	.81	.72

Effect of Race on the Correlation of Childhood with Young Adult BMI

Reference	Population	Childhood Age, Years	Males	Females
Hulman et al 1998[86]	100% black (n=137)	13	.37	
Wattigney et al 1995[87]	100% black (n=147)	13 to 17	.69	.72
Wattigney et al 1995[87]	100% white (n=327)	13 to 17	.63	.48
Freedman et al 2004[91]	67% white (n=2,212)	14 to 17	.76	.73

Table 4. Probability of Adult Obesity (Body Mass Index [BMI] ≥30) Based on Childhood BMI Percentile Measures at Various Ages

Study Identification	Overweight Measure in Childhood, BMI Percentile	Child's Age When Measured	Adult's Age When Measured	Probability of Adult Overweight (Male & Female Combined)	Probability of Adult Overweight (Males)	Probability of Adult Overweight (Females)
Gortmaker et al 1993[88] (n=10,039) 80% white, 14% black, 6% Hispanic 51% female	> 95	16-24	23-31	--	.77*	.66*
Freedman et al 2001[89] (n=2617) 67% white, 32% black, 57% female	< 50	5-17	18-37	.07	--	--
Freedman et al 2001[89] (as above)	85-94	5-17	18-37	.51	--	--
Freedman et al 2001[89] (as above)	≥ 95	5-17	18-37	.77	--	--
Guo et al 2002[90] (n=347) 100% white, 52% female	≥ 75	3	35	--	.1	.14
	≥ 85			--	.1	.17
	≥95				.2	.24
Guo et al 2002[90] (as above)	≥ 75	8	35	--.	.1	.16
	≥ 85				.1	.23
	≥ 95				.2	.46
Guo et al 2002[90] (as above)	≥ 75	13	35	--.	.2	.16
	≥ 85				.2	.27
	≥ 95				.5	.64
Guo et al 2002[90] (as above)	≥ 75	18	35	--	.2	.15
	≥ 85				.3	.26
	≥ 95				.8	.68

* In this study adult overweight was defined as >95%ile on NHANES.

Table 5. Randomized Controlled Trials Addressing Overweight in Children and Adolescents

Study Reference	N Randomized Country	Age % Male % Non-White	Baseline Measure of Overweight	Intervention characteristics* Components Comprehensive? Parent Participation? Group vs.	Time Period # of Sessions Session Length Total Contact Time (min)
Berkowitz et al 2003[94]	82 adolescents USA	13-17 33% 45%	BMI 37.8 kg/m^2 (3.8); BMI z-score: 2.4 (0.2)	BM,D,E yes yes G	6 mo (phase I) 19 (phase I) NR NR
Duffy and Spence 1993[95]	29 children Australia	7-13 21% NR, Australian	48.4% overweight	BM,D,E yes yes NR	8 wk 8 90 in 720 min
Ebbeling et al 2003[96]	16 adolescents USA	13-21 31% 19%	BMI 34.9 kg/m^2 (reduced glycemic group); 37.1 kg/m^2 (reduced fat diet group)	BM,D No No NR	12 mo 14 NR NR
Epstein et al 1985[97]	41 families USA	8-12 40% NR	48% overweight	BM,D,E yes yes NR	12 mo 18 NR NR
Epstein et al 1985[98]	23 children USA	8-12 0% NR	48 % overweight	BM,D,E yes yes NR	12 mo NR NR NR
Epstein et al 1985[99]	24 children USA	5-8 0% NR	39-42% overweight	BM,D,E yes yes G, unclear if I	12 mo unclear, approx 26 NR
Epstein et al 1994[100]	44 families USA	8-12 26% NR	59.6% over the 50th%ile for BMI	BM,D,E yes yes unclear	1 yr 32 NR NR
Epstein et al 1995[101]	61 families USA	8-12 27% 4%	51.8% overweight	BM,D,E yes yes I + G	6 mo 18 NR NR
Epstein et al 2000[102]	90 families USA	8-12 32% NR	62% overweight	BM,D,E yes yes I + G	6 mo 20 45-60 min 900-1200 min

Intervention characteristics: BM=behavior modification; D = special diet; E = exercise program; G = group; I = individual;
Other outcomes: B = behavioral ; P = physiological; H = childhood health outcomes; A = adverse effects

Table 5. Randomized Controlled Trials Addressing Overweight in Children and Adolescents

Study Reference	Group	Units of Measure	Study Duration	Outcome at Latest Follow-up Time	P Value for Comparisons between Groups**	Other Outcomes	USPSTF Quality Grade
Berkowitz et al 2003[94]	Sibutramine	change in BMI (% change from entry BMI)	6 months	-8.5%	p = 0.001	P, A	Good
	Placebo			-4.0%			
Duffy and Spence 1993[95]	BT + cognitive self-	% overweight change	6 months	-8.9%	n.s.	B	Fair-to-Poor
	BT + relaxation placebo			-9.2%			
Ebbeling et al 2003[96]	Reduced glycemic load	absolute change in BMI	12 months	-1.2 kg/m2	p < 0.05	B, P	Fair
	Reduced fat diet			0.6 kg/m^2			
Epstein et al 1985[97]	Lifestyle PA	% overweight change	24 months	-18.0%	<0.05, lifestyle PA vs. aerobic PA; <0.05, lifestyle PA vs. calisthenics	B, P	Fair
	Aerobic PA			-6.8%			
	Low-intensity calisthenics PA			-7.2%			
Epstein et al 1985[98]	Diet + PA	% overweight change	12 months	-25.4%	n.s.	B, P	Fair
	Diet alone			-18.7%			
Epstein et al 1985[99]	BT	% overweight change	12 months	-26.3%	< 0.05	B	Fair
	Education only			-11.2%			
Epstein et al 1994[100]	Mastery criteria & contingent reinforcement	% overweight change	24 months	-15.4%	n.s.	B	Fair
	Comparison group			-10.6%			
Epstein et al 1995[101]	Combined	% overweight change	12 months	-8.7%	p < 0.05, combined vs. increased PA	B, P	Fair
	Decreased SB			-10.3%			
	Increased PA			-18.7%			
Epstein et al 2000[102]	Decreased SB high dose	% overweight change	24 months	-14.3%	n.s.	B, P	Fair
	Decrease SB low dose			-11.6%			
	PA high dose			-13.2%			
	PA low dose			-12.4%			

Intervention characteristics: BM=behavior modification; D = special diet; E = exercise program; G = group; I = individual;
Other outcomes: B = behavioral ; P = physiological; H = childhood health outcomes; A = adverse effects

Table 5. Randomized Controlled Trials Addressing Overweight in Children and Adolescents

Study Reference	N Randomized Country	Age % Male % Non-White	Baseline Measure of Overweight	Intervention characteristics* Components Comprehensive? Parent Participation? Group vs.	Time Period # of Sessions Session Length Total Contact Time (min)
Epstein et al 2000[103]/ Epstein et al 2001[104]	67 children USA	NR, mean (SD) 10.3 (1.1) yrs 48% 4%	BMI 27.4 kg/m^2 (3.2)	BM,D,E yes yes I + G	6 mo 18 45-60 min 810-1080 min
Epstein et al 2001[104]	67 families USA	8-12 52% NR	60.2% overweight (compared to the 50%ile BMI for age and sex); BMI 27.4 kg/m^2 (3.6 kg/m^2)	BM, D,E yes yes I + G	6 mo 20 30 min 600 min
Flodmark et al 1993[105]	44 children (plus 50 matched controls) Sweden	10-11 48% NR (Swedish)	24.7 kg/m^2 (family therapy group); 25.5 kg/m^2 (conventional treatment group); 25.1 kg/m^2 (control group)	D,E no yes I	14-18 mo 5 + 6 family therapy sessions NR NR
Golan et al 1998[106]	60 children Israel	6-11 38% NR (Israeli)	39.1%overweight (conventional group); 39.6% (parents agents of change group)	BM,D,E yes no* G+I	1 yr 30 60 min 1800 min
Graves et al 1988[107]	40 children USA	6-12 NR NR	52%-56% overweight	BM,D,E yes yes G	8 wk 8 60 min 480 min

Intervention characteristics: BM=behavior modification; D = special diet; E = exercise program; G = group; I = individual;
Other outcomes: B = behavioral ; P = physiological; H = childhood health outcomes; A = adverse effects

Table 5. Randomized Controlled Trials Addressing Overweight in Children and Adolescents

Study Reference	Group	Units of Measure	Study Duration	Outcome at Latest Follow-up Time	P Value for Comparisons between Groups**	Other Outcomes	USPSTF Quality Grade
Epstein et al 2000[103]/ Epstein et al 2001[104]	PS to parent and child	change in BMI z-score	24 months	-0.5	p < 0.05, PS to parent and child vs. no PS; p < 0.05, PS to parent and child vs. PS to child only	H, A	Fair
	PS to child only			-0.9			
	No PS			-1.1			
Epstein et al 2001[104]	Increased PA	change in absolute BMI (statistical comparisons done on percent overweight change)	12 months	girls: -0.27 kg/m^2; boys: -0.65 kg/m^2	p < 0.01, interaction of group by sex; p < 0.001, boys in combined group vs. girls in combined group p < 0.05, boys in combined group vs. girls in increased PA group	none	Fair
	Combined increased PA + decreased SB			girls: 1.0 kg/m^2; boys: -1.76 kg/m^2			
Flodmark et al 1993[105]	Family therapy	change in BMI (kg/m2)	26 - 30 months	1.1 kg/m^2	p < 0.05, family therapy vs. untreated controls	P	Fair
	Conventional treatment			1.6 kg/m^2			
	Matched controls - untreated			2.8 kg/m^2			
Golan et al 1998[106]	Conventional: children responsible for own wt loss	% overweight change	12 months	-8.1%	p < 0.05	none	Fair
	Parents exclusive agents of			-14.7%			
Graves et al 1988[107]	BT + parent PS	% overweight change	6 months	-24.5%	p < 0.05, PS vs BT only; p < 0.05, PS vs. instruction only	B	Fair
	BT only			-10.2%			
	Instruction only			-9.5%			

Intervention characteristics: BM=behavior modification; D = special diet; E = exercise program; G = group; I = individual;
Other outcomes: B = behavioral ; P = physiological; H = childhood health outcomes; A = adverse effects

Table 5. Randomized Controlled Trials Addressing Overweight in Children and Adolescents

Study Reference	N Randomized Country	Age % Male % Non-White	Baseline Measure of Overweight	Intervention characteristics* Components Comprehensive? Parent Participation? Group vs.	Time Period # of Sessions Session Length Total Contact Time (min)
Israel 1985[108]	33 children USA	8-12 30% NR	45.88% overweight (parent training group); 53.13% (BT only); 56.02% (controls)	BM,D,E yes yes G	12 mo 17 same +2-60 min sessions >930 min
Israel et al 1994[109]	36 families USA	8-13 NR NR	48.1% overweight (enhanced child involvement group); 46.0% (standard treatment group)	BM,D,E yes yes G	26 wk 17 90 min 1530 min
Kang et al 2002[111] **/ Gutin et al 2002**[110]	80 adolescents USA	13-16 33% 69%	40.7% body fat (white boys); 45.8% body fat (white girls); 43.9% body fat (black boys); 45.2% body fat (black girls)	BM,E no no G	8 mo 160 60 min for LSE, variable for PA NR
Mellin et al 1987[112]	66 adolescents USA	12-18 21% 22%	30-37% overweight	BM,D,E yes yes G	14 wk 16 90 min 1440 min
Saelens et al 2002[113]	44 adolescents USA	12-16 59% 30%	BMI 30.7 kg/m^2 (3.1)	BM,D,E yes no I	4 mo 13 10-20 min for TC, NR for visit NR, >200 min

Intervention characteristics: BM=behavior modification; D = special diet; E = exercise program; G = group; I = individual;
Other outcomes: B = behavioral ; P = physiological; H = childhood health outcomes; A = adverse effects

Table 5. Randomized Controlled Trials Addressing Overweight in Children and Adolescents

Study Reference	Group	Units of Measure	Study Duration	Outcome at Latest Follow-up Time	P Value for Comparisons between Groups**	Other Outcomes	USPSTF Quality Grade
Israel 1985[108]	BT + parent training in child management	% overweight change	12 months	-10.2%	p < 0.001	B	Fair-to-Poor
	BT only			-1.3%			
	Wait list controls			NR			
Israel et al 1994[109]	Enhanced child involvement	% overweight change	36 months	-4.8%	n.s.	none	Fair-to-Poor
	Standard treatment (parents primarily responsible)			6.4%			
Kang et al 2002[111] **/ Gutin et al 2002**[110]	LSE + high intensity PA	change in % body fat	8 months	-2.9%	n.s.	B, P	Fair-to-Poor
	LSE + moderate PA			-1.4%			
	LSE			-0.1%			
Mellin et al 1987[112]	SHAPEDOWN group (Cognitive, behavioral, affective	% overweight change	15 months	-9.9%	Between group comparison NR (15 months vs. baseline: p < 0.01, SHAPEDOWN; n.s., control group)	B, H	Fair
	No treatment controls			-0.1%			
Saelens et al 2002[113]	Healthy habits intervention	% overweight change & change in BMI (statistical analyses on BMI z-scores)	7 months	-2.4%, 0.1 kg/m2	n.s.	B, A	Good
	Typical care			4.1%, 1.4 kg/m2			

Intervention characteristics: BM=behavior modification; D = special diet; E = exercise program; G = group; I = individual;
Other outcomes: B = behavioral ; P = physiological; H = childhood health outcomes; A = adverse effects

Table 5. Randomized Controlled Trials Addressing Overweight in Children and Adolescents

Study Reference	N Randomized Country	Age % Male % Non-White	Baseline Measure of Overweight	Intervention characteristics* Components Comprehensive? Parent Participation? Group vs.	Intervention characteristics* Time Period # of Sessions Session Length Total Contact Time (min)
Senediak and Spence 1985[114]	45 children USA	6-13 approximately 66% NR	37.22% overweight	BM,D,E yes yes G	4 wk 8 90 min 720 min
Wadden et al 1990[115]	47 girls USA	12-16 0% 100% black	95.1 kg; BMI 35.6 kg/m^2	BM,D,E yes yes G	10 mo 22 60 min (first 16 sessions), others NR >960 min
White 2003[116]/ Williamson unpublished data	57 adolescents USA	11-15 0% 100%	BMI 36.34 kg/m^2; 98.3 BMI %ile	BM,D,E yes yes I	6 mo 4 + weekly website logins NR NR

*for most intensive intervention which is listed first

Intervention characteristics: BM=behavior modification; D = special diet; E = exercise program; G = group; I = individual;
Other outcomes: B = behavioral ; P = physiological; H = childhood health outcomes; A = adverse effects

Table 5. Randomized Controlled Trials Addressing Overweight in Children and Adolescents

Study Reference	Group	Units of Measure	Study Duration	Outcome at Latest Follow-up Time	P Value for Comparisons between Groups**	Other Outcomes	USPSTF Quality Grade
Senediak and Spence 1985[114]	Rapid schedule BT	% overweight change	6 months	-14.7%	p < 0.05, rapid and gradual schedule BT groups combined vs. non-specific controls; (comparison of rapid vs. gradual schedule BT groups n.s.)	B	Fair-to-Poor
	Gradually decreasing schedule BT			-18.3%			
	Non-specific treatment controls			-10.9%			
	Wait list controls			NR			
Wadden et al 1990[115]	Mother and child together	change in weight	6 months	1.7 kg	n.s.	P, H	Fair-to-Poor
	Child alone			3.0 kg			
	Mother and child separate			3.5 kg			
White 2003[116]/ Williamson unpublished data	Behavioral	change in % body fat; change in BMI	6 months	-1.12%; -0.19 kg/m2	p < 0.05 (% body fat); P < 0.05 (change in BMI)	B	Good
	Education only			0.43%; 0.65 kg/m2			

**if multiple comparisons, then presented only if p < 0.05

Intervention characteristics: BM=behavior modification; D = special diet; E = exercise program; G = group; I = individual;
Other outcomes: B = behavioral ; P = physiological; H = childhood health outcomes; A = adverse effects

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

Key Question		Study Hierarchy	Overall USPSTF Quality
1.	Screening	-	Poor.
2a.	Prevalence	II-2	Good, but lacking for specific non-white racial/ethnic subgroups.
2b,c.	Screening tests as a risk factor	II-2	Fair. Data for BMI as a risk factor for adult overweight from childhood overweight are the most valid but are very limited for non-whites. Data for BMI as a risk factor for adult morbidities generally do not control for confounding by adult BMI.
3.	Screening harms	-	Poor. Due to lack of screening studies, possible harms can only be inferred from other sources.
4,5.	BCI	I	Fair-to-poor. Data are limited by very small samples, non-comparable interventions, & not using intent-to-treat analyses. Little reporting of intermediate outcomes—including risk factor changes, or changes in health outcomes. Poor generalizability due to specialist interventions not widely available and addressing mostly 8-12 years. No data in 2-5. Few trials include non-whites.
4,5.	Pharmacology with BCI	I	Fair. One good-quality trial in adolescents.
4,5.	Surgery	-	Poor.
6.	Intervention harms	I, II-2	Fair-to-Poor. Very limited reporting of harms for BCI.

Table 7. Pending Studies / Studies Awaiting Assessment

Study Identification Completion Date, Funding Agency	Trial Name/Title	Participants	Interventions	Study Aim
Epstein, LH, et al In press, *Health Psychology*	The effect of reinforcement or stimulus control to reduce sedentary behavior in the treatment of pediatric obesity	N/A	N/A	N/A
Epstein, LH March 2006 National Institute of Child Health and Human Development (NICHD)	A Behavioral Economic Approach to Childhood Obesity	120 obese children, followed for two years	Randomized to one of two groups based on behavioral economic theory or standard family-based behavioral intervention program.	To test an innovative program for pediatric obesity based on behavioral economic theory that provides reinforcement for obese children for alternatives to their usual high-fat/low nutrient density eating.
NICHD	Safety and Efficacy of Orlistat in African American and Caucasian Children and Adolescents with Obesity-Related Co morbid Conditions	12-17 year old African American or Caucasian with BMI above the 95th percentile, evidence of quantifiable obesity-related comorbidities; Control group: Healthy children and adolescents with BMI above the 5th but below the 85th percentile.	Investigation drug: Orlistat	Determine the safety, tolerability, and efficacy of Orlistat in children and adolescent with comorbidities
Reilly, John J	The Scottish Childhood Obesity Treatment Trial (SCOTT)	6-10 year old children	140 children randomized to treatment (24-week behavioral change program) or control group (typical care)	Primary outcome is change in BMI, secondary outcomes are physical activity, blood pressure, body fat distribution, body fatness, and adverse effects.

Table 7. Pending Studies / Studies Awaiting Assessment

Study Identification Completion Date, Funding Agency	Trial Name/Title	Participants	Interventions	Study Aim
Reynolds, Kim D. June 2005 National Cancer Institute	Obesity Prevention Tailored for Health	Families with children 10-11 years old	A motivational Interviewing protocol will be developed for delivery to the parents	Measurements of diet, psychosocial variables, fruit and vegetable availability, and BMI will be completed on children and one parent
Ariza, Adolfo J. December 2004 AHRQ	Tools to Improve Nutritional Health in Primary Care (pilot study)	Two diverse medical practices, one with a majority of white, privately insured patients and the other a community health center serving low-income African American and Hispanic patients.	The program includes a practice educational intervention and the use of a computerized system using newly developed software that has the ability to track growth and provide personalized handouts about child nutrition according to child age and nutritional status categories.	Determine changes effected by the program in the rates of identification of overweight or at risk for overweight children, in the provision of counseling on healthy behaviors, and in-patient flow.
Barlow, Sarah E. August 2005 AHRQ	Improving Obesity Care in Pediatric Offices	6-16-year-olds	Intervention is with pediatricians	To improve communication about childhood and adolescent obesity in the offices of pediatricians.
Saelens, Brian E. March 2007 National Institute of Diabetes and Digestive and Kidney Diseases	Body Fat and Hormones in Adolescent Obesity Treatment	NR	NR	Examining the time course of total body fat and intra-abdominal fat accretion through early puberty among already overweight youth. Investigate the differential impact of providing similar behavioral weight control intervention to overweight youth at different time periods of intra-abdominal fat mass accumulation.

Table 7. Pending Studies / Studies Awaiting Assessment

Study Identification Completion Date, Funding Agency	Trial Name/Title	Participants	Interventions	Study Aim
Wilfley, Denise E. July 2004 NICHD	Childhood Obesity Treatment: A Maintenance Approach	216 9-11-year-old children with one obese parent	Two strategies designed to improve the long-term maintenance of weight loss in children	The study will examine whether the content of the maintenance sessions or extended treatment is important in improving long-term maintenance.
Golan, M. February 2004 No outside funding	Targeting Parents Exclusively in the Treatment of Childhood Obesity: Long-Term Results	50 of the 60 children from the original study located 7 years later. Now 14-19 years of age.	Two different target groups attended sessions (parent-only group, child-only group) to test the effect on children's percent overweight.	To report the long-term change in children's overweight following a family-based health-centered approach where only parents were targeted compared with a control intervention where only children were targeted.
Epstein, H 2004 NICHD	The Effect of Reinforcement or Stimulus Control to Reduce Sedentary Behavior in the Treatment of Pediatric Obesity	72 families with a child 8-12 years old with BMI > 85th percentile.	Two treatment groups: reinforced reduced sedentary behavior or stimulus control of sedentary behaviors.	To determine whether different methods of reducing targeted sedentary behaviors as associated with differences in the pattern of change in behaviors and in percentage overweight change.

Abbreviations

Abbreviation	Definition
AHRQ	Agency for Healthcare Research and Quality
AR	adiposity rebound
b or B	black
BCI	behavioral counseling interventions
BHS	Bogalusa Heart Study
BIA	bioelectrical impedance analysis
BMC	bone mineral content
BMI	body mass index
BP	blood pressure
bpm	beats per minute
BT	behavioral therapy
CCT	controlled clinical trial
CDC	Centers for Disease Control and Prevention
CHD	coronary heart disease
CHO	carbohydrate
CI or C.I.	confidence interval
cm	centimeter(s)
comp	comparison intervention
cont	control group
CT	computed tomography
CTF	Community Task Force
CV	cardiovascular
CVD	cardiovascular disease
DBP	diastolic blood pressure
DEXA	dual energy X-ray absorptiometry
DM	diabetes mellitus
DMII	diabetes mellitus II
ECG	electrocardiogram
Exp	experimental intervention
f or F	female
FFM	fat-free mass
FM	fat mass
f/u	follow up
g	gram(s)
GBD	gallbladder disease
GTT	glucose tolerance test
H	height *or* Hispanic
HDL	high-density lipoprotein
HDLC	high-density lipoprotein cholesterol
HH	healthy habits
HHANES	Hispanic Health and Nutrition Examination Survey
HIPT	high-intensity physical activity treatment
HMO	health maintenance organization
HOMA	homeostatic model of insulin sensitivity
HR	hazards ratio *or* heart rate
ht or Ht	height
IBW	ideal body weight
in	inches
int	intervention
IOTF	International Obesity Task Force
kcal	kilocalorie(s)

Appendix A. Abbreviations

kcal/d	kilocalorie(s)/day
kg	kilogram(s)
kg/m²	kilograms divided by meters squared
KQ	Key Question
lbs	pounds
LDL	low-density lipoprotein
LSE	lifestyle education
m or M	male
MA	Mexican American
med	medium
METS	metabolic equivalents
mg	milligram(s)
mg/dL	milligram(s)/deciliter
mi	mile(s)
min	minute(s)
MIPT	medium-intensity physical activity treatment
mL	milliliter(s)
mm	millimeter(s)
mmHg	millimeters of mercury
mo(s)	month(s)
mod	moderate
MRI	magnetic resonance imaging
n or N	number
NA	Native American
N/A	not applicable *or* not available
NAA	neutron activation analysis
NASH	non-alcoholic steatohepatitis
NCHS	National Center for Health Statistics
NHANES	National Health and Nutrition Examination Survey
NHB	non-Hispanic black
NHES	National Health Examination Survey
NHW	non-Hispanic white
NIH	National Institutes of Health
NNS	number needed to screen
NNT	number needed to treat
NR	not reported
NS or n.s.	not significant
OA	osteoarthritis *or* overweight adults
obs	observations
OC	overweight children
O:E	observed: expected
OR	odds ratio
OW	overweight
p	p value
PA	physical activity
PC	phone call
PCP	primary care provider
PI	ponderal index (w/h^3)--same measure as Rohrer index
pop	population
Prev	prevalence
Prob	probability
PS	problem solving
pts	patients
QD	every day
QI	Quetelet index (w/h^2)
r	regression coefficient

Appendix A. Abbreviations (continued)

RCT	randomized clinical trial
RF	reduced fat
RGL	reduced glycemic load
RI	Rohrer index (w/h^3)
ROC	receiver-operating characteristic
RR	relative risk
SA	sleep apnea
SB	sedentary behavior
SBP	systolic blood pressure
SD	standard deviation
SE	standard error
sens	sensitivity
SKF	skinfold thickness
spec	specificity
SSF	subscapular skinfold thickness
TBW	total body weight
TC	total cholesterol *or* typical care
TG	triglycerides
TLD	Traffic Light Diet
TOBEC	total body electrical conductivity
TSF	triceps skinfold
UK	United Kingdom
US	United States *or* ultrasound
USPSTF	U.S. Preventive Services Task Force
VLDL	very low density lipoprotein
VO2	volume of oxygen
w or W	white
W/H	weight for height
WHO	World Health Organization
wk	week(s)
WLBS	Weight Loss Behavior Scale
wm	white male
wt	weight
yo	years old
yr	year(s)

Measuring Adiposity in Children and Adolescents

In this section, we review the challenges in measuring adiposity through direct or indirect measurements of body composition, since these challenges directly affect the choice of appropriate and available clinical measurement techniques, and determine the acceptability of reference standards used to validate clinical measures in screening and intervention (Table B-1). We then examine the advantages and limitations of clinical measures of overweight, such as body mass index (BMI).

Measures of Adiposity

In obesity, adiposity (total body fat expressed as a percentage of total body weight) is the body composition measure of primary physiologic interest.[1] Body fat mass (FM) measurement through direct or indirect approaches is preferable, but not practical for routine use. FM is one of two main components of body composition, along with fat-free mass (FFM)—which can be further divided into subcompartments of total body water, body protein, and body mineral.[2,3] Body composition is measured directly using dual x-ray absorptiometry (DEXA) or hydrodensitometry, with FM determined from this measurement in combination with measured results for other body components.[4] Body composition must be measured and not inferred as its components change with growth and development throughout childhood and adolescence, with expected variations due to sex, age, pubertal status, race/ethnicity, athletic status, and disease state.[3,5,6] Laboratory-based body composition approaches that directly measure multiple (three or more) body compartments are accepted as valid for estimating body fat in children,[7,8] particularly after age three-five when hydration levels of fat-free mass reach adult values.[3] Multi-component body composition measures offer greater accuracy and precision in children[7] but are not practical for clinical practice or large-scale epidemiologic surveys on the basis of cost, portability, and acceptability to the patient.[8]

Clinical Measures of Total Body Adiposity

Portable, non-invasive measures of body fat appropriate for clinical practice, such as skinfold thickness (SKF) and, more recently, bioelectrical impedance analysis (BIA), are proxy (indirect) measures for determining total body fat. These measures are used in prediction equations to estimate a simpler two-component model of body composition--FM and FFM--in order to determine whether there is excess fat (obesity). The two-component body composition model is increasingly recognized as insufficient for estimating body fat in children, because the subcomponents of FFM in children are not constant as they generally are in adults. Children and prepubescent adolescents exhibit chemical immaturity,[6] with a gradual change in FFM composition through infancy, childhood, and adolescence to adult values.[8]

The two-component model is based on assumptions that ignore other sources of interindividual variability in the composition of FFM in children, such as variations in bone mineral content due to race/ethnicity.[9] In 20 black and 20 white normal-weight girls aged 7-10 years who were matched for BMI, bone age, chronological age, Tanner

breast stage, and socioeconomic status, black girls had significantly greater bone mineral density and less total adipose tissue than white girls.[10] This finding of increased bone mineral content in prepubescent black girls, compared with age- and weight-matched white girls, has been confirmed in other studies. Ethnic differences in skeletal growth patterns, as well as effects of puberty on bone density, have also been reported.[9]

Prediction equations used to determine body fat from anthropometric measurements in two-component or other models are problematic. These equations have often been derived from samples including a wide range of prepubescent, pubescent, and postpubescent subjects, and therefore do not apply well to any of these subgroups, as each group differs in the relationship between skinfold thickness and body density.[6] Equations developed to predict body fat based on BIA tend to lack cross-validity even when they have been "validated," (i.e., they do not provide accurate estimates in populations other than those in which they were derived).[8] Different prediction equations in the same children result in very different estimates of mean body fat: estimates range from 5.0 percentage points below to 3.0 points above the reference method for prediction equations based on skinfold measurements, and from 6.8 percentage points below to 8.6 points above for prediction equations based on BIA measurements.[8] The ability of BIA to predict body composition in children varies by equation, and its accuracy cannot be safely assumed.[11] Few prediction equations based on BIA measurements have been rigorously evaluated in obese persons.[12] Finally, few prediction equations have been validated in people of diverse racial backgrounds,[6] although some reports are appearing.

Indirect measures of body fat often have additional measurement limitations due to variation in the selection of sites and in measurement techniques.[6] In some circumstances the assessment of body composition by such measures is compromised more by measurement variability than by overall inaccuracy.[13] Skinfold measurements with a single observer can be reproducible,[12] but interrater reliability is often poor.[14,15 16] Skinfold measures are even less reliable for those with markedly increased body fat.[17] Instruments under development that may have future clinical application in pediatric body composition measurement, such as air-displacement plethysmography and bioelectrical impedance, still show considerable variability between instruments and methods.[3,18,19]

Clinical Measures of Intra-Abdominal Adiposity

In adults, intra-abdominal body fat (around visceral organs) is related to negative health outcomes independent of total body fat.[4,20] Intra-abdominal fat as measured anthropometrically in children and adolescents has been related cross-sectionally to presence and clustering of cardiovascular risk factors within black and white children.[21-23] Research in this area is active. It is currently unclear whether and when visceral fat accumulates in children as an appropriate part of growth and development. Intra-abdominal adipose tissue has been observed in healthy, non-obese children as young as four-seven years of age, as well as non-obese adolescents.[4] Waist circumference correlates most strongly with DEXA-determined fat distribution in 201 black and white children aged 7-17 years, compared with other indirect measures of intra-abdominal obesity including BMI, waist-hip circumference ratio, subscapular/triceps skinfold ratio, and subscapular + suprailiac/triceps skinfold ratio.[24] However, in studies using more

accurate imaging techniques for assessing body fat distribution in children (CT and MRI), neither waist circumference nor other indirect indicators of body fat distribution correlate well with intra-abdominal adipose tissue.[4] Given the uncertainties about the clinical meaning of indirect central obesity measures, including the extent of normal variations due to age, gender, race, and percentage body fat[4,24]and whether observed extremes in these measures have an impact on current and future morbidity,[25] these tools are not appropriate for clinical obesity screening at this time.

Comparability of Clinical Measures for Adiposity

Even if acceptable, clinical measures for adiposity cannot be assumed to substitute for one another. Several recent studies illustrate the current limits of utility and consistency between various clinical adiposity measures in children. Researchers categorized 625 overweight and obese white youths aged 12-18 years in the NHANES III study as obese if they were above the 80th percentile in their age and-sex group for BMI, triceps and subscapular skinfolds, sum of four skinfolds, waist circumference, or percentage body fat determined by BIA.[26] Agreement between the anthropometric methods for overweight categorization ranged from kappas of 0.065-0.85 in males and 0.050-0.79 in females, and showed further within-sex variation by age. The overall agreement between waist circumference and percent body fat was the lowest of the tested measures. When SKF measures were compared to a four-component criterion model for 40 African American and 72 white adolescent girls, individual percent fat mass could be over- or under-estimated by as much as 10%.[27] Racial/ethnic subgroups may need separate reference standards. Important age, sex, and ethnic differences in body fat growth have been described, when comparing percent body fat growth estimates from two different sum of skinfold measures and those from bioelectrical impedance combined with body measurements, body mass index, and abdominal circumference in 678 boys and girls (20% black, 75% non-Hispanic white, and 5% other).[28] Fat growth patterns showed marked differences for each index, and within indices, varied by sex, age, and race/ethnicity. For three measures (percent body fat, sum of two skinfolds, and sum of six skinfolds), black females had the highest values, followed by non-black females and non-black males, with lowest values among black males. However, the shapes of the curves were not comparable either within or across measures. The authors point out that the distinct growth patterns for different indices of body fat indicate that they likely measure different tissue content, and thus should not be expected to relate in the same way to intermediate or actual health outcomes.

Appendix Table B-1. Reference Standards for Pediatric Obesity Screening[2,3,6,8,12,17,19,29-37]

Preferable/Highest validity 4-C model (independent measurement of bone mineral content, TBW, and NAA measurement or extrapolation of body protein from bone mineral measurement) 3-C model at age five or above (independent measurement of TBW via isotope dilution and body density via BODPOD, isotope dilution, or hydrodensitometry using Lohman's equations) (except in racial and ethnic subgroups or athletes, then becomes only acceptable)
Acceptable 3-C model at age five or above (independent measurement of TBW via isotope dilution and FFM from total body potassium) 3-C model (independent measurement of TBW via means other than isotope dilution (e.g., TOBEC) and body density via BODPOD, isotope dilution, or hydrodensitometry not clearly using Lohman's equations) 3-C model (independent measurement of bone mineral content via DEXA and hydrodensitometry using Lohman's equations) DEXA alone in a 3C model (used to measure BMC, lean tissue mass to derive fat mass)
Barely acceptable 2-C model at age five or above (measurement of TBW via isotope dilution) 2-C model using Lohman's equation or equivalent
Unacceptable MRI (due to cost, duration of testing) Whole body CT (due to cost, radiation exposure) Bioelectrical impedance (BIA)-potential screening test Electromagnetic induction alone or in children under five(e.g., TOBEC) 2-C model not using Lohman's age and sex-specific equations 2-C model, measurement of total body K+ -Near infrared interactance (NIRI) -Skinfold thickness, other anthropomorphic measures

References

1. Oken E, Lightdale JR. Updates in pediatric nutrition. *Curr Opin Pediatr* 2000; 12(3):282-290.

2. Sutcliffe JF. A review of in vivo experimental methods to determine the composition of the human body. *Phys Med Biol* 1996; 41(5):791-833.

3. Ellis KJ. Human body composition: in vivo methods. *Physiol Rev* 2000; 80(2):649-680.

4. Goran MI. Measurement issues related to studies of childhood obesity: assessment of body composition, body fat distribution, physical activity, and food intake. *Pediatrics* 1998; 101(3 Pt 2):505-518.

5. Lohman T. *Advances in Body Composition Assessment*. 3rd ed. Champaign, IL: Human Kinetics Publishers, 1992.

6. Lohman TG. Applicability of body composition techniques and constants for children and youths. *Exerc Sport Sci Rev* 1986; 14:325-357.

7. Wells JC, Fuller NJ, Dewit O, Fewtrell MS, Elia M, Cole TJ. Four-component model of body composition in children: density and hydration of fat-free mass and comparison with simpler models. *Am J Clin Nutr* 1999; 69(5):904-912.

8. Reilly JJ. Assessment of body composition in infants and children. *Nutrition* 1998; 14(10):821-825.

9. Gasperino J. Ethnic differences in body composition and their relation to health and disease in women. *Ethnicity & Health* 1996; 1(4):337-347.

10. Yanovski JA, Yanovski SZ, Filmer KM, Hubbard VS, Avila N, Lewis B et al. Differences in body composition of black and white girls. *American Journal of Clinical Nutrition* 1996; 64(6):833-839.

11. Reilly JJ, Wilson J, McColl JH, Carmichael M, Durnin JV. Ability of biolectric impedance to predict fat-free mass in prepubertal children. *Pediatr Res* 1996; 39(1):176-179.

12. Jebb SA, Elia M. Techniques for the measurement of body composition: a practical guide. *Int J Obes Relat Metab Disord* 1993; 17(11):611-621.

13. Elia M. Body composition analysis: an evaluation of 2 component models, multicomponent models and bedside techniques. *Clinical Nutrition* 1992; 11:114-127.

14. Marks GC, Habicht JP, Mueller WH. Reliability, dependability, and precision of anthropometric measurements. The Second National Health and Nutrition Examination Survey 1976-1980. *Am J Epidemiol* 1989; 130(3):578-587.

15. Chumlea WC, Guo SS, Kuczmarski RJ, Flegal KM, Johnson CL, Heymsfield SB et al. Body composition estimates from NHANES III bioelectrical impedance data. *International Journal of Obesity & Related Metabolic Disorders* 2002; 26(12):1596-1609.

16. Dietz WH, Bellizzi MC. Introduction: the use of body mass index to assess obesity in children. *Am J Clin Nutr* 1999; 70(1):123S-125S.

17. Kuczmarski RJ. Trends in body composition for infants and children in the U.S. *Crit Rev Food Sci Nutr* 1993; 33(4-5):375-387.

18. Fields DA, Goran MI, McCrory MA. Body-composition assessment via air-displacement plethysmography in adults and children: a review. *Am J Clin Nutr* 2002; 75(3):453-467.

19. Horlick M, Arpadi SM, Bethel J, Wang J, Moye J, Jr., Cuff P et al. Bioelectrical impedance analysis models for prediction of total body water and fat-free mass in healthy and HIV-infected children and adolescents. *Am J Clin Nutr* 2002; 76(5):991-999.

20. Bjorntorp P. Abdominal obesity and the metabolic syndrome. *Ann Med* 1992; 24(6):465-468.

21. Morrison JA, Barton BA, Biro FM, Daniels SR, Sprecher DL. Overweight, fat patterning, and cardiovascular disease risk factors in black and white boys. *Journal of Pediatrics* 1999; 135(4):451-457.

22. Morrison JA, Sprecher DL, Barton BA, Waclawiw MA, Daniels SR. Overweight, fat patterning, and cardiovascular disease risk factors in black and white girls: The National Heart, Lung, and Blood Institute Growth and Health Study. *Journal of Pediatrics* 1999; 135(4):458-464.

23. Freedman DS, Serdula MK, Srinivasan SR, Berenson GS. Relation of circumferences and skinfold thicknesses to lipid and insulin concentrations in children and adolescents: the Bogalusa Heart Study. *Am J Clin Nutr* 1999; 69(2):308-317.

24. Daniels SR, Khoury PR, Morrison JA. Utility of different measures of body fat distribution in children and adolescents. *Am J Epidemiol* 2000; 152(12):1179-1184.

25. McCarthy HD, Ellis SM, Cole TJ. Central overweight and obesity in British youth aged 11-16 years: cross sectional surveys of waist circumference. *BMJ* 2003; 326(7390):624.

26. Himes JH. Agreement among anthropometric indicators identifying the fattest adolescents. *Int J Obes Relat Metab Disord* 1999; 23 Suppl 2: S18-S21.

27. Wong WW, Stuff JE, Butte NF, Smith EO, Ellis KJ. Estimating body fat in African American and white adolescent girls: a comparison of skinfold-thickness equations with a 4-compartment criterion model. *American Journal of Clinical Nutrition* 2000; 72(2):348-354.

28. Dai S, Labarthe DR, Grunbaum JA, Harrist RB, Mueller WH. Longitudinal analysis of changes in indices of obesity from age 8 years to age 18 years. Project HeartBeat! *American Journal of Epidemiology* 2002; 156(8):720-729.

29. Ellis KJ. Measuring body fatness in children and young adults: comparison of bioelectric impedance analysis, total body electrical conductivity, and dual-energy X-ray absorptiometry. *Int J Obes Relat Metab Disord* 1996; 20(9):866-873.

30. Fomom SJ, Haschke F, Ziegler EE, Nelson SE. Body composition of reference children from birth to age 10 years. *Am J Clin Nutr* 1982; 35(5 Suppl):1169-1175.

31. Gately PJ, Radley D, Cooke CB, Carroll S, Oldroyd B, Truscott JG et al. Comparison of body composition methods in overweight and obese children. *J Appl Physiol* 2003; 95(5):2039-2046.

32. Gutin B, Litaker M, Islam S, Manos T, Smith C, Treiber F. Body-composition measurement in 9-11-y-old children by dual-energy X-ray absorptiometry, skinfold-thickness measurements, and bioimpedance analysis. *Am J Clin Nutr* 1996; 63(3):287-292.

33. Houtkooper LB, Going SB, Lohman TG, Roche AF, Van Loan M. Bioelectrical impedance estimation of fat-free body mass in children and youth: a cross-validation study. *J Appl Physiol* 1992; 72(1):366-373.

34. Laskey MA. Dual-energy X-ray absorptiometry and body composition. *Nutrition* 1996; 12(1):45-51.

35. Treuth MS, Butte NF, Wong WW, Ellis KJ. Body composition in prepubertal girls: comparison of six methods. *Int J Obes Relat Metab Disord* 2001; 25(9):1352-1359.

36. Parker L, Reilly JJ, Slater C, Wells JC, Pitsiladis Y. Validity of six field and laboratory methods for measurement of body composition in boys. *Obes Res* 2003; 11(7):852-858.

37. Roemmich JN, Clark PA, Weltman A, Rogol AD. Alterations in growth and body composition during puberty. I. Comparing multicompartment body composition models. *J Appl Physiol* 1997; 83(3):927-935.

Clinical Measures of Overweight

The limitations of the clinically feasible adiposity measures in children necessitate adopting different measures for obesity diagnosis. Height and weight measures are simple, practical, and reliable.[1] Their use to estimate overweight has traditionally involved classifying children by percentage deviation from mean weight or weight-for-height on age-standardized growth charts.[2] Weight must be adjusted for height to reflect age-normal differences in growth and body build. Measures of weight per height include weight/height; weight/height^2 (body mass index [BMI] or Quetelet index); weight/height^3 (ponderal index); weight/height^k ; standardized weight (measured weight minus mean weight for that height divided by standard deviation of weight for that height); and relative weight (weight X 100 /mean weight for that height).[3] An important limitation of all weight-for-height measures is that they cannot distinguish between excess weight due to increased adiposity from that due to increased lean body mass. Thus, use of these measures defines obesity as excess body weight rather than excess body fat.

Among the many weight-for-height adjusted measures, BMI has become the primary index of relative weight.[4] BMI equals kilograms of weight divided by height in meters squared (kg/m^2), and is compared to population-based reference standards. In 1997, a workshop to assess the strengths and limitations of practical measurements to assess overweight and obesity in children and adolescents worldwide was convened by the International Obesity Task Force.[5] In reviewing the use of BMI, other weight-for-height indicators, and indirect measures of body fat, experts concluded that BMI offered a reasonable measure of fatness in children and adolescents. They based their conclusion on moderate to high correlations (.44-.82) between BMI and percentage body fat (measured by DEXA or by underwater weighing) in non-obese boys and girls aged 4-18 years, and the lack of reproducibility of direct measures of body fat, such as triceps skinfolds. Further, the authors noted that use of BMI in children and adolescents would provide a consistent assessment of obesity across the lifespan. However, they also noted several problems with the use of BMI as an adiposity index, including the fact that although BMI is highly correlated with measures of body fat, it is not a precise indicator of overweight or obesity, since high BMI for an individual child or adult can be due to increased fat-free mass.[6] This concern has been amplified in a study in which a large proportion of the between-child variations in BMI were explained by variability in fat-free mass and not just differences in fat mass.[7] Also, there is limited data on BMI's appropriateness in non-white ethnic groups. Among black and white nine-year-old girls in the National Heart, Lung and Blood Institute Growth and Health study, BMIs are consistently higher in black girls of all ages.[8] This does not necessarily reflect overall greater adiposity among black females, since across these ages the BMIs of black girls in the 15th percentile by race consistently exceed those of white girls in the 15th percentile by 0.5 kg/m^2. In a cross-sectional study of 192 healthy boys and girls aged 7-17 years old (46% black and 54% white), the relationship between BMI and percentage body fat measured by DEXA was dependent on several factors in addition to the known differences due to sex[9]: sexual maturation (body fat is lower in those of greater sexual maturity), race (at a given BMI, whites have higher body fat than blacks), and waist: hip ratio (at the same BMI, higher ratios are associated with greater body fatness). Stage of

sexual maturation was a more important correlate of percent body fat than age; thus, the percent body fat for a given BMI may differ depending on the level of maturation.

Another concern about BMI as a measure of fatness in individual children comes from reliance on strength of correlation rather than calculated agreement between methods in determining the validity of BMI.[10]

Although a number of studies have found that BMI cutoffs at the upper end of the distribution are reasonably specific for classifying the fattest children,[11] this body of literature is comprised of studies using different BMI cutoff definitions and varying criterion measures of body fat, many of which are not directly comparable or are of questionable validity.[12-14] (See Appendix B, Table B-1) There is little evidence on the sensitivity and specificity of BMI as a screening tool for obesity compared to valid reference standards in large U.S. samples of boys and girls of all ages and races, with the body composition range from low to the excess adiposity seen in clinical practice. For example, in a study of 1,570 children aged 9-19 years of BMI validity in obesity, triceps skinfold (TSF) was the fat criterion used for most of the sample; as a reference criterion TSF is insufficient.[15] Among the remainder of the sample with a valid fat criterion measure (98 Mexican American boys and 104 white girls), none had BMI measures at or above the 95th percentile. BMI at the 85th percentile was reasonably specific in boys (98.8%) and girls (95.0%) but very insensitive (values of 30.8% or less). Studies such as these are inadequate to answer how validly BMI identifies excess fat. Given the current state of this literature on BMI, many have shifted their focus to the validity of BMI cutpoints as a pragmatic measure of risk for adult overweight and as an indicator of future morbidity or mortality.[16,17]

Measures other than BMI, such as skinfold thicknesses and waist circumference, are not sufficiently well-developed in terms of reference values, measurement approaches, or established relationships to criterion methods or to morbid outcomes to serve as independent clinical measures.[18] Other clinical measures such as these may eventually prove useful in supplementing BMI to define overweight in particular subgroups, such as adolescent males.[13]

References

1. Kuczmarski RJ. Trends in body composition for infants and children in the U.S. *Crit Rev Food Sci Nutr* 1993; 33(4-5):375-387.

2. Oken E, Lightdale JR. Updates in pediatric nutrition. *Curr Opin Pediatr* 2000; 12(3):282-290.

3. Thomas PW, Peters TJ, Golding J, Haslum MN. Weight-for-height in two national cohorts with particular reference to 10-year-old children. *Annals of Human Biology* 1989; 16(2):109-119.

4. Poskitt EM. Defining childhood obesity: the relative body mass index (BMI). European Childhood Obesity group. *Acta Paediatr* 1995; 84(8):961-963.

5. Dietz WH, Bellizzi MC. Introduction: the use of body mass index to assess obesity in children. *Am J Clin Nutr* 1999; 70(1):123S-125S.

6. Maynard LM, Wisemandle W, Roche AF, Chumlea WC, Guo SS, Siervogel RM. Childhood body composition in relation to body mass index. *Pediatrics* 2001; 107(2):344-350.

7. Wells JC. A Hattori chart analysis of body mass index in infants and children. *Int J Obes Relat Metab Disord* 2000; 24(3):325-329.

8. Kimm SY, Barton BA, Obarzanek E, McMahon RP, Sabry ZI, Waclawiw MA et al. Racial divergence in adiposity during adolescence: The NHLBI Growth and Health Study. *Pediatrics* 2001; 107(3):E34.

9. Daniels SR, Khoury PR, Morrison JA. The utility of body mass index as a measure of body fatness in children and adolescents: differences by race and gender. *Pediatrics* 1997; 99(6):804-807.

10. Reilly JJ. Assessment of body composition in infants and children. *Nutrition* 1998; 14(10):821-825.

11. Reilly JJ, Wilson ML, Summerbell CD, Wilson DC. Obesity: diagnosis, prevention, and treatment; evidence based answers to common questions. *Arch Dis Child* 2002; 86(6):392-394.

12. Ellis KJ. Human body composition: in vivo methods. *Physiol Rev* 2000; 80(2):649-680.

13. Himes JH. Agreement among anthropometric indicators identifying the fattest adolescents. *Int J Obes Relat Metab Disord* 1999; 23 Suppl 2: S18-S21.

14. Wells JC, Fuller NJ, Dewit O, Fewtrell MS, Elia M, Cole TJ. Four-component model of body composition in children: density and hydration of fat-free mass and comparison with simpler models. *Am J Clin Nutr* 1999; 69(5):904-912.

15. Malina RM, Katzmarzyk PT. Validity of the body mass index as an indicator of the risk and presence of overweight in adolescents. *Am J Clin Nutr* 1999; 70(1):131S-136S.

16. Reilly JJ, Methven E, McDowell ZC, Hacking B, Alexander D, Stewart L et al. Health consequences of obesity. *Arch Dis Child* 2003; 88(9):748-752.

17. Dietz WH. Childhood weight affects adult morbidity and mortality. *Journal of Nutrition* 1998; 128(2 Suppl):411S-414S.

18. Lobstein T, Baur L, Uauy R. Obesity in children and young people: a crisis in public health. *Obes Rev* 2004; 5 Suppl 1:4-85.

Search Strategies

Databases searched: Medline®, PsycINFO, DARE, CINAHL, Cochrane Database of Systematic Reviews, Cochrane Central Register of Controlled Trials

KQ 1 and KQ 2 Years searched: 1966-June 4, 2004

1 obesity/
2 obesity in diabetes/
3 obesity, morbid/
4 (obesity or obese).ti,ab.
5 overweight.ti,ab.
6 1 or 2 or 3 or 4 or 5
7 body mass index/
8 skinfold thickness/
9 Body Height/
10 body weight/
11 9 and 10
12 bmi.ti,ab.
13 body mass indices.ti,ab.
14 body mass index$.ti,ab.
15 skinfold.ti,ab.
16 skin fold.ti,ab.
17 weight for height.ti,ab.
18 height for weight.ti,ab.
19 weight for length.ti,ab.
20 weight for age.ti,ab.
21 weight for stature.ti,ab.
22 self report$.ti,ab.
23 parent$ report$.ti,ab.
24 patient report$.ti,ab.
25 screen$.ti,ab.
26 mass screening/
27 7 or 8 or 11 or 12 or 13 or 14 or 15 or 16 or 17 or 18 or 19 or 20 or 21 or 22 or 23 or 24 or 25 or 26
28 6 and 27
29 obesity/di
30 obesity in diabetes/di
31 obesity, morbid/di
32 ((obesity or obese or overweight) adj5 diagnos$).ti,ab.
33 29 or 30 or 31 or 32
34 28 or 33
35 limit 34 to (preschool child <2 to 5 years> or child <6 to 12 years> or adolescent <13 to 18 years>)
36 child.ti,ab.
37 children$.ti,ab.

38 childhood.ti,ab.
39 preschool$.ti,ab.
40 teen.ti,ab.
41 teens.ti,ab.
42 teenage$.ti,ab.
43 pediatric$.ti,ab.
44 paediatric$.ti,ab.
45 adolescen$.ti,ab.
46 boys.ti,ab.
47 girls.ti,ab.
48 youth.ti,ab.
49 youths.ti,ab.
50 36 or 37 or 38 or 39 or 40 or 41 or 42 or 43 or 44 or 45 or 46 or 47 or 48 or 49
51 34 and 50
52 35 or 51
53 limit 52 to english language
54 clinical trials/ or clinical trials, phase i/ or clinical trials, phase ii/ or clinical trials, phase iii/ or clinical trials, phase iv/ or controlled clinical trials/ or randomized controlled trials/ or multicenter studies/
55 research design/ or control groups/ or double-blind method/ or meta-analysis/
56 evaluation studies/ or program evaluation/
57 epidemiologic research design/ or control groups/ or cross-over studies/ or double-blind method/ or matched-pair analysis/ or meta-analysis/ or random allocation/ or "sensitivity and specificity"/ or predictive value of tests/ or roc curve/ or single-blind method/
58 PLACEBOS/
59 Comparative Study/
60 (clinical trial or clinical trial phase i or clinical trial phase ii or clinical trial phase iii or clinical trial phase iv or controlled clinical trial or meta analysis or multicenter study or randomized controlled trial).pt.
61 clinical trial$.ti,ab.
62 (control$ adj (trial$ or stud$ or evaluation$ or experiment$)).ti,ab.
63 ((singl$ or doubl$ or trebl$ or tripl$) adj5 (blind$ or mask$)).ti,ab.
64 placebo$.ti,ab.
65 random$.ti,ab.
66 evaluation stud$.ti,ab.
67 matched pair$.ti,ab.
68 control group$.ti,ab.
69 (outcome study or outcome studies).ti,ab.
70 (quasiexperimental or quasi experimental or pseudoexperimental or pseudo experimental).ti,ab.
71 (nonrandomi?ed or non randomi?ed or pseudorandomi?ed or pseudo randomi?ed).ti,ab.
72 54 or 55 or 56 or 57 or 58 or 59 or 60 or 61 or 62 or 63 or 64 or 65 or 66 or 67 or 68 or 69 or 70 or 71
73 72 or random allocation/
74 epidemiologic studies/ or case-control studies/ or retrospective studies/ or cohort studies/ or longitudinal studies/ or follow-up studies/ or prospective studies/ or cross-sectional studies/

75 (sensitivit$ or specificit$).ti,ab.
76 (meta analy$ or metaanaly$).ti,ab.
77 (systematic$ review$ or systematic$ overview$).ti,ab.
78 (quantitative$ review$ or quantitative$ overview$).ti,ab.
79 Evidence-Based Medicine/
80 evidence based review$.ti,ab.
81 73 or 74 or 75 or 76 or 77 or 78 or 79 or 80
82 53 and 81
83 (comment or editorial or letter).pt.
84 case report/
85 83 or 84
86 82 not 85

KQ 3: Years searched: 1966-June 4, 2004
1 OBESITY/di
2 Obesity in Diabetes/di
3 Obesity, Morbid/di
4 (diagnos$ adj3 obes$).ti,ab.
5 (diagnos$ adj3 overweight).ti,ab.
6 (screen$ adj5 obes$).ti,ab.
7 (screen$ adj5 overweight).ti,ab.
8 1 or 2 or 3 or 4 or 5 or 6 or 7
9 limit 8 to (preschool child <2 to 5 years> or child <6 to 12 years> or adolescent <13 to 18 years>)
10 child.ti,ab.
11 children$.ti,ab.
12 childhood.ti,ab.
13 teen.ti,ab.
14 teens.ti,ab.
15 teenage$.ti,ab.
16 pediatric$.ti,ab.
17 paediatric$.ti,ab.
18 adolescen$.ti,ab.
19 boys.ti,ab.
20 girls.ti,ab.
21 youth.ti,ab.
22 youths.ti,ab.
23 10 or 11 or 12 or 13 or 14 or 15 or 16 or 17 or 18 or 19 or 20 or 21 or 22
24 8 and 23
25 9 or 24
26 ae.fs.
27 advers$ effect$.ti,ab.
28 (harm or harms or harmed or harmful).ti,ab.
29 quality of life/
30 depression/
31 depressive disorder/

32 (depression or depressed).ti,ab.
33 stress, psychological/
34 adaptation, psychological/
35 anxiety/
36 (anxiety or anxious).ti,ab.
37 px.fs.
38 suicide/
39 self concept/
40 self esteem.ti,ab.
41 body image/
42 social isolation/
43 risk/
44 risk factors/
45 risky behavior$.ti,ab.
46 risky behaviour$.ti,ab.
47 risk taking/
48 Professional-Patient Relations/
49 Physician-Patient Relations/
50 Patient Compliance/
51 Patient Acceptance of Health Care/
52 Patient Participation/
53 Treatment Refusal/
54 Patient Dropouts/
55 eating disorders/
56 Anorexia Nervosa/
57 bulimia/
58 weight cycling.ti,ab.
59 weight fluctuat$.ti,ab.
60 fasting/
61 laxative$.ti,ab.
62 (overweight adj4 concern$).ti,ab.
63 (weight adj4 concern$).ti,ab.
64 family relations/
65 intergenerational relations/
66 parent-child relations/
67 parenting/
68 sibling relations/
69 family/
70 interpersonal relations/
71 False Positive Reactions/
72 26 or 27 or 28 or 29 or 30 or 31 or 32 or 33 or 34 or 35 or 36 or 37 or 38 or 39 or 40 or 41 or 42 or 43 or 44 or 45 or 46 or 47 or 48 or 49 or 50 or 51 or 52 or 53 or 54 or 55 or 56 or 57 or 58 or 59 or 60 or 61 or 62 or 63 or 64 or 65 or 66 or 67 or 68 or 69 or 70 or 71
73 25 and 72
74 limit 73 to english language

KQ 4 and KQ 5: Years searched: 2001-June 4, 2004

1 exp "Obesity"/
2 "Weight-Gain"/
3 "Weight-Loss"/
4 (obesity or obese).mp.
5 (weight gain or weight loss).mp.
6 (overweight or over weight or overeat$ or over eat$).mp.
7 weight change$.mp.
8 ((bmi or body mass index) adj2 (gain or loss or change)).mp.
9 1 or 2 or 3 or 4 or 5 or 6 or 7 or 8
10 limit 9 to child <6 to 12 years>
11 limit 9 to adolescent <13 to 18 years>
12 limit 9 to preschool child <2 to 5 years>
13 (child$ or adolescen$).mp.
14 (teenage$ or young people or young person or young adult$).mp.
15 (schoolchildren or school children).mp.
16 (pediatr$ or paediatr$).ti,ab.
17 (boys or girls or youth or youths).mp.
18 10 or 11 or 12 or 13 or 14 or 15 or 16 or 17
19 exp "Behavior-Therapy"/
20 Social Support/
21 Family-Therapy/
22 exp "Psychotherapy-Group"/
23 ((psychological or behavio?r$) adj (therapy or modif$ or strateg$ or intervention$)).mp.
24 (group therapy or family therapy or cognitive therapy).mp.
25 ((lifestyle or life style) adj (chang$ or intervention$)).mp.
26 counsel?ing.mp.
27 social support.mp.
28 (peer adj2 support).mp.
29 ((children adj3 parent$) and therapy).mp.
30 19 or 20 or 21 or 22 or 23 or 24 or 25 or 26 or 27 or 28 or 29
31 exp OBESITY/dt
32 exp Anti-Obesity Agents/
33 lipase inhibitor$.mp.
34 (orlistat or xenical or tetrahydrolipstatin).mp.
35 (appetite adj (suppressant$ or depressant$)).mp.
36 sibutramine.mp. or meridia.ti,ab.
37 (dexfenfluramine or fenfluramine or phentermine).mp.
38 bulking agent$.mp.
39 (methylcellulose or celevac).mp.
40 ((antiobesity or anti obesity) adj (drug$ or agent$)).mp.
41 guar gum.mp.
42 (metformin or glucophage).mp.
43 31 or 32 or 33 or 34 or 35 or 36 or 37 or 38 or 39 or 40 or 41 or 42
44 exp OBESITY/dh
45 "Diet-Fat-Restricted"/

46 "Diet-Reducing"/
47 "Diet-Therapy"/
48 "Fasting"/
49 (diet or diets or dieting).mp.
50 (diet$ adj (modif$ or therapy or intervention$ or strateg$)).mp.
51 (low calorie or calorie control$ or healthy eating).mp.
52 (fasting or modified fast$).mp.
53 exp "Dietary-Fats"/
54 (fruit or vegetable$).mp.
55 (high fat$ or low fat$ or fatty food$).mp.
56 formula diet$.mp.
57 44 or 45 or 46 or 47 or 48 or 49 or 50 or 51 or 52 or 53 or 54 or 55 or 56
58 "Exercise"/
59 "Exercise-Therapy"/
60 exercis$.mp.
61 (aerobics or physical therapy or physical activity or physical inactivity).mp.
62 (fitness adj (class$ or regime$ or program$)).mp.
63 (physical training or physical education).mp.
64 dance therapy.mp.
65 sedentary behavio?r reduction.mp.
66 58 or 59 or 60 or 61 or 62 or 63 or 64 or 65
67 exp OBESITY/su
68 "Surgical-Staplers"/
69 "Surgical-Stapling"/
70 "Lipectomy"/
71 "Gastric-Bypass"/
72 "Gastroplasty"/
73 (dental splinting or jaw wiring).mp.
74 (gastroplasty or gastric band$ or gastric bypass).mp.
75 (intragastric balloon$ or vertical band$).mp.
76 (stomach adj (stapl$ or band$ or bypass)).mp.
77 liposuction.mp.
78 67 or 68 or 69 or 70 or 71 or 72 or 73 or 74 or 75 or 76 or 77
79 exp "Alternative-Medicine"/
80 (alternative medicine or complementary therap$ or complementary medicine).mp.
81 (hypnotism or hypnosis or hypnotherapy).mp.
82 (acupuncture or homeopathy).mp.
83 (chinese medicine or indian medicine or herbal medicine or ayurvedic).mp.
84 79 or 80 or 81 or 82 or 83
85 ((diet or dieting or slim$) adj (club$ or organi?ation$)).mp. g]
86 (weightwatcheR$ or weight watcher$).mp.
87 (correspondence adj (course$ or program$)).mp.
88 (fat camp$ or diet$ camp$).mp.
89 85 or 86 or 87 or 88
90 (family intervention$ or parent$ intervention$).mp.
91 (parent$ adj2 (behavio?r or involve$ or control$ or attitude$ or educat$)).mp.

92 90 or 91
93 exp OBESITY/pc
94 secondary prevention.mp.
95 (preventive measure$ or preventative measure$).mp.
96 (preventive care or preventative care).mp.
97 (obesity adj2 (prevent$ or treat$)).mp.
98 93 or 94 or 95 or 96 or 97
99 (systematic$ review$ or systematic$ overview$).mp.
100 (quantitative$ review$ or quantitative$ overview$).mp.
101 Evidence-Based Medicine/
102 evidence based review$.mp.
103 exp "Controlled-Clinical-Trials"/
104 exp "Research-Design"/
105 ((singl$ or doubl$ or trebl$ or tripl$) adj5 (blind$ or mask$)).mp.
106 (CONTROLLED-CLINICAL-TRIAL or RANDOMIZED CONTROLLED TRIAL or META-ANALYSIS).pt.
107 (control$ and (trial$ or stud$ or evaluation$ or experiment$)).ti,ab.
108 (comparison group$ or control group$).mp.
109 matched pairs.mp.
110 (outcome study or outcome studies).mp.
111 (quasiexperimental or quasi experimental or pseudo experimental).mp.
112 (nonrandomi?ed or non randomi?ed or pseudo randomi?ed).mp.
113 99 or 100 or 101 or 102 or 103 or 104 or 105 or 106 or 107 or 108 or 109 or 110 or 111 or 112
114 9 and 18
115 30 or 43 or 57 or 66 or 78 or 84 or 89 or 92 or 98
116 113 and 114 and 115
117 limit 116 to animals
118 limit 116 to human
119 117 not (117 and 118)
120 116 not 119
121 limit 120 to yr=2001-2004
122 limit 121 to english language

KQ 6: Years searched: 2001-June 4, 2004
1 obesity/
2 obesity in diabetes/
3 obesity, morbid/
4 (obesity or obese).ti,ab.
5 overweight.ti,ab.
6 1 or 2 or 3 or 4 or 5
7 child, preschool/
8 child/
9 adolescent/
10 7 or 8 or 9
11 6 and 10

12 obesity/th
13 obesity in diabetes/th
14 obesity, morbid/th
15 obesity/dt
16 obesity in diabetes/dt
17 obesity, morbid/dt
18 drug therapy.ti,ab.
19 exp anti-obesity agents/
20 (orlistat or xenical or tetrahydrolipstatin).mp.
21 (sibutramine or meridia in ti,ab).mp. [mp=title, abstract, name of substance, mesh subject heading]
22 (metformin or glucophage).mp.
23 (behavior$ therapy or behaviour$ therapy).ti,ab.
24 behavior therapy/
25 (fat camp$ or diet$ camp$).ti,ab.
26 (family intervention$ or parent$ intervention$).ti,ab.
27 ((lifestyle or life style) adj (chang$ or intervention$)).ti,ab.
28 obesity/dh
29 obesity, morbid/dh
30 obesity in diabetes/dh
31 (low calorie or calorie control$).ti,ab.
32 "Diet-Fat-Restricted"/
33 "Diet-Reducing"/
34 "Diet-Therapy"/
35 exercise/
36 physical activity.ti,ab.
37 sedentary behavio?r reduction.ti,ab.
38 obesity/su
39 obesity, morbid/su
40 obesity in diabetes/su
41 gastric bypass/
42 gastroplasty/
43 (gastroplasty or gastric band$ or gastric bypass).ti,ab.
44 weight loss intervention$.ti,ab.
45 diet$ intervention$.ti,ab.
46 12 or 13 or 14 or 15 or 16 or 17 or 18 or 19 or 20 or 21 or 22 or 23 or 24 or 25 or 26 or 27 or 28 or 29 or 30 or 31 or 32 or 33 or 34 or 35 or 36 or 37 or 38 or 39 or 40 or 41 or 42 or 43 or 44 or 45
47 11 and 46
48 ae.fs.
49 adverse effect$.ti,ab.
50 (harm or harms or harmful or harmed).ti,ab.
51 (risky behavior$ or risky behaviour$).ti,ab.
52 to.fs.
53 weight gain/
54 (overeat$ or over$ eat$).ti,ab.

55 weight change$.ti,ab.
56 Weight regain.ti,ab.
57 weight cycling.ti,ab.
58 weight fluctuation$.ti,ab.
59 mo.fs.
60 Mortality/
61 Morbidity/)
62 Postoperative complications/
63 Postoperative complication$.ti,ab.
64 Survival rate/
65 Survival rate.ti,ab.
66 Reoperation/
67 Reoperation.ti,ab.
68 eating disorders/
69 Eating disorder$.ti,ab.
70 anorexia nervosa/
71 Anorexia nervosa.ti,ab.
72 bulimia/
73 Bulimi$.ti,ab.
74 Laxative$.ti,ab.
75 Binging.ti,ab.
76 Purging.ti,ab.
77 depression/
78 depressive disorder/
79 (depression or depressed).ti,ab.
80 anxiety/
81 (anxiety or anxious).ti,ab.
82 suicide/
83 Suicid$.ti,ab.
84 body image/
85 Body image.ti,ab.
86 self esteem.ti,ab.
87 Self concept/
88 quality of life/
89 Quality of life.ti,ab.
90 in.fs.
91 Athletic injuries/
92 School functioning.ti,ab.
93 (Behavior$ problems or behaviour$ problems).ti,ab. (4405)
94 (Growth impairment or Growth retardation or Growth failure or impaired growth).ti,ab. (12511)
95 Growth disorders/ (10322)
96 (linear growth and (stunt$ or retard$)).ti,ab. (235)
97 Growth spurt$.ti,ab. (1096)
98 (Secondary amenorrhea or secondary amenorrhoea).ti,ab. (956)
99 or/48-98 (1816670)

100 47 and 99 (1194)
101 limit 100 to english language (1009)
102 limit 101 to human (1009)

Previously Conducted Systematic Evidence Reviews of Interventions for Childhood Overweight

A large number of reviews related to obesity interventions were located during this process (n=17). Seven of them were review articles that did not meet criteria for systematic reviews,[1-7] such as not stating literature retrieval strategies or inclusion/exclusion criteria or not systematically assessing methodologic quality. One systematic evidence review (SERs) in process at the time of our review shared their list of studies addressing childhood obesity treatments (M. Maglione [maglione@rand.org], e-mail, January 23, 2004). We located six SERs or meta-analyses of treatment; three were rated good[8-10] and two were rated fair due to limiting searching to published studies[11] or to lack of recency.[12] One[13] was rated poor due to including a wide range of short-term, uncontrolled or unrandomized studies and to not formally rating methodologic quality of included studies, as was its follow-up.[14] Two articles were evidence-based guidelines based on systematic reviews we had already located.[15,16] Two other systematic reviews addressing obesity prevention were excluded as outside our scope due to primarily school-based research.[17,18]

In addition to the intervention trials located through our literature searches, we examined all trials (n=18) in the Summerbell review. We also searched the same databases and time period for publications cited as "awaiting assessment". We examined all intervention articles that were not included in the Summerbell SER but were listed in the other five SERs/meta-analyses, were located as part of the ongoing RAND obesity review, or were cited in the two evidence-based guidelines for pediatric obesity treatment.

Appendix E. Previously Conducted Systematic Evidence Reviews of Interventions for Childhood Overweight (continued)

Table E-1. Previously Conducted Systematic Evidence Reviews (SERs) of Interventions for Childhood Overweight

SER Title	Author, Source, Pub Date	End Search Date	Scope and Number of Studies	Relevance to Key Questions	USPSTF Quality Rating
Interventions for treating obesity in children	Summerbell et al 2003[8]	1985-7/2001	Children Treatment: N=18	KQ #4	GOOD
Obesity: diagnosis, prevention, and treatment; evidence based answers to common questions	Reilly et al 2002[9]	1/1981-6/2000	Children Prevention: N=2 Screening: N=16 Treatment: N=3 Prevalence in UK: N=3	KQ #2 KQ #4	GOOD
Physical activity interventions in the prevention and treatment of pediatric obesity: systematic review and critical appraisal	Reilly JJ. In Press. *Proc Nutr Soc.*	6/2000-5/2002	Children Prevention: N=3 Treatment: N= 1 RCT, 1 meta-analysis (Le Mura & Maziekas 2002 – negative quality rating)	KQ #4 (KQ# 3,6 – discusses studies that report adverse effects but does not systematically review)	GOOD
The treatment and prevention of obesity: A systematic review of the literature	Glenny et al 1997[12]	1/1997	Adults and children N=13 studies in children (1 prevention, 12 treatment)	KQ #4	FAIR due to lack of recency
Family involvement in weight control, weight maintenance and weight-loss interventions: a systematic review of randomised trials	McLean et al 2003[11]	Medline® 1966-2000; PsycLIT 1971-2000	Adults and children Treatment studies with family involvement N=8 studies in children or adolescents	KQ #4	FAIR due to limiting searching to published studies
Factors that alter body fat, body mass, and fat-free mass in pediatric obesity	LeMura and Maziekas 2002[13]	1960-2001	Children Treatment studies involving exercise N=7 RCTs, N=6 CCTs, N= 17 uncontrolled studies	KQ #4	POOR due to including a wide range of short-term, uncontrolled, non-randomized studies and not formally rating methodological quality of included studies

Appendix E. Previously Conducted Systematic Evidence Reviews of Interventions for Childhood Overweight (continued)

SER Title	Author, Source, Pub Date	End Search Date	Scope and Number of Studies	Relevance to Key Questions	USPSTF Quality Rating
Follow up exercise studies in paediatric obesity: implications for long term effectiveness	Maziekas et al 2003[14]	1960-2002	Children Treatment studies involving exercise N = 2 RCTs N = 2 CCTs N = 4 uncontrolled studies	KQ #4	POOR due to including a wide range of uncontrolled, non-randomized studies and not formally rating methodological quality of included studies

References

1. Epstein LH, Myers MD, Raynor HA, Saelens BE. Treatment of pediatric obesity. *Pediatrics* 1998; 101(3 Pt 2):554-570.

2. Goldfield GS, Raynor HA, Epstein LH. Treatment of pediatric obesity. In: Wadden TA, Stunkard AJ, editors. *Handbook of Obesity Treatment.* New York: Guilford Press, 2002: 532-555.

3. Jelalian E, Saelens BE. Empirically supported treatments in pediatric psychology: pediatric obesity. *Journal of Pediatric Psychology* 1999; 24(3):223-248.

4. Epstein LH, Coleman KJ, Myers MD. Exercise in treating obesity in children and adolescents. *Med Sci Sports Exerc* 1996; 28(4):428-435.

5. Robinson TN. Behavioural treatment of childhood and adolescent obesity. *International Journal of Obesity & Related Metabolic Disorders* 1999; 23 (Suppl 2):552-557.

6. Jeffery RW, Drewnowski A, Epstein LH, Stunkard AJ, Wilson GT, Wing RR et al. Long-term maintenance of weight loss: current status. *Health Psychol* 2000; 19(1 Suppl):5-16.

7. Yanovski JA. Intensive therapies for pediatric obesity. *Pediatric Clinics of North America* 2001; 48(4):1041-1053.

8. Summerbell CD, Ashton V, Campbell KJ, Edmunds L, Kelly S, Waters E. Interventions for treating obesity in children [Systematic Review]. *Cochrane Database of Systematic Reviews* 2003;(3):CD001872.

9. Reilly JJ, Wilson ML, Summerbell CD, Wilson DC. Obesity: diagnosis, prevention, and treatment; evidence based answers to common questions. *Arch Dis Child* 2002; 86(6):392-394.

10. Reilly JJ, McDowell ZC. Physical activity interventions in the prevention and treatment of paediatric obesity: systematic review and critical appraisal. *Proceedings of the Nutrition Society* 2003; 62(3):611-619.

11. McLean N, Griffin S, Toney K, Hardeman W. Family involvement in weight control, weight maintenance and weight-loss interventions: a systematic review of randomised trials. *International Journal of Obesity & Related Metabolic Disorders* 2003; 27(9):987-1005.

12. Glenny AM, O'Meara S, Melville A, Sheldon TA, Wilson C. The treatment and prevention of obesity: a systematic review of the literature. *Int J Obes Relat Metab Disord* 1997; 21(9):715-737.

13. LeMura LM, Maziekas MT. Factors that alter body fat, body mass, and fat-free mass in pediatric obesity. *Medicine & Science in Sports & Exercise* 2002; 34(3):487-496.

14. Maziekas MT, LeMura LM, Stoddard NM, Kaercher S, Martucci T. Follow up exercise studies in paediatric obesity: implications for long term effectiveness. *British Journal of Sports Medicine* 2003; 37(5):425-429.

15. Scottish Intercollegiate Guidelines Network. *Management of Obesity in Children and Young People. A National Clinical Guideline.* 2003. Edinburgh, Scottish Intercollegiate Guidelines Network.

16. The prevention and treatment of childhood obesity. *Effective Health Care 2002;* 7(6):1-11 (67 ref).

17. Campbell K, Waters E, O'Meara S, Kelly S, Summerbell C. Interventions for preventing obesity in children.[update of Cochrane Database Syst Rev. 2001;(3):CD001871; PMID: 11686999]. *Cochrane Database of Systematic Reviews* 2002;(2):CD001871.

18. Jerum A, Melnyk BM. Effectiveness of interventions to prevent obesity and obesity-related complications in children and adolescents. *Pediatr Nurs* 2001; 27(6):606-610.

Appendix F. Inclusion Criteria and Results of Searches

Key Questions	Inclusion Criteria	# of Abstracts* Reviewed	Articles reviewed from Abstracts and Outside Sources	# of Articles Meeting Inclusion Criteria
KQ1: Is there direct evidence that screening for overweight in childhood improves age-appropriate behavioral or physiologic measures, or health outcomes?	English abstract; RCT or CCT; US population; Age ≥2 and ≤18; Primary care feasible screening test; Acceptable reference standard; met USPSTF quality grade of good or fair.	2162	0	0
KQ2a: What are appropriate standards for overweight in childhood and what is the prevalence of overweight based on these?	Same as KQ1; for prevalence, representative sample or underreported special populations	2162	69	5
KQ2b: What clinical screening tests for overweight in childhood are reliable and valid in predicting obesity in adulthood? **KQ2c:** What clinical screening tests for overweight in childhood are reliable and valid in predicting poor health outcomes in adulthood?	English, US population; examine the relationship between overweight in childhood and adult health status relating to overweight; used primary care feasible measures; longitudinal studies; children aged 2-17, adults aged 18 or older; clear definition of childhood overweight measures and status; using adult measure of health status; clear definition of adult disease/health status, or overweight status, controlled for appropriate confounders (age, sex); clearly defined cohort; all examined at the same stage (age group) used representative population standard as comparison; met USPSTF quality grade of good or fair.	2162	284	36
KQ3: What are the adverse effects of screening, including labeling? Is screening acceptable to patients?	Study includes a screening or intervention component, delivered by a professional; study explicitly evaluates or discusses harms; Screen/intervention is one that has been included in report; Population 2-18 or sub-group analysis for this age group; Meets study quality criteria	312	9	0

Appendix F. Inclusion Criteria and Results of Searches (continued)

KQ4: Do weight control interventions lead to improved intermediate outcomes, including behavioral, physiologic or weight-related measures? **KQ5:** Do weight control interventions lead to improved health outcomes, including decreased morbidity, and/or improved functioning (school attendance, self-esteem and other psychosocial indicators)?	English; RCT; US or similarly industrialized countries; age ≥2 and ≤18 with or without family; not specific patient groups such as disease specific populations; enrolled children who are overweight/at-risk for overweight; delivered by any professional; primary care feasible; intervention treats obesity; ≥ 6 mo. follow-up; report baseline and post-intervention measures of weight; clear definition of overweight/at-risk for overweight; good or fair USPSTF quality grade.	949	198	22 3
KQ6: What are the adverse effects of interventions? Are interventions acceptable to patients?	Study includes a screening or intervention component, delivered by a professional; study explicitly evaluates or discusses harms; Screen/intervention is one that has been included in report; Longitudinal; Population 2-18 or sub-group analysis for this age group; Meets study quality criteria	864	27	4
KQ7: Are improvements in intermediate outcomes associated with improved health outcomes? (Only evaluated if there is no direct evidence for KQ1 or KQ5 and if there is sufficient evidence for KQ4.)				

*All abstracts were reviewed for applicability to other key questions.

Appendix G. Exclusion Table for Key Questions 2b and 2c (continued)

Reference	Reason for Exclusion
Abraham S; Nordsieck M. Relationship of excess weight in children and adults. *Public Health Rep* 1960:75;263-73.	Unusable reference standard
Abraham S.; Collins G.; Nordsieck M. Relationship of childhood weight status to morbidity in adults. *HSMHA Health Rep* 1971:86 (3);273-284.	Unusable reference standard Poor quality rating
Abrantes MM; Lamounier JA; Colosimo EA. Comparison of body mass index values proposed by Cole et al. (2000) and Must et al. (1991) for identifying obese children with weight-for-height index recommended by the World Health Organization. *Public Health Nutrition.* 2003:6(3);307-311.	Non-US population
Allison DB; Zannolli R; Faith MS; Heo M; Pietrobelli A; VanItallie TB; Pi-Sunyer FX; Heymsfield SB. Weight loss increases and fat loss decreases all-cause mortality rate: results from two independent cohort studies. *International Journal of Obesity & Related Metabolic Disorders.* 1999:23 (6);603-611.	Wrong age group
Aristimuno GG; Foster TA; Voors AW; Srinivasan SR; Berenson GS. Influence of persistent obesity in children on cardiovascular risk factors: the Bogalusa Heart Study. *Circulation* 1984:69(5);895-904.	Wrong age group Not an outcome of interest
Arslanian S. Insulin secretion and sensitivity in healthy African-American vs American white children. *Clin Pediatr(Phila).* 1998:37(2);81-88.	Not a longitudinal study Not an outcome of interest Wrong measure of OW
Arslanian S. Type 2 diabetes in children: clinical aspects and risk factors. *Hormone Research.* 2002:57 Suppl 1;19-28.	Not a longitudinal study
Bao W.; Srinivasan SR; Valdez R; Greenlund KJ; Wattigney WA; Berenson GS. Longitudinal changes in cardiovascular risk from childhood to young adulthood in offspring of parents with coronary artery disease: the Bogalusa Heart Study.[comment]. *JAMA.* 1997:278 (21);1749-1754.	Wrong measure of OW
Bao W; Srinivasan SR; Wattigney WA; Berenson GS. Persistence of multiple cardiovascular risk clustering related to syndrome X from childhood to young adulthood. The Bogalusa Heart Study. *Arch Intern Med* 1994:154 (16);1842-1847.	Poor data on obesity tracking Wrong age group
Bao W; Srinivasan SR; BerensonGS. Persistent elevation of plasma insulin levels is associated with increased cardiovascular risk in children and young adults. The Bogalusa Heart Study. *Circulation.* 1996:93 (1);54-59.	No report of childhood weight
Barlow SE; Dietz WH. Obesity evaluation and treatment: Expert Committee recommendations. The Maternal and Child Health Bureau, Health Resources and Services Administration and the Department of Health and Human Services. *Pediatrics* 1998:102 (3);E29.	Not a longitudinal study
Barnekow-Bergkvist M; Hedberg G; Janlert U; Jansson E. Adolescent determinants of cardiovascular risk factors in adult men and women. *Scandinavian Journal of Public Health.* 2001:29(3);208-217.	Non-US population
Belamarich PF; Luder E; Kattan M; Mitchell H; Islam S; Lynn H; Crain EF. Do obese inner-city children with asthma have more symptoms than nonobese children with asthma? *Pediatrics* 2000:106 (6);1436-1441.	Not a longitudinal study
Berenson GS; Wattigney WA; Tracy RE; Newman WP III; Srinivasan SR; Webber LS; Dalferes ER Jr.; Strong JP. Atherosclerosis of the aorta and coronary arteries and cardiovascular risk factors in persons aged 6 to 30 years and studied at necropsy (The Bogalusa Heart Study). *Am J Cardiol* 1992:70(9);851-858.	Age at BMI measurement not reported

Appendix G. Exclusion Table for Key Questions 2b and 2c (continued)

Berenson GS, Srinivasan SR, Bao W, Newman WP 3rd, Tracy RE, Wattigney WA. Association between multiple cardiovascular risk factors and atherosclerosis in children and young adults. The Bogalusa Heart Study. N Engl J Med. 1998 Jun 4;338(23):1650-1656.	Age at BMI measurement not reported
Bhargava SK; Sachdev HS; Fall CH; Osmond C; Lakshmy R; Barker DJ; Biswas SK; Ramji S; Prabhakaran D; Reddy KS. Relation of serial changes in childhood body-mass index to impaired glucose tolerance in young adulthood. *New England Journal of Medicine.* 2004:350(9);865-875.	Non-US population
Biro FM; Lucky AW; Simbartl LA; Barton BA; Daniels SR; Striegel-Moore R; Kronsberg SS; Morrison JA. Pubertal maturation in girls and the relationship to anthropometric changes: pathways through puberty. *Journal of Pediatrics.* 2003:142(6);643-646.	Wrong measure of OW
Blackett PR; Taylor T; Russell D; Lu M; Fesmire J; Lee ET. Lipoprotein changes in relation to body mass index in Native American adolescents. *Pediatric Research.* 1996:40(1);77-81.	Not a longitudinal study
Bognetti E; Macellaro P; Novelli D; Meschi F; Ciralli F; Chiumello G. Prevalence and correlates of obesity in insulin dependent diabetic patients. *Archives of Disease in Childhood.* 1995:73(3);239-242.	Non-US population
Boxer GH; Bauer AM; Miller BD. Obesity-hypoventilation in childhood. *J Am Acad Child Adolesc Psychiatry* 1988:27(5);552-558.	Not a longitudinal study Limited to a specific disease
Braddon FE; Rodgers B; Wadsworth ME; Davies JM. Onset of obesity in a 36 year birth cohort study. *Br Med J (Clin Res Ed)* 1986:293(6542);299-303.	Non-US population
Bray GA. Overweight is risking fate. Definition, classification, prevalence, and risks. *Ann N Y Acad Sci* 1987:499;14-28.	Not a longitudinal study
Briend A, Zimicki S. Validation of arm circumference as an indicator of risk of death in one to four year old children. *Nutrition Research* 1986: 6;249-261.	Non-US population
Bringer J, Lefebvre P, Boulet F, Grigorescu F, Renard E, Hedon B et al. Body composition and regional fat distribution in polycystic ovarian syndrome. Relationship to hormonal and metabolic profiles. *Ann N Y Acad Sci* 1993: 687;115-123.	Non-US population
Brinton LA, Swanson CA. Height and weight at various ages and risk of breast cancer. *Annals of Epidemiology* 1992: 2(5);597-609.	Wrong measure of OW
Britz B, Siegfried W, Ziegler A, Lamertz C, Herpertz-Dahlmann BM, Remschmidt H et al. Rates of psychiatric disorders in a clinical study group of adolescents with extreme obesity and in obese adolescents ascertained via a population based study. *International Journal of Obesity & Related Metabolic Disorders* 2000: 24(12);1707-1714.	Non-US population
Burns TL, Moll PP, Lauer RM. The relation between ponderosity and coronary risk factors in children and their relatives. The Muscatine Ponderosity Family Study. *American Journal of Epidemiology* 1989: 129(5);973-987.	Wrong measure of OW
Byrnes SE, Baur LA, Bermingham M, Brock K, Steinbeck K. Leptin and total cholesterol are predictors of weight gain in pre-pubertal children. *International Journal of Obesity & Related Metabolic Disorders* 1999: 23(2);146-150.	Non-US population
Campbell K, Waters E, O'Meara S, Summerbell C. Interventions for preventing obesity in childhood. A systematic review. *Obesity Reviews* 2001: 2(3);149-157.	Non-US population
Caprio S, Hyman LD, Limb C, McCarthy S, Lange R, Sherwin RS et al. Central adiposity and its metabolic correlates in obese adolescent girls. *Am J Physiol* 1995: 269(1 Pt 1);E118-E126.	Not a longitudinal study Wrong measure of OW
Caprio S, Bronson M, Sherwin RS, Rife F, Tamborlane WV. Co-existence of severe insulin resistance and hyperinsulinaemia in pre-adolescent obese children. *Diabetologia* 1996: 39(12);1489-1497.	Wrong measure of OW Not a longitudinal study

Caprio S, Hyman LD, McCarthy S, Lange R, Bronson M, Tamborlane WV. Fat distribution and cardiovascular risk factors in obese adolescent girls: importance of the intraabdominal fat depot. *Am J Clin Nutr* 1996: 64(1Jul);12-17.	Not a longitudinal study
Casey VA, Dwyer JT, Berkey CS, Bailey SM, Coleman KA, Valadian I. The distribution of body fat from childhood to adulthood in a longitudinal study population. *Ann Hum Biol* 1994: 21(1);39-55.	No BMI tracking data
Charney E, Goodman HC, McBride M, Lyon B, Pratt R. Childhood antecedents of adult obesity. Do chubby infants become obese adults? *New England Journal of Medicine* 1976: 295(1);6-9.	Wrong age group
Chen W, Srinivasan SR, Elkasabany A, Berenson GS. Cardiovascular risk factors clustering features of insulin resistance syndrome (Syndrome X) in a biracial (black-white) population of children, adolescents, and young adults: the Bogalusa Heart Study. *Am J Epidemiol* 1999: 150(7);667-674.	Not a longitudinal study
Chen Y, Dales R, Krewski D, Breithaupt K. Increased effects of smoking and obesity on asthma among female Canadians: the National Population Health Survey, 1994-1995. *Am J Epidemiol* 1999: 150(3);255-262.	Not a longitudinal study Wrong age OW measure
Chervin RD, Clarke DF, Huffman JL, Szymanski E, Ruzicka DL, Miller V et al. School performance, race, and other correlates of sleep-disordered breathing in children. *Sleep Medicine* 2003: 4(1);21-27.	Not a longitudinal study
Chinn S. Obesity and asthma: evidence for and against a causal relation. *Journal of Asthma* 2003: 40(1);1-16.	Non-US population
Chinn S, Rona RJ. Can the increase in body mass index explain the rising trend in asthma in children? *Thorax* 2001: 56(11);845-850.	Non-US population
Chu NF, Rimm EB, Wang DJ, Liou HS, Shieh SM. Clustering of cardiovascular disease risk factors among obese schoolchildren: the Taipei Children Heart Study. *American Journal of Clinical Nutrition* 1998: 67(6);1141-1146.	Non-US population
Chu NF, Wang DJ, Shieh SM, Rimm EB. Plasma leptin concentrations and obesity in relation to insulin resistance syndrome components among school children in Taiwan--The Taipei Children Heart Study. *International Journal of Obesity & Related Metabolic Disorders* 2000: 24(10);1265-1271.	Non-US population
Clarke,W.R.; Woolson,R.F.; Lauer,R.M. Changes in ponderosity and blood pressure in childhood: the Muscatine Study. *Am J Epidemiol* 1986:124 (2);195-206.	Wrong age group
Cook DG, Mendall MA, Whincup PH, Carey IM, Ballam L, Morris JE et al. C-reactive protein concentration in children: relationship to adiposity and other cardiovascular risk factors. *Atherosclerosis* 2000: 149(1);139-150.	Non-US population
Cowell JM, Montgomery AC, Talashek M. Cardiovascular risk stability: from grade school to high school. *Journal of Pediatric Health Care* 1992: 6(6);349-354.	Wrong measure of OW Wrong age group
Cowin I, Emmett P. Cholesterol and triglyceride concentrations, birthweight and central obesity in pre-school children. ALSPAC Study Team. Avon Longitudinal Study of Pregnancy and Childhood. *Int J Obes Relat Metab Disord* 2000: 24(3);330-339.	Non-US population
Cronk CE, Roche AF, Kent R, Berkey C, Reed RB, Valadian I et al. Longitudinal trends and continuity in weight/stature from 3 months to 18 years. *Hum Biol* 1982: 54(4);729-749.	Trajectory study
Dai S, Labarthe DR, Grunbaum JA, Harrist RB, Mueller WH. Longitudinal analysis of changes in indices of obesity from age 8 years to age 18 years. Project HeartBeat! *American Journal of Epidemiology* 2002: 156(8);720-729.	Normative data only Trajectory study
Daniels SR, Kimball TR, Morrison JA, Khoury P, Witt S, Meyer RA. Effect of lean body mass, fat mass, blood pressure, and sexual maturation on left ventricular mass in children and adolescents. Statistical, biological, and clinical significance. *Circulation* 1995: 92(11);3249-3254.	Not a longitudinal study

Daniels SR, Obarzanek E, Barton BA, Kimm SY, Similo SL, Morrison JA. Sexual maturation and racial differences in blood pressure in girls: the National Heart, Lung, and Blood Institute Growth and Health Study. *Journal of Pediatrics* 1996: 129(2);208-213.	Not a longitudinal study
Datar A, Sturm R, Magnabosco JL. Childhood overweight and academic performance: national study of kindergartners and first-graders 4904. *Obesity Research.* 2004:12 (1);58-68.	No adult outcomes
Davis SP, Arthur C, Davis M, Goldberg D, Moll G, Davis G. Assessing cardiovascular risk in children: the Jackson, Mississippi CRRIC Study. *Journal of Cultural Diversity* 2002: 9(3);67-72.	Not a longitudinal study
Dietz WH. Childhood weight affects adult morbidity and mortality. *Journal of Nutrition* 1998: 128(2 Suppl);411S-414S.	Not a longitudinal study
Dietz WH. Overweight in childhood and adolescence. *N Engl J Med* 2004: 350(9);855-857.	Not a longitudinal study
DiPietro L, Mossberg HO, Stunkard AJ. A 40-year history of overweight children in Stockholm: life-time overweight, morbidity, and mortality. *International Journal of Obesity & Related Metabolic Disorders* 1994: 18(9);585-590.	Non-US population
Douglas MB, Birrer RB, Medidi S, Schlussel YR. Obese children should be screened for hypercholesterolemia. *Journal of Health Care for the Poor & Underserved* 1996:7(1);24-35.	Not a longitudinal study
Dowling AM, Steele JR, Baur LA. Does obesity influence foot structure and plantar pressure patterns in prepubescent children? *International Journal of Obesity & Related Metabolic Disorders* 2001: 25(6);845-852.	Non-US population
Dyer AR, Stamler J, Garside DB, Greenland P. Long-term consequences of body mass index for cardiovascular mortality: The Chicago Heart Association Detection Project in Industry Study. *Ann Epidemiol* 2003:14;101-108.	Wrong age group
Ebbeling CB, Pawlak DB, Ludwig DS. Childhood obesity: public-health crisis, common sense cure. *Lancet* 2002: 360(9331);473-482.	Not a longitudinal study
Ellis KJ, Abrams SA, Wong WW. Monitoring childhood obesity: assessment of the weight/height index. *American Journal of Epidemiology* 1999: 150(9);939-946.	Not a longitudinal study
Engeland A, Bjorge T, Sogaard AJ, Tverdal A. Body mass index in adolescence in relation to total mortality: 32-year follow-up of 227,000 Norwegian boys and girls. *Am J Epidemiol* 2003: 157(6);517-523.	Non-US population
Erickson SJ, Robinson TN, Haydel KF, Killen JD. Are overweight children unhappy?: Body mass index, depressive symptoms, and overweight concerns in elementary school children.[comment]. *Archives of Pediatrics & Adolescent Medicine* 2000: 154(9);931-935.	Not a longitudinal study
Eriksson JG, Forsen T, Tuomilehto J, Osmond C, Barker DJ. Early adiposity rebound in childhood and risk of Type 2 diabetes in adult life. *Diabetologia* 2003: 46(2);190-194.	Non-US population
Ferraro KF, Thorpe RJ, Jr., Wilkinson JA. The life course of severe obesity: does childhood overweight matter? *Journals of Gerontology Series B-Psychological Sciences & Social Sciences* 2003: 58(2);S110-S119.	Wrong measure of OW
Figueroa-Munoz JI, Chinn S, Rona RJ. Association between obesity and asthma in 4-11 year old children in the UK. *Thorax* 2001: 56(2);133-137.	Non-US population
Ford ES, Galuska DA, Gillespie C, Will JC, Giles WH, Dietz WH. C-reactive protein and body mass index in children: findings from the Third National Health and Nutrition Examination Survey, 1988-1994. *J Pediatr* 2001 Apr:138(4);486-92.	Not a longitudinal study
Freedman DS, Srinivasan SR, Burke GL, Shear CL, Smoak CG, Harsha DW et al. Relation of body fat distribution to hyperinsulinemia in children and adolescents: the Bogalusa Heart Study. *Am J Clin Nutr* 1987: 46(3);403-410.	Not a longitudinal study

Appendix G. Exclusion Table for Key Questions 2b and 2c (continued)

Freedman DS, Srinivasan SR, Harsha DW, Webber LS, Berenson GS. Relation of body fat patterning to lipid and lipoprotein concentrations in children and adolescents: the Bogalusa Heart Study. *Am J Clin Nutr* 1989: 50(5);930-939.	Not a longitudinal study
Freedman DS; Srinivasan SR; Valdez RA; Williamson DF; Berenson GS. Secular increases in relative weight and adiposity among children over two decades: the Bogalusa Heart Study. *Pediatrics.* 1997:99 (3);420-426.	Not a longitudinal study
Freedman DS, Dietz WH, Srinivasan SR, Berenson GS. The relation of overweight to cardiovascular risk factors among children and adolescents: the Bogalusa Heart Study. *Pediatrics* 1999: 103(6 Pt 1);1175-1182.	Not a longitudinal study
Freedman DS, Serdula MK, Srinivasan SR, Berenson GS. Relation of circumferences and skinfold thicknesses to lipid and insulin concentrations in children and adolescents: the Bogalusa Heart Study. *Am J Clin Nutr* 1999: 69(2);308-317.	Not a longitudinal study
Freedman DS, Dietz WH, Tang R, Mensah GA, Bond MG, Urbina EM et al. The relation of obesity throughout life to carotid intima-media thickness in adulthood: the Bogalusa Heart Study. *International Journal of Obesity & Related Metabolic Disorders* 2004: 28(1);159-166.	Not an outcome of interest
Friedman MA, Brownell KD. Psychological correlates of obesity: moving to the next research generation. *Psychol Bull* 1995: 117(1);3-20.	Not a longitudinal study
Fung KP, Lee J, Lau SP, Chow OK, Wong TW, Davis DP. Properties and clinical implications of body mass indices. *Archives of Disease in Childhood* 1990: 65(5);516-519.	Non-US population
Garn SM, Clark DC. Trends in fatness and the origins of obesity Ad Hoc Committee to Review the Ten-State Nutrition Survey. *Pediatrics* 1976: 57(4);443-456.	Wrong measure of OW
Garn SM, Cole PE. Do the obese remain obese and the lean remain lean? Am J Public Health. 1980 Apr:70(4);351-2.	Not a longitudinal study
Garn SM, Pilkington JJ, Lavelle M. Relationship between initial fatness level and long-term fatness change. *Ecology of Food and Nutrition* 1984: 14;85-92.	No clear age at OW measurement
Gasperino J. Ethnic differences in body composition and their relation to health and disease in women. *Ethnicity & Health* 1996: 1(4);337-347.	Not a longitudinal study Wrong age group
Gasser T, Ziegler P, Seifert B, Molinari L, Largo RH, Prader A. Prediction of adult skinfolds and body mass from infancy through adolescence. *Annals of Human Biology* 1995: 22(3);217-233.	Non-US population
Giacchi M, Mattei R, Rossi S. Correction of the self-reported BMI in a teenage population. *International Journal of Obesity & Related Metabolic Disorders* 1998: 22(7);673-677.	Not a longitudinal study
Gidding SS, Bao W, Srinivasan SR, Berenson GS. Effects of secular trends in obesity on coronary risk factors in children: the Bogalusa Heart Study. *Journal of Pediatrics* 1995: 127(6);868-874.	Wrong outcome measure
Gidding SS, Leibel RL, Daniels S, Rosenbaum M, Van Horn L, Marx GR. Understanding obesity in youth. A statement for healthcare professionals from the Committee on Atherosclerosis and Hypertension in the Young of the Council on Cardiovascular Disease in the Young and the Nutrition Committee, American Heart Association. Writing Group. *Circulation* 1996: 94(12);3383-3387.	Not a longitudinal study
Gillum RF, Taylor HL, Brozek J, Polansky P, Blackburn H. Indices of obesity and blood pressure in young men followed 32 years. *J Chronic Dis* 1982: 35(3);211-219.	Wrong age group
Golan M, Weizman A. Reliability and validity of the Family Eating and Activity Habits Questionnaire. *European Journal of Clinical Nutrition* 1998: 52(10);771-777.	No link between OW and subsequent health measures

Appendix G. Exclusion Table for Key Questions 2b and 2c (continued)

Goodman E, Adler NE, Daniels SR, Morrison JA, Slap GB, Dolan LM. Impact of objective and subjective social status on obesity in a biracial cohort of adolescents. *Obesity Research* 2003: 11(8);1018-1026.	Not a longitudinal study
Goran MI. Measurement issues related to studies of childhood obesity: assessment of body composition, body fat distribution, physical activity, and food intake. *Pediatrics* 1998: 101(3 Pt 2);505-518.	Not a longitudinal study
Gortmaker SL, Dietz WH, Jr., Sobol AM, Wehler CA. Increasing pediatric obesity in the United States. *Am J Dis Child* 1987: 141(5);535-540.	Not a longitudinal study
Grilo CM, Wilfley DE, Brownell KD, Rodin J. Teasing, body image, and self-esteem in a clinical sample of obese women. *Addict Behav* 1994: 19(4);443-450.	Wrong age group Not a longitudinal study
Gunnell DJ, Frankel SJ, Nanchahal K, Peters TJ, Davey SG. Childhood obesity and adult cardiovascular mortality: a 57-y follow-up study based on the Boyd Orr cohort. *American Journal of Clinical Nutrition* 1998: 67(6);1111-1118.	Non-US population
Gutin B, Owens S, Treiber F, Islam S, Karp W, Slavens G. Weight-independent cardiovascular fitness and coronary risk factors. *Arch Pediatr Adolesc Med* 1997: 151(5);462-465.	Not a longitudinal study Wrong measure of OW
Gutin B, Treiber F, Owens S, Mensah GA. Relations of body composition to left ventricular geometry and function in children. *J Pediatr* 1998: 132(6);1023-1027.	Not a longitudinal study
Hales CN, Barker DJ, Clark PM, Cox LJ, Fall C, Osmond C et al. Fetal and infant growth and impaired glucose tolerance at age 64. *BMJ* 1991: 303(6809);1019-1022.	Non-US population
Halpern CT, Udry JR, Campbell B, Suchindran C. Effects of body fat on weight concerns, dating, and sexual activity: a longitudinal analysis of black and white adolescent girls. *Developmental Psychology* 1999: 35(3);721-736.	Not an outcome of interest
Hansson LE, Nyren O, Bergstrom R, Wolk A, Lindgren A, Baron J et al. Nutrients and gastric cancer risk. A population-based case-control study in Sweden. *Int J Cancer* 1994: 57(5);638-644.	Non-US population
Hardy R, Wadsworth M, Kuh D. The influence of childhood weight and socioeconomic status on change in adult body mass index in a British national birth cohort. *Int J Obes Relat Metab Disord* 2000: 24(6);725-734.	Non-US population
Hartz AJ, Rimm AA. Natural history of obesity in 6,946 women between 50 and 59 years of age. *Amer J Pub Health*. 1980:70(4);385-8.	Wrong measure of OW
He Q, Karlberg J. Prediction of adult overweight during the pediatric years. *Pediatric Research* 1999: 46(6);697-703.	Non-US population
He Q, Karlberg J. Probability of adult overweight and risk change during the BMI rebound period. *Obesity Research* 2002: 10(3);135-140.	Non-US population
He Q, Ding ZY, Fong DY, Karlberg J. Blood pressure is associated with body mass index in both normal and obese children. *Hypertension* 2000: 36(2);165-170.	Non-US population
Health implications of obesity. National Institutes of Health Consensus Development Conference. 11-13 February 1985. *Ann Intern Med.* 1985 Dec:103(6 (Pt 2));1073-1077.	Wrong age group Not a longitudinal study
Higgins PB, Gower BA, Hunter GR, Goran MI. Defining health-related obesity in prepubertal children. *Obes Res* 2001: 9(4);233-240.	Not a longitudinal study
Himes JH, Dietz WH. Guidelines for overweight in adolescent preventive services: recommendations from an expert committee. The Expert Committee on Clinical Guidelines for Overweight in Adolescent Preventive Services. *Am J Clin Nutr* 1994: 59(2);307-316.	Not an outcome of interest
Himes JH, Roche AF. Subcutaneous fatness and stature: relationship from infancy to adulthood. *Hum Biol* 1986: 58(5);737-750.	Wrong outcome measure Wrong measure of OW
Hoffmans MD, Kromhout D, de Lezenne Coulander C. The impact of body mass index of 78,612 18-year old Dutch men on 32-year mortality from all causes. J Clin Epidemiol. 1988:41(8);749-56.	Non-US population

Appendix G. Exclusion Table for Key Questions 2b and 2c (continued)

Hoffmans MD, Kromhout D, Coulander CD. Body Mass Index at the age of 18 and its effects on 32-year-mortality from coronary heart disease and cancer. A nested case-control study among the entire 1932 Dutch male birth cohort. *J Clin Epidemiol* 1989: 42(6);513-520.	Non-US population
Holbrook TL, Barrett-Connor E, Wingard DL. The association of lifetime weight and weight control patterns with diabetes among men and women in an adult community. *Int J Obes* 1989: 13(5);723-729.	Wrong age group
Hwang HK, Chen MR, Lee YJ, Lee HC, Huang CY, Kao HA et al. Metabolic disturbance in obese children: glucose, insulin levels and lipid profile. *Acta Paediatrica Taiwanica* 2001: 42(2);75-80.	Non-US population
Hypponen E, Virtanen SM, Kenward MG, Knip M, Akerblom HK, Childhood Diabetes in Finland Study Group. Obesity, increased linear growth, and risk of type 1 diabetes in children. *Diabetes Care* 2000: 23(12);1755-1760.	Non-US population
Hypponen E, Power C, Smith GD. Prenatal growth, BMI, and risk of type 2 diabetes by early midlife. *Diabetes Care*. 2003 Sep:26(9);2512-7.	Non-US population
Israel AC, Ivanova MY. Global and dimensional self-esteem in preadolescent and early adolescent children who are overweight: age and gender differences. *Int J Eat Disord* 2002: 31(4);424-429.	Not a longitudinal study
Jedrychowski W, Maugeri U, Flak E, Mroz E, Bianchi I. Predisposition to acute respiratory infections among overweight preadolescent children: an epidemiologic study in Poland. *Public Health* 1998: 112(3);189-195.	Non-US population
Johnson JG, Cohen P, Kasen S, Brook JS. Childhood adversities associated with risk for eating disorders or weight problems during adolescence or early adulthood. *American Journal of Psychiatry* 2002: 159(3);394-400.	Wrong topic area
Kaplan TA, Digel SL, Scavo VA, Arellana SB. Effect of obesity on injury risk in high school football players. *Clinical Journal of Sport Medicine* 1995: 5(1);43-47.	Limited to specific group Not an outcome of interest
Kawabe H, Shibata H, Hirose H, Tsujioka M, Saito I, Saruta T. Determinants for the development of hypertension in adolescents. A 6-year follow-up. *Journal of Hypertension* 2000: 18(11);1557-1561.	Non-US population
Kelly JL, Stanton WR, McGee R, Silva PA. Tracking relative weight in subjects studied longitudinally from ages 3 to 13 years. *J Paediatr Child Health* 1992: 28(2);158-161.	Non-US population
Kempers KG, Foote JW, DiFlorio-Brennan T. Obesity: prevalence and considerations in oral and maxillofacial surgery. *Journal of Oral & Maxillofacial Surgery* 2000: 58(2);137-143.	Not a longitudinal study
Kikuchi DA, Srinivasan SR, Harsha DW, Webber LS, Sellers TA, Berenson GS. Relation of serum lipoprotein lipids and apolipoproteins to obesity in children: the Bogalusa Heart Study. *Preventive Medicine* 1992: 21(2);177-190	Not a longitudinal study
Klish WJ. Childhood obesity. *Pediatr Rev* 1998: 19(9);312-315.	Not a longitudinal study
Koplan JP, Dietz WH. Caloric imbalance and public health policy. *JAMA* 1999: 282(16);1579-1581.	Not a longitudinal study
Labarthe DR, Mueller WH, Eissa M. Blood pressure and obesity in childhood and adolescence. Epidemiologic aspects. *Annals of Epidemiology* 1991:1(4);337-345.	Not a longitudinal study
Lai SW, Ng KC, Lin HF, Chen HL. Association between obesity and hyperlipidemia among children. *Yale Journal of Biology & Medicine* 2001: 74(4);205-210.	Non-US population
Laitinen J, Power C, Jarvelin MR. Family social class, maternal body mass index, childhood body mass index, and age at menarche as predictors of adult obesity. *American Journal of Clinical Nutrition* 2001: 74(3);287-294.	Non-US population
Lake JK, Power C, Cole TJ. Child to adult body mass index in the 1958 British birth cohort: associations with parental obesity. *Archives of Disease in Childhood* 1997: 77(5);376-381.	Wrong measure of OW

Lake JK, Power C, Cole TJ. Women's reproductive health: the role of body mass index in early and adult life. *Int J Obes Relat Metab Disord* 1997: 21(6);432-438.	Non-US population
Laskarzewski P, Morrison JA, Mellies MJ, Kelly K, Gartside PS, Khoury P et al. Relationships of measurements of body mass to plasma lipoproteins in schoolchildren and adults. *Am J Epidemiol* 1980: 111(4);395-406.	Not a longitudinal study
Lauer RM, Lee J, Clarke WR. Factors affecting the relationship between childhood and adult cholesterol levels: the Muscatine Study. *Pediatrics* 1988: 82(3);309-318.	Same data as Lauer 89
Lauer RM, Lee J, Clarke WR. Predicting adult cholesterol levels from measurements in childhood and adolescence: the Muscatine Study. *Bulletin of the New York Academy of Medicine* 1989: 65(10);1127-1142.	Wrong measure of OW
Lauer RM, Burns TL, Clarke WR, Mahoney LT. Childhood predictors of future blood pressure. Hypertension. 1991 Sep:18(3 Suppl);I74-81.	Not a longitudinal study Wrong measure of OW
Laurier D, Guiguet M, Chau NP, Wells JA, Valleron AJ. Prevalence of obesity: a comparative survey in France, the United Kingdom and the United States. *International Journal of Obesity & Related Metabolic Disorders* 1992: 16(8);565-572.	Not a longitudinal study Wrong age group
Lazarus R, Colditz G, Berkey CS, Speizer FE. Effects of body fat on ventilatory function in children and adolescents: cross-sectional findings from a random population sample of school children. *Pediatric Pulmonology* 1997: 24(3);187-194.	Non-US population
Le Stunff,C.; Bougneres,P. Early changes in postprandial insulin secretion, not in insulin sensitivity, characterize juvenile obesity. *Diabetes* 1994:43 (5);696-702.	Non-US population
Lin-Su K, Vogiatzi MG, New MI. Body mass index and age at menarche in an adolescent clinic population. *Clinical Pediatrics* 2002: 41(7);501-507.	Not a longitudinal study
Lissau-Lund-Sorensen I, Sorensen TI. Prospective study of the influence of social factors in childhood on risk of overweight in young adulthood. *International Journal of Obesity & Related Metabolic Disorders* 1992: 16(3);169-175.	Non-US population
Lissner L, Odell PM, D'Agostino RB, Stokes J, III, Kreger BE, Belanger AJ et al. Variability of body weight and health outcomes in the Framingham population. *N Engl J Med* 1991: 324(26);1839-1844.	Wrong age group
Lloyd JK, Wolff OH. Childhood obesity. *Br Med J*. 1961 Jul 15:5245;145-8.	Non-US population
Loder RT, Aronson DD, Greenfield ML. The epidemiology of bilateral slipped capital femoral epiphysis. A study of children in Michigan. *Journal of Bone & Joint Surgery - American Volume* 1993: 75(8);1141-1147.	Not a longitudinal study
Maffeis C, Pietrobelli A, Grezzani A, Provera S, Tato L. Waist circumference and cardiovascular risk factors in prepubertal children. *Obes Res* 2001: 9(3);179-187.	Non-US population
Mallory GB, Jr., Fiser DH, Jackson R. Sleep-associated breathing disorders in morbidly obese children and adolescents. *J Pediatr* 1989:115(6);892-897.	Not a longitudinal study
Mamalakis G, Kafatos A, Manios Y, Kalogeropoulos N, Andrikopoulos N. Adipose fat quality vs. quantity: relationships with children's serum lipid levels. *Preventive Medicine* 2001: 33(6);525-535.	Non-US population
Martin MM, Martin AL. Obesity, hyperinsulinism, and diabetes mellitus in childhood. *J Pediatr* 1973: 82(2);192-201.	Not a longitudinal study
Martini G, Riva P, Rabbia F, Molini V, Ferrero GB, Cerutti F et al. Heart rate variability in childhood obesity. *Clinical Autonomic Research* 2001: 11(2);87-91.	Non-US population
McGill HC, Jr., McMahan CA, Malcom GT, Oalmann MC, Strong JP. Relation of glycohemoglobin and adiposity to atherosclerosis in youth. Pathobiological Determinants of Atherosclerosis in Youth (PDAY) Research Group. *Arterioscler Thromb Vasc Biol* 1995: 15(4);431-440.	Not a longitudinal study Wrong measure of OW
McGill HC, Jr., McMahan CA, Herderick EE, Zieske AW, Malcom GT, Tracy RE et al. Obesity accelerates the progression of coronary atherosclerosis in young men. *Circulation* 2002: 105(23);2712-2718.	Not a longitudinal study

Appendix G. Exclusion Table for Key Questions 2b and 2c (continued)

McTigue KM, Garrett JM, Popkin BM. The natural history of the development of obesity in a cohort of young U.S. adults between 1981 and 1998.[comment][summary for patients in Ann Intern Med. 2002 Jun 18;136(12):I-24; PMID: 12069570]. *Annals of Internal Medicine* 2002: 136(12);857-864.	Not predicting adult OW by youth OW status
Miller FJ, Billewicz WZ, Thomson AM. Growth from birth to adult life of 442 Newcastle upon Tyne children. *Br J Prev Soc Med* 1972: 26(4);224-230.	Non-US population
Mitchell BM, Gutin B, Kapuku G, Barbeau P, Humphries MC, Owens S et al. Left ventricular structure and function in obese adolescents: relations to cardiovascular fitness, percent body fat, and visceral adiposity, and effects of physical training. *Pediatrics* 2002: 109(5);E73.	Wrong age group Not a longitudinal study
Mokdad AH, Marks JS, Stroup DF, Gerberding JL. Actual causes of death in the United States, 2000. *JAMA* 2004: 291(10);1238-1245.	Not a longitudinal study
Molnar D, Livingstone B. Physical activity in relation to overweight and obesity in children and adolescents. *European Journal of Pediatrics* 2000: 159 Suppl 1;S45-S55.	Not a longitudinal study
Monti LD, Brambilla P, Stefani I, Caumo A, Magni F, Poma R et al. Insulin regulation of glucose turnover and lipid levels in obese children with fasting normoinsulinaemia. *Diabetologia* 1995: 38(6);739-747.	Non-US population
Moon OR, Kim NS, Jang SM, Yoon TH, Kim SO. The relationship between body mass index and the prevalence of obesity-related diseases based on the 1995 National Health Interview Survey in Korea. *Obesity Reviews* 2002: 3(3);191-196.	Non-US population
Morgan CM, Tanofsky-Kraff M, Wilfley DE, Yanovski JA. Childhood obesity. *Child & Adolescent Psychiatric Clinics of North America* 2002: 11(2); 257-278.	Not a longitudinal study
Morrison JA, Barton BA, Biro FM, Daniels SR, Sprecher DL. Overweight, fat patterning, and cardiovascular disease risk factors in black and white boys. *Journal of Pediatrics* 1999: 135(4);451-457.	Not a longitudinal study
Morrison JA, Sprecher DL, Barton BA, Waclawiw MA, Daniels SR. Overweight, fat patterning, and cardiovascular disease risk factors in black and white girls: The National Heart, Lung, and Blood Institute Growth and Health Study. *Journal of Pediatrics* 1999: 135(4);458-464.	Not a longitudinal study
Mossberg HO. 40-year follow-up of overweight children. *Lancet* 1989: 2(8661);491-493.	Non-US population
Mo-suwan L, Tongkumchum P, Puetpaiboon A. Determinants of overweight tracking from childhood to adolescence: a 5 y follow-up study of Hat Yai schoolchildren. *International Journal of Obesity & Related Metabolic Disorders* 2000: 24(12);1642-1647.	Non-US population
Must A. Morbidity and mortality associated with elevated body weight in children and adolescents. *Am J Clin Nutr* 1996: 63(3 Suppl);445S-447S.	Not a longitudinal study
Must A. Does overweight in childhood have an impact on adult health? *Nutr Rev* 2003: 61(4);139-142.	Non-US population
Must A, Spadano J, Coakley EH, Field AE, Colditz G, Dietz WH. The disease burden associated with overweight and obesity. *JAMA* 1999: 282(16);1523-1529.	Wrong age group Not a longitudinal study
National Center for Health Statistics. Cited 3/29/04. www.cdc.gov/nchs/products/pubs/pubd/hestats/overwght99.htm Prevalence of overweight among children and adolescents: United States, 1999-2000.	Not a longitudinal study
Newman WP 3rd, Freedman DS, Voors AW, Gard PD, Srinivasan SR, Cresanta JL, Williamson GD, Webber LS, Berenson GS. Relation of serum lipoprotein levels and systolic blood pressure to early atherosclerosis. The Bogalusa Heart Study. N Engl J Med. 1986 Jan 16:314(3);138-44.	Do not report age at OW measurement
Oken E, Lightdale JR. Updates in pediatric nutrition. *Curr Opin Pediatr* 2000: 12(3);282-290.	Not a longitudinal study

Oren A, Vos LE, Uiterwaal CS, Bak AA, Gorissen WH, Grobbee DE et al. The Atherosclerosis Risk in Young Adults (ARYA) study: rationale and design. *European Journal of Epidemiology* 2003: 18(7);715-727.	Non-US population
Paffenbarger RS Jr, Wolf PA, Notkin J, Thorne MC. Chronic disease in former college students. I. Early precursors of fatal coronary heart disease. *Am J Epidemiol*. 1966 Mar:83(2);314-28.	Poor quality rating
Palasciano G, Portincasa P, Vinciguerra V, Velardi A, Tardi S, Baldassarre G et al. Gallstone prevalence and gallbladder volume in children and adolescents: an epidemiological ultrasonographic survey and relationship to body mass index. *American Journal of Gastroenterology* 1989: 84(11);1378-1382.	Non-US population
Peckham CS, Stark O, Simonite V, Wolff OH. Prevalence of obesity in British children born in 1946 and 1958. *British Medical Journal Clinical Research Ed* 1983: 286(6373);1237-1242.	Non-US population
Phillips RG, Hill AJ. Fat, plain, but not friendless: self-esteem and peer acceptance of obese pre-adolescent girls. *International Journal of Obesity & Related Metabolic Disorders* 1998: 22(4);287-293.	Non-US population
Pinhas-Hamiel O, Dolan LM, Daniels SR, Standiford D, Khoury PR, Zeitler P. Increased incidence of non-insulin-dependent diabetes mellitus among adolescents. *J Pediatr*. 1996 May:128(5 Pt 1);608-15.	Not a longitudinal study
Plourde G. Impact of obesity on glucose and lipid profiles in adolescents at different age groups in relation to adulthood. *BMC Family Practice* 2002: 3(1);18.	Non-US population
Power C, Li L, Manor O. A prospective study of limiting longstanding illness in early adulthood. *International Journal of Epidemiology* 2000: 29(1);131-139.	Non-US population
Prokopec M, Bellisle F. Adiposity in Czech children followed from 1 month of age to adulthood: analysis of individual BMI patterns. *Annals of Human Biology* 1993: 20(6);517-525.	Non-US population
Ramirez-Lopez G, Gonzalez-Villalpando C, Sanchez-Corona J, Salmeron-Castro J, Gonzalez-Ortiz M, Celis-de la Rosa A et al. Weight, physical activity, and smoking as determinants of insulinemia in adolescents. *Archives of Medical Research* 2001: 32(3);208-213.	Non-US population
Redline S, Tishler PV, Schluchter M, Aylor J, Clark K, Graham G. Risk factors for sleep-disordered breathing in children. Associations with obesity, race, and respiratory problems. *American Journal of Respiratory & Critical Care Medicine* 1999: 159(5 Pt 1);1527-1532.	Not a longitudinal study
Reilly JJ. Assessment of childhood obesity: national reference data or international approach? *Obes Res* 2002: 10(8);838-840.	Not a longitudinal study
Reilly JJ, Dorosty AR. Epidemic of obesity in UK children. *Lancet* 1999: 354(9193);1874-1875.	Non-US population
Reilly JJ, Ventham JC, Newell J, Aitchison T, Wallace WH, Gibson BE. Risk factors for excess weight gain in children treated for acute lymphoblastic leukaemia. *International Journal of Obesity & Related Metabolic Disorders* 2000: 24(11);1537-1541.	Not a longitudinal study
Reinehr T, Andler W. Thyroid hormones before and after weight loss in obesity. *Archives of Disease in Childhood* 2002:87(4);320-323.	Not a longitudinal study
Resnicow K, Futterman R, Vaughan RD. Body mass index as a predictor of systolic blood pressure in a multiracial sample of US schoolchildren. *Ethnicity & Disease* 1993: 3(4);351-361.	Not a longitudinal study
Rhoads GG, Kagan A. The relation of coronary disease, stroke, and mortality to weight in youth and in middle age. *Lancet* 1983: 1(8323);492-495.	Wrong age group
Ribeiro J, Guerra S, Pinto A, Oliveira J, Duarte J, Mota J. Overweight and obesity in children and adolescents: relationship with blood pressure, and physical activity. *Annals of Human Biology* 2003: 30(2);203-213.	Non-US population

Appendix G. Exclusion Table for Key Questions 2b and 2c (continued)

Richards GE, Cavallo A, Meyer WJ, III, Prince MJ, Peters EJ, Stuart CA et al. Obesity, acanthosis nigricans, insulin resistance, and hyperandrogenemia: pediatric perspective and natural history. *J Pediatr* 1985: 107(6);893-897.	Not a longitudinal study Limited to a specific disease group
Rich-Edwards JW, Goldman MB, Willett WC, Hunter DJ, Stampfer MJ, Colditz GA et al. Adolescent body mass index and infertility caused by ovulatory disorder. *American Journal of Obstetrics & Gynecology* 1994: 171(1);171-177.	Wrong age group
Riddiford-Harland DL, Steele JR, Storlien LH. Does obesity influence foot structure in prepubescent children? *Int J Obes Relat Metab Disord* 2000: 24(5);541-544.	Non-US population
Riley DJ, Santiago TV, Edelman NH. Complications of obesity-hypoventilation syndrome in childhood. *Am J Dis Child* 1976: 130(6);671-674.	Not a longitudinal study Limited to a specific disease group
Rimm IJ, Rimm AA. Association between juvenile onset obesity and severe adult obesity in 73, 532 women. *Am J Public Health* 1976: 66(5);479-481.	Wrong measure of OW
Robinson C, Tamborlane WV, Maggs DG, Enoksson S, Sherwin RS, Silver D, Shulman GI, Caprio S. Effect of insulin on glycerol production in obese adolescents. *Endocrinol. Metab.* 1998:37;E737-43.	Not a longitudinal study Wrong measure of OW
Rolland-Cachera MF, Deheeger M, Bellisle F, Sempe M, Guilloud-Bataille M, Patois E. Adiposity rebound in children: a simple indicator for predicting obesity. *Am J Clin Nutr* 1984: 39(1);129-135.	Non-US population
Rolland-Cachera MF, Deheeger M, Guilloud-Bataille M, Avons P, Patois E, Sempe M. Tracking the development of adiposity from one month of age to adulthood. *Ann Hum Biol* 1987: 14(3);219-229.	Non-US population
Rolland-Cachera MF, Bellisle F, Sempe M. The prediction in boys and girls of the weight/height index and various skinfold measurements in adults: a two-decade follow-up study. *International Journal of Obesity* 1989: 13(3);305-311.	Non-US population
Rudolf MC, Greenwood DC, Cole TJ, Levine R, Sahota P, Walker J et al. Rising obesity and expanding waistlines in schoolchildren: a cohort study. *Archives of Disease in Childhood* 2004: 89(3);235-237.	Non-US population
Russo A, Franceschi S, La Vecchia C, Dal Maso L, Montella M, Conti E et al. Body size and colorectal-cancer risk. *International Journal of Cancer* 1998: 78(2);161-165.	Non-US population
Saitoh H, Kamoda T, Nakahara S, Hirano T, Nakamura N. Serum concentrations of insulin, insulin-like growth factor(IGF)-I, IGF binding protein (IGFBP)-1 and -3 and growth hormone binding protein in obese children: fasting IGFBP-1 is suppressed in normoinsulinaemic obese children. *Clinical Endocrinology* 1998: 48(4);487-492.	Non-US population
Salbe AD; Weyer C; Lindsay RS; Ravussin E; Tataranni PA. Assessing risk factors for obesity between childhood and adolescence: I. Birth weight, childhood adiposity, parental obesity, insulin, and leptin. *Pediatrics.* 2002:110 (2 Pt 1);299-306,.	Wrong age group
Sangi H, Mueller WH, Harrist RB, Rodriguez B, Grunbaum JG, Labarthe DR. Is body fat distribution associated with cardiovascular risk factors in childhood? *Annals of Human Biology* 1992:19(6);559-578.	Not a longitudinal study
Sargent JD, Blanchflower DG. Obesity and stature in adolescence and earnings in young adulthood. Analysis of a British birth cohort. *Arch Pediatr Adolesc Med*. 1994 Jul:148(7);681-7.	Non-US population
Schachter LM, Peat JK, Salome CM. Asthma and atopy in overweight children. *Thorax* 2003: 58(12);1031-1035.	Non-US population
Seminara S, Filpo A, La Cauza F, Faedda A, Miola A, Pellizzone S et al. Growth hormone binding protein activity in obese children. *Journal of Endocrinological Investigation* 1998: 21(7);441-444.	Non-US population

Appendix G. Exclusion Table for Key Questions 2b and 2c (continued)

Sharp GB, Cole P. Identification of risk factors for diethylstilbestrol-associated clear cell adenocarcinoma of the vagina: similarities to endometrial cancer. *American Journal of Epidemiology* 1991: 134(11);1316-1324.	Not a longitudinal study Not an outcome of interest
Sharp TA, Grunwald GK, Giltinan KE, King DL, Jatkauskas CJ, Hill JO. Association of anthropometric measures with risk of diabetes and cardiovascular disease in Hispanic and Caucasian adolescents. *Preventive Medicine* 2003: 37(6 Pt 1);611-616.	Not a longitudinal study
Shear CL, Freedman DS, Burke GL, Harsha DW, Berenson GS. Body fat patterning and blood pressure in children and young adults: The Bogalusa Heart Study. *Hypertension*. 1987:9;236-244.	Not a longitudinal study
Siervogel RM, Roche AF, Guo SM, Mukherjee D, Chumlea WC. Patterns of change in weight/stature2 from 2 to 18 years: findings from long-term serial data for children in the Fels longitudinal growth study. *Int J Obes* 1991: 15(7);479-485.	Wrong age group
Siervogel RM, Wisemandle W, Maynard LM, Guo SS, Chumlea WC, Towne B. Lifetime overweight status in relation to serial changes in body composition and risk factors for cardiovascular disease: The Fels Longitudinal Study. *Obesity Research* 2000: 8(6);422-430.	Wrong age group
Sinha R, Fisch G, Teague B, Tamborlane WV, Banyas B, Allen K et al. Prevalence of impaired glucose tolerance among children and adolescents with marked obesity. *New England Journal of Medicine* 2002: 346(11);802-810.	Not a longitudinal study
Slyper AH. Childhood obesity, adipose tissue distribution, and the pediatric practitioner. *Pediatrics*. 1998:102(1) Part 1 of 3; 131.	Not a longitudinal study
Smoak CG, Burke GL, Webber LS, Harsha DW, Srinivasan SR, Berenson GS. Relation of obesity to clustering of cardiovascular disease risk factors in children and young adults. The Bogalusa Heart Study. *Am J Epidemiol* 1987: 125(3);364-372.	Not a longitudinal study
Sokol RJ. The chronic disease of childhood obesity: the sleeping giant has awakened. *J Pediatr* 2000: 136(6);711-713.	Not a longitudinal study
Somerville SM, Rona RJ, Chinn S. Obesity and respiratory symptoms in primary school. *Archives of Disease in Childhood* 1984: 59(10);940-944.	Non-US population
Sorensen TI, Sonne-Holm S. Mortality in extremely overweight young men. *J Chronic Dis* 1977: 30(6);359-367.	Non-US population
Sorensen TI, Sonne-Holm S. Risk in childhood of development of severe adult obesity: retrospective, population-based case-cohort study. *American Journal of Epidemiology* 1988: 127(1);104-113.	Non-US population
Stark O, Atkins E, Wolff OH, Douglas JW. Longitudinal study of obesity in the National Survey of Health and Development. *British Medical Journal Clinical Research Ed* 1981: 283(6283);13-17.	Non-US population
Steinbeck KS, Bermingham MA, Mahajan D, Baur LA. Low-density lipoprotein subclasses in children under 10 years of age. *Journal of Paediatrics & Child Health* 2001: 37(6);550-553.	Non-US population
Stettler N, Kumanyika SK, Katz SH, Zemel BS, Stallings VA. Rapid weight gain during infancy and obesity in young adulthood in a cohort of African Americans.[comment]. *American Journal of Clinical Nutrition* 2003: 77(6);1374-1378.	Wrong age group
Stewart KJ, Brown CS, Hickey CM, McFarland LD, Weinhofer JJ, Gottlieb SH. Physical fitness, physical activity, and fatness in relation to blood pressure and lipids in preadolescent children. Results from the FRESH Study. *Journal of Cardiopulmonary Rehabilitation* 1995: 15(2);122-129.	Not a longitudinal study
Stoll BA. Teenage obesity in relation to breast cancer risk. *International Journal of Obesity & Related Metabolic Disorders* 1998: 22(11);1035-1040.	Not a longitudinal study Not an outcome of interest

Stradmeijer M, Bosch J, Koops W, Seidell J. Family functioning and psychosocial adjustment in overweight youngsters. *Int J Eat Disord*. 2000 Jan:27(1);110-4.	Non-US population
Strauss R. Childhood obesity. *Curr Probl Pediatr* 1999: 29(1);1-29.	Not a longitudinal study
Strauss RS, Mir HM. Smoking and weight loss attempts in overweight and normal-weight adolescents. *International Journal of Obesity & Related Metabolic Disorders* 2001: 25(9);1381-1385.	Not a longitudinal study Not an outcome of interest
Strauss RS, Pollack HA. Social marginalization of overweight children. *Arch Pediatr Adolesc Med* 2003: 157(8);746-752.	Not a longitudinal study
Striegel-Moore RH, Schreiber GB, Lo A, Crawford P, Obarzanek E, Rodin J. Eating disorder symptoms in a cohort of 11 to 16-year-old black and white girls: the NHLBI Growth and Health Study. National Heart, Lung, and Blood Institute. *Int J Eat Disord* 2000: 27(1Jan);49-66.	Not a longitudinal study for obesity
Stunkard AJ, Sobal J. Psychosocial consequences of obesity. In: Brownell KD, Fairburn C, editors. *Eating Disorders and Obesity: A Comprehensive Handbook*. New York: Guilford Press, 1995: 417-421.	Not a longitudinal study
Tanofsky-Kraff M, Yanovski SZ, Wilfley DE, Marmarosh C, Morgan CM, Yanovski JA. Eating-Disordered Behaviors, Body Fat, and Psychopathology in Overweight and Normal-Weight Children. *Journal of Consulting & Clinical Psychology* 2004:Vol 72(1);61.	Not a longitudinal study
Tershakovec AM, Jawad AF, Stallings VA, Cortner JA, Zemel BS, Shannon BM. Age-related changes in cardiovascular disease risk factors of hypercholesterolemic children. *J Pediatr*. 1998 Mar:132(3 Pt 1);414-20.	Not a longitudinal study Limited to a specific disease group
Tershakovec AM, Kuppler KM, Zemel BS, Katz L, Weinzimer S, Harty MP et al. Body composition and metabolic factors in obese children and adolescents. *International Journal of Obesity & Related Metabolic Disorders* 2003: 27(1);19-24.	Not a longitudinal study Wrong measure of OW
Thomson CC, Clark S, Camargo CA, Jr., Investigators MARC. Body mass index and asthma severity among adults presenting to the emergency department. *Chest* 2003: 124(3);795-802.	Wrong age group
Tounian P, Aggoun Y, Dubern B, Varille V, Guy-Grand B, Sidi D et al. Presence of increased stiffness of the common carotid artery and endothelial dysfunction in severely obese children: a prospective study. *Lancet* 2001:358(9291);1400-1404.	Non-US population
Tulio S, Egle S, Greily B. Blood pressure response to exercise of obese and lean hypertensive and normotensive male adolescents. *Journal of Human Hypertension*. 1995:9;953-8.	Non-US population
Uchiyama M. Risk factors for the development of essential hypertension: long-term follow-up study in junior high school students in Niigata, Japan. *Journal of Human Hypertension* 1994: 8(5);323-325.	Non-US population
Urbina EM, Gidding SS, Bao W, Pickoff AS, Berdusis K, Berenson GS. Effect of body size, ponderosity, and blood pressure on left ventricular growth in children and young adults in the Bogalusa Heart Study. *Circulation* 1995: 91(9);2400-2406.	Wrong age group
Valle M, Gascon F, Martos R, Ruz FJ, Bermudo F, Rios R et al. Infantile obesity: a situation of atherothrombotic risk? *Metabolism: Clinical & Experimental* 2000: 49(5);672-675.	Non-US population
Valle M, Gascon F, Martos R, Ruz FJ, Bermudo F, Morales R et al. Metabolic cardiovascular syndrome in obese prepubertal children: the role of high fasting insulin levels. *Metabolism: Clinical & Experimental* 2002: 51(4);423-428.	Non-US population
Valle M, Gascon F, Martos R, Bermudo F, Ceballos P, Suanes A. Relationship between high plasma leptin concentrations and metabolic syndrome in obese pre-pubertal children. *International Journal of Obesity & Related Metabolic Disorders* 2003: 27(1);13-18.	Non-US population

Appendix G. Exclusion Table for Key Questions 2b and 2c (continued)

Vanhala MJ, Vanhala PT, Keinanen-Kiukaanniemi SM, Kumpusalo EA, Takala JK. Relative weight gain and obesity as a child predict metabolic syndrome as an adult. *International Journal of Obesity & Related Metabolic Disorders* 1999: 23(6);656-659.	Non-US population
Vastag B. Obesity Is Now on Everyone's Plate. *JAMA* 2004: 291(10);1186-1188.	Not a longitudinal study
Visser M, Bouter LM, McQuillan GM, Wener MH, Harris TB. Low-grade systematic inflammation in overweight children. *Pediatrics*. 2001:107(1);e13.	Not a longitudinal study
Von Eyben FE, Mouritsen E, Holm J, Montvilas P, Dimcevski G, Suciu G, Helleberg I, Kristensen L, von Eyben R. Intra-abdominal obesity and metabolic risk factors: A study of young adults. *International Journal of Obesity* 2003:27;941-9.	Non-US population
von Mutius E, Schwartz J, Neas LM, Dockery D, Weiss ST. Relation of body mass index to asthma and atopy in children: the National Health and Nutrition Examination Study III. *Thorax* 2001: 56(11);835-838.	Not a longitudinal study
Wabitsch M, Hauner H, Heinze E, Muche R, Bockmann A, Parthon W, Mayer H, Teller W. Body-fat distribution and changes in the atherogenic risk-factor profile in obese adolescent girls during weight reduction. *Am J Clin Nutr*. 1994 Jul:60(1);54-60.	Non-US population
Waldhor T, Schober E, Rami B, Austrian Diabetes Incidence Study Group. Regional distribution of risk for childhood diabetes in Austria and possible association with body mass index. *European Journal of Pediatrics* 2003: 162(6);380-384.	Non-US population
Wang,G.; Dietz,W.H. Economic burden of obesity in youths aged 6 to 17 years: 1979-1999. *Pediatrics.* 2002:109 (5);E81.	Not a longitudinal study
Washino K, Takada H, Nagashima M, Iwata H. Significance of the atherosclerogenic index and body fat in children as markers for future, potential coronary heart disease. *Pediatrics International* 1999: 41(3);260-265.	Non-US population
Wattigney WA, Harsha DW, Srinivasan SR, Webber LS, Berenson GS. Increasing impact of obesity on serum lipids and lipoproteins in young adults. The Bogalusa Heart Study. *Archives of Internal Medicine* 1991: 151(10);2017-2022.	Not a longitudinal study
Watts K, Beye P, Siafarikas A, O'Driscoll G, Jones TW, Davis EA, Green DJ. Effects of exercise training on vascular function in obese children. *J Pediatr*. 2004 May:144(5);620-5.	Non-US population
Wild RA. Hyperandrogenism in the adolescent. *Pediatric & Adolescent Gynecology*. 1992:19(1);71-89.	Not a longitudinal study Limited to a specific disease group
Williams CL, Hayman LL, Daniels SR, Robinson TN, Steinberger J, Paridon S et al. Cardiovascular health in childhood: A statement for health professionals from the Committee on Atherosclerosis, Hypertension, and Obesity in the Young (AHOY) of the Council on Cardiovascular Disease in the Young, American Heart Association. *Circulation* 2002: 106(1);143-160.	Not a longitudinal study
Williams DP, Going SB, Lohman TG, Harsha DW, Srinivasan SR, Webber LS et al. Body fatness and risk for elevated blood pressure, total cholesterol, and serum lipoprotein ratios in children and adolescents. *Am J Public Health* 1992: 82(3Mar);358-363.	Not a longitudinal study
Williams S, Dickson N. Early growth, menarche, and adiposity rebound. *Lancet* 2002: 359(9306);580-581.	Non-US population
Williams S, Davie G, Lam F. Predicting BMI in young adults from childhood data using two approaches to modelling adiposity rebound. *Int J Obes Relat Metab Disord* 1999: 23(4);348-354.	Non-US population
Xu B, Pekkanen J, Laitinen J, Jarvelin MR. Body build from birth to adulthood and risk of asthma. *European Journal of Public Health* 2002: 12(3);166-170.	Non-US population

Appendix G. Exclusion Table for Key Questions 2b and 2c (continued)

Young TK; Dean HJ; Flett B; Wood-Steiman P. Childhood obesity in a population at high risk for type 2 diabetes. *Journal of Pediatrics.* 2000:136 (3);365-369.	Non-US population
Young-Hyman D, Schlundt DG, Herman L, De Luca F, Counts D. Evaluation of the insulin resistance syndrome in 5- to 10-year-old overweight/obese African-American children. *Diabetes Care* 2001: 24(8);1359-1364.	Not a longitudinal study
Youssef AA, Valdez R, Elkasabany A, Srinivasan SR, Berenson GS. Time-course of adiposity and fasting insulin from childhood to young adulthood in offspring of parents with coronary artery disease: the Bogalusa Heart Study. *Annals of Epidemiology* 2002: 12(8);553-559.	Wrong exposure measure
Zack PM, Harlan WR, Leaverton PE, Cornoni-Huntley J. A longitudinal study of body fatness in childhood and adolescence. *Journal of Pediatrics* 1979: 95(1);126-130.	Wrong age group

Exclusion Table for Key Questions 4 and 5

Reference	Reason for Exclusion
Abu-Abeid,S., Gavert,N., Klausner,J.M., Szold,A. Bariatric surgery in adolescence. *Journal of Pediatric Surgery.* 2003:38(9);1379-1382.	Study design (case-series)
Allen GS: A double-blind clinical trial of diethylpropion hydrochloride, mazindol, and placebo in the treatment of exogenous obesity. *Current Therapeutic Research* 1977: 22;678-385	Mazindol study; published prior to 1985
Andelman MB, Jones C, Nathan S: Treatment of obesity in underprivileged adolescents. Comparison of diethylpropion hydrochloride with placebo in a double-blind study. *Clin Pediatr* (Phila) 1967: 6;327-330	Less than 6 mos f/u
Anderson JW, Hamilton CC, Crown-Weber E, Riddlemoser M, Gustafson NJ: Safety and effectiveness of a multidisciplinary very-low-calorie diet program for selected obese individuals. *J Am Diet Assoc* 1991: 91;1582-1584	Peds data not reported separately; study design (case series)
Baird IM, Howard AN: A double-blind trial of mazindol using a very low calorie formula diet. *International Journal of Obesity.* 1977: 1;271-278	Mazindol study; published prior to 1985
Bal' LV, Shugaeva EN, Deev AA, Maslova AR, Aleksandrov AA. Results of a three-year trial of arterial hypertension prevention in a population of children aged 11-15 years by overweight control. *Cor et Vasa* 1990: 32(6);448-456.	Not in exclusively overweight population; not primary care feasible/referable
Balagopal P, Bayne E, Sager B, Russell L, Patton N, George D. Effect of lifestyle changes on whole-body protein turnover in obese adolescents. *International Journal of Obesity & Related Metabolic Disorders* 2003: 27(10);1250-1257.	Less than 6 mos f/u
Barbeau P, Gutin B, Litaker M, Owens S, Riggs S, Okuyama T. Correlates of individual differences in body-composition changes resulting from physical training in obese children. *Am J Clin Nutr* 1999; 69(4):705-711.	Less than 6 mos f/u
Barbeau P, Litaker MS, Woods KF, Lemmon CR, Humphries MC, Owens S et al. Hemostatic and inflammatory markers in obese youths: effects of exercise and adiposity. *Journal of Pediatrics* 2002: 141(3);415-420.	Baseline and post-intervention weights are not reported
Barbeau P, Gutin B, Litaker MS, Ramsey LT, Cannady WE, Allison J et al. Influence of physical training on plasma leptin in obese youths. *Canadian Journal of Applied Physiology* 2003: 28(3);382-396.	Baseline and post-intervention weights are not reported
Barry H. Is a low-carbohydrate diet more effective in adolescents than a low-fat diet? *Evidence-Based Practice* 2003:6(7);11-2, 2p.	Summary of Sondike, 2003 which is less than 6 mos f/u
Becque et al. Coronary risk incidence obese adolescents: reduction by exercise plus diet intervention. *Pediatrics* 1988: 81; 605-612.	Less than 6 mos f/u
Black DR. Lantz CE. Spouse involvement and a possible long-term follow-up trap in weight loss. *Behav Res Ther* 1984: 22; 557-562	Published prior to 1985
Braet C, Van Winckel M, Van Leeuwen K. Follow-up results of different treatment programs for obese children. *Acta Paediatr* 1997: 86; 397-402.	CCT, not primary care feasible or referable
Braet C, Tanghe A, Bode PD, Franckx H, Winckel MV. Inpatient treatment of obese children: a multicomponent programme without stringent calorie restriction. *European Journal of Pediatrics* 2003: 162(6);391-396.	Not primary care referable

Appendix H. Exclusion Table for Key Questions 4 and 5 (continued)

Reference	Reason for Exclusion
Brandou F, Dumortier M, Garandeau P, Mercier J, Brun JF. Effects of a two-month rehabilitation program on substrate utilization during exercise in obese adolescents. *Diabetes & Metabolism* 2003: 29(1);20-27.	Less than 6 mos f/u
Brownell KD, Stunkard AJ. Couples training, pharmacotherapy and behavior therapy in the treatment of obesity. *Arch Gen Psychiatry*. 1981:38;1224-1229.	Published prior to 1985
Brownell KD, Kelman JH, Stunkard AJ. Treatment of obese children with and without their mothers: changes in weight and blood pressure. *Pediatrics* 1983: 71; 515-523.	Published prior to 1985
Canty LM. Teen Obesity Addressed in the Military. *Military Medicine* 2003:168(2);139-142.	No controls
Chen MY, Huang LH, Wang EK, Cheng NJ, Hsu CY, Hung LL et al. The effectiveness of health promotion counseling for overweight adolescent nursing students in Taiwan. *Public Health Nursing* 2001: 18(5);350-356.	Study design
Christakis G, Sajecki S, Hillman RW, Miller E, Blumenthal S, Archer M: Effect of a combined nutrition education and physical fitness program on the weight status of obese high school boys. *Fed.Proc* 1966: 25;15-19	School-based; only 40% of pop is overweight
Coney PJ WK. Weight change and adverse event incidence with a low-dose oral contraceptive: Two randomized, placebo-controlled trials. *Contraception* 2001:63(6);297-302.	Baseline and post-intervention weights are not reported
Cuellar, GE et al. Six-month treatment of obesity with sibutramine 15 mg; a double-blind, placebo-controlled monocenter clinical trial in a Hispanic population. *Obesity Research* 2000: 8(1); 71-82.	Peds data not reported separately
Davis BR, Blaufox MD, Oberman A, Wassertheil-Smoller S, Zimbaldi N, Cutler JA, Kirchner K, Langford HG: Reduction in long-term antihypertensive medication requirements. Effects of weight reduction by dietary intervention in overweight persons with mild hypertension. *Arch Intern Med* 1993: 153;1773-1782	Not a pediatric study (age 21-65 yrs)
Davis K, Christoffel KK: Obesity in preschool and school-age children. Treatment early and often may be best. *Arch Pediatr Adolesc Med* 1994: 148;1257-1261	Less than 6 mos f/u
Denzer C, Reithofer E, Wabitsch M, Widhalm K. The outcome of childhood obesity management depends highly upon patient compliance. *European Journal of Pediatrics* 2004; 163(2):99-104.	No untreated or comparison group
DeWolfe JA, Jack E. Weight control in adolescent girls: a comparison of the effectiveness of three approaches to follow up. *J Sch Health* 1984: 54; 347-349.	School-based; published prior to 1985
Dolan K; Creighton L; Hopkins G; Fielding G. Laparoscopic gastric banding in morbidly obese adolescents. *Obes Surg.* 2003:13(1);101-104.	Study design (case-series)
Dubbert PM, Wilson GT. Goal-setting and spouse involvement in the treatment of obesity. *Behav Res Ther*. 1984:22;227-242.	Published prior to 1985
Ebbeling CB, Rodriguez NR: Effects of exercise combined with diet therapy on protein utilization in obese children. *Med Sci Sports Exerc* 1999: 31;378-385	Less than 6 mos f/u

Appendix H. Exclusion Table for Key Questions 4 and 5 (continued)

Reference	Reason for Exclusion
Ekelund U, Aman J, Yngve A, Renman C, Westerterp K, Sjostrom M. Physical activity but not energy expenditure is reduced in obese adolescents: a case-control study.[see comment]. *American Journal of Clinical Nutrition* 2002: 76(5);935-941.	Study design; less than 6 mos f/u
Eliakim A, Kaven G, Berger I, Friedland O, Wolach B, Nemet D. The effect of a combined intervention on body mass index and fitness in obese children and adolescents - a clinical experience. *European Journal of Pediatrics* 2002: 161(8);449-454.	CCT
Epstein LH Wing RR, Koeske R, et al. Child and parent weight loss in family-based behavior modification programs. *J Consult Clin Psychol* 1981: 49; 674-685.	Published prior to 1985
Epstein LH, Koeske R, Wing RR. Adherence to Exercise in Obese Children. *Journal of Cardiac Rehabilitation* 1984; 4:185-195.	Published prior to 1985
Epstein LH, Wing RR, Koeske R. Effects of diet plus exercise on weight change in parents and children. *J Consult Clin Psychol* 1984: 52; 429-437.	Published prior to 1985
Epstein LH, Wing RR, Koeske R, Valoski A. Effect of parent weight on weight loss in obese children. *J Consult Clin Psychol*. 1986:54;400-401.	Baseline and post-intervention weights are not reported
Epstein LH, Wing RR, Valoski A, Gooding W. Long-term effects of parent weight on child weight loss. *Behav Ther.* 1987;18:219-226.	Evaluates parent weight as prognostic factor; does not present baseline characteristics or attrition rates stratified by intervention group, therefore unable to assess USPSTF quality grade.
Estelles A, Dalmau J, Falco C, Berbel O, Castello R, Espana F et al. Plasma PAI-1 levels in obese children--effect of weight loss and influence of PAI-1 promoter 4G/5G genotype. *Thrombosis & Haemostasis* 2001: 86(2);647-652.	Study design; less than 6 mos f/u
Faith MS, Berman N, Heo M, Pietrobelli A, Gallagher D, Epstein LH et al. Effects of contingent television on physical activity and television viewing in obese children. *Pediatrics* 2001: 107(5);1043-1048.	Less than 6 mos f/u
Fanghanel G, Cortinas L, Sanchez-Reyes L, Berber A: A clinical trial of the use of sibutramine for the treatment of patients suffering essential obesity. *International Journal of Obesity & Related Metabolic Disorders.* 2000: 24;144-150	Peds data not reported separately
Fanghanel G, Cortinas L, Sanchez-Reyes L, Berber A: Second phase of a double-blind study clinical trial on Sibutramine for the treatment of patients suffering essential obesity: 6 months after treatment cross-over. *International Journal of Obesity & Related Metabolic Disorders.* 2001: 25;741-747	Peds data not reported separately
Favretti F, Cadiere GB, Segato G, Himpens J, De Luca M, Busetto L, De Marchi F, Foletto M, Caniato D, Lise M, Enzi G: Laparoscopic banding: selection and technique in 830 patients. *Obesity Surgery*. 2002: 12;385-390	Peds data not separate

Appendix H. Exclusion Table for Key Questions 4 and 5 (continued)

Reference	Reason for Exclusion
Field AE, Austin SB, Taylor CB, Malspeis S, Rosner B, Rockett HR et al. Relation between dieting and weight change among preadolescents and adolescents. *Pediatrics* 2003: 112(4);900-906.	Intervention not administered by a professional
Figueroa-Colon R, von Almen TK, Franklin FA, Schuftan C, Suskind RM: Comparison of two hypocaloric diets in obese children. *American Journal of Diseases of Children*. 1993: 147;160-166	CCT
Foster GD, Wadden TA, Brownell KD: Peer-led program for the treatment and prevention of obesity in the schools. *J Consult Clin Psychol* 1985: 53;538-540	Less than 6 mos f/u
Gately PJ, Cooke CB, Butterly RJ, Mackreth P, Carroll S. The effects of a children's summer camp programme on weight loss, with a 10 month follow-up. *International Journal of Obesity & Related Metabolic Disorders* 2000: 24(11);1445-1452.	Study design (case-series)
Gehrman CA. Effects of a physical activity and nutrition intervention on body image in pre-adolescents. *Dissertation Abstracts International: Section B: the Sciences & Engineering* 2003:Vol 64(3-B).	Not in overwt or at risk for overwt population
Gortmaker SL, Peterson K, Wiecha J, Sobol AM, Dixit S, Fox MK, Laird N: Reducing obesity via a school-based interdisciplinary intervention among youth: Planet Health. *Archives of Pediatrics & Adolescent Medicine*. 1999: 153;409-418	Not primary care-feasible or referable (school-based)
Grignard,S.; Jean-Pierre,B.; Michel,B.; Philippe,M.; Chantal,V. Characteristics of adolescent attempts to manage overweight. *Patient Education & Counseling.* 2003:51(2);183-189.	Study design
Gutin B, Cucuzzo N, Islam S, Smith C, Stachura ME. Physical training, lifestyle education, and coronary risk factors in obese girls. *Medicine & Science in Sports & Exercise* 1996:28(1);19-23.	Less than 6 mos f/u
Gutin B, Owens S, Okuyama T, Riggs S, Ferguson M, Litaker M. Effect of physical training and its cessation on percent fat and bone density of children with obesity. *Obesity Research* 1999; 7(2):208-214.	Less than 6 mos f/u
Hamilton JL; James FW; Bazargan M. Provider practice, overweight and associated risk variables among children from a multi-ethnic underserved community. *J Natl Med Assoc.* 2003:95(6);441-448.	Not a CCT/RCT
Herrera EA, Johnston CA, Steele RG. A comparison of cognitive and behavioral treatments for pediatric obesity. *Children's Health Care* 2004:33(2); 151-167.	Less than 6 mos f/u
Hills AP, Parker AW. Obesity management via diet and exercise intervention. *Child Care Health* 1988:14; 409-416.	Less than 6 mos f/u
Hipsky J, Kirk S. HealthWorks! Weight management program for children and adolescents. *Journal of the American Dietetic Association.* 2002:102(3S);S64-S67.	Study design
Hoerr SL, Nelson RA, Essex-Sorlie D. Treatment and follow-up of obesity in adolescent girls. *Journal of Adolescent Health Care* 1988; 9(1):28-37.	No controls

Appendix H. Exclusion Table for Key Questions 4 and 5 (continued)

Reference	Reason for Exclusion
Hoffman RP, Stumbo PJ, Janz KF, Nielsen DH. Altered insulin resistance is associated with increased dietary weight loss in obese children. *Horm Res* 1995; 44(1):17-22.	Less than 6 mos f/u
Hoie LH, Bruusgaard D: Compliance, clinical effects, and factors predicting weight reduction during a very low calorie diet regime. *Scand J Prim Health Care* 1995: 13;13-20	Less than 6 mos f/u; peds data not reported separately
Hoos MB, Gerver WJ, Kester AD, Westerterp KR. Physical activity levels in children and adolescents. *International Journal of Obesity & Related Metabolic Disorders.* 2003:27(5);605-609.	No wt loss intervention tested
Inge TH; Garcia V; Daniels S; Langford L; Kirk S; Roehrig H; Amin R; Zeller M; Higa K. A multidisciplinary approach to the adolescent bariatric surgical patient. *Journal of Pediatric Surgery*.2004:39(3);442.-7.	Study design (case-series)
Israel AC, Saccone AJ. Follow-up of effects of choice of mediator and target of reinforcement on weight loss. *Behav Ther*. 1979:10;260-265.	Published prior to 1985
James WP, Astrup A, Finer N, Hilsted J, Kopelman P, Rossner S, Saris WH, Van Gaal LF. Effect of sibutramine on weight maintenance after weight loss: a randomised trial. STORM Study Group. Sibutramine Trial of Obesity Reduction and Maintenance. *Lancet* 2000: 356;2119-2125	Peds data not reported separately
Jiao et al. Clinical study on rhubarb extract tablet in treating simple obesity. *CJIM* 2001: 7(1); 33-35.	Less than 6 mos f/u
Johnson WG, Hinkle LK Carr RE, et al. Dietary and exercise interventions for juvenile obesity: long-term effects of behavioural and public health models. *Obs Res* 1997: 5; 257-61	Poor USPSTF Quality grade
Karvetti RL, Hakala P. A seven-year follow-up of a weight reduction programme in Finnish primary health care. *Eur J Clin Nutr* 1992: 46;743-752	Peds data not reported separately
Kay JP, Alemzadeh R, Langley G, D'Angelo L, Smith P, Holshouser S. Beneficial effects of metformin in normoglycemic morbidly obese adolescents. *Metabolism: Clinical & Experimental* 2001: 50(12);1457-1461.	Less than 6 mos f/u; disease-specific patient population: hyperinsulinemic, non-diabetic obese adolescents
Kirschenbaum DS, Harris ES, Tomarken AJ. Effects of parental involvement in behavioral weight loss therapy for preadolescents. *Behav Ther* 1984: 15; 485-500.	Published prior to 1985
Levine MD, Ringham RM, Kalarchian MA, Wisniewski L, Marcus MD. Is family-based behavioral weight control appropriate for severe pediatric obesity? *International Journal of Eating Disorders* 2001: 30(3);318-328.	Less than 6 mos f/u; no control group
Lorber J. Obesity in childhood. A controlled trial of anorectic drugs. *Arch Dis Child* 1966: 41;309-312	Less than 6 mos f/u

Appendix H. Exclusion Table for Key Questions 4 and 5 (continued)

Reference	Reason for Exclusion
Luepker RV, Perry CL, McKinlay SM, Nader PR, Parcel GS, Stone EJ, Webber LS, Elder JP, Feldman HA, Johnson CC. Outcomes of a field trial to improve children's dietary patterns and physical activity. The Child and Adolescent Trial for Cardiovascular Health. CATCH collaborative group. *JAMA* 1996: 275;768-776	Not primary care-feasible or referable (school-based)
Maclay WP, Wallace MG: A multi-centre general practice trial of mazindol in the treatment of obesity. *Practitione*r 1977: 218;431-434	Mazindol study; published prior to 1985
Manios Y, Moschandreas J, Hatzis C, Kafatos A: Evaluation of a health and nutrition education program in primary school children of Crete over a three-year period. *Prev Med* 1999: 28;149-159	Not primary care-feasible or referable (school-based)
Manning RM, Jung RT, Leese GP, Newton RW: The comparison of four weight reduction strategies aimed at overweight diabetic patients. *Diabet Med* 1995: 12;409-415	All diabetic patients
McCann S, McDuffie J, Nicholson J, Sastry L, Calis K, Yanovski J. A pilot study of the efficacy of orlistat in overweight adolescents. *Obes.Res.* 2000:8[S1], 58S.	Less than 6 mos f/u; no controls
McDuffie JR, Calis KA, Uwaifo GI, Sebring NG, Fallon EM, Frazer TE et al. Efficacy of orlistat as an adjunct to behavioral treatment in overweight African American and Caucasian adolescents with obesity-related co-morbid conditions. *Journal of Pediatric Endocrinology* 17(3):307-319, 2004.	Study design (case series); also required to have obesity-related comorbid condition
Miller K, Hell E. Laparoscopic adjustable gastric banding: a prospective 4-year follow-up study. *Obesity Surgery.* 1999:9(2);183-187.	Study design (case series); results for pediatric age group not reported separately
Mirouze J, Mary P, Schmouker Y, Lapinski H, Chauchard C, Prunac N: [Alternating low calory diets (200 to 1000 calories), their value in the treatment of resistant obesity]. *Sem.Hop*. 1976: 52;2255-2261	Published prior to 1985
Molnar D, Torok K, Erhardt E, Jeges S. Safety and efficacy of treatment with an ephedrine/caffeine mixture. The first double-blind placebo-controlled pilot study in adolescents. *International Journal of Obesity & Related Metabolic Disorders* 2000; 24(12):1573-1578.	Less than 6 mos f/u
Molokhia M: Obesity wars: a pilot study of very low calorie diets in obese patients in general practice. *Br J Gen Pract* 1998: 48;1251-1252	Not a pediatric study (age 27-83 yrs)
Moon YI, Park HR, Koo HY, Kim HS. Effects of behavior modification on body image, depression and body fat in obese Korean elementary school children. *Yonsei Medical Journal* 2004; 45(1):61-67.	CCT
Murphy JK. The long-term effects of spouse involvement upon weight loss and maintenance. *Behav Ther*. 1982:13;681-693.	Published prior to 1985
Norgren S, Danielsson P, Jurold R, Lotborn M, Marcus C. Orlistat treatment in obese prepubertal children: a pilot study. *Acta Paediatrica* 2003: 92(6);666-670.	Less than 6 mos f/u; no controls
Nova A, Russo A, Sala E. Long-term management of obesity in paediatric office practice: experimental evaluation of two different types of intervention. *Ambul Child Health* 2001: 7; 239-47.	Poor USPSTF Quality grade

Appendix H. Exclusion Table for Key Questions 4 and 5 (continued)

Reference	Reason for Exclusion
Nuutinen O. Long-term effects of dietary counselling on nutrient intake and weight loss in obese children. *European Journal of Clinical Nutrition* 1991; 45(6):287-297.	CCT, inappropriate control group
Nuutinen O, Knip M. Predictors of weight reduction in obese children. *European Journal of Clinical Nutrition* 1992: 46(11);785-794.	No wt loss intervention tested
Obarzanek E, Hunsberger SA, Van Horn L, Hartmuller VV, Barton BA, Stevens VJ, Kwiterovich PO, Franklin FA, Kimm SY, Lasser NL, Simons-Morton DG, Lauer RM: Safety of a fat-reduced diet: the Dietary Intervention Study in Children (DISC). *Pediatrics* 1997: 100;51-59	All children with high LDL
Owens S, Gutin B, Allison J et al. Effect of physical training on total and visceral fat in obese children. *Med Sci Sports Exerc*. 1999: 31; 143-148.	Less than 6 mos f/u
Pearce JW, LeBow MD, Orchard J. Role of spouse involvement in the behavioral treatment of overweight women. *J Consult Clin Psychol.* 1981:49;236-244.	Published prior to 1985
Pidlich J, Pfeffel F, Zwiauer K, Schneider B, Schmidinger H: The effect of weight reduction on the surface electrocardiogram: a prospective trial in obese children and adolescents. *Int J Obes Relat Metab Disord* 1997: 21;1018-1023	Less than 6 mos f/u
Pittler MH. Rhubarb extract -- helpful in treating obesity? *Focus on Alternative & Complementary Therapies* 2001:6(3);195-6.	Summary of Jiao et al 2001, which is less than 6 mos f/u
Raeburn JM, Atkinson JM: A low-cost community approach to weight control: initial results from an evaluated trial. *Prev Med* 1986: 15;391-402.	Peds data not reported separately
Randolph JG, Weintraub WH, Rigg A: Jejunoileal bypass for morbid obesity in adolescents. *J Pediatr Surg* 1974: 9;341-345.	Study design (case-series); jejunoileal bypass surgery
Reinehr T, Kersting M, Alexy U, Andler W. Long-term follow-up of overweight children: after training, after a single consultation session, and without treatment. *Journal of Pediatric Gastroenterology & Nutrition* 2003: 37(1);72-74.	CCT
Reiterer EE, Sudi KM, Mayer A, Limbert-Zinterl C, Stalzer-Brunner C, Fuger G et al. Changes in leptin, insulin and body composition in obese children during a weight reduction program. *Journal of Pediatric Endocrinology & Metabolism* 1999: 12(6);853-862.	Less than 6 mos f/u; no controls
Rigg CA: Proceedings: Jejunoileal bypass by morbidly obese adolescent. *Acta Paediatr Scand Suppl* 1975:62-64	Jejunoileal bypass surgery; published prior to 1985
Robinson,T.N. Behavioural treatment of childhood and adolescent obesity. *International Journal of Obesity & Related Metabolic Disorders.* 1999:23(S2)2;S52-7.	Not primary care-feasible or referable (school-based)
Rocchini AP, Katch V, Anderson J, Hinderliter J, Becque D, Martin M, Markes C. Blood pressure in obese adolescents: effect of weight loss. *Pediatrics* 1988:82;16-23.	Less than 6 mos f/u

Appendix H. Exclusion Table for Key Questions 4 and 5 (continued)

Reference	Reason for Exclusion
Rolland-Cachera MF, Thibault H, Souberbielle JC, Soulie D, Carbonel P, Deheeger M, Roinsol D, Longueville E, Bellisle F, Serog P. Massive obesity in adolescents: dietary interventions and behaviours associated with weight regain at 2 y follow-up. *International Journal of Obesity* 2004: 28; 514-519.	Not primary care-feasible
Rosenthal B, Allen GJ, Winter C. Husband involvement in the behavioral treatment of overweight women: initial effects and long-term follow-up. *Int J Obes Relat Metab Disord*. 1980:4;165-173.	Published prior to 1985
Saccone AJ, Israel AC. Effects of experimenter versus significant other-controlled reinforcement and choice of target behavior on weight loss. *Behav Ther*. 1978:9;271-278.	Published prior to 1985
Sartorio A, Lafortuna CL, Marinone PG, Tavani A, La Vecchia C, Bosetti C. Short-term effects of two integrated, non-pharmacological body weight reduction programs on coronary heart disease risk factors in young obese patients. *Diabetes, Nutrition & Metabolism - Clinical & Experimental* 2003; 16(4):262-265.	Not a pediatric study
Savage MP, Petratis MM, Thomson WH, Berg K, Smith JL, Sady SP. Exercise training effects on serum lipids of prepubescent boys and adult men. *Med Sci Sports Exerc* 1986; 18(2):197-204.	Less than 6 mos f/u
Schwingshandl J, Sudi K, Eibl B, Wallner S, Brokenstein M. Effect of an individualised training programme during weight reduction on body composition: a randomised trial. *Arch Dis Child* 1999: 81; 426-428.	Less than 6 mos f/u
Sondike SB, Copperman N, Jacobson MS. Effects of a low-carbohydrate diet on weight loss and cardiovascular risk factor in overweight adolescents. *Journal of Pediatrics* 2003: 142(3);253-258.	Less than 6 mos f/u
Sothern, Udall JN, Jr., Suskind RM, Vargas A, Blecker U: Weight loss and growth velocity in obese children after very low calorie diet, exercise, and behavior modification. *Acta Paediatrica*. 2000: 89;1036-1043	Study design (case-series)
Spieth LE, Harnish JD, Lenders CM, Raezer LB, Pereira MA, Hangen SJ, Ludwig DS: A low-glycemic index diet in the treatment of pediatric obesity. *Archives of Pediatrics & Adolescent Medicine*. 2000: 154;947-951	Less than 6 mos f/u
Stallings VA, Archibald EH, Pencharz PB. Potassium, magnesium, and calcium balance in obese adolescents on a protein-sparing modified fast. *Am J Clin Nutr* 1988; 47(2):220-224.	Less than 6 mos f/u; not primary care-feasible or referable (inpatient hospital setting)
Stanford A; Glascock JM; Eid GM; Kane T; Ford HR; Ikramuddin S; Schauer P. Laparoscopic Roux-en-Y gastric bypass in morbidly obese adolescents. *Journal of Pediatric Surgery.* 2003:38(3);430-433.	Study design
Strauss RS, Bradley LJ, Brolin RE: Gastric bypass surgery in adolescents with morbid obesity. *J Pediatr* 2001: 138;499-504	Study design (case-series)
Sugerman HJ, Sugerman EL, DeMaria EJ, Kellum JM, Kennedy C, Mowery Y et al. Bariatric surgery for severely obese adolescents. *Journal of Gastrointestinal Surgery* 2003: 7(1);102-107.	Study design (case-series)

Appendix H. Exclusion Table for Key Questions 4 and 5 (continued)

Reference	Reason for Exclusion
Sung RY, Yu CW, Chang SK, Mo SW, Woo KS, Lam CW. Effects of dietary intervention and strength training on blood lipid level in obese children. *Archives of Disease in Childhood* 2002: 86(6);407-410.	Less than 6 mos f/u
Suskind R; Blecker U; Udall J Jr; Von Almen T; Schumacher H; Carlisle L; Sothern M. Recent advances in the treatment of childhood obesity. *Pediatric Diabetes* 2000:1(1);23-33.	Study design
Suttapreyasri D, Suthontan N, Kanpoem J, Krainam J, Boonsuya C. Weight-control training-models for obese pupils in Bangkok. *Journal of the Medical Association of Thailand* 1990: 73(7);394-400.	Not primary care-feasible or referable (school-based)
Talvia S, Lagstrom H, Rasanen M, Salminen M, Rasanen L, Salo P, Viikari J, Ronnemaa T, Jokinen E, Vahlberg T, Simell O. A randomized intervention since infancy to reduce intake of saturated fat: calorie (energy) and nutrient intakes up to the age of 10 years in the Special Turku Coronary Risk Factor Intervention Project. *Arch Pediatr Adolesc Med* 2004:158(1);41-47.	Not in an overweight population; baseline & post-intervention weight not measured
Tershakovec AM, Jawad AF, Stallings VA, Zemel BS, McKenzie JM, Stolley PD, Shannon BM: Growth of hypercholesterolemic children completing physician-initiated low-fat dietary intervention. *J Pediatr* 1998: 133:28-34	All patients had high cholesterol
Tounian P, Frelut ML, Parlier G, Abounaufal C, Aymard N, Veinberg F, Fontaine JL, Girardet JP: Weight loss and changes in energy metabolism in massively obese adolescents. *Int J Obes Relat Metab Disord* 1999: 23;830-837	Less than 6 mos f/u
Treuth MS, Sunehag AL, Trautwein LM, Bier DM, Haymond MW, Butte NF. Metabolic adaptation to high-fat and high-carbohydrate diets in children and adolescents. *American Journal of Clinical Nutrition* 2003; 77(2):479-489.	Intervention not tested in overweight or at-risk for overweight population
Valverde MA, Patin RV, Oliveira FL, Lopez FA, Vitolo MR. Outcomes of obese children and adolescents enrolled in a multidisciplinary health program. *International Journal of Obesity & Related Metabolic Disorders* 1998: 22(6);513-519.	No controls
Vandenplas Y, Bollen P, De Langhe K, Vandemaele K, De Schepper J. Intragastric balloons in adolescents with morbid obesity. *European Journal of Gastroenterology & Hepatology* 1999; 11(3):243-245.	No controls
Vandongen R, Jenner DA, Thompson C, Taggart AC, Spickett EE, Burke V, Beilin LJ, Milligan RA, Dunbar DL: A controlled evaluation of a fitness and nutrition intervention program on cardiovascular health in 10- to 12-year-old children. *Prev Med* 1995: 24;9-22	Not primary care-feasible or referable (school-based)
Vogiatzi MG; Boeck MA; Vlachopapadopoulou E; el Rashid R; New MI. Dehydroepiandrosterone in morbidly obese adolescents: effects on weight, body composition, lipids, and insulin resistance. *Metabolism: Clinical & Experimental.* 1996:45(8);1011-1015.	Less than 6 mos f/u

Appendix H. Exclusion Table for Key Questions 4 and 5 (continued)

Reference	Reason for Exclusion
Wabitsch M; Hauner H; Heinze E; Bockmann A; Benz R; Mayer H; Teller W. Body fat distribution and steroid hormone concentrations in obese adolescent girls before and after weight reduction. *Journal of Clinical Endocrinology & Metabolism.* 1995:80(12):3469-3475.	Less than 6 mos f/u
Wabitsch M, Braun U, Heinze E, Muche R, Mayer H, Teller W et al. Body composition in 5-18-y-old obese children and adolescents before and after weight reduction as assessed by deuterium dilution and bioelectrical impedance analysis. *American Journal of Clinical Nutrition* 1996: 64(1);1-6.	Less than 6 mos f/u
Wallace AG: AN 448 Sandoz (Mazindol) in the treatment of obesity. *Med J Aust* 1976: 1;343-345	Mazindol study; published prior to 1985
Warschburger P, Fromme C, Petermann F, Wojtalla N, Oepen J. Conceptualisation and evaluation of a cognitive-behavioural training programme for children and adolescents with obesity. *International Journal of Obesity & Related Metabolic Disorders* 2001:25(Suppl 1);S93-S95.	Poor USPSTF Quality grade; not primary care feasible or referable
White JJ, Cheek D, Haller JA, Jr.: Small bowel bypass is applicable for adolescents with morbid obesity. *American Surgeon*. 1974: 40;704-708	Study design (case-series)
Wilmore JH, Despres JP, Stanforth PR, Mandel S, Rice T, Gagnon J et al. Alterations in body weight and composition consequent to 20 wk of endurance training: the HERITAGE Family Study. *American Journal of Clinical Nutrition* 1999: 70(3);346-352.	Less than 6 mos f/u
Wing RR, Marcus MD, Epstein LH, Jawad A. A "family-based" approach to the treatment of obese Type II diabetic patients. *J Consult Clin Psychol*. 1991:59;156-162.	Study in adults only; population with specific disease (type II DM)
Wong ML, Koh D, Lee MH, Fong YT. Two-year follow-up of a behavioural weight control programme for adolescents in Singapore: predictors of long-term weight loss. *Annals of the Academy of Medicine, Singapore* 1997: 26(2);147-153.	Peds data not reported separately; no controls who did not receive intervention
Ylitalo VM: Treatment of obese schoolchildren. *Klin Padiatr*. 1982: 194;310-314	CCT
Yoshinaga M, Sameshima K, Miyata K, Hashiguchi J, Imamura M. Prevention of mildly overweight children from development of more overweight condition. *Preventive Medicine* 2004; 38(2):172-174.	CCT
Zhi J; Moore R; Kanitra L. The effect of short-term (21-day) orlistat treatment on the physiologic balance of six selected macrominerals and microminerals in obese adolescents. *Journal of the American College of Nutrition*. 2003:22(5);357-62.	Less than 6 mos f/u

USPSTF Hierarchy of Research Design and Quality Rating Criteria

Hierarchy of Research Design

I Properly conducted randomized controlled trial (RCT)
II-1: Well-designed controlled trial without randomization
II-2: Well-designed cohort or case-control analytic study
II-3: Multiple time series with or without the intervention; dramatic results from uncontrolled experiments
III: Opinions of respected authorities, based on clinical experience; descriptive studies or case reports; reports of expert committees

Design-Specific Criteria and Quality Category Definitions

Systematic Reviews

Criteria:

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

Definition of ratings from above criteria:

Good: Recent, relevant review with comprehensive sources and search strategies; explicit and relevant selection criteria; standard appraisal of included studies; and valid conclusions.
Fair: Recent, relevant review that is not clearly biased but lacks comprehensive sources and search strategies.
Poor: Outdated, irrelevant, or biased review without systematic search for studies, explicit selection criteria, or standard appraisal of studies.

Case-Control Studies

Criteria:

- Accurate ascertainment of cases
- Nonbiased selection of cases/controls 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
- Measurement of exposure accurate and applied equally to each group
- Appropriate attention to potential confounding variables

Definition of ratings based on criteria above:

Good: Appropriate ascertainment of cases and nonbiased selection of case and control participants; exclusion criteria applied equally to cases and controls; response rate equally to or greater than 80 percent; diagnostic procedures and measurements accurate and applied equally to cases and controls; and appropriate attention to confounding variables.

Fair: Recent, relevant, without major apparent selection or diagnostic work-up bias but with response rates less than 80 percent or attention to some but not all important confounding variables.

Poor: Major section or diagnostic work-up biases, response rates less than 50 percent, or inattention to confounding variables.

Randomized Controlled Trials and Cohort Studies

Criteria:

- Initial assembly of comparable groups
  - -for RCTs: 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, cross-overs, adherence, contamination)
- Important differential loss to follow-up or overall high loss to follow-up
- Measurements: equal, reliable, and valid (includes masking of outcome assessment)
- Clear definition of the interventions
- All important outcomes considered

Definition of ratings based on above criteria:

Good: Evaluates relevant available screening tests; uses a credible reference standard; interprets reference standard independently of screening test; reliability of test assessed; has few or handles indeterminate results in a reasonable manner; includes large number (more than 100 broad-spectrum of patients.

Fair: Evaluates relevant available screening tests; uses reasonable although not best standard; interprets reference standard independent of screening test; moderate sample size (50 to 100 subjects) and a “medium” spectrum of patients.

Poor: Has fatal flaw such as: Uses inappropriate reference standard; screening test improperly administered; biased ascertainment of reference standard; very small sample size or very narrow selected spectrum of patients.

Diagnostic Accuracy Studies

Criteria:

- Screening test relevant, available for primacy care, adequately described
- Study uses a 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

Definition of ratings based on above criteria:

Good: Evaluates relevant available screening test; uses a credible reference standard; interprets reference standard independently of screening test; reliability of test assessed; has few or handles indeterminate results in a reasonable manner; includes large number (more than 100) broad-spectrum patients with and without disease.

Fair: Evaluates relevant available screening test; uses reasonable although not best standard; interprets reference standard independent of screening test; moderate sample size (50-100 subjects) and a "medium" spectrum of patients.

Poor: Has fatal flaw such as: Uses inappropriate reference standard; screening test improperly administered; biased ascertainment of reference standard; very small sample size or very narrow selected spectrum patients.

References

1. Harris R, Atkins D, Berg AO, Best D, Eden KB, Feightner JW et al. *US Preventive Services Task Force Procedure Manual*. Rockville, MD: Agency for Healthcare Research and Quality, 2001.

2. Harris RP, Helfand M, Woolf SH, Lohr KN, Mulrow CD, Teutsch SM et al. Current methods of the US Preventive Services Task Force: a review of the process. *Am J Prev Med* 2001; 20(3 Suppl):21-35.

Childhood Overweight Clinical Screening Tests and Predicting Adult Obesity

Prior Reviews

A fair-quality systematic evidence review identified one previous review article representing international literature through 1992[1] and identified six reports addressing obesity persistence from childhood into adulthood.[2] The systematic review indicated a tendency for childhood overweight to persist, particularly in older children, in overweight children with one or more obese parents, and in children with more severe overweight.[2] Quantitative relationships between childhood overweight measures and adult obesity measures were not systematically reported. Another non-systematic but extensive review through 1996 identified 25 longitudinal studies worldwide examining the relationship between childhood and adult adiposity.[3] Persistence (tracking) of adiposity from childhood and adolescence to adulthood varied with the measure of adiposity, the cutoff used to define overweight/obesity, the age of childhood assessment, and the age at which adulthood overweight/obesity was assessed.[3] In restricting their analysis to large British cohort studies, less variability was seen in the risk estimations, perhaps due to reduced variation in cutoff values used. Relative risks for adult obesity at age 33 years were higher for boys (7.0, 95% CI 5.7-8.4) and girls (5.5, 95% CI 4.8-6.4) with BMIs over the 98th percentile at age 16, than for boys and girls aged 7 with the same BMI (4.2, 95% CI 3.3-5.5 and 4.2, 95% CI 3.5-5.0, respectively). Similarly, correlations for BMI and other indices of adiposity were higher (0.46-0.91 in males and 0.60-0.78 in females) between adolescence and young adulthood than for younger ages (r of about 0.30). BMI between childhood and adulthood was more strongly correlated than skinfolds or waist:hip ratio. Few studies examined the impact of change in adiposity in childhood or timing of adiposity rebound. However, the majority of obese adults were not fat in childhood or adolescence. For example, in a 1958 British birth cohort, 13% and 11% of obese 33-year-old men and women were overweight at age 16, but only 8% and 9% were overweight at age seven. The authors concluded that, while adiposity tracks from childhood--particularly from adolescence–to adulthood, the prevention of adult obesity cannot rely solely on identification of a high-risk group in childhood.[3]

Longitudinal U.S. Studies

We examined 19 fair- or good-quality retrospective or prospective observational studies (in 20 publications) from the United States (Table J-1) reporting on the relationship of different childhood overweight measures (BMI values or percentiles), ponderal index (values or percentiles), skinfolds (triceps and/or subscapular), and/or relative weight (one study) to adult measures of overweight and obesity (usually based on BMI or skinfolds). Few studies used ponderal index or relative weight. Studies that compared ponderal index or skinfolds to BMI showed consistent superiority for BMI in measurement correlations between childhood and adulthood (Table J-1). Since these measures are not as widely used clinically and have not been shown to be superior to BMI, which was confirmed in the studies reviewed here, we limit our discussion to BMI values or percentiles. Studies used a variety of reference populations for age- and sex-specific childhood BMI percentiles, including some listed in Appendix R and the 2000

CDC sex-specific BMI-for-age charts, and also used several definitions for adult overweight based on BMI.

General Trends in the Data

Tracking. Across a normal range of childhood BMI levels, early adult (37 years or less) BMI levels tend to correlate more strongly with childhood BMI levels measured at older ages than younger ages, and slightly better in boys than girls.[4] With a few exceptions, BMI at age 12 or above correlated above 0.6 with adult values up to age 37 in white boys and girls, with somewhat lower values generally (but not always) seen in girls. One study among black children showed strikingly lower correlations of childhood BMI at ages 7 and 14[5] compared with whites. Another study showed better correlation of BMI at ages 4-7 and ages 13-17 with adult BMI in black children than in whites, particularly in adolescent black females.[6] Because of the way the data were reported, many studies did not contribute much to the understanding of differences in the strength of the relationship between childhood and adulthood at different ages. This is particularly true for non-BMI measures, such as triceps skinfold (TSF). Correlations of TSF measures in childhood and adulthood were not available for those between two and nine, except in a small study[7] in which very modest correlations (0.35 for boys and 0.18 for girls) were seen between TSF at or above the 85th percentile at age seven and the same value at age 25. In studies that reported both BMI and TSF in white boys and girls (ages 9-18) and in adulthood (ages 20-35), TSF had lower correlations (0.26-0.58) than BMI (0.58-0.91) with the same adult measure,[8] across all ages and in boys as well as girls.

Risks or odds of overweight in adulthood. No large U.S. studies provide relative risks for adult obesity across the age ranges 2-18 years. Guo et al 1994[9] compiled data from four cohort studies and computed the relative risk (RR) of overweight at age 35 (BMI >28 in males and >26 in females) in white adults who were measured in the age-specific 95th percentile of BMI or more vs. the 75th percentile or less at ages 3, 8, 13, and 18 years. RRs did not differ significantly between boys and girls and were modest, with non-significant trends upward with age (1.37 at age 3, 2.03 at age 8, 2.57 at age 18, and 6.05 at age 18 in boys). Statistically significant differences were seen only between those at age 18 and those at age 8 or younger.

In a retrospective HMO medical record cohort study, much higher odds (7.9 to 44.3) were seen for adult obesity (BMI 27.8 or greater for males and 27.3 or greater in females) at age 30 or younger for white adults whose childhood BMIs measured at or above the 95th percentile compared to all others.[10] Odds ratios tended to increase non-significantly with age but with no statistically significant differences, due to very wide, overlapping 95% confidence intervals. Since this was not estimating a rare condition, odds ratios in this case are not comparable with relative risks. However, this study importantly indicated that parental obesity is an important moderator, as the odds of young adult overweight were at least doubled for non-obese and obese children at all ages when one or more parent was obese.

BMI changes over time. Few U.S. studies examine the impact of change in adiposity in childhood or timing of adiposity rebound in adult overweight, although experts have recently noted three times in childhood (prenatal period, period of adiposity rebound [AR], and adolescence) as potentially critical in overweight development.[11] (Adiposity rebound is the nadir of the longitudinal growth curve for an individual child occurring, on average, at 5-6 years). In a

retrospective cohort study among predominately non-Hispanic white, primarily middle and upper-middle class, long-term HMO members, obesity rates in early adulthood (ages 21-29) were higher (25%) in children with earlier age (before 4.8 years) at AR compared to those with a later rebound (10%).[12] Controlling for the other factors associated with increased prevalence of early adult obesity (parental obesity, BMI at time of AR, and sex), boys and girls with early age at AR had an increased odds (6.0, 95% CI 1.3-26.6) of adult obesity compared with those whose AR occurred at 6.2 years or after. Paternal obesity and maternal obesity independently increased the odds of adult obesity in the offspring (OR 4.1, 95% CI 1.5-11.4 and OR 3.2, 95% CI 1.1-9.5, respectively).

Mean age of AR for boys and girls and correlations between age at AR and adult BMI were similar in this study and in a study of the older Fels longitudinal cohort consisting of mostly white individuals.[13] Using serial records of childhood weight and height measures in the Fels cohort, children who reached a BMI > 25 before age 25 compared to those who never did, or who reached it after age 25, were not reported to have an earlier age at adiposity rebound, but had a steeper slope of weight and increase in BMI after AR.[14] Particularly in girls who reached an adult overweight BMI before age 25, the BMI pattern from ages two through six showed little evidence of the usual decline and rebound seen in normal or later-onset overweight children. It should be noted that the adult definition of overweight (BMI > 25) was applied to these children, which corresponds to BMI percentile for both males and females in the 75-85th percentile at age 18 for males (the mean age at which this BMI was reached), in the 90-95th percentile at age 14 (one standard deviation), and substantially above the 95th percentile at age 10 (two standard deviations).[15]

Bogalusa Heart Study investigators confirmed the relationship between early AR (age five or before) and increased adult weight. However, this relationship was not independent of childhood BMI at age seven or eight years.[4] Exploratory analyses suggested that childhood height between the 25th and 75th percentile at age five appeared to modestly modify the relationship of childhood BMI at age 5 to young adult BMI.

Probability of adult obesity. Table J-1 examines the probability of adult obesity for children by BMI percentile at ages 3-18. For black and white children between 5 and 17 years of age, there is a low probability of adult obesity for those below the 50th percentile.[16] For those between the 85th and 94th percentiles (at risk for overweight), about half were found to be obese at age 18-37. The probability of obesity was relatively high (0.7) for children who measured in the overweight category (BMI $\geq$95th percentile) between ages 5 and 17 years. By combining age groups, these data do not tell us much about the differences in the probability of overweight between younger and older children with BMIs at the 95th percentile or above. Data on whites only from the Fels longitudinal study (1929 through 1960) suggests that the probability of adult obesity at age 35 does not exceed 0.5 for children at any of the BMI cutpoints examined before age 13.[17] After age 13, white boys at the 95th percentile or above had a relatively high probability of adult obesity (0.5-0.8), with similar results for white girls (0.64-0.68). This was not a large sample, and there were no confidence intervals reported for these estimates. Looking at the same sample another way, researchers found very poor sensitivity and moderate specificity for the "optimal" BMI cutpoint (based on maximizing the area under the curve) of the 72nd percentile at age 18 for detecting those that became obese at age 35. Of note, based on analysis of a series of ROC curves, the best overall test performance using BMI percentiles to "screen" for adult obesity was in children aged 18.

Children aged 2-5. There is limited data on tracking. A study of 555 white three-year-olds with mean BMI of 16 (1.2) for males and 16 (1.4) for females (around the 50th percentile)[15] found a poor correlation with overweight at age 35 (defined as BMI >28 in males and BMI >26 in females) with r = 0.18 and 0.22 in males and females, respectively.[9] In the same study, three-year-old boys and girls at the 95th percentile (NHANES II) had only a 0.2 probability of overweight at age 35. Using a portion of the same cohort (n=347) in a later study using CDC growth chart definitions for childhood BMI percentiles,[17] children in the 95th percentile for BMI at age three had a 0.2-0.24 probability of adult obesity (BMI 30 or greater), but a much higher probability (0.7 in males and 0.58 in females) of being overweight (BMI 25 or greater). This series of reports over time (from the same research group using essentially the same set of data) illustrates that no matter what BMI-based definition is used for childhood overweight, the tracking into adulthood for this age is minimal. However, the apparent probability of overweight varied substantially (from 0.18 to 0.7 in males and from 0.22 to 0.58 in females) depending on how broadly adulthood overweight was defined.

In a commonly cited separate study addressing predominantly white three- to six-year-old boys and girls, those with BMIs in the 85th percentile or above (NHANES I-II) had an increased risk of overweight (BMI 27.8 or above in males and 27.3 or above in females) (RR of 4.1, 95% CI 2.5-6.7).[10] These elevated relative risks can be somewhat misleading when absolute risk information is not also presented. The absolute risk here was 19% (14 of 73 children with BMIs at the 85th percentile or above became overweight as adults). For young children above the 95th percentile, 52% (14/27) were overweight at age 35.

Children aged 6-11. Tracking data between childhood and adult BMI measures that have been examined for children aged 3-7, 4-7, 7-8, and 9-10 are relevant to this age group. In general, correlations between BMIs measured in predominantly white 6-11-year-olds and again as adults up to age 37 range from 0.36 to 0.73 in males and from 0.21 to 0.63 in females. The few comparable studies done in blacks suggest correlations between 0.28 and 0.68 in males and 0.28 and 0.65 in females. There is not enough data to clearly determine racial/ethnic differences. Considering probability of adult obesity from BMI measures at this age, limited data in whites suggest a probability at or below 0.5. Higher probabilities were reported for overweight (defined as BMI > 27) in black and white boys at age 10 from the Bogalusa Heart Study. Boys with BMI values of 18 had about a 0.5 probability of overweight 15 years later, as did girls with BMI values of 20 (white girls) and 17 (black girls). These BMI values convert to about the 75th percentile for black and white boys, the 85th percentile for white girls, and the 50th percentile for black girls on the 2000 CDC growth charts.[15]

Adolescents aged 12-18. Tracking between childhood and adulthood in weight-for-height measures such as BMI increases[18] with attainment of peak height.[19] Therefore it makes sense that adolescents will generally show higher levels of BMI tracking than younger children. However, the data between studies show a range of results, depending on the age of adolescents measured and the age of measurement in adulthood. Presumably some of this variability also reflects a mixture of pubertal stages in younger adolescents. By age 17-18, most children can be assumed to have gone through puberty. BMIs in white children aged 17-18 years correlate with adult BMIs up to age 35 between 0.58 and 0.81 for males and 0.63 to 0.81 for females.[19-22] Data for black children separately are limited to boys and girls at age 13 and 14[5] or 13-17.[6] In these studies, young black adolescents' BMIs correlated 0.37-0.72 with their BMIs at age 28-32.

These values were the same or better than the correlation in white children in the only study that measured and reported results for both black and white children.[6] Considering probability of adult overweight, there are more data available for this age group than for younger children. After age 13, white boys at or above the 95th percentile had a relatively high probability of adult obesity (0.5-0.8), with similar results for white girls (0.64-0.68). The Bogalusa Heart Study found that boys with BMI values of 22 had about a 0.5 probability of overweight 15 years later, as did girls with BMI values of 25 (white girls) and 23 (black girls).[23] Using the CDC 2000 charts, these BMI values convert to the 75th percentile for boys, between the 85th and 90th percentiles for white girls, and between the 75th and 85th percentiles for black girls.

References

1. Reilly JJ, Methven E, McDowell ZC, Hacking B, Alexander D, Stewart L et al. Health consequences of obesity. *Arch Dis Child* 2003; 88(9):748-752.

2. Serdula MK, Ivery D, Coates RJ, Freedman DS, Williamson DF, Byers T. Do obese children become obese adults? A review of the literature. *Prev Med* 1993; 22(2):167-177.

3. Power C, Lake JK, Cole TJ. Measurement and long-term health risks of child and adolescent fatness. *Int J Obes Relat Metab Disord* 1997; 21(7):507-526.

4. Freedman DS, Khan LK, Serdula MK, Dietz WH, Srinivasan SR, Berenson GS. Inter-relationships among childhood BMI, childhood height, and adult obesity: the Bogalusa Heart Study. *International Journal of Obesity & Related Metabolic Disorders* 2004; 28(1):10-16.

5. Hulman S, Kushner H, Katz S, Falkner B. Can cardiovascular risk be predicted by newborn, childhood, and adolescent body size? An examination of longitudinal data in urban African Americans. *Journal of Pediatrics* 1998; 132(1):90-97.

6. Wattigney WA, Webber LS, Srinivasan SR, Berenson GS. The emergence of clinically abnormal levels of cardiovascular disease risk factor variables among young adults: the Bogalusa Heart Study. *Preventive Medicine* 1995; 24(6):617-626.

7. Garn SM, Lavelle M. Two-decade follow-up of fatness in early childhood. *Am J Dis Child* 1985; 139(2):181-185.

8. Lauer RM, Clarke WR, Burns TL. Obesity in childhood: the Muscatine Study. *Chung-Hua Min Kuo Hsiao Erh Ko i Hsueh Hui Tsa Chih* 1997; 38(6):432-437.

9. Guo SS, Roche AF, Chumlea WC, Gardner JD, Siervogel RM. The predictive value of childhood body mass index values for overweight at age 35 y. *American Journal of Clinical Nutrition* 1994; 59(4):810-819.

10. Whitaker RC, Wright JA, Pepe MS, Seidel KD, Dietz WH. Predicting obesity in young adulthood from childhood and parental obesity. *N Engl J Med* 1997; 337(13):869-873.

11. Dietz WH. Health consequences of obesity in youth: childhood predictors of adult disease. *Pediatrics* 1998; 101(3 Pt 2):518-525.

12. Whitaker RC, Pepe MS, Wright JA, Seidel KD, Dietz WH. Early adiposity rebound and the risk of adult obesity. *Pediatrics* 1998; 101(3):E5.

13. Siervogel RM, Roche AF, Guo SM, Mukherjee D, Chumlea WC. Patterns of change in weight/stature2 from 2 to 18 years: findings from long-term serial data for children in the Fels longitudinal growth study. *Int J Obes* 1991; 15(7):479-485.

14. Wisemandle W, Maynard LM, Guo SS, Siervogel RM. Childhood weight, stature, and body mass index among never overweight, early-onset overweight, and late-onset overweight groups. *Pediatrics* 2000; 106(1):E14.

15. Kuczmarski RJ, Ogden CL, Guo SS, Grummer-Strawn LM, Flegal KM, Mei Z et al. 2000 CDC Growth Charts for the United States: methods and development. *Vital Health Stat 11* 2002;(246):1-190.

16. Freedman DS, Khan LK, Dietz WH, Srinivasan SR, Berenson GS. Relationship of childhood obesity to coronary heart disease risk factors in adulthood: the Bogalusa Heart Study. *Pediatrics* 2001; 108(3):712-718.

17. Guo SS, Wu W, Chumlea WC, Roche AF. Predicting overweight and obesity in adulthood from body mass index values in childhood and adolescence.[comment]. *American Journal of Clinical Nutrition* 2002; 76(3):653-658.

18. Freedman DS, Shear CL, Burke GL, Srinivasan SR, Webber LS, Harsha DW et al. Persistence of juvenile-onset obesity over eight years: the Bogalusa Heart Study. *American Journal of Public Health* 1987; 77(5):588-592.

19. Casey VA, Dwyer JT, Coleman KA, Valadian I. Body mass index from childhood to middle age: a 50-y follow-up. *Am J Clin Nutr* 1992; 56(1):14-18.

20. Lauer RM, Lee J, Clarke WR. Factors affecting the relationship between childhood and adult cholesterol levels: the Muscatine Study. *Pediatrics* 1988; 82(3):309-318.

Appendix J. Childhood Overweight Clinical Screening Tests and Predicting Adult Obesity (continued)

21. Lauer RM, Clarke WR. Childhood risk factors for high adult blood pressure: the Muscatine Study. *Pediatrics* 1989; 84(4):633-641.

22. Clarke WR, Lauer RM. Does childhood obesity track into adulthood? *Crit Rev Food Sci Nutr* 1993; 33(4-5):423-430.

23. Valdez R, Greenlund KJ, Wattigney WA, Bao W, Berenson GS. Use of weight-for-height indices in children to predict adult overweight: the Bogalusa Heart Study. *International Journal of Obesity & Related Metabolic Disorders* 1996; 20(8):715-721.

24. Sinaiko AR, Donahue RP, Jacobs DR, Jr., Prineas RJ. Relation of weight and rate of increase in weight during childhood and adolescence to body size, blood pressure, fasting insulin, and lipids in young adults. The Minneapolis Children's Blood Pressure Study. *Circulation* 1999; 99 (11):1471-1476.

25. Gortmaker SL, Must A, Perrin JM, Sobol AM, Dietz WH. Social and economic consequences of overweight in adolescence and young adulthood. *New England Journal of Medicine* 1993; 329 (14):1008-1012.

26. Srinivasan SR, Bao W, Wattigney WA, Berenson GS. Adolescent overweight is associated with adult overweight and related multiple cardiovascular risk factors: the Bogalusa Heart Study. *Metabolism: Clinical & Experimental* 1996; 45 (2):235-240.

27. Lauer RM, Clarke WR, Mahoney LT, Witt J. Childhood predictors for high adult blood pressure. The Muscatine Study. *Pediatric Clinics of North America* 1993; 40 (1):23-40.

28. Webber LS, Cresanta JL, Croft JB, Srinivasan SR, Berenson GS. Transitions of cardiovascular risk from adolescence to young adulthood--the Bogalusa Heart Study: II. Alterations in anthropometric blood pressure and serum lipoprotein variables. *Journal of Chronic Diseases* 1986; 39 (2):91-103.

Table J-1. Evidence Table: Relationship between Childhood Overweight and Adult Overweight

Author QUALITY	N	% White	% Female	Age		OW Defined as			M	F	Measure	Reference Standard
				Initial	Follow-up	Youth	BMI Range (Youth)	Adult				
BMI (kg/m^2): continuous												
Freedman et al 2004[4] **FAIR**	885	67% overall; NR for this age group	62%	3 to 7	18 to 37 (mean: 25)	BMI	NR: mean BMI for study pop is 17.7+/-3.4	BMI	0.59	0.51	Correlation	N/A
								TSF + SSF (sum)	0.47	0.38		
Wattigney et al 1995[6]	222	100%	57%	4 to 7	19 to 22	BMI	NR	BMI	0.43	0.43	Correlation	N/A
FAIR	132	100% black	58%						0.68	0.65		
Casey et al 1992a[19] **FAIR**	114	100%	50% overall; NR this age group	5 to 7	30	BMI	Mean BMI: 15.4 +/-.9 (m); 15.7 +/- 1.5 (f)	BMI	0.41	0.21	Correlation	N/A
	104				40				0.36	-0.03		
	84				50				0.41	0.05		
Freedman et al 2001[16] **FAIR**	2617	67% white, 32% black	57%	5 to 17	18 to 37	BMI	Mean childhood BMI: 17.8 +/- 3.5	BMI	0.58		Correlation	N/A
Hulman et al 1998b[5] **FAIR**	137	0% white; 100% black	49%	7	28	BMI	Mean BMI 16.7 +/-3.0 (m); 16.4 +/- 2.4 (f)	BMI	0.28		Correlation	N/A
Sinaiko et al 1999[24] **FAIR**	679	66% white, 25% black, 4% NA	48%	8	24	BMI	8 to 34	BMI	0.612		Correlation	N/A
Freedman et al 2004[4] **FAIR**	2444	67% overall; NR for this age group	56%	8 to 13	18 to 37	BMI: continuous	NR: mean BMI for study pop is 17.7+/-3.4	BMI (continuous)	0.68	0.63	Correlation	N/A
								TSF + SSF (sum)	0.53	0.49		

Table J-1. Evidence Table: Relationship between Childhood Overweight and Adult Overweight

Author QUALITY	N	% White	% Female	Age		OW Defined as			M	F	Measure	Reference Standard
				Initial	Follow-up	Youth	BMI Range (Youth)	Adult				
Valdez et al 1996d[23] **FAIR**	835	68%	54%	10 to 15	25 to 30	BMI	Means: 19.1+/- .23 (wm); 18.5 +/-0.33 (bm); 18.9 +/-0.21 (wf); 19.6 +/-0.30 (bf)	BMI > 27	0.64		Correlation	N/A
									1.65 (1.48-1.84)	1.58 (1.43-1.74)	RR or OR	
Hulman et al 1998b[5] **FAIR**	137	100% black	49%	13	28	BMI	Mean BMI 19.7 +/- 4.1 (m); 20.6 +/- 4.2 (f)	BMI	0.37		Correlation	N/A
Wattigney et al 1995[6] **FAIR**	327	100%	51%	13 to 17	28 to 32	BMI: kg/m^2	NR	BMI: kg/m^2	0.63	0.48	Correlation	N/A
	147	100% black	55%						0.69	0.72		
Hulman et al 1998b[5] **FAIR**	137	100% black	49%	14	28	BMI	Mean BMI 20.4 +/- 4.4 (m); 22.2 +/- 4.8 (f)	BMI	0.13		Correlation	N/A
Freedman et al 2004[4] **FAIR**	2212	67% overall; NR for this age group	56%	14 to 17	18 to 37	BMI: continuous	NR: mean BMI for study pop is17.7+/- 3.4	BMI: continuous	0.76	0.73	Correlation	N/A
								TSF + SSF sum	0.57	0.56		
Casey et al 1992[19] **FAIR**	95	100%	50% overall; NR this age group	18	30	BMI (W/H^2)	Mean BMI: 21.3 +/- 1.9 (m); 20.7 +/- 2.6 (f)	BMI (W/H^2)	0.67	0.66	Correlation	N/A
	84				40				0.61	0.62		
	67				50				0.51	0.44		

Table J-1. Evidence Table: Relationship between Childhood Overweight and Adult Overweight

Author QUALITY	N	% White	% Female	Age		OW Defined as			M	F	Measure	Reference Standard
				Initial	Follow-up	Youth	BMI Range (Youth)	Adult				
BMI %ile (kg/m^2): continuous												
Guo et al 1994[9] **FAIR**	555	100%	50%	3	35	BMI	Mean baseline BMI range: 16+/-1.2 (3 yo m) to 25 +/- 3.4 (18 yo m); 16+/- 1.4 (3 yo f) to 26 +/- 5.0 (18 yo f)	BMI	0.18	0.22	Correlation	N/A
Lauer et al 1988, Lauer and Clarke 1989[20,21] **FAIR**	109 obs	100%	52%	7 to 8	20 to 25	W/H^2: assume kg/m^2	Approx 17	W/H^2 (QI): assume kg/m^2	0.57	0.45	Correlation	N/A
Guo et al 1994[9] **FAIR**	555	100%	50%	8	35	BMI	Mean baseline BMI range: 16+/-1.2 (3 yo m) to 25 +/- 3.4 (18 yo m); 16+/- 1.4 (3 yo f) to 26 +/- 5.0 (18 yo f)	BMI	0.28	0.60	Correlation	N/A
Clarke and Lauer 1993c[22] **FAIR**	1286 obs	100%	53%	9 to 10	21 to 25	BMI	Baseline NR	BMI	0.61	0.59	Correlation	N/A
	304 obs		58%		26 to 30				0.73	0.6		
Lauer et al 1988, Lauer and Clarke 1989[20,21] **FAIR**	603 obs	100%	52%	9 to 10	20 to 25	W/H^2: assume kg/m^2	Approx 18	W/H^2 (QI): assume kg/m^2	0.63	0.61	Correlation	N/A
Lauer et al 1997[8] **FAIR**	2631	100%	NR	9 to 18	23 to 33	BMI	NR	BMI	.58 - .91		Correlation	N/A
Lauer et al 1988, Lauer and Clarke 1989[20,21] **FAIR**	*1018 obs	100%	53%	11 to 12	20 to 25	W/H^2: assume kg/m^2	Approx 20	W/H^2 (QI): assume kg/m^2	0.67	0.65	Correlation	N/A

Table J-1. Evidence Table: Relationship between Childhood Overweight and Adult Overweight

Author QUALITY	N	% White	% Female	Age		OW Defined as			M	F	Measure	Reference Standard
				Initial	Follow-up	Youth	BMI Range (Youth)	Adult				
Guo et al 1994[9] **FAIR**	555	100%	50%	13	35	BMI	Mean baseline BMI range: 16+/-1.2 (3 yo m) to 25 +/- 3.4 (18 yo m); 16+/- 1.4 (3 yo f) to 26 +/- 5.0 (18 yo f)	BMI	0.50	0.65	Correlation	N/A
Lauer et al 1988, Lauer and Clarke 1989[20,21] **FAIR**	1041 obs	100%	53%	13 to 14	20 to 25	W/H^2: assume kg/m^2	Approx 21	W/H^2 (QI): assume kg/m^2	0.64	0.68	Correlation	N/A
					26 to 30				0.74	0.67		
Clarke and Lauer 1993c[22] **FAIR**	1104 obs	100%	54%	13 to 14	21-25	BMI	Baseline NR	BMI	0.7	0.7	Correlation	N/A
	713 obs		56%		26 to 30				0.79	0.68		
	104 obs		52%		31-35				0.91	0.77		
Lauer et al 1988, Lauer and Clarke 1989[20,21] **FAIR**	767 obs	100%	54%	15 to 16	20 to 25	W/H^2: assume kg/m^2	Approx 22	W/H^2 (QI): assume kg/m^2	0.68	0.68	Correlation	N/A
	568 obs				26 to 30				0.66	0.69		
Clarke and Lauer 1993c[22] **FAIR**	631 obs	100%	52%	17 to 18	21 to 25	BMI	Baseline NR	BMI	0.81	0.72	Correlation	N/A
	676 obs		52%		26 to 30				0.77	0.7		
	218 obs		51%		31-35				0.58	0.63		
Lauer et al 1988, Lauer and Clarke 1989[20,21] **FAIR**	615 obs	100%	51%	17 to 18	20 to 25	W/H^2: assume kg/m^2	Approx 23	W/H^2 (QI): assume kg/m^2	0.74	0.73	Correlation	N/A
	469 obs		52%		26 to 30				0.68	0.52		

Table J-1. Evidence Table: Relationship between Childhood Overweight and Adult Overweight

Author QUALITY	N	% White	% Female	Age		OW Defined as			M	F	Measure	Reference Standard
				Initial	Follow-up	Youth	BMI Range (Youth)	Adult				
Guo et al 1994[9] **FAIR**	555	100%	50%	18	35	BMI	Mean baseline BMI range: 16+/-1.2 (3 yo m) to 25 +/- 3.4 (18 yo m); 16+/- 1.4 (3 yo f) to 26 +/- 5.0 (18 yo f)	BMI	0.65	0.77	Correlation	N/A
BMI ≥ 95th %ile												
Freedman et al 2001[16] **FAIR**	60	67% white, 32% black in whole pop	57% overall; NR in this age group	< 8	18 to 37	BMI ≥ 95%ile	N/A	BMI ≥ 30	87%		% OC who became OA	CDC / NCHS growth charts for 1963-94
Whitaker et al 1997[10] **FAIR**	27	94% overall	64% overall; NR this age group	3 to 5	21 to 29	BMI ≥ 95%ile	N/A	BMI > 27.8 (m), 27.3 (f)	7.9 (3.6 - 17.3)		RR or OR	NHANES I-II
Freedman et al 2001[16] **FAIR**	186 581 adults BMI > 30	67% white, 32% black overall	57% overall; NR in this age group	5 to 17	18 to 37	BMI ≥ 95%ile	N/A	BMI ≥ 30	77% 25%	 -	% OC who became OA % OA who were OC	CDC / NCHS growth charts for 1963-94
Whitaker et al 1997[10] **FAIR**	35 29 30	94% overall, NR this age group	64% overall, NR this age group	6 to 9 10 to 14 15 to 17	21 to 29	BMI ≥ 95%ile	N/A	BMI ≥ 27.8 (m), 27.3 (f)	18.5 (8.8-38.8) 44.3 (16.3 - 120) 32.5 (13.1 - 80.6)		RR or OR	NHANES I-II
Gortmaker et al 1993[25] **FAIR**	10,039; 56-79% follow up by question	80% white, 14% black, 6% Hispanic full cohort	100%	16 to 24	23 to 31	BMI > 95th %ile for age, sex	N/A	BMI > 95th %ile for age, sex	77%	66%	% OC who became OA	NHANES I

Table J-1. Evidence Table: Relationship between Childhood Overweight and Adult Overweight

Author QUALITY	N	% White	% Female	Age		OW Defined as			M	F	Measure	Reference Standard
				Initial	Follow-up	Youth	BMI Range (Youth)	Adult				
BMI at 95th %ile												
Guo et al 1994e[9] **FAIR**	555 in whole cohort	100%	50%	3	35	BMI at the 95th %ile	N/A	BMI > 28 (m), > 26 (f)	0.21	0.20	Other	NHANES II
Guo et al 2002e[17] **FAIR**	347 whole cohort	100%	52% overall	3	35	BMI at the 95th %ile	N/A	BMI ≥ 25	0.71	0.58	Other	CDC growth charts NHANES I-III
								BMI ≥ 30	0.15	0.24		
Guo et al 1994e[9] **FAIR**	555 in whole cohort	100%	50%	8	35	BMI at the 95th %ile	N/A	BMI > 28 (m), > 26 (f)	0.37	0.39	Other	NHANES II
Guo et al 2002e[17] **FAIR**	347 whole cohort	100%	52% overall	8	35	BMI at the 95th %ile	N/A	BMI ≥ 25	0.72	0.76	Other	CDC growth charts NHANES I-III
								BMI ≥ 30	0.22	0.46		
Guo et al 1994e[9] **FAIR**	555 in whole cohort	100%	50%	13	35	BMI at the 95th %ile	N/A	BMI > 28 (m), > 26 (f)	0.40	0.32	Other	NHANES II
Guo et al 2002e[17] **FAIR**	347 whole cohort	100%	52% overall	13	35	BMI at the 95th %ile	N/A	BMI ≥ 25	0.91	0.92	Other	CDC growth charts NHANES I-III
								BMI ≥ 30	0.46	0.64		
Guo et al 1994e[9] **FAIR**	555 in whole cohort	100%	50%	18	35	BMI at the 95th %ile	N/A	BMI > 28 (m), > 26 (f)	0.78	0.66	Other	NHANES II
Guo et al 2002e[17] **FAIR**	347 whole cohort	100%	52% overall	18	35	BMI at the 95th %ile	N/A	BMI ≥ 25	0.98	0.95	Other	CDC growth charts NHANES I-III
								BMI ≥ 30	0.77	0.68		

Table J-1. Evidence Table: Relationship between Childhood Overweight and Adult Overweight

Author QUALITY	N	% White	% Female	Age		OW Defined as			M	F	Measure	Reference Standard
				Initial	Follow-up	Youth	BMI Range (Youth)	Adult				
BMI 85th-94th %ile												
Freedman et al 2001[16]	2617	67% white, 32% overall	57% overall; NR in this age group	5 to 17	18 to 37	BMI 85-94th %ile	N/A	BMI ≥ 30	51%		% OC who became OA	CDC / NCHS growth charts for 1963-94
FAIR	581 adults BMI ≥ 30								22%	-	% OA who were OC	
BMI ≥ 85th %ile												
Whitaker et al 1997[10]	86	94% overall	64% overall	3 to 5	21 to 29	BMI ≥ 85th%ile	N/A	BMI ≥ 27.8 (m), 27.3 (f)	4.1 (2.5-6.7)		RR or OR	NHANES I-II
FAIR	NR:w/ parental data too (f)								4.7 (2.5 - 8.8)			
	80			6 to 9					10.3 (6.2 - 17.3)			
	NR:w/ parental data too (f)								8.8 (4.7 - 16.5)			
	61			10 to 14					28.3 (15.0-35.5)			
	NR:w/ parental data too (f)								22.3 (10.5 - 47.1)			
	55			15 to 17					20.3 (10.4-39.6)			
	NR:w/ parental data too (f)								17.5 (7.7 - 39.5)			

Table J-1. Evidence Table: Relationship between Childhood Overweight and Adult Overweight

Author QUALITY	N	% White	% Female	Age		OW Defined as			M	F	Measure	Reference Standard
				Initial	Follow-up	Youth	BMI Range (Youth)	Adult				
BMI at 85th %ile												
Guo et al 2002e[17] **FAIR**	347 whole cohort	100%	52% whole cohort	3	35	85th %ile	N/A	BMI ≥ 25	0.59	0.45	Other	CDC growth charts NHANES I-III
								BMI ≥ 30	0.12	0.17		
				8				BMI ≥ 25	0.57	0.54		
								BMI ≥ 30	0.14	0.23		
				13				BMI ≥ 25	0.71	0.66		
								BMI ≥ 30	0.22	0.27		
				18				BMI ≥ 25	0.87	0.72		
								BMI ≥ 30	0.33	0.26		
BMI ≥ 80th %ile (top quintile)												
Clarke and Lauer 1993c[22] **FAIR**	1286 obs	100%	53%	9 to 10	21 to 25	BMI ≥ 80th %ile	N/A	BMI ≥ 80th %ile	62%	65%	% OC who became OA	Muscatine study population
	304 obs		58%		26 to 30				65%	65%		
Lauer et al 1997[8] **FAIR**	2631	100%	NR	9 to 18	23 to 33	BMI ≥ 80th %ile	N/A	BMI ≥ 80th %ile	48-75%		% OC who became OA	age, sex, year specific %iles: study pop; also NHANES II
Clarke and Lauer 1993c[22] **FAIR**	1104 obs	100%	54%	13 to 14	21 to 25	BMI ≥ 80th %ile	N/A	BMI ≥ 80th %ile	58%	68%	% OC who became OA	Muscatine study population
	713 obs		56%		26 to 30				70%	65%		
	104 obs		52%		31 to 35				75%	87%		
	631 obs		52%	17 to 18	21 to 25	BMI ≥ 80th %ile	N/A	BMI ≥ 80th %ile	75%	62%		Muscatine study population
	676 obs		52%		26 to 30				65%	59%		
	218 obs		51%		31 to 35				62%	50%		

Table J-1. Evidence Table: Relationship between Childhood Overweight and Adult Overweight

Author QUALITY	N	% White	% Female	Age		OW Defined as			M	F	Measure	Reference Standard
				Initial	Follow-up	Youth	BMI Range (Youth)	Adult				
BMI > 75th %ile												
Srinivasan et al 1996[26] **FAIR**	191	33% w m, 35% w f, 13% b m, 19% b f	NR	13 to 17	27 to 31	BMI > 75th %ile	N/A	BMI > 75th %ile: kg/m^2	58%	57%	% OC who became OA	in-study
		100% black	0%						52%	-		
		100% black	100%						-	62%		
BMI at 75th %ile												
Guo et al 1994e[9] **FAIR**	555 in whole cohort	100%	50%	3	35	BMI at the 75th %ile	N/A	BMI > 28 (m), > 26 (f)	0.16	0.15	Other	NHANES II
Guo et al 2002e[17] **FAIR**	347 whole cohort	100%	52% overall	3	35	BMI at the 75th %ile	N/A	BMI ≥ 25	0.53	0.38	Other	CDC growth charts NHANES I-III
								BMI ≥ 30	0.1	0.14		
Guo et al 1994e[9] **FAIR**	555 in whole cohort	100%	50%	8	35	BMI at the 75th %ile	N/A	BMI > 28 (m), > 26 (f)	0.23	0.20	Other	NHANES II
Guo et al 2002e[17] **FAIR**	347 whole cohort	100%	52% overall	8	35	BMI at the 75th %ile	N/A	BMI ≥ 25	0.49	0.44	Other	CDC growth charts NHANES I-III
								BMI ≥ 30	0.12	0.16		
Guo et al 1994e[9] **FAIR**	555 in whole cohort	100%	50%	13	35	BMI at the 75th %ile	N/A	BMI > 28 (m), > 26 (f)	0.21	0.19	Other	NHANES II
Guo et al 2002e[17] **FAIR**	347 whole cohort	100%	52% overall	13	35	BMI at the 75th %ile	N/A	BMI ≥ 25	0.58	0.48	Other	CDC growth charts NHANES I-III
								BMI ≥ 30	0.15	0.16		

Table J-1. Evidence Table: Relationship between Childhood Overweight and Adult Overweight

Author QUALITY	N	% White	% Female	Age		OW Defined as			M	F	Measure	Reference Standard
				Initial	Follow-up	Youth	BMI Range (Youth)	Adult				
Guo et al 1994e[9] **FAIR**	555 in whole cohort	100%	50%	18	35	BMI at the 75th %ile	N/A	BMI > 28 (m), > 26 (f)	0.37	0.32	Other	NHANES II
Guo et al 2002e[17] **FAIR**	347 whole cohort	100%	52% overall	18	35	BMI at the 75th %ile	N/A	BMI ≥ 25	0.72	0.52	Other	CDC growth charts NHANES I-III
								BMI ≥ 30	0.17	0.15		
BMI > 72nd %ile												
Guo et al 2002[17] **FAIR**	78	100%	52% overall	18	35	≥ 72nd %ile	N/A	BMI ≥ 30	.36, sens / spec .83, .79	.36, sens / spec .76, .83	Other	CDC growth charts NHANES I-III
									19.3 (5.20, 71.4)	15.7 (4.69, 52.5)	RR or OR	
BMI ≥ 60th %ile												
Guo et al 1994[9] **FAIR**	115	100%	50%	18	35	BMI ≥ 60th %ile	N/A	BMI > 28 (m), > 26 (f)	14.9 (5.01, 52.6)	27.7 (7.35, 151.3)	RR or OR	NHANES II
									0.34: sens .81, spec .77	0.37 (sens .86, spec .81)	Other	
BMI at 60th %ile												
Guo et al 1994[9] **FAIR**	115	100%	50%	18	35	BMI at 60th %ile	N/A	BMI > 28 (m), > 26 (f)	false positive: 18% (m), 17% (f); sensitivity 80%		Other	NHANES II

Table J-1. Evidence Table: Relationship between Childhood Overweight and Adult Overweight

Author QUALITY	N	% White	% Female	Age		OW Defined as			M	F	Measure	Reference Standard
				Initial	Follow-up	Youth	BMI Range (Youth)	Adult				
BMI > 50th %ile												
Freedman et al 2001g[16] **FAIR**	2617	67% white, 32% black	57% overall; NR in this age group	5 to 17	18 to 37	BMI < 50%ile	N/A	BMI ≥ 30	7%		% OC who became OA	CDC / NCHS growth charts for 1963-94
Freedman et al 2001h[16] **FAIR**	581 adults BMI ≥30	67% white, 32% black in whole pop	57% overall; NR in this age group	5 to 17	18 to 37	BMI < 50%ile	N/A	BMI ≥ 30	17%		% OA who were OC	CDC / NCHS growth charts for 1963-94
Guo et al 2002[17] **FAIR**	140	100%	52% overall	18	35	≥ 50th %ile	N/A	BMI ≥ 25	12.1 (5.49, 27.3)	7.92 (3.61, 17.4)	RR or OR	CDC growth charts NHANES I-III
									.69, sens/spec .83, .72	.56, sens/spec .76, .73	Other	
BMI at 50th %ile												
Guo et al 1994e[9] **FAIR**	555 in whole cohort	100%	50%	3	35	BMI at the 50th %ile	N/A	BMI > 28 (m), > 26 (f)	0.12	0.10	Other	NHANES II
				8					0.11	0.08		
				13					0.07	0.09		
				18					0.06	0.07		

Table J-1. Evidence Table: Relationship between Childhood Overweight and Adult Overweight

Author QUALITY	N	% White	% Female	Age		OW Defined as			M	F	Measure	Reference Standard
				Initial	Follow-up	Youth	BMI Range (Youth)	Adult				
BMI: 95th vs. 75th %ile												
Guo et al 1994[9] **FAIR**	555 in whole cohort	100%	50%	3	35	At 95th vs at 75th %ile	N/A	BMI > 28 (m), > 26 (f)	1.37 (0.99, 1.89)	1.41 (1.00, 1.98)	RR or OR	NHANES II
				8					2.03 (1.39, 2.99)	2.45 (1.54, 3.89)		
				13					2.57 (1.76, 3.75)	2.04 (1.41, 2.96)		
				18					6.05 (3.03, 12.08)	4.08 (2.34, 7.12)		
BMI: 95th vs. 50th %ile												
Guo et al 1994[9] **FAIR**	555 in whole cohort	100%	50%	3	35	At 95th vs at 50th %ile	N/A	BMI > 28 (m), > 26 (f)	2.02 (0.98, 4.17)	2.17 (1.01, 4.64)	RR or OR	NHANES II
				8					4.94 (2.08, 11.72)	7.49 (2.64, 21.22)		
				13					8.39 (3.59, 19.61)	5.00 (2.17, 11.52)		
				18					57.46 (12.15, 271.84)	23.69 (6.78, 82.82)		

Table J-1. Evidence Table: Relationship between Childhood Overweight and Adult Overweight

Author QUALITY	N	% White	% Female	Age		OW Defined as			M	F	Measure	Reference Standard
				Initial	Follow-up	Youth	BMI Range (Youth)	Adult				
BMI: 90th vs. 50th %ile												
Lauer et al 1993[27] **FAIR**	About 375 obs	100%	N/A	16	20 to 25	QI %ile > 90 %ile vs 'at the 50th %ile'	N/A	QI %ile > 90	9	10	RR or OR	age, sex, year specific %iles: study pop
	About 375 obs	100%	N/A	16	20 to 25	QI %ile 'at the 90 %ile' vs 'at the 50th %ile'	N/A	QI %ile > 90	9	10	RR or OR	age, sex, year specific %iles: study pop
BMI: 75th vs. 50th %ile												
Guo et al 1994[9] **FAIR**	555 in whole cohort	100%	50%	3	35	At 75th vs at 50th	N/A	BMI > 28 (m), > 26 (f)	1.48 (0.99, 2.21)	1.54 (1.01, 2.35)	RR or OR	NHANES II
				8					2.43 (1.50, 3.92)	3.06 (1.72, 5.46)		
				13					3.26 (2.03, 5.23)	2.44 (1.54, 3.89)		
				18					9.49 (4.00, 22.51)	5.80 (2.90, 11.63)		

Table J-1. Evidence Table: Relationship between Childhood Overweight and Adult Overweight

Author QUALITY	N	% White	% Female	Age		OW Defined as			M	F	Measure	Reference Standard
				Initial	Follow-up	Youth	BMI Range (Youth)	Adult				
BMI: specific values												
Valdez et al 1996[23]	148	100% black	100%	10	30	BMI > 17 kg/m^2	N/A	BMI > 27	-	50%	% OC who became OA	N/A
FAIR	302	100%				BMI > 20 kg/m^2			-	50%		
	121	100% black	0%			BMI > 18 kg/m^2			50%	-		
	264	100%				BMI > 18 kg/m^2			50%	-		
	148	100% black	100%	15	30	BMI > 23 kg/m^2			-	50%		
	302	100%				BMI > 25 kg/m^2			-	50%		
	121	100% black	0%			BMI > 22 kg/m^2			50%	-		
	264	100%				BMI > 22 kg/m^2			50%	-		
W/H^3 (aka Rohrer Index, Ponderal Index)												
Freedman et al 1987i[18]	1490	59%	52%	2 to 14	10 to 24	RI: kg/m^3	Mean at baseline: 13.1 kg/m^3	RI: kg/m^3	0.67 (.64, .70)		Correlation	N/A
FAIR	440	100%	0%						0.70 (.65, .74)			
	432	100%	100%							0.57 (.50, .63)		
	281	100% black	0%						0.72 (.66, .77)			
	337	100% black	100%							0.72 (.66, .77)		

Table J-1. Evidence Table: Relationship between Childhood Overweight and Adult Overweight

Author QUALITY	N	% White	% Female	Age		OW Defined as			M	F	Measure	Reference Standard
				Initial	Follow-up	Youth	BMI Range (Youth)	Adult				
Freedman et al 2004[4] **FAIR**	885	67% overall; NR for this age group	62%	3 to 7	18 to 37	W/H^3	NR: mean BMI for study pop is 17.7+/-3.4	BMI (continuous)	0.49	0.45	Correlation	N/A
								TSF + SSF sum	0.38	0.34		
	2444		56%	8 to 13	18 to 37			BMI (continuous)	0.62	0.61		
								TSF + SSF sum	0.48	0.46		
Freedman et al 1987[18] **FAIR**	255	59% overall	52% overall	9 to 10	18 to 19	RI: kg/m^3	NR by groups	RI: kg/m^3	0.76 (.70, .81)		Correlation	N/A
Webber et al 1986[28] **FAIR**	42	NR: 67% in other BHS	NR: 57% in other BHS	10 to 11	17 to 18	PI: W/H^3	Mean PI at baseline ranged from 11.0 +/- 0.5 to 13.3 +/- 2.1 (m), from 11.6 +/- 1.2 to 16.4 +/- 2.9 (f); varied by age and race; T 1-2	PI: W/H^3	0.93	0.73	Correlation	N/A
Valdez et al 1996j[23] **FAIR**	835	68%	54%	10 to 15	25 to 30	PI	Mean 12.5 +/- 0.13 (wm); 12.1 +/- 0.19 (bm); 12.4 +/- 0.12 (wf); 12.7 +/-0.17 (bf)	BMI > 27	0.64		Correlation	N/A
									2.18 (1.82 - 2.61)	2.04 (1.75 - 2.38)	RR or OR	
Freedman et al 1987[18] **FAIR**	288	59% overall	52% overall	11 to 14	20 to 24	RI: kg/m^3	NR by groups	RI: kg/m^3	0.72 (.66, .77)		Correlation	N/A
	57	100% black	100%							0.83 (.73, .90)		

Table J-1. Evidence Table: Relationship between Childhood Overweight and Adult Overweight

Author QUALITY	N	% White	% Female	Age		OW Defined as			M	F	Measure	Reference Standard
				Initial	Follow-up	Youth	BMI Range (Youth)	Adult				
Webber et al 1986[28] **FAIR**	162	NR: 67% in other BHS	NR: 57% in other BHS	12 to 13	19 to 20	PI: W/H^3	Mean PI at baseline ranged from 11.0 +/- 0.5 to 13.3 +/- 2.1 (m), from 11.6 +/- 1.2 to 16.4 +/- 2.9 (f); varied by age and race; T 1-2	PI: W/H^3	0.82	0.82	Correlation	N/A
	80	NR: 67% in other BHS	NR: 57% in other BHS	14 to 15	21 to 22	PI: W/H^3	Mean PI at baseline ranged from 11.0 +/- 0.5 to 13.3 +/- 2.1 (m), from 11.6 +/- 1.2 to 16.4 +/- 2.9 (f); varied by age and race; T 1-2	PI: W/H^3	0.7	0.91	Correlation	N/A
Freedman et al 2004[4] **FAIR**	2212	67% overall; NR for this age group	56%	14 to 17	18 to 37	W/H^3 (assume kg/m^3)	NR: mean BMI for study pop is17.7+/- 3.4	BMI (continuous) TSF + SSF sum	0.73 0.53	0.72 0.54	Correlation	N/A
W/H^3 > 95 %ile												
Freedman et al 1987[18] **FAIR**	74	59% overall	52% overall	2 to 14	10 to 24	RI > 95th %ile: kg/m^3	N/A	RI > 95th %ile: kg/m^3	72%		% OC who became OA	in-study
W/H^3 > 85 %ile												
Freedman et al 1987[18] **FAIR**	1490	59%	52%	2 to 14	10 to 24	RI > 85th %ile: kg/m^3	N/A	RI > 85th %ile: kg/m^3	50%		% OC who became OA	in-study

Table J-1. Evidence Table: Relationship between Childhood Overweight and Adult Overweight

Author QUALITY	N	% White	% Female	Age		OW Defined as			M	F	Measure	Reference Standard
				Initial	Follow-up	Youth	BMI Range (Youth)	Adult				
W/H^3 specific values												
Valdez et al 1996[23]	148	100% black	100%	10	30	PI > 12 kg/m^3	N/A	BMI > 27	-	50%	% OC who became OA	N/A
FAIR	302	100%	100%			PI >14 kg/m^3			-	50%		
	121	100% black	0%			PI > 13 kg/m^3			50%	-		
	264	100%	0%			PI > 13 kg/m^3			50%	-		
	148	100% black	100%	15	30	PI > 14 kg/m^3			-	50%		
	302	100%	100%			PI > 16 kg/m^3			-	50%		
	121	100% black	0%			PI > 13 kg/m^3			50%	-		
	264	100%	0%			PI > 13 kg/m^3			50%	-		
Triceps Skinfold												
Freedman et al 1987[18]	1490	59%	52%	2 to 14	10 to 24	TSF	12 mm NR for groups	TSF	0.54 (.50, .57)		Correlation	N/A
FAIR	440	100%	0%						0.52 (.45, .59)			
	432	100%	100%							0.45 (.37, .52)		
	281	100% black	0%						0.59 (.51, .66)			
	337	100% black	100%							0.64 (.57, .70)		
Clarke and Lauer 1993c[22]	1286 obs	100%	53%	9 to 10	21 to 25	TSF	Baseline NR	TSF	0.49	0.44	Correlation	N/A
	304 obs		58%		26 to 30				0.58	0.50		
FAIR												

Table J-1. Evidence Table: Relationship between Childhood Overweight and Adult Overweight

Author QUALITY	N	% White	% Female	Age		OW Defined as			M	F	Measure	Reference Standard
				Initial	Follow-up	Youth	BMI Range (Youth)	Adult				
Freedman et al 1987[18] **FAIR**	255	59% overall	52% overall	9 to 10	18 to 19	TRSF	NR for groups	TSF	0.56 (.47, .64)		Correlation	N/A
Lauer et al 1997[8] **FAIR**	2631	100%	NR probably around 50	9 to 18	23 to 33	TSF	NR	TSF	.26 - .58		Correlation	N/A
Webber et al 1986a[28] **FAIR**	359	64% overall	48% overall	11 to 12	17 to 18	TSF	Baseline mean TSF range: 10 +/- 3 mm - 17+/-7 mm; varies by age, study yr at baseline	TSF	0.88		Correlation	N/A
Freedman et al 1987[18] **FAIR**	288	59% overall	52% overall	11 to 14	20 to 24	TRSF	NR for groups	TRSF	0.57 (.49, .64)		Correlation	N/A
	57	100% black	100%		10 to 24					0.65 (.47, .78)		
Clarke and Lauer 1993c[22] **FAIR**	1104 obs	100%	54%	13 to 14	21 to 25	TSF	baseline NR	TSF	0.45	0.49	Correlation	N/A
	713 obs		56%		26 to 30				0.48	0.48		
	104 obs		52%		31 tp 35				0.72	0.55		
Webber et al 1986a[28] **FAIR**	188	64% overall	48% overall	13 to 14	17 to 18	TSF	Baseline mean TSF range: 10 +/- 3 mm - 17+/-7 mm; varies by age, study yr at baseline	TSF	0.77		Correlation	N/A
					19 to 20				0.92			

Table J-1. Evidence Table: Relationship between Childhood Overweight and Adult Overweight

Author QUALITY	N	% White	% Female	Age		OW Defined as			M	F	Measure	Reference Standard
				Initial	Follow-up	Youth	BMI Range (Youth)	Adult				
Clarke and Lauer 1993c[22] **FAIR**	631 obs	100%	52%	17 to 18	21 to 25	TSF	Baseline NR	TSF	0.43	0.5	Correlation	N/A
	676 obs		52%		26 to 30				0.44	0.49		
	218 obs		51%		31 to 35				0.26	0.53		
Webber et al 1986b[28] **FAIR**	3051	64% overall	48% overall	10 to 15	15 to 20	TSF	Baseline mean TSF range: 10 +/- 3 mm - 17+/-7 mm; varies by age, study yr at baseline	TSF	about .65			N/A
Triceps Skinfold: > 95th %ile												
Freedman et al 1987[18] **FAIR**	74	59% overall	52% overall	2 to 14	10 to 24	TSF > 95th %ile	N/A	TSF > 95th %ile	66%		% OC who became OA	in-study
Triceps Skinfold 86th-95th %ile												
Freedman et al 1987[18] **FAIR**	148	59% overall	52% overall	2 to 14	10 to 24	TSF 86-95th%ile	N/A	TSF 86-95th%ile	32%		% OC who became OA	in-study

Table J-1. Evidence Table: Relationship between Childhood Overweight and Adult Overweight

Author QUALITY	N	% White	% Female	Age		OW Defined as			M	F	Measure	Reference Standard
				Initial	Follow-up	Youth	BMI Range (Youth)	Adult				
Triceps Skinfold ≥ 85th %ile												
Garn and Lavelle 1985[7] **FAIR**	383	100%	56%	0.5 to 5.5	21 to 25	TSF ≥ 85th %ile	N/A	TSF ≥ 85th %ile	0.11	0.15	Correlation	Met Life Ins tables 1960
									1.73		RR or OR	
									26%		% OC who became OA	
Freedman et al 1987[18] **FAIR**	1490	59%	52%	2 to 14	10 to 24	TSF > 85th %ile	N/A	TSF > 85th %ile	43%		% OC who became OA	in-study
									43%		% OA who were OC	
	63	100%	100%						-	35%	% OC who became OA	
Garn and Lavelle 1985[7] **FAIR**	79	100%	56%	5	25	TSF ≥ 85th %ile	N/A	TSF ≥ 85th %ile	0.35	0.18	Correlation	Met Life Ins tables 1960
									0.87		RR or OR	
									13%		% OC who became OA	
Freedman et al 1987[18] **FAIR**	223	59% whole cohort	52% whole cohort	11 to 14	10 to 24	TSF > 85th %ile	N/A	TSF > 85th %ile	59%		% OC who became OA	in-study

Table J-1. Evidence Table: Relationship between Childhood Overweight and Adult Overweight

Author QUALITY	N	% White	% Female	Age		OW Defined as			M	F	Measure	Reference Standard
				Initial	Follow-up	Youth	BMI Range (Youth)	Adult				
Triceps Skinfold ≥ 80th %ile												
Clarke and Lauer 1993c[22] **FAIR**	1286 obs	100%	53%	9 to 10	21 to 25	TSF top quintile	N/A	TSF top quintile	46%	45%	% OC who became OA	Muscatine study population
	304 obs		58%		26 to 30				45%	40%		
	1104 obs		54%	13 to 14	21 to 25				44%	50%		
	713 obs		56%		26 to 30				51%	42%		
	104 obs		52%		31 to 35				57%	25%		
	631 obs		52%	17 to 18	21 to 25				44%	50%		
	676 obs		52%		26 to 30				52%	47%		
	218 obs		51%		31 to 35				42%	42%		
Lauer et al 1997[8] **FAIR**	2631	100%	NR, probably around 50	9 to 18	23 to 33	Highest TSF quintile	N/A	Highest TSF quintile	25-56%		% OC who became OA	NHANES II
Other measures: SSF												
Freedman et al 1987[18] **FAIR**	1490	59% overall	52% overall	7 to 19	10 to 24	SSF	N/A	SSF	0.8		Correlation	N/A
Garn and Lavelle 1985[7] **FAIR**	79	100%	56%	5	25	SSF ≥ 85th %ile	N/A	SSF ≥ 85th %ile	0.22	0.13	Correlation	Met Life Ins tables 1960
									0.80		RR or OR	
									25.0%		% OC who became OA	
	383			0.5 to 5.5	21 to 25				0.13	0.20	Correlation	
									1.77		RR or OR	
									27.1%		% OC who became OA	
Freedman et al 1987[18] **FAIR**	195	59% overall	52% overall	11 to 14	10 to 24	SSF > 85th %ile	N/A	SSF > 85th %ile	70%		% OC who became OA	in-study

Table J-1. Evidence Table: Relationship between Childhood Overweight and Adult Overweight

Author QUALITY	N	% White	% Female	Age		OW Defined as					Measure	Reference Standard
				Initial	Follow-up	Youth	BMI Range (Youth)	Adult	M	F		
Notes to this table:												

a Data also available for youth measurements at early adolescence (2 yr before yr of peak height velocity), yr of peak ht velocity, and 2 yr after yr of peak velocity; this table includes info on ages 5-7, 18).

b Estimated from bar graph.

c The number of children measured at baseline is not given, but rather the number of observations in that age group.

d Black females 3.4x more likely to become OA as white females of same BMI, age.

e Statistics represent probability of OW at 35.

f These data are for children for whom parental data available: controlling for parents' obesity status.

g 7% of normal weight children became OA.

h 17% of OW adults were normal weight children.

I Tracking differs by Tanner stage at baseline.

j Black females 3.8x more likely to become OA as white females with same BMI, age.

Childhood Overweight Clinical Screening Tests to Predict Poorer Health Outcomes

Overview of Methods and Prior Reviews

For the relationship between clinical screening measures of overweight and health consequences, we located three overview or review articles[1 2,3], one extensive(but non-systematic) review,[4] and one fair- or good-quality systematic review[5] at the time we conducted our literature search. The fair-quality systematic evidence review examined the consequences of childhood obesity in childhood and longer term through a comprehensive literature review from January 1981 through December 2001.[5] This review used established critical appraisal methodology, including explicit methodological quality rating and a hierarchical study design approach. However, it did not distinguish between cohort and cross-sectional studies in examining the childhood or adult consequences of overweight, nor did it systematically report the quantitative relationship between childhood overweight measures and health consequences. We used this review only as a source of potentially relevant studies. The extensive non-systematic review of long-term health risks of child and adolescent fatness that searched through 1996[4] located seven U.S. studies addressing childhood overweight and adult health outcomes.[6-12] This review concluded that there were too few studies on which to base firm conclusions about long-term health risks in relation to childhood and adolescent adiposity.[4]

In addition to screening prognosis trials located in our searches, we retrieved all non-duplicated possibly relevant citations from the five review articles, and from continuing to check bibliographies. Through this process we located another four non-systematic reviews related to health consequences and obesity measures[13-16]; which we reviewed for additional articles not previously located through other sources. None were found.

There were insufficient studies from all of these sources and from our searches to critically appraise the prospective relationship of childhood overweight to childhood health outcomes. We therefore focused on the prospective studies addressing childhood overweight and adult health outcomes.

Longitudinal U.S. Studies of Adult Health Consequences of Childhood Overweight

Table K-1 lists the 11 U.S. studies we examined for this key question.[7,8,10-12,17-22] We excluded a large number of cross-sectional studies identified in previous reviews and other studies for quality reasons (Appendix G). Among the longitudinal studies identified above, we excluded one that had follow-up measures in late adolescence (15-18 years) and not adulthood.[9] We excluded another[6] for using a non-comparable overweight definition and weight reference standard, for incomplete follow-up of the cohort (700/2,000), and for failing to address loss due to mortality in their morbidity analyses.

Morbidity and mortality. One often-cited fair- to poor-quality cohort study used a constructed sample (n=508, 27% of the original cohort) from the Third Harvard Growth Study conducted from 1922 to 1935.[7] A subsample of this group (n=309, 61%) had adult BMIs measured at mean age of 55 years that could be used in adjusted analyses. For most other analyses it is not clear whether the entire sample (n=508) was used, and if so, what assumptions were made to include those lost to follow-up or who declined participation (166, 32%). The study reported the impact of adolescent BMI (>75th percentile according to NHANES I compared with 25th-50th percentile) between ages 13 and 18 on all-cause mortality, coronary heart disease (CHD) mortality, atherosclerotic heart disease mortality, and colorectal cancer mortality in white males and females over 50 years later. Males in the higher BMI quartile had small increased relative risks for all-cause mortality (RR 1.8, 95% CI 1.2-2.7) and for mortality from CHD (RR 2.3, 95% CI1.4-4.1), atherosclerotic cardiovascular disease (RR 13.2, 95% CI 1.6-108.0), and colorectal cancer (RR 9.1, 95% CI 1.1-77.5). Females in the higher quartile of adolescent BMI did not show significantly elevated risks for mortality from these conditions or from breast cancer. In the subsample (n=309) with measured adult weights at 55 years of age, adjustment for adult BMI slightly decreased the RR for all-cause mortality in males and removed the increased RR for CHD mortality. However, the RR for all-cause mortality cited in the text as "before adult BMI adjustment" in males (2.9, 95% CI 1.5-5.8) does not match that reported for the entire sample in their Table 2, raising the question of the generalizability of the subsample analysis of adjustment for adult BMI on the risk of adult mortality and morbidity from childhood overweight. Morbidity analyses used a further subsample interviewed in 1988 (n=181). Lack of details about the selection and characteristics of this subsample preclude its use.

In another often-cited fair- to poor-quality nested case-control study,[8] mortality odds were calculated for measured relative weight (defined internally using the sample) in a population-based study of 13,146 children ages 5 and 18 in 1933-1945. Death certificate information was available for 5,471/13,146 of the population (42%). Pre-pubertal relative weight measures were those before age 10 in girls and age 12 in boys, while post-pubertal measures were those after age 13.5 in girls and 15.5 in boys. A total of 509 deaths were identified, 308 in males (median age at death, 50 years) and 201 in females (median age at death, 51 years). Controlling for sex and year of birth, those in the highest quintile of pre-pubertal and post-pubertal weight had the same, slightly increased mortality odds (OR 1.5, 95% CI 1.0-2.4). The analysis did not control for adult BMI, race, or other sociodemographic factors significantly related to mortality.

Socioeconomic outcomes. A fair- to good-quality longitudinal cohort study using the National Longitudinal Survey of Labor Market Experience Youth Cohort from 1979 examined the risk of lower household income, household poverty, likelihood of marriage, years completed of school, and self-esteem in young adulthood (23-31 years of age) for overweight 16-24 year olds.[12] Overweight was defined as those above the 95th percentile of BMI (in NHANES I), and compared with all others in the cohort.[12] The cohort was 51% female, 80% white, 14% black, and 6% Hispanic. Between 3.0% and 3.4% were overweight (BMI >95th percentile) at baseline, with black adolescent females significantly more overweight than non-Hispanic whites (5.8% vs. 2.5%, P<.001). At follow-up, 77% of the men and 66% of the women were still overweight. For females, but not males, overweight in adolescence was associated with completing 0.3 mean fewer years of schooling, $6,710 lower household income, and 10% higher rate of poverty. Overweight males and females were less likely to have married (11% and 20% less likely, respectively) but had no differences in self-esteem in young adulthood. Among males only,

being 12 inches shorter in height at baseline was independently associated with a 10% higher prevalence of poverty (95% CI 6-13%). The researchers did not control for adult BMI in their analyses but did control for baseline socioeconomic status and aptitude. Redefining overweight as over the 85th percentile increased the prevalence of overweight at baseline and reduced some (and eliminated others) of the reported risk relationships with adult social and economic factors in women and the risk relationship to marriage in men.

Cardiovascular disease and diabetes risk factors. One good-quality cohort study used data from the Bogalusa Heart Study (32% black and 57% female) to examine the longitudinal relationship between childhood BMI or triceps skinfold thickness measured at a mean age of 10 +/- 3 years and adult BMI; lipids (total cholesterol, LDL and HDL cholesterol); insulin; and systolic and diastolic blood pressure after a mean of 17 years of follow-up (adult ages of 18 to 37 years, mean age 26.2 +/- 6.3 years).[19] BMI levels in childhood and adulthood were more strongly associated with all adult risk factors than TSF measures in childhood and adulthood. When examined separately, adult BMI levels were moderately correlated with adult cardiovascular risk factors (r=0.21-0.59), as were childhood BMI levels, although the correlations tended to be about 50% as strong (r=0.09-0.26). Controlling for adult BMI eliminated the association of childhood BMI with adult CV risk factors, indicating that the effect of childhood weight status was mediated through its relationship to adult BMI. For all six risk factors, adjusting for adult BMI actually reversed the relationship between greater childhood BMI and greater adult CV risk factors, although these correlations tended to be quite small (<0.15 absolute value). Compared with adults who had normal childhood BMIs (<50th percentile), adults who were overweight in childhood (BMIs $\geq$95th percentile) had significantly higher adult BMI (34.9 +/- 7 vs. 22.5 +/- 4) and significantly higher CVD risk factor measures (although HDL cholesterol was significantly lower).

However, in an analysis stratifying by both adult and childhood BMI, obese adults who had been overweight as children had similar adult CVD risk factors compared to obese adults who had been normal weight as children. Obese adults who had been overweight children were significantly more obese (mean BMI 38.1) than obese adults who had been normal weight as children (mean BMI 33.2, $p<0.05$). A similar pattern was seen when comparing normal-weight adults who had been overweight or not as children. The authors did not compare the proportion of adults who were hypertensive or hyperlipidemic, although the proportion diagnosed with diabetes did not differ between those who were overweight vs. normal weight as children, once adult BMI was taken into account. A subset analysis on timing of obesity and adult CV risk factors was reported, but is not considered here due to its limited power and our inability to confirm lack of selection bias.

Seven fair-quality longitudinal cohort studies examined the relationship between childhood overweight and adult CVD risk factors.[10,11,17,18,20-22] None of these adjusted for adult BMI in examining this relationship, although four studies included the change from childhood to adult BMI in their analyses.[10,11,18,22] Given the potential confounding of adult BMI on the relationship between childhood overweight and adult risk factors, we confine our discussion to the four studies considering change. Three of these studies included racial/ethnic minorities, with about one-third consisting of blacks [11,22] or blacks and Native Americans.[18] The most informative study examined the relationship between elevated childhood lipid levels (according to National Cholesterol Education Program [NCEP] guidelines) at age 5-14 and adult dyslipidemia (elevated total cholesterol, LDL-C, HDL-C, or triglycerides according to NCEP guidelines) at age 20-34 in

children from the Bogalusa Heart Study, controlling for race, sex, age, baseline BMI, baseline lipids, and change in BMI from childhood to adulthood.[22] Children with baseline LDL-C above 101 had the greatest odds (2.5, 95% CI 2.0-3.1) for adult dyslipidemia. Baseline BMI, change in BMI from childhood to adulthood, and older age all independently raised the odds of adult dyslipidemia (OR 1.7-1.9). Controlling for other factors, females and blacks were significantly less likely to have adult dyslipidemia (61% and 42%, $p<0.01$). These data are suggestive of a role for each of these factors, including greater weight gain from childhood to adulthood. Without controlling for adult BMI, however, it is not clear that these are independent of adult overweight.

In another study using the Bogalusa Heart study cohort, CVD risk factors at ages 27-31 years were compared in adults who had become overweight as adolescents (above the 75th percentile for age- and sex-specific BMI) and remained overweight as adults with those who were consistently lean (25th-50th percentiles).[11] Among 191/783 adolescents ages 13-17 years identified as overweight, 110 (58%) remained overweight as adults, 64 were between the 50th and 75th percentiles, and 17 were below the 25th percentile. The predictive value of overweight status was lowest in black males (52%) and highest in black females (62%). Conversely, the predictive value of lean status was lowest in black males (28%) and highest in white males (52%). Compared with the consistently lean cohort, higher adolescent BMI and change in BMI were associated with increased blood pressure, lipids (decreased HDL-C), glucose, and insulin levels. With the relatively strong tracking of adolescent to adult BMI, it is not possible to understand the independent contribution of these factors beyond adult BMI.

Using the Muscatine cohort, childhood blood pressure, childhood BMI, change in BMI from childhood to adulthood, family history, and behavioral risks at ages 7-18 years were related to adult systolic blood pressure at ages 20-30.[10] At age 16, probability of adult systolic blood pressure above the 90th percentile reached about 0.3 in females and 0.4 in males at the 90th percentile for BMI, and increased steeply with greater BMI percentile. This analysis did not consider adult BMI. Across ages and sex, without considering adult BMI, change in BMI from childhood to adulthood and childhood blood pressure level were independent predictors of adult systolic blood pressure, jointly explaining 14%-24% of the variance.

Childhood BMI at age eight and change in BMI in childhood and adolescence were examined in 679 children across a broad range of BMI measures (8 to 34, mean 16.5 in relation to blood pressure, fasting insulin, and lipids at age 24.[18] Childhood and adult BMI were strongly correlated ($r=0.61$). Childhood BMI was not independently related to adult CVD risk factors, after childhood and adolescent weight gain were considered. Weight gain in childhood and adulthood could be surrogates for eventual adult overweight.

References

1. Serdula MK, Ivery D, Coates RJ, Freedman DS, Williamson DF, Byers T. Do obese children become obese adults? A review of the literature. *Prev Med* 1993; 22(2):167-177.
2. Dietz WH. Childhood weight affects adult morbidity and mortality. *Journal of Nutrition* 1998; 128(2 Suppl):411S-414S.
3. Must A. Morbidity and mortality associated with elevated body weight in children and adolescents. *Am J Clin Nutr* 1996; 63(3 Suppl):445S-447S.
4. Power C, Lake JK, Cole TJ. Measurement and long-term health risks of child and adolescent fatness. *Int J Obes Relat Metab Disord* 1997; 21(7):507-526.
5. Reilly JJ, Methven E, McDowell ZC, Hacking B, Alexander D, Stewart L et al. Health consequences of obesity. *Arch Dis Child* 2003; 88(9):748-752.
6. Abraham S, Collins G, Nordsieck M. Relationship of childhood weight status to morbidity in adults. *HSMHA Health Rep* 1971; 86(3):273-284.
7. Must A, Jacques PF, Dallal GE, Bajema CJ, Dietz WH. Long-term morbidity and mortality of overweight adolescents. A follow-up of the Harvard Growth Study of 1922 to 1935.. *New England Journal of Medicine* 1992; 327(19):1350-1355.
8. Nieto FJ, Szklo M, Comstock GW. Childhood weight and growth rate as predictors of adult mortality. *American Journal of Epidemiology* 1992; 136(2):201-213.
9. Clarke WR, Woolson RF, Lauer RM. Changes in ponderosity and blood pressure in childhood: the Muscatine Study. *Am J Epidemiol* 1986; 124(2):195-206.
10. Lauer RM, Lee J, Clarke WR. Predicting adult cholesterol levels from measurements in childhood and adolescence: the Muscatine Study. *Bulletin of the New York Academy of Medicine* 1989; 65(10):1127-1142.
11. Srinivasan SR, Bao W, Wattigney WA, Berenson GS. Adolescent overweight is associated with adult overweight and related multiple cardiovascular risk factors: the Bogalusa Heart Study. *Metabolism: Clinical & Experimental* 1996; 45(2):235-240.
12. Gortmaker SL, Must A, Perrin JM, Sobol AM, Dietz WH. Social and economic consequences of overweight in adolescence and young adulthood. *New England Journal of Medicine* 1993; 329(14):1008-1012.
13. Zametkin AJ, Zoon CK, Klein HW, Munson S. Psychiatric aspects of child and adolescent obesity: a review of the past 10 years. *J Am Acad Child Adolesc Psychiatry* 2004; 43(2):134-150.
14. Dietz WH. Health consequences of obesity in youth: childhood predictors of adult disease. *Pediatrics* 1998; 101(3 Pt 2):518-525.
15. Must A, Strauss RS. Risks and consequences of childhood and adolescent obesity. *Int J Obes Relat Metab Disord* 1999; 23 Suppl 2:S2-11.:S2-11.
16. Must A. Does overweight in childhood have an impact on adult health? *Nutr Rev* 2003; 61(4):139-142.
17. Freedman DS, Khan LK, Serdula MK, Dietz WH, Srinivasan SR, Berenson GS. Inter-relationships among childhood BMI, childhood height, and adult obesity: the Bogalusa Heart Study. *International Journal of Obesity & Related Metabolic Disorders* 2004; 28(1):10-16.
18. Sinaiko AR, Donahue RP, Jacobs DR, Jr., Prineas RJ. Relation of weight and rate of increase in weight during childhood and adolescence to body size, blood pressure, fasting insulin, and lipids in young adults. The Minneapolis Children's Blood Pressure Study. *Circulation* 1999; 99(11):1471-1476.
19. Freedman DS, Khan LK, Dietz WH, Srinivasan SR, Berenson GS. Relationship of childhood obesity to coronary heart disease risk factors in adulthood: the Bogalusa Heart Study. *Pediatrics* 2001; 108(3):712-718.
20. Lauer RM, Clarke WR, Burns TL. Obesity in childhood: the Muscatine Study. *Chung-Hua Min Kuo Hsiao Erh Ko i Hsueh Hui Tsa Chih* 1997; 38(6):432-437.
21. Srinivasan SR, Myers L, Berenson GS. Predictability of childhood adiposity and insulin for developing insulin resistance syndrome (syndrome X) in young adulthood: the Bogalusa Heart Study. *Diabetes* 2002; 51(1):204-209.
22. Bao W, Srinivasan SR, Wattigney WA, Bao W, Berenson GS. Usefulness of childhood low-density lipoprotein cholesterol level in predicting adult dyslipidemia and other cardiovascular risks. The Bogalusa Heart Study. *Archives of Internal Medicine* 1996; 156(12):1315-1320

Table K-1. Evidence Table: Relationship between Childhood Overweight and Adult Health Measures Other than Overweight

Author QUALITY	N	% White	% Female	Age		Health Measure			M	F	Measure	Reference Standard	Adjusted for Adult BMI
				Initial	Follow-up	Youth	Range	Adult					
BMI: kg/m^2													
Freedman et al 2004[17] **FAIR**	885	67% overall; NR for this age group	62%	3 to 7	18 to 37	BMI	NR: mean BMI for study pop is 17.7+/-3.4	Fasting insulin level	0.27	0.21	Correlation	N/A	No
Lauer et al 1989[10] **FAIR**	109 obs	100%	52%	7 to 8	20 to 25	W/H^2: assume kg/m^2	around 17	SBP	0.38	-.16 ns	Correlation	N/A	No
								DBP	0.27	-.03 ns			
Sinaiko et al 1999[18] **FAIR**	679	66% white, 25% black, 4% NA	48%	8	24	BMI	8 to 34: mean 16.5	Triglycerides	0.19		Correlation	N/A	No
								HDL-C	-0.18				
								SBP	0.27				
								Fasting insulin	0.28				
								LDL-C	ns				
Freedman et al 2004[17] **FAIR**	2444	67% overall; NR for this age group	56%	8 to 13	18 to 37	BMI	NR: mean BMI for study pop is 17.7+/-3.4	Fasting insulin level	0.31	0.26	Correlation	N/A	No
Lauer et al 1989[10] **FAIR**	603 obs	100%	52%	9 to 10	20 to 25	W/H^2: assume kg/m^2	around 18	SBP	.10 ns	0.17	Correlation	N/A	No
								DBP	-.05 ns	0.13			
	1018 obs		53%	11 to 12	20 to 25		around 20	SBP	0.17	0.21			
								DBP	.05 ns	0.17			
	1041 obs		53%	13 to 14	20 to 25		around 21	SBP	0.16	0.15			
								DBP	.05 ns	0.1			
	339 obs		54%	13 to 14	26 to 30		around 21	SBP	0.29	0.3			
								DBP	.14 ns	0.34			

Table K-1. Evidence Table: Relationship between Childhood Overweight and Adult Health Measures Other than Overweight

Author QUALITY	N	% White	% Female	Age		Health Measure			M	F	Measure	Reference Standard	Adjusted for Adult BMI
				Initial	Follow-up	Youth	Range	Adult					
Freedman et al 2004[17] **FAIR**	2212	67% overall; NR for this age group	56%	14 to 17	18 to 37	BMI	NR: mean BMI for study pop is 17.7+/-3.4	Fasting insulin level	0.36	0.28	Correlation	1963-94: CDC growth charts	No
Lauer et al 1989[10] **FAIR**	767 obs	100%	54%	15 to 16	20 to 25	W/H^2: assume kg/m^2	around 22	SBP	0.14	.05 ns	Correlation	N/A	No
								DBP	.04 ns	.04 ns			
	568 obs		54%	15 to 16	26 to 30		around 22	SBP	0.14	0.24			
								DBP	0.17	0.23			
	615 obs		51%	17 to 18	20 to 25		around 23	SBP	0.29	-.09 ns			
								DBP	.10 ns	.01 ns			
	469 obs		52%	17 to 18	26 to 30		around 23	SBP	0.19	0.21			
								DBP	0.15	.09 ns			
BMI %ile													
Freedman et al 2001[b,19] **GOOD**	2617	67% white, 33% black	58%	5 to 17 (mean 10 yrs)	18 to 37 (mean 27)	BMI %ile	mean childhood BMI: 17.8 +/- 3.5	TC	0.10		Correlation	CDC / NCHS growth charts for 1963-94	Yes
								Triglycerides	0.16				
								LDL-C	0.11				
								HDL-C	-0.14				
								Insulin	0.26				
								SBP	0.08				
								DBP	0.09				

Table K-1. Evidence Table: Relationship between Childhood Overweight and Adult Health Measures Other than Overweight

Author QUALITY	N	% White	% Female	Age		Health Measure			M	F	Measure	Reference Standard	Adjusted for Adult BMI
				Initial	Follow-up	Youth	Range	Adult					
BMI > 95th %ile vs < 95th %ile													
Gortmaker et al 1993[12] **FAIR**	10,039 original cohort; but 56-79% follow up by question	80% white, 14% black, 6% Hispanic original cohort	100%	16 to 24	23 to 31	BMI > 95th %ile vs < 95th %ile	N/A	Years of school	F: OW completed .3 (0.1 - 0.6) yrs less school (p = .009)		Other	NHANES I	No
								Married	-	2.5 (1.8 - 3.5)	RR or OR		
									F: OW 20% (13-27%) less likely to be married (p < .0001)		Other		
								Household income	F: household income lower by $6,710/yr ($3942-9478), p<.0001		Other		
								Household poverty	-	2.0 (1.1 - 2.4)	RR or OR		
									F: 10% (4 - 16%) higher rates of household poverty, p <.0001		Other		
			0%					Married	1.6 (1.2 - 2.3)	-	RR or OR		
									M: 11% (3 - 18%) less likely to be married p = .005		Other		

Table K-1. Evidence Table: Relationship between Childhood Overweight and Adult Health Measures Other than Overweight

Author QUALITY	N	% White	% Female	Age		Health Measure			M	F	Measure	Reference Standard	Adjusted for Adult BMI
				Initial	Follow-up	Youth	Range	Adult					
								Household income	M: household income lower by $5,454/yr ($2488-8420), p= 0.05		Other		
BMI > 90th %ile vs < 90th %ile													
Lauer et al 1997[c,20] **FAIR**	384	100%	NR	9 to 18	32 to 41	upper vs lower 9 deciles BMI adjusted for age, sex	N/A	Coronary artery calcification	.58 to .91		Correlation	age, sex, year specific %iles: study pop; also NHANES II	No
									1.9 (0.8 - 4.2)		RR or OR[c]		
									6.1 (2.4 - 15.1)	-	RR or OR[d]		
BMI > 85th %ile													
Gortmaker et al 1993[12] **FAIR**	10,039 original cohort; but 56-79% follow up by question	80% white, 14% black, 6% Hispanic original cohort	100%	16 to 24	23 to 31?	BMI > 85th %ile	N/A	Married	F: OW 6% less likely to be married (p < .0001)		Other	NHANES I	No
								Household income	F: hhold income lower by $3602/yr, p < .0001				

Table K-1. Evidence Table: Relationship between Childhood Overweight and Adult Health Measures Other than Overweight

Author QUALITY	N	% White	% Female	Age		Health Measure			M	F	Measure	Reference Standard	Adjusted for Adult BMI
				Initial	Follow-up	Youth	Range	Adult					
BMI > 75th %ile													
Srinivasan et al 1996[11] **FAIR**	110	65% white, 35% black in cohort	NR	13-17	27 to 31	BMI > 75th %ile	N/A	> 75th %ile for TC:HDLC ratio, plasma insulin level	5.8		RR for O:E	in-study	No
								> 75th %ile for plasma insulin level, SBP	5.4				
								> 75th %ile for TC:HDLC ratio, plasma insulin level, SBP	3				
Srinivasan et al 2002[e,21] **FAIR**	745	67%	61%	8 to 17	12 years later	BMI top quartile	N/A	Insulin resistance syndrome (X) y/n	11.7 (3.4 - 39.7) M: in stepwise regression, childhood BMI, insulin were the best predictors of adult syndrome X presence		RR for O:E Other		No

Table K-1. Evidence Table: Relationship between Childhood Overweight and Adult Health Measures Other than Overweight

Author QUALITY	N	% White	% Female	Age		Health Measure			M	F	Measure	Reference Standard	Adjusted for Adult BMI
				Initial	Follow-up	Youth	Range	Adult					
BMI > 75th %ile vs BMI 25th-50th %ile													
Must et al 1992[f,7] **FAIR**	508	100%	50%	13 to 18 in 1922-35	In 1988: 66-84?	BMI >75th vs 25th-50th %ile	BMI 22-25 (m), 22-24 (f)	All-cause mortality	1.8 (1.2, 2.7)	1.0 (0.6-1.6) ns	RR or OR	NHANES I (1971-4)	Yes
								CHD mortality	2.3 (1.4, 4.1)	0.8 (0.3-2.1) ns			
								Atherosclerotic cerebrovascular disease mortality	13.2 (1.6-108.0)	0.4 (0.1-1.8) ns			
								Colorectal cancer mortality	9.1 (1.1 - 77.5)	1.0 (0.1 - 7.0) ns			
	181	100%	50%	13 to 18 in 1922-35	In 1988: 66-84?	BMI >75th vs 25th-50th %ile	BMI 22-25 (m), 22-24 (f)	CHD	2.8 (1.1 - 7.2)	2.5 (0.9-7.1) ns	RR or OR	NHANES I (1971-4)	
								Arthritis	1.6 (0.8-3.2) ns	2.0 (1.1 - 3.7)			
								Gout	3.1 (1.1 - 9.3)	2.2 (0.7-6.9) ns			

Table K-1. Evidence Table: Relationship between Childhood Overweight and Adult Health Measures Other than Overweight

Author QUALITY	N	% White	% Female	Age		Health Measure			M	F	Measure	Reference Standard	Adjusted for Adult BMI
				Initial	Follow-up	Youth	Range	Adult					
Srinivasan et al 1996[11] **FAIR**	191 OW, 199 lean	65% white, 35% black in cohort	NR	13 to 17	27 to 31	BMI > 75th vs 25th-50th %ile	N/A	SBP > 140 mmHg, or on meds	4.1		M: regression coefficients		No
								DBP > 90 mm Hg, or on meds	2.7				
								TC > 240 mg/dL	14.3				
								VLDL C	5.1				
								LDLC > 160 mg/dL	12.4				
								HDLC < 35 mg/dL	-3.4				
								Triglycerides > 259 mg/dL	37.5				
								Insulin	5.1				
								Glucose > 115 mg/dL	5.3				
BMI > 75th %ile vs BMI 25th %ile													
Bao et al 1996[22] **FAIR**	1169	64% white, 36% black	NR	5 to 14	20 to 34	BMI 75th vs. 25th %ile: 19.7 vs. 15.3	NR	Existence of adult dyslipidemia: total chol ≥240 mg/dL, triglycerides > 400 mg/dL, HDL-C ≤ 35 mg/dL, LDL-C ≥ 160 mg/dL, or hypertension > 140/>90 mmHg	1.7 (1.1-2.6)		RR or OR		No

Table K-1. Evidence Table: Relationship between Childhood Overweight and Adult Health Measures Other than Overweight

Author QUALITY	N	% White	% Female	Age		Health Measure			M	F	Measure	Reference Standard	Adjusted for Adult BMI
				Initial	Follow-up	Youth	Range	Adult					
PI: W/H^3													
Freedman et al 2004[17]	885	67% overall; NR for this age group	62%	3 to 7	18 to 37	W/H^3	NR: mean BMI for study pop is 17.7+/-3.4	Fasting insulin level	0.17	0.2	Correlation	N/A	No
	2444		56%	8 to 13					0.28	0.24			
FAIR	2212		56%	14 to 17					0.32	0.27			
PI: W/H(p)													
Freedman et al 2004[17]	885	67% overall; NR for this age group	62%	3 to 7	18 to 37	W/H(p)	NR: mean BMI for study pop is 17.7+/-3.4	Fasting insulin level	0.18	0.21	Correlation	N/A	No
	2444		56%	8 to 13					0.21	0.24			
FAIR	2212		56%	14 to 17					0.32	0.28			
Triceps skinfold													
Freedman et al 2001[19]	2617	67% white, 33% black	58%	5 to 17	18 to 37	TSF	mean childhood BMI: 17.8 +/-3.5	TC	0.10		Correlation	CDC / NCHS growth charts for 1963-94	Yes
								Triglycerides	0.12				
FAIR								LDL-C	0.08				
								HDL-C	-0.12				
								Insulin	0.21				
								SBP	0.07				
								DBP	0.07				

Table K-1. Evidence Table: Relationship between Childhood Overweight and Adult Health Measures Other than Overweight

Author QUALITY	N	% White	% Female	Age		Health Measure						Reference Standard	Adjusted for Adult BMI
				Initial	Follow-up	Youth	Range	Adult	M	F	Measure		
Top vs bottom quintiles of relative weight													
Nieto et al 1992[8] **FAIR**	About 225 cases matched to about 2250 controls	99%	37% of cases	5-18 in 1933-45	45 to 69	highest vs lowest quintile of pre-pubertal relative weight	NR	Death by 1985	1.5 (1.0-2.4) 1.5 (0.9-2.7)	1.5 (0.8-3.1)	RR or OR	Relative weight defined internal to study pop	No
						highest vs lowest quintile of post-pubertal relative weight			1.6 (1.0-2.4) 1.2 (0.6-2.2)	2.0 (1.1-3.6)			

Notes to this table:

a p = .05; not significant.

b Controlling for adult BMI eliminated these positive associations between childhood BMI and adult measures. Also, there are data on mean levels of these risk factors by youth BMI but these differences are also mediated by adult BMI.

c OR for presence of coronary artery calcification: univariate.

d OR for coronary artery calcification: identified from stepwise regression.

e Syndrome X: defined as clustering of highest quartile for BMI, insulin, BP, TC:HDLC or trigs:HDLC.

f NHANES I standards used to determine %iles in youth (1922-35). There are also data on risk of difficulties in tasks of daily living.

Appendix L. Evidence Table: Intervention Trials 2001-Present

Study	Methods	Participants	Interventions	Outcomes	Results	Adverse Effects	Comment
Berkowitz et al 2003[1] **Setting:** University-based specialty research clinic **Location:** Philadelphia	**Random allocation:** Yes, but neither randomization procedure nor allocation concealment are described. **Blinding:** Yes, participants, parents, all study personnel. Only research pharmacist was aware of treatment status **Length of Intervention and f/u:** 6 mos of RCT + 6 mos during which all participants received sibutramine (open label extension) **Unit of allocation:** individual adolescents **Unit of analysis**: individuals **Protection against contamination** : Control and intervention subjects differed only by receiving sibutramine vs. placebo **Drop outs** – 9.8%	**N randomized, completed:** 82, 74 (completed 6 mos) **Age range:** *Eligible:* ages 13-17 yrs (only postmenarcheal girls) *Actual:* 14.1 yrs (1.2) **% male**: 33% **% minority or non-white:** 45% **Weight entry criteria**: BMI 32-44 **Weight on entry**: BMI: 37.8 (3.8) BMI z-score: 2.4 (0.2) **Recruitment pool:** NR	**Description of intervention:** Randomized, double-blinded, placebo-controlled trial of pharmacological agent sibutramine plus comprehensive, family-based, behavioral wt loss program vs. behavioral wt loss program alone. ***Exp:*** *Pharmacological treatment*: Wk 1 – placebo (all); Wk 2 - 5 mg sibutramine QD; Wk 3 – 10 mg/day*; Wk 7 – 15 mg/day* *unless BP or HR elevated *Behavioral treatment*: Diet: 1200-1500 kcal/d, 30% fat/15% protein/ remainder CHO. Physical activity: ≥ 120 min/wk walking or other aerobic activity. Daily eating and activity logs submitted. Parents met separately from children on same schedule. ***Comp:*** *Pharmacological treatment:* Placebo capsules identical in appearance to sibutramine were administered on same schedule as experimental group. *Behavioral treatment:* Same as experimental group. **Intensity level of intervention** *group vs. individual*: group *# sessions, over what time period:* *Phase I:* 19 sessions Over 6 months; (*Phase II:* 9 sessions over 6 months) *length of sessions:* NR *total contact time (min):* unable to calculate	**Weight status:** Weight, height, waist circumference **Time points when weight was measured:** Baseline, months 3,6,9, plus at each treatment visit **Behavioral:** *Diet-* NR *PA-* NR **Physiological:** *Lipids/ lipoproteins:* yes, reported* *Glucose tolerance:* yes, reported* (fasting serum glucose & insulin levels, HOMA) *blood pressure:* yes, reported*(systolic BP diastolic BP) *physical fitness:* NR *Other:* ECG Pulse rate* Correlation between %ΔBMI and physiologic outcomes. *= treatment groups lumped together & only completers are analyzed **Adverse events:** Yes, reported **Health Outcomes:** NR **Others:** -Adherence to lifestyle program -Adherence to medication -Hunger	**Results at 6 months:** *Change in initial BMI (%):* Exp: -8.5 %(6.8) Comp: -4.0 %(5.4) Mean difference (95%CI): 4.5 % (1.8-7.2), p = 0.001 *Weight:* Exp: -7.8 kg (6.3) Comp: -3.2 kg (6.1) Mean difference (95% CI): 4.6 kg (2.0-7.4), p=0.001 *Waist circumference:* Exp: -8.2 cm (6.9) Comp: -2.8 cm (5.6) Mean difference (95% CI): 5.4 cm (2.5-8.2), p < 0.001 *Reduction in initial BMI, ≥ 5%* Exp: 27/43 (63%) Comp: 14/39 (36%) OR: 3.0 (1.2-7.4) χ^2=5.92, p=0.02 *Reduction in initial BMI, ≥ 10%* Exp: 17/43 (40%) Comp: 6/39 (15%) OR: 3.6 (1.2-10.4) χ^2=5.91, p=0.02 *Reduction in initial BMI, ≥15%* Exp: 8/43 (19%) Comp: 1/39 (3%) OR: 8.7(1.0-73.0) χ^2=5.39,p=0.02	*Elevated BP and/or pulse rate @6 mos:* Comp: 0/39 (0%) Exp: 5/43 (12%) P=0.06 During first 6 months, 19/43 (44%) patients required reduction in dose of sibutramine due to increased BP or pulse rate. During entire study, 5/64 (8%) patients had marked and sustained increases in BP (≥ 10 mmHg) that required discontinuation of medication. *Other reasons for discontinuing medication (n):* Ecchymoses (2); Ventricular premature complexes (1); Rash (1)	**Quality:** Good **Financial deposits:** NR **Financial disclosures:** Investigators have served as consultants, on the speakers bureau, and have received funding from Knoll Pharmaceutical and Abbott Laboratories.

Appendix L. Evidence Table: Intervention Trials 2001-Present

Study	Methods	Participants	Interventions	Outcomes	Results	Adverse Effects	Comment
			Primary care feasible/referable? – Yes, referable				
Ebbeling et al 2003[2] **Setting:** Research clinic **Location:** Boston, MA	**Random allocation:** Yes, but neither randomization procedure nor allocation concealment are described. **Blinding: (children, providers, outcome assessors):** not reported **Length:** 6 mos of Intervention + 6 mos f/u **Unit of allocation:** individual adolescents **Unit of analysis:** individuals **Protection against contamination:** Yes, took several strategies to prevent **Drop outs (%):** 12.5%	**N randomized, completed**: 16, 14 **Age range:** 13-21 yrs exp: 16.9 (1.3) cont: 15.3(0.9) **% male:** 31.25% **% minority or non-white:** 18.75% **Weight entry criteria:** BMI > 95th %ile for sex and age **Weight on entry:** *BMI:* Exp:34.9(1.0) Cont:37.1(1.2) **Recruitment pool:** Not stated	**Description of intervention:** Randomized controlled trial of reduced glycemic load diet vs. conventional reduced fat diet. For both groups, educational and behavioral components of treatment delivered using a social cognitive theory conceptual framework. *Exp: reduced glycemic load diet* – patients instructed to select carbohydrates characterized by low-mod glycemic index. No energy restriction**.** Target CHO 45-50%/fat 30-35%/ remainder protein at snacks and meals. *Comp: Conventional reduced fat diet* – pts instructed to limit dietary fat, increase grains, fruits, & vegetables. Negative energy balance 250-500 kcal/day. CHO 55-60%/fat 25-30%/remainder protein. **Intensity level of intervention:** *group vs. individual*: NR *# sessions, over what time period:* 12 dietary counseling sessions over 6 mos during intervention, 2 dietary counseling sessions over 6 mos during f/u. *length of sessions:* not stated *total contact time:* unable to calculate **Primary care feasible/referable:** Yes, referable	**Weight status:** Total body mass and fat mass measured by dual-energy x-ray absorptiometry, ht using a wall-mounted stadiometer. **Time points**: 0, 6, 12 months **Behavioral:** *Diet*- yes, reported Glycemic load (g/1000 kcal) Fat (% energy) Energy (kcal) *PA* - NR **Physiologic:** *Lipids* – NR *Glucose tolerance* – Yes, reported (HOMA) *Blood pressure*: NR *Physical fitness*: NR **Adverse events**: NR **Health Outcomes:** NR **Others:** Adherence to diet	*Change in BMI:* Comp: 0.6 kg/m^2 (0.5) Exp: -1.2 kg/m^2 (0.7) P=0.02 Note: baseline group differences in fat mass were not controlled for in outcome analyses.	Did not assess	**Quality:** Fair **Financial deposits:** NR **Financial disclosures:** NR
Epstein et al 2001[3]	**Random allocation:** yes,	**N randomized, completed:** 67,	**Description of intervention:** Compares two groups that	**Weight status:** *Ht** using a	*Note:* Primary goal was to detect interaction effect of	Did not assess	**Quality:** Fair

Appendix L. Evidence Table: Intervention Trials 2001-Present

Study	Methods	Participants	Interventions	Outcomes	Results	Adverse Effects	Comment
Setting: Research clinic **Location:** Buffalo, NY	but unclear how randomization scheme was generated **Blinding:** Not mentioned **Length of Intervention and f/u** *Int*: 6 months *F/u:* 12 months **Unit of allocation:** Families **Unit of analysis:** targeted child and sibling **Protection against contamination:** unclear **Drop outs (%)** 16%	56 **Age range:** 8-12 years ; 10.4 (1.2) yrs **% male:** 51.8% **% minority or non-white**: not reported **Weight entry criteria:** > 85th %ile BMI for age and sex and < 100% over the average BMI for age and sex **Weight on entry:** *% overweight:* 60.2 % (18.9); (based on comparisons of the participant BMI to the 50th BMI %ile for age and sex *BMI* 27.4 kg/m^2 (3.6) **Recruitment pool:** Children and parents living at home from families who participated in the Childhood Weight Control Program at the University of Buffalo and expressed interest.	both received comprehensive, family-based behavior change program but differed in physical activity component (increased PA vs combined increased PA + decreased sedentary behavior). Goal of study is to assess sex differences. For both intervention groups, families met with individual therapist and separate parent and child group meetings on alternate weeks. Diet was Traffic Light Diet. *Increased PA*: Reinforced for increasing moderate or greater PA (3 METS or greater). Goal was to increase activity in 30 min increments from baseline up to 180 min/week. *Combined increased PA + decreased sedentary activity:* Same increased PA goals. Additional goals: decrease 5 hr/wk of watching TV from baseline to meet goal of 15 hrs/wk. **Intensity level of intervention** *group vs. individual:* Individual and group *# sessions*: 20 *over what time period*: 6 months *length of sessions*: 30 minutes *total contact time in minutes:* 600 minutes **Primary care feasible/referable:** Yes, referable	laboratory-constructed height board or a stadiometer *Weight** using a medical balance beam scale *% overweight* *Self-reported data were used when individuals were unable to attend assessments (7.3% of observations) and were adjusted. **Time points :** Baseline, 6 months, 12 months **Behavioral:** *Diet:* NR *PA:* NR **Physiological:** *Lipids:* NR *glucose tolerance*: NR *blood pressure*: NR *physical fitness*: NR **Adverse Events:** NR **Health Outcomes:** NR **Others:** adherence to diet and PA, but data not reported by treatment subgroups	gender with treatment group. Also, statistics were done on % overweight change, which is only depicted graphically. **Change in BMI (kg/m^2)** *Increase PA + decrease SB:* Girls: +1.00 kg/m2 (1.73) Boys: -1.76 kg/m2 (1.86) *Increase PA alone:* Girls: -0.27 (1.37) Boys: -0.65 (1.37) Group x sex interaction in rate of change in percentage overweight: $F_{(2,102)}$=5.16, p=0.007, ES=0.25) (Boys lost more weight than girls within each treatment group, especially in increased PA + decreased SB group)		**Financial deposits:** $75 deposited by parents return contingent on completing 75% of sessions and attending 6 & 12 month f/u. **Financial disclosures**: NR
Gutin et al / Kang et al 2002[4,5]	**Random allocation:** Yes, but do not state	**N randomized, completed:** 80, number	**Description of intervention/ control conditions:** Lifestyle education (LSE) only was	**Weight status:** • Total body composition and %	**Results for 8 months:** **Change in body fat:** *LSE alone:* -0.11 % (0.57)	NR	**Quality:** Fair-to-poor

Appendix L. Evidence Table: Intervention Trials 2001-Present

Study	Methods	Participants	Interventions	Outcomes	Results	Adverse Effects	Comment
Setting: Research clinic **Location:** Georgia	method of generating randomization or concealment. **Blinding:** Not reported **Length of Intervention and f/u:** 8 months intervention period **Unit of allocation:** individual adolescents **Unit of analysis:** individuals **Protection against contamination:** Tried to prevent by having PA group subjects have lifestyle class at different time than control group **Drop outs (%):** unclear, 29%	completed is unclear-57 **Age range:** 13-16 yrs, mean not reported. **% male:** 32.5% **% minority or non-white**: 68.8% black **Weight entry criteria:** triceps skinfold thickness > 85th %ile for sex, ethnicity, and age. **Weight on entry:** *BMI*–NR; *% body fat:* white boys-40.7%(2.2) white girls-45.8%(1.5) black boys-43.9%(2.3) black girls-45.2%(0.9) **Recruitment pool:** children who attended schools near research institute and advertisements in community and hospital newspapers	compared to LSE + moderate and LSE + high intensity PA *LSE:* classes that included principles of learning and behavior modification, information on nutrition and PA, psychosocial factors, problem solving, and coping skills. *LSE + mod PA*: same LSE plus exercise sessions. Individual goals based on VO2 and energy expenditure. Target HR calculated. *LSE + high intensity PA:* Same as above except target HR was higher. Overall goal calorie expenditure was the same as in moderate PA group. **Intensity level of intervention** *group vs. individual*: group *# sessions:* 16 for LSE group; 160 (5 sessions/wk) for LSE + PA groups *length of sessions*: 1 hour for LSE, variable for physical training *total contact time in minutes:* unable to calculate *over what time period:* 8 months **Primary care feasible/referable:** Yes, referable	body fat by DEXA; • Visceral adipose tissue and subcutaneous abdominal adipose tissue by MRI • Body wt by electronic scale • Ht by stadiometer **Time points:** Baseline, 4 mos, 8 mos **Behavioral:** *Diet*: yes, reported *PA* **:** yes, reported **Physiological:** *Lipids:* yes, reported *Glucose tolerance:* yes, reported (fasting insulin and glucose levels) *Blood pressure:* yes, reported *Physical fitness:* yes, reported (VO2 at HR of 170 bpm (mL*Kg^{-1}*min^{-1})) **Adverse events:** NR **Health outcomes:** NR	*LSE + MIPT:* -1.42 (0.84) *LSE + HIPT:* -2.85 (1.25) P=0.111 **Change in visceral adipose tissue:** *LSE alone:* -11.56 cm^3 (10.47) *LSE + MIPT:* -49.00 cm^3 (15.73) *LSE + HIPT:* -48.73 cm^3 (15.71) P=0.066 Also present "effectiveness analyses" including only participants who attended > 40% of training sessions.		**Financial deposits:** No, but received financial compensation for attending PA training ($1/session) sessions and LSE ($5 per class) **Financial disclosures:** NR
Saelens et al 2002[6] **Setting**	**Random allocation:** Yes, but do not state how	**N randomized, completed:** 44, 39 completed treatment, 37	**Description of intervention/ control conditions:** *Exp: Healthy Habits (HH):* Developmentally-tailored	**Weight status:** Weight using calibrated standard digital scale or beam	**Results at 7 months:** **BMI:** Base-line: [illegible] 7 mos: [illegible]	No significant differences in problematic eating from	**Quality:** Good

Appendix L. Evidence Table: Intervention Trials 2001-Present

Study	Methods	Participants	Interventions	Outcomes	Results	Adverse Effects	Comment
Primary care **Location:** southern California	randomization scheme was generated. Randomization assignment concealed in opaque envelopes. **Blinding:** Not reported **Length of Intervention and f/u:** 4 mos, 7 mos (total 7 mos) **Unit of allocation:** individual patients **Unit of analysis:** individual patients **Protection against contamination** – not described **Drop outs (%)** 11% at 4 mos, 16% at 7 mos.	completed f/u assessment **Age range:** 12-16 yrs; 14.2 (1.2) yrs **% male:** 59.1% **% minority or non-white:** 30% non-white (4.5% African American) **Weight entry criteria:** 20% to 100% above the median (50th %ile) for BMI for sex and age **Weight on entry:** BMI 30.7 (3.1) **Recruitment pool:** two pediatric primary care clinics	behavioral skills training program delivered via computer and telephone materials. Computer program assessment of PA and eating behavior. Individually tailored plan for increasing PA, decreasing SB, reducing dietary fat, overeating, snacking, and increasing fruit/vegetable intake discussed with pediatrician. Followed by phone calls by counselors with bachelor's degree in psychology or nutrition. Self-monitoring of food, beverage, intake and PA. Parent information sheets. *Comp: Typical Care (TC)*: Single non-tailored counseling session with a pediatrician covering motivation for wt-related change, health consequences, review of food guide pyramid and PA recommendations for adolescents, and encourage persistence with health behavior changes. Same pediatricians performed counseling for both HH and TC groups. **Intensity level of intervention** *group vs. individual*: individual *# sessions:* 13 sessions *length of sessions:* 10-20 min phone calls, not reported for pediatrician visit *total contact time in minutes:* around 200 minutes, *over what time period*: 4 months **Primary care**	scale; height using a stadiometer. Calculated BMI, BMI z scores, and % overweight. **Time points:** baseline, 4 mos, 7 mos. **Behavioral:** *Diet:* yes, assessed *PA:* yes, assessed **Physiologic:** *Lipids:* NR *Glucose tolerance:* NR *Blood pressure:* NR *Physical fitness:* NR **Adverse events:** see health outcomes **Health outcomes:** eating disorder psychopathology **Other outcomes:** Behavior skills use; Participant satisfaction	<table><tr><td>Comp</td><td>30.7 (3.1)</td><td>32.1 (3.8)</td></tr><tr><td>Exp</td><td>31.0 (3.5)</td><td>31.1 (4.5)</td></tr></table> **Percentage overweight:** <table><tr><td></td><td>Base-line</td><td>7 mos</td></tr><tr><td>Comp</td><td>62.3 (17.4)</td><td>66.4 (20.1)</td></tr><tr><td>Exp</td><td>62.0 (20.5)</td><td>59.6 (24.6)</td></tr></table> Change in baseline to follow-up BMI z-score between groups: $F_{(1,42)} = 3.11$, $p < 0.09$ **Decreased BMI z-score from baseline:** Comp: 15.8% Exp: 55.6% $\chi^2_{(1)} = 6.41$, $p < 0.02$	baseline to post-treatment (4 mos)	**Financial deposits:** NR, but patients received $25 for post-treatment and $25 follow-up assessments Financial disclosures: NR

Appendix L. Evidence Table: Intervention Trials 2001-Present

Study	Methods	Participants	Interventions	Outcomes	Results	Adverse Effects	Comment
			feasible/referable: yes, referable				
White 2003[7]/Williamson unpublished data **Setting** : Research clinic (internet-based) **Location:** Louisiana	**Random allocation:** Yes, stratified by age and BMI but neither randomization procedure nor allocation concealment are described. **Blinding:** not described **Length of Intervention and f/u:** 6 months for this report (planned 2 years for full study) **Unit of allocation:** Child & parent **Unit of analysis**: Child & parent separately **Protection against contamination**: Experimental and comparison group participants logged onto separate password-protected websites and could access only the website that was appropriate for their treatment	**N randomized, completed:** 57, 50 **Age range:** *Eligible:* 11-15 years *Actual:* 13.19 years (1.37) **% male**: 0% **% minority or non-white:** 100% African American **Weight entry criteria**: > 85th BMI %ile (using BMI-for-age growth charts from the National Center for Health Statistics) AND one obese biological parent (BMI>27 and willing to participate in study) **Weight on entry**: BMI: 36.34 kg/m^2 (7.89); DEXA %fat: 45.84 (7.4); BIA-fat: 42.41 (6.52) **Recruitment pool:** Community	**Description of intervention:** Randomized controlled trial of the Health Improvement Program for Teens (HIP-Teens) program, a primarily internet- and e-mail-based, family-based intervention study in African American adolescent girls comparing two treatment conditions: *Exp:* a comprehensive behavioral intervention *Comp:* an education only intervention (diet and broad physical activity). Full study period is 2 years but this publication reports results from first 6 months. **Intensity level of intervention** *group vs. individual*: individual *# sessions, over what time period:* weekly website logins over 6 months; approximately 600 website logins for experimental group and 200 logins for comparison group. *length of sessions:* NR *total contact time (min):* NR **Primary care feasible/referable?** Yes, feasible or referable	**Weight status:** *Primary measure:* Change in % body fat measured by dual-energy x-ray absorptiometry (DEXA) *Secondary measures:* Change in % body fat using bioelectrical impedance analysis; BMI **Time points when weight was measured:** Baseline, 6 months **Behavioral:** *Diet-* yes, reported (Multi-pass 24-hr recall of dietary intake; Food Frequency Questionnaire; Weight Loss Behavior Scale (WLBS), which measures concern about weight loss behaviors)) *PA-* yes, reported (WLBS) **Physiological:** *Lipids/ lipoproteins:* NR *Glucose tolerance:* NR *blood pressure:* NR *physical fitness:* NR **Adverse events:** See health outcomes **Health Outcomes:** Depression, self-esteem, eating	**6 month results:** Change % body fat – DEXA: *Exp:* -1.04 (2.00) *Comp:* 0.38 (2.95) (t=2.11, p=0.02) Change in % body fat-BIA: *Exp*: -0.05 (1.20) *Comp*: 0.75 (1.49) (t=2.23, p=0.01) Change in BMI (kg/m2): *Exp*: -0.24 (1.38) *Comp*: 0.71 (1.19) (t=2.77, p < 0.01) Change in wt (kg): *Exp*: 0.55 (3.26) *Comp*: 2.40 (2.86) (t=2.28, p=0.03)	Depression, self-esteem, eating disorder pathology, and satisfaction with life were measured at baseline but changes over time are not reported	**Quality:** Good **Financial deposits:** No, but received financial incentives from a computer purchase voucher ($700), free internet-services, gifts for attending sessions, and payment for assessments. **Financial disclosures:** NR

Appendix L. Evidence Table: Intervention Trials 2001-Present

Study	Methods	Participants	Interventions	Outcomes	Results	Adverse Effects	Comment
	assignment **Drop outs** 12%			disorder pathology, and satisfaction with life were measured at baseline but changes over time are not reported **Others:** -Child dietary self-efficacy -adherence measured by # website "hits", completing weekly quizzes -Weight loss behavior scale (measuring concern for wt loss behaviors) -computer anxiety -Parent measures (multiple measures)			

References

1. Berkowitz RI, Wadden TA, Tershakovec AM, Cronquist JL. Behavior therapy and sibutramine for the treatment of adolescent obesity: a randomized controlled trial. *JAMA* 2003; 289(14):1805-1812.

2. Ebbeling CB, Leidig MM, Sinclair KB, Hangen JP, Ludwig DS. A reduced-glycemic load diet in the treatment of adolescent obesity. *Archives of Pediatrics & Adolescent Medicine* 2003; 157(8):773-779.

3. Epstein LH, Paluch RA, Raynor HA. Sex differences in obese children and siblings in family-based obesity treatment. *Obesity Research* 2001; 9(12):746-753.

4. Gutin B, Barbeau P, Owens S, Lemmon CR, Bauman M, Allison J et al. Effects of exercise intensity on cardiovascular fitness, total body composition, and visceral adiposity of obese adolescents. *American Journal of Clinical Nutrition* 2002; 75(5):818-826.

5. Kang HS, Gutin B, Barbeau P, Owens S, Lemmon CR, Allison J et al. Physical training improves insulin resistance syndrome markers in obese adolescents. *Medicine & Science in Sports & Exercise* 2002; 34(12):1920-1927.

6. Saelens BE, Sallis JF, Wilfley DE, Patrick K, Cella JA, Buchta R. Behavioral weight control for overweight adolescents initiated in primary care. *Obesity Research* 2002; 10(1):22-32.

7. White MA. *Mediators of Weight Loss in an Internet-Based Intervention for African-American Adolescent Girls*. Louisiana State University, 2003.

Appendix M. Evidence Table: Intervention Study Characteristics

Study Reference	N Randomized Setting Country	Age % Male, % Non-White	Entry Wt Criteria	Mean Entry Wt	Group#	Weight Outcome: Units of Measure	Weight Outcome: 6-8 mo	Weight Outcome: 12-18 mo	Weight Outcome: 24-30 mo	Physiological Outcomes	Childhood Health Outcomes	Study Quality
Pharmacologic												
Berkowitz et al 2003[1]	82 adolescents University-based specialty research clinic USA	13-17 33% 45%	BMI 32-44	BMI 37.8 (3.8); BMI z-score: 2.4 (0.2)	#1: Sibutramine #2: Placebo	change in BMI (% change from entry BMI)	-8.5% -4.0%	N/A N/A	NR NR	lipids yes; glucose tolerance yes; blood pressure yes; physical fitness NR	NR	Good
Low-glycemic load												
Ebbeling et al 2003[2]	16 adolescents Research clinic USA	13-21 31% 19%	BMI > 95%ile for sex, age	BMI 34.9 kg/m2 (reduced glycemic group); 37.1 kg/m2 (conventional diet group)	#1: Reduced glycemic load diet #2: Reduced fat diet	absolute change in BMI	NR NR	-1.2 kg/m2 0.6 kg/m2	NR NR	lipids NR; glucose tolerance yes; blood pressure NR; physical fitness NR	NR	Fair
Changes in physical activity												
Epstein et al 1985a[3]	41 families NR; presumably a research clinic USA	8-12 40% NR	> 20% over ideal weight for height, age, sex	48% overweight	#1: aerobic PA #2: lifestyle PA #3: low-intensity calisthenics PA	% overweight change	-17.4% -19.6% -20.7%	-16.3% -16.1% -17.5%	-6.8% -18.0% -7.2%	lipids NR, glucose tolerance NR, blood pressure NR, physical fitness yes	NR	Fair
Epstein et al 1985b[4]	23 children NR; presumably a research clinic USA	8-12 0% NR	at least 20% overweight for height and age	48 % overweight for age and height	#1: diet + PA #2: diet alone	% overweight change	-27.5% -18.8%	-25.4% -18.7%	NR NR	lipids NR; glucose tolerance NR; blood pressure NR; physical fitness yes	NR	Fair

Appendix M. Evidence Table: Intervention Study Characteristics

Study Reference	N Randomized Setting Country	Age % Male, % Non-White	Entry Wt Criteria	Mean Entry Wt	Group#	Weight Outcome: Units of Measure	Weight Outcome: 6-8 mo	Weight Outcome: 12-18 mo	Weight Outcome: 24-30 mo	Physiological Outcomes	Childhood Health Outcomes	Study Quality
Epstein et al 1995[5]	61 families NR; presumably a research clinic USA	8-12 27% 4%	20-100% overweight	51.8% overweight	#1: increased PA	% overweight change	NR	-18.7%	NR	lipids NR; glucose tolerance NR; blood pressure NR; physical fitness yes	NR	Fair
					#2: decreased sedentary behavior		NR	-10.3%	NR			
					#3: combined		NR	-8.7%	NR			
Epstein et al 2000b[6]	90 families Childhood obesity research clinic USA	8-12 32% NR	20-100% overweight	62% overweight	#1: PA low dose	% overweight change	-25.6%	NR	-12.4%	lipids NR; glucose tolerance NR; blood pressure NR; physical fitness yes	NR	Fair
					#2: PA high dose		-26.4%	NR	-13.2%			
					#3: decreased sedentary behavior low dose		-22.4%	NR	-11.6%			
					#4: decreased sedentary behavior high dose		-27.4%	NR	-14.3%			
Gutin et al[7]/ Kang et al[8] 2002	80 Research clinic USA	13-16 33% 69%	triceps skinfold thickness > 85%ile for sex, ethnicity, and age	40.7 % body fat (white boys); 45.8% body fat (white girls); 43.9% body fat (black boys); 45.2% body fat (black girls)	#1: lifestyle education only (LSE)	change in % body fat	-0.1%	NR	NR	lipids yes; glucose tolerance yes; blood pressure yes; physical fitness yes	NR	Fair-to-Poor
					#2: LSE + moderate PA		-1.4%	NR	NR			
					#3: LSE + high intensity PA		-2.9%	NR	NR			
Epstein et al 2001[9]	67 families NR; presumably a research clinic USA	8-12 52% NR	at or > 85%ile BMI for age and sex and < 100% over the average BMI for age and sex	60.2% overweight (compared to the 50%ile BMI for age and sex); BMI 27.4 kg/m2 (3.6 kg/m2)	#1: increased PA	change in absolute BMI	reported graphic-ally	girls: -0.27 kg/m2; boys: -1.76 kg/m2	NR	lipids NR; glucose tolerance NR; blood pressure NR; physical fitness NR	NR	Fair
					#2: increased PA + decreased SB		reported graphic-ally	girls: 1.0 kg/m2; boys: -1.76 kg/m2	NR			

Appendix M. Evidence Table: Intervention Study Characteristics

Study Reference	N Randomized Setting Country	Age % Male, % Non-White	Entry Wt Criteria	Mean Entry Wt	Group#	Weight Outcome: Units of Measure	Weight Outcome: 6-8 mo	Weight Outcome: 12-18 mo	Weight Outcome: 24-30 mo	Physiological Outcomes	Childhood Health Outcomes	Study Quality
Problem solving vs. usual care or behavioral therapy												
Graves et al 1988[10]	40 children NR; presumably a research clinic USA	6-12 NR NR	at least 20% overweight for age, sex, and height	52%-56% overweight for age, sex, and height	#1: BT + parent problem solving	% overweight change	-24.5%	NR	NR	lipids NR; glucose tolerance NR; blood pressure NR; physical fitness NR	NR	Fair
					#2: BT only		-10.2%	NR	NR			
					#3: instruction only		-9.5%	NR	NR			
Epstein et al 2000a[11]	67 children NR; presumably a research clinic USA	NR, mean (sd) 10.3 (1.1) yrs 48% 4% (2% black, 2% Hispanic)	> 20% overweight	BMI 27.4 (3.2)	#1: PS to parent and child	change in absolute weight (kg) (analyzed change in BMI z-score)	-6.8 kg	-1.2 kg	11.9 kg	lipids NR; glucose tolerance NR; blood pressure NR; physical fitness NR	problem behaviors	Fair
					#2: PS to child only		-7.0 kg	-2.4 kg	7.2 kg			
					#3: no PS		-6.2 kg	-1.3 kg	7.2 kg			
Behavioral therapy vs no treatment/usual care												
Epstein 1985c[12]	24 children NR; presumably a research clinic USA	5-8 0% NR	No details given	39-42% overweight	#1: BT	% overweight change	-23.7%	-26.3%	NR	lipids NR; glucose tolerance NR; blood pressure NR; physical fitness NR	NR	Fair
					#2: Education only		-11.6%	-11.2%	NR			
Senediak and Spence 1985[13]	45 children NR; presumably a research clinic USA	6-13 approximately 66% NR	at least 20% overweight for height, age, and sex	37.22% overweight	#1: rapid schedule BT	% overweight change	-14.7%	NR	NR	lipids NR; glucose tolerance NR; blood pressure NR; physical fitness NR	NR	Fair-to-Poor
					#2: gradually decreasing schedule BT		-18.3%	NR	NR			
					#3: non-specific treatment controls		-10.9%	NR	NR			
					#4: wait list controls		NR	NR	NR			

Appendix M. Evidence Table: Intervention Study Characteristics

Study Reference	N Randomized Setting Country	Age % Male, % Non-White	Entry Wt Criteria	Mean Entry Wt	Group#	Weight Outcome: Units of Measure	Weight Outcome: 6-8 mo	Weight Outcome: 12-18 mo	Weight Outcome: 24-30 mo	Physiological Outcomes	Childhood Health Outcomes	Study Quality
Mellin et al 1987[14]	66 adolescents Rural health dept; rural nutrition private practice, suburban medical clinic; urban outpatient clinic USA	12-18 21% 22%	No details given	30-37% overweight for age, sex, and height	#1: SHAPEDOWN group (Cognitive, behavioral, affective treatment)	% overweight change	NR	-9.9%	NR	lipids NR; glucose tolerance NR; blood pressure NR; physical fitness NR	depression; self-esteem	Fair
					#2: no treatment controls		NR	-0.1%	NR			
Flodmark et al 1993[15]	44 children (plus 50 matched controls) Outpatient referral clinical setting Sweden	10-11 48% NR (Swedish)	BMI > 23.0 kg/m^2	25.5 kg/m^2 (conventional treatment group); 24.7 kg/m^2 (family therapy group): 25.1 kg/m^2 (control group)similar to	#1: conventional treatment	change in BMI (kg/m^2)	NR	0.6 kg/m^2	1.6 kg/m^2	lipids NR; glucose tolerance NR; blood pressure NR; physical fitness yes	NR	Fair
					#2: family therapy		NR	0.3 kg/m^2	1.1 kg/m^2			
					#3: matched controls - untreated		NR	NR	2.8 kg/m^2			
Saelens et al 2002[16]	44 Primary care clinical setting USA	12-16 59% 30%	20-100% above median (50%ile) for BMI for sex and age	BMI 30.7 (3.1);	#1: Healthy habits intervention	% overweight change & change in BMI	-2.4%, 0.1 kg/m2	NR	NR	lipids NR; glucose tolerance NR; blood pressure NR; physical fitness NR	eating disorder psychopathology (adverse effect)	Good
					#2: Typical care		4.1%, 1.4 kg/m2	NR	NR			
White 2003[17]/ Williamson unpublished data	57 adolescents research clinic USA	11-15 0% 100%	BMI > 85th %ile for age and gender	BMI 36.34 kg/m^2; 98.3 BMI %ile	#1: Behavioral	change in % body fat; change in BMI	-1.12%; 0.19 kg/m2	NR	NR	lipids NR; glucose tolerance NR; blood pressure NR; physical fitness NR	depression, self-esteem, satisfaction with life, and eating disorder pathology measured but change over time NR	Good
					#2: Education only		0.43%; 0.65 kg/m2	NR	NR			

Appendix M. Evidence Table: Intervention Study Characteristics

Study Reference	N Randomized Setting Country	Age % Male, % Non-White	Entry Wt Criteria	Mean Entry Wt	Group#	Weight Outcome: Units of Measure	Weight Outcome: 6-8 mo	Weight Outcome: 12-18 mo	Weight Outcome: 24-30 mo	Physiological Outcomes	Childhood Health Outcomes	Study Quality
Varying degrees of family involvement												
Israel and Shapiro 1985[18]	33 children NR; presumably a research clinic USA	8-12 30% NR	at least 20% overweight for height	53.13% overweight (BT only); 45.88% (parent training group); 56.02% (controls)	#1: BT only #2: BT + parent training in child management #3: wait list controls	% overweight change	NR NR NR	-1.3% -10.2% NR	NR NR NR	lipids NR; glucose tolerance NR; blood pressure NR; physical fitness NR	NR	Fair-to-Poor
Wadden et al 1990[19]	47 girls NR; presumably a research clinic USA	12-16 0% 100% black	> 10 kg overweight for age, sex, and height	95.1 kg; BMI 35.6 kg/m^2	#1: child alone #2: mother and child together #3: mother and child separate	change in weight	3.0 kg 1.7 kg 3.5 kg	NR NR NR	NR NR NR	lipids yes; glucose tolerance NR; blood pressure yes; physical fitness NR (physiological outcomes at 4 mo)	self-esteem; depression (reported at 4 mo)	Fair-to-Poor
Israel et al 1994[20]	36 families NR; presumably a research clinic USA	8-13 NR NR	at least 20% overweight for weight, height, and sex	46.0% overweight (standard treatment group); 48.1% (enhanced child involvement group)	#1: standard treatment (parents primarily responsible) #2: enhanced child involvement	% overweight change	-12.5% -15.6%	-0.8% -5.8%	6.4% -4.8%	lipids NR; glucose tolerance NR; blood pressure NR; physical fitness NR	NR	Fair-to-Poor

Appendix M. Evidence Table: Intervention Study Characteristics

Study Reference	N Randomized Setting Country	Age % Male, % Non-White	Entry Wt Criteria	Mean Entry Wt	Group#	Weight Outcome: Units of Measure	Weight Outcome: 6-8 mo	Weight Outcome: 12-18 mo	Weight Outcome: 24-30 mo	Physiological Outcomes	Childhood Health Outcomes	Study Quality
Golan et al 1998[21]	60 children NR; presumably a research clinic Israel	6-11 38% NR (Israeli)	> 20 % overweight for age, height and gender	39.6% overweight (experimental group); 39.1% (conventional group)	#1: parents exclusive agents of change	% overweight change	NR	-14.7%	NR	lipids NR; glucose tolerance NR; blood pressure NR; physical fitness NR	NR	Fair
					#2: conventional: children responsible for own wt loss		NR	-8.1%	NR			
Cognitive behavioral therapy												
Duffy and Spence 1993[22]	29 children NR; presumably a research clinic Australia	7-13 21% NR, Australian	> 15% overweight for age, ht and sex	48.4% overweight	#1: BT + cognitive self-management	% overweight change	-8.9%	NR	NR	lipids NR; glucose tolerance NR; blood pressure NR; physical fitness NR	NR	Fair-to-Poor
					#2: BT + relaxation placebo		-9.2%	NR	NR			
Mastery criteria												
Epstein et al 1994[23]	44 families NR; presumably a research clinic USA	8-12 26% NR	20-100% overweight for height	59.6% over the 50th%ile for BMI (equivalent to > 95%ile BMI for age; similar to 50-60% overweight range)	#1: mastery criteria & contingent reinforcement	% overweight change	-30.1%	-26.5%	-15.4%	lipids NR; glucose tolerance NR; blood pressure NR; physical fitness NR	NR	Fair
					#2: comparison group		-20.6%	-16.7%	-10.6%			

References

1. Berkowitz RI, Wadden TA, Tershakovec AM, Cronquist JL. Behavior therapy and sibutramine for the treatment of adolescent obesity: a randomized controlled trial. *JAMA* 2003; 289(14):1805-1812.

2. Ebbeling CB, Leidig MM, Sinclair KB, Hangen JP, Ludwig DS. A reduced-glycemic load diet in the treatment of adolescent obesity. *Archives of Pediatrics & Adolescent Medicine* 2003; 157(8):773-779.

3. Epstein LH, Wing RR, Koeske R, Valoski A. A comparison of lifestyle exercise, aerobic exercise, and calisthenics on weight loss in obese children. *Behavior Therapy Vol 16(4) Sep* 1985;345-356.

4. Epstein LH, Wing RR, Penner BC, Kress MJ. Effect of diet and controlled exercise on weight loss in obese children. *J Pediatr* 1985; 107(3):358-361.

5. Epstein LH, Valoski AM, Vara LS, McCurley J, Wisniewski L, Kalarchian MA et al. Effects of decreasing sedentary behavior and increasing activity on weight change in obese children 3814. *Health Psychology* 1995; 14(2):109-115.

6. Epstein LH, Paluch RA, Gordy CC, Dorn J. Decreasing sedentary behaviors in treating pediatric obesity. *Arch Pediatr Adolesc Med* 2000; 154(3):220-226.

7. Gutin B, Barbeau P, Owens S, Lemmon CR, Bauman M, Allison J et al. Effects of exercise intensity on cardiovascular fitness, total body composition, and visceral adiposity of obese adolescents. *American Journal of Clinical Nutrition* 2002; 75(5):818-826.

8. Kang HS, Gutin B, Barbeau P, Owens S, Lemmon CR, Allison J et al. Physical training improves insulin resistance syndrome markers in obese adolescents. *Medicine & Science in Sports & Exercise* 2002; 34(12):1920-1927.

9. Epstein LH, Paluch RA, Raynor HA. Sex differences in obese children and siblings in family-based obesity treatment. *Obesity Research* 2001; 9(12):746-753.

10. Graves T, Meyers AW, Clark L. An evaluation of parental problem-solving training in the behavioral treatment of childhood obesity. *J Consult Clin Psychol* 1988; 56(2):246-250.

11. Epstein LH, Paluch RA, Gordy CC, Saelens BE, Ernst MM. Problem solving in the treatment of childhood obesity. *Journal of Consulting & Clinical Psychology* 2000; 68(4):717-721.

12. Epstein LH. Effects of family-based behavioral treatment on obese 5-to-8-year-old children. *Behavior Therapy Vol 16(2) Mar* 1985;-212.

13. Senediak C, Spence SH. Rapid versus gradual scheduling of therapeutic contact in a family based behavioural weight control programme for children. *Behavioural Psychotherapy Vol 13(4) Oct* 1985;-287.

14. Mellin LM, Slinkard LA, Irwin CE, Jr. Adolescent obesity intervention: validation of the SHAPEDOWN program 3987. *Journal of the American Dietetic Association* 1987; 87(3):333-338.

15. Flodmark CE, Ohlsson T, Ryden O, Sveger T. Prevention of progression to severe obesity in a group of obese schoolchildren treated with family therapy. *Pediatrics* 1993; 91(5):880-884.

16. Saelens BE, Sallis JF, Wilfley DE, Patrick K, Cella JA, Buchta R. Behavioral weight control for overweight adolescents initiated in primary care. *Obesity Research* 2002; 10(1):22-32.

17. White MA. *Mediators of weight loss in an internet-based intervention for African-American adolescent girls*. Louisiana State University, 2003.

18. Israel AC, Shapiro LS. Behavior problems of obese children enrolling in a weight reduction program. *J Pediatr Psychol* 1985; 10(4):449-460.

19. Wadden TA, Stunkard AJ, Rich L, Rubin CJ, Sweidel G, McKinney S. Obesity in black adolescent girls: a controlled clinical trial of treatment by diet, behavior modification, and parental support 3928. *Pediatrics* 1990; 85(3):345-352.

20. Israel AC, Guile CA, Baker JE, Silverman WK. An evaluation of enhanced self-regulation training in the treatment of childhood obesity. *Journal of Pediatric Psychology* 1994; 19(6):737-749.

21. Golan M, Fainaru M, Weizman A. Role of behaviour modification in the treatment of childhood obesity with the parents as the exclusive agents of change. *Int J Obes Relat Metab Disord* 1998; 22(12):1217-1224.

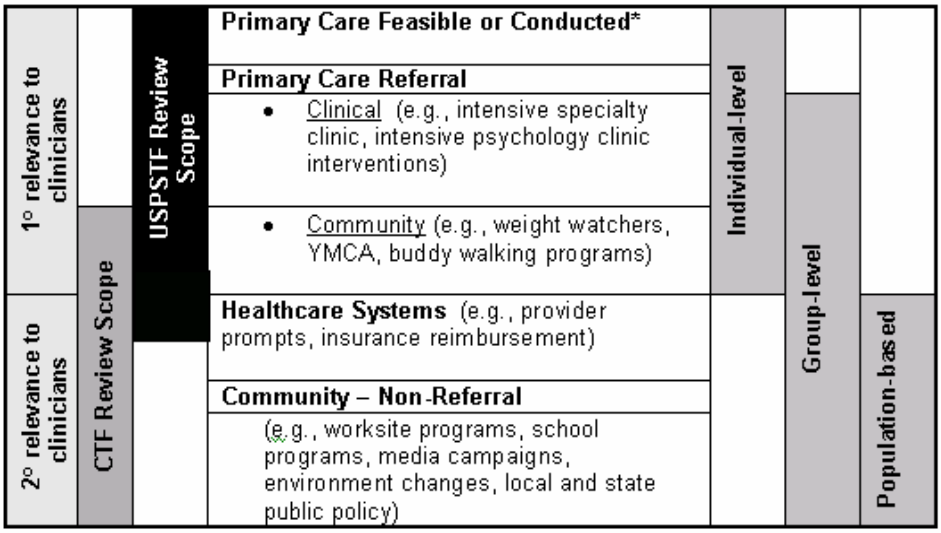
22. Duffy G, Spence SH. The effectiveness of cognitive self-management as an adjunct to a behavioural intervention for childhood obesity: a research note. *J Child Psychol Psychiatry* 1993; 34(6):1043-1050.

23. Epstein LH, McKenzie SJ, Valoski A, Klein KR, Wing RR. Effects of mastery criteria and contingent reinforcement for family-based child weight control 3838. *Addictive Behaviors* 1994; 19(2):135-145.

Screening and Intervention for Overweight in Children and Adolescents Categorization Scheme for Settings

The existing systematic evidence reviews of the childhood obesity literature have categorized the study settings as follows: school-based, clinical, or community-based. These interventions may or may not involve parents, may target individuals or groups, and vary in intensity and duration.

We have developed the diagram below to clarify which types of interventions are within the scope of the U.S. Preventive Services Task Force (USPSTF), the Community Task Force (CTF), or both. Most of the studies evaluated in the Cochrane review of treatment were set in specialist/research clinics and thus fall under clinical primary care referral interventions. In contrast, studies in the Cochrane review of obesity *prevention* were set in schools and fall under community non-referral. The diagram indicates which settings are of primary or secondary relevance for clinicians. Once refined, such an approach could be used to illustrate settings for reviews of other preventive topics relevant to clinicians, or for other audiences in addition to clinicians, such as health departments.



*An intervention must be judged as feasible to be conducted in primary care by demonstration of being conducted in primary care research setting or by judgment that it would be feasible in "usual" primary care; see criteria below.

Criteria for Interventions Judged to Be Relevant/Feasible to Primary Care

WHO TARGETED: Somehow involve individual-level identification of being a patient/in need of intervention

WHO DELIVERED: Usually involve primary care clinicians (physicians in family practice, internal medicine, ob-gyn, pediatrics, general practitioner), other physicians, nurses, nurse practitioners, physician assistants or related clinical staff (dietitians, health educators, other counselors) in some direct or indirect way—or, at least, the intervention would be seen as connected to the health care system by the participant.

HOW DELIVERED: To individuals or in small groups (15 or less). Do not involve only or primarily group-level interventions outside the primary care setting to achieve behavioral changes. Generally involve no more than 8 group sessions total, and intervention time period is no longer than 12 months.

WHERE DELIVERED: Could be delivered anywhere (including via the web, interactive technologies, in the home) if linked to primary care as above.

Appendix O. Intervention Characteristic Definitions [1]

Intervention Characteristic Definitions

Comprehensive behavioral treatment: those using a combination of three types of interventions: behavioral modification procedures, a special diet, and an exercise program

Exercise programs

- Broad
- Specific
  - *Aerobic (*e.g., running, jogging, swimming)
    - High intensity
    - Low intensity
  - *Calisthenic* (e.g., sit-ups, toe-touches)
  - *Lifestyle* (e.g., taking staircase, walking to store)

Decreased sedentary behavior [2] (e.g., limiting watching television or video games)

Diet programs

Behavioral modification components

- *Self-monitoring:* having the child document diet-related behaviors or physical activity
- *Stimulus control:* modifying factors that appear to serve as cues leading to inappropriate eating, such as while watching television
- *Eating management:* techniques specifically aimed at modifying the act of eating, such as eating slowly
- *Contingency management:* contingency contracting, where rewards are given for desired eating or exercise behaviors, weight loss, or treatment attendance
- *Cognitive-behavioral techniques:* the attempt to alter maladaptive cognitions related to health behaviors, such as problem-solving during high-risk situations

Parent participation

- *High:* family participated in all aspects of treatment including attending treatment sessions, dietary choices, and behavioral modification techniques
- *Medium:* family involved in many components of treatment, but the child was solely responsible for some significant aspects of treatment, such as dietary choices
- *Low:* parents were minimally involved in treatment such as attending some sessions and providing encouragement

1. Based on Haddock CK, Shadish WR, Klesges RC, Stein RJ. Treatments for childhood and adolescent obesity. *Ann Behav Med* 16 (3):235-244, 1994.
2. Not included in Haddock et al 1994.

Methods for Figures 4 and 5

Ages 8 to 12 Years: Modeled Data

Percent overweight was a common measure used to describe the entry weights and post-intervention weights of participants in studies of children aged 8-12 years. Percent overweight is calculated as follows: 100% x (actual weight – ideal weight for age, height, & sex) ÷ ideal weight for age, height, & sex. The ideal weight for age, height, and sex was typically taken from a reference dataset such as the World Health Organization's report on the assessment of community nutritional status published in 1966.[1] In a majority of studies conducted in children aged 8-12, the participants were on average 40%-60% overweight prior to the intervention. The average change in percent overweight at the time of the last post-intervention follow-up measurement was –10% to –20%. To model these typical entry weight and results, we have calculated the BMI associated with being 50% overweight (i.e., typical mean entry weight), 40% overweight (i.e., change in percent overweight of –10%), 30% overweight (i.e., change in percent overweight of –20%), and ideal weight. These BMI data were plotted on the gender-specific CDC 2000 BMI-for-age growth charts to visually demonstrate how percentage overweight corresponds to BMI percentiles. The entry BMI that is equivalent to 50% overweight is displayed for ages 8, 10, and 12 years and corresponding –10% and –20% changes in overweight are displayed for ages 9, 11, and 13 years in order to demonstrate that the participants grew older during the course of the study. Plots for boys and girls were similar, therefore we present the data for girls only.

Calculation of age-, height-, and sex-specific BMI based on percent overweight: For ages 8-13, the median height was estimated for each age based on the CDC's 2000 sex-specific stature-for-age growth charts. Ideal weight for height, age, and sex was then taken from the same reference dataset used in several of the original studies.[1] Weight if 50%, 40%, or 30% overweight was determined by multiplying the ideal weight by 1.5, 1.4, or 1.3, respectively. The BMI for each age for each category of % overweight was then calculated as follows: 10,000 x weight in kg ÷ (height in cm)^2. Data for each age between 8 and 13 years are displayed in Table P-1.

Table P-1. Data for Modeled Results in Girls Ages 8 to 13 years (Figure 4)

			If Ideal Body Weight[b]		If 50% Overweight[b]		If 40% Overweight[b]		If 30% Overweight[b]	
Gender	Age	Median Ht for Age[a] (cm)	Wt[c] (kg)	BMI[e] (kg/m^2)	Wt[d] (kg)	BMI[e] (kg/m^2)	Wt[d] (kg)	BMI[e] (kg/m^2)	Wt[d] (kg)	BMI[e] (kg/m^2)
Girls	8	128	25.8	15.7	38.7	23.6	36	22.0	33.5	20.5
	9	133	28.6	16.2	42.9	24.3	40	22.6	37.2	21.0
	10	138	31.6	16.6	47.4	24.9	44	23.2	41.1	21.6
	11	144	35.6	17.2	53.4	25.8	50	24.0	46.3	22.3
	12	151	40.8	17.9	61.2	26.8	57	25.1	53	23.3
	13	157	46.5	18.9	69.8	28.3	65	26.4	60.5	24.5

[a] Median height for age based on CDC 2000 stature-for-age growth charts.
[b] Height-, age-, and sex-specific.
[c] Ideal weight for height, age, sex from Jelliffe 1966.[1]
[d] Calculated by multiplying ideal weight by 1.5, 1.4, or 1.3, respectively for 50%, 40%, or 30% overweight.
[e] BMI calculated as follows: 10,000 x (wt in kg) ÷ (ht in cm)^2.

Data from Individual Studies in Adolescents (Figure 5)

For studies that included mostly participants 13 years and older, actual entry weight and outcomes are plotted if these measures were presented using BMI or percentage overweight.[2-7] One study of participants in this age group[8] presented triceps skinfold thickness and percentage body fat and is therefore not included in the figure.

In general, the mean entry BMI and post-intervention change in BMI are reported for the treatment group that had the larger decrease in BMI, and these data are plotted at the mean age at entry for the group with the better result. The center of the box symbol corresponds to the entry BMI. The tip of the arrow drawn for each box represents the average change in BMI at the time of the latest post-intervention measurement. Data for plotted values are presented in Table P-2.

Four studies reported actual mean entry BMI and post-intervention BMI.[3,5-7] However, only two of these reported both the BMI data and mean entry age stratified by treatment groups.[3,7] One study[5] presents mean entry age for all participants across treatment groups, so the BMI data for the treatment group with the best results (Healthy Habits group) are plotted at that age. One study[6] presents entry and post-intervention BMI results across treatment subgroups but presents mean age at entry stratified by treatment subgroup. For this study, the BMI data across subgroups are presented in Figure 4 at the age corresponding to the mean of the mean ages presented for each subgroup.

Of the two studies that did not present mean entry BMI and post-intervention BMI, one[2] presented all age and weight data stratified by treatment subgroups, but the outcomes as percent change in BMI from baseline BMI. In this study, the group that received both sibutramine and behavioral therapy had the better results on average, -8.5% change in BMI from baseline. The actual post-intervention BMI for this treatment group was calculated as follows: mean BMI at entry + (% change in BMI from baseline) x (mean BMI at entry). Entry BMI and post-intervention BMI data were plotted at the mean age at entry for the sibutramine plus behavioral therapy group. The other study[4] presented entry and post-intervention data as percent overweight. Age and height specific BMI were calculated similar to how they were calculated for the 8 to 12 year old age group using the mean age at entry for the group with the best result. For this study, post-intervention BMI was calculated for a participant 17 years old.

Table P-2. Data for Treatment Group with Best Post-Intervention Outcome in Studies of Adolescents That Report BMI-Based or % Overweight-Based Outcomes

Study	Treatment Group with Best Result	Mean Age at Entry	Mean BMI (kg/m^2)at Entry	Post-Intervention Outcomes[1]		Length of f/u Period
				Non-BMI Outcome	Mean BMI (kg/m^2)	
Berkowitz et al 2003[2]	sibutramine + BT	14.1 years	37.5	-8.5% (% decrease in BMI from baseline)	34.3	6 mos
Ebbeling et al 2003[3]	Reduced glycemic load diet	16.9 years	34.9	N/A	33.7	6 mos
Mellin et al 1987[4]	SHAPEDOWN	15.6 years	27.6[2]	-9.9% (change in % overweight)	25.8[2]	15 mos
Saelens et al 2002[5]	Healthy Habits	14.2 years[3]	31.0	N/A	31.1	7 mos
Wadden et al 1990[6]	Mother and Child together	14.0[4]	35.2[5]	N/A	35.4[5]	6 mos
White 2003[7]	Behavioral	13.1	35.3	N/A	35.1	6 mos

[1] All results are reported at the latest time point measured.
[2] Calculated from mean % overweight.
[3] Mean age for participants in both treatment groups.
[4] Mean of mean ages for each subgroup.
[5] BMI values reported across subgroups.

References

1. Jelliffe DB. *The Assessment of the Nutritional Status of the Community*. Monograph Series #53 ed. Geneva: World Health Organization, 1966.

2. Berkowitz RI, Wadden TA, Tershakovec AM, Cronquist JL. Behavior therapy and sibutramine for the treatment of adolescent obesity: a randomized controlled trial. *JAMA* 2003; 289(14):1805-1812.

3. Ebbeling CB, Leidig MM, Sinclair KB, Hangen JP, Ludwig DS. A reduced-glycemic load diet in the treatment of adolescent obesity. *Archives of Pediatrics & Adolescent Medicine* 2003; 157(8):773-779.

4. Mellin LM, Slinkard LA, Irwin CE, Jr. Adolescent obesity intervention: validation of the SHAPEDOWN program 3987. *Journal of the American Dietetic Association* 1987; 87(3):333-338.

5. Saelens BE, Sallis JF, Wilfley DE, Patrick K, Cella JA, Buchta R. Behavioral weight control for overweight adolescents initiated in primary care. *Obesity Research* 2002; 10(1):22-32.

6. Wadden TA, Stunkard AJ, Rich L, Rubin CJ, Sweidel G, McKinney S. Obesity in black adolescent girls: a controlled clinical trial of treatment by diet, behavior modification, and parental support 3928. *Pediatrics* 1990; 85(3):345-352.

7. White MA, Kohlmaier JR, Varnado-Sullivan P, Williamson DA. Racial/ethnic differences in weight concerns: protective and risk factors for the development of eating disorders and obesity among adolescent females. *Eating & Weight Disorders: EWD* 2003; 8(1):20-25.

8. Kang HS, Gutin B, Barbeau P, Owens S, Lemmon CR, Allison J et al. Physical training improves insulin resistance syndrome markers in obese adolescents. *Medicine & Science in Sports & Exercise* 2002; 34(12):1920-1927.

Outcomes Table Modeling Potentially Prevented Adult Cases of Disease by Screening 1,000 Adolescents and Treating Overweight Adolescents (≥ 95th Percentile)

Using established cutpoints for overweight (≥ 95th percentile) in adolescents, the group with the highest probability of developing adult overweight or obesity, we modeled the number needed to screen (NNS) to prevent one excess case of adult morbidity associated with overweight. We used nationally representative prevalence figures[1] for Mexican Americans and non-Hispanic blacks and whites, and a large population-based study for Native Americans.[2] We assumed screening 1,000 adolescents of the same sex and race in a clinic setting would yield these proportions. To model the risk of health conditions, we used sex-specific disease prevalence estimates attributable to severity of overweight and obesity from cross-sectional nationally representative data (NHANES III).[3] For the prevalence of disease associated with adult obesity, we chose the lowest estimates (associated with Obesity Class I (BMI) rather than Classes II or III) to avoid overestimating the excess cases associated with adult obesity. Based on the assumptions used to produce this outcomes table, we assumed that weight-related disease estimates applied equally across racial and ethnic minorities. We also assumed that the probability of adult disease in those initially overweight but achieving and maintaining a non-overweight BMI would equal that of adolescents at the non-overweight BMI initially. In the absence of adequate treatment data, we assumed that a low (0.10) or moderate (0.30) proportion of adolescents would respond to treatment by reducing and maintaining their BMI at normal adult levels (18.5 to 24.9).

Considering all cases of disease as occurring in different people and as of equal morbidity importance, there was a low of 18 excess cases of disease (diabetes mellitus II, gallbladder disease, coronary heart disease, osteoarthritis) due to adolescent overweight in adult non-Hispanic white males and a high of 58 excess cases in adult non-Hispanic black females. Similarly, there was a low of 38 excess cases with increased cardiovascular (CV) risk factors due to adult obesity or overweight in non-Hispanic white males and a high of 87 in non-Hispanic black females. The NNS varies with the presumed efficacy of treatment. If all patients enroll in treatment, and treatment is effective in 30% of participants, the NNS to prevent one adult case of disease ranges from a low of 59 in non-Hispanic black females to a high of 200 in non-Hispanic white males. If treatment is 10% effective, the NNS to prevent one case of adult disease ranges from a low of 200 in non-Hispanic black females to a high of 1,000 in non-Hispanic white males. No groups have an NNS of 100 or below for a treatment that returns 10% of overweight adolescent patients to normal weight. For a treatment that returns 30% of overweight adolescent patients to normal weight, four groups have an NNS of 100 or below: non-Hispanic black females (59), Mexican American females (77), Mexican American males (91), and Native American males (100). NNSs are more favorable when considering prevented CV risk factors or when combining diseases and risk factors. The number needed to treat (NNT) for both estimated levels of treatment efficacy are also reported.

Appendix Q. Outcomes Table Modeling Potentially Prevented Adult Cases of Disease by Screening 1,000 Adolescents and Treating Overweight Adolescents (≥ 95th Percentile) (continued)

We examined the proportion of the total disease or risk factor burden potentially prevented by screening and treating overweight adolescents by dividing the excess cases due to overweight that would be prevented with a low (0.10) or moderate (0.30) treatment regimen by the total burden of adult disease in all weight categories. One to five percent of the overall disease burden could be prevented through this approach, with similar proportions of the overall risk factor burden.

Using an outcomes table approach, we modeled the impact on adult weight-associated morbidities of identification and treatment of overweight adolescents based on BMI measurements (with 100% accuracy). For the treatment effect that seems most reasonable given the limited existing evidence (0.10 long-term treatment benefit), NNS is greater than 100 for all subgroups of adolescents. If a more robust sustained treatment effect could be sustained (0.30), then non-Hispanic black females, Mexican American males and females, and Native American males would all have an NNS of 100 or less. However, looked at a different way, the proportion of the overall disease prevented would be quite small (1%-7%), probably reflecting the fact that despite relatively strong tracking of adolescent overweight to adult obesity, most adults (75%) who are obese were not overweight as children.[4]

Appendix Q. Outcomes Table Modeling Potentially Prevented Adult Cases of Disease by Screening 1,000 Adolescents and Treating Overweight Adolescents (≥ 95th Percentile) (continued)

Table Q-1. Outcomes Table Modeling Potentially Prevented Adult Cases of Disease by Screening 1,000 Adolescents and Treating Overweight Adolescents (≥ 95th Percentile)

		Mexican American				Non-Hispanic Black				Native American				Non-Hispanic White			
		Males		Females		Males		Females		Males		Females		Males		Females	
Age		12 thru 19				12 thru 19				17				12 thru 19			
Prev/1000 ≥95th percentile[1, 2]		275		194		207		266		259		143		128		124	
Adult BMI		25-29.9	≥30	25-29.9	≥30	25-29.9	≥30	25-29.9	≥30	25-29.9	≥30	25-29.9	≥30	25-29.9	>30	25-29.9	≥30
Prob Adult Over-weight or Obesity[5]		0.2	0.8	0.3	0.7	0.2	0.8	0.3	0.7	0.2	0.8	0.3	0.7	0.2	0.77	0.3	0.7
Excess Adult Cases Due to Overweight or Obesity[3]	**DMII**	19		9		14		12		18		7		9		6	
	GBD	8		16		6		22		8		12		4		10	
	CHD	3		9		3		13		3		7		2		6	
	OA	6		8		4		11		5		6		3		5	
	TC >240mg/dl	21		22		16		31		20		16		10		14	
	BP>140/90 mmHg	60		41		45		56		57		30		28		26	
		0.30	**0.10**	**0.30**	**0.10**	**0.30**	**0.10**	**0.30**	**0.10**	**0.30**	**0.10**	**0.30**	**0.10**	**0.30**	**0.10**	**0.30**	**0.10**
Prevented excess cases of disease. (Treatment efficacy of 0.30 and 0.10.)	**DMII**	6	2	3	1	4	1	4	1	5	2	2	1	3	1	2	1
	GBD	2	1	5	2	2	1	6	2	2	1	3	1	1	0	3	1
	CHD	1	0	3	1	1	0	4	1	1	0	2	1	0	0	2	1
	OA	2	1	2	1	1	0	3	1	2	1	2	1	1	0	2	1
	total diseases	**11**	**4**	**13**	**5**	**8**	**2**	**17**	**5**	**10**	**4**	**9**	**4**	**5**	**1**	**9**	**4**
	TC >240mg/dl	6	2	7	2	5	2	9	3	6	2	5	2	3	1	4	1
	BP>140/90 mmHg	18	6	12	4	14	5	17	6	17	6	9	3	8	3	8	3
	total risk factors	**24**	**8**	**19**	**6**	**19**	**7**	**26**	**9**	**23**	**8**	**14**	**5**	**11**	**4**	**12**	**4**
Total burden of adult disease per 1000[3]	**DMII**	54		69		50		74		44		57		51		57	
	GBD	29		123		34		127		30		108		34		105	
	CHD	106		97		104		95		100		93		104		91	
	OA	41		85		39		88		37		91		40		76	
	total diseases	**230**		**374**		**227**		**384**		**211**		**349**		**229**		**329**	
	TC >240mg/dl	333		375		319		372		316		350		327		344	
	BP>140/90 mmHg	352		386		339		394		317		348		342		340	
	total risk factors	**685**		**761**		**658**		**766**		**633**		**698**		**669**		**684**	
		0.30	**0.10**	**0.30**	**0.10**	**0.30**	**0.10**	**0.30**	**0.10**	**0.30**	**0.10**	**0.30**	**0.10**	**0.30**	**0.10**	**0.30**	**0.10**
Proportion prevented	**diseases**	0.05	0.02	0.03	0.01	0.04	0.01	0.04	0.01	0.05	0.02	0.03	0.01	0.02	0.00	0.03	0.01
	risk factors	0.04	0.01	0.02	0.01	0.03	0.01	0.03	0.01	0.04	0.01	0.02	0.01	0.02	0.01	0.02	0.01
NNS	**diseases**	91	250	77	200	125	500	59	200	100	250	111	250	200	1000	111	250

Appendix Q. Outcomes Table Modeling Potentially Prevented Adult Cases of Disease by Screening 1,000 Adolescents and Treating Overweight Adolescents (≥ 95th Percentile) (continued)

	risk factors	42	125	53	167	53	143	38	111	43	125	71	200	91	250	83	250
NNT	**diseases**	25	69	15	39	26	104	16	53	26	65	16	36	26	128	14	31
	risk factors	11	34	10	32	11	30	10	30	11	32	10	29	12	32	10	31

References

1. Ogden CL, Flegal KM, Carroll MD, Johnson CL. Prevalence and trends in overweight among US children and adolescents, 1999-2000. *JAMA* 2002; 288(14Oct 9):1728-1732.

2. Zephier E, Himes JH, Story M. Prevalence of overweight and obesity in American Indian School children and adolescents in the Aberdeen area: a population study. *Int J Obes Relat Metab Disord* 1999; 23 Suppl 2:S28-30.:S28-S30.

3. Must A, Spadano J, Coakley EH, Field AE, Colditz G, Dietz WH. The disease burden associated with overweight and obesity. *JAMA* 1999; 282(16):1523-1529.

4. Freedman DS, Khan LK, Dietz WH, Srinivasan SR, Berenson GS. Relationship of childhood obesity to coronary heart disease risk factors in adulthood: the Bogalusa Heart Study. *Pediatrics* 2001; 108(3):712-718.

5. Guo SS, Wu W, Chumlea WC, Roche AF. Predicting overweight and obesity in adulthood from body mass index values in childhood and adolescence. *American Journal of Clinical Nutrition* 2002; 76(3):653-658.

Population-Based Obesity Prevalence Surveys

Survey	Dates	Age/years	Race/Ethnicity	Measures
NHES I	1960-62	18-79	Predominately white	Unable to locate
NHES II	1963-65	6-11	14% black	wt=lbs; ht=cm Skinfold (3)
NHES III	1966-70	12-17	10% black	wt=lbs; ht=cm Skinfold (4)
NHANES I	1971-74	1-74	*Race recorded as b, w, other	wt=lbs; ht=cm & in Skinfold (2)
****NHANES II**	1976-80	6 mos-74	*Race recorded as b, w, other	wt=lbs; ht=cm & in Skinfold (2)
HHANES	1982-84	6 mos-74	Predominately Hispanic from SW USA	wt=lbs; ht=cm & in
NHANES III	1988-94	2 mos +	***Over sampled MA, NHB	wt = kg, ht=cm Skinfold (4)
NHANES	1999-2000	Birth +	***Over sampled preschool age, MA, NHB	BIA, DEXA, ht, wt, Skinfold (1)

* Observed race recorded.
** Self-reported Hispanic origin.
*** Race self-reported as NHW, NHB, MA, Other.

References

1. *Plan and Operation of the Second National Health and Nutrition Examination Survey, 1976-1980.* Series 1 ed. Hyattsville: U.S. Department of Health and Human Services Public Health Service, Office of Health Research, Statistics, and Technology, National Center for Health Statistics, 1981.

2. *Plan and Operation of a Health Examination Survey of U.S. Youths 12-17 Years of Age*. Series 1 ed. Rockville: U.S. Department of Health, Education, and Welfare, Public Health Service, National Center for Health Statistics, 1974.

3. *Plan and Operation of the Health and Nutrition Examination Survey, United States 1971-1973.* Series 1 ed. Hyattsville: U.S. Department of Health, Education, and Welfare, Public Health Service, Office of Health Research, Statistics, and Technology, National Center for Health Statistics, 1973.

4. *Plan, Operation, and Response Results of a Program of Children's Examinations*. Series 1 ed. Washington, D.C.: U.S. Department of Health, Education, and Welfare, Public Health Service, National Center for Health Statistics, 1967.

5. *Plan and Initial Program of the Health Examination Survey*. Series 1 ed. Washington, D.C.: U.S. Department of Health, Education, and Welfare, Public Health Service, National Center for Health Statistics, 1965.

6. Flegal KM, Carroll MD, Kuczmarski RJ, Johnson CL. Overweight and obesity in the United States: prevalence and trends, 1960-1994. *Int J Obes Relat Metab Disord* 1998; 22(1):39-47.

7. Troiano RP, Flegal KM, Kuczmarski RJ, Campbell SM, Johnson CL. Overweight prevalence and trends for children and adolescents. The National Health and Nutrition Examination Surveys, 1963 to 1991. *Archives of Pediatrics & Adolescent Medicine* 1995; 149(10):1085-1091.

8. Killeen J, Vanderburg D, Harlan WR. Application of weight-height ratios and body indices to juvenile populations--the National Health Examination Survey Data. *J Chronic Dis* 1978; 31(8):529-537.

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