Original Investigation

Reducing Racial/Ethnic Disparities in Childhood Obesity The Role of Early Life Risk Factors

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IMPORTANCE Many early life risk factors for childhood obesity are more prevalent among blacks and Hispanics than among whites and may explain the higher prevalence of obesity among racial/ethnic minority children.

OBJECTIVE To examine the extent to which racial/ethnic disparities in adiposity and overweight are explained by differences in risk factors during pregnancy (gestational diabetes and depression), infancy (rapid infant weight gain, feeding other than exclusive breastfeeding, and early introduction of solid foods), and early childhood (sleeping <12 h/d, presence of a television set in the room where the child sleeps, and any intake of sugar-sweetened beverages or fast food).

DESIGN Prospective prebirth cohort study.

SETTING Multisite group practice in Massachusetts.

PARTICIPANTS Participants included 1116 mother-child pairs (63% white, 17% black, and 4% Hispanic)

EXPOSURE Mother's report of child's race/ethnicity.

MAIN OUTCOMES AND MEASURES Age- and sex-specific body mass index (BMI) *z* score, total fat mass index from dual-energy x-ray absorptiometry, and overweight or obesity, defined as a BMI in the 85th percentile or higher at age 7 years.

RESULTS Black (0.48 U [95% CI, 0.31 to 0.64]) and Hispanic (0.43 [0.12 to 0.74]) children had higher BMI *z* scores, as well as higher total fat mass index and overweight/obesity prevalence, than white children. After adjustment for socioeconomic confounders and parental BMI, differences in BMI *z* score were attenuated for black and Hispanic children (0.22 U [0.05 to 0.40] and 0.22 U [-0.08 to 0.52], respectively). Adjustment for pregnancy risk factors did not substantially change these estimates. However, after further adjustment for infancy and childhood risk factors, we observed only minimal differences in BMI *z* scores between whites, blacks (0.07 U [-0.11 to 0.26]), and Hispanics (0.04 U [-0.27 to 0.35]). We observed similar attenuation of racial/ethnic differences in adiposity and prevalence of overweight or obesity.

CONCLUSIONS AND RELEVANCE Racial/ethnic disparities in childhood adiposity and obesity are determined by factors operating in infancy and early childhood. Efforts to reduce obesity disparities should focus on preventing early life risk factors.

JAMA Pediatr. 2013;167(8):731-738. doi:10.1001/jamapediatrics.2013.85 Published online June 3, 2013.

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Corresponding Author: Elsie M. Taveras, MD, MPH, Division of General Pediatrics, MassGeneral Hospital for Children, 100 Cambridge St, 15th Floor, Boston, MA 02114 (etaveras@partners.org). n the past 3 decades, rates of overweight and obesity among children have substantially increased worldwide, with all but the poorest countries now struggling with a high prevalence of obesity and its related noncommunicable diseases.¹ In the United States alone, the prevalence of overweight and obesity in children and adolescents is 32%.²⁻⁴ Although childhood overweight and obesity rates may have plateaued in some US population subgroups, such as non-Hispanic whites and those of higher socioeconomic status, overall rates remain high, and racial/ethnic and socioeconomic disparities seem to be widening.⁵⁻⁸

There is growing evidence that risk factors in the prenatal period and early childhood are crucial to the development and thus prevention—of obesity and its consequences. Epidemiologic studies suggest that adverse exposures, such as intrauterine exposure to maternal smoking, excessive weight gain, or elevated glucose levels, and, in early childhood, rapid infant weight gain, poor feeding practices, too much television viewing, and short sleep duration, may increase short- and long-term risks for obesity and its sequelae.⁹⁻²⁶ For these reasons, the 2010 White House Task Force Report, *Solving the Problem of Childhood Obesity Within a Generation*,²⁷ and 2 Institute of Medicine reports^{28,29} emphasize the role of early life risk factors in obesity development and the need for interventions in early life to prevent obesity.

Research has demonstrated racial/ethnic and socioeconomic disparities across most known risk factors for childhood obesity from the prenatal period through childhood. In a study reported elsewhere,³⁰ we found that children from racial/ethnic minority groups had a higher prevalence of several early life risk factors for obesity than did their white counterparts. Among blacks and Hispanics, these differences included higher rates of maternal depression during pregnancy. Differences during infancy included more rapid infant weight gain, lower rates of exclusive breastfeeding, fewer infants sleeping at least 12 h/d, and more infants receiving solid foods before 4 months of age, and differences after age 2 years included a higher prevalence of a television set in the room where the child sleeps and higher intake of sugarsweetened beverages or fast food. These differences may very well explain the observed racial/ethnic disparities in elevated adiposity and prevalence of overweight and obesity as children grow up, but, to our knowledge, no study has yet explored this hypothesis.

The purpose of this study was to examine the extent to which racial/ethnic disparities in childhood adiposity and overweight and obesity among a cohort of school-age children are explained by differences in pregnancy, infancy, and early childhood risk factors.

Methods

Subjects and Study Design

Study subjects were participants in Project Viva, a prospective, prebirth cohort study that recruited women during early pregnancy from Harvard Vanguard Medical Associates, a multispecialty group practice in eastern Massachusetts.³¹ Details of recruitment and retention procedures are available elsewhere.³¹ Of the 2128 women who delivered a live newborn, 1579 were eligible for 7-year follow-up, of whom 1116 attended a 7-year in-person visit.

After obtaining informed consent, we performed inperson study visits with mothers at the end of the first and second trimesters of pregnancy and with mothers and children in the first few days after delivery and at 6 months, 3 years, and 7 years after birth. Mothers completed mailed questionnaires 1, 2, 4, 5, and 6 years after birth. Institutional review boards of Harvard Pilgrim Health Care, Brigham and Women's Hospital, and Beth Israel Deaconess Medical Center approved the study protocols.

Measurements

Main Exposure

At the 3-year interview, research assistants asked mothers the question, "Which of the following best describes your child's race or ethnicity?" Mothers had a choice of 1 or more of the following racial/ethnic groups: Hispanic or Latino, white or Caucasian, black or African American, Asian or Pacific Islander, American Indian or Alaskan Native, and other (specification was requested). For the participants who chose "other," we compared the specified responses to US census definitions³² for the other 5 race/ethnicities and reclassified the children where appropriate.

Outcome Measures

We measured children's height and weight using a calibrated stadiometer (Shorr Productions) and scale (Seca model 881; Seca Corp). We calculated age- and sex-specific body mass index (BMI) z scores using US national reference data.33 We defined overweight or obesity as a BMI in the 85th percentile or higher for age and sex.³³ In children at age 7 years, we also measured total fat with dual-energy x-ray absorptiometry and calculated the fat mass index (in kilograms per square meter), waist circumference (measured in centimeters with a Lufkin woven tape), and the sum of subscapular and triceps skinfold thickness measurements (obtained with Holtain calipers [Holtain Ltd]). Research assistants performing all measurements followed standardized techniques³⁴ and participated in in-service training to ensure measurement validity (Shorr Productions). Interrater and intrarater measurement error were well within published reference ranges for all measurements.35

Intermediate Variables: Early Life Risk Factors

The main intermediate variables were risk factors during pregnancy, infancy, and early childhood that are associated with childhood obesity in the medical literature and were significantly more prevalent among black and Hispanic children than their white counterparts in our previous Project Viva analysis of obesity-related risk factors.³⁰

In pregnancy, these risk factors included (1) gestational diabetes, defined as 2 or more abnormal fasting glucose tolerance test results at 26 to 28 weeks of gestation based on published criteria,³⁶ and (2) maternal depression in the second trimester of gestation, defined as a score of 13 or more on the Edinburgh Postnatal Depression Scale.³⁷ We did not include gestational weight gain or smoking during pregnancy because our earlier study did not show racial/ethnic differences in either after adjustment for maternal socioeconomic characteristics.³⁰

Risk factors during infancy included (1) rapid infant weight gain, defined as being in the highest quartile of change in weight-for-age *z* score between birth and age 6 months based on US reference data³³; (2) early introduction of solid foods, defined as introduction of complementary foods before 4 months of age; and (3) feeding other than exclusive breastfeeding, defined as mixed breastfeeding and formula feeding, weaned from breastfeeding, or formula feeding only in the first 6 months of life.

Early childhood risk factors (age 1-3 years) included (1) insufficient sleep, defined as less than 12 h/d of sleep from age 6 months to 2 years⁹; (2) any intake of sugar-sweetened beverages at age 2 years, defined as intake of soda (other than sugar-free soda), flavored milks, and fruit drinks (eg, Hi-C, Kool-Aid, and lemonade)³⁸; (3) any intake of fast food³⁹ at age 3 years; and (4) the presence of a television set in the room where the child sleeps at age 4 years, according to the mother's report.

Confounding Factors

We also collected information about maternal age, educational level, parity, and household income. Mothers reported their prepregnancy weight and height as well as the father's weight and height, from which we calculated maternal and paternal BMIs.

Statistical Analysis

We first examined bivariate relationships between child race/ ethnicity and each risk factor, covariate, and anthropometric outcome. We then used multivariable linear or logistic regression models to examine the association between the child's race/ethnicity and our anthropometric outcomes, with and without the inclusion of confounding factors or early life risk factors. Our first model, model 1, was adjusted for child age and sex only. We then also adjusted the multivariable models for potential confounders, including socioeconomic variables (model 2) and maternal and paternal BMI (model 3). In subsequent models, we adjusted for risk factors during pregnancy (gestational diabetes and depression; model 4), infancy (rapid infant weight gain, feeding other than exclusive breastfeeding, and early introduction of solid foods; model 5), and early childhood (sleeping <12 h/d, presence of a television set in the room where the child sleeps, and any intake of sugar-sweetened beverages or fast food; model 6). Each subsequent model includes adjustments in the prior model, with further adjustments.

The confounding and intermediate variables were not observed in all subjects. Using only subjects with all data observed would have resulted in a smaller sample, and most subjects lost to analysis would have been missing only 1 or 2 values. Reducing the sample in this manner loses information and can produce a selected subset. We therefore used multiple imputation to include the information known about subjects with missing values.^{40,41}

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In the multiple-imputation process, a model is used to generate or "impute" a plausible value that might have been observed for each missing datum; this process incorporates known variability in the data. A "completed" data set comprises the observed data and 1 imputed value for each missing datum. The analysis is then performed with this completed data set. Next, these initial imputed values are discarded, and a new set of imputed values (ie, a new completed data set) is generated, and the analysis is performed in the new completed data set. After several repetitions of this process, the analysis results—here, parameter estimates—are then combined in a structured fashion that accurately reflects the true amount of information in the observed data.

The advantage of this process is that it does not erroneously presume that any of the imputed values are known true values. Instead, the information in partially observed subjects is recovered rather than being discarded, as is typically done. Theory and experience with multiple imputation show that the results can be unbiased and confidence intervals narrower, and avoiding the selection bias implied by using only complete cases can meaningfully change parameter estimates.⁴² We generated 50 complete data sets and combined multivariable modeling results with SAS software, version 9.3 (Proc MIANALYZE software; SAS Institute, Inc).

From these multiple imputation results, we report adjusted differences estimated from regression models and 95% CIs for black and Hispanic children compared with non-Hispanic white children as the reference group.

Results

Consistent with national statistics, we found that black and Hispanic children, compared with non-Hispanic white children, had substantially higher BMI z scores, total fat mass index at dual-energy x-ray absorptiometry, and prevalence of overweight and obesity at age 7 years (Table 1). Waist circumference and the sum of subscapular and triceps skin-fold thickness measurements were all higher among black and Hispanic children than among non-Hispanic white children. Because of the significant heterogeneity among the children categorized as multiracial or "other" and because we did not observe differences in BMI z score or childhood overweight or obesity prevalence between these children and non-Hispanic white children in our study, we report all subsequent results only for black and Hispanic children compared with non-Hispanic white children. With the exception of excessive gestational weight gain, we observed that all the early life risk factors we measured were more prevalent among blacks and Hispanic children than among non-Hispanic white children (Table 2).

In models adjusted only for child age and sex, BMI *z* scores were higher among black and Hispanic children (differences, 0.48 U [95% CI, 0.31 to 0.64] and 0.43 U[0.12 to 0.74], respectively) compared with non-Hispanic white children (**Table 3** and **Figure**). After adjustment for socioeconomic confounders and parental BMI, these differences were attenuated for black and Hispanic children (0.22 U [95% CI, 0.05 to 0.40] and 0.22 U [-0.08 to 0.52], respectively). Adjustment for pregnancy risk

Table 1. Selected Parent and Child Characteristics According to Child Race/Ethnicity^a

	Race/Ethnicity of Child				
Characteristic	White, Non-Hispanic (63%)	Black, Non-Hispanic (17%)	Hispanic or Latino (4%)	Other (16%)	<i>P</i> Value
Maternal age, mean (SE), y	33.4 (0.16)	28.9 (0.49)	28.9 (0.93)	30.8 (0.44)	<.001
Maternal prepregnancy BMI, mean (SE)	24.0 (0.17)	27.3 (0.52)	25.5 (0.81)	24.6 (0.40)	<.001
Paternal BMI, mean (SE)	26.4 (0.14)	26.9 (0.35)	27.3 (0.58)	25.8 (0.33)	.05
Maternal educational level, college graduate or above, %	80.3	38.8	21.1	60.3	<.001
Primiparous mother, %	49.4	42.7	35.1	48.3	.15
Annual household income >\$70 000, %	73.3	25.1	23.1	56.8	<.001
Characteristics of children					
Female sex, %	51.9	45.5	36.8	50.1	.15
Measurements at age 7 y, mean (SE)					
BMI z score	0.29 (0.04)	0.79 (0.08)	0.74 (0.15)	0.29 (0.08)	
SS + TR skin-fold thickness, mm	18.7 (0.31)	23.5 (0.98)	21.9 (1.70)	20.2 (0.79)	<.001
Total FMI at DXA, kg/m ²	4.26 (0.06)	5.01 (0.20)	4.96 (0.33)	4.41 (0.15)	<.001
Waist circumference, cm	59.2 (0.26)	63.3 (0.84)	62.1 (1.32)	59.4 (0.68)	<.001
BMI, %					
≥85th Percentile	21.2	39.9	39.7	24.7	<.001
≥95th Percentile	7.2	27.3	25.4	13.0	<.001

Abbreviations: BMI, body mass index (calculated as weight in kilograms divided by height in meters squared); DXA, dual-energy x-ray absorptiometry; FMI, fat mass index; SS, subscapular; TR, triceps.

^a Data from 1116 mother-child pairs from Project Viva.

Table 2. Early Life Risk Factors for Childhood Obesity According to Child Race/Ethnicity^a

	Children, %			
Factor	White, Non-Hispanic	Black, Non-Hispanic	Hispanic or Latino	P Value
Pregnancy exposures				
Excessive gestational weight gain	59.9	58.2	52.7	.16
Gestational diabetes	4.6	5.2	5.2	.99
Mother smoking during early pregnancy	7.8	13.5	16.3	.01
Prenatal depression	6.4	15.6	20.8	.001
Infancy exposures				
Rapid infant weight gain ^b	20.5	36.8	39.7	<.001
Not exclusively breastfed at age 6 mo	68.6	81.5	88.3	.001
Introduction of solid foods before age 4 mo	13.1	33.9	41.2	<.001
Early childhood exposures				
Average daily sleep duration <12 h/d at age 6 mo to 2 y	30.2	60.4	64.1	<.001
Any sugar-sweetened beverage intake at age 2 y	46.3	84.4	81.3	<.001
Any fast food intake at age 3 y	64.5	80.9	83.7	<.001
Television in room where child sleeps at age 4 y	6.4	52.8	61.2	<.001

^a Data from 1116 mother-child pairs from Project Viva.

^b Rapid infant weight gain was defined as being in the highest quartile of change in weight-for-age z score between birth and age 6 months based on US reference data.³³

factors did not substantially change these estimates (differences, 0.23 U [0.06 to 0.40] for black and 0.24 U [-0.07 to 0.54] for Hispanic children). However, after further adjustment for infancy and early childhood risk factors, we observed only minimal differences in BMI *z* scores between white children and black or Hispanic children (0.07 U [-0.11 to 0.26] and 0.04 U [-0.27 to 0.35], respectively). Each infancy and early childhood risk factor contributed to the observed racial/ethnic differences. The degree of attenuation ranged from 10% after adjustment for having a television set in the room where the child sleeps to 23% after adjustment for rapid infant weight gain (data not shown).

We observed similar patterns of attenuation by socioeconomic factors, parental BMI, and infancy and early childhood risk factors for all our anthropometric outcomes (Table 3). However, racial/ethnic disparities were less attenuated for the prevalence of overweight or obesity than for BMI *z* score. For example, in models adjusted for socioeconomic factors and parental BMI, the odds of overweight or obesity was higher among black and Hispanic children (odds ratio, 1.60 [95% CI, 1.09-2.56] and 1.88 [0.89-3.94]) than among non-Hispanic white children. After adjustment for infancy and early childhood risk factors, these odds ratios were attenuated to 1.35 (95% CI, 0.84-2.16) in black and 1.46 (0.65-3.28) in Hispanic children.

Table 3. Anthropometric Outcomes at Age 7 Years According to Child Race/Ethnicity Before and After Adjustment for Confounders and Early Life Risk Factors for Obesity

	Estimated Difference or OR (95% CI)			
Measure and Multivariable Model ^a	White, Non-Hispanic ^b	Black, Non-Hispanic	Hispanic	
Difference in BMI z score				
Model 1	0	0.48 (0.31 to 0.64)	0.43 (0.12 to 0.74)	
Model 2	0	0.35 (0.17 to 0.53)	0.27 (-0.06 to 0.59)	
Model 3	0	0.22 (0.05 to 0.40)	0.22 (-0.08 to 0.52)	
Model 4	0	0.23 (0.06 to 0.40)	0.24 (-0.07 to 0.54)	
Model 5	0	0.16 (-0.01 to 0.33)	0.14 (-0.16 to 0.44)	
Model 6	0	0.07 (-0.11 to 0.26)	0.04 (-0.27 to 0.35)	
Difference in SS + TR skin-fold thickness, mm				
Model 1	0	3.95 (2.43 to 5.47)	2.93 (0.02 to 5.85)	
Model 2	0	2.64 (0.96 to 4.32)	1.20 (-1.84 to 4.24)	
Model 3	0	1.56 (-0.04 to 3.16)	0.81 (-2.07 to 3.68)	
Model 4	0	1.57 (-0.03 to 3.18)	0.84 (-2.05 to 3.74)	
Model 5	0	1.01 (-0.60 to 2.61)	-0.01 (-2.89 to 2.86)	
Model 6	0	0.25 (-1.46 to 1.97)	-0.86 (-3.83 to 2.11)	
Difference in FMI at DXA, kg/m ²				
Model 1	0	0.66 (0.35 to 0.97)	0.72 (0.13 to 1.30)	
Model 2	0	0.42 (0.07 to 0.76)	0.39 (-0.22 to 1.00)	
Model 3	0	0.18 (-0.14 to 0.51)	0.31 (-0.27 to 0.89)	
Model 4	0	0.19 (-0.14 to 0.51)	0.33 (-0.26 to 0.91)	
Model 5	0	0.08 (-0.25 to 0.40)	0.16 (-0.43 to 0.74)	
Model 6	0	-0.05 (-0.40 to 0.30)	0.02 (-0.59 to 0.62)	
Difference in waist circumference, cm				
Model 1	0	2.85 (1.57 to 4.13)	1.89 (-0.54 to 4.31)	
Model 2	0	2.13 (0.71 to 3.55)	0.94 (-1.59 to 3.48)	
Model 3	0	1.09 (-0.25 to 2.43)	0.60 (-1.78 to 2.99)	
Model 4	0	1.11 (-0.23 to 2.45)	0.65 (-1.74 to 3.04)	
Model 5	0	0.66 (-0.69 to 2.00)	-0.01 (-2.39 to 2.38)	
Model 6	0	0.12 (-1.32 to 1.56)	-0.61 (-3.07 to 1.85)	
OR for overweight or obesity (BMI ≥85th vs <85th percentile)				
Model 1	1 [Reference]	2.35 (1.69 to 3.34)	2.34 (1.22 to 4.51)	
Model 2	1 [Reference]	2.03 (1.36 to 3.02)	1.92 (0.96 to 3.84)	
Model 3	1 [Reference]	1.67 (1.09 to 2.56)	1.88 (0.89 to 3.94)	
Model 4	1 [Reference]	1.71 (1.12 to 2.62)	1.95 (0.92 to 4.14)	
Model 5	1 [Reference]	1.54 (1.00 to 2.39)	1.69 (0.78 to 3.67)	
Model 6	1 [Reference]	1.35 (0.84 to 2.16)	1.46 (0.65 to 3.28)	

Abbreviations: BMI, body mass index; DXA, dual-energy x-ray absorptiometry; FMI, fat mass index; OR, odds ratio; SS, subscapular; TR, triceps.

^a Model 1 is adjusted for child age and sex. Each subsequent model includes adjustments in the preceding model, with the following further adjustments: model 2, the confounders of maternal age, educational level, parity, and household income: model 3. the confounders of maternal and paternal BMI; model 4, the pregnancy factors of gestational diabetes and second-trimester depression; model 5, the infancy factors of rapid infant weight gain (defined as being in the highest quartile of change in weight-for-age z score between birth and age 6 months), feeding other than exclusive breastfeeding, and introduction of solid foods before age 4 months; and model 6, the early childhood risk factors of insufficient sleep, presence of a television set in the room where the child sleeps, and intake of sugar-sweetened beverages and fast food.

^b The values in this column are reference values.

Discussion

In this prospective prebirth cohort, we found that the prevalence of overweight and obesity among black and Hispanic children at age 7 years was almost double that in white children. The observed racial/ethnic disparities in adiposity and the prevalence of overweight or obesity were partially but not entirely explained by confounding socioeconomic factors and parental obesity. Although adjustment for obesity risk factors during pregnancy did not substantially change our observed associations, after adjustment for infancy and early childhood risk factors we observed only small differences in BMI *z* score between non-Hispanic white, black, and Hispanic children. Our findings suggest that racial/ethnic disparities in childhood obesity may be explained by factors operating in infancy and early childhood and that eliminating these factors could eliminate the disparities in childhood obesity. These factors include differences in modifiable early feeding behaviors, such as breastfeeding and timing of the introduction of solid foods, accelerated infant weight gain, and early childhood obesity-related risk factors, including insufficient sleep, the presence of a television set in the room where the child sleeps, and consumption of sugar-sweetened beverages and fast food, all of which have been found previously to be more prevalent among blacks and Hispanics than among whites.^{30,43-47} In our study, adjustment for obesity-related risk factors in infancy and early childhood attenuated the observed differences in BMI z score between blacks and

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Figure. Body Mass Index z Score at Age 7 Years



Body mass index (BMI) *z* score at age 7 years among black and Hispanic children compared with non-Hispanic white children (reference group) before and after adjustment for confounders and early life risk factors for obesity. Model 1 includes adjustment for child age and sex. Each subsequent model includes the adjustments in the preceding model with the following further adjustments: model 2 includes maternal age, educational level, parity, and household income; model 3, maternal and paternal BMI; model 4, the pregnancy factors of gestational diabetes and second-trimester depression; model 5, the infancy factors of rapid infant weight gain (defined as being in the highest quartile of change in weight-for-age *z* score between birth and age 6 months), feeding other than exclusive breastfeeding, and introduction of solid foods before age 4 months; and model 6, the early childhood risk factors of insufficient sleep, presence of a television set in the room where the child sleeps, and intake of sugar-sweetened beverages and fast food.

whites by as much as 69% and between Hispanics and whites by as much as 83%. Perhaps more complete measurement of and adjustment for infancy and childhood risk factors could attenuate the difference still further. Thus, our study implies that interventions to modify early life risk factors may greatly reduce disparities in the prevalence of childhood obesity.

Substantial evidence suggests that the best times in the life course to prevent obesity are infancy and early childhood, when behaviors are modifiable and/or physiologic characteristics are plastic.¹⁸ The infancy and early childhood periods seem particularly promising and highly sensitive to interventions; there are multiple settings in which to access parents (eg, primary care, child care, and early education), and parents and other caregivers are highly sensitized to the child's needs. Habits and tastes develop early in children, so it is critical to future behavioral patterns to establish tastes for a variety of foods, enjoyment of active play, and good sleep habits.

Despite growing evidence for the role of early life interventions in preventing childhood obesity, a 2010 review of interventions to prevent or treat overweight among children younger than 2 years yielded only 10 published studies of poor or fair quality.⁴⁸ In addition, national funding initiatives continue to exclude children younger than 2 years, a missed opportunity for both obesity prevention and reduction of related racial/ethnic disparities. For example, in 2011 the Centers for Disease Control and Prevention awarded approximately \$18 million to fund Childhood Obesity Research Demonstration grants,⁴⁹ intended to prevent and reduce obesity among children aged 2 to 12 years covered by the Children's Health Insurance Program, which provides low-cost health insurance to children from working families. Similar to findings of previous studies, we found that socioeconomic factors confounded the observed associations between race/ethnicity and our anthropometric outcomes. Adjustment for parental and household socioeconomic factors attenuated the observed difference in BMI *z* score between black and white children by about 27% and between Hispanic and white children by 37%.

Previous studies among both children⁵⁰ and adults⁵¹ have examined the extent to which racial/ethnic disparities in obesity are confounded or explained by socioeconomic status. In a study of 8984 children aged 12 to 17 years participating in the National Longitudinal Study of Youth, Powell et al⁵⁰ found that, although parental income and maternal educational level partially explained observed black-white and Hispanic-white differences in BMI, a large portion of these differences remained unexplained. In a study of 4356 US adults from the 1994-1996 Continuing Survey of Food Intakes by Individuals and the Diet and Health Knowledge Survey, Wang and Chen⁵¹ found that black-white differences in risk of overweight and obesity were reduced by 38% after adjustment for educational level and income, which partially explain the observed differences. Overall, our findings and those in the published literature suggest that social conditions and their effects on children's environments are important for understanding how disparities in childhood obesity originate.

In this study, we considered maternal and paternal BMI to be confounders of the relationship between race/ethnicity and obesity. However, parental BMI also could be an upstream variable in the association between race/ethnicity and obesity. Thus, obesity-related behaviors by overweight or obese parents could influence early life behaviors and in utero or early childhood physiologic programming in their offspring, leading to unhealthy weight trajectories.⁵² In our study, differences in maternal and paternal BMI partially explained the observed gaps between racial/ethnic groups. Our findings emphasize the importance of intergenerational effects on obesity and the need for interventions to interrupt the cycle of obesity in families.

Strengths of our study included prospectively collected data on a wide range of risk factors extending from pregnancy through early childhood; objectively measured weights, heights, and anthropometric outcomes; and adjustment for several important confounding socioeconomic factors. The study also had several potential limitations. First, although we used validated survey items, most of our exposure measures were from self-report. These factors could have introduced social desirability bias because participants could exaggerate selfreported behaviors. However, we have no reason to believe that self-report of behaviors would differ by racial/ethnic group. Second, although we studied many risk factors for childhood obesity that had plausible hypotheses, we did not measure others, such as lifestyle, cultural, or environmental determinants of dietary and sedentary practices. The incomplete measure of other important infancy and early childhood risk factors or incomplete adjustment for residual confounders may account for our inability to explain all the racial/ethnic disparities in the prevalence of overweight and obesity. Third, our sample of Hispanics was small, and the 95% CIs for some estimates were wide. However, the observed effect estimates of BMI *z* score for black and Hispanic children were almost identical, lending support to our conclusions for both groups.

In conclusion, obesity is disproportionately prevalent among racial/ethnic minority children,⁶ and recent trends suggest that these disparities are widening.⁵ Our findings suggest that modifiable risk factors throughout the life course,⁵³ including infancy and early childhood, as well as social conditions in childhood and transgenerational obesity, are critical to understanding how disparities in childhood obesity arise. Our findings also imply that efforts to eliminate racial/ethnic disparities in childhood obesity should focus on preventing these early life risk factors within their socioenvironmental context.

ARTICLE INFORMATION

Accepted for Publication: December 5, 2012.

Published Online: June 3, 2013. doi:10.1001/jamapediatrics.2013.85.

Author Contributions: Dr Taveras had full access to all the data in the study and takes responsibility for the integrity of the data and the accuracy of the data analysis.

Study concept and design: Taveras, Gillman, and Kleinman.

Acquisition of data: Rich-Edwards.

Analysis and interpretation of data: Taveras, Kleinman, Rich-Edwards, and Rifas-Shiman. Drafting of the manuscript: Taveras. Critical revision of the manuscript for important intellectual content: Gillman, Kleinman,

Rich-Edwards, and Rifas-Shiman. Statistical analysis: Kleinman, Rich-Edwards, and

Rifas-Shiman.

Administrative, technical, and material support: Taveras.

Obtained funding: Taveras and Gillman. *Study supervision:* Taveras and Gillman.

Conflict of Interest Disclosures: None reported.

Funding/Support: This study was supported by grant MD 003963 from the National Institute on Minority Health and Health Disparities.

REFERENCES

1. World Health Organization. Global strategy on diet, physical activity and health. http://www.who .int/dietphysicalactivity/en/. December 14, 2010. Accessed December 10, 2012.

2. Ogden CL, Carroll MD, Curtin LR, Lamb MM, Flegal KM. Prevalence of high body mass index in US children and adolescents, 2007-2008. *JAMA*. 2010;303(3):242-249.

3. Ogden CL, Carroll MD, Flegal KM. High body mass index for age among US children and adolescents, 2003-2006. *JAMA*. 2008;299(20): 2401-2405.

4. Ogden CL, Carroll MD, Curtin LR, McDowell MA, Tabak CJ, Flegal KM. Prevalence of overweight and obesity in the United States, 1999-2004. *JAMA*. 2006;295(13):1549-1555.

5. Olds T, Maher C, Zumin S, et al. Evidence that the prevalence of childhood overweight is plateauing: data from nine countries. *Int J Pediatr Obes*. 2011;6(5-6):342-360.

6. Ogden CL, Carroll MD, Kit BK, Flegal KM. Prevalence of obesity and trends in body mass index among US children and adolescents, 1999-2010. *JAMA*. 2012;307(5):483-490.

7. Wang YC, Gortmaker SL, Taveras EM. Trends and racial/ethnic disparities in severe obesity among US children and adolescents, 1976-2006 [published online March 17, 2010]. *Int J Pediatr Obes*. 2011;6(1): 12-20. doi:10.3109/17477161003587774.

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8. Wen X, Gillman MW, Rifas-Shiman SL, Sherry B, Kleinman K, Taveras EM. Decreasing prevalence of obesity among young children in Massachusetts from 2004 to 2008. *Pediatrics*. 2012;129(5): 823-831.

9. Taveras EM, Rifas-Shiman SL, Oken E, Gunderson EP, Gillman MW. Short sleep duration in infancy and risk of childhood overweight. *Arch Pediatr Adolesc Med*. 2008;162(4):305-311.

10. Ong KK, Loos RJ. Rapid infancy weight gain and subsequent obesity: systematic reviews and hopeful suggestions. *Acta Paediatr*. 2006;95(8): 904-908.

11. Baird J, Fisher D, Lucas P, Kleijnen J, Roberts H, Law C. Being big or growing fast: systematic review of size and growth in infancy and later obesity [published online October 14, 2005]. *BMJ*. 2005;331(7522):929.

12. Monteiro PO, Victora CG. Rapid growth in infancy and childhood and obesity in later life—a systematic review. *Obes Rev.* 2005;6(2):143-154.

13. Ylihärsilä H, Kajantie E, Osmond C, Forsén T, Barker DJ, Eriksson JG. Body mass index during childhood and adult body composition in men and women aged 56-70 y. *Am J Clin Nutr.* 2008;87(6): 1769-1775.

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

15. Reilly JJ, Armstrong J, Dorosty AR, et al; Avon Longitudinal Study of Parents and Children Study Team. Early life risk factors for obesity in childhood: cohort study [published online May 20, 2005]. *BMJ*. 2005;330(7504):1357.

16. Owen CG, Martin RM, Whincup PH, Smith GD, Cook DG. Effect of infant feeding on the risk of obesity across the life course: a quantitative review of published evidence. *Pediatrics*. 2005;115(5): 1367-1377.

17. Oken E, Levitan EB, Gillman MW. Maternal smoking during pregnancy and child overweight: systematic review and meta-analysis. *Int J Obes* (*Lond*). 2008;32(2):201-210.

18. Gillman MW, Rifas-Shiman SL, Kleinman K, Oken E, Rich-Edwards JW, Taveras EM. Developmental origins of childhood overweight: potential public health impact. *Obesity (Silver Spring)*. 2008;16(7):1651-1656.

19. Oken E, Rifas-Shiman SL, Field AE, Frazier AL, Gillman MW. Maternal gestational weight gain and offspring weight in adolescence. *Obstet Gynecol.* 2008;112(5):999-1006.

20. Oken E, Taveras EM, Kleinman KP, Rich-Edwards JW, Gillman MW. Gestational weight gain and child adiposity at age 3 years. *Am J Obstet Gynecol*. 2007;196(4):322.e1-322.e8. doi:10.1016/j.ajog.2006.11.027. **21.** Wright CS, Rifas-Shiman SL, Rich-Edwards JW, Taveras EM, Gillman MW, Oken E. Intrauterine exposure to gestational diabetes, child adiposity, and blood pressure. *Am J Hypertens*. 2009;22(2): 215-220.

22. Gillman MW, Oakey H, Baghurst PA, Volkmer RE, Robinson JS, Crowther CA. Effect of treatment of gestational diabetes mellitus on obesity in the next generation. *Diabetes Care*. 2010;33(5): 964-968.

23. Taveras EM, Rifas-Shiman SL, Belfort MB, Kleinman KP, Oken E, Gillman MW. Weight status in the first 6 months of life and obesity at 3 years of age. *Pediatrics*. 2009;123(4):1177-1183.

24. Belfort MB, Rifas-Shiman SL, Rich-Edwards J, Kleinman KP, Gillman MW. Size at birth, infant growth, and blood pressure at three years of age. *J Pediatr*. 2007;151(6):670-674.

25. Taveras EM, Rifas-Shiman SL, Scanlon KS, Grummer-Strawn LM, Sherry B, Gillman MW. To what extent is the protective effect of breastfeeding on future overweight explained by decreased maternal feeding restriction? *Pediatrics*. 2006;118(6):2341-2348.

26. van Rossem L, Taveras EM, Gillman MW, et al. Is the association of breastfeeding with child obesity explained by infant weight change? *Int J Pediatr Obes*. 2011;6(2-2):e415-e422. doi:10.3109 /17477166.2010.524700.

27. White House Task Force. Solving the Problem of Childhood Obesity Within a Generation: White House Task Force Report on Childhood Obesity Report to the President. Washington, DC; 2010. http://www.letsmove.gov/white-house-task-force -childhood-obesity-report-president. Accessed May 9, 2013.

28. Institute of Medicine. *Early Childhood Obesity Prevention Policies*. Washington, DC: National Academies Press; 2011.

29. Institute of Medicine. Accelerating Progress in Obesity Prevention: Solving the Weight of the Nation. Washington, DC: National Academy of Sciences; 2012.

30. Taveras EM, Gillman MW, Kleinman K, Rich-Edwards JW, Rifas-Shiman SL. Racial/ethnic differences in early-life risk factors for childhood obesity. *Pediatrics*. 2010;125(4):686-695.

31. Gillman MW, Rich-Edwards JW, Rifas-Shiman SL, Lieberman ES, Kleinman KP, Lipshultz SE. Maternal age and other predictors of newborn blood pressure. *J Pediatr*. 2004;144(2):240-245.

32. US Census Bureau, US Department of Commerce. Race. http://www.census.gov /population/race/about. Accessed May 9, 2013.

33. National Center for Health Statistics. CDC growth charts, United States. 2000. http://www.cdc.gov/growthcharts/. Accessed May 9, 2013. **34**. Shorr IJ. *How to Weigh and Measure Children.* New York, NY: United Nations; 1986.

35. Mueller WH, Martorell R. Reliability and accuracy of measurement. In: Lohman TG, Roche AF, Martorell R, eds. *Anthropometric Standardization Reference Manual*. Champaign, IL: Human Kinetics Books; 1988.

36. American Diabetes Association. Gestational diabetes mellitus. *Diabetes Care*. 2004;27(suppl 1): \$88-\$90.

37. Cox JL, Holden JM, Sagovsky R. Detection of postnatal depression: development of the 10-item Edinburgh Postnatal Depression Scale. *Br J Psychiatry*. 1987;150(6):782-786.

38. Blum RE, Wei EK, Rockett HR, et al. Validation of a food frequency questionnaire in Native American and Caucasian children 1 to 5 years of age. *Matern Child Health J.* 1999;3(3):167-172.

39. Pereira MA, Kartashov AI, Ebbeling CB, et al. Fast-food habits, weight gain, and insulin resistance (the CARDIA study): 15-year prospective analysis. *Lancet*. 2005;365(9453):36-42.

40. Rubin DB. *Multiple Imputation for Nonresponse in Surveys*. New York, NY: Wiley & Sons; 1987.

41. Horton NJ, Kleinman KP. Much ado about nothing: a comparison of missing data methods and software to fit incomplete data regression models. *Am Stat.* 2007;61(1):79-90.

42. Ertel KA, Kleinman K, van Rossem L, et al. Maternal perinatal depression is not independently associated with child body mass index in the Generation R Study: methods and missing data matter. *J Clin Epidemiol.* 2012;65(12):1300-1309.

43. Dennison BA, Erb TA, Jenkins PL. Television viewing and television in bedroom associated with overweight risk among low-income preschool children. *Pediatrics*. 2002;109(6):1028-1035.

44. Welsh JA, Cogswell ME, Rogers S, Rockett H, Mei Z, Grummer-Strawn LM. Overweight among low-income preschool children associated with the consumption of sweet drinks: Missouri, 1999-2002. *Pediatrics*. 2005;115(2):e223-e229. doi:10.1542/peds.2004-1148.

45. 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(6): e1604-e1614. doi:10.1542/peds.2007-2834.

46. Piernas C, Popkin BM. Increased portion sizes from energy-dense foods affect total energy intake at eating occasions in US children and adolescents: patterns and trends by age group and sociodemographic characteristics, 1977-2006. *Am J Clin Nutr.* 2011;94(5):1324-1332.

47. Nevarez MD, Rifas-Shiman SL, Kleinman KP, Gillman MW, Taveras EM. Associations of early life risk factors with infant sleep duration. *Acad Pediatr*. 2010;10(3):187-193. Reducing Racial Disparities in Childhood Obesity

48. Ciampa PJ, Kumar D, Barkin SL, et al. Interventions aimed at decreasing obesity in children younger than 2 years: a systematic review. *Arch Pediatr Adolesc Med*. 2010;164(12):1098-1104.

49. Centers for Disease Control and Prevention. Childhood Obesity Research Demonstration Project. http://www.cdc.gov/obesity/childhood /researchproject.html. Accessed May 9, 2013.

50. Powell LM, Wada R, Krauss RC, Wang Y. Ethnic disparities in adolescent body mass index in the United States: the role of parental socioeconomic status and economic contextual factors. *Soc Sci Med*. 2012;75(3):469-476.

51. Wang Y, Chen X. How much of racial/ethnic disparities in dietary intakes, exercise, and weight status can be explained by nutrition- and health-related psychosocial factors and socioeconomic status among US adults? *J Am Diet Assoc.* 2011;111(12):1904-1911.

52. Nader PR, Huang TT, Gahagan S, Kumanyika S, Hammond RA, Christoffel KK. Next steps in obesity prevention: altering early life systems to support healthy parents, infants, and toddlers. *Child Obes*. 2012;8(3):195-204.

53. Dixon B, Peña MM, Taveras EM. Lifecourse approach to racial/ethnic disparities in childhood obesity. *Adv Nutr*. 2012;3(1):73-82.