The Role of Adolescent Behaviors in the Female–Male Disparity in Obesity Incidence in US Black and White Young Adults

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In the United States, black women are at much greater risk for obesity than black men. We explored whether adolescent behaviors (family dinners, hours of television, playing sports with mother, playing sports with father, bouts of physical activity) were associated with gender disparity in 6-year obesity incidence in young adulthood. We used data from the nationally representative National Longitudinal Study of Adolescent Health to examine adolescent behaviors in nonimmigrant black (*n* = 1,503) and white (*n* = 4,452) youths in 1994–95 (aged 11–19 years) and 1995–96 (aged 12–20). We assessed gender disparity in obesity incidence (female incidence minus male incidence) during young adulthood (2001–02; aged 18–26). Standardized gender disparities were calculated using race- and gender-stratified, covariate-adjusted logistic regression models in which males and females were set to the same distributions of adolescent behaviors. In adolescence, black females reported less leisure-time physical activity and lower likelihood of playing sports with either parent compared with black males. Setting adolescent behaviors equal for black males and females did not reduce the estimated gender disparity in obesity incidence (nonstandardized: 9.8 percentage points (95% confidence interval (CI): 4.5, 15.1); fully standardized: 10.2 percentage points (5.2, 15.2)). There was little gender disparity in whites before or after adjustments. To our knowledge, this is the first study to examine to what extent behavioral differences during adolescence might account for gender disparity in obesity incidence in black young adults. Male-female differences in these adolescent behaviors did not appear to underlie the gender gap in young adult obesity.

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INTRODUCTION

In the United States, black women are at much greater obesity risk than black men. In fact, the gender disparity in obesity between black women and men is much larger than the racial disparity between blacks and whites (1). In the 2003–2006 National Health and Nutrition Examination Surveys, the difference in obesity prevalence between black and white adults was 12.1 percentage points (black: ~44.5% obese; white: ~32.4%) (2). However, the difference between black women and men was 17.4 percentage points (black women: 53.2% obese; black men: 35.8%) (2). There was virtually no gender difference between white women and men (white women: 32.2% obese; white men: 32.6%) (2).

Although the gender gap in obesity prevalence in blacks is well-known, it is unclear what factors underlie this disparity (3). The obesity gender disparity in black adults may be influenced by gender differences in behaviors during childhood (which we define as the period between birth and adulthood) (4). Childhood experiences appear to have lasting consequences for adult obesity risk (5). Further, some of these childhood factors, such as socioeconomic position, are differentially associated with adult obesity risk in men vs. women (3,4,6). These differential associations suggest that gender differences in behavioral responses to childhood environments may contribute to the gender disparity in obesity in black adults. Lower levels of physical activity in black females vs. black males may be an important factor in the adult gender gap (7,8). Further, parenting behaviors in the childhood family may have lasting consequences for adult obesity risk (8–10). If boys and girls living together in the same households were treated differently by their parents in regard to physical activity and eating behaviors, this differential treatment could partially explain why men and women from similar family backgrounds arrive at different obesity outcomes.

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We investigated whether gender disparities in obesity in young adulthood may be attributable to gender differences in behaviors during late childhood. We specifically examined behaviors associated with physical activity, physical inactivity, and parental interactions. Using data from a nationally representative prospective cohort study of US adolescents followed into young adulthood, we examined whether selected adolescent behaviors (family dinners, television-viewing, playing sports with a mother, playing sports with a father, and bouts of leisure-time physical activity) might contribute to gender disparities in obesity incidence in black and white young adults. We conducted all analyses in whites as well as blacks to provide a comparison group in which to test the consistency of our findings in blacks.

METHODS AND PROCEDURES

Population

Data were from the National Longitudinal Study of Adolescent Health (Add Health) longitudinal cohort, which began as a nationally representative survey of US public and private school students enrolled in grades seven through 12 in 1994–95. The survey was cluster sampled by school and oversampled some subgroups, including black students with a parent who completed college or attained a professional degree.

At wave I (1994–95), detailed questionnaires were administered to each student and to the student's primary in-residence caregiver, preferentially a female. At the wave II visit (1995–96), all students except those who were in twelfth grade at wave I were reinterviewed, and height and weight were measured. At wave III (2001–02), all study respondents surveyed at wave I were reinterviewed; height and weight were measured for the second time. All study procedures described below were approved by the Institutional Review Board of the University of North Carolina at Chapel Hill.

Exclusions

The sample was limited to those eligible to be interviewed at all three study visits, e.g., those in twelfth grade at wave I were not interviewed at wave II and thus were excluded from our analysis. Also, we restricted the analysis sample to non-Hispanic blacks and whites (defined by a combination of child self-report and parent self-report (3)) who had at least one parent born in the United States because of evidence that the health behaviors and obesity prevalence of immigrant black adults are different than in blacks with multiple generations of residence in the US (11,12).

Overall, 71.6% of those eligible for follow-up at waves II and III participated in both follow-up interviews (74.2% of black females, 64.3% of black males, 75.8% of white females, 68.9% of white males). Of these 7,679 respondents, 9.4% were excluded for one or more of the following reasons: outside the desired age range (0.1%); missing baseline exposure or covariate information (5.9%); missing measured and self-reported height or weight at 1- or 7-year follow-up (1.3%); or pregnant at wave II or wave III weighing (3.6% overall (8.4% of black women, 5.9% of white women)). Self-reported data were substituted when measured height or weight were missing: 1.3% of respondents at wave II and 4.3% of respondents at wave III. Finally, 1,063 (13.8%) of eligible respondents were excluded because they were already obese at wave II and thus not at risk for incident obesity. The final analysis sample consisted of 1,503 black and 4,452 white respondents.

Those who were excluded from the analysis were older (χ^2 $P < 0.01$) and more likely to be black, especially black and female ($P < 0.01$). Those excluded were also more likely to eat dinner with their parents daily ($P = 0.01$), were less likely to have played a sport with a father in the past month ($P < 0.01$), and reported fewer bouts of leisure-time physical activity ($P < 0.01$). There was no difference in sport with a mother between the excluded and the analysis sample in blacks or whites ($P = 0.63$ and $P = 0.42$, respectively).

Outcomes

The main outcome was gender disparity in young adult obesity incidence: female obesity incidence minus male obesity incidence. Incident obesity, defined as adult BMI ≥30 kg/m^2 (13), was assessed at wave III in those who were nonobese (i.e., "underweight," "normal weight," and "overweight") at wave II. Wave II obesity was defined as (i) BMI ≥95th percentile of the age- and sex-specific CDC 2000 cutpoint or (ii) BMI ≥30.0 kg/m^2 (9).

Exposures: adolescent behaviors

Exposure variables were self-reported by the respondent on the Add Health wave I and wave II in-home questionnaires. The behaviors were (i) frequency of dinners with parents (how many days in the past week a parent was present during the respondent's evening meal, average of wave I and wave II); (ii) hours of television-viewing (number of hours the respondent watched television in a typical week, wave II); (iii) sports participation with a mother (whether respondent reported playing a sport with his/her biological mother or resident female caregiver in the past month at either wave I or wave II); (iv) sports participation with a father (whether respondent reported playing a sport with his/her biological father or resident male caregiver in the past month at either wave I or wave II); and (v) frequency of leisure-time moderate-to-vigorous physical activity (average of wave I and wave II moderate-to-vigorous physical activity scores). Moderate-to-vigorous physical activity score ranged from 0 to 9 and was the sum of responses to three questions (each scored from 0 to 3) about the past week's engagement in selected activities of 5–8 metabolic equivalents: (i) "active sports," (ii) "exercise," and (iii) skating/biking. The three questions were scored according to reported frequency of the activity: 0 for "0 times per week", 1 for "1–2 times per week", 2 for "3–4 times per week," and 3 for "5 or more times per week".

Covariates

Parental education, based on adolescent and parental report, was defined as the highest education attained by either of the respondent's biological parents: less than high school graduate; high school graduate; vocational degree or some college; or college or professional degree. Age at last birthday was modeled categorically, in 1-year increments. When small samples sizes did not support 1-year increments, age categories were collapsed into 2- or 3-year groupings. Gender, which we conceptualized as the joint expression of biological and cultural influences (14), was defined by a variable for biological sex.

Data analysis

All variables were analyzed as nominal categorical variables. χ^2-tests were used to evaluate whether the distributions of the behavior variables differed by gender in each race group. Then, as explained below, we evaluated whether setting females and males equal on the distributions of the behavior variables reduced estimated female–male differences in obesity incidence. All tests were adjusted for Add Health's complex sampling design and weighted to correct for loss to follow-up (15).

We examined the female–male difference in obesity incidence before and after standardizing for the behavior exposure variables. Standardizing was used to set males and females to be equal on distributions of the variables. First, using multivariable-adjusted logistic regression, we generated coefficients to estimate the obesity incidence associated with each category of each exposure variable. The logistic regression models were race-stratified with obesity at wave III as the dependent variable; age, parental education, and gender as covariates; and interactions between gender and all other variables. The interactions allowed us to calculate coefficients separately for males and females, which was indicated by previous research showing gender-specific effects of obesity risk factors

Table 1 Sample characteristics before exclusion of those obese in adolescence, the National Longitudinal Study of Adolescent Health, 2001–02

	Black		White	
	Male	Female	Male	Female
	n = 831	*n* = 1,018	*n* = 2,499	*n* = 2,648
	% (s.e.)[a]	% (s.e.)[a]	% (s.e.)[a]	% (s.e.)[a]
Mean age (years), young adulthood	21.6 (0.2)	21.4 (0.2)	21.4 (0.1)	21.2 (0.1)
Parental education (%)				
<High school degree	14.7 (2.9)	19.3 (2.8)	8.8 (1.1)	7.8 (1.0)
High school graduate	34.6 (2.8)	36.9 (2.3)	32.2 (1.7)	34.0 (1.5)
Some college/vocational degree	28.6 (2.8)	25.9 (2.5)	31.3 (1.2)	29.5 (1.2)
College or professional degree	22.1 (3.0)	17.9 (2.3)	27.8 (2.1)	28.6 (2.1)
Weight status, adolescence (%)[b]				
Underweight	1.2 (0.4)	2.5 (0.7)	4.4 (0.5)	2.4 (0.4)
Normal weight	63.9 (2.3)	55.7 (1.8)	63.7 (1.4)	72.7 (1.4)
Overweight	18.4 (1.6)	21.4 (1.2)	16.6 (0.9)	13.6 (0.9)
Obese	16.5 (2.0)	20.5 (1.7)	15.4 (1.2)	11.3 (1.0)
Weight status, young adulthood (%)[c]				
Underweight	1.3 (0.4)	3.0 (0.7)	2.6 (0.5)	4.4 (0.5)
Normal weight	48.3 (2.5)	35.7 (1.7)	45.8 (1.3)	55.0 (1.5)
Overweight	26.6 (2.0)	26.3 (1.6)	30.6 (1.1)	19.6 (1.0)
Obese	23.7 (2.3)	35.0 (2.2)	20.9 (1.2)	21.1 (1.4)
Incident obesity, young adulthood (%)[d]	12.3 (1.8)	21.2 (2.1)	11.1 (0.9)	12.2 (1.0)

[a]Percentages and s.e. are adjusted for Add Health's complex sampling design and weighted to correct for loss to follow-up. [b]Consistent with Expert Committee Recommendations Regarding the Prevention, Assessment, and Treatment of Child and Adolescent Overweight and Obesity: Underweight: <5th percentile, age- and sex-specific CDC 2000, and BMI <18.5; Normal weight: (BMI ≥5th percentile or BMI ≥18.5) and (BMI <85th percentile and BMI <25.0); Overweight: (BMI ≥85th percentile or BMI ≥25.0) and (BMI <95th percentile and BMI <30.0); Obesity: BMI ≥95th percentile or BMI ≥30.0 (6). [c]WHO/NIH cutpoints: BMI <18.5, 18.5–24.9, 25.0–29.9, ≥30.0 (5). [d]Samples exclude those obese in adolescence. Black males: *n* = 700; Black females: *n* = 803; white males: *n* = 2,140; white females: *n* = 2,312.

(16,17). We used gender interactions rather than run gender-stratified models in order to keep males and females in the same models and thus facilitate calculation of 95% confidence intervals (CIs) for the female–male incidence difference.

In the second step of the standardization process, the coefficients obtained from logistic regression were used to form race- and gender-specific formulas for the calculation of obesity incidence standardized across gender for the exposures and covariates (18). Applying these formulas, values of each exposure and covariate category were set to the race-specific average of the proportion of male and female respondents in that category. Thus, males and females of the same race group were set to equal values for every exposure and covariate category. We then calculated standardized incidence differences as the standardized obesity incidence in females minus that in males.

An obesity incidence difference of 0 represented equal obesity incidence for men and women. Values greater than 0 indicated greater incidence in women than men; less than 0 indicated greater incidence in men. The 95% CIs for the incidence differences were calculated using the delta method (15,18).

Further, we calculated standardized incidence differences in blacks and whites whose parents did not complete high school, given the relatively large gender disparities observed in these groups (3). We also ran analyses examining transitions across finer categories of weight status. We examined incident overweight among those nonoverweight (i.e., normal weight or underweight) at baseline; incident obesity among those nonoverweight (i.e., normal weight or underweight) at baseline; and incident obesity among those overweight but nonobese at baseline. Incident overweight and obesity at wave III were defined as adult BMI between 25.0 and 30.0 kg/m^2 and adult BMI ≥30.0 kg/m^2, respectively (13). Baseline nonoverweight was defined as (i) BMI <85th percentile of the age- and sex-specific CDC 2000 cutpoint and (i) BMI <25.0 kg/m^2 (9). Baseline overweight but nonobese was defined as (BMI ≥85th percentile of the CDC 2000 pediatric cutpoints or BMI ≥25.0 kg/m^2) and (BMI <95th percentile of the CDC 2000 pediatric cutpoints and BMI <30.0 kg/m^2) (9).

RESULTS

Table 1 shows the distributions of age, parental education, and weight status before exclusion of the respondents who were obese in adolescence. The ages of respondents ranged from 12 to 20 years when adolescent weight status was measured. When young adult weight status was measured 6 years later, respondents were aged 18–26 years. In both adolescence and young adulthood, black females were more likely than black males to be obese. Among whites, males were more likely than females to be obese in adolescence; by young adulthood, obesity prevalence was similar in white males and females. Over the 6-year period between wave II and wave III, the estimated incidence difference for obesity in blacks was 8.9 percentage points (95% CI: 3.8, 14.0); the incidence difference in whites was −1.1 percentage points (−1.3, 3.5).

Table 2 Distributions of exposure variables in adolescence in analysis sample, by race and gender, with tests for gender differences in each race group, the National Longitudinal Study of Adolescent Health, 1995–1996

	Black			White		
	Male	Female		Male	Female	
	n = 700	*n* = 803		*n* = 2,140	*n* = 2,312	
	% (s.e.)[b]	% (s.e.)[b]	*P* value[a]	% (s.e.)[b]	% (s.e.)[b]	*P* value[a]
Dinners with parent per week			0.40			0.29
0–2.5	36.9 (2.2)	32.1 (2.2)		14.1 (1.2)	15.8 (1.5)	
3–4.5	32.2 (2.8)	31.2 (2.6)		22.0 (1.4)	23.7 (1.3)	
5–6.5	16.4 (1.9)	19.3 (2.1)		34.8 (1.2)	32.2 (1.4)	
7	14.6 (2.2)	17.3 (1.9)		29.1 (1.9)	28.4 (1.9)	
Sport with mother			0.02			0.66
In past month	15.8 (2.1)	10.4 (1.6)		17.8 (1.2)	18.4 (1.1)	
Sport with father			<0.01			<0.01
In past month	33.1 (3.2)	16.7 (1.6)		49.7 (1.6)	38.7 (1.6)	
Bouts of MVPA per week			<0.01			<0.01
0–1.5	7.1 (1.1)	22.0 (2.3)		10.4 (0.9)	16.6 (1.3)	
2–3.5	31.1 (3.0)	49.5 (2.7)		29.3 (1.5)	39.4 (1.3)	
4–5	34.5 (3.2)	19.4 (2.2)		27.8 (1.1)	26.3 (1.1)	
5.5+	27.4 (2.7)	9.1 (1.5)		32.5 (1.6)	17.7 (1.5)	
Television hours per week			0.25			<0.01
0–4	15.4 (1.8)	15.3 (1.4)		20.4 (1.1)	29.8 (1.7)	
5–8	20.7 (2.1)	19.4 (1.8)		20.3 (1.2)	24.3 (1.2)	
9–13	9.8 (1.3)	14.1 (1.9)		17.1 (1.0)	16.0 (1.1)	
14–18	8.6 (1.4)	6.7 (1.2)		13.7 (1.0)	11.0 (1.1)	
19–21	10.2 (1.5)	8.7 (1.2)		11.2 (0.9)	7.2 (0.8)	
22+	35.4 (2.6)	35.9 (2.6)		17.2 (1.2)	11.8 (1.1)	

[a]*P* values test gender differences in distributions of variables. The *P* values are from χ^2-tests adjusted for Add Health's complex sampling design and weighted to correct for loss to follow-up. [b]Variable distributions and standard errors are adjusted for Add Health's complex sampling design and weighted to correct for loss to follow-up.

Table 2 shows the distributions of the exposure variables in the analysis sample, which excluded respondents who were obese in adolescence. In whites and blacks, adolescent males reported more bouts of leisure-time physical activity than adolescent females and were more likely to report sport with a father. White males reported watching more television than white females. In blacks, there was no gender difference in television-viewing, but black males and females as a group reported watching more television than either white males or females.

None of the adolescent behaviors was consistently related to obesity incidence in young adulthood. For instance, χ^2-tests examining the associations between adolescent physical activity and obesity incidence yielded *P* values >0.20 in all four race/gender groups. The same was true for adolescent television-viewing and dinner with parents (data not shown). Sports participation with a father tended to be associated with lower obesity incidence in black men (34.2% of nonobese did sports with father vs. 24.9% in obese; $P = 0.09$) but not in black women (16.0% of nonobese did sports with father vs. 19.3% in obese; $P = 0.48$). Sports participation with a mother tended to be in the protective direction (associated with lower obesity incidence) in black men ($P = 0.55$) and black women ($P = 0.07$). In white males and females, neither sports participation with a mother nor sports participation with a father was associated with obesity incidence (sports with mother: male $P = 0.20$, female $P = 0.17$; sports with father: male $P = 0.16$; female $P = 0.99$).

To assess the degree to which behavioral differences during adolescence might contribute to the gender gap in obesity in young adulthood, we compared behavior-standardized incidence differences to the incidence difference standardized only for age and parental education (Table 3). If gender differences in the adolescent behaviors contributed to the young adult obesity gender gap, then we would expect the behavior-standardized incidence differences to be smaller than the incidence differences standardized only for age and parental education. This was not seen in our analyses. In blacks, there was little suggestion that setting males and females at the same distribution of any given adolescent behavior resulted in a smaller gender difference in obesity incidence. The same was true even when all behaviors were included in the same model

Table 3 Incidence of young adult obesity (BMI ≥30.0), by race and gender, with males and females standardized to the same race-specific distributions of adolescent exposure variables

	Black			White		
Standardization variables	Incidence difference (95% CI)	Female incidence (s.e.)	Male incidence (s.e.)	Incidence difference (95% CI)	Female incidence (s.e.)	Male incidence (s.e.)
Parental education and age[a,b]	9.8 (4.5, 15.1)	20.2 (2.2)	10.4 (1.5)	0.7 (–1.5, 3.0)	11.3 (0.9)	10.6 (0.9)
Dinner with parent per week[b,c]	9.9 (4.7, 15.0)	20.2 (2.1)	10.3 (1.4)	0.8 (–1.5, 3.0)	11.3 (0.9)	10.5 (0.9)
Sport with mother[b,c]	9.2 (4.1, 14.4)	19.7 (2.1)	10.4 (1.5)	0.8 (–1.5, 3.1)	11.3 (0.9)	10.5 (0.9)
Sport with father[b,c]	10.0 (4.8, 15.2)	20.6 (2.1)	10.6 (1.5)	0.8 (–1.5, 3.1)	11.4 (0.9)	10.6 (0.9)
Bouts of MVPA per week[b,c]	10.9 (5.7, 16.0)	20.9 (2.2)	10.1 (1.6)	0.8 (–1.5, 3.1)	11.1 (0.9)	10.3 (0.9)
Television hours per week[b,c]	9.3 (4.1, 14.6)	19.6 (2.1)	10.3 (1.5)	0.9 (–1.3, 3.2)	11.4 (0.9)	10.4 (0.9)
All behavioral variables[b,d]	10.2 (5.2, 15.2)	20.1 (2.2)	9.8 (1.5)	1.1 (–1.1, 3.4)	11.3 (0.9)	10.1 (0.9)

[a]Independent variables in model: sex + age + sex × (age) + parental education + sex × (parental education). [b]For age variable, ages 18 and 19 and ages 25 and 26 were collapsed into categories of 2-year increments. [c]Other independent variables in model: sex + age + sex × (age) + parental education + sex × (parental education) + sex × (standardization variable). [d]Independent variables in model: sex + age + sex × (age) + parental education + sex × (parental education) + dinners per week + sex × (dinners per week) + sport with mom + sex × (sport with mom) + sport with dad + sex × (sport with dad) + bouts of physical activity + sex × (bouts of physical activity) + hours of television + sex × (hours of television).

(Table 3). Likewise, in whites, the gender difference remained virtually unchanged after standardization.

We also computed standardized incidence differences for young men and women whose parents did not complete high school. The incidence differences were computed using the same logistic models described above, but, for all respondents, parental education was set to "less than high school graduate." In blacks, the incidence difference standardized for all behaviors (28.8 percentage points (95% CI: 13.3, 44.2)) was similar to the incidence difference standardized only for age (26.6 (95% CI: 12.9, 40.4)). The same was true in whites: the fully behavior-standardized incidence difference was 14.9 percentage points (95% CI: 3.2, 26.7); the age-standardized incidence difference was similar: 12.3 percentage points (95% CI: –0.7, 25.4).

Similarly, in supplementary analyses of incident overweight or obesity among those normal weight or overweight at baseline, standardizing for the behaviors did not consistently change incidence differences for overweight or obesity between women and men (see **Supplementary Tables S1–S4** online). However, the tables demonstrate that the magnitude and even direction of gender disparity varies for different body size transitions. For instance, overweight black female adolescents were overwhelmingly more likely than overweight black male adolescents to become obese as young adults (age- and parental education-adjusted incidence difference: 32.6 percentage points), but the gender difference for nonoverweight adolescents becoming overweight as young adults was much smaller (age- and parental education-adjusted incidence difference: 6.4 percentage points). In whites, patterns of gender disparity reversed in some cases. Although overweight white female adolescents were more likely than their overweight male counterparts to become obese as young adults (age- and parental education-adjusted incidence difference: 11.1 percentage points), nonoverweight white females were less likely than their male counterparts to become overweight as young adults (age- and parental education-adjusted incidence difference: –10.6 percentage points). Patterns of gender disparity in excess weight gain appear to vary across BMI. These variations merit further research.

DISCUSSION

The adolescent behaviors examined here did not appear to have contributed to the higher incidence of obesity in young black women vs. young black men. Although black adolescent females engaged in less leisure-time physical activity and were less likely to participate in sports with their parents than black adolescent males, these behaviors were not associated with obesity incidence over the 6-year follow-up period. Therefore, setting these physical activity-related variables to the same distributions for males and females did not reduce the estimated obesity gender gap in obesity incidence in black young adults.

It is possible that gender differences in body size preferences (which we did not examine) contribute to the large gender difference in obesity risk in black young adults. Available evidence suggests that, adjusted for socioeconomic position and BMI, blacks and whites of the same-sex share similar ideal body sizes (19) but diverge on perceptions of their current body sizes: blacks appear less likely to describe themselves as "overweight" (20,21) and, on average, choose smaller figures to represent their current body sizes compared to same-sex whites of the same BMI (19,22). Although self-perception of current body size differs by sex, the sex differences between black men and women appear similar in magnitude to the differences between white men and women (19–22), implying little role for body size self-perception in the greater gender disparity observed in blacks vs. whites, implying, imply. Additionally, it remains unclear whether preference for a smaller body size leads to lower obesity incidence (23): available evidence suggests that, in the US, greater body size dissatisfaction is associated with greater weight gain (24–28). Although the literature is sparse, there is little evidence to suggest that a cultural preference

for larger female body size underlies the gender disparity in obesity prevalence in US blacks.

There are important limitations of our work. First, the behavioral factors we investigated were measured late in childhood: the average age of respondents was 15 and 16 years when the behavioral factors were measured. Gender differences in obesity risk may have their roots earlier in childhood. Alternatively, behaviors may not be associated with obesity incidence across a 6-year lag if the behaviors are short-acting and unstable over time or if middle to late adolescence is not the relevant age window. Further, our failure to find associations between the adolescent behaviors and obesity incidence could be due to measurement error in the exposure variables (29,30). For instance, our measure of physical activity was self-reported; did not capture intensity or duration of activities; nor did it capture activity associated with occupational and other nonleisure-time activities. Failure to find associations could also be attributable to reverse causation (i.e., adolescents with an unmeasured propensity towards obesity may be more likely to engage in behaviors perceived to prevent obesity), which could have obscured relationships between behaviors and incident obesity. Additionally, BMI may be less sensitive in identifying obesity in men than in women (31), which could lead to overestimation of females' excess obesity risk in both black and white samples. Finally, we were unable to incorporate dietary intake into our analyses. Depending on the relationships among dietary intake, the independent variables, and incident obesity, failure to model dietary intake could have obscured protective or positive effects of the adolescent behaviors.

To our knowledge, our study is the first to investigate whether adolescent and parental behaviors might be associated with the female–male disparity in obesity incidence in young adulthood. Our study sheds light on potential mechanisms behind gender differences in obesity development. Understanding these mechanisms is critical for tailoring interventions that will be effective in reducing obesity prevalence in US males and females. Other study strengths are that the dataset was prospective, nationally representative, and had sufficient sample size and diversity to allow stratified analysis by both race and gender. For the majority of respondents, height and weight were measured, which may be especially important in investigating gender differences because reporting bias for height and weight varies by gender (32). Additionally, we assessed the gender disparity on an absolute scale, which gives a better sense of public health impact than disparities assessed on relative scales (33). Moreover, the disparity measure we used was "decomposable": we could quantify how much of the disparity might be associated with each behavior (33,34). Finally, our study is one of the few to stratify by both race and gender rather than adjust for them as covariates (35). Failure to jointly investigate race and gender as obesity risk factors may obscure the true relationships between gender and obesity and between race and obesity.

Although the examined behaviors differed by gender during adolescence, the behaviors were not strongly associated with obesity incidence. These weak findings replicate the results of population-based randomized trials aimed at preventing excess weight gain (36–38). Although experts can control the weights of research participants in clinic- and lab-based settings by manipulating dietary intake and physical activity, that knowledge does not translate perfectly to free-living populations: interventions in naturalistic settings do not consistently prevent weight gain over the long term (36–38). Future research on the gender disparity in black Americans should investigate behavioral traits which have predicted excess weight gain in adolescents or young adults in longitudinal, observational studies. For example, lower fitness is associated with weight gain, and black females appear to be less fit than black males (39,40). Second, a fast rate of decline in physical activity may be associated with excess weight gain independent of baseline activity level (41–43). Although little is known about declines in physical activity in black adolescent males, physical activity declines dramatically in most black girls during adolescence (44). Other topics worthy of further investigation include breakfast-skipping (45,46); dieting behaviors, which are more common in females than males and which may predispose adolescents to excess weight gain (47); pregnancy-related weight gain (48); differential weight-related responses to stress in males and females (3); and differential responses to food insecurity (49).

Despite longstanding evidence of a gender disparity in obesity prevalence in US blacks, the factors underlying this disparity remain unclear. In recognition that men and women mostly share residential environments and originate from similar socioeconomic backgrounds, we recommend three conceptual frameworks that may be useful for guiding future research on gender disparity. First, the framework grounding the present research is that differential treatment of or behaviors in males and females in the same environment may contribute to differential obesity risk. A second relevant framework is an ecological perspective which emphasizes ways in which "shared" environments may be inhabited differently, spatially and socially, by males and females. Third, research should explore whether different pathways of obesity development predominate in one sex vs. another. For example, women may be more physiologically susceptible to obesity given certain environmental stressors than men, or gender-specific life events such as pregnancy may put women at additional risk. Identifying the mechanisms that underlie gender disparity in US blacks will advance efforts to reduce the obesity burden in black Americans and other populations in which obesity disproportionately affects women (3,50–52). Moreover, advancing our understanding of gender disparity in US blacks will help elucidate key aspects of obesity development that remain poorly understood.

SUPPLEMENTARY MATERIAL

Supplementary material is linked to the online version of the paper at http://www.nature.com/oby

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DISCLOSURE

The authors declared no conflict of interest.

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