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Childhood Obesity: Update on Predisposing Factors and Prevention Strategies

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Abstract Obesity is a global epidemic and children are affected in increasing numbers. Overweight children are at increased risk of becoming overweight adults with associated chronic diseases. In this update, we present key findings from a review of the current literature focused on potential causes and strategies for preventing childhood obesity. We highlight recent evidence regarding the role of genetics, maternal body mass index, postnatal influences, and environmental effects throughout childhood in predicting overweight. We also summarize the results of new research that examined the effectiveness of intervention strategies implemented in a variety of settings: home, school, community, and health care system. Statements recently released by the Centers for Disease Control and Prevention (CDC) and the US Department of Health and Human Services emphasize the need for effective policy and environmental change to promote healthy lifestyle change at the individual and population levels.

Keywords Obesity · Children · Predisposing factors · Prevention · Genetics · Physical activity · Caloric sweeteners · Added sugars

Introduction

Childhood obesity is increasingly common worldwide and is associated with comorbidities in childhood and in adulthood. In the United States, 17% of children are overweight, and overweight children are likely to be overweight as adults [1, 2]. These children suffer from psychosocial issues (eg, depression, being bullied, and decreased school performance) and physical complications (eg, hypertension, nonalcoholic fatty liver disease, type 2 diabetes, and hyperlipidemia). Being overweight in childhood significantly increases future mortality in adulthood. In a study of American Indian children, Franks et al. [3••] found that after a median follow-up of 24 years, children in the highest quartile of body mass index (BMI) had more than double the mortality rate.

The factors that predispose an individual child to excess weight gain are complex, as highlighted by the current lack of effective prevention strategies despite the tremendous interest and concern surrounding childhood obesity. At its simplest, excess weight gain is the result of burning fewer calories than are consumed on an ongoing basis. At a more complex level, obesity is a failure of the ability to self-regulate—to match intake to energy needs. Thus, the question becomes: what causes the failure of this innate regulatory balance in children? In this review, we discuss selected literature published in 2009 and early 2010 on excess weight gain in children, first examining biologic and environmental predictive factors, and then discussing new developments in preventive strategies.

Predisposing Factors

Genetics

Obesity tracks in families, and one of the strongest predictors of child overweight is the BMI of the mother and father. In recent years, progress was made in identifying genes that may contribute to this effect. The

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FTO (fat mass and obesity-associated) gene is a large gene on chromosome 16; in 2007, three independent studies identified associations between single nucleotide polymorphisms on *FTO* and BMI, creating much excitement [4]. The absolute risk generated by the *FTO* gene is relatively modest, is found in Caucasian populations, and is modifiable by environmental factors. Cecil et al. [4] studied 2,726 children for associations with the rs9939609 variant of the *FTO* gene and found strong associations with BMI and weight. They further studied 97 of these children, and found that this allele predicted increased energy intake at a test meal that was independent of body weight [5]. Interestingly, the weight of the food consumed was not substantially different, suggesting that it was selection of high energy-dense foods that accounted for the increase in energy consumption by the children with the variant *FTO*.

A defect in the melanocortin 4 receptor gene (*MC4R*) is associated with a severe, early form of monogenic obesity in children. *MC4R* deficiency is characterized by hyperphagia, hyperinsulinemia, and increased fat mass [6]. Recent studies showed that variants in *MC4R* are also associated with fat mass, weight, and risk of obesity and, in children, possibly associations with regulation of weight through energy intake and energy expenditure [7, 8]. In a study of *MCR4* and *FTO* genetic variants in Finnish adolescents, the gene effects were additive but, again, were modifiable by physical activity [9].

The genetic studies are exciting because behavioral studies support early childhood personality traits as important longitudinal predictors of obesity. For example, low inhibitory control at age 7 years was predictive of increased weight gain in Hispanic children [10•]. In 4-year-old children, those with less ability to delay gratification were 30% more likely to be overweight at age 11 years; however, maternal BMI explained part of the effect [11]. In children age 3 to 12 years, those with both decreased self-control and less ability to delay gratification had significantly higher BMI and rate of BMI increase, and the two behaviors were additive [12]. The authors of this last study explain that these self-regulatory behaviors are more "trait than state." The consistency of these three studies showing behaviors so early in life makes it seem more likely that they are inherited traits rather than taught behaviors.

Prenatal and Perinatal Influences

Researchers have considered effects that may occur in utero to "program" for obesity. In particular, as the number of women who are overweight early in pregnancy increases, the effects of this altered metabolism on the fetus may be important. Catalano et al. [13] examined 89 women either with impaired glucose tolerance or normal glucose tolerance in a prospective study of in utero effects on childhood obesity. They assessed these women during pregnancy and at delivery, and assessed their children at an average of 8.8 years later. The strongest predictor of being in the highest tertile for weight for the children was the maternal pregravid weight, and this predictor was independent of maternal glucose tolerance and infant birth weight. Similarly, a study of 11,653 school children in England found that increased current maternal and paternal BMI and increased pre-pregnancy BMI of the mother were significantly associated with rapid weight gain between the ages of 3 and 5 years [14]. The challenge/confounder for both studies (and for most studies of maternal weight status) is that lifestyle habits of the parents track with their BMI. Although some habits can be directly measured, issues with validity of self-report and difficulty in accurately measuring diet and activity levels complicate the separation of early family environmental effects on the child from the effect of in utero programming.

Postnatal Effects

Hitze et al. [15] re-examined the phenomena of rapid postnatal weight gain in German infants. Of 351 infants born appropriate for gestational age (AGA), they found that 22% gained weight rapidly, which was defined as a greater than average gain in weight from birth to age 2 years. In this group of rapid gainers, the prevalence of obesity at age 12 years was 26.2% among girls and was 28.9% among boys, compared to the significantly lower prevalence in the slower gainers (2.8% among girls and 5.0% among boys). Gestational age accounted for some of the effect, but other early life factors such as breastfeeding, maternal smoking, parental BMI, and socioeconomic status did not. These investigators found that the infants who were rapid gainers had lower birth weights and earlier gestational age, although they fit within the guidelines of AGA. Because small for gestational age (SGA) infants are prone to rapid weight gain and later obesity, this study suggests that the effect of intrauterine weight gain is a spectrum and even small shifts toward SGA may have later effects. Breastfeeding is another well-studied postnatal effect that was previously found to reduce the risk of obesity. However, studying the phenomenon is a challenge because controlling completely for parenting and environmental effects that track with breastfeeding is difficult. Perhaps the strongest evidence that breastfeeding is protective was published recently by Metzger et al. [16••]. In a longitudinal, nationally representative, US survey, they compared breastfed and non-breastfed siblings and examined the presence of obesity in adolescence. In sibling pairs in which one was breastfed and one was not, the breastfed sibling had a lower BMI in adolescence and was less likely to be overweight and obese.

Parent Restriction

Parent feeding behaviors (eg, restriction, food prompting, and pressuring) were known to be associated with the child's weight, but it was unclear whether parents develop these mechanisms in response to a challenge with their child (eg, a picky eater or rapid gainer) or if the parent behaviors predict the child's behavior and weight. Hennessy et al. [17] examined the association between parent restrictiveness and child's disinhibited eating to see if other parental characteristics might modify the effect. They found that parents who are more restrictive have children with more disinhibited eating, and that the child's disinhibited eating is positively associated with BMI. Negative parenting characteristics (eg, coerciveness, inconsistency, and unpredictability with regard to feeding) strengthened the association between restrictiveness and BMI. Furthermore, a trend existed toward less association of parent restrictiveness and child's disinhibited eating with greater parent supportiveness, suggesting that parental style is the modifiable effect. A study by Anzman et al. [10•] supported these results; they found that restrictive parenting exacerbated the association between uninhibited eating and increased BMI.

Sedentary Behavior

The association of low physical activity and increased sedentary time with increased risk of obesity is well established, but recent studies have helped to clarify the precise associations. Screen time (ie, television and computer time) is an important potentially modifiable component of sedentary behavior. Almost half of obese children engaged in ≥2 h a day of screen time, compared to 33% of normal-weight children [18, 19]. African American children had the highest prevalence, with 66% watching more than 2 h a day compared to European American (43%) and Mexican American (48%) children. Factors that are associated with increased screen time are lower family income and the presence of a TV in the child's bedroom [20]. Conversely, participating in sports, being a girl, having a negative attitude toward screen time, participating in after-school programs, and participating in the "Turn off the Screen Week" campaign were protective. All screen time may not be equal. New evidence supports this idea by showing that for children under the age of 7 years, hours of commercial-containing TV are positively associated with BMI, whereas other categories (eg, videos and noncommercial-containing children's TV) are not [21]. Programming for children under the age of 5 years has a food advertisement every 5 min, 95% of which are for poor-quality food. This may partly explain why eating in front of the TV was associated with higher BMI in a 3-year longitudinal study of children [22].

Mitchell et al. [23] studied the association of hours of objectively measured sedentary behavior and odds of being obese, and confirmed that sedentary behavior was positively associated with obesity. However, when they further adjusted their models for minutes of moderate-to-vigorous physical activity (MVPA), the findings for sedentary behavior were no longer significant, suggesting that the two are closely interrelated. As expected, MVPA and sedentary behavior were closely negatively correlated (ie, sedentary children engage in less MVPA.)

Decreased Physical Activity

Prospective examination of cardiorespiratory fitness in school-aged children demonstrated that children with low fitness had significantly higher risk (3.5-fold) of being overweight and had disproportionate increase in weight gain [24]. Together with the study by Roberts et al. [25] showing that low aerobic fitness predicts lower test scores in math, reading, and language—a finding that was independent of parent education level and the child's BMI—these studies confirm that increasing cardiorespiratory fitness should be a key area of focus for school-aged children.

Increased Caloric Sweeteners

The evidence is solid that caloric sweetened beverages (CSBs) are associated with excess weight gain, as reviewed in a meta-analysis of 30 studies published in 2006 [26]. CSBs may act by several mechanisms, including by increasing fructose. In children, the largest dietary source of fructose (a lipogenic sugar) is CSBs [27], which provide extra calories that are somehow "less recognized" than solid food, and which replace milk and decrease calcium consumption [28]. In a longitudinal study of girls, Fiorito et al. [29] showed that CSB consumption is moderately stable over time, that the increased BMI associated with this consumption is durable, and that, when present in a 5-year-old child, increased BMI will persist into adolescence. Lim et al. [30] prospectively followed young (3–5 years old) African American children, and showed that increased fruit juice and CSB intake at baseline predicted increased weight gain at follow-up 2 years later. In the study by Fiorito et al. [29], no associations with weight were found for fruit juice, but the difference in the two studies likely is from the length of follow-up, because fruit juice consumption decreases as children age and thus would have less effect in adolescents. Although the amount of CSB consumed in childhood does not seem to be associated with BMI in adulthood, in a 21-year, longitudinal study of Finnish children [31], increases in CSB intake over time predicted increased BMI in women, suggesting that the persistent intake of CSB promotes weight gain.

Although individual physical activity behaviors and feeding/eating behaviors predispose to obesity, many of these behaviors occur together in the same family and likely are synergistic. More evidence for the influence of maternal BMI on the environment of the child comes from the study by Gubbels et al. [32] in which they showed that obesogenic behaviors occur in clusters in 2-year-old Dutch children. They demonstrated that, even in these very young children, TV watching was associated with increased consumption of snacks and sugar-sweetened beverages, and lower maternal education and higher maternal BMI were associated with increased sedentary behavior and snacking in the children. Papas et al. [33] studied the diets of African American adolescent mothers and their children, and found that diets were similar between parent and child and that both consumed more sweets and less fruits and vegetables than recommended. Both studies support that the strong association between maternal BMI and child overweight is at least partially mediated via the child-raising behaviors of the mother.

Strategies for Childhood Obesity Prevention

Given that even where one lives influences the likelihood of being overweight (South vs North, rural vs urban), it is clear that obesity prevention is not simply an issue of individual responsibility. Successful strategies for obesity prevention need to effect change on the societal and governmental levels, the community, school, and childcare levels, and the individual and family levels. Although it would appear that simply targeting the well-established predictors, such as those discussed earlier, would be effective, such may not be the case. Because of the complexity of change and the clustering of obesogenic habits, prevention is not straightforward. To be most useful, successful strategies must be generalizable, durable, and cost-effective. Unfortunately, few studies address the issue of cost-effectiveness of their interventions.

Dodson et al. [34] evaluated enablers and barriers to passing legislation that influenced childhood obesity prevention by interviewing legislators and their staff in 11 states. They found that factors promoting passage included support from parents, physicians, and schools, sponsorship by senior leaders, and national media exposure [34]. Not surprisingly, barriers included lobbying by companies that produce unhealthy foods and misunderstandings regarding the legislation of school foods. In the United States, states have substantial power over policies that affect child health, particularly through governance of the school systems and decision making about the transportation infrastructure, safety improvements, and access to parks. To help guide states and communities, the CDC in 2009 released a comprehensive statement of recommended community strategies for obesity reduction (Table 1) [35••]. For guidance at all levels, in 2010, the US Department of Health and Human Services released "The Surgeon General's Vision for a Healthy and Fit Nation," including a concise list of individual healthy choices in the section on opportunities (Table 2) [36].

School-Based Strategies

School-based programs have great opportunity to promote healthy nutrition and physical activity because most children attend school, and a child spends more than half his/her waking hours at school on any given school day. A 2010 review of 23 home-based and preschool/childcare-based obesity prevention studies in children ages 0 to 5 years concluded that although the interventions varied widely, most included multiple modalities to achieve behavior change, and only some were successful on behaviors that contribute to obesity, more often in the preschool-based studies [37]. The authors note that many of the preschool studies lack a parent component and hypothesized that this may contribute to the lack of success. Gonzalez-Suarez et al. [38] recently published a meta-analysis of school-based programs and found that, in general, school-based programs were effective in preventing overweight/obesity and that longer programs (>1 year) were more likely to be effective.

An example of a randomized, controlled study of a school-based program is the "STOPP" school-based intervention study in Sweden, in which low-fat dairy and whole grains were promoted at school and all sugar-sweetened beverages were eliminated [39]. Children at intervention schools were less likely to become overweight and had healthier eating habits at home and school. Walther et al. [40] demonstrated that aerobic capacity (VO_2 max) can be improved through school-based activity, and they showed a trend toward improvement in BMI, although it was not significant in this 1-year study. A randomized, controlled, school-based trial of 12-year-old boys also showed benefits; the boys who received two additional 20-min lunchtime physical activity sessions per week plus some health education had improved waist circumference, body fat, and physical fitness level [41]. One recent school-based study showed what does not work—Cullen et al. [42] reported on the Free Fruit and Vegetable program in a Houston, TX, high school and found that it had no impact on student exposure and preference for fruits and vegetables. Ultimately, school-based programs need to be supported and integrated with community- and family-based changes. This requires cooperative efforts and education—possibly easier said than done. In an interesting focus group study of student, parent, and teacher views, the authors

Table 1 Recommended community-based strategies

• Increasing availability of healthier foods and beverages in public service venues and restricting access to less healthy foods and beverages
• Improving affordability of healthier food and beverage choices in public service venues
• Improve geographic availability of supermarkets in underserved areas
• Provide incentives to food retailers to offer healthier foods and beverages in underserved areas
• Increase access to farmers' markets
• Increase incentives for use of foods from local farms
• Limit advertising of less healthy foods and beverages
• Discourage consumption of sugar-sweetened beverages
• Increase support for breastfeeding
• Require physical education in schools and increase amount of physical activity in such programs
• Increase extracurricular physical activity
• Reduce screen time in public service venues
• Improve access to parks and other outdoor recreational facilities
• Enhance infrastructure supporting bicycling and walking
• Locate schools within easy walking distance of neighborhoods
• Improve access to public transportation
• Zone for mixed-use development
• Enhance personal safety in areas where persons are or could be physically active
• Participate in community coalitions or partnerships to address obesity

(*Adapted from* Khan et al. [35].)

found that all groups had attribution errors: the early adolescents blamed their unhealthy behaviors on situational factors, the teachers blamed the parents, and the parents blamed their children [43].

Primary Health Care-Based Strategies

Because primary care physicians see children at regularly scheduled well-child check-ups and are involved in parent education to promote wellness in children, these visits would seem to be an effective mechanism to prevent obesity. However, simply implementing current guidelines in the clinic is a challenge. Dorsey et al. [44] studied the charts of children ranging in age from 3 to 18 years and found that even when BMI was addressed at initial visits, attention to BMI quickly dropped off, and by the fifth clinic visit was only documented 27% of the time. Another study reported that pediatricians felt the barriers included that the parents were not motivated, the child was not motivated, the parent was overweight, and the families had poor habits (eg, eating fast food, not exercising) [45]. Interestingly, only a minority of the pediatricians in this study knew the correct definition and prevalence of obesity in children, and less than half were familiar with the American Academy of Pediatrics recommendations for exercise and juice consumption. Woolford et al. [46] developed a one-page tool to facilitate physician assessment of obesity with parents of preschoolers; however, only 23% of the 25 pediatricians who tried the tool reported that they were very likely to continue using it. The greatest barrier to its use was time. The matter of time brings up the issue of cost-effectiveness that was studied in an Australian randomized controlled trial in which general practitioners intervened with overweight and obese children [47]. The researchers found that the brief office counseling interventions were ineffective in improving physical activity, nutrition, and BMI and that the

Table 2 Recommended prevention strategies

• Reducing consumption of sodas and juice containing added sugars
• Reducing consumption of energy-dense foods that primarily contain added sugars or added fats
• Eating more fruits, vegetables, whole grains, and lean proteins
• Controlling portions
• Drinking more water
• Choosing low-fat or nonfat dairy
• Limiting TV viewing time and consider keeping TV out of children's rooms
• Becoming more physically active throughout the day
• Breastfeeding exclusively to 6 months

(*Adapted from* US Department of Health and Human Services [36].)

costs were high. Thus, although the pediatrician's office must be part of the solution, cost-effective systems and strategies are needed if they are to have a positive impact.

Home-Based Programs

Because obesity occurs at all ages and usually clusters in families, attention may need to shift from the individual to the family as the unit of change and measurement [48]. Sonneville et al. [49] conducted an interesting focus group study with parents to understand the barriers to and facilitators of obesity prevention. The parents identified numerous barriers, including child preference for the current status, difficulty with other family members adopting the behaviors, and difficulty in committing to the changes themselves. For facilitators, parents thought that changing their own habits and shopping would facilitate adherence as well as having better access to physical activities to replace the TV time.

Home-based strategies to improve physical activity have had varying success. In particular, changing screen time seems difficult. In one US study, 67% percent of racial/ethnic minority children age 2 to 13 years already had a TV in their bedroom [50]. This situation is particularly problematic, because parents believe that having the TV already in the bedroom was difficult to change [49]. Parents report that they use the TV to keep their child occupied so they can do other things, to help the child sleep, and to free up the other TV so other family members can watch [50]. To change TV time, parents will have to be convinced that health benefits outweigh the inconvenience incurred. In one study to reduce television time, they were able to achieve significant reductions in the number of TVs in bedrooms, but the actual time spent watching TV by the study subjects was unchanged despite the 2-h workshop and newsletters [51]. Another strategy to increase physical activity was more successful. Eiholzer et al. [52] assigned two groups of 13-year-old boys to either physical activity (playing hockey) with additional resistance training, or hockey alone. At the end of 4 months, spontaneous physical activity among boys with resistance training increased 25% and at 12 months was still increased by 13%.

A reduction in added sugar has potential for benefiting children. Ventura et al. [53] found that children who reduced added sugars (by the equivalent of one soda) had improved insulin and glucose levels, and children who increased fiber had improved BMI and visceral fat. One of the questions surrounding reduction of CSBs is whether a child will compensate for those calories. Using nationally representative data for 2 days of diet in children, Wang et al. [54] estimated that replacing sweetened beverages with water would result in an average reduction of 235 cal/day.

Although most studies continue to target a single level (community, school, family, or individual), Gentile et al. [55] attempted a multi-ecologic prevention study at the family, community, and school levels in which the goals were to increase fruits and vegetables, decrease TV time, and increase physical activity. At the end of 6 months post-program, they demonstrated success in increasing fruits and vegetables and reducing screen time, but reported no effect on BMI or physical activity in the intervention group. However, 6 months may be too short a follow-up to expect a change in BMI; it is hoped that the investigators will reassess at a longer time point.

Conclusions

In summary, new research continues to add important details to our understanding of the prevention of obesity in children, including increased physical activity and improved nutrition. One of the most important clarifications is the role of maternal obesity—this influence may be the strongest predictor of all, at least partially because overweight mothers teach their children similar unhealthy habits. In this finding lies an opportunity. If we can educate and improve the health habits of young women before or near the time that they decide to bear children, we could shift the epidemic. Another central finding is that children who are high risk can be identified early by parent BMI. Because no simple answers are available and obesity prevention in high-risk children will require multilevel, multicomponent interventions, focusing on higher risk children may be a way to improve the cost-effectiveness of such programs. Finally, family-based interventions early in a child's life, before habits are set, are likely to be most effective.

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References

Papers of particular interest, published recently, have been highlighted as:
• Of importance
•• Of major importance

1. Deshmukh-Taskar P, Nicklas TA, Morales M, et al.: Tracking of overweight status from childhood to young adulthood: the Bogalusa Heart Study. *Eur J Clin Nutr* 2006, 60:48–57.
2. Ogden C, Carroll M, Curtin L, et al.: Prevalence of overweight and obesity in the United States, 1999–2004. *JAMA* 2006, 295:1549–1555.

3. ••Franks P, Hanson R, Knowler W, et al.: Childhood obesity, other cardiovascular risk factors and premature death. *N Engl J Med* 2010, 362:485–493. *This article reports the association between childhood obesity and premature death*.
4. Loos RJ, Bouchard C: FTO: the first gene contributing to common forms of human obesity. *Obes Rev* 2008, 9:246–250.
5. Cecil J, Tavendale R, Watt P, et al.: An obesity-associated FTO gene variant and increased energy intake in children. *N Engl J Med* 2008, 359:2558–2566.
6. Farooqi IS, Yeo GS, Keogh JM, et al.: Dominant and recessive inheritance of morbid obesity associated with melanocortin 4 receptor deficiency. *J Clin Invest* 2000, 106:271–279.
7. Loos RJ, Lindgren CM, Li S, et al.: Common variants near MC4R are associated with fat mass, weight and risk of obesity. *Nat Genet* 2008, 40:768–775.
8. Cole SA, Butte NF, Voruganti S, et al.: Evidence that multiple genetic variants of MC4R play a functional role in the regulation of energy expenditure and appetite in Hispanic children. *Am J Clin Nutr* 2010, 91:191–199.
9. Cauchi S, Stutzmann F, Cavalcanti-Proenca C, et al.: Combined effects of MC4R and FTO common genetic variants on obesity in European general populations. *J Mol Med* 2009, 87:537–546.
10. •Anzman SL, Birch LL. Low inhibitory control and restrictive feeding practices predict weight outcomes. *J Pediatr* 2009, 155:651–656. *This article demonstrated that low inhibitory control is associated with increased BMI in children and, importantly, that parenting practices can modify the risk associated with this trait*.
11. Seeyave D, Coleman S, Appugliese D, et al.: Ability to delay gratification at age 4 years and risk of overweight at age 11 years. *Arch Pediatr Adolesc Med* 2009, 163:303–308.
12. Francis LA, Susman E: Self-regulation and rapid weight gain in children from age 3 to 12 years. *Arch Pediatr Adolesc Med* 2009, 163:297–302.
13. Catalano PM, Farrell K, Thomas A, et al.: Perinatal risk factors for childhood obesity and metabolic dysregulation. *Am J Clin Nutr* 2009, 90:1303–1313.
14. Griffiths LJ, Hawkins SS, Cole TJ, Dezateux C: Risk factors for rapid weight gain in preschool children: findings from a UK-wide prospective study. *Int J Obes (Lond)* 2010, 34:624–632.
15. Hitze B, Bosy-Westphal A, Plachta-Danielzik S, et al.: Long-term effects of rapid weight gain in children, adolescents and young adults with appropriate birth weight for gestational age: the Kiel Obesity Prevention Study. *Acta Paediatr* 2010, 99:256–262.
16. ••Metzger MW, McDade TW: Breastfeeding as obesity prevention in the United States: a sibling difference model. *Am J Hum Biol* 2010, 22:291–296. *This article reports the effect of breastfeeding on BMI in a sibling study. This study decreases many of the confounders typically found in association studies and demonstrates a strong protective effect of breastfeeding*.
17. Hennessy E, Hughes SO, Goldberg JP, et al.: Parent behavior and child weight status among a diverse group of underserved rural families. *Appetite* 2010, 54:369–377.
18. Sisson SB, Church TS, Martin CK, et al.: Profiles of sedentary behavior in children and adolescents: the US National Health and Nutrition Examination Survey, 2001–2006. *Int J Pediatr Obes* 2009, 4:353–359.
19. Fulton JE, Wang X, Yore MM, et al.: Television viewing, computer use, and BMI among US children and adolescents. *J Phys Act Health* 2009, 6(Suppl 1):S28–S35.
20. He M, Harris S, Piche L, Beynon C: Understanding screen-related sedentary behavior and its contributing factors among school-aged children: a social-ecologic exploration. *Am J Health Promot* 2009, 23:299–308.
21. Zimmermann F, Bell J: Associations of television content type and obesity in children. *Am J Public Health* 2010, 100:334–340.
22. MacFarlane A, Cleland V, Crawford D, et al.: Longitudinal examination of the family food environment and weight status among children. *Int J Pediatr Obes* 2009, 4:343–352.
23. Mitchell JA, Mattocks C, Ness AR, et al.: Sedentary behavior and obesity in a large cohort of children. *Obesity (Silver Spring)* 2009, 17:1596–1602.
24. McGavock JM, Torrance BD, McGuire KA, et al.: Cardiorespiratory fitness and the risk of overweight in youth: the Healthy Hearts Longitudinal Study of Cardiometabolic Health. *Obesity (Silver Spring)* 2009, 17:1802–1807.
25. Roberts CK, Freed B, McCarthy WJ: Low aerobic fitness and obesity are associated with lower standardized test scores in children. *J Pediatr* 2010, 156:711–718.
26. Malik VS, Schulze MB, Hu FB: Intake of sugar-sweetened beverages and weight gain: a systematic review. *Am J Clin Nutr* 2006, 84:274–288.
27. Vos M, Kimmons J, Gillespie C, et al.: Dietary fructose consumption among US children and adults: the Third National Health and Nutrition Examination Survey. *Medscape J Med* 2008, 10:160.
28. Keller KL, Kirzner J, Pietrobelli A, et al.: Increased sweetened beverage intake is associated with reduced milk and calcium intake in 3- to 7-year-old children at multi-item laboratory lunches. *J Am Diet Assoc* 2009, 109:497–501.
29. Fiorito L, Marini MF, Francis LA, et al.: Beverage intake of girls at age 5 y predicts adiposity and weight status in childhood and adolescence. *Am J Clin Nutr* 2009, 90:935–942.
30. Lim S, Zoellner JM, Lee JM, et al.: Obesity and sugar-sweetened beverages in African-American preschool children: a longitudinal study. *Obesity (Silver Spring)* 2009, 17:1262–1268.
31. Nissinen K, Mikkila V, Mannisto S, et al.: Sweets and sugar-sweetened soft drink intake in childhood in relation to adult BMI and overweight. The Cardiovascular Risk in Young Finns Study. *Public Health Nutr* 2009, 12:2018–2026.
32. Gubbels JS, Kremers SP, Stafleu A, et al.: Clustering of dietary intake and sedentary behavior in 2-year-old children. *J Pediatr* 2009, 155:194–198.
33. Papas MA, Hurley KM, Quigg AM, et al.: Low-income, African American adolescent mothers and their toddlers exhibit similar dietary variety patterns. *J Nutr Educ Behav* 2009, 41:87–94.
34. Dodson EA, Fleming C, Boehmer TK, et al.: Preventing childhood obesity through state policy: qualitative assessment of enablers and barriers. *J Public Health Policy* 2009, 30(Suppl 1): S161–S176.
35. ••Khan LK, Sobush K, Keener D, et al.: Recommended community strategies and measurements to prevent obesity in the United States. *MMWR Recomm Rep* 2009, 58(RR-7):1–26. *This article provides an important summary of recommended community strategies and measurements to prevent obesity in the United States*.
36. US Department of Health and Human Services: The Surgeon General's Vision for a Healthy and Fit Nation. Rockville, MD: US Department of Health and Human Services, Office of the Surgeon General; 2010.
37. Hesketh KD, Campbell KJ: Interventions to prevent obesity in 0–5 year olds: an updated systematic review of the literature. *Obesity (Silver Spring)* 2010, 18(Suppl 1):S27–S35.
38. Gonzalez-Suarez C, Worley A, Grimmer-Somers K, Dones V: School-based interventions on childhood obesity: a meta-analysis. *Am J Prev Med* 2009, 37:418–427.
39. Marcus C, Nyberg G, Nordenfelt A, et al.: A 4-year, cluster-randomized, controlled childhood obesity prevention study: STOPP. *Int J Obes (Lond)* 2009, 33:408–417.
40. Walther C, Gaede L, Adams V, et al.: Effect of increased exercise in school children on physical fitness and endothelial progenitor

cells: a prospective randomized trial. *Circulation* 2009, 120:2251–2259.

41. Peralta LR, Jones RA, Okely AD: Promoting healthy lifestyles among adolescent boys: the Fitness Improvement and Lifestyle Awareness Program RCT. *Prev Med* 2009, 48:537–542.
42. Cullen KW, Watson KB, Konarik M: Differences in fruit and vegetable exposure and preferences among adolescents receiving free fruit and vegetable snacks at school. *Appetite* 2009, 52:740–744.
43. Power TG, Bindler RC, Goetz S, Daratha KB: Obesity prevention in early adolescence: student, parent, and teacher views. *J Sch Health* 80:13–19.
44. Dorsey KB, Mauldon M, Magraw R, et al.: Applying practice recommendations for the prevention and treatment of obesity in children and adolescents. *Clin Pediatr (Phila)* 49:137–145.
45. Spivack JG, Swietlik M, Alessandrini E, Faith MS: Primary care providers' knowledge, practices, and perceived barriers to the treatment and prevention of childhood obesity. *Obesity (Silver Spring)* 2009 (Epub ahead of print).
46. Woolford SJ, Clark SJ, Ahmed S, Davis MM: Feasibility and acceptability of a 1-page tool to help physicians assess and discuss obesity with parents of preschoolers. *Clin Pediatr (Phila)* 2009, 48:954–959.
47. Wake M, Baur LA, Gerner B, et al.: Outcomes and costs of primary care surveillance and intervention for overweight or obese children: the LEAP 2 randomised controlled trial. *BMJ* 2009, 339: b3308.
48. Gruber KJ, Haldeman LA: Using the family to combat childhood and adult obesity. *Prev Chronic Dis* 2009, 6:A106.
49. Sonneville KR, La Pelle N, Taveras EM, et al.: Economic and other barriers to adopting recommendations to prevent childhood obesity: results of a focus group study with parents. *BMC Pediatr* 2009, 9:81.
50. Taveras EM, Hohman KH, Price S, et al.: Televisions in the bedrooms of racial/ethnic minority children: how did they get there and how do we get them out? *Clin Pediatr (Phila)* 2009, 48:715–719.
51. Escobar-Chaves SL, Markham CM, Addy RC, et al.: The Fun Families Study: intervention to reduce children's TV viewing. *Obesity (Silver Spring)* 2010, 18(Suppl 1):S99–S101.
52. Eiholzer U, Meinhardt U, Petro R, et al.: High-intensity training increases spontaneous physical activity in children: a randomized controlled study. *J Pediatr* 156:242–246.
53. Ventura E, Davis J, Byrd-Williams C, et al.: Reduction in risk factors for type 2 diabetes mellitus in response to a low-sugar, high-fiber dietary intervention in overweight Latino adolescents. *Arch Pediatr Adolesc Med* 2009, 163:320–327.
54. Wang YC, Bleich SN, Gortmaker SL: Increasing caloric contribution from sugar-sweetened beverages and 100% fruit juices among US children and adolescents, 1988–2004. *Pediatrics* 2008, 121:e1604–e1614.
55. Gentile DA, Welk G, Eisenmann JC, et al.: Evaluation of a multiple ecological level child obesity prevention program: switch what you do, view, and chew. *BMC Med* 2009, 7:49.