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The female-male disparity in obesity prevalence among black American young adults: contributions of sociodemographic characteristics of the childhood family[1–4]

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ABSTRACT

Background: In the United States, black women are at much greater risk of obesity than are black men. Little is known about the factors underlying this disparity.

Objective: We explored whether childhood sociodemographic factors (parental education, single-mother household, number of siblings, number of minors in household, birth order, and female caregiver's age) were associated with the gender disparity in obesity prevalence in young black adults in the United States.

Design: An analytic data set ($n = 7747$) was constructed from the nationally representative National Longitudinal Study of Adolescent Health. Childhood sociodemographic factors were assessed in 1994–1995 in nonimmigrant black and white youths aged 11–19 y. Obesity was assessed in 2001–2002. For each childhood sociodemographic factor, we evaluated whether the prevalence difference (female obesity minus male obesity) was modified by the factor. We described the contribution of each variable category to the overall prevalence difference.

Results: In unadjusted and multivariable-adjusted models, parental education consistently modified gender disparity in blacks ($P = 0.01$). The gender gap was largest with low parental education (16.7% of men compared with 45.4% of women were obese) and smallest with high parental education (28.5% of men compared with 31.4% of women were obese). In whites, there was little overall gender difference in obesity prevalence.

Conclusions: To our knowledge, this was the first study to document that the gender disparity in obesity prevalence in young black adults is concentrated in families with low parental education. In these low-socioeconomic-status families, obesity development is either under the control of distinct mechanisms in each gender, or men and women from these households adopt different obesity-related behaviors. *Am J Clin Nutr* 2009;89:1204–12.

INTRODUCTION

It is well established that prevalence of obesity is higher in black women than in black men (1, 2). In the 1999–2002 National Health and Nutrition Examination Survey (NHANES), the gender difference in obesity prevalence in blacks was 21.1 percentage points: 49.0% of black women were obese, whereas only 27.9% of black men were obese (3). In contrast, there was virtually no gender disparity in obesity prevalence in whites: 30.7% of white women were obese compared with 28.2% of white men (3).

NHANES has monitored gender-specific obesity prevalence for the past 35 y (4). During most of that time, the gender disparity among US blacks remained stable at ≈15 percentage points, but has increased to 20 percentage points in more recent surveys (2, 4). Although this gender disparity is well documented, little is known about factors underlying the disparity: in fact, we know of no studies that have investigated factors associated with this gender gap in obesity in blacks.

Because this gender disparity in obesity prevalence emerges in childhood (2, 5), we hypothesized that the disparity observed in young adults is associated with characteristics of the childhood environment. Several sociodemographic characteristics of the childhood family environment, such as parental education and birth order, have been found to be associated with obesity risk in adolescence and adulthood (5–17). We hypothesized that sociodemographic characteristics of the childhood family environment may differentially affect obesity risk in male and female children (18) and thus affect the magnitude of the gender disparity in obesity prevalence in black adults.

We were particularly interested in whether socioeconomic status (SES) was associated with gender disparity in obesity. For

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[2] This study used data from Add Health, a program project designed by J Richard Udry, Peter S Bearman, and Kathleen Mullan Harris and that was funded by grant P01-HD31921 from the National Institute of Child Health and Human Development, with cooperative funding from 17 other agencies. Persons interested in obtaining data files from Add Health should contact Add Health, Carolina Population Center, 123 West Franklin Street, Chapel Hill, NC 27516-2524 (addhealth@unc.edu).

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young black men and women in NHANES III (1988–1994), the relations between SES and ''overweight/obesity'' (19) indicated that the gender disparity would be larger in low-SES than in high-SES black young adults. Nonetheless, a recent review suggested that, between 1988–1994 and 1999–2000, the relations between SES and obesity reversed in black men and women (20). This finding implied that the gender disparity would be larger in high-SES than in low-SES black adults. However, this more recent research focused on an older population. In younger adults, it remains unclear whether, in the more recent time period, the gender disparity in obesity was larger in low-SES or in high-SES blacks.

We used data from a nationally representative prospective cohort study of adolescents followed into young adulthood to investigate the associations between sociodemographic characteristics of the childhood family environment and gender disparity in obesity prevalence in young black adults in the United States. We replicated all analyses in US whites to detect common patterns in blacks and whites and to contextualize the disparities observed in blacks.

SUBJECTS AND METHODS

Population

Data were from the National Longitudinal Study of Adolescent Health (Add Health). Add Health began as a nationally representative survey of all US public and private school students enrolled in grades 7 through 12. The Add Health survey focused on adolescent risk behaviors and includes a wealth of behavior data. The survey was cluster-sampled by school and also oversampled some subgroups, including black students with a parent who had completed college or attained a professional degree.

In 1994–1995 (baseline), detailed questionnaires were administered to each student and to the student's primary in-residence caregiver, preferentially a female. A year later, in 1995–1996, all students except those in 12th grade at baseline were re-interviewed. In 2001–2002, 7 y after baseline, all study respondents who participated in the 1994–1995 baseline visit were re-interviewed, and height and weight were measured.

We restricted our sample to non-Hispanic blacks and whites, at least one of whose parents was born in the United States. Race was defined by a combination of child self-report and parent self-report data (21). We restricted the sample to adolescents with a US-born parent (22) because our theoretical framework presumed that shared cultural and historical experiences shape how obesity-related beliefs, behaviors, and desired norms may be transmitted differently to each gender. In immigrant families, these constructs could be additionally influenced by the cultural context of their parents' countries of origin. We also restricted our analysis sample to those eligible to be interviewed at all 3 study time points, ie, those in the 12th grade in 1994–1995 were excluded. All study procedures were approved by the Institutional Review Board of the University of North Carolina at Chapel Hill.

Outcome

The main study outcome was the prevalence difference for obesity (obesity prevalence in women minus that in men) at the 7-y follow-up visit. Obesity was defined as body mass index (BMI; in kg/m^2) $\geq$30 (23). A prevalence difference of 0 represents an equal obesity prevalence for men and women, $>$0 indicates that women were more likely to be obese, and $<$0 indicates that men were more likely to be obese.

We considered modeling 6-y incidence rather than prevalence. Incidence is advantageous when one is estimating causal associations and seeks to exclude bias from reverse causation and from confounding by differential outcome duration. However, our intention was not to calculate causal estimates. Furthermore, we believe that reverse causation and differential outcome duration are of limited importance in this analysis. First, it is unlikely or impossible that a child's obesity status would affect parental education, family structure, female caregiver's age at child's birth, number of minors in household, birth order, or number of siblings. Second, obesity is generally a persistent state. In Add Health, over a 6-y period, obesity was maintained 80–90% of the time in black and white males and females (1). By modeling prevalence, we produced estimates that can be directly compared with other surveillance data and can be easily incorporated into estimates of public health burden.

Exposures and covariates

Exposure variables were derived from the baseline (1994–1995) interviews of respondents and their caregivers. Six exposure variables were examined: number of full siblings (including respondent), birth order, number of minors (age $\leq$ 18 y) living in the respondent's household (including respondent), parental education, family structure, and female caregiver's age at the time of the respondent's birth. Both birth order and number of siblings were defined in terms of the respondent's full sibship, ie, all children of both respondents' biological parents. Parental education was defined as the highest education attained by either of the respondent's biological parents (5). We categorized family structure into 4 groups: household headed by single mother; by both biological parents; by 2 parents, at least one of whom was nonbiological; and other (22). Ninety-two percent of the identified female caregivers reported being the respondents' biological mothers.

Other variables included in all multivariable models were categorical age and the respondent's biological sex. Although we use a variable for biological sex, our theoretical framework presumes that the obesity disparity observed between black men and women results from both biological (''sex'') and cultural (''gender'') influences (24). For lack of a more precise term, throughout this article, we use the word ''gender'' to connote the confluence of biological and cultural influences.

Exclusions

Overall, 78.3% of those eligible participated in the 7-y follow-up (80.9% of black females, 71.2% of black males, 82.0% of white females, and 75.9% of white males). Of the respondents, 7.7% were excluded from the present analysis. About 5% of respondents were excluded because they were missing baseline exposure information (4.6%), mostly female caregiver's age at child's birth, which was missing for 3.6% of respondents. Others were excluded because they were outside the desired age range (0.01%), lived alone at baseline (0.2%), were pregnant at the time of the follow-up visit [2.2% overall

(4.5% black women; 4.0% white women)], or were missing measured and self-reported height or weight data (0.9%). Self-reported height or weight was substituted for missing measured data for 5.4% of respondents (7.2% of black females, 6.4% of black males, 5.6% of white females, and 4.2% of white males). The final analysis sample consisted of 2096 black and 5651 white respondents.

Data analysis

Effect modification of the prevalence difference

Our primary hypothesis was tested by examining whether there was effect modification of the prevalence difference in obesity by any of 6 exposure variables. Specifically, this analysis tested whether the prevalence difference varied across categories of each childhood sociodemographic factor. All sociodemographic factors were analyzed as nominal categorical variables. We first calculated obesity prevalence by race for each gender within each exposure category. We then estimated the obesity prevalence difference in each stratum of each exposure. Finally, effect modification of the prevalence difference by each exposure was tested by using modified Wald tests (25).

All estimates were corrected for Add Health's complex survey design (25). Both unadjusted and multivariable-adjusted estimates were calculated for all analyses (Stata 9; Stata Corp, College Station, TX). To allow multivariable-adjusted associations to vary independently by gender, all variable categories had interactions with gender. Calculating adjusted prevalence estimates from multivariable logistic regression models required us to set model covariates to specific values (26). We chose to standardize the multivariable-adjusted estimates to hypothetical race-stratified populations with covariate distributions similar to those of the sample population. For each race group, we set each covariate category's value at the mean proportion of respondents in that category over the 2 genderes (Microsoft Excel, Redmond, WA). The one exception was respondent's age, for which data from the 2 race groups were combined before determining the mean proportions. The delta method was used to calculate 95% CIs for prevalence differences.

Because some of our 6 exposures were likely to be associated with each other, we screened for multicollinearity. We used bivariate multinomial logistic regression models with each variable as an outcome and each of the other 5 variables separately as an exposure. We found that female caregiver's age at the time of the respondent's birth was strongly associated [odds ratio (OR) $\geq$ 3.0] with most other exposure variables. To examine this further, we ran multivariable models both including and excluding the female caregiver's age variable. The estimates for other variables changed very little, so we report estimates from multivariable models including female caregiver's age at the time of the respondent's birth.

We also observed a strong bivariate association (OR $\geq$ 3.0) between birth order and number of siblings. To separate the associations with birth order compared with those with the number of siblings, we created joint variables that allowed us to examine associations between prevalence differences in obesity and either birth order or size of sibship while holding the other factor constant. These analyses indicated that birth order was the factor more strongly associated with differential obesity prevalence by gender (data not shown). Therefore, our final multivariable logistic regression models included gender, categorical age, 5 exposure variables (excluding number of full siblings), and gender interaction terms with all variables.

Decomposition of the prevalence difference

We used the Kitagawa decomposition technique to divide the overall gender disparity into components due to differences between men and women in their stratum-specific obesity prevalences (27). For each variable, the standard population was assigned the average exposure distribution of the 2 gender groups. We modified the Kitagawa method by dividing each stratum-specific component by the sum of the absolute values of the stratum-specific components instead of dividing by the overall prevalence difference. This approach simplifies interpretation when stratum-specific estimates have different signs. In addition, this approach explores only disparity due to differences in stratum-specific obesity prevalence by gender, not that which may be due to men and women having different likelihoods of being in a given stratum. Any disparity due to differences in the percentage of men and women in each stratum is reflected in the difference between the overall gender disparity and the sum of the stratum-specific components.

RESULTS

The mean age, obesity prevalence, and distributions of the exposure variables across the 4 race-gender groups are shown in **Table 1**. The distributions of the exposure variables differed between blacks and whites but generally varied little between males and females of the same race. As expected, obesity prevalence was higher in black women than in black men: the estimated prevalence difference was 11.9 percentage points (95% CI: 7.0, 16.7). Among whites, there was no gender difference in obesity prevalence: the prevalence difference was only 0.9 percentage points (95% CI: -1.9, 3.8).

The distributions of unadjusted prevalence differences across the 6 exposure variables in whites and blacks are shown in **Table 2**. In blacks, the prevalence difference, or magnitude of black women's excess obesity prevalence compared with black men, was not uniform across the exposure variables. For instance, the prevalence difference in blacks varied markedly depending on parental education ($P = 0.01$). In families in which neither parent completed high school, obesity prevalence in black women was 45.4% compared with only 16.7% in black men, which corresponded to a large prevalence difference of 28.8 percentage points (Table 2). In contrast, in blacks with at least one parent with a college degree, the prevalence difference was only 2.9 percentage points. There was also some statistical evidence that the gender disparity in blacks varied with birth order ($P = 0.11$). Because tests of modification are weakly powered and therefore prone to type II error, cutoffs as high as 0.20 are often recommended to identify variation in an effect measure (28–30). In whites, the prevalence difference did not vary across any exposures to the same degree as it did for parental education in blacks, but the prevalence difference in whites did tend to vary with parental education ($P = 0.05$), age of female caregiver ($P = 0.08$), and birth order ($P = 0.14$).

Multivariable-adjusted prevalence differences are shown in **Figure 1**, A–E. The overall multivariable-adjusted prevalence differences for blacks and whites were 12.5 percentage points

TABLE 1
Characteristics of US black and white young adults, adjusted for sampling design: National Longitudinal Study of Adolescent Health, 2001–2002[1]

	Black		White	
	Females ($n = 1153$)	Males ($n = 943$)	Females ($n = 2909$)	Males ($n = 2742$)
Age (y)[2–4]	21.5 ± 0.2	21.7 ± 0.2	21.2 ± 0.1	21.5 ± 0.1
Obese [% (n)][5–7]	35.6 (355)	23.7 (207)	21.6 (597)	20.7 (546)
Parental education [%][4–8]				
Less than HS graduate	21.0 (191)	15.3 (94)	9.2 (268)	8.9 (220)
HS graduate	37.3 (337)	36.7 (283)	33.7 (971)	32.8 (880)
Some college	24.5 (302)	27.0 (282)	29.6 (855)	31.3 (861)
College graduate	17.2 (323)	21.1 (284)	27.6 (815)	27.0 (781)
Family structure [% (n)][4,6–8]				
Single mother	44.6 (490)	44.9 (381)	15.6 (451)	14.4 (390)
Two biological parents	32.8 (399)	28.3 (335)	63.2 (1812)	65.0 (1741)
Two parents, ≥1 nonbiological	12.6 (158)	14.6 (146)	18.0 (554)	17.6 (538)
Other	10.0 (106)	12.2 (81)	3.2 (92)	3.0 (73)
Female caregiver's age at birth [% (n)][4,6–8]				
<19 y	13.2 (125)	12.9 (96)	5.4 (168)	6.4 (159)
19–24 y	37.9 (434)	40.8 (358)	35.1 (1034)	38.3 (1023)
25–34 y	37.9 (473)	33.5 (375)	52.8 (1518)	48.4 (1377)
35–44 y	8.8 (93)	8.9 (86)	5.6 (162)	5.9 (157)
≥45 y	2.3 (28)	4.0 (28)	1.1 (27)	1.0 (26)
No. of children in household [% (n)][4,6–8]				
1	25.3 (266)	25.8 (238)	26.7 (701)	27.0 (703)
2	33.3 (389)	32.3 (335)	43.9 (1266)	41.3 (1147)
3	18.8 (255)	21.5 (213)	20.1 (645)	21.7 (605)
≥4	22.6 (243)	20.4 (157)	9.3 (297)	10.0 (287)
No. of children in full sibship [% (n)][4,6–8]				
1	28.0 (306)	31.0 (259)	20.6 (543)	17.5 (463)
2	30.5 (381)	28.3 (284)	40.8 (1190)	41.1 (1114)
3	20.6 (241)	18.8 (207)	25.9 (767)	26.9 (736)
≥4	21.1 (225)	22.0 (193)	12.7 (409)	14.5 (429)
Birth order [% (n)][4,6–8]				
First born	53.7 (609)	54.3 (478)	52.5 (1474)	52.3 (1386)
Second born	25.0 (315)	24.2 (252)	31.3 (932)	31.0 (875)
Third born	11.1 (126)	12.4 (125)	11.6 (359)	11.0 (320)
≥Fourth born	10.2 (103)	9.1 (88)	4.6 (144)	5.7 (161)

[1] Percentages are adjusted for the clustered sampling design of and unequal probability of selection into the data set. Numbers are the absolute unadjusted numbers in each stratum. HS, high school.

[2] All values are means ± SEs adjusted for the clustered sampling design of and unequal probability of selection into the data set. The age range was 18–26 y for all 4 race-gender groups. Estimated SDs for age, adjusted for the clustered sampling design and unequal probability of selection into the data set, were as follows: 1.8 for black men, 2.0 for black women, 1.4 for white men, and 1.4 for white women.

[3] Statistically significant difference ($P \leq 0.05$) in variable distribution between white females and white males (Pearson chi-square test adjusted for the clustered sampling design of and unequal probability of selection into the data set).

[4] Statistically significant difference ($P \leq 0.05$) in variable distribution between white females and black males (Pearson chi-square test adjusted for the clustered sampling design of and unequal probability of selection into the data set).

[5] Statistically significant difference ($P \leq 0.05$) in variable distribution between black females and black males (Pearson chi-square test adjusted for the clustered sampling design of and unequal probability of selection into the data set).

[6] Statistically significant difference ($P \leq 0.05$) in variable distribution between black females and white females (Pearson chi-square test adjusted for the clustered sampling design of and unequal probability of selection into the data set).

[7] Statistically significant difference ($P \leq 0.05$) in variable distribution between black females and white males (Pearson chi-square test adjusted for the clustered sampling design of and unequal probability of selection into the data set).

[8] Statistically significant difference ($P \leq 0.05$) in variable distribution between black males and white males (Pearson chi-square test adjusted for the clustered sampling design of and unequal probability of selection into the data set).

(95% CI: 7.8, 17.2) and 0.4 percentage points (95% CI: −2.4, 3.3), respectively. These prevalence differences are indicated on the figures by the dashed lines. The P values associated with Figure 1 (A–E) are from tests of the association between each sociodemographic factor and the gender disparity in obesity prevalence: the tests evaluated whether the prevalence difference varied between categories of each exposure variable. The most striking association with the obesity gender disparity was found for parental education in blacks (Figure 1A; $P = 0.01$). The prevalence difference was greatest among those whose parents did not complete high school and was smallest among those with a parent who completed college. In whites, as in blacks, the overall trend was that women from the lowest-educated families had a higher risk of obesity risk than did men from similar families. In whites, the test for effect modification of the prevalence difference in obesity by parental education was statistically

TABLE 2
Unadjusted prevalence of obesity and prevalence differences in US black and white young adults stratified by race, gender, and childhood sociodemographic factors: National Longitudinal Study of Adolescent Health, 2001–2002[1]

	Black			White		
	Prevalence in females ($n = 1153$)	Prevalence in males ($n = 943$)	Prevalence difference[2]	Prevalence in females ($n = 2909$)	Prevalence in males ($n = 2742$)	Prevalence difference[2]
	%	%	% points	%	%	% points
Overall	35.6	23.7	11.9 ± 2.4	21.6	20.7	0.9 ± 1.4
Parental education[3,4]						
Less than HS graduate	45.4	16.7	28.8 ± 6.5	41.3	27.5	13.8 ± 6.1
HS graduate	30.3	23.6	6.7 ± 4.0	23.9	22.2	1.7 ± 2.4
Some college	38.2	24.2	14.0 ± 5.6	20.2	20.7	−0.4 ± 2.1
College graduate	31.4	28.5	2.9 ± 4.8	13.9	16.7	−2.8 ± 2.1
Family structure						
Single mother	37.4	22.4	15.0 ± 4.5	24.6	20.3	4.3 ± 3.2
Two biological parents	35.8	24.5	11.2 ± 3.5	21.1	20.6	0.5 ± 1.8
Two parents, ≥1 nonbiological	32.1	20.8	11.3 ± 7.0	20.1	21.7	−1.5 ± 3.1
Other	31.5	30.5	1.0 ± 9.6	26.3	19.4	6.9 ± 8.9
Female caregiver's age at birth						
<19 y	39.1	26.7	12.4 ± 9.7	34.7	23.5	11.2 ± 6.0
19–24 y	35.9	22.9	13.1 ± 4.2	23.0	22.3	0.8 ± 2.3
25–34 y	34.4	25.2	9.2 ± 3.8	18.3	19.3	−1.1 ± 1.8
35–44	28.1	13.3	14.8 ± 7.9	29.1	19.3	9.9 ± 5.8
≥45 y	58.7	34.0	24.7 ± 16.7	37.9	18.7	19.2 ± 14.8
No. of children in household						
1	32.9	25.0	7.8 ± 5.9	24.9	20.2	4.7 ± 2.8
2	38.3	27.7	10.6 ± 5.4	21.8	22.7	−0.8 ± 2.3
3	31.4	21.7	9.6 ± 5.5	17.1	18.8	−1.7 ± 3.3
≥4	38.2	17.9	20.3 ± 5.6	21.2	18.1	3.1 ± 5.6
No. of children in full sibship						
1	32.0	21.4	10.6 ± 4.5	21.1	22.0	−0.9 ± 3.2
2	45.1	28.9	16.2 ± 5.3	20.8	21.1	−0.3 ± 2.3
3	26.9	24.9	2.0 ± 5.9	21.4	20.5	0.9 ± 2.4
≥4	35.1	19.4	15.7 ± 6.7	25.9	18.5	7.3 ± 3.7
Birth order						
First born	37.4	21.9	15.5 ± 3.0	20.8	22.2	−1.4 ± 2.2
Second born	33.7	32.0	1.6 ± 5.1	20.0	19.0	1.1 ± 2.2
Third born	29.4	13.6	15.8 ± 5.8	26.7	19.9	6.8 ± 4.1
≥Fourth born	37.5	26.2	11.3 ± 10.8	29.3	17.9	11.5 ± 6.4

[1] All prevalence and prevalence difference statistics were adjusted for the clustered sampling design of and unequal probability of selection into the data set. HS, high school.

[2] All values are percentage points ± SEs.

[3] In blacks, statistical evidence ($P \leq 0.05$) indicated that the prevalence difference varied over the strata of this variable (modified Wald test, adjusted for the clustered sampling design of and unequal probability of selection into the data set).

[4] In whites, statistical evidence ($P \leq 0.05$) indicated that the prevalence difference varied over the strata of this variable (modified Wald test, adjusted for the clustered sampling design of and unequal probability of selection into the data set).

significant in unadjusted (Table 2) but not in multivariable-adjusted models ($P = 0.34$).

For 2 other sociodemographic exposures, there were suggestions of associations with the gender difference in obesity prevalence. Birth order appeared somewhat associated with gender difference in both blacks and whites (Figure 1E). Having a female caregiver who was relatively young (≤18 y at the time of the respondent's birth) or relatively old (≥35 y old at the time of the respondent's birth) appeared to be associated with a higher prevalence of obesity for white women than for their white male counterparts (Figure 1C).

Results from the decomposition analyses are shown in **Table 3**. Blacks from the lowest parental-education families represented <20% of the population but contributed >40% of the gender gap in obesity in blacks (≈5 percentage points). In contrast, black children of college graduates also made up ≈20% of the population but contributed only ≈5% of the gender gap (<1 percentage point). In earlier analyses, whites from families in which parents did not complete high school also appeared to show a gender gap in obesity. However, this group made up less than 10% of the white population and thus contributed only ≈1 percentage point of gender disparity.

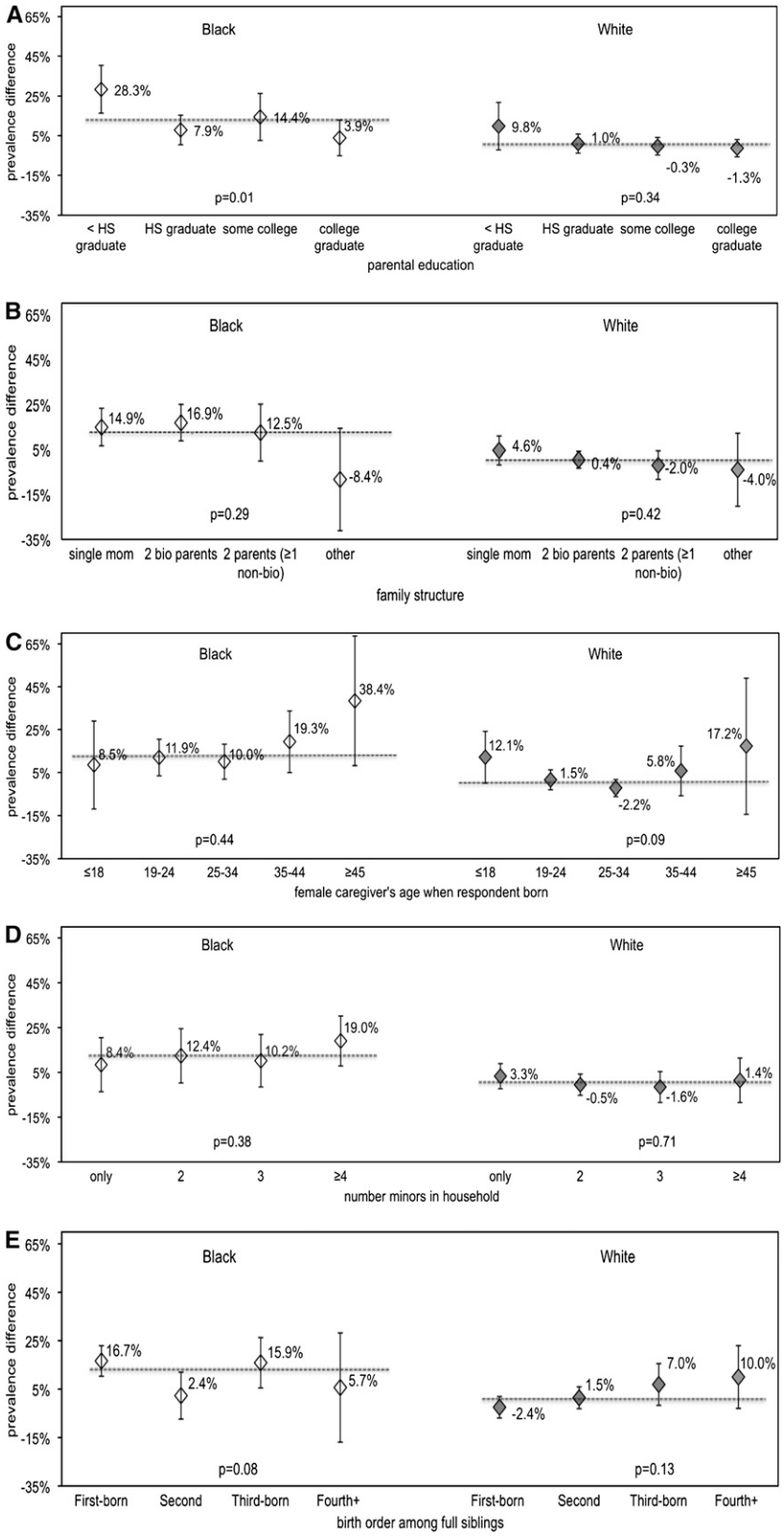

FIGURE 1. (A–E) Estimated multivariable-adjusted differences in obesity prevalence between young US black ($n = 2096$) and white ($n = 5651$) women and men in the by race and childhood family exposures. Data are from the National Longitudinal Study of Adolescent Health, 2001–2002. Error bars represent 95% CIs. *P* values are from race-specific tests of modification of the differences in prevalence of obesity by the exposure variable. The tests assessed whether the prevalence difference varies across categories of the variable (modified Wald test, adjusted for the clustered sampling design of and unequal probability of selection into the data set). The dotted lines represent the overall multivariable-adjusted prevalence difference in blacks (12.5 percentage points) and whites (0.4 percentage points). bio, biological; HS, high school.

TABLE 3
Contributions of population subgroups, defined by childhood sociodemographic factors, to the female-male disparity in the prevalence of obesity in US black and white young adults: National Longitudinal Study of Adolescent Health, 2001–2002[1]

	Black (n = 2096)			White (n = 5651)		
		Contribution to gender gap			Contribution to gender gap	
	Population weights[2]	Unadjusted model	Multivariable model	Population weights[2]	Unadjusted model	Multivariable model
	%	*percentage points (%)*		%	*percentage points (%)*	
Total	100.0	11.9[3] (100[4])	12.5[3] (100[4])	100.0	0.9[3] (100[4])	0.4[3] (100[4])
Parental education						
Less than HS graduate	18.1	5.2 (44)	5.1 (41)	9.0	1.2 (46)	0.9 (53)
HS graduate	37.0	2.5 (21)	2.9 (23)	33.2	0.6 (21)	0.3 (20)
Some college	25.7	3.6 (30)	3.7 (30)	30.5	−0.1 (−5)	−0.1 (−6)
College graduate	19.1	0.6 (5)	0.7 (6)	27.3	−0.8 (−28)	−0.4 (−21)
Family structure						
Single mother	44.8	6.7 (57)	6.7 (46)	15.0	0.6 (44)	0.7 (48)
Two biological parents	30.5	3.4 (29)	5.2 (36)	64.1	0.3 (23)	0.3 (18)
Two parents, ≥1 nonbiological	13.6	1.5 (13)	1.7 (12)	17.8	−0.3 (−19)	−0.4 (−25)
Other	11.1	0.1 (1)	−0.9 (−6)	3.1	0.2 (15)	−0.1 (−9)
Female caregiver's age at child's birth						
<19 y	13.1	1.6 (13)	1.1 (9)	5.9	0.7 (29)	0.7 (24)
19–24 y	39.3	5.1 (42)	4.7 (38)	36.7	0.3 (12)	0.6 (20)
25–34 y	35.7	3.3 (27)	3.6 (29)	50.6	−0.5 (−24)	−1.1 (−38)
35–44 y	8.8	1.3 (11)	1.7 (14)	5.7	0.6 (25)	0.3 (11)
≥45	3.1	0.8 (6)	1.2 (10)	1.1	0.2 (9)	0.2 (6)
No. of children in household						
1	25.5	2.0 (17)	2.1 (17)	26.9	1.3 (56)	0.9 (56)
2	32.8	3.5 (29)	4.1 (33)	42.6	−0.4 (−16)	−0.2 (−14)
3	20.2	1.9 (16)	2.1 (17)	20.9	−0.4 (−15)	−0.3 (−21)
≥4	21.5	4.4 (37)	4.1 (33)	9.7	0.3 (13)	0.1 (9)
No. of children in full sibship						
1	29.5	3.1 (27)	—	19.0	−0.2 (−12)	—
2	29.4	4.8 (41)	—	41.0	−0.1 (−7)	—
3	19.7	0.4 (3)	—	26.4	0.2 (15)	—
≥4	21.5	3.4 (29)	—	13.6	1.0 (66)	—
Birth order						
First born	54.0	8.4 (71)	9.0 (75)	52.4	−0.7 (−30)	−1.3 (−42)
Second born	24.6	0.4 (3)	0.6 (5)	31.2	0.3 (14)	0.5 (15)
Third born	11.8	1.9 (16)	1.9 (16)	11.3	0.8 (32)	0.8 (26)
≥Fourth born	9.7	1.1 (9)	0.6 (5)	5.2	0.6 (25)	0.5 (17)

[1] HS, high school.
[2] These population weights are race-specific averages of males' and females' population distributions of the variable, adjusted for Add Health's clustered sampling design and unequal probability of selection.
[3] These decomposition estimates (from the modified Kitagawa decomposition technique) divide the overall female-male disparity into stratum-specific components. Because of rounding, the components may not add up to the total female-male disparity (all values in this column).
[4] These decomposition estimates (from modified Kitagawa decomposition) express the stratum-specific components of the female-male disparity as percentages of the sum of the absolute values of the stratum-specific components of the female-male disparity (all values in this column).

DISCUSSION

We used an innovative methodologic approach to directly study the gender disparity in obesity prevalence in young black adults in the United States. To our knowledge, this study was the first to examine family factors as possible correlates of the gender gap in obesity prevalence in blacks. Parental education was the only sociodemographic variable that was strongly associated with the gender disparity in obesity in blacks. In fact, nearly half of the overall gender gap was concentrated among the one-fifth of young black adults whose parents did not complete high school. Whereas young black women from low-education families were at the greatest risk of obesity, young black men from these same families appeared to be at the lowest risk.

Although US research addressing gender disparities in obesity prevalence is scarce, there is a body of relevant work examining international differences in obesity prevalence (31–35). The association between SES and obesity varies both by gender and by a country's degree of economic development [as assessed by the United Nations' Human Development Index (HDI)] (35). For men in poor, low-HDI nations, SES is positively associated with obesity; studies of men in medium-HDI nations (eg, Brazil and Saudi Arabia) find fewer positive associations and more null associations; and in men from high-HDI nations (eg, the United States and Australia), null and negative associations are most common. Women in low-HDI and medium-HDI countries tend to show trends similar to those observed for men, but, in high-

HDI countries, negative associations are more common in women than in men (35).

Our findings for whites conformed to patterns observed for high-HDI countries, but our findings for blacks did not. The gender disparity in young black adults more closely resembled the profile of a medium-HDI country, where obesity prevalence is usually much higher in women than in men (32, 36). Within medium-HDI and high-HDI nations, historical subgroups can be classified as living in contexts less developed than the larger nation's because the subgroup is not fully integrated into the nation's social and economic systems (33, 35). As in American Indians in the United States or in Maoris in New Zealand, black Americans in the high-HDI United States could be classified as living in a medium-HDI context: legally sanctioned mechanisms barred them from full participation in American social and economic life until well into the 20th century (37).

Moreover, the divergent associations between SES and obesity that we observed in young US blacks (men: positive; women: inverse) resemble those of a medium-HDI country in the midst of rapid nutrition transition. In these countries, the obesity burden shifts from high-SES individuals to low-SES individuals (34), and this shift generally occurs in women before it occurs in men (31, 34, 38). Thus, the prevalence of obesity in women may be inversely associated with SES, while the prevalence of obesity in men may retain its positive association. Furthermore, as obesity prevalence stabilizes or decreases among women of high SES, it may continue to increase among men, causing the gender gap to decrease. However, over the past 35 y, the magnitude of the gender disparity in obesity among US black adults has not decreased (2, 4). Further research should explore whether the obesity patterns of black Americans conform to the model observed in rapidly developing medium-HDI nations.

Our results appear to differ from conclusions reached by a recent review, which argued that, in blacks with less than a high school education, women had a lower and men had a higher prevalence of obesity relative to other black adults of the same gender (20). This conclusion was based largely on Zhang and Wang's analysis of the 1999–2000 NHANES data set (39). Several aspects of that analysis differed from ours. Most notably, the 1999–2000 NHANES analysis studied adults aged 20–60 y (39), whereas the average age of our cohort was ≈21 y; a previous study found age differences in the gender-specific relations between obesity and SES for young black adults aged <30 y relative to older black adults (19). In addition, the 1999–2000 NHANES analysis (39) used ORs, whereas we used prevalence differences; they examined an adult's own educational attainment whereas we used parental education; their education categories (≤9th grade, 10th–12th grade, and college or higher) differed from ours (<12th grade, some college, college degree, and higher than college); and their sample of blacks was smaller than ours, which resulted in more unstable estimates.

Our analysis was not without limitations. First, we examined a narrow age range within young adulthood; therefore, our findings may not generalize to older adults. Second, we did not investigate the respondent's adult SES independent of his or her parents' educational attainment; the age range examined is a highly complex transitional period, in which it is difficult to classify SES independent of family of origin. Third, we were limited in using preexisting data. For instance, family structure and number of minors in the household were only examined during adolescence; perhaps examination at a younger age or of cumulative exposure over a longer period would have produced different results. Furthermore, compared with tests of main effects in data sets of the same size, tests of modification tend to have relatively low power, which would increase the likelihood of failing to detect an association (28, 29, 40, 41). Finally, there was a possibility of differential selection bias by gender, especially among blacks. Only students enrolled in school were eligible for the study, and black males drop out of school at a higher rate than do black females. In addition, fewer black male respondents than female respondents were retained in the young adult follow-up sample. However, the Add Health sample weights were designed to account for this differential loss-to-follow-up.

Our study had many strengths. To our knowledge, Add Health is the only nationally representative data set with an adequate sample size and diversity of variables suitable for this work. Furthermore, blacks with college-educated parents were oversampled, which allowed us to produce relatively precise estimates for parental education. In addition, we had access to both individual-level (reported by the respondents) and household-level (reported by the respondents as well as their parents) variables, which is rare in a study of this size. Height and weight data were measured by trained staff; there is evidence that associations between obesity and SES differ when anthropometric data are self-reported rather than measured (35). Additionally, retention rates were good over the 7-y follow-up period. Finally, our analysis of effect modification of prevalence differences allowed us to make meaningful comparisons across race and across exposure variables.

It remains a puzzle why black boys and girls from similar genetic, family, and community backgrounds have such different risks of developing obesity by young adulthood. In low-SES families, it is apparent that black sons and daughters either adopt very different and gender-specific obesity-related behaviors or that obesity development occurs through different mechanisms for these young women and men. We found that parental education, but not other examined family factors, strongly predicted the degree to which obesity prevalence differed by gender. Perhaps, community characteristics related to SES are more important than within-family dynamics in explaining the gender difference in obesity prevalence in young adults. Future research on the causes underlying the decades-old gender difference in obesity prevalence among US blacks should examine mechanisms by which behavioral and community characteristics of blacks from low-SES families may differentially affect obesity risk in males and females during adolescence and young adulthood.

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