

# Lifecourse Approach to Racial/Ethnic Disparities in Childhood Obesity<sup>1–3</sup>

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## ABSTRACT

Eliminating racial/ethnic disparities in health and health care is a national priority, and obesity is a prime target. During the last 30 y in the United States, the prevalence of obesity among children has dramatically increased, sparing no age group. Obesity in childhood is associated with adverse cardio-metabolic outcomes such as hypertension, hyperlipidemia, and type II diabetes and with other long-term adverse outcomes, including both physical and psychosocial consequences. By the preschool years, racial/ethnic disparities in obesity prevalence are already present, suggesting that disparities in childhood obesity prevalence have their origins in the earliest stages of life. Several risk factors during pregnancy are associated with increased risk of offspring obesity, including excessive maternal gestational weight gain, gestational diabetes, smoking during pregnancy, antenatal depression, and biological stress. During infancy and early childhood, rapid infant weight gain, infant feeding practices, sleep duration, child's diet, physical activity, and sedentary practices are associated with the development of obesity. Studies have found substantial racial/ethnic differences in many of these early life risk factors for childhood obesity. It is possible that racial/ethnic differences in early life risk factors for obesity might contribute to the high prevalence of obesity among minority preschool-age children and beyond. Understanding these differences may help inform the design of clinical and public health interventions and policies to reduce the prevalence of childhood obesity and eliminate disparities among racial/ethnic minority children. *Adv. Nutr.* 3: 73–82, 2012.

## Introduction

In the past 3 decades, rates of overweight and obesity among adults and children have substantially increased worldwide with all but the poorest countries now struggling with a growing obesity problem (1). In the United States alone, the prevalence of overweight and obesity in school-aged children and adolescents approaches one-third (2–4). The epidemic has not spared even our youngest children (5,6). Even though U.S. childhood obesity rates in some population subgroups, such as whites and those of higher

socioeconomic status (SES), may have peaked, overall rates remain stubbornly high and racial/ethnic disparities appear to be widening (7–9).

The lifecourse approach to chronic disease prevention posits that factors may act in the prenatal period into infancy, childhood, and beyond to determine risk of chronic disease (10). These factors can range from the social/built environment (macro) through behavior, physiology, and genetics (micro). Factors interact with each other over the lifecourse, with different determinants being more or less important at different life stages. When risk factors have much more influence at a particular life stage than either before or after, it is called a sensitive period (10).

Today, we recognize that the prenatal, infancy, and early childhood periods are likely to be key to the development and thus prevention of obesity and its consequences in children. In addition, given the high prevalence of obesity among racial/ethnic minority children < 6 y of age, the lifecourse approach could provide a framework both for understanding the etiology of early differences in risk factors and disparities in prevalence and for intervening during these periods to eliminate racial/ethnic disparities.

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In the subsequent sections we review the prevalence of childhood obesity and its comorbidities, summarize evidence on racial/ethnic differences in early life risk factors for obesity, and discuss potential approaches for reducing early childhood obesity and eliminating racial/ethnic disparities in obesity.

## Methods

We conducted a systematic review of the published literature on childhood obesity prevalence; obesity risk factors in pregnancy, infancy, and childhood; and racial/ethnic disparities in these areas. We also conducted a review of current childhood obesity prevention efforts and efforts to eliminate racial/ethnic disparities in the United States. Databases searched included MEDLINE/Pub Med, Academic Search Premier, RePort, ERIC, the Cochrane Database of Systematic Reviews, and the Cochrane Register of Controlled Trials. We also reviewed reference lists of included papers and other relevant reviews and meta-analyses.

## Current status of knowledge

### Childhood obesity is prevalent, of consequence, and has its origins in the earliest stages of life

In the US, ~32% of children ages 2–19 y are overweight (age- and sex-specific BMI 85th to 94th percentile) or obese (BMI  $\geq$  95th percentile) (2). Even among preschool-age children 2–5 y of age, studies also suggest a high prevalence of obesity. The NHANES 2007–2008 reported an obesity prevalence of 10.4% in the 2- to 5-y-old group. Furthermore, a 2008 report by the CDC Pediatric Nutrition Surveillance System showed an obesity prevalence rate of 14.6% among low-income, preschool-aged children (ages 2–4 y) (11). Obesity in children is associated with both short- and long-term adverse outcomes, including both physical and psycho-social consequences (12–16) and even perhaps shortened lifespan (17). Children who are overweight tend to become overweight adults and, once present, obesity is notoriously hard to treat (18–20). The obesity epidemic has spared no age group, including young children. Recent data show that even infants have experienced a dramatic rise in excess weight in the past 20 y, implying that the roots of the epidemic can be found as early as infancy, and before (21). Taken together, these facts indicate that finding new etiologic clues, starting at the very earliest stages of human development, is potentially crucial to stemming the rising tide of obesity.

### Substantial racial/ethnic disparities exist in childhood obesity and its related comorbidities

Racial/ethnic minority children bear a disproportionate share of the burden of obesity and its related comorbidities (22). According to the most recent NHANES 2007–2008, the prevalence of children ages 2–19 y having a BMI  $\geq$  95th percentile was 15.3% among non-Hispanic white children, 20.0% among non-Hispanic black children, and 20.8% among Mexican American children (2). By the preschool years, racial/ethnic disparities in obesity prevalence are already present and are particularly evident among non-Hispanic black girls and Mexican American boys (3). According to a 2008 survey of low-income, preschool-aged

children by the CDC Pediatric Nutrition Surveillance System, obesity prevalence was highest among American Indian/Alaska Native (21.2%) and Hispanic (18.5%) children and lowest among non-Hispanic white (12.6%), non-Hispanic black (11.8%), and Asian/Pacific Islander (12.3%) children (11).

The degree of obesity also differs among racial/ethnic minority children. Compared to non-Hispanic white children, non-Hispanic black girls and Hispanic boys had almost 2-fold greater odds of being severely obese (BMI  $\geq$  97th percentile) in 2007–2008 (2). In another study examining severe obesity from 1976 to 2006, Wang et al. (7) found that the prevalence of severe obesity in the 2- to 19-y age range was much higher in non-Hispanic black girls (9.1%) compared to non-Hispanic white girls (3.5%) and Hispanic girls (5.1%). Similarly, the age-adjusted prevalence of severe obesity was higher in non-Hispanic black boys (7.1%) and Hispanic boys (6.9%) compared to non-Hispanic white boys (4.0%).

The adverse sequelae of obesity are also more common among racial/ethnic minority children. Obesity in childhood is a significant risk factor for obesity in adulthood. According to results from the Bogalusa Heart Study, obese black children are much more likely than their white counterparts to remain obese as adults (23,24). The epidemiologic shift of obesity to younger ages is also expected to have a profound impact on rates of type 2 diabetes in young adults (25). Evidence from the SEARCH for Diabetes in Youth Study, a multicenter population-based study, has shown higher incidence rates of type 2 diabetes compared with type 1 diabetes mellitus among African American, Hispanic, Asian American, and American Indian adolescents (26). The proportion of all diabetes that was diagnosed as type 2 among 10–19 y olds were: 6% for non-Hispanic whites, 22% for Hispanics, 33% for African Americans, 40% for Asian/Pacific Islanders, and 76% for American Indians. Among children aged 8–17 y in the NHANES 1999–2000, blood pressure levels were higher among African Americans and Mexican Americans compared with non-Hispanic whites. These differences were reduced or eliminated after adjustment for BMI, suggesting that higher adiposity accounted for much of the elevated blood pressure (27). Racial/ethnic differences also exist for fatty liver related to obesity. In a study of obese children ages 2–19 y, fatty liver disease was present in 50% of Hispanics, 35% of whites, and 10% of blacks (28).

In sum, existing evidence overwhelmingly suggests that racial/ethnic disparities exist in childhood obesity and its related sequelae. The consistency of these disparities across the spectrum of obesity-related sequelae underscores the importance of identifying the underlying causes of the disparities.

### Established early life risk factors for childhood obesity

#### *Prepregnancy and pregnancy risk factors.*

Maternal obesity is one of the strongest and most reliable predictors of later obesity in children (18). Maternal overweight or obesity entering pregnancy is also a risk factor for childhood

obesity. Infants born to overweight mothers are more likely to be born large for gestational age, are less likely to be breastfed, and are at higher risk for obesity and type 2 diabetes in later life (29–36).

Gestational weight gain (GWG) is an important and independent predictor of child weight and health. Higher GWG is associated with greater birth weight for gestational age, which predicts offspring weight and risk of overweight in later life (37). Independent of its relationship with fetal growth, we and others have found that even among overweight mothers, higher GWG is a strong risk factor for child obesity and its sequelae (38–41).

Children born to mothers with impaired glucose tolerance during pregnancy are more likely to be macrosomic and have higher body fat at birth and subsequently may be at elevated risk for becoming overweight and developing related complications such as higher blood pressure and risk for type 2 diabetes (42–49). The link between a mother's glucose tolerance during pregnancy and her child's weight and glucose tolerance appears not only to result from shared genes and behaviors but also from a direct influence of the adverse intrauterine environment on the fetus (43,50,51).

A recent meta-analysis of 14 studies showed that smoking during pregnancy is associated with a 50% increased odds of obesity during childhood (52). We previously reported that maternal smoking during early pregnancy is associated with elevated risk for having a BMI that exceeds the 85th percentile for age and sex, higher BMI Z-score, and higher systolic blood pressure at age 3 y (53).

Finally, previous studies found that maternal stressors, such as depression, are associated with childhood obesity (54,55). Other characteristics and behaviors during pregnancy, including diet and diet quality, may influence weight gain and obesity among offspring, but these factors remain understudied.

**Infancy risk factors.** In the first year of life, the primary determinants of later obesity appear to be rapid weight gain and type and duration of infant feeding. Both fetal growth and rapid, early, infancy weight gain are associated with later BMI in childhood or adulthood (56–59). Furthermore, 2 meta-analyses (60,61) found that breastfeeding is associated with a 10–20% reduction in obesity prevalence in childhood or adulthood, with duration showing a dose-response relationship (62). Previous studies also found that introduction of solids at < 4 mo old is a risk factor for increased infant weight gain (63–65).

Children naturally regulate their intake, but parents' feeding behaviors may override children's internal appetite signals. Birch et al. (66) reported that overly restrictive feeding behaviors are associated with overeating and poorer self-regulation of energy intake in preschool-aged children and, among girls, increased adiposity. It is possible that, compared with parents who bottle-feed, mothers who breastfeed may be more responsive to the infant's signals for frequency and volume of feedings (67). In some studies, mothers' pressure on their children to eat in the absence of hunger has

also been associated with disinhibited eating (68), increased child energy intake and body weight (69).

An inverse association between sleep duration and obesity has been observed in cross-sectional studies of children and adolescents (70–73). Taveras et al. (74) recently reported that infant sleep of <12 h/d was associated with a 2-fold increased odds of obesity at age 3 y. This association has been observed in infants as young as 6 mo old. Tikotzky et al. (75) found that short nocturnal sleep was associated with increased weight and weight:length ratio in 6-mo-old infants. Finally, early child care attendance has been shown to be a risk factor for greater weight gain in the first year of life (63).

**Risk factors in early childhood through preschool age.** After the first year of life, several risk factors emerge that have been found to be associated with later obesity. Among school-aged children, there is strong evidence from both observational (76–78) and experimental (79,80) research that television viewing is positively associated with risk of overweight. In preschool-aged children, Dennison et al. (81) found that the number of hours a child spends watching TV is associated with increased risk of obesity. Additionally, Lumeng et al. (82) found that exposure of  $\geq 2$  h/d of TV was positively associated with an increased risk of overweight at ages 36 and 54 mo. Having a TV in the child's bedroom is also associated with greater amounts of time watching TV and with increased risk of overweight (81). Additionally, while watching TV, children are exposed to substantial amounts of advertisements for food products. Two- to seven-year-old children view an average of 2:23 min of food advertising per hour (83). In a study of 2- to 6-year-old children, children who watched a cartoon with commercials were more likely to prefer the products advertised than children who watched the same cartoon but were not exposed to commercials (84). These findings suggest that food advertising can affect children's food preferences and could contribute to greater energy intake and increase obesity risk.

Short sleep duration persists as a potential risk factor for obesity in childhood as well. Accumulating evidence links short sleep with increased risk of obesity in children, with short sleep duration and late bedtime showing a dose-response relationship with increased odds of obesity (71). Evidence suggests that sleep insufficiency in early childhood may be associated with a long-term risk of obesity in later years. Insufficient nighttime sleep in children 0–4 y old has been associated with increased odds of overweight (vs. normal weight) and obesity (vs. overweight) 5 y later (85). In a population-based birth cohort study, Landhuis et al. (86) found that the mean childhood sleep duration from ages 5 to 11 y was inversely associated with risk of adult obesity at 32 y of age.

Sugar-sweetened beverage consumption has increased rapidly among children. Between 1977 and 2001, consumption of sugar-sweetened beverages increased from 3.0 to 6.9% among children ages 2–18 y in the US (87). Children start drinking sugar-sweetened beverages at a remarkably young age and consumption increases through young

adulthood. Among school-aged children, intake of sugar-sweetened beverages has been associated with greater caloric intake (88) and increased BMI (89). Results from both cross-sectional and prospective studies suggest that intake of sweetened beverages may be associated with increased risk of obesity in preschool-aged children (90–92).

Over the last 30 y, consumption of fast food has paralleled the rise in childhood obesity prevalence (93–95). Consumption of fast food by children increased 5-fold from 2% of total energy in the late 1970s to 10% of total energy in the mid-1990s (94). Greater consumption of fast food has been found to be associated with poorer diet quality among children (96) and is usually accompanied by other unhealthy dietary practices like large portion sizes (93) and high energy density, which are known to contribute to body weight (97).

Finally, regular participation in physical activity has multiple health benefits for children (98). Evidence suggests that physical activity has a substantial influence on BMI (99) and that children who are active early in life have a reduced risk for large adiposity gains in elementary school through adolescence (100–102).

Parents play a key role in childhood obesity prevention interventions. Several studies have examined the effects of particular household routines on the prevalence of childhood overweight and obesity. For example, fewer family meals and increased TV viewing have been associated with an increased risk for childhood overweight (103). In a cross-sectional study of 8550 4-y-old children, Anderson et al. (104) found 3 household routines, e.g. eating family dinners, getting sufficient nocturnal sleep, and limiting screen time, as protective of children's risk for obesity.

### **Racial/ethnic and socioeconomic differences in early life risk factors for childhood obesity**

Previous studies have examined racial/ethnic differences in pregnancy-related risk factors for childhood obesity (105–108). In a national study by Chu et al. (105), black and Hispanic women were more likely than white women to begin their pregnancies already overweight or obese and gained less weight during pregnancy. One study (107) found that Hispanic women have a higher risk of gestational diabetes. Few studies have examined racial/ethnic differences in smoking during pregnancy (108) and in maternal antenatal depression (106).

A recent study by Taveras et al. (109) showed that racial/ethnic differences in risk factors for obesity exist prenatally and in early childhood. In this study, children from underrepresented racial/ethnic groups were found to have a higher risk of various obesity risk factors compared to their white counterparts. Specifically, black and Hispanic children had higher odds of antenatal depression, weight gain during infancy, introduction of solid foods before 4 mo of age, maternal restrictive feeding, television in their bedrooms after age 2 y, and intake of sugar-sweetened beverages and fast food. Conversely, black and Hispanic children had lower odds of protective factors, including exclusive breastfeeding and sleep for >12 h/d during infancy. These findings may help

to explain racial/ethnic disparities in early childhood obesity rates while justifying the need for early childhood interventions to reduce these disparities in obesity prevalence.

Several studies of older children have found obesity-related risk factors to be more prevalent among racial/ethnic minority youth. Previous studies have found higher levels of TV viewing and more televisions in bedrooms, higher consumption of sugar-sweetened beverages (110), increased fast food consumption (111), and lower levels of physical activity among black and Hispanic youth compared to white youth (112,113).

### **What is the role of SES in explaining racial/ethnic differences in obesity and obesity-related risk factors?**

SES is a multidimensional construct that is known to exert a profound influence on health (114). The relationship between SES, usually measured as parental income or education, and childhood obesity is complex (28). Although several U.S. studies of the relationship between SES and childhood obesity have shown that low SES and minority groups have a higher prevalence of obesity, more recent data have shown some conflicting results (115–119). A recent study by Wang et al. (116) of secular trends in the relationship between SES and obesity did not find consistent associations between SES and overweight among black and Mexican American children. Among white children, a reverse association was observed between SES and obesity prevalence. Furthermore, the study by Wang et al. (116) showed that although the relationship between SES and obesity has weakened over time, the gap between ethnic groups has become wider.

The exact role of SES in the relationship between race/ethnicity and obesity is unclear. Singh et al. (120) found that the magnitude of the effects of SES on obesity among 10–17 y olds varied by race/ethnicity. For example, the effects of SES on the odds of obesity were larger for Hispanics than non-Hispanic whites and blacks. Additionally, after adjusting for maternal education, household income, and children's food security status, Whitaker et al. (121) found that the increased odds of obesity risk among Hispanic 3-y-old children did not change substantially, suggesting that the increased prevalence of obesity in Hispanics compared to blacks or whites was not explained by racial/ethnic differences in socioeconomic indicators. Taveras et al. (109) recently found that socioeconomic factors did not explain most racial/ethnic differences in prenatal and early childhood risk factors for obesity. In this study, socioeconomic factors markedly attenuated (>30%) racial/ethnic differences in smoking during pregnancy, maternal depression, breast feeding initiation and duration, and having a TV where the child sleeps. However, adjusting for SES did not completely eliminate observed racial/ethnic differences in obesity risk factors. Thus, existing evidence does not suggest that differences in SES primarily explain racial/ethnic disparities in obesity.

Caprio et al. (28) have discussed the limited nature of using household income and parental education as markers of

SES, because such variables may not fully capture the child's access to resources or the family's socioeconomic position. Braveman et al. (122) found that a given income and education level may not reflect the same amount of wealth across all races/ethnicities. For example, they found that compared to African Americans in the lowest income quintile, whites in the same income quintile had >400 times as many resources. Environmental factors can also contribute to SES. Williams et al. (123) suggested that residential segregation may be responsible for disparities in SES and health. A review by Lovasi et al. (124) suggested that increased access to supermarkets and exercise facilities and better safety perceptions may help foster better diets and physical activity frequency among low-income, black, and Hispanic populations, targeting obesity disparities. However, it was noted that the association between built environment components and obesity prevalence has not been consistent in the literature. Such studies demonstrate that other aspects of socioeconomic position should be examined to better understand the role of SES in racial/ethnic disparities in obesity prevalence.

### **Contribution of culture and acculturation to racial/ethnic differences in obesity risk factors**

Given that SES may not fully explain racial/ethnic differences in obesity prevalence, increased attention has been given to culture and acculturation as underlying factors that may contribute to racial/ethnic disparities in obesity. Accumulating evidence has shown a change in health status with acculturation and more time spent in the US. In particular, studies have shown changes in traditional diet components across generations. Allen et al. (125) found that across generations, Latinos' diets have transitioned from better to worse than whites, with Latinos consuming fewer fruits and vegetables and drinking more soda across generations. McArthur et al. (126) showed that in Hispanic immigrant households, high-fat and -sugar foods, not in the traditional Hispanic diet, were regularly available for children, suggesting that children may have experienced transitions in food preferences. Furthermore, Popkin et al. (127) showed that US-born Asian American and Hispanic adolescents were more than 2 times as likely to be obese compared to foreign-born adolescents. Immigration and nativity status may also interact with other socioeconomic factors. For example, Balistreri et al. (128) found that income was inversely associated with BMI among kindergarten children of US-born Hispanic and white parents. On the other hand, income was positively associated with BMI among foreign-born Hispanic families. Cultural differences among immigrants may explain the different associations and interactions.

Culture may also play a role in shaping parental perceptions of their children's health status. Mothers may have different preferences for what they consider a "healthy" child. In some cultures, mothers may view thinness as a reflection of poor health and malnutrition. For example, some evidence suggests that Hispanic mothers may perceive heavier

children as healthier children (129–131). Thus, they may encourage their children to eat more. A qualitative study also showed that Hispanic mothers may perceive a child who is "not hungry" as worrisome (132). At the same time, some parents may not perceive their children as obese. In a study including overweight to very obese 5- to 10-y-old African American children, Young-Hyman et al. (133) found that even though 90% of boys and 80% of girls were obese or very obese, only 30% of their parents classified their child as being very overweight. Such culturally defined perceptions of body image may influence parenting strategies and decisions regarding eating habits, affecting the amount of food children eat and their risk of overweight and obesity.

### **Opportunities for reducing childhood obesity prevalence in the US**

From a lifecourse perspective, the pregnancy and early childhood periods may provide a critical period and unique opportunity for preventing childhood obesity. Pregnancy and early childhood represent periods of maximal environmental dependence and parental care and a developmentally plastic period during which behaviors are modifiable and the physiologic signals among adipocytes, the brain, and other organs are being set (134). Early childhood seems particularly promising and highly sensitive to interventions; there are multiple settings to access parents (e.g. primary care, child care, early education settings, etc.), and parents and caregivers are highly sensitized to children's needs. Habits and tastes develop early in children and thus establishing the tastes for a variety of foods, enjoying active play, developing motor skills, and good sleep habits are all critical for future healthy behavioral patterns.

In recognition of the growing evidence of the importance of assessing the beginnings of obesity and instituting preventive measures in the early years, the Institute of Medicine's Standing Committee on Childhood Obesity Prevention (135) recently published a report examining strategies to prevent overweight and obesity from birth to age 5 y, with a focus on nutrition, physical activity, and sedentary behavior, and made policy recommendations on obesity prevention in the first 5 y. The goals, recommendations, and potential actions for implementation were in the areas of growth monitoring and assessing risk, physical activity, healthy eating, food marketing and screen time, and sleep. The recommendations provide examples of potential intervention targets for prevention policies across a range of settings and types of programs that can influence the environments in which young children develop and grow.

Evidence for effective obesity prevention interventions is increasing rapidly, with the most promising approaches to date being changes in environments and policies aimed at young children (136). Obesity and intervention experts have advocated for the development and testing of multi-level, multi-sector prevention strategies that invoke change at the environment level (137). Recent results from whole-of-community interventions, such as Shape Up Somerville (138) in the US and Romp and Chomp (139) in Australia,

provide further evidence that multi-level interventions can be effective in obesity prevention among children. Few interventions have targeted children < 5 y of age (140,141) and questions remain regarding sources of policy resistance, strategies to overcome such resistance, and the most effective levers of sustainable change in environmental-level interventions (142).

A recent national effort to prevent obesity is the *Let's Move!* campaign. In 2010, First Lady Michelle Obama helped launch the *Let's Move!* campaign with the goal of reducing childhood obesity and establishing a healthier generation of youth by involving participants at multiple levels. *Let's Move!* encompasses 5 pillars: early childhood obesity prevention; parent and caregiver empowerment; healthier food in schools; access to healthy, affordable foods; and increased physical activity (143). The early childhood component of *Let's Move!* outlines specific recommendations for pregnant women and those involved in the care of young children. Women are advised to achieve a healthy prenatal weight and adequate GWG in addition to seeking prenatal care. The early childhood component also promotes breastfeeding due to its association with lower rates of obesity in children. The initiative supports research of environmental chemicals that may promote weight gain or obesity in children. *Let's Move!* discourages television and media exposure during early childhood in favor of increased physical activity. Lastly, the campaign calls for interventions in child care settings that are in alignment with the initiative's overarching goals of fitness and nutrition. The guidelines of the *Let's Move!* campaign strive to ignite a national movement in the fight against childhood obesity.

In parallel with the *Let's Move!* campaign and childhood obesity prevention set as a national priority, President Barack Obama created the White House Task Force on Childhood Obesity in February 2010. The task force was charged to develop an action plan (144), including recommendations and concrete goals to eliminate the childhood obesity epidemic within the next generation. The 70 recommendations, which are based on the latest research, focus on the same pillars as the *Let's Move!* public awareness efforts and both efforts strive to jointly engage society to immediately take action against the obesity epidemic.

### Approaches to eliminating racial/ethnic disparities in obesity prevalence

Substantial evidence suggests that interventions to modify early life risk factors for obesity and use of the lifecourse approach as a conceptual framework may have substantial impact on reducing disparities in childhood obesity prevalence and its related comorbidities. To date, however, few interventions have been developed to target the prenatal, infancy, and early childhood periods in an attempt to prevent obesity. Although more studies are needed to understand the underlying reasons for the development of these racial/ethnic disparities in obesity prevalence, existing evidence provides strong rationale for testing comprehensive interventions in early life to reduce disparities in obesity prevalence.

Another approach to addressing racial/ethnic disparities in obesity prevalence is to apply biomedical science and technology to evidence-based medicine, public policy, and social programs. Dankwa-Mullan et al. (145) described the need for a paradigm shift in health disparities science to incorporate translational, transformational, and trans-disciplinary approaches to health disparities research. In this way, pioneering research could more efficiently lead from etiology and detection of differences and disparities to intervention studies. With this proposed paradigm shift and research triad, the field of health of disparities science may be able to better accommodate new findings and implement interventions more efficiently. In accordance with the new paradigm shift, the NIH held a summit, titled "The Science of Eliminating Health Disparities," in December 2008. The purpose of the summit was to assess national and global efforts in eliminating health disparities and provide a framework for future endeavors (146). The conclusions of the summit were that health disparities research: 1) should focus on social determinants of health; 2) needs to be collaborative and promote community engagement; 3) should promote effective and sustainable partnership models; 4) should promote infrastructure and capacity building for health disparities research; and 5) should consider media outreach and communication as an essential component of dissemination of the research findings. The NIH summit recommendations provide a framework for bridging science, practice, and policy in the health disparities field and could be an important model for guiding obesity prevention among racial/ethnic minority populations.

### Conclusions

Effective interventions for addressing childhood obesity and eliminating racial/ethnic disparities will require multi-level, multi-sector strategies, especially those that invoke change at the social environment level and draw lessons from a lifecourse understanding of the etiology of obesity. In addition, to eliminate racial/ethnic disparities there is an important need to identify effective levers of sustainable change and a move toward paradigm-shifting research and interventions that integrate translational, transformational, and trans-disciplinary thinking and approaches.

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