

# Original Reports: Weight Control

---

## OVERWEIGHT AS A RISK FACTOR IN CHILDREN: A FOCUS ON ETHNICITY

The prevalence of overweight in youth is increasing dramatically in the United States. The intimate relationship of obesity and overweight with cardiovascular risk factors and diabetes in adults raises concern for the likelihood of subsequent disease development in children. Ethnic minorities are so disproportionately affected by overweight that a call to action is necessary. The International Society on Hypertension in Blacks convened this work group as part of a larger effort to focus on cardiovascular risk protection beginning in childhood and adolescence, entitled the "Children are Our Messengers: Changing the Health Message" initiative. This summary article reviews the data on cardiovascular risk factors and overweight in ethnic children and adolescents, and culminates in a practical algorithm for evaluating overweight children for cardiovascular risk. (*Ethn Dis.* 2003;14:94–110)

**Key Words:** Obesity, Overweight, Children, Adolescents, African American/Blacks, Hispanics, Ethnicity, Cardiovascular Disease, Hypertension, Diabetes, Insulin Resistance, Dyslipidemia

---

From the University of Texas Southwestern Medical Center at Dallas, Dallas (SDN), University of Texas Medical School, Houston Pediatric Adolescent Hypertension Program, Houston (JMS), Texas; Centers for Disease Control and Prevention, Emerging Investigations and Analytic Methods Branch (MOA), The Children's Hospital of Philadelphia, Division of Gastroenterology and Nutrition (NS); University of Southern California Institute for Prevention Research (MIG); Johns Hopkins University, Department of Pediatrics and Medicine (RP); and Thomas Jefferson University, Department of Medicine (BEF).

Address correspondence and reprint requests to Shawna D. Nesbitt, MD, MS; University of Texas Southwestern Medical Center at Dallas; 5323 Harry Hines Blvd. CS8.102A; Dallas, TX 75390-8899; 214-648-2103; 214-648-7979 (fax); shawna.nesbitt@utsouthwestern.edu

Shawna D. Nesbitt, MD, MS; Modele O. Ashaye, MD, MPH; Nicolas Stettler, MD, MSCE; Jonathan M. Sorof, MD; Michael I. Goran, PhD; Rulan Parekh, MD, MS; Bonita E. Falkner, MD

### OBJECTIVE

During the past 2 decades, the prevalence of overweight has increased dramatically in the US adult population.<sup>1</sup> With that increase, there has been a concurrent increase in the prevalence of diabetes, and other well-known cardiovascular risk factors.<sup>2</sup> Subsequent to these trends in the adult population, similar trends of overweight are manifesting in US children and adolescents.<sup>3</sup> The rates of type 2 diabetes and insulin resistance are also increasing in the pediatric population.<sup>4</sup> Type 2 diabetes and cardiovascular disease are typically thought of as health problems affecting those of middle or older ages. This assumption is based on the appearance of risk factors in mid-to-late adult life. The current trends of increasing risk at earlier ages may signify an emerging trend toward earlier manifestation of clinical cardiovascular disease.

Overweight and obesity have direct links to adverse health outcomes; however, the more potent relationship may be their indirect association with a litany of other cardiovascular risk factors. While attention is focused on these risk factors, it is of paramount importance to address overweight as a pervasive and centralizing theme. The approaches to weight loss in adults often have limited success, because lifestyle patterns are set early in life. Perhaps a more effective strategy is to concentrate on overweight treatment and risk-factor assessment in childhood, when lifestyle habits are being formed.

Like other disease patterns, trends of

overweight differ according to ethnicity and gender. However, the approach to treating overweight individuals has not been targeted accordingly. In this article, we review the literature on obesity and overweight in children and adolescents, as related to ethnicity, risk factors, and clinical disease; determinants of overweight; and clinical trials. Ethnic classifications are derived from region of descent, cultural origin, and cultural identification. In this paper, we have not sought to resolve the issue of clearly defining ethnicity, but to present current data published according to these classifications. Finally, this article reviews the optimal approach to treating an overweight child, and risk factor assessment.

### PARTICIPANTS

The authors of this summary article are members of the "Children Are Our Messengers: Changing the Health Message" initiative sponsored and selected by the International Society on Hypertension in Blacks (ISHIB). The membership of this initiative was convened in October 2001, and the writing group first met in April 2002. Members of this group are from multi-specialty backgrounds, and include health service researchers, internists, and pediatricians, who share an interest in cardiovascular risk-factor assessment, and prevention of cardiovascular disease.

## EVIDENCE

The work group identified a list of important issues relevant to the development of cardiovascular disease in obese children. The paper was divided into sections addressing key components of the subject of overweight ethnic children in the United States, including epidemiology, risk factors for obesity, hypertension, dyslipidemia, cardiovascular disease, diabetes, effectiveness of weight loss, and evidence-based treatment recommendations. The review focused on overweight or obese ethnic children in the United States, and the association of their condition with cardiovascular risk factors. A systematic review was not possible, due to the limited number of studies of ethnic children in the United States. The data review was conducted as follows: A Medline/PubMed search (from 1966–2002) was performed on each focus area, using key words such as “overweight children,” “obesity,” and “ethnicity or race,” and reviewed by an assigned member of the work group. The papers collected were then summarized and presented to the work group. The data on cardiovascular risk factors in hypertension, dyslipidemia, and diabetes, were compiled into risk-factor epidemiology, pathophysiology, and treatment. These sections were compiled and reviewed by the entire work group. The drafted document was then read by 2 external reviewers with expertise in the focus areas of the manuscript, and returned for revision. The final draft was submitted to the ISHIB board of directors.

### FOCUS AREA 1: EPIDEMIOLOGY OF OVERWEIGHT AND OBESITY IN CHILDREN AND ADOLESCENTS

#### Definitions of Overweight and Obesity

Obesity is defined as excess adipose tissue or body fat, in relation to lean

---

*The current trends of increasing risk at earlier ages may signify an emerging trend toward earlier manifestation of clinical cardiovascular disease.*

---

body mass, while overweight refers to increased body weight compared to height. Body mass index (BMI), a measure of body weight related to height (calculated as weight in kilograms divided by height in meters squared [ $\text{kg}/\text{m}^2$ ]), is gender- and age-specific for children and adolescents, but not for adults. Body mass index (BMI) growth charts developed by the Centers for Disease Control and Prevention (CDC) were derived using National Health and Nutrition Examination Surveys (NHANES) data, and serve as guidelines for assessing BMI cut points expressed as percentiles.<sup>5</sup> Children and adolescents with BMI values at, or above, the 95th percentile of the sex-specific BMI growth charts are classified as overweight, while those with BMI between the 85th and 95th percentile are considered to be at risk for becoming overweight.<sup>6</sup> Because obesity is defined as excessive adiposity, this term should only be used in children when the high BMI is confirmed by assessment of adiposity (see <http://www.cdc.gov/growthcharts> for the CDC growth charts). The standards for these growth charts are based on large data sets collected in children during the 1960s and 1970s. Several of the studies referred to in this manuscript actually assessed obesity, as they measured adiposity, in addition to weight and height. Therefore, it is appropriate to use “overweight” in some parts of the manuscript and “obese” elsewhere.

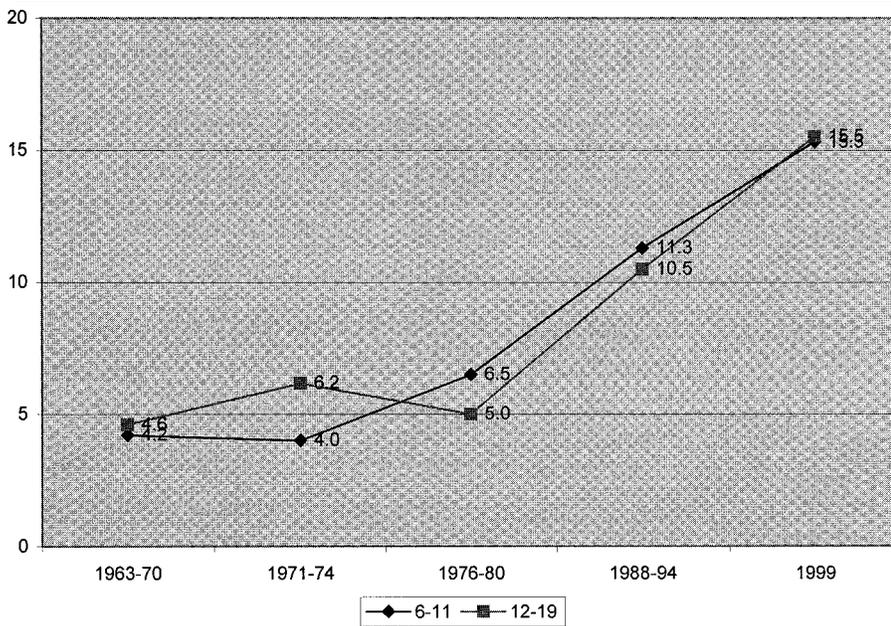
#### Prevalence

The epidemic of childhood obesity is an issue of global importance. Worldwide, approximately 22 million children under 5 years of age are overweight.<sup>7</sup> In the United States, overweight and obesity have been recognized as leading health indicators in *Healthy People 2010*, a national health promotion and disease prevention initiative seeking to reduce the prevalence of overweight and obesity among children and adolescents to  $\leq 5\%$ .<sup>8</sup> The 1999–2000 NHANES data indicate that the prevalence of overweight is now 15.3% and 15.5% in children and adolescents, respectively (Figure 1). In addition, the 2001 Youth Risk Behavior Surveillance System (YRBSS), a self-reported, school-based survey conducted by the CDC to track health-risk behaviors and prevalence trends among youth and young adults, found that 10.5% of US high school students reported being overweight, and 13.6% are at risk for overweight.<sup>10</sup>

#### Trends and Disparities

Trends in the United States over the past 4 decades indicate a worsening picture. The prevalence of pediatric overweight has increased 3-fold since the 1960s, and doubled since the early 1970s. From 1963 to 2000, the prevalence of overweight rose from 4% to 15.3% in children [aged 6–11 years], and from 5% to 15.5% in adolescents [aged 12–19 years], respectively (Figure 1). This increase in overweight prevalence extends across racial and ethnic groups; however, there are some differences by gender and ethnicity. At younger ages, boys have a higher prevalence of overweight than girls (16% vs 14.5%, respectively), while adolescent boys and girls have similar prevalence rates of overweight (15.5%).<sup>9</sup>

In the YRBSS, male students (14.2%) were significantly more likely than female students (6.9%) to report being overweight. Across grades 9 to 12, prevalence among all students ranged from 9.6% to 10.8%, with adolescents



**Fig 1. Trends in the prevalence of overweight among children and adolescents, United States, 1963–2000**

Source: Centers for Disease Control and Prevention (CDC), National Center for Health Statistics (NCHS); National Health Examination Survey (NHES II/III [1963–70]), National Health and Nutritional Examination Surveys NHANES I [1971–74]; NHANES II [1976–80]; NHANES III [1988–94]; NHANES (1999–2000)

exhibiting the highest prevalence in tenth grade, and the lowest in twelfth grade. This survey is useful in monitoring trends, yet the accuracy of self-reported data suffers due to under-reporting.<sup>11,12</sup> In ethnic comparisons from 1999–2000 NHANES data, among boys, Mexican Americans had the highest rates of overweight (27.3% at ages 6–11 years, and 27.5% at ages 12–19 years). Among girls, non-Hispanic Blacks have the highest rates of overweight (22.2% at ages 6–11 years, and 26.6% at ages 12–19 years)<sup>2</sup> (Figure 2). Strauss and Pollack, using data from the National Longitudinal Survey of Youth (NLSY), have documented racial and ethnic differences, with the greatest increase in overweight found among African Americans and Hispanics.<sup>2</sup> Although there is no nationally representative study of obesity in Native American children to date, this ethnic group also appears to have an increased risk for

obesity.<sup>13</sup> Data are still sparse on Asian-American, Pacific Islander, or Alaska Native children. Also not well researched are prevalence rates of obesity among children of recent immigrants to the United States from Latino countries (other than Mexico), or from Caribbean, Asian, European, Middle-Eastern, or African countries.

The high prevalence rates of obesity in children and adolescents of minority populations underscore the need for timely intervention in this population. In developing approaches to this public health issue, it is critical to identify those at greatest risk for obesity.

## FOCUS AREA 2: RISK FACTORS FOR OBESITY IN CHILDHOOD

### Energy Balance

In children, obesity and overweight results from failure to balance energy in-

take with energy expenditure.<sup>14</sup> The main component of energy expenditure is resting energy expenditure, which is closely associated with body composition and size. Therefore, much inter-individual variability in metabolism and resting energy expenditure is probably explained by differences in body composition.<sup>14–16</sup> The component of energy expenditure exhibiting the greatest variability is physical activity energy.<sup>14</sup> For example, compared to watching television, energy expenditure is about 15% higher for doing school work, 20%–30% higher for creating arts and crafts projects, and 150% higher for engaging in mild-to-moderate physical activity.<sup>17</sup> Therefore, the time spent in these various activities is an important determinant of the overall energy balance. For instance, a 4-year-old child who increased his energy intake by ~30 kcal (~2.5 oz of soda), or who spent ~15 minutes more per day watching television, rather than playing, could potentially develop obesity, if this trend continued for 1 to 2 years.<sup>14</sup> While comprehensive reviews of factors associated with energy imbalance have been published<sup>18</sup>; the focus in this report is on US minority children.

### Unusual Underlying Conditions

In the United States, most cases of childhood obesity are primary or exogenous. A small percentage of cases are due to co-existing medical conditions, such as hypothyroidism, Cushing's syndrome, Down's syndrome, Turner's syndrome, Prader-Willi syndrome, excessive tube feeding, cancer survival, or steroid therapy. Most underlying conditions are easily recognizable, and obese children who do not exhibit short stature, developmental delay, dysmorphic signs, or abnormal genitalia, are unlikely to have an underlying medical condition. Energy balance is regulated by leptin, ghrelin, neuropeptides, and other hormones.<sup>19</sup> Only a few cases of leptin insufficiency have been documented, but

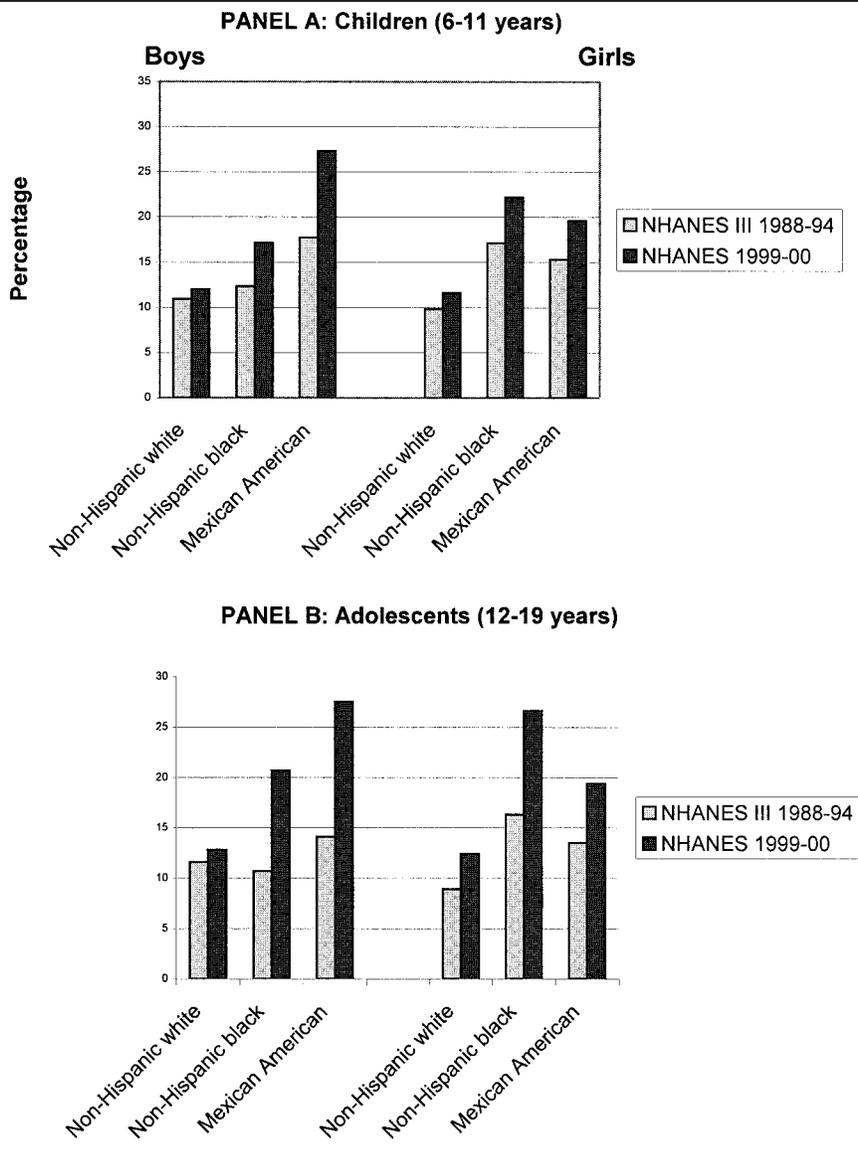


Fig 2. Overweight prevalence by race/ethnicity for children (6–11 years) and adolescents (12–19 years)

Source: Ogden CL, Flegal KM, Carroll MD, Johnson CL. Prevalence and Trends in Overweight Among US Children and Adolescents, 1999–2000. *JAMA*. Oct 9 2002; 288(14): 1728–32

leptin resistance may provide a partial explanation for individual predisposition to obesity. In adults, some evidence suggests that leptin levels in non-Hispanic Blacks and Mexican Americans are higher than in Whites; however, after adjusting for body weight, these differences are minimized in children.<sup>20,21</sup>

### Constitutional and Non-Modifiable Factors

Because families usually share environmental and genetic backgrounds, the most compelling evidence for a genetic predisposition to obesity is derived from studies of twins,<sup>22–25</sup> and subjects adopted at birth.<sup>26,27</sup> Although such evidence

is scarce in minorities, many studies in diverse populations have shown a familial clustering of obesity<sup>28–31</sup>; however, genetic and environmental factors cannot be separated in these studies. Gender differences in the prevalence of overweight status vary among US ethnic groups (Figure 2),<sup>32</sup> suggesting a cultural, or perhaps ethnic, variation in gene expression. Studies of both Black and White children confirm an increased risk of obesity and overweight in children from small families,<sup>30,31,33–36</sup> and in first-born children.<sup>30,33</sup> An increased maternal age has been associated with an increased risk for obesity in Black and White girls.<sup>36</sup> The prevalence rates of overweight vary among racial groups of US children (Figure 2); however, whether these differences are racial, genetic, and biological, or ethnic, environmental, and cultural, remains controversial.<sup>14,37–39</sup>

### Socioeconomic and Environmental Factors

In high-income countries, children from lower-income families are at increased risk for obesity and overweight, while in low-income countries, the opposite is true.<sup>40</sup> In the United States, this association varies by sex, age, and ethnic group. The prevalence of obesity is higher in US non-Hispanic White children from low-income families,<sup>31,32,41</sup> but higher in middle- or upper-class families of Mexican-American, or non-Hispanic Black, children,<sup>32</sup> as is the case in low-income countries.<sup>40</sup> Lower parental education has been associated with obesity in non-Hispanic White and Black boys,<sup>32</sup> and in non-Hispanic White, but not Black, girls.<sup>41</sup> In young children, maternal feeding method and control have been associated with obesity in Black and White children.<sup>42,43</sup>

Environmental influences on obesity and overweight may be present early in life. Maternal diabetes during pregnancy has been associated with the development of obesity in Pima Indian children.<sup>44</sup> Observational studies suggest

that breastfeeding may decrease the risk for obesity.<sup>45-47</sup> An association of total caloric and fat intake with obesity has been documented in later childhood, at ages 9 to 10 years<sup>41,48</sup>; however, many studies failed to show an association in children aged 4 to 7 years, possibly due to the difficulties in assessing dietary intake in younger children. The consumption of sugar-containing beverages,<sup>49,50</sup> large portion sizes,<sup>49,51</sup> and meal structure and patterns have also been associated with overweight in US children.<sup>34,35,52</sup>

Several studies demonstrate the association between television viewing and excessive weight gain in childhood,<sup>31,53,54,55</sup> including randomized controlled treatment and prevention trials.<sup>52,53</sup> These studies were conducted in mostly European-American children, however, and may not generalize to minority children. An inverse association of physical activity with body mass index was demonstrated in studies of Native American and African-American children,<sup>56,57</sup> but successful randomized trials are limited.<sup>58</sup> Goran<sup>14</sup> has shown in longitudinal studies that a low level of fitness (as measured by  $VO_2$  max), but not energy expenditure, predicts greater likelihood of developing obesity, in both African-American and White children. In recent decades, opportunities for physical activity have decreased, in conjunction with an increase in automobile use,<sup>59</sup> and a decrease in school physical education.<sup>49</sup> For unknown reasons, fitness levels, as measured by  $VO_2$  max, are lower among African Americans, compared to Whites.<sup>14</sup> Unpublished data in Hispanics show results similar to those for African Americans (personal communication, Goran). Additionally, children's eating and drinking opportunities have increased, with greater numbers of vending machines being located in schools. Many schools exclusively contract with soft-drink companies, and an increasing number of fast food restaurants,<sup>49</sup> leading to what some have called a "toxic environment."<sup>60</sup>

### Growth Patterns and Critical Periods

Using a life-course approach to chronic disease,<sup>61</sup> sensitive or critical periods in obesity development have been investigated as important periods for prevention.<sup>62</sup> Fetal life is recognized as one of these critical periods.<sup>63-65</sup> Rapid infancy weight gain has also been associated with childhood obesity, particularly in African Americans,<sup>66-69</sup> while early malnutrition and stunting are associated with an increased risk for obesity in low-income countries.<sup>70,71</sup> The period of adiposity rebound, between ages 3 and 6 years, also appears to be a critical for obesity development,<sup>72-73</sup> as is adolescence.<sup>72</sup> Most studies, however, did not include a large number of minority children.

Among overweight children, those who have additional cardiovascular risk factors are at greatest risk for developing cardiovascular disease. It is critical to understand the relationship of overweight in children to known cardiovascular risk factors.

### FOCUS AREA 3: OBESITY-RELATED HYPERTENSION IN CHILDREN

#### Epidemiology

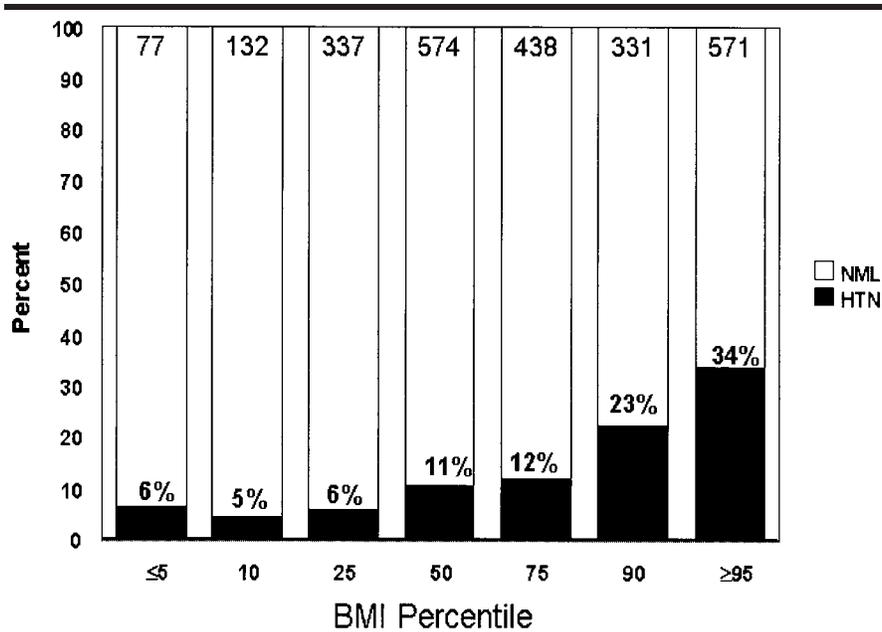
Numerous studies in a variety of ethnic and racial groups have reported an association between obesity and hypertension in children, and virtually all studies find higher blood pressure levels, or higher prevalence of hypertension, in obese, compared to lean, children.<sup>74-84</sup> Rosner et al pooled data from 8 large US epidemiological studies, involving more than 47,000 children, to describe blood pressure differences between Black and White children in relation to body size.<sup>85</sup> Regardless of ethnicity, gender, or age range, children in the upper decile of BMI had an odds ratio of systolic hypertension ranging from 2.5 to 3.7. Similarly, Sorof et al found that

obese adolescents exhibited an approximately 3 times greater prevalence of hypertension, compared to non-obese adolescents, in a school-based screening study of predominantly ethnic minorities.<sup>84</sup> Further analysis by Rosner et al revealed that among lean children, the risk of hypertension was greater in Blacks, whereas among obese children, hypertension risk was greater in Whites.<sup>85</sup> These data suggest that hypertension in Black children, although exacerbated by obesity, is likely mediated by mechanisms that are, to some extent, obesity-independent.

The risk of hypertension in children increases across the entire range of BMI values, and is not defined by a simple threshold effect. Rosner et al reported a progressive increase in the prevalence of diastolic hypertension in children of all race, gender, and age combinations, as BMI increased across the normal range.<sup>85</sup> Sorof et al found an increased prevalence of systolic hypertension as relative BMI increased from the 5th to the 95th percentile (Figure 3).<sup>84</sup> Although this latter study showed a higher prevalence of hypertension in Black, compared to White, adolescents, the difference in prevalence was not significant, after controlling for the higher BMI in the Black adolescents.

#### Pathophysiology

Most studies in children have focused on the investigation of 3 main pathophysiological mechanisms for obesity-related hypertension: over-activity of the sympathetic nervous system (SNS), insulin resistance, and abnormalities in vascular structure and function. Evidence for SNS over-activity derives from studies demonstrating correlations between heart rate and subscapular skinfold thickness,<sup>86</sup> and between hyperdynamic cardiovascular states and several measures of obesity.<sup>87</sup> Obese hypertensive children also have higher heart rates,<sup>84</sup> increased heart-rate variability,<sup>88</sup> and increased 24-hour blood pressure variability,<sup>84</sup> compared to lean



**Fig 3. Distribution of BMI percentiles and the prevalence of hypertension within each BMI percentile category. Values above bars indicate number of children within each BMI category.**

NML=normotensive;  
HTN=hypertensive

Sorof JM, Poffenbarger T, Franco K, Bernard L, Portman RJ: Isolated systolic hypertension, obesity, and hyperkinetic hemodynamic states in children. *J Pediatr* 2002;140:660-666

hypertensive children. Evidence for insulin resistance comes from reported positive associations between fasting insulin levels and resting blood pressure in obese children and young adults.<sup>89-94</sup> In addition, a direct relationship between insulin and the sodium sensitivity of blood pressure has been demonstrated in obese adolescents.<sup>95</sup> Evidence of altered vascular structure and function comes from reports of lower arterial compliance, lower distensibility function, and lower endothelium-dependent and -independent function in severely obese, compared to control, children.<sup>96</sup> Further, obese adolescents are reported to have decreased maximal forearm blood flow, and increased minimal forearm vascular resistance, compared to healthy controls.<sup>97</sup> Other factors implicated in the pathogenesis of obesity-related hypertension include structural changes in the kidney due to encapsu-

lation by fatty tissue,<sup>98</sup> alteration of the renin-angiotensin-aldosterone system,<sup>99</sup> and alterations of the hypothalamic-pituitary-adrenal axis.<sup>100</sup> Although racial predispositions to risk factors for hypertension, such as salt sensitivity, are likely to be important, no studies to date have systematically investigated the influence of ethnicity on the putative mechanisms of obesity-related hypertension in children. Further studies in children are needed to determine whether racial factors moderate the pathophysiology of obesity-related hypertension.

### Treatment

The relationship between weight loss and blood-pressure reduction in children is demonstrated in several interventional studies.<sup>101-104</sup> The only controlled trial was performed by Rocchini et al, who randomized overweight adolescents to 3 interventions over a 20-

week period: diet alone, diet plus exercise, and control (no intervention).<sup>105</sup> Changes in systolic blood pressure from baseline in the diet plus exercise group, diet alone group, and control group, were -16 mm Hg, -10 mm Hg, and +4 mm Hg, respectively. This latter study provides the most definitive evidence that weight loss, particularly in conjunction with exercise, is beneficial in treating obesity-related hypertension in children. The mechanisms by which weight loss results in blood-pressure reduction have been investigated. Both heart rate<sup>106</sup> and fasting insulin levels<sup>103,106</sup> are lower after weight loss occurs in obese adolescents. Weight loss in obese adolescents also renders previously salt-sensitive individuals insensitive to the hypertensive effects of salt-loading,<sup>95</sup> and reverses impaired post-ischemic maximal forearm blood flow.<sup>105</sup> Therefore, the beneficial effects of weight loss on blood pressure in obese adolescents appear to be mediated through a combination of neurohumoral, metabolic, and vascular changes. It is important to note that each of these studies was conducted either exclusively in White children, or provided no description of the ethnic distribution of study subjects. To date, no studies have focused specifically on racial or ethnic differences in the effect of weight loss on blood-pressure reduction in children.

## FOCUS AREA 4: DYSLIPIDEMIA AND CARDIOVASCULAR DISEASE IN OVERWEIGHT AND OBESE CHILDREN

### Epidemiology

Dyslipidemias have been described in obese adults, and there is a linear relationship between increasing BMI and increases in levels of total cholesterol, triglycerides, and low-density lipoproteins (LDL), and decreases in levels of high-density lipoproteins (HDL), in both women and men.<sup>107</sup> Abnormal lip-

id levels, and the frequently associated insulin resistance syndrome, are related to adverse atherosclerotic disease in the obese adult population. In children, however, lipids are required for growth and maturation. Lipid levels change with age and puberty, and differ by gender.<sup>108</sup>

In cross-sectional data, dyslipidemia is prevalent in obese children, but is most striking in the ethnic minorities, including Blacks and Latinos. Data from 11,389 school-aged children showed that among Whites and Latinos, mean cholesterol and odds of hypercholesterolemia increase as BMI increases, especially in children with BMI greater than the 95%.<sup>109</sup> Although young Mexican Americans have levels of total cholesterol and LDL similar to those of the general population, consistently higher levels of triglycerides are found in males, while lower levels of HDL are found in females.<sup>110</sup> All obese children have higher odds of developing dyslipidemia.<sup>111</sup> The association of dyslipidemia and obesity is most striking in Black boys and girls, where obese children and adolescents had higher levels of total cholesterol, triglycerides, and LDL, and lower levels of HDL, compared to leaner children. Obese Black boys were 9 times more likely than non-obese Black boys to have hypertriglyceridemia >200 mg/dL, and 4 times more likely to have HDL <35 mg/dL.<sup>112</sup>

In the longitudinal Princeton School Study, BMI increased in both Black and White children, but was most prominent in Black females aged 7–12 years.<sup>113</sup> There was a parallel rise in total cholesterol, particularly among Blacks.<sup>113</sup> In the Child and Adolescent Trial for Cardiovascular Health (CATCH) study, overweight multi-ethnic children, as young as 8 years old, were monitored for 2.5 years.<sup>114</sup> Twenty-five percent of these children remained obese, while another 7% gained weight despite weight-loss interventions. These overweight children also had an increase in levels of total cholesterol and

apoprotein B, and a large decrease in HDL levels.<sup>114</sup>

Distribution of abdominal fat appears to be closely associated with dyslipidemias in adults, as defined by a waist-to-hip ratio (WHR). Central adiposity is associated with an increase in levels of LDL and triglycerides, in both Black and White girls aged 9–10 in the National Heart, Lung, and Blood Institute's (NHLBI) Growth and Health Study.<sup>115</sup> Black girls, however, are more likely than White girls to exhibit central adiposity.<sup>115</sup> Body fat distribution and age at obesity onset are strong determinants of obesity tracking into adulthood.<sup>116</sup> A cross-sectional analysis of NHANES data shows an association with WHR in pre-pubertal White and Mexican-American children. After adjustment for BMI and age, WHR is not important in determining apo A or apo B, and lipoprotein (a) >30 mg/d L in all children; however, WHR is important for determining levels of triglycerides for both White and Mexican-American children.<sup>117</sup>

### Cardiovascular Disease

Recent data from the Pathobiological Determinants of Atherosclerosis in Youth (PDAY) study provide compelling evidence in the general pediatric population that initial fatty streaks in adolescents may become atheromatous plaques in young adults.<sup>118</sup> Obesity in young Black and White men is associated with a 2-times greater prevalence of an initial atheromatous lesion in the coronary artery, and a 5-times greater prevalence of advanced coronary plaque, as compared to non-obese men. Left anterior descending artery stenosis also occurred in 2 times as many obese men, compared to non-obese males.<sup>119,120</sup> In addition, the long-term risk for the development of atherosclerotic disease in obese men is 2.3–13.2 times higher, than in those without obesity, and 0.4–0.8 times higher, in obese, compared to non-obese, women.<sup>121</sup> This appears to be independent of weight at later stages,

and may represent the weight gain of central body fat.

### Pathophysiology and Treatment

Adipose tissue is an active endocrine organ primarily used for storage of fatty acids. The release of free fatty acids is thought to be involved in the development of dyslipidemia and insulin resistance in children. Given the fatty acid requirements of children, the development of dyslipidemia in atherosclerosis in children must be clarified to properly prescribe treatment. In adults, exercise has been shown to be beneficial in modifying lipid levels. In children, one 7-year study of Black and White children showed a beneficial effect of dietary intervention with a low-fat diet to reduce lipid levels, but the impact of these changes on coronary disease has not been investigated.<sup>122</sup> Although exercise and diet may affect HDL, it is not clear that these treatments affect levels of total cholesterol and triglycerides in adolescents.<sup>123</sup> Drug therapy is currently reserved for children with severe dyslipidemias, and those at high risk for early CVD.

## FOCUS AREA 5: OVERWEIGHT AND OBESITY AND DIABETES IN CHILDREN

Type 2 diabetes has emerged as a critical health issue in overweight children, especially overweight African-American, Hispanic, and Native American adolescents.<sup>124,125</sup> More recently, pre-diabetes (previously termed impaired glucose tolerance) has emerged as a major concern in obese children and adolescents, with several studies demonstrating that 20%–30% of obese children have pre-diabetes.<sup>4,126</sup> These studies included children from various ethnic groups, and reported no differences across groups (although, in some cases, the sample sizes of ethnic groups were small).

Extensive work has elucidated the natural history of type 2 diabetes in adults. During the long period preceding the development of the disease, there is a progressive increase in fasting insulin and, subsequently, glucose.<sup>127</sup> At a critical moment, insulin fails to increase in proportion to glucose, and hyperglycemia ensues. While impaired glucose tolerance is regarded as a state of early risk for type 2 diabetes, recent studies have shown that the ability of the beta cell to compensate for insulin resistance may be just as important.<sup>128,129</sup> In youth, risk factors may be similar (increased body fat, decreased physical activity), but the time course is different, and type 2 diabetes in adolescence is further confounded by transient insulin resistance associated with puberty.

The most significant factor contributing to increased risk of type 2 diabetes in children is increased body fat, and possibly specific body fat deposits, such as visceral fat.<sup>130-135</sup> The strong relationship between body fat and insulin resistance has been shown to occur across different ethnic groups of children, including Caucasian, African-American, and Hispanic. Several studies show that African-American children are more insulin-resistant than are Caucasian children, independent of body composition and obesity status.<sup>14,130,134,136,137</sup> Lower insulin sensitivity in African-American children is associated with a higher-than-expected acute insulin response to glucose.<sup>134</sup> These higher insulin levels are partly attributable to increased secretion and a lower hepatic extraction.<sup>138</sup> Studies of obesity, insulin resistance, insulin secretion, and the beta-cell response in other ethnic groups of children are extremely limited. In one study comparing Caucasian, African-American, and Hispanic children in Los Angeles, Hispanic and African-American children were both, and to an equal degree, more insulin-resistant than were Caucasian children.<sup>139</sup> This difference in insulin resistance is independent of ad-

iposity. The compensatory response to the same degree of insulin resistance was different in Hispanic, compared to African-American children. African-American children compensated with a higher acute insulin response to glucose, an effect that may be partly due to a reduction in hepatic insulin extraction, which spares the need to increase insulin secretion. Hispanic children compensated with greater insulin secretion.

Puberty is another important factor affecting insulin resistance in children.<sup>140,141</sup> Pubertal development is associated with a 25%–30% reduction in insulin sensitivity, with the peak reduction occurring at Tanner stage III, followed by recovery by Tanner stage V.<sup>142</sup> Given the increased risk of developing type 2 diabetes among ethnic groups, and the role of puberty in this pathogenesis, it is important to know whether the influence of puberty on insulin resistance varies across ethnic groups; however, this has not yet been studied.

Several recent studies have suggested that low birth weight may contribute to greater insulin resistance among children in different age groups among Caucasians, Asians, Mexican-Americans, Pima Indians, and Black South Africans.<sup>143</sup> Only one study, however, has studied possible associations between low birth weight and greater insulin resistance across multiethnic groups,<sup>144</sup> finding that ethnicity and birth weight had a significant influence on fasting insulin among African Americans.

Previous studies in adults suggest a strong hereditary component in risk for type 2 diabetes.<sup>145-153</sup> Very few studies have looked at the relationship between family history and insulin resistance in children.<sup>126,154,155</sup> Collectively, the studies in children indicate that fasting glucose and insulin may not be affected by a positive family history of type 2 diabetes. Family history may affect the glucose and insulin response to an oral glucose load and insulin sensitivity, as assessed by the clamp technique; however, this was not found to be true for insulin

sensitivity when measured by the intravenous glucose tolerance test.

Several studies in children have examined treatment and prevention issues relating to obesity, insulin resistance, and type 2 diabetes, showing moderate, but inconsistent, improvements in insulin resistance.<sup>156-159</sup> Treatment studies have been limited to a study of metformin, with no specific studies comparing effects across ethnic groups. Three published studies of adults provide convincing evidence that type 2 diabetes can be prevented<sup>160-162</sup> with lifestyle intervention or drug treatment; however, there are no studies of children to support this notion. In addition, it is well known that exercise training can improve insulin resistance and reduce risk of type 2 diabetes in adults,<sup>163</sup> but remarkably few studies exist for children and ethnic minorities.

## FOCUS AREA 6: EFFECTIVENESS OF WEIGHT LOSS METHODOLOGY IN CHILDHOOD OVERWEIGHT AND OBESITY

Clinical trial data on weight loss in children have been published primarily based on small single-site trials. The focus of these trials has been on dietary, behavioral, physical activity, and multidisciplinary modalities, and on limited drug interventions. Few trials have included ethnic minorities, and even fewer have been designed to focus on the special needs of these groups.

### Dietary Trials

There are few dietary trials in children. One of the most frequently studied diets is the protein-sparing modified fast. This diet typically consists of low-calorie, high-protein meals containing lean meats, poultry, fish, and low-carbohydrate vegetables. Several small studies have shown significant weight losses

in overweight children. The dietary phase of these trials has lasted from 10 to 30 weeks. All participants in each study lost weight during the intense phase. In the 1-year maintenance phase, most maintained their weight reduction. However, the dropout rate during the maintenance phases was considerable.<sup>102,164-166</sup> While such diets reduce fat mass, lean body mass and growth velocity were unaffected in both Black and White children.<sup>167-171</sup> The focus of these studies was not on ethnicity, and only a few subjects were African-American. However, researchers observed notable differences in responses according to ethnicity.

The low-glycemic-index diet, which emphasizes food selection rather than calorie restriction, was more effective than a low-fat diet in overweight children after 4 months and maintained at 1 year.<sup>172</sup> Similar results were seen recently in adolescents.<sup>173</sup> Although these findings are encouraging, the data are not from a randomized controlled clinical trial, and should be further investigated.

These diets are not without some small risk of side effects, such as cholelithiasis, hyperuricemia, low serum protein, orthostatic hypotension, halitosis, and diarrhea. The indication for wide-ranging use of these diets in overweight children remains uncertain; therefore, such diets should be reserved for individuals who require rapid weight loss, and those who will be monitored closely under medical supervision.<sup>174,175</sup>

A recent trial of dietary intervention in children revealed that a low-fat diet lowered lipid levels with no adverse effects on growth or pubertal development; however, there were also no significant reductions in body mass index.<sup>122</sup> Another trial, performed in school children to assess the effects of a school-based dietary intervention on fruit and vegetable consumption, demonstrated that the intervention increased fruit and vegetable intake in both children and their parents at 1 year, with a

diminishing effect over time. This study showed similar results in the African-American sub-sample of this population.<sup>176</sup>

### Behavioral Modification

Clinical studies of behavioral modification programs in children and adolescents have focused on the mode of delivery and the additional effect of exercise.<sup>177</sup> Key findings are that: behavioral therapy is effective in facilitating weight loss, regardless of whether children are treated alone, separate from parents, or simultaneously with parents; physical activity improves the response to behavioral modification in the short term, but not necessarily in the long term; lifestyle exercise is more successful than structured exercise programs; longer-term cognitive behavior therapy is superior to short-term treatment; and, although short-term results of individual and group therapy are apparently better than a school model of treatment, long-term success was similar for all methods.<sup>178-184</sup>

Cultural differences contribute to the eating and lifestyle habits of individuals. Despite the trend of higher rates of overweight among Blacks and Hispanics, few clinical trials have focused on these groups.<sup>185</sup> Low socioeconomic status (SES) and African-American ethnicity overshadow other indicators of obesity. In overweight males, engaging in one high-intensity physical activity 3 to 5 days per week decreases the ethnic and SES-adjusted relative risk of being overweight.<sup>186</sup> In a dietary hospital-based program, although weight losses were similar for Blacks and Whites, the completion rate for low-income Black children was far below that of middle-income Black and White children.<sup>165</sup> This highlights the need for studies to address ethnic groups as a separate priority.

Culturally specific studies of African-American girls and their mothers that include behavioral modification, exercise, and food preparation, demon-

strated that: fat intake was reduced after 12 weeks<sup>187</sup>; as shown in other groups, children were more successful than their parents in maintaining weight loss<sup>188,189</sup>; academic performance and family support were determinants of successful weight loss, while early onset of obesity predicted less weight reduction<sup>190</sup>; and parental support was not necessarily important for weight loss in White children, but was clearly important for overweight Black girls and Hispanic children.<sup>101,191-194</sup>

The clinical response to weight loss interventions is affected by attitudes about weight. White adolescent girls from middle- and upper-middle-class families are more likely to aspire to be very thin.<sup>40,195-199</sup> Conversely, Blacks and Hispanics do not idealize ultra-thin body habitus.<sup>200-203</sup> In a recent study of African-American girls (8-10 years old) and their parents, most of the girls were happy with their weight or did not think about it; however, African-American girls living in predominantly White environments were more likely to report weight dissatisfaction, compared to girls living in predominantly Black environments. However 28% of parents were concerned about their daughters' weight, and 71% were concerned about their own weight.<sup>204</sup>

Overall, long-term behavioral studies show that behavioral modification is a vital part of sustained weight loss programs in all children, and family-based therapy may be more successful than individual therapy, particularly for African Americans.

### Multidisciplinary School-Based Programs

Although school-based programs have inconsistent success as measured by BMI reduction, they are overwhelmingly successful in changing behavior patterns following these interventions.<sup>205-211</sup> Children emerge with improved health knowledge and awareness, and healthier patterns of eating and exercise. These changes are the beginning of a healthier

lifestyle that is less likely to result in further overweight.

### Medications to Reduce Weight

Although phentermine and dexfenfluramine previously showed some success in small trials,<sup>212-216</sup> only subitramine and orlistat remain under study in children and adolescents.<sup>217,218</sup> A recent study of subitramine with behavioral therapy in adolescents demonstrated more weight loss with subitramine, compared to placebo, over 1 year, with minimal side effects.<sup>219</sup> Orlistat, along with diet and exercise, has shown promising results, including weight reduction and improvement in lipid levels and insulin resistance, with minimal side effects in the adolescent population.<sup>220</sup> The diabetes drug metformin has been studied in insulin-resistant and diabetic adolescents, showing significant weight reduction and improvement in insulin sensitivity, without adverse effects.<sup>156-158</sup>

### Surgery as a Treatment Modality

The results of gastric bypass surgery have been positive in small case-series reports. However, there are several possible complications from this surgery, and it remains a last resort for severely obese adolescents.<sup>174,221</sup>

It is clear that there is a role for dietary, exercise, and behavioral modification in successful weight-loss programs for children, and those incorporating these methods have longer-term results. Since rates of overweight among children continue to rise, the question of whether school-based or clinic-based programs are most successful is academic. It is necessary to combine all efforts to address the problem, in order to avert further increases in the prevalence of childhood overweight. It would seem appropriate for future clinical trials to place greater focus on high-risk groups such as African Americans and Hispanics, given the disparity in their prevalence rates compared to Whites of overweight and associated risk factors.

## CONCLUSIONS

### Evidence-Based Treatment Recommendations for Obese and Overweight Children

Obesity in the young, as in adults, results from an imbalance between energy intake and energy expenditure. This imbalance occurs when more calories are consumed than are expended in physical activity and physiological growth. There is ample evidence that maneuvers undertaken to restore energy balance through an increase in physical activity, and a reduction in excessive dietary intake, can correct this imbalance (thereby reducing excess body fat) over a period of time. However, treatment strategies that can be used to achieve and maintain a correction in body size remain elusive. This conundrum applies to children and adults of all ethnic groups, although there are some aspects to the causal pathway that render minority children uniquely vulnerable to the development of obesity. The preceding sections discussed the available evidence, and provided a rationale for a treatment plan. Health risks associated with obesity indicate that obesity is a medical problem that must be addressed by clinicians for the care of individual patients. Obesity is also a health problem with a rising prevalence among the pediatric population. Obesity is, therefore, a public health concern requiring public health strategies and policies that facilitate the implementation and effectiveness of individual case management and prevention efforts.

### Prevention

Obesity prevention interventions should be included in well-child care provided by primary care clinicians. Physicians or trained support staff should provide diet counseling and dietary guidelines to parents for their children as part of well-child visits. These anticipatory guidance measures should include parent education on food quality and quantity, as well as on eating

---

*Physicians or trained support staff should provide diet counseling and dietary guidelines to parents for their children as part of well-child visits.*

---

patterns. A clinician should calculate and plot the child's BMI on the BMI growth curves. Our work group recommends that efforts to prevent obesity with diet and activity interventions should begin when BMI exceeds the 75th percentile.

In a recent survey of health practitioners' needs in treating overweight children and adolescents, the majority felt that childhood obesity needs treatment due to its effects on chronic disease and future quality of life. Although the most recent Expert Committee Recommendations on Obesity Evaluation and Treatment in Children and Adolescents recommends the use of BMI as a clinically useful assessment of excess body fat, very few practitioners utilize this measure.<sup>222,223</sup>

### Treatment of Overweight/Obesity

Overweight/obesity in childhood is a risk factor that contributes to chronic diseases, and therefore should be treated. Modest reductions in caloric intake and modest increases in physical activity can have positive effects. Treatment should include measures to reverse diet and activity behaviors that promote obesity. Parents should be educated about heart-healthy diets that engage the entire family. The treatment plan should use behavioral change strategies, along with nutrition education, to lower energy intake. Parents should be strongly encouraged to enable the obese or overweight child to shift from sedentary

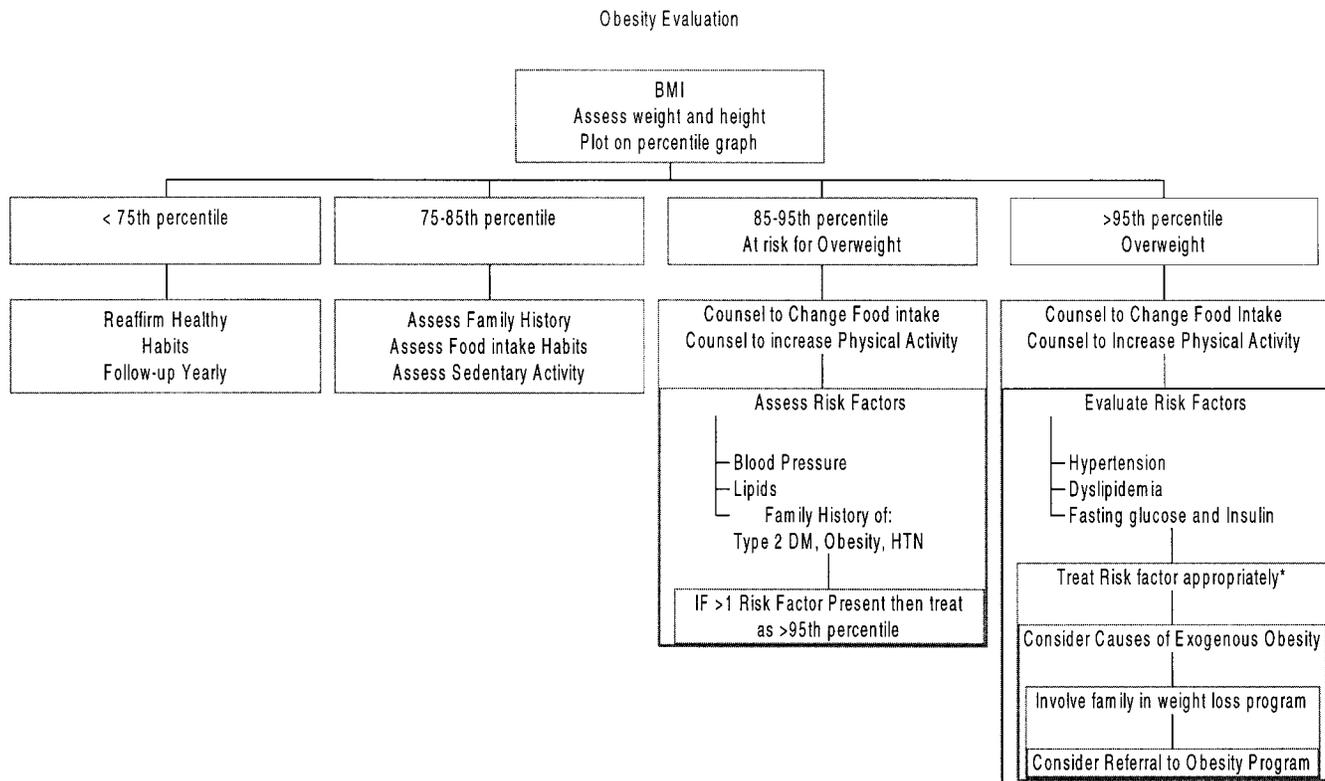


Fig 4. Overweight/obesity evaluation for cardiovascular risk

\* Risk factors such as hypertension, dyslipidemia and diabetes should be treated with medication and diet as prescribed by physician. Algorithm useful for evaluating overweight children and adolescents, with greater focus on evaluating concurrent cardiovascular risk factors in overweight children

behaviors, like television viewing, to more physically demanding activities. Counseling on behavioral or “lifestyle” changes must be delivered in consideration of ethnicity, particularly among minority children and their families. Recommendations on diet and physical activity will be more effective when appropriately tailored to the family’s culture/ethnicity and available resources.

### Risk-Factor Screening

Overweight/obese children should be screened for the presence of other factors that heighten risk for premature onset of obesity-related chronic disease, such as hypertension, dyslipidemia, diabetes, and subsequent atherosclerosis. While most practitioners report routinely evaluating levels of blood pressure

and lipids in overweight children, few examine for the presence of insulin resistance and early onset of type 2 diabetes.<sup>224</sup> Some ethnic groups appear to be at higher risk than others for obesity-related diseases. African Americans are at greater risk for hypertension, while Native Americans, African Americans, and Hispanics are all at greater risk for diabetes. Initial screening includes evaluating family history for hypertension, type 2 diabetes (including maternal gestational diabetes), stroke, myocardial infarction, or other premature cardiovascular events. The child’s blood pressure should be measured to determine if the blood pressure is elevated for age and height, according to hypertension guidelines for children. An obese or overweight child with another risk fac-

tor, elevated blood pressure, or positive family history, should have a medical evaluation. The medical evaluation should examine blood pressure, glucose metabolism, plasma lipid levels, and the presence of multiple risk factors. Figure 4 presents a useful algorithm for evaluating overweight children and adolescents.

### Public Health Issues

Despite the basic concept of changing diet patterns and increasing physical activity, the medical management of an overweight/obese child is extremely difficult. The best efforts of clinicians and parents to effect change in diet and exercise patterns can be disrupted by powerful environmental forces and conditions, such as:

- Inadequate safe and accessible recreation facilities.
- Lack of available supervised after-school programs that promote physical activity.
- Increasing public reliance on processed and fast foods that are less expensive and easily accessible.
- Food-label nutrition information that is difficult to understand.

Health agencies and communities need to confront the adverse effects of these environmental conditions on the health status and health future of the young. Public health approaches must be developed to address the problem of obesity/overweight in the young, particularly among minority youth. These approaches include developing and articulating policies; acquiring resources necessary to enact the policies; and educating the public on the health benefits of the policies vs the health risks of obesity.

REFERENCES

1. Flegal KM, Carroll MD, Johnson CL. Prevalence and trends in obesity among US adults, 1999–2000. *JAMA*. 2002;288(14):1723–1727.
2. Strauss RS, Pollack HA. Epidemic increase in childhood overweight, 1986–1998. *JAMA*. 2001;286(22):2845–2848.
3. 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.
4. Sinha R, Fisch G, Teague B, et al. Prevalence of impaired glucose tolerance among children and adolescents with marked obesity. *N Engl J Med*. 2002;346:802–810.
5. Kuczmarski RJ, Ogden CL, Guo SS, et al. 2000 CDC growth charts for the United States: methods and developments. *Vital Health Stat 11*. 2002;246:1–190.
6. Himes JH, Dietz WH. Guidelines for overweight in adolescent preventive services; recommendations from an expert committee. *Am J Clin Nutr*. 1994;59:307–316.
7. Deckelbaum RJ, Williams CL. Childhood obesity: the health issue. *Obes Res*. November 2001;9(suppl 4):239S–243S.
8. US Department of Health and Human Services. *Healthy People 2010: Understanding and Improving Health*. 2nd ed. Washington, DC: US Government Printing Office; November 2000.
9. Pastor P, Makuc DM, Reuben C, Xia H. *Chartbook on Trends in the Health of Americans*. Health, United States, 2002. Hyattsville, Md: National Center for Health Statistics; 2002.
10. US Department of Health and Human Services. Centers for Disease Control and Prevention. Youth Risk Behavior Surveillance System (YRBSS), United States, 2001. *MMWR*. 2001;51(SS04):1–64.
11. Bowlin SJ, Morrill BD, Nafziger AN, Jenkins PJ, Lewis C, Pearson TA. Validity of cardiovascular disease risk factors assessed by telephone survey: the Behavioral Risk Factor Survey. *J Clin Epidemiol*. 1993;46:561–571.
12. Hauck FR, White L, Cao G, Woolf N, Strauss K. Inaccuracy of self-reported weights and heights among American Indian adolescents. *Ann Epidemiol*. 1995;5:386–392.
13. Broussard BA, Johnson A, Himes JH, et al. Prevalence of obesity in American Indians and Alaska Natives. *Am J Clin Nutr*. 1991;53:1535S–1542S.
14. Goran MI. Metabolic precursors and effects of obesity in children: a decade of progress, 1990–1999. *Am J Clin Nutr*. 2001;73:158–171.
15. Heymsfield SB, Gallagher D, Kotler DP, Wang Z, Allison DB, Heshka S. Body-size dependence of resting energy expenditure can be attributed to nonenergetic homogeneity of fat-free mass. *Am J Physiol Endocrinol Metab*. January 2002;282(1):E132–E138.
16. Ravussin E, Bogardus C, Lillioja S. Familial dependence of the resting metabolic rate. *N Engl J Med*. 1986;315:96–100.
17. Treuth MS, Butte NF, Wong WW. Effects of familial predisposition to obesity on energy expenditure in multiethnic prepubertal girls. *Am J Clin Nutr*. 2000;71:893–900.
18. Parsons TJ, Power C, Logan S, Summerbell CD. Childhood predictors of adult obesity: a systematic review. *Int J Obes*. 1999;23:S1–S107.
19. Friedman JM. Obesity in the new Millennium. *Nature*. 2000;404:632–634.
20. Ruhl CE, Everhart JE. Leptin concentrations in the United States: relations with demographic and anthropometric measures. *Am J Clin Nutr*. September 2001;74(3):295–301.
21. Danadian K, Suprasongsin C, Janosky JE, Arslanian S. Leptin in African-American children. *J Pediatr Endocrinol Metab*. Sep–Oct 1999;12(5):639–644.
22. Stunkard AJ, Foch TT, Hrubec Z. A twin study of human obesity. *JAMA*. 1986;256:51–54.
23. Allison DB, Heshka S, Neale MC, Heymsfield SB. Race effects in the genetics of adolescents' body mass index. *Int J Obes Relat Metab Disord*. 1994;18:363–368.
24. Fabitz RR, Carmelli D, Hewitt JK. Evidence for independent genetic influences on obesity in middle age. *Int J Obes Relat Metab Disord*. 1992;16:657–666.
25. Pietilainen KH, Kaprio J, Rissanen A, et al. Distribution and heritability of BMI in Finnish adolescents aged 16y and 17y a study of 4884 twins and 2509 singletons. *Int J Obes Relat Metab Disord*. 1999;23:107–115.
26. Stunkard AJ, Sorensen TI, Hanis C, et al. The adoption study of human obesity. *N Engl J Med*. 1986;314:193–198.
27. Sorensen TI. Genetic aspects of obesity. *Int J Obes Relat Metab Disord*. 1992;16:227–236.
28. Morrison JA, Payne G, Barton BA, Khoury PR, Crawford P. Mother-daughter correlations of obesity and cardiovascular disease risk factor in Black and White households: the NHLBI Growth and Health Study. *Am J Public Health*. 1994;84:1761–1767.
29. Colilla S, Rotimi C, Cooper R, Goldberg J, Cox N. Genetic inheritance of body mass index in African-American and African families. *Genet Epidemiol*. 2000;18:360–376.
30. Stettler N, Tereshakovec AM, Zemel BS, et al. Early risk factors for increased adiposity: a cohort study of African-American subjects followed from birth to young adulthood. *Am J Clin Nutr*. August 2000;72(2):378–383.
31. Dowda M, Ainsworth Be, Addy CL, Saunders R, Riner W. Environmental influences, physical activity, and weight status in 8- to 16-year-olds. *Arch Pediatr Adolesc Med*. 2001;155:711–717.
32. Troiano RP, Flegal KM. Overweight children and adolescents: description, epidemiology, and demographics. *Pediatrics*. 1999;101:497–504.
33. Ravelli GP, Belmont L. Obesity in nineteen-year-old men: family size and birth order associations. *Am J Epidemiol*. 1973;109:66–70.
34. Wolfe WS, Campbell CC, Frongillo EA Jr, Haas JD, Melnik TA. Overweight school children in New York State: prevalence and characteristics. *Am J Public Health*. 1994;84:807–813.
35. Melnik TA, Rhoades SJ, Wales KR, Cowell C, Wolfe WS. Overweight school children in New York City: prevalence estimates and characteristics. *Int J Obes Relat Metab Disord*. 1998;22:7–13.
36. Patterson ML, Stern S, Crawford PB, et al. Sociodemographic factors and obesity in preadolescent Black and White girls: NHLBI's Growth and Health Study. *J Natl Med Assoc*. 1997;89:594–600.
37. Sun M, Gower BA, Nagy TR, Trowbridge CA, Dezenberg C, Goran MI. Total, resting, and activity-related energy expenditures are similar in Caucasian and African-American children. *Am J Physiol*. 1998;274:E232–E237.
38. DeLany JP, Bray GA, Harsha DW, Volau-

- fova J. Energy expenditure in preadolescent African-American and White boys and girls: the Baton Rouge Children's Study. *Am J Clin Nutr.* 2002;75:705-713.
39. Tershakovec AM, Kuppler KM, Zemel B, Stallings VA. Age, sex, ethnicity, body composition, and resting energy expenditure of obese African-American and White children and adolescents. *Am J Clin Nutr.* 2002;75: 867-871.
  40. Sobal J, Stunkard AJ. Socioeconomic status and obesity: a review of the literature. *Psychol Bull.* 1989;105:260-275.
  41. Kimm SY, Obarzanek E, Barton BA, et al. Race, socioeconomic status, and obesity in 9- to 10-year-old girls: the NHLBI Growth and Health Study. *Ann Epidemiol.* 1996;6: 266-275.
  42. Birch LL, Fisher JO. Mothers' child-feeding practices influence daughters' eating and weight. *Am J Clin Nutr.* 2000;71:1054-1061.
  43. Spruijt-Metz D, Lindquist CH, Birch LL, Fisher JO, Goran MI. Relation between others' child-feeding practices and children's adiposity. *Am J Clin Nutr.* 2002;75:581-586.
  44. Pettitt DJ, Nelson RG, Saad MF, Bennett PH, Knowler WC. Diabetes and obesity in the offspring of Pima Indian women with diabetes during pregnancy. *Diabetes Care.* 1993;16:310-314.
  45. Von Kries R, Koletzko B, Saurerwald T, et al. Breastfeeding and Obesity Cross Section Study. *Br Med J.* 1999;319:147-150.
  46. Gillman MW, Rifas-Shiman SL, Camargo CA Jr, et al. Risk of overweight among adolescents who were breastfed as infants. *JAMA.* 2001;285:2461-2467.
  47. Hediger ML, Overpeck MD, Kuczmarski RJ, Ruan WJ. Association between infant breastfeeding and overweight in young children. *JAMA.* 2001;285:2453-2460.
  48. Nguyen VT, Larson DE, Johnson RK, Goran MI. Fat intake and adiposity in children of lean and obese parents. *Am J Clin Nutr.* 1996;63:507-513.
  49. French SA, Story M, Jeffery RW. Environmental influences on eating and physical activity. *Annu Rev Public Health.* 2001;22: 309-335.
  50. Ludwig DS, Peterson KE, Gortmaker SL. Relation between consumption of sugar-sweetened drinks and childhood obesity: a prospective, observational analysis. *Lancet.* February 2001;35:505-508.
  51. McConahy KL, Smiciklas-Wright H, Birch LL, Mitchell DC, Picciano MF. Food portions are positively related to energy intake and body weight in early childhood. *J Pediatr.* 2002;140:340-347.
  52. Siega-Riz AM, Popkin BM, Carson T. Trends in breakfast consumption for children in the United States from 1965 to 1991. *Am J Clin Nutr.* 1998;67:748S-756S.
  53. Gortmaker SL, Must A, Sobol AM, Peterson K, Colditz GA, Dietz WH. Television viewing as a cause of increasing obesity among children in the United States, 1986-1990. *Arch Pediatr Adolesc Med.* 1996;150:356-362.
  54. Epstein LH, Valoski AM, Vara LS, et al. Effects of decreasing sedentary behavior and increasing activity on weight change in obese children. *Health Psychol.* 1995;14: 109-115.
  55. Robinson TN. Reducing children's television viewing to prevent obesity: a randomized controlled trial. *JAMA.* 1999;282: 1561-1567.
  56. Horn OK, Paradis G, Potvin L, Macaulay AC, Desrosiers S. Correlates and predictors of adiposity among Mohawk children. *Prev Med.* 2001;33:274-281.
  57. Ward DS, Trost SG, Felton G, et al. Physical activity and physical fitness in African-American girls with and without obesity. *Obes Res.* 1997;5:572-577.
  58. 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. *Obes Res.* 1999;7:208-214.
  59. National Personal Transport Survey, 1995. Available at: <http://www.cta.ornl.gov.npts.1995/doc/publications.html>. Access July 6, 2001.
  60. Horgen KB, Brownell KD. Confronting the toxic environment: environmental and public health actions in a world crisis. In: Wadden TA, Stunkard AJ, eds. *Handbook of Obesity Treatment.* New York, NY: Guildford Press; 2002:95-106.
  61. Ben-Sholomo Y, Kuh Diana. A life course approach to chronic disease epidemiology: conceptual models, empirical challenges, and interdisciplinary perspectives. *Int J Epidemiol.* 2002;31:285-293.
  62. Dietz WH. Critical periods in childhood for the development of obesity. *Am J Clin Nutr.* May 1994;59(5):955-959.
  63. Ravelli GP, Stein ZA, Susser MW. Obesity in young men after famine exposure in utero and early infancy. *N Engl J Med.* 1976;295: 349-353.
  64. Curhan GC, Willett WC, Rimm EB, Spiegelman D, Ascherio AL, Stampfer MJ. Birth weight and adult hypertension, diabetes mellitus, and obesity in US men. *Circulation.* 1996;94:3246-3250.
  65. Curhan GC, Chertow GM, Willett WC, et al. Birth weight and adult hypertension and obesity in women. *Circulation.* 1996;94: 1310-1315.
  66. Eid EE. Follow-up study of physical growth of children who had excessive weight gain in first six months of life. *BMJ.* 1970;2:74-76.
  67. Ong KKL, Ahmed ML, Emmett PM, Preece MA, Dunger DB. Association between postnatal catch-up growth and obesity childhood: prospective cohort study. *BMJ.* 2000; 320:967-971.
  68. Stettler N, Zemel BS, Kumanyika S, Stallings VA. Infant weight gain and childhood overweight status in a multicenter, cohort study. *Pediatrics.* 2002;109:194-199.
  69. Stettler N, Kumanyika SK, Katz SH, Zemel BS, Stallings VA. Rapid weight gain during infancy and obesity in young adulthood in a cohort of African Americans. *Am J Clin Nutr.* June 2003;77(6):1374-1378.
  70. Popkin BM, Richards MK, Montiero CA. Stunting is associated with overweight in children of four nations that are undergoing the nutrition transition. *J Nutr.* December 1996;126:3009-3016.
  71. Sichieri R, Siqueira KS, Moura AS. Obesity and abdominal fatness associated with undernutrition early in life survey in Rio de Janeiro. *Int J Obes Relat Metab Disord.* 2000; 24:614-618.
  72. Guo SS, Huang C, Maynard LM, et al. Body mass index during childhood, adolescence, and young adulthood in relation to adult overweight and adiposity: the Fels Longitudinal Study. *Int J Obes Relat Metab Disord.* 2000;24:1628-1635.
  73. Rolland-Cachera MF, Deheeger M, Bellisle F, Sempe M, Guillaud-Bataille M, Patois E. Adiposity rebound in children: a simple indicator for predicting obesity. *Am J Clin Nutr.* 1984;39:129-135.
  74. Chu NF, Rimm EB, Wang DJ, Liou HS, Shieh SM. Clustering of cardiovascular disease risk factors among obese school children: the Taipei Children Heart Study. *Am J Clin Nutr.* 1998;67:1141-1146.
  75. Macedo ME, Trigueiros D, de Freitas F. Prevalence of high blood pressure in children and adolescents. Influence of obesity. *Rev Port Cardiol.* 1997;16:27-28.
  76. Verma M, Chhatwal J, George SM. Obesity and hypertension in children. *Indian Pediatr.* 1994;31:1065-1069.
  77. Mo-Suwan L, Lebel L. Risk factors for cardiovascular disease in obese and normal school children: association of insulin with other cardiovascular risk factors. *Biomed Environ Sci.* 1996;9:269-275.
  78. Freedman DS, Dietz WH, Srinivasan SR, Berenson GS. The relation of overweight to cardiovascular risk factors among children and adolescents: the Bogalusa Heart Study. *Pediatrics.* 1999;103:1175-1182.
  79. Morrison JA, Barton BA, Biro FM, Daniels SR, Sprecher DL. Overweight, fat patterning, and cardiovascular disease risk factors in Black and White boys. *J Pediatr.* 1999;135: 451-457.
  80. Guillaume M, Lapidus L, Beckers F, Lambert A, Bjornorp P. Cardiovascular risk factors in children from the Belgian province of Luxembourg. The Belgian Luxembourg Child Study. *Am J Epidemiol.* 1996;144: 867-880.

81. 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;69:895-904.
82. Elcarte LR, Villa EI, Sada GJ, et al. The Navarra study. Prevalence of arterial hypertension, hyperlipidemia, and obesity in the infant-child population of Navarra. Association of risk factors. *An Esp Pediatr*. 1993;38:428-436.
83. Goldring D, Hernandez A, Choi S, et al. Blood pressure in a high school population. II. Clinical profile of the juvenile hypertensive. *J Pediatr*. 1979;95:298-304.
84. Sorof JM, Poffenbarger T, Franco K, Bernard L, Portman RJ. Isolated systolic hypertension, obesity, and hyperkinetic hemodynamic states in children. *J Pediatr*. 2002;140:660-666.
85. Rosner B, Prineas R, Daniels SR, Loggie J. Blood pressure differences between Blacks and Whites in relation to body size among US children and adolescents. *Am J Epidemiol*. 2000;151:1007-1019.
86. Voors AW, Webber LS, Berenson GS. Resting heart rate and pressure-rate product of children in a total biracial community: the Bogalusa Heart Study. *Am J Epidemiol*. 1982;116:276-286.
87. Jiang X, Srinivasan SR, Urbina E, Berenson GS. Hyperdynamic circulation and cardiovascular risk in children and adolescents. The Bogalusa Heart Study. *Circulation*. 1995;91:1101-1106.
88. Riva P, Martini G, Rabbia F, et al. Obesity and autonomic function in adolescence. *Clin Exp Hypertens*. 2001;23:57-67.
89. Voors AW, Radhakrishnamurthy B, Srinivasan SR, Webber LS, Berenson GS. Plasma glucose level related to blood pressure in 272 children, ages 7-15 years, sampled from a total biracial population. *Am J Epidemiol*. 1981;113:347-356.
90. Kanai H, Matsuzawa Y, Tokunaga K, et al. Hypertension in obese children: fasting serum insulin levels are closely correlated with blood pressure. *Int J Obes*. 1990;14:1047-1056.
91. Saito I, Nishino M, Kawabe H, et al. Leisure time physical activity and insulin resistance in young obese students with hypertension. *Am J Hypertens*. 1992;5:915-918.
92. Saito I, Kawabe H, Takeshita E, Wainai H, Murata K, Saruta T. Insulin resistance syndrome in adolescents and adults. *Hypertens Res*. 1996;19(suppl 1):S19-S22.
93. 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;150:667-674.
94. 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;24:1359-1364.
95. Rocchini AP, Key J, Bondie D, et al. The effect of weight loss on the sensitivity of blood pressure to sodium in obese adolescents. *N Engl J Med*. 1989;321:580-585.
96. Tounian P, Aggoun Y, Dubern B, et al. Presence of increased stiffness of the common carotid artery and endothelial dysfunction in severely obese children: a prospective study. *Lancet*. 2001;358:1400-1404.
97. Rocchini AP, Moorehead C, Katch V, Key J, Finta KM. Forearm resistance vessel abnormalities and insulin resistance in obese adolescents. *Hypertension*. 1992;19:615-620.
98. Hall JE, Brands MW, Henegar JR, Shek EW. Abnormal kidney function as a cause and a consequence of obesity hypertension. *Clin Exp Pharmacol Physiol*. 1998;25:58-64.
99. Rocchini AP, Moorehead C, Deremer S, Goodfriend TL, Ball DL. Hyperinsulinemia and the aldosterone and pressor responses to angiotensin II. *Hypertension*. 1990;15:861-866.
100. Bjorntorp P, Rosmond R. Neuroendocrine abnormalities in visceral obesity. *Int J Obes Relat Metab Disord*. 2000;24(suppl 2):S80-S85.
101. 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.
102. Figueroa-Colon R, von Almen TK, Franklin FA, Schuftan C, Suskind RM. Comparison of two hypocaloric diets in obese children. *Am J Dis Child*. 1993;147:160-166.
103. Wabitsch M, Hauner H, Heinze E, et al. Body-fat distribution and changes in the atherogenic risk-factor profile in obese adolescent girls during weight reduction. *Am J Clin Nutr*. 1994;60:54-60.
104. Gallistl S, Sudi KM, Aigner R, Borkenstein M. Changes in serum interleukin-6 concentrations in obese children and adolescents during a weight reduction program. *Int J Obes Relat Metab Disord*. 2001;25:1640-1643.
105. Rocchini AP, Katch V, Anderson J, et al. Blood pressure in obese adolescents: effect of weight loss. *Pediatrics*. 1988;82:16-23.
106. Rocchini AP, Katch V, Schork A, Kelch RP. Insulin and blood pressure during weight loss in obese adolescents. *Hypertension*. 1987;10:267-273.
107. Pi-Sunyer FX, Becker DM, Bouchard C, et al. *Clinical Guidelines on the Identification, Evaluation, and Treatment of Overweight and Obesity in Adults: the Evidence Report. National Institutes of Health*. Bethesda, Md: National Heart, Lung, and Blood Institute; 1998.
108. Owens S, Gutin B, Ferguson M, Allison J, Karp W, Le NA. Visceral adipose tissue and cardiovascular risk factors in obese children. *J Pediatr*. 1998;133(1):41-45.
109. Resnicow K, Morabia A. The relation between body mass index and plasma total cholesterol in a multiracial sample of US school children. *Am J Epidemiol*. 1990;132(6):1083-1090.
110. Hanis CL, et al. Lipoprotein and apolipoprotein levels among Mexican Americans in Starr County, Texas. *Arterioscler Thromb*. 1991;11(1):123-129.
111. Freedman DS, et al. Differences in the relation of obesity to serum triacylglycerol and VLDL subclass concentrations between Black and White children: the Bogalusa Heart Study. *Am J Clin Nutr*. 2002;75(5):827-833.
112. Freedman DS, et al. The relation of overweight to cardiovascular risk factors among children and adolescents: the Bogalusa Heart Study. *Pediatrics*. 1999;103(6, pt 1):1175-1182.
113. Morrison JA, et al. Sex and race differences in cardiovascular disease risk factor changes in school children, 1975-1990: the Princeton School Study. *Am J Public Health*. 1999;89(11):1708-1714.
114. Dwyer JT, et al. Predictors of overweight and overfatness in a multiethnic pediatric population. Child and Adolescent Trial for Cardiovascular Health Collaborative Research Group. *Am J Clin Nutr*. 1998;67(4):602-610.
115. Morrison JA, et al. Overweight, fat patterning, and cardiovascular disease risk factors in Black and White girls: The National Heart, Lung, and Blood Institute Growth and Health Study. *J Pediatr*. 1999;135(4):458-464.
116. Freedman DS, et al. Relationship of childhood obesity to coronary heart disease risk factors in adulthood: the Bogalusa Heart Study. *Pediatrics*. 2001;108(3):712-718.
117. Gillum RF. Indices of adipose tissue distribution, apolipoproteins B and AI, lipoprotein (a), and triglyceride concentration in children aged 4-11 years: the Third National Health and Nutrition Examination Survey. *J Clin Epidemiol*. 2001;54(4):367-375.
118. Strong JP. Natural history and risk factors for early human atherogenesis. Pathobiological Determinants of Atherosclerosis in Youth (PDAY) Research Group. *Clin Chem*. 1995;41(1):134-138.
119. McGill HC Jr, et al. Obesity accelerates the progression of coronary atherosclerosis in young men. *Circulation*. 2002;105(23):2712-2718.
120. McGill HC Jr, et al. Effects of nonlipid risk factors on atherosclerosis in youth with a favorable lipoprotein profile. *Circulation*. 2001;103(11):1546-1550.
121. Must A, et al. Long-term morbidity and mortality of overweight adolescents. A fol-

- low-up of the Harvard Growth Study of 1922 to 1935. *N Engl J Med.* 1992;327(19):1350-1355.
122. Obarzanek E, Kimm SY, Barton BA, Van Horn L, et al. Long-term safety and efficacy of cholesterol: seven-year results of the Dietary Intervention Study in Children (DISC). *Pediatrics.* 2001;107(2):256-264.
  123. Becque MD, et al. Coronary risk incidence of obese adolescents: reduction by exercise plus diet intervention. *Pediatrics.* 1988; 81(5):605-612.
  124. American Diabetes Association. Type 2 diabetes in children and adolescents. *Pediatrics.* 2000;105:671-680.
  125. 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;128:608-615.
  126. Paulsen EP, Richenderfer L, Ginsberg-Fellner F. Plasma glucose, free fatty acids, and immunoreactive insulin in sixty-six obese children. *Diabetes.* 1968;17:261-269.
  127. Saad MF, Knowler WC, Pettitt DJ, Nelson RG, Mott DM, Bennett PH. The natural history of impaired glucose tolerance in the Pima Indians. *N Engl J Med.* 1988;319:1500-1506.
  128. Weyer C, Bogardus C, Mott DM, Pratley RE. The natural history of insulin secretory dysfunction and insulin resistance in the pathogenesis of type 2 diabetes mellitus. *J Clin Invest.* 1999;104:787-794.
  129. Kahn SE. The importance of b-cell failure in the development and progression of type 2 diabetes. *JCEM.* 2001;86(9):4047-4058.
  130. Freedman DS, Srinivasan SR, Burke GL, et al. Relation of body fat distribution to hyperinsulinemia in children and adolescents: the Bogalusa Heart Study. *Am J Clin Nutr.* 1987;46:403-410.
  131. Gutin B, Islam S, Manso T, Cucuzzo N, Smith C, Stachura ME. Relation of percentage of body fat and maximal aerobic capacity to risk factors for atherosclerosis and diabetes in Black and White seven- to eleven-year-old children. *J Pediatr.* 1994;125:847-852.
  132. Caprio S, Hyman LD, Limb C, et al. Central adiposity and its metabolic correlates in obese adolescent girls. *Am J Physiol Endocrinol Metab.* 1995;269(1):E118-E126.
  133. 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;64:12-17.
  134. Gower BA, Nagy TR, Goran MI. Visceral fat, insulin sensitivity, and lipids in prepubertal children. *Diabetes.* 1999;48:1515-1521.
  135. Cruz ML, Bergman RN, Goran MI. Unique effect of visceral fat on insulin sensitivity in obese Hispanic children with a positive family history for type 2 diabetes. *Diabetes Care.* In press.
  136. Burke GL, Webber LS, Srinivasan SR, Radhakrishnamurthy B, Freedman DS, Berenson GS. Fasting plasma glucose and insulin levels and their relationship to cardiovascular risk factors in children: Bogalusa Heart Study. *Metabolism.* 1986;35:441-446.
  137. Arslanian S, Suprasongsin C, Janosky JE. Insulin secretion and sensitivity in Black versus White prepubertal healthy children. *JCEM.* 1997;82(6):1923-1927.
  138. Gower BA, Granger WM, Franklin F, Shewchuk RM, Goran MI. Contribution of insulin secretion and clearance to the greater acute insulin response to glucose in African-American vs Caucasian children and adolescents. *JCEM.* 2002;87(2218):2224.
  139. Goran MI, Cruz ML, Bergman RN, Watanabe RM. Insulin resistance and the associated compensatory response in Caucasian, African-American, and Hispanic children. *Diabetes Care.* In press.
  140. Amiel SA, Sherwin RS, Simonson DC, Lauritano AA, Tamborlane WV. Impaired insulin action in puberty: a contributing factor to poor glycemic control in adolescents with diabetes. *N Engl J Med.* 1986;315(4):215-219.
  141. Goran MI, Gower BA. Longitudinal study of pubertal insulin resistance. *Diabetes.* 2001;50:2444-2450.
  142. Moran A, Jacobs DR Jr, Steinberger J, et al. Insulin resistance during puberty: result from clamp studies in 357 children. *Diabetes.* 1999;48(10):2039-2044.
  143. Barker DJ, Hales CN, Fall CHD, Osmond C, Phipps K, Clark PM. Type 2 (non-insulin-dependent) diabetes mellitus, hypertension, and hyperlipidemia (Syndrome X): relation to reduced fetal growth. *Diabetologia.* 1993;36:62-67.
  144. Li C, Johnson MS, Goran MI. Effects of low birth weight on insulin resistance syndrome in Caucasian and African-American children. *Diabetes Care.* 2001;24(12):2035-2042.
  145. Haffner SM, Stern MP, Hazuda HP, Mitchell BD, Patterson JK. Increased insulin concentrations in nondiabetic offspring of diabetic parents. *N Engl J Med.* 1988;319(20):1297-1301.
  146. Elbein SC, Maxwell TM, Schumacher MC. Insulin and glucose levels and prevalence of glucose intolerance in pedigrees with multiple diabetic siblings. *Diabetes.* 1991;40(8):1024-1032.
  147. Warram JH, Martin BC, Krolewski AS, Soeldner JS, Kahn CR. Slow glucose removal rate and hyperinsulinemia precede the development of type II diabetes in the offspring of diabetic parents. *Ann Intern Med.* 1990;113(12):909-915.
  148. Barnett AH, Eff C, Leslie RD, Pyke DA. Diabetes in identical twins. A study of 200 pairs. *Diabetologia.* 1981;20(2):87-93.
  149. Osei K, Cottrell DA, Orabella MM. Insulin sensitivity, glucose effectiveness, and body fat distribution pattern in nondiabetic offspring of patients with NIDDM. *Diabetes Care.* 1991;14(10):890-896.
  150. Johnston C, Ward WK, Beard JC, McKnight B, Porte DP Jr. Islet function and insulin sensitivity in the non-diabetic offspring of conjugal type 2 diabetic patients. *Diabet Med.* 1990;7:119-125.
  151. Weijnen CF, Rich SS, Meigs JB, Krolewski AS, Warram JH. Risk of diabetes in siblings of index cases with type 2 diabetes: implications for genetic studies. *Diabet Med.* 2002;19(1):41-50.
  152. Martin BC, Warram JH, Rosner B, Rich SS, Soeldner JS, Krolewski AS. Familial clustering of insulin sensitivity. *Diabetes.* 1992; 41(7):850-854.
  153. Lillioja S, Mott DM, Zawadzki JK, et al. In vivo insulin action is familial characteristic in nondiabetic Pima Indians. *Diabetes.* 1987;36(11):1329-1335.
  154. Danadian K, Balasekaran G, Lewy V, Meza MP, Robertson R, Arslanian SA. Insulin sensitivity in African-American children with and without family history of type 2 diabetes. *Diabetes Care.* 1999;22:1325-1329.
  155. Goran MI, Coronges K, Bergman RN, Cruz ML, Gower BA. Influence of family history of type 2 diabetes on insulin resistance in children. 2002. Unpublished work.
  156. Freemark M, Bursey D. The effects of metformin on body mass index and glucose tolerance in obese adolescents with fasting hyperinsulinemia and a family history of type 2 diabetes. *Pediatrics.* 2001;107(4):1-7.
  157. Jones KL, Arslanian S, Peterokova VA, Park JS, Tomlinson MJ. Effect of metformin in pediatric patients with type 2 diabetes: a randomized controlled trial. *Diabetes Care.* 2002;25(1):89-94.
  158. Arslanian SA, Lewy V, Danadian K, Saad R. Metformin therapy in obese adolescents with polycystic ovary syndrome and impaired glucose tolerance: amelioration of exaggerated adrenal response to adrenocorticotropin with reduction of insulinemia/insulin resistance. *J Clin Endocrinol Metab.* 2002;87(4):1555-1559.
  159. McDuffie JR, Calis KA, Uwaifo GI, et al. Three-month tolerability of orlistat in adolescents with obesity-related comorbid conditions. *Obes Res.* 2002;10(7):642-650.
  160. Knowler WC, Barrett-Connor E, Fowler SE, et al. Reduction in the incidence of type 2 diabetes with lifestyle intervention or metformin. *N Engl J Med.* 2002;346(6):393-403.
  161. Pan XR, Li GW, Hu YH, et al. Effects of diet and exercise in preventing NIDDM in people with impaired glucose tolerance. The

- Da Qing IGT and Diabetes Study. *Diabetes Care*. 1997;20(4):537-544.
162. Tuomilehto J, Lindstrom J, Eriksson JG, et al. Prevention of type 2 diabetes mellitus by changes in lifestyle among subjects with impaired glucose tolerance. *N Engl J Med*. 2001;344(18):1343-1350.
  163. Kelley DE, Goodpaster BH. Effects of physical activity on insulin action and glucose tolerance in obesity. *Med Sci Sports Exerc*. 1999;31:S619-S623.
  164. Stallings VA, Archibald EH, Pencharz PB, et al. One-year follow-up of weight, total body potassium, and total body nitrogen in obese adolescents treated with the protein-sparing modified fast. *Am J Clin Nutr*. 1988;48:91-94.
  165. Suskind RM, Sothorn MS, Farris RP, et al. Recent advances in the treatment of childhood obesity. *Ann N Y Acad Sci*. 1993;699:181-199.
  166. Figueroa-Colon R, Franklin FA, Lee JY, von Almen TK, Suskind RM. Feasibility of a clinic-based hypocaloric dietary intervention implemented in a school setting for obese children. *Obes Res*. 1996;4:419-429.
  167. Figueroa-Colon R, Mayo MS, Aldridge RA, Winder T, Weinsier RL. Body composition changes in Caucasian and African-American children and adolescents with obesity using dual-energy X-ray absorptiometry measurements after a 10-week weight loss program. *Obes Res*. 1998;6:326-331.
  168. Epstein LH, Valoski A, McCurley. Effect of weight loss by obese children on long-term growth. *Am J Dis Child*. 1993;147:1076-1080.
  169. Sothorn MS. Childhood and adolescent obesity: exercise as a modality in the treatment of childhood obesity. *Pediatr Clin North Am*. 2001;48.
  170. Sothorn MS, Loftin M, Suskind RM, Udall JN Jr, Blecker U. The impact of significant weight loss on resting energy expenditure in obese youth. *J Investig Med*. 1999;47:222-226.
  171. Sothorn MS, 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 Paediatr*. 2000;89:1036-1043.
  172. Spieth LE, Harnish JD, Lenders CM, et al. A low-glycemic index diet in the treatment of pediatric obesity. *Arch Pediatr Adolesc Med*. 2000;154:947-952.
  173. Ebbeling CB, Leidig MM, Sinclair KB, Hangen JP, Ludwig DS. A reduced-glycemic load diet in the treatment of adolescent obesity. *Arch Pediatr Adolesc Med*. 2003;157:773-779.
  174. Yanovski JA. Intensive therapies for pediatric obesity. *Pediatr Clin North Am*. 2001;48:1041-1053.
  175. Merritt RJ, Bistran BR, Blackburn GL, Suskind RM. Consequences of modified fasting in obese pediatric and adolescent patients. *J Pediatr*. 1980;96:13-19.
  176. Reynolds KD, Franklin FA, Binkley D, et al. Increasing the fruit and vegetable consumption of fourth-graders: results from the High 5 Project. *Prev Med*. 2000;30:309-319.
  177. Jelilian E, Saelens BE. Empirically supported treatments in pediatric psychology: pediatric obesity. *J Pediatr Psychol*. 1999;24:223-248.
  178. Epstein LH, Valoski A, Wing RR, McCurley J. Ten-year follow-up of behavioral, family-based treatment for obese children. *JAMA*. 1990;264:2519-2523.
  179. Epstein LH, Wing RR, Penner BC, Kress MJ. Effect of diet and controlled exercise on weight loss in obese children. *J Pediatr*. 1985;107:358-361.
  180. Braet C, Van Winckel M, Van Leeuwen K. Follow-up results of different treatment programs for obese children. *Acta Paediatr*. 1997;86:397-402.
  181. Knip M, Nuutinen O, Uhari M, Forsius H. Treatment of obesity in children and adolescents (Swedish). *Nord Med*. 1991;106:186-188.
  182. Duffy G, Spence SH. The effectiveness of cognitive self-management as an adjunct to a behavioral intervention for childhood obesity: a research note. *J Child Psychol Psychiatry*. 1993;34:1043-1050.
  183. Sothorn MS, Hunter S, Suskind RM, Brown R, Udall JN Jr, Blecker U. Motivating the obese child to move: the role of structured exercise in pediatric weight management. *South Med J*. 1999;92:577-584.
  184. Israel AC, Guile CA, Baker JE, Silverman WK. An evaluation of enhanced self-regulation training in the treatment of childhood obesity. *J Pediatr Psychol*. 1994;19:737-749.
  185. Crawford PB, Story M, Wang MC, Ritchie LD, Sabry ZI. Ethnic issues in the epidemiology of childhood obesity. *Pediatr Clin North Am*. 2001;48:855-878.
  186. McMurray RG, Harrell JS, Deng S, Bradley CB, Cox LM, Bangdiwala SI. The influence of physical activity, socioeconomic status, and ethnicity on the weight status of adolescents. *Obes Res*. 2000;8:130-139.
  187. Stolley MR, Fitzgibbon ML. Effects of an obesity prevention program on the eating behavior of African-American mothers and daughters. *Health Educ Behav*. 1997;24:152-164.
  188. Epstein LH, Valoski AM, Kalarchian MA, McCurley J. Do children lose and maintain weight easier than adults: a comparison of child and parent weight changes from six months to ten years. *Obes Res*. 1995;3:411-417.
  189. Resnicow K, Yaroch AL, Davis A, et al. GO GIRLS!: results from a nutrition and physical activity program for low-income, overweight African-American adolescent females. *Health Educ Behav*. 2000;27:616-631.
  190. Gross I, Wheeler M, Hess K. The treatment of obesity in adolescents using behavioral self-control: an evaluation. *Clin Pediatr*. 1976;15:920-924.
  191. Baskin ML, Ahluwalia HK, Resnicow K. Obesity intervention among African-American children and adolescents. *Pediatr Clin North Am*. 2001;48:1027-1039.
  192. 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. *Pediatrics*. 1990;85:345-352.
  193. Cousins JH, Rubovits DS, Dunn JK, Reeves RS, Ramirez AG, Foreyt JP. Family versus individually oriented intervention for weight loss in Mexican-American women. *Public Health Rep*. 1992;107:549-555.
  194. Epstein LH, McCurley J, Wing RR, Valoski A. Five-year follow-up of family-based behavioral treatments for childhood obesity. *J Consult Clin Psychol*. 1990;58:661-664.
  195. Flores R. Dance for Health: improving fitness in African-American and Hispanic adolescents. *Public Health Rep*. 1995;110:189-193.
  196. Field AE, Camargo CA Jr, Taylor CB, Berkley CS, Roberts SB, Colditz GA. Peer, parent, and media influences on the development of weight concerns and frequent dieting among preadolescent and adolescent girls and boys. *Pediatrics*. 2001;107:54-60.
  197. Wadden TA, Foster GD, Stunkard AI, et al. Dissatisfaction with weight and figure in obese girls: discontent but not depression. *Int J Obes*. 1989;13:89-97.
  198. Striegel-Moore RH, Silbertson LR, Rodin J. Toward an understanding of risk factors in bulimia. *Am J Psychol*. 1986;41:246-263.
  199. Dornbusch SM, Carl Smith JM, Duncan PD, et al. Sexual maturation, social class, and the desire to be thin among adolescent females. *J Dev Behav Pediatr*. 1984;5:308-314.
  200. Huenemann RL, Shapiro LR, Hampton MC, et al. A longitudinal study of gross body composition and body conformation and their association with food and activity in a teenage population: view of teenage subjects on body conformation, food, and activity. *Am J Clin Nutr*. 1966;18:325-328.
  201. Levinson R, Powell B, Steelman LC. Social location, significant others, and body image among adolescents. *Soc Psychol Q*. 1986;49:330-337.
  202. Maddox GL, Black KW, Liederman VR. Overweight as social deviance and disability. *J Health Soc Behav*. 1968;4:287-298.
  203. Stern MP, Pugh JA, Gaskill SP, et al. Knowledge, attitudes, and behavior related to obesity and dieting in Mexican Americans and Anglos: the San Antonio Heart Study. *Am J Epidemiol*. 1982;115:917-927.
  204. Sherwood NE, Story M, Beech B, et al. Body image perceptions and dieting among

## OVERWEIGHT IN ETHNIC CHILDREN - Nesbitt et al

- African-American pre-adolescent girls and parents/caregivers. *Ethn Dis*. 2003;13:200–207.
205. Sothorn MS, Loftin JM, Udall JN Jr, et al. Inclusion of resistance exercise in a multidisciplinary outpatient treatment program for preadolescent obese children. *South Med J*. 1999;92:585–592.
206. Mo-suwan L, Pongprapai S, Junjana C, Puetpaiboon A. Effects of a controlled trial of a school-based exercise program on the obesity indexes of preschool children. *Am J Clin Nutr*. 1998;68:1006–1011.
207. Sothorn MS, Schumacher H, von Almen TK, Carlisle LK, Udall JN Jr. Committed to kids: an integrated, 4-level team approach to weight management in adolescents (review). *J Am Diet Assoc*. 2002;102(suppl 3):S81–S85.
208. Sahota P, Rudolf MCJ, Dixey R, Hill AJ, Barth JH, Cade J. Randomized controlled trial of primary school based intervention to reduce risk factors for obesity. *BMJ*. 2001;323:1–5.
209. Sahota P, Rudolf MCJ, Cixey R, Hill AJ, Barth JH, Cade J. Evaluation of implementation and effect of primary school based intervention to reduce risk factors for obesity. *BMJ*. 2001;323:1–4.
210. Holcomb JD, Lira J, Kingery PM, Smith DW, Lane D, Goodway J. Evaluation of Jump Into Action: a program to reduce the risk of non-insulin dependent diabetes mellitus in school children on the Texas-Mexico border. *J Sch Health*. 1998;68:1–12.
211. Weber JL, Cunningham-Sabo L, Skipper B, et al. Portion-size estimation training in second- and third-grade American Indian children. *Am J Clin Nutr*. 1999;64(suppl 4):782S–787S.
212. Lorber J. Obesity in childhood: a controlled trial of anorectic drugs. *Arch Dis Child*. 1966;41:309–312.
213. Spranger J. Phentermine resinate in obesity: clinical trial of mirapront in adipose children (German). *Munch Med Wochenschr*. 1965;107:1833–1834.
214. Malecka-Tendera E, Koehler B, Muchacka M, et al. Efficacy and safety of dexfenfluramine treatment in obese adolescents (Polish). *Pediatr Pol*. 1996;71:431–436.
215. Bacon GE, Lowrey GH. A clinical trial of fenfluramine in obese children. *Curr Ther Res Clin Exp*. 1967;9:626–630.
216. Pedrinola F, Cavaliere H, Lima N, Medeiros-Neto G. Is DL-fenfluramine a potentially helpful drug therapy in overweight adolescent subjects? *Obes Res*. 1994;2:1–4.
217. James TPT, Astrup A, Finer N, et al. Effect of sibutramine on weight maintenance after weight loss: a randomized study. *Lancet*. 2000;356:2119–2125.
218. 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. *Int J Obes Relat Disord*. 2000;24:1573–1578.
219. Berkowitz RI, Wadden TA, Terhakovec AM, Cronquist JL. Behavior therapy and sibutramine for the treatment of adolescent obesity: a randomized controlled trial. *JAMA*. 2003;289:1805–1812.
220. McDuffie JR, Calis KA, Uwaifo GI, et al. Three-month tolerability of orlistat in adolescents with obesity-related comorbid conditions. *Obes Res*. 2002;10(7):642–650.
221. Glenny AM, O'Meara S, Melville A, Sheldon TA, Wilson C. The treatment and prevention of obesity: a systematic review of the literature [review]. *Int J Obes Relat Metab Disord*. 1997;21:715–737.
222. Barlow SE, Dietz WH. Obesity evaluation and treatment: expert committee recommendations. *Pediatrics*. 1998;102(3).
223. Story MT, Neumark-Stzainer DR, Sherwood NE, et al. Treatment of overweight children and adolescents: a needs assessment of health practitioners. Attitudes, barriers, skills, and training needs among healthcare professionals. *Pediatrics*. 2002;110:210–214.
224. Barlow SE, Dietz WH, Klish WJ, Trowbridge FL. Treatment of overweight children and adolescents: a needs assessment of health practitioners. Reports from Pediatricians, Pediatric Nurse Practitioners, and Registered Dietitians. *Pediatrics*. 2002;110:229–235.

### AUTHOR CONTRIBUTIONS

*Design and concept of study:* Nesbitt, Ashaye, Stettler, Sorof, Goran, Falkner

*Acquisition of data:* Nesbitt, Ashaye, Sorof, Goran, Parekh, Falkner

*Data analysis and interpretation:* Nesbitt, Ashaye, Stettler, Goran, Parekh, Falkner

*Manuscript draft:* Nesbitt, Ashaye, Stettler, Sorof, Goran, Parekh, Falkner

*Statistical expertise:* Stettler, Ashaye, Parekh

*Administrative, technical, or material assistance:* Ashaye, Falkner

*Supervision:* Nesbitt