Under the same roof: Understanding the gender disparity in obesity prevalence in U.S. Black young adults

Whitney Ragan Robinson

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Approved by:

June Stevens (chair)

Penny Gordon-Larsen

Jay S. Kaufman

Kari E. North

Chirayath M. Suchindran

Abstract

WHITNEY ROBINSON: Under the same roof: Understanding the gender disparity in obesity prevalence in U.S. Black young adults
(Under the direction of June Stevens)

Background: In the United States, Black women are at much greater risk for obesity than Black men. Little is known about the factors underlying this disparity. **Objectives:** We explored whether, in U.S. Black young adults, childhood sociodemographic factors (parental education, single-mother household, number of siblings, number of minors in household, birth order, and female caregiver's age) and adolescent behaviors (family dinners, hours of television, playing sports with mother, playing sports with father, bouts of physical activity) were associated with gender disparities in obesity. **Methods:** Analysis datasets were constructed from the nationally representative National Longitudinal Study of Adolescent Health. The datasets included non-immigrant Black and White youths aged 11 to 19 years in 1994-95. Childhood sociodemographic factors (n=7,747) were assessed in 1994-95. Adolescent behaviors (n=5,955) were assessed in 1994-95 and 1995-96. Obesity was measured in 1995-96 and again in 2001-02. For each assessed childhood sociodemographic factor, we evaluated whether the factor modified the female-male prevalence difference. Second, we evaluated whether standardizing Black males and females to the same distributions of the adolescent behaviors reduced the size of the predicted gender disparity in young Blacks. **Results:** In unadjusted and multivariable-adjusted models, parental education consistently modified Blacks' gender disparity (p=0.01). The gender gap was largest at low

parental education (16.7% men obese vs. 45.4% women obese) and smallest at high parental education (28.5% men obese vs. 31.4% women obese). In Whites, there was little overall gender difference in obesity prevalence. Blacks females reported less leisure-time physical activity and lower likelihood of sport with either parent than did Black males. Standardizing by these behaviors did not reduce the predicted gender disparity in obesity incidence.

Discussion: Black young adults' gender disparity in obesity prevalence was concentrated in families with low parental education. Male-female differences in the adolescent behaviors examined did not appear to underlie the obesity gender gap in young U.S. Blacks. Future research should investigate environmental, physiologic, and behavioral factors related to the differential regulation of energy balance in young Black males and females.

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Introduction

> "Probably the most controversial finding from twin studies is the relatively low shared-environment effect . . .. Discussions about the obesity epidemic almost invariably ascribe a key role to the family, but, in the present study . . . siblings from the same family were only slightly more similar in adiposity than would be expected from their genetic similarity . . .." -- Wardle, J. et al. 2008 (1).

In the lay press, childhood obesity often seems to be portrayed as a syndrome partially attributable to poor parental oversight of children's health. However, in my lay experience in Black communities, many of the heavier boys and girls were living in families where their health, well-being, and educational achievement were being attended to. In particular, most of the heavy Black boys I knew were being raised by attentive and financially secure parents. On the other hand, the children, especially the boys, with the most dysfunctional families often seemed to me to be at uniquely low risk for obesity.

Against this backdrop, I became animated by the idea of studying the gender disparity in obesity risk in Black young adults. I believed that the topic presented an original perspective from which to investigate the role of the childhood family environment in influencing adult obesity risk. Because Black boys and girls are largely raised in the same families, differences in the distribution of childhood sociodemographic factors could not be responsible for the female-male disparity. In addition, because I was studying people of the same racial background, I would be free to set aside the view that Black Americans' obesity patterns are determined by unique attributes of their genetic profiles. This work presented a

novel perspective from which to explore how childhood family context may influence later susceptibility to obesity.

Critical Review of the Literature

It is well established that Black women have higher obesity prevalence than Black men (2, 3). According to the 1999-2002 National Health and Nutrition Examination Survey (NHANES), Blacks' gender difference in obesity prevalence was 21.1 percentage points: 49.0% of Black women were obese while only 27.9% of Black men were obese (4). This prevalence difference was much larger than that observed in Whites, in whom there was virtually no gender disparity in obesity prevalence. In the 1999-2002, 30.7% of White women were obese compared to 28.2% of White men (4).

NHANES has monitored gender-specific obesity prevalence for the past 35 years (5). During most of that time, the gender disparity among U.S. Blacks remained stable at about 15 percentage points but has increased to 20 percentage points in the most recent surveys (5, 6). In adults aged 20 to 40 years old, recent data do not indicate secular changes in the magnitude of the gender gap. The 1999-2002 estimate was 21.6 percentage points (5). The 2001-2004 estimate was 21.5 percentage points. Although the obesity gender disparity is well documented, little is known about the causes of the disparity.

While gender differences in overweight and obesity do exist among Whites, their gender disparity is less consistent and much less pronounced than among Blacks. For instance, among Whites, the direction of the gender disparity is different for overweight and obesity. White women are *less* likely to be overweight compared to White men, but White

women are slightly *more* likely to be obese (4). In contrast, Black women are more likely than Black men to be *both* overweight and obese (4).

Because Black men have not historically had a disproportionately high risk of excess weight, obesity research in Black populations has concentrated on understanding risks for Black females. However, now there is an urgent need to understand obesity risk factors for Black males. As recently as the 1988-1994 NHANES III survey, White and Black boys had a similar prevalence of "obesity" ($\geq 95^{th}$ percentile) (7). Nevertheless, in the late 1990s, young Black males' incidence of overweight/obesity accelerated rapidly. From NHANES III to 1999-2002, the prevalence of overweight among Black teenage boys nearly doubled. For the first time since national record-keeping began in the 1960s, Black adolescent boys were more likely to be overweight and obese compared to White boys (4, 7). Other population-based surveys have seen a similar escalation in obesity prevalence among young Black males (2, 8). In the National Longitudinal Study of Adolescent Health (Add Health), the prevalence of extreme obesity (BMI>40) was especially high in young Black men: 5.1% [se = 1.1%] (versus 3.3% [0.6] in young White men) (2). At a time when obesity risk is accelerating disproportionately in young Black men, we know little about the determinants of obesity in Black males. New race-specific and gender-specific research is needed to understand these recent increases.

Adolescence and early adulthood may be critical life periods for obesity prevention. The incidence of obesity and weight gain accelerate during the late teens and early 20s (2, 8-10). In fact, the late teens and early 20s are the time when Americans gain the greatest amount of weight the most quickly (8-10). In the Add Health study, a nationally

representative longitudinal cohort, the prevalence of obesity among those aged 13-20 years (10.9% obese) doubled by ages 19-26 years (22.1% obese) (2).

Further, adolescent overweight is associated with greater morbidity and mortality later in life, specifically cardiovascular problems (11, 12). Heavier adolescents tend to experience a large weight gain during early adulthood (late teens, early 20s) and to be obese as older adults (11, 13-18). These factors – early adult weight gain and adult obesity – put heavy adolescents at increased risk of coronary heart disease, certain cancers, and higher rates of overall mortality (15, 19) (12, 20). Therefore, the determinants of excess weight and excess weight gain among adolescents and young adults merits investigation.

According to the ecological systems theory of obesity incidence, obesity is determined not only by individual behaviors but through the influences of family, neighborhoods, local government bodies, and even national governmental policy (21-25). Further, these multiple contexts interact with each other and with an individual's characteristics to influence obesity risk (21, 24). Below I briefly review what is known about the determinants of obesity in adolescent and young adult populations. I organize the review around the contexts described in the ecological model. I pay special attention to research in U.S. Blacks.

Individual-level factors. The literature on individual-level obesity determinants in youth and young adults consists mostly of cross-sectional studies and studies in young children, both of which have important limitations. Cross-sectional studies are limited in establishing causal relationships (21, 26, 27). Studies in young children may not be generalizable to older populations. Below, I briefly discuss the main features of this research.

Young Black females are much less physically active than young Black males (26-29). In young adulthood, White women were about half as likely as Black and White men to meet physical activity guidelines (≥5 bouts of moderate or vigorous activity per week) (30). However, Black women were only a quarter as likely to meet the guidelines. In regard to sedentary behavior, an analogous pattern was observed. As both White and Black girls aged, they spent less time than their male counterparts in TV-watching and video-viewing (28) (30). However, in both adolescence and early adulthood, the amount of sedentary behavior for Black females was much higher than for White females. While White males and females differed in their weekly sedentary time, Black males and females both spent a lot of time in sedentary pursuits.

In children and youth, studies have failed to establish a consistent relationship between with childhood overweight and diet (21, 26, 29). Physical activity research has shown somewhat stronger results than studies of diet: children with greater or increasing bouts of physical activity show modest reductions in overweight status (27, 28). However, several school-based physical activity interventions failed to reduce BMI or obesity prevalence (31-34). The lack of strong associations between obesity and diet and physical activity could be due to poor measurement of diet and activity (35). The relationship between sedentary behavior and body fatness is somewhat more consistent. More time spent in "inactivity" in childhood, especially television-watching, may contribute to weight gain and obesity development (28, 36-39), perhaps even into early adulthood (40). However, these associations appear to be modest (39). A recent meta-analysis described the association between hours of television-viewing and fatness as inverse but small (41).

On average, Black girls reach menarche at an earlier age than White girls. Earlier menarche is positively associated with adolescent and adult obesity in girls (29, 42-44). However, it is unclear whether early weight gain causes early sexual maturation or whether sexual maturation itself predisposes girls to obesity (44, 45).

The prevalence of individual-level risk factors fails to fully explain the high obesity prevalence of Black females or the accelerating prevalence among young Black males. The effects of these risk factors are often weak. Further, factors found to confer risk in White populations often show different effects among Blacks (26). For instance, in U.S. cohorts of Black and White young adults, sedentary behavior, fast-food consumption, and physical activity appear to show weaker associations to obesity among Blacks (27, 28, 46).

Few studies have examined risk factors for obesity in adolescent and young adult populations and none directly address the obesity gender disparity among U.S. Blacks. While some individual-level factors are weakly associated with obesity prevalence, these factors are not so much more common among Black females that they fully explain this group's excess prevalence of obesity. The few studies that have examined risk factor associations by race and gender have observed differences in the directions and strengths of associations between obesity and individual-level risk factors.

Neighborhoods, communities, and macro-level policy. Beyond individual risk factors, characteristics of one's larger community are believed to affect obesity risk. Amount of food advertising on television; less physical education in the schools; less walkable communities; more limited access to fresh produce; increased average portion sizes; more sedentary transportation, work, and leisure activities – some of these community and macro-level characteristics may promote obesity among adolescents and young adults (42).

In a longitudinal analysis of the National Longitudinal Survey of Health dataset, region of the country was examined as an obesity risk factor for a cohort of 16-23-year-olds [Must, 1994]. For no group was region a statistically significant predictor of five-year overweight incidence, but, among young White men, overweight prevalence (BMI $\geq$ 27.8) was significantly associated with living in the North Central region or the South (versus the West). Most multiracial cohorts, like NGHS and CARDIA, sample a majority of their Black population from one or two metro areas. This kind of sampling does not allow one to examine the impact of region on racial differences.

Multiple studies have examined the associations between obesity and individual- and family-level income. However, many fewer been able to examine community-level socioeconomic status as a risk factor for obesity among Black and White adolescents and young adults. I am unaware of the studies on this topic in adolescents and young adults.

Childhood-family factors. Longitudinal studies show a strong and consistent relationship between childhood SES and risk of obesity later in adulthood (26) even though higher SES does not predictably predict obesity among children and adult men in cross-sectional research in mostly White populations (26, 47). The fact that childhood SES fails to predict childhood obesity but does predict obesity in adulthood indicates that childhood may be a unique critical period for increasing risk for adult obesity. Although childhood family environment may appreciably affect adult obesity risk, the influence of family factors has not been extensively researched (26). However, one important theme to emerge from this literature is that associations may differ by age and gender (48-51). Below I briefly discuss the influence of childhood family factors on adolescent and young adult obesity.

Family SES. Lower childhood socioeconomic status increases one's risk of being obese in adulthood (26). However, the association between SES and obesity risk varies by race and gender (52, 53). For example, a paper from the CARDIA cohort found that a person's father's education (a surrogate measure for childhood SES) predicted decreased body size in White women and Black men but not in White men or Black women (52). The relationship between SES of origin and later risk of obesity merits further investigation, especially in regard to racial and gender differences.

Mother's age at child's' birth. In a cohort of Black and White girls aged 9 and 10 years, Patterson et al. observed that both Black and White girls with older female guardians were more likely to be overweight: OR=1.14 for each 5-year increase in maternal age (54). In their Discussion, Patterson et al speculated that, " . . . age of mother may affect attitudes toward food, parenting styles, and other family dynamics." I found only one other study of this topic (in primary and secondary schoolchildren in central Italy), but the authors did not report the results of the analysis.

Birth order. In numerous foreign populations, being an only child or being the oldest child are associated with greater risk of obesity (55-59). However, I could only find one study that addressed this question in a U.S. population. In a cohort of inner-city African-Americans, Stettler et al found that first-born status was associated with obesity risk at ages 18-22: RR=4.0 (1.4, 11.2) (60).

Number of siblings. Again, nearly all of the studies of this topic were in foreign populations. In contemporary Italy, mid-twentieth-century the Netherlands, and England in the late 1960s, having fewer siblings, and especially being an only child, was associated with greater prevalence of overweight in adolescents. In one U.S. study of Black and White pre-

adolescent girls, Patterson et al. observed that the odds of being overweight decreased by 14% with each additional sibling (54).

Single-parent household. The only study I found addressing this topic among adolescents was from England in the late 1960s. Children who were "fatherless," especially those being raised by widowed mothers, were more likely to be "obese" (as defined by a local doctor). In the National Growth and Health study, White pre-adolescent girls with a single guardian were more likely to be overweight. There was not a statistically significant effect among Black girls.

Few studied populations have been sufficiently racially diverse to assess interactions of childhood family factors with race. Those studies with multiracial populations have typically observed some differences in effects among the different race and gender groups. For instance, in Add Health, family income and education exerted different effects in Whites versus Blacks and in males versus females (61). In the NGHS study, Patterson et al found the same patterns: Some risk factors were shared between White and Black pre-adolescent girls, but others, like parent SES, varied by race (54).

In Add Health, at every level of income and at every level of parental education, Black girls were more likely than Black boys to be overweight. However, the magnitude of this gender disparity varied considerably by income and education level (61). (In contrast, White boys' and girls' overweight prevalences were never more than 5 percentage points different). At the highest family incomes, overweight prevalence in Black girls and boys differed by 25 percentage points (45% vs. 20%). However, at the middle incomes, Blacks' gender difference was as small as 5 percentage points (32% vs. 27%). Stratifying by parental education revealed even more variation in the gender difference. In families where parents

had less than a high school education, overweight prevalence was 45% in females and 15% in males. When parents had at least a college or professional degree, the gender difference practically disappeared (30% females, 28% males). Family socioeconomic factors were strongly associated with Gender differences in overweight prevalence in Black adolescents.

Because Blacks' obesity gender gap emerges in childhood and increases dramatically during the transition to young adulthood (6, 61), we hypothesized that the gap observed in young adults was associated with childhood and adolescent family factors. In some studies, childhood family factors such as parental education, lower birth order, single-mother household, and fewer siblings were associated with obesity risk in adolescence and adulthood (53-57, 60-67).

Researchers investigating cigarette smoking, illegal substance use, and early sexual debut have proposed that family sociodemographic factors are related to parents' social control of their children (68, 69). For instance, having two parents in the home may engender greater social control because two parents can provide greater parental supervision and mutual reinforcement of the others' authority (69). Additionally, social control may have a stronger effect on health behaviors among men than among women (70). In families with progressively better social support and social control, males' health outcomes may converge towards those of women. In fact, some research has found that being married is associated with more social control of males and that males show more variability in their health behaviors due to the stabilizing influences of marriage than do women (70). However, these results may be less relevant for the adolescent context: in one study, adolescent family structure was not strongly associated with cigarette smoking, illegal substance use, or early sexual debut.

The idea that childhood family social control has stronger effects on males than females may be relevant to understanding the gender disparity in obesity prevalence in Black adolescents and young adults. If Black families have norms, habits, and beliefs that promote obesity, being more closely connected to the family may be associated with higher obesity incidence. In families with loose social control, adolescent boys may be less connected to the family than their female counterparts. Therefore, the males will be less influenced by the family environment than their female counterparts, and the obesogenic norms of the family may not elevate males' obesity risk. On the other hand, in families with greater social control, both boys and girls are more likely to be controlled and connected to the family environment. Therefore, there will be less gender disparity in obesity-related behaviors in boys and girls with uniformly high social control.

Parent-child interactions. The effect of parental control on adolescents, whether perceived or objectively measured, has not been extensively researched. There is little research on parental influence on children's eating habits. Instead, much of the existing research on parental influence concerns physical activity.

Family dinners. However, in one older population (the GUTS study, a predominantly White, middle-class national cohort of early adolescents), more frequent family dinners were associated with a decreased prevalence of overweight status in teenage girls and pre-adolescent boys (71). Family dinner also predicted a slight (10%) decrease in overweight incidence over 1 year. The effect was seen only in boys and young teenage girls. Those pre-adolescent girls reporting family dinner every day showed a slightly increased risk of overweight.

Parental influence on physical activity. A review on parents' influence on their children's physical activity noted that most studies were cross-sectional. The review concluded that concluded that parents probably do influence children's physical activity through multiple pathways: modeling behavior, providing social support, and genetics (50). However, the strength of parents' influence on physical activity appears to diminish as the child ages (50).

Parental participation with kids in physical activity. In a cluster analysis of the Add Health cohort, kids who participated in sports with their parents were most likely to meet physical activity recommendations in young adulthood (72). Few young adults, however, (~10 percent) met the guidelines: While children with involved parents did sustain physical activity more than others, the absolute effects were small.

Parental modeling of physical activity. Another recent study concluded that parental effects through modeling are probably weak (73). In an eight-year study of Norwegian 13-year-olds, standard regression models showed no effects. Multilevel growth models found that father's baseline physical activity was slightly associated with increased child physical activity. In addition, the only maternal effect was less decline in child physical activity when mother's activity increased over time.

Parental influence on sedentary behavior. In Add Health, children whose parents limited their television decisions were among the least active as young adults (72). Interestingly, adolescents who reported high sedentary activity but were free from parental television control were much less likely than the other groups to have excessive "screen time" as young adults (72).

Parental involvement in weight loss. In clinical trials of the obese, family involvement appears to aid short-term weight (49). However, I could identify only two studies in adolescents or young adults that assessed how parental involvement affected long-term (≥6 months) weight loss (51, 74). Both studies targeted only adolescent girls. Neither of the interventions achieved long-term weight loss. However, the two studies suggested possible racial differences in the impact of parental involvement on girls' weight loss. In the study of White girls and their mothers, greater maternal involvement was associated with poorer outcomes (74); in the study of Black girls, the opposite was true (49, 51).

Genetic heritability studies: family environment

Two 1997 reviews argued that the family environment does not contribute to risk of obesity (75, 76). They argue that genetics and the extra-family environment explain all of the person-to-person variation in obesity. However, the genetic studies cited have several limitations. First, the studies were conducted predominantly in populations of White middle-class twins. It may be difficult to detect family influence if studied families are fairly homogenous. In addition, effects may be different in minority populations. Second, most studies investigated variation in BMI, rather than the likelihood of obesity. There is evidence that, compared to BMI variation within the normal range, obesity is more heavily influenced by the environment (62).

Furthermore, twin studies in general are likely to underestimate household environment effects. For instance, calculated genetic effects are often inflated because the twins do share the same household. In twin and sibling studies, it is wrongly assumed that family environmental variance is the same (a) for both twins in a pair. However, even in utero, the experience of offspring differs: For instance, opposite-sex fraternal twins experience

different hormonal environments in utero. Also, while fraternal twins each have their own chorions and amnions in utero, identical twins may or may not share the same amnion. Once born, twins are distinct individuals and do not share the exact same "home environment." No two children experience exactly the same home environment: families relate differently to boys and girls, to older children and younger children, to children with different personality traits (21).

Another assumption of twin studies is that the experiences of twins are representative of the entire population. However, it is likely that twins, especially identical twins, are treated differently than other siblings in their households and than other children in the outside world. Finally, studying more than one type of family relationship (besides twins) would allow more precision when predicting the genetic component of body size variance. Better measures of household variation or even more variation in household types would also improve the precision of measuring the environmental variance.

Conclusion

Children's families are frequently cited as being important for obesity development (73). Yet the role of family has not been extensively researched. Research into racial disparities in obesity often uses a deficit theoretical model (77). For instance, traditional research has investigated why Black girls (who are more likely to be overweight) are not more like White girls (who are seen as healthier because they are less likely to be overweight). This research paradigm ignores Black males' historically low prevalence of obesity and factors which may protect against obesity risk in the Black community. The factors most commonly investigated in Blacks are those previously found to be associated with obesity in White populations.

Instead, a more asset-driven paradigm is needed (77). An asset-driven approach would broaden investigation beyond factors shown to be associated with obesity in Whites. Additionally, an asset-driven paradigm would pursue factors that are associated with *lower* obesity risk in Black populations, factors such as male gender. Uncovering factors that influence Black males' lower obesity risk may help uncover factors associated with

Black Americans have a high prevalence of obesity, but the reasons behind their greater obesity burden are not fully understood. Gender exerts an important effect on obesity risk in Blacks. Presumably, Black boys and girls being raised in the same environments end up having dramatically different obesity risks. The mechanisms behind this different response to a shared environment are not fully understood. A small body of research has demonstrated that childhood family environment has strong associations to adult obesity risk. However, these effects often vary by race and gender. The limited research on family factors, such as family size and SES, underscores this heterogeneity. Although the role of family environment in obesity risk in Black youth and young adults is understudied, family factors may be especially important mediators for obesity development in Blacks. We know of no studies that have investigated factors associated with the gender gap in obesity in Black adults. Understanding the causes underlying these differential outcomes will help elucidate distinct mechanisms of obesity development in males and females.

Specific Aims

Black Americans have a high prevalence of obesity compared to other U.S. ethnic groups. Among Blacks, one obesity risk factor is uniquely strong and consistent: female gender. Black adult females have an obesity prevalence that is 21.1 percentage points higher than Black adult males (4). In contrast, the difference for Whites is only 2.5 percentage points. This marked gender difference in Black populations has not been explained or even well-researched.

I investigate this gender difference in obesity prevalence among U.S. Blacks, with a particular focus on the influence of family environment during adolescence. Through the differential intergenerational transmission of obesity-related norms and behavior modeling, family members may influence gender-based obesity differences. I investigate these questions in a cohort of Black and White U.S. adolescents. While this research primarily aims to better understand obesity determinants among Blacks, all analyses are replicated in the cohort's White population. Replicating results among Whites provides a comparison group for analyses, allowing me to interpret the primary results within a broader context.

The specific aims of this research were as follows:

1) Investigate whether adolescent sociodemographics (two-parent households, fewer siblings, fewer children in the household, lower birth order, and higher parental education) are associated with gender disparity in obesity prevalence in U.S. Black young adults.

2) Estimate to what extent differences in adolescent behaviors (family dinners, hours of television, playing sports with mother, playing sports with father, bouts of physical activity) might account for the gender disparity in obesity incidence in U.S. Black young adults.

These aims are met through secondary data analysis of the National Longitudinal Study of Adolescent Health. This longitudinal, nationally representative study includes data on more than 2,000 Black and more than 5,000 White adolescents followed into early adulthood. The results of this study provide insight into how obesity risk in U.S. Blacks is shaped by gender-specific determinants in the childhood family environment. This knowledge improves understanding of the observed gender disparity in obesity among U.S. Blacks.

Methods

I used a nationally prospective cohort study of young adults to investigate the associations of selected childhood family characteristics and behaviors with gender disparity in obesity prevalence in U.S. Black young adults. We replicated all analyses in U.S. Whites for contextualization and comparison. We hypothesized that greater social control; greater parental involvement in nutrition and activity in adolescence; fewer hours of television in adolescence; and more frequent bouts of physical activity in adolescence are associated with gender disparities in obesity. This study uses extant data from an observational cohort study. No new data collection was conducted.

Population

Add Health is a prospective, nationally representative school-based study initiated in 1994 under a grant from the National Institute of Child Health and Human Development (NICHD). The study was designed to explore the health-related behaviors of adolescents in grades 7 through 12 and their outcomes in young adulthood. Add Health seeks to examine how social contexts (families, friends, peers, schools, neighborhoods, and communities) influence adolescents' health and risk behaviors. Some populations were oversampled, including Blacks from well-educated families. Students who were strategically sampled and for whom sample weights are available comprise the Core sample.

Beginning with an in-school questionnaire administered to students in grades 7 through 12 (Wave I), Add Health followed up with a series of in-home interviews several months afterward (Wave I), a year later (Wave II), and six years later (Wave III). Other sources of data include questionnaires for parents, siblings, fellow students, and school administrators. Participants were aged 13 to 20 years old at the first series of questionnaires (Wave I). Those still in grades 7 through 12 in 1996 were included in the next wave (Wave II). All those who completed the in-home questionnaire at Wave I (even those ineligible for Wave II) were eligible for the next follow-up (Wave III).

Table 1. Sample sizes from each wave of the National Longitudinal Study of Adolescent Health (source: http://www.cpc.unc.edu/projects/addhealth/designfacts)

		Wave I *(1994-95)*		*Wave II* *(1995-1996)*	*Wave III* *(2001-02)*
	Student *in-school*	*Student* *in-home*	*Parent* *in-home*	*Student* *in-home*	*Student* *in-home*
Sample sizes	90,118	20,745	17,700	14,738	15,197

Variables

Data on hundreds of variables have been collected from the Add Health population. Most of the study variables were assessed by questionnaires administered to students and their parents. Students, their parents, and/or school administrators were interviewed at 3 time points: Wave I, Wave II, and Wave III. Data on community characteristics were collected from school administrators or were derived from independent data sources, such as the U.S.

census. Table 2 shows selected pertinent variables and the frequency of collection. Detailed variable descriptions are available on the Internet: http://www.cpc.unc.edu/projects/Add Health/codebooks.

Table 2. Pertinent variables in the National Longitudinal Study of Adolescent Health

	Wave I ***(1994-95)***			***Wave II*** ***(1996)***	***Wave III*** ***(2001-02)***
	School administrator	*Student in-home*	*Parent in-home*	*Student in-home*	*Student in-home*
Family Characteristics					
Household roster					
Number		X		X	X
Ages		X		X	X
Genders		X		X	X
Relationships		X		X	X
Shared housing history		X		X	
Family income (1994)			X		X
Parental education			X		
Number siblings		X			X
Child Characteristics					
Weight (measured)				X	X
Height (measured)				X	X
Physical development, e.g., voice change (M) or menstruation (F)		X		X	
Child Behaviors					
During past week, times you did...					

work around house		X	X
roller-blade, skate, or bike		X	X
sports, e.g., soccer		X	X
exercise, e.g., walking		X	X
During past week, hours you did…			
watch television		X	X
watch videos		X	X
play video/computer games		X	X
Parental Influence			
sport w/ mom past month			X
sport w/ dad past month			X
parents control TV			X
parents control eating			X
1+ parent around during dinner past wk			X
Community Characteristics			
urbanicity	X		
region	X		

Below I describe the anthropometric variables in more detail. Body mass index (BMI) was the anthropometric measure used to classify obesity. For each individual, body mass index (BMI) is calculated as follows: weight (in kilograms) divided by squared-height (in meters squared). Height and weight were measured at Waves II and III. (At Wave I, height and weight were self-reported but not measured.) Wave II BMI is evaluated based on age- and sex-specific cutpoints that are were developed for an American pediatric population (CDC, 2000): obesity was defined as body mass index (BMI) $\geq 95^{th}$ percentile of the age- and sex-specific CDC 2000 cutpoint or BMI > 30.0 kg/m^2 (78). Wave III obesity was defined as BMI $\geq$ 30 kg/m^2 (79).

I chose to examine BMI $\geq$ 30.0 as an outcome rather than BMI $\geq$ 25.0. According to an analysis that compared BMI to adiposity assessed via bioelectrical impedance analysis (with a standard race-neutral formula), a measure which performs less accurately in classifying percent body fat in U.S. men than in U.S. women (80). Any BMI cutpoint used to classify obesity has poorer sensitivity and specificity in men than in women (80). We chose the cutpoint, BMI $\geq$ 30.0, because it is a standard, commonly used definition of obesity that has high specificity in both men and women (~95% and ~99%, respectively (80)). Therefore, $\geq$95% of men and women who had BMI$\geq$30.0 were correctly classified as having excess adiposity. In contrast, at 25.0 $\leq$ BMI $\leq$ 29.9, more than 25% of men are actually not over-fat.

However, this cutpoint, BMI $\geq$ 30.0, has only moderate sensitivity, and the sensitivity is poorer for men (~36%) than for women (~49%) for women (80). Thus, our cutpoint misses relatively more "overfat" men than women and may overestimate the magnitude of the obesity gender gap in Blacks (and fail to detect an excess of obesity in White males compared to White females). (Romero-Corral did not find important race differences, but

another study that used a race-correction in BIA formula did. So the following is an open question: the degree to which race differences in the accuracy of BMI differentially misclassifies men and women of different racial categories.) For analyses that seek to understand gender differences in the accumulation of excess adiposity, I would recommend using BMI $\geq$ 30.0 as a cutpoint rather than BMI $\geq$ 25.0.

I also chose not to conduct a proposed analysis comparing obese young adults who were also obese in adolescence with obese young adults were not obese as adolescents. This analysis was proposed to compare the determinants of early-onset obesity to the determinants of later onset obesity. If there were 3 time points available (pre-adolescent, adolescent, young adult), I would do this analysis. I would measure exposures in pre-adolescence (time 1). Then I would compare these exposures in two outcome conditions (incident adolescent obesity [time1->time2] and incident young adult obesity [time2->time3]). However, our dataset has only 2 time points (times 2 and 3 above). With only 2 time points, the proposed analysis would compare *prevalent* adolescent obesity to *incident* young adult obesity, which I did not believe was very valuable for informing causal inference. In my papers, I note that the critical time period for exposure could be earlier in childhood than we examined and that we did not have the proper data to test that hypothesis. We encourage other researchers to investigate the importance of behavioral factors occurring at younger ages.

Analytic strategy

All analyses began with exploratory analysis. I examined distributions of all variables and generated simple tables to increase my familiarity with the variables. Next, I modeled unadjusted bivariate relationships between exposures and the relevant outcomes. I

then proceeded to multivariable modeling. For all analyses, Stata 9 Survey procedures were used. I only used data from the Core dataset, which was sampled using Add Health multi-stage stratified sampling design. I used the weights provided by Add Health to calculate nationally representative estimates.

Statistical Models

Overview

The objective of Aim 1 was to investigate the associations of selected childhood family characteristics with gender disparity in obesity prevalence in U.S. Black young adults. The exposures were derived from the following items in the Waves I and II adolescent in-home questionnaires: Maternal age at child's birth, respondent's birth order among biological siblings, respondent's number of biological siblings, number of minors in the household, parental education, and single-mother household. Aim 1 focuses on surveillance and describing population-level relationships. This analysis tested whether the prevalence difference varied across categories of each childhood family factor. A limitation of this aim is our inability to control for parents' social environment or body size.

The objective of Aim 2 was to investigate behavioral factors that might contribute to gender disparities in obesity prevalence. The exposures were derived from questions asked at the Wave I and Wave II adolescent questionnaires, including the following:

- On how many of the past 7 days was at least one of your parents in the room with you when you ate your evening meal?

- Which of [these] things have you done with your mother in the past four weeks? . . . Played a sport?
- Which of [these] things have you done with your father in the past four weeks? . . . Played a sport?

A major limitation of this analysis is that the relationships among the exposures, obesity, and possible confounders are dynamic and inter-related. In addition, unmeasured social factors introduce confounding relationships among the child's own behaviors. I somewhat tempered these effects by adjusting some analyses for Wave II sedentary behavior and physical activity. Unfortunately, I did not have detailed diet data and so could not adjust for diet. I did not adjust for adolescent body size: because the initial body size is independently associated with later body size (my outcome), this strategy would be overadjusting, which would dilute true effects between parental control of diet and later body size. We did examine incident rather than prevalent obesity. Looking at obesity that was incident after exposure was assessed reduces some of the impact of feedback, that is exposures like parenting style simply responding to a child's previous characteristics rather than determining that child's future characteristics.

Aim 1 and Aim 2 employ similar statistical models. The main differences are the outcome variables (obesity prevalence in Aim 1, obesity incidence in Aim 2) and the exposure variables. The exposure variables in Aim 1 are all childhood family sociodemographic factors. The exposure variables in Aim 2 are adolescent behaviors. Additionally, each model includes parameters for the given exposure variable and for female gender. A third term quantifies the multiplicative interaction between female gender and the

exposure. There are also terms for age and the multiplicative interactions between female gender and age.

For each aim, we examined the female-male difference in obesity prevalence or incidence before and after standardizing for the exposure variables. Standardizing obesity prevalences or incidences were calculated separately for males and females using coefficients generated by multivariable-adjusted logistic regression. Logistic regression models were race-stratified with obesity at wave III as the dependent variable and age, parental education, and gender as covariates. In addition, interactions with gender were included for all variables due to previous evidence of gender-specific effects of obesity risk factors (28, 81, 82). We opted to use gender interactions with males and females in the same regression models rather than run gender-stratified models in order to facilitate calculation of 95% confidence intervals for the female-male prevalence or incidence difference.

The coefficients obtained from the logistic regression models were used to form race- and gender-specific equations for the calculation of standardized obesity prevalences or incidences. In these formulas, values of each categorical covariate and exposure variable were set to the race-specific average of the proportion of male and female respondents in that category. Thus, the proportion of respondents in each category of every variable was set to the same value for males and females of the same race group. We then calculated differences as the standardized obesity prevalences or incidence in females minus that in males. For instance, an obesity incidence difference of 0 represented equal projected obesity incidence for men and women. Greater than 0 indicated that women were projected to be more likely to become obese; less than 0 indicated that men were more likely to become obese.

All initial data analysis will be run separately for each race group. All variables were analyzed as nominal categorical variables. All tests were adjusted for Add Health's complex sampling design and weighted to correct for loss-to-follow-up (83). The 95% confidence intervals for the incidence differences were calculated using the delta method (83).

Other statistical approaches considered

Before deciding on my statistical approach, I tested numerous other options. I sought a statistical approach that was (1) appropriate for a dichotomous outcome, (2) would produce effect estimates on an absolute scale rather than a ratio scale, and (3) would allow me to produce survey-weighted and –adjusted effect estimates and confidence intervals using standard statistical software. To evaluate statistical approaches, I used each to run survey-adjusted multivariable models adjusting for Black/White race, gender, and the interaction of Black/White race and gender. I used the results of these models to estimate obesity prevalence difference in Black and White young adults. Then, I compared each estimate to a statistical gold standard: obesity prevalence difference estimated from survey-adjusted non-parametric cross tabulations (using Stata 9 command svy: proportion). Below is a description of my findings:

1. Generalized linear model with identity link: problems with convergence
2. Poisson model with sandwich estimator: estimates were slightly different from non-parametric estimates
3. Binomial regression: not a standard procedure in Stata survey software. However, when I programmed it myself, directing the problem to include the statistical weights and use a sandwich estimator, the results were similar to the non-parametric estimates

4. Marginal effects, conditional on mean values of all covariates. This methods takes the derivative a coefficient estimated from logistic regression. The main problem with simply taking the derivative of the "gender" variable is that the gender variable is also included in interaction terms with other variables. Heuristically, the effect of gender is spread among the gender variable and all other variables consisting of an interaction with gender. Therefore, this marginal effects approach, which considers variables to be completely independent of all other variables and holds all other variables at the mean values while calculating the derivative of the coefficient in question, does not work in models with interactions. Also, the marginal effect conditional on the mean values of other covariates is an abstract concept rather than a population-based one.
5. Marginal effects, set non-default covariate pattern. A possible solution to the problem described above is to code variables so that interaction variables should be equal to 0 for the subgroup under investigation. However, this would require constantly changing the coding depending on the subgroup under investigation.
6. Average marginal effects. An alternative to conditional marginal effects is average marginal effects where the covariates are set to mimic the joint distributions of covariates in the population of those in the dataset. There is user-written code that will calculate average marginal effects along with confidence intervals (using bootstrapping techniques). However, interaction variables complicate the calculations; I could not identify any user-written code that could handle more than one interaction term. It would be possible to fit average marginal effects for males

and females separately, but I do not know how to get confidence intervals for the difference of the male and female estimates.

7. Survey-adjusted logistic regression model with categorized variables, saturated with gender interactions; compute difference measures by taking weighted linear combinations of logistic regression coefficients. This allowed stratified estimates for males and females to be in one model, while also allowing us to compute difference measures standardized to meaningful (if hypothetical) populations, and calculate confidence intervals (Huber & White/robust/sandwich estimator) and test the resulting estimates (modified Wald test of equality). This appeared to work best.

Covarying variables

A final methodologic decision involved the multivariable modeling strategy in Aim 1. Because birth order and number of siblings were highly associated, I investigated which factor was more closely associated with the prevalence differences in obesity in U.S. White and Black adults. There was variability in the gender prevalence difference (PD) when stratifying by birth order, but there was not strong evidence of variability in the obesity PD when stratifying by number of siblings. For instance, we compared gender difference among first-born and among second-born respondents from sibships of the same size (sibships of 2) in Black and White adolescents and young adults. In all cases, there was more evidence of gender difference among first-borns than among second-borns. In contrast, when we compared respondents of the same birth order (second-born) but from sibships of different sizes (2 versus 3 or more), estimated gender differences were similar regardless of number of siblings. Therefore, our final multivariable variable models included 5 of the exposure

variables: parental education, female caregiver's age, number of minors in household, family structure, and birth order.

The results are described in greater detail below: When we compared first-born and second-born participants from sibships of the same size (sibships of 2), their patterns of gender disparity were not the same. Among the second-born siblings, there was no evidence of gender difference (RD=-0.01), while first-borns did appear to show gender difference (RD=-0.08). In contrast, when we compared adolescents of the same birth order (second-born) but in sibships of different sizes, we saw no differences (RD[sibship of 2]= -0.02; RD [sibship of 3+]= -0.01). Additionally, we compared first-born children who did not have siblings (only-children) to those who had siblings. The RDs were not statistically different (p=0.34; RD[only]=-0.10; RD[siblings]=-0.06).

Multinomial models: Aim 2

To address concerns that including "overweight" adolescents and young adults in the at-risk population and outcome referent category, respectively, biased my Aim 2 findings, it was suggested that I run multinomial logistic models comparing two sets of analyses:

1) adolescent normal-weight → young adult overweight ($25.0 \leq BMI \leq 29.9$) versus adolescent normal-weight → young adult underweight/normal-weight
2) adolescent normal-weight → young adult obesity ($BMI \geq 30.0$) versus adolescent normal-weight → young adult underweight/normal-weight

Instead of using a multinomial logistic model, I estimated these contrasts using sets of logistic models (see Appendix A).

Aim 1. Association with childhood sociodemographic factors

Abstract

Background: In the United States, Black women are at much greater risk for obesity than Black men. Little is known about the factors underlying this disparity. **Objective:** We explored whether childhood family 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 U.S. Black young adults. **Design:** An analysis data set (n=7,747) was constructed from the nationally representative National Longitudinal Study of Adolescent Health. Childhood family factors were assessed in 1994-95 in non-immigrant Black and White youths aged 11 to 19 years. Obesity was assessed in 2001-02. For each assessed childhood family factor, we evaluated whether the prevalence difference (female obesity minus male obesity) was modified by the factor; and we described the contribution of each variable category to the overall prevalence difference. **Results:** In unadjusted and multivariable-adjusted models, parental education consistently modified Blacks' gender disparity (p=0.01). The gender gap was largest at low parental education (16.7% men obese vs. 45.4% women obese) and smallest at high parental education (28.5% men obese vs. 31.4% women). In Whites, there was little overall gender difference in obesity prevalence. **Discussion:** To our knowledge, this study is the first to document that Black young adults' gender disparity in obesity prevalence is concentrated in

families with low parental education. In these low-SES families, obesity development is either under the control of distinct mechanisms in each gender, or men and women from these households adopt very different obesity-related behaviors.

Introduction

It is well established that Black women have higher obesity prevalence than Black men (2, 3). According to 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 while only 27.9% of Black men were obese (4). This prevalence difference was much larger than that observed in Whites, in whom there was virtually no gender disparity in obesity prevalence. In the 1999-2002 NHANES, 30.7% of White women were obese compared to 28.2% of White men (4).

NHANES has monitored gender-specific obesity prevalence for the past 35 years (5). During most of that time, the gender disparity among U.S. Blacks remained stable at about 15 percentage points but has increased to 20 percentage points in more recent surveys (5, 6). 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 the Black obesity gender gap.

Because this gender disparity in obesity prevalence emerges in childhood and increases dramatically during the transition to young adulthood (6, 61), we hypothesized that the gap observed in young adults was associated with childhood and adolescent family factors. Childhood family factors such as parental education, lower birth order, single-mother household, and fewer siblings have been found to be associated with obesity risk in

adolescence and adulthood (53-57, 60-67). We hypothesized that these factors were related to parental social control, which may differentially affect health behaviors in male and female children (70). Thus, with greater parental social control, health outcomes in males may converge towards those of females. Therefore, we hypothesized that two-parent households, fewer siblings, fewer children in the household, lower birth order, and higher parental education (all putative markers of greater family social control) would be associated with a smaller gender gap in obesity prevalence between young Black men and women.

We were particularly interested in the gender disparity's relationship with parental education. In NHANES III (1988-1994), the relationship between socioeconomic status (SES) and "overweight/obesity" in Black young adults (aged 20 to 30 years) was negative for women but strongly positive for men (82), with the implication that the gender disparity was larger in low- versus high-SES young adults. A recent review suggests that, between NHANES III (1988-1994) and 1999-2000, the relationship between SES and obesity became positive in Black women and negative in Black men (84), implying a potentially smaller gender disparity in low- versus high-SES adults. To address this lack of consensus on the gender disparity's association with SES and other sociodemographic factors, we used data from a nationally representative prospective cohort study of adolescents followed into young adulthood to investigate the associations of selected childhood family characteristics with gender disparity in obesity prevalence in U.S. Black young adults. We also estimated the contributions of specific family types, as well as birth order and female caregiver's age at one's birth, to Black young adults' female-male obesity gap. We replicated all analyses in U.S. Whites for contextualization and comparison.

Methods

Population

Data were from the National Longitudinal Study of Adolescent Health (Add Health). Add Health began as a nationally representative survey of all U.S. 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 1994-95 (baseline), detailed questionnaires were administered to each student and the student's primary in-residence caregiver, preferentially a female. A year later, in 1995-96, all students except those in twelfth grade at baseline were re-interviewed. In 2001-02, seven years after baseline, all study respondents who participated in the 1994-95 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 were born in the United States. Race was defined by a combination of child self-report and parent self-report data (28). We restricted the sample to adolescents whose parents were born in the United States (85) 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 boys and to girls. 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 three study time points, i.e., those in twelfth grade in 1994-95 were excluded.

Outcome

The main outcome was prevalence difference: obesity prevalence in women minus that in men, at the 7-year follow-up visit. Obesity was defined as body mass index (BMI) ≥30 kg/m^2 (79). A prevalence difference of 0 represents equal obesity prevalence for men and women. Greater then 0 indicates that women were more likely to be obese; less than 0 indicates more male obesity.

We considered modeling six-year 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, we are not calculating causal estimates, and 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 the 6-year observation period, obesity was maintained 80%-90% of the time in Black and White males and females (2). By modeling prevalence, we produce estimates that can be directly compared to other surveillance data and be easily incorporated into estimates of public health burden.

Exposures and covariates

Exposure variables were derived from the baseline (1994-95) interviews of respondents and their caregivers. Six exposure variables were examined: number of full-siblings (including respondent); birth order; number of minors (aged ≤18 years) 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, i.e., all children of both respondents' biological parents. Parental education was defined as the highest education attained by either of the respondent's biological parents (61). We categorized family structure into four groups: household headed by single mother; by both biological parents; by two parents, at least one of whom was non-biological; and other (85). Ninety-two percent of the identified female caregivers were 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 (86). For lack of a better term, throughout this paper, 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-year follow-up (80.9% of Black females, 71.2% of Black males, 82.0% of White females, 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, 4.2% of

White males]. The final analysis sample consisted of 2,096 Black and 5,651 White respondents.

Data analysis

Effect modification of the prevalence difference

Our primary hypothesis was tested by examining whether there was effect modification of the obesity prevalence difference by any of six exposure variables. Specifically, this analysis tested whether the prevalence difference varied across categories of each childhood family factor. All family 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 using modified Wald tests (83).

All estimates were corrected for Add Health's complex survey design (83). Both unadjusted and multivariable-adjusted estimates were calculated for all analyses. 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 (87). We chose to standardize the multivariable-adjusted estimates to hypothetical race-stratified populations with similar covariate distributions as 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 two genders. The one exception was respondent's age, for which data from the two race groups were combined before determining the mean proportions. The delta method was used to calculate 95% confidence intervals for prevalence differences.

Because some of our six 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 five variables separately as an exposure. We found that female caregiver's age at respondent's birth was strongly associated (OR≥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 respondent's birth.

We also observed a strong bivariate association (OR≥3.0) between birth order and number of siblings. To separate the associations with birth order versus those with number of siblings, we created joint variables that allowed us to examine associations between obesity prevalence difference 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 (results not shown). Therefore, our final multivariable logistic regression models included gender, categorical age, five exposure variables (excluding number of full-siblings), and gender interaction terms with all variables.

Decomposition of the prevalence difference

We used Kitagawa decomposition to divide the overall gender disparity into components due to differences in men's and women's stratum-specific obesity prevalences (88). For each variable, the standard population was assigned the average exposure distribution of the two 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 diving by the overall prevalence difference. This approach explores

only disparity due to differences in stratum-specific obesity prevalence not that which may be due to men's and women's differences in exposure distributions. 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. In addition, this approach simplifies interpretation when stratum-specific estimates have different signs.

Results

Table 3 shows the distributions of the exposure and outcome variables. There were striking differences between Blacks and Whites, especially for parental education and family structure. As expected, Black women were much more likely than Black men to be obese. The estimated prevalence difference was 11.9 percentage points (95% CI: 7.0, 16.7). Among Whites, there was not a gender difference in obesity prevalence: the prevalence difference estimate was only 0.9 percentage points (95% CI: -1.9, 3.8).

In Blacks, obesity prevalence was greater in women than in men in every stratum of every childhood family variable (see **Table 4**). However, the magnitude of women's excess prevalence varied across some of the family factors. For instance, in families in which neither parent completed high school, Black women's unadjusted obesity prevalence was 45.4% compared to only 16.7% for Black men, corresponding to a large prevalence difference of 28.8 percentage points (standard error [se]=6.5). In contrast, in Blacks from families in which a parent had a college degree, the unadjusted prevalence difference was only 2.9 percentage points (se=4.8): Black men's obesity prevalence was 28.5%, and Black women's was 31.4%. In Whites, there was not this much variation in gender difference by any of the exposure

variables, but obesity prevalence did tend to be higher in White women than in White men for parental education less than high school; young or old female caregivers; and in large sibships/high birth order.

Multivariable-adjusted prevalence differences are shown in **Figures 1-5**. The overall multivariable-adjusted prevalence differences for Blacks and Whites were 12.5 percentage points (95% CI: 7.8, 17.2) and 0.4 percentage points (95% CI: -2.4, 3.3), respectively. The most striking association with obesity gender disparity was found for parental education in Blacks (Figure 1) (p=0.01). The prevalence difference was greatest among those whose parents did not complete high school and smallest among those with a parent who completed college. As in Blacks, the overall trend in Whites was that women from the lowest education families were at elevated obesity risk compared to men from similar families. In Whites, the test for effect modification of the obesity prevalence difference by parental education was statistically significant in unadjusted (p=0.05) but not multivariable-adjusted models (p=0.34).

For two other childhood family exposures, there were suggestions of moderate associations with gender difference in obesity prevalence. Birth order was somewhat associated with gender difference in both Blacks and Whites (see Figure 3). Having a female caregiver who was relatively young (≤18 years at respondent's birth) or relatively old (≥35 years old at respondent's birth) appeared associated with higher obesity prevalence for White women relative to their White male counterparts (see Figure 5).

Results from the decomposition analyses are shown in **Table 5**. Blacks from the lowest parental-education families represented less than 20% of the population but contributed more than 40% of Blacks' gender gap (~5 percentage points). In contrast, Black

children of college graduates also made up about 20% of the population but contributed only about 5 percent of the gender gap (<1 percentage point). In earlier analyses, Whites from families where parents did not complete high school also appeared to show a gender gap. However, this group made up less than 10% of the White population and so contributed only about 1 percentage point of gender disparity.

Discussion

We used an innovative methodological approach to directly study the gender disparity in obesity prevalence in U.S. Black young adults. To our knowledge, this study is the first to examine family factors as possible correlates of Blacks' gender gap in obesity prevalence. With the exception of parental education, none of the family variables were strongly associated with the gender disparity. Thus, we believe that this research does not implicate differential parental social control in adolescence as a cause of the gender disparity in non-immigrant Black young adults. However, we found that the obesity gender gap varied by parental education. Nearly half of the overall gender gap was concentrated among the fifth of Black young adults whose parents did not complete high school. While young Black women from low-education families were at the greatest risk of obesity, young Black men from these same families were at the lowest risk.

Although U.S. work addressing gender disparities in obesity prevalence is scarce, there is a body of relevant work examining international differences in obesity prevalence (89-93). The association between socioeconomic status 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]) (93). For men in poor, low-HDI nations, socioeconomic

status is positively associated with obesity. In medium-HDI nations (e.g., Brazil, Saudi Arabia), studies of men find fewer positive associations and more null associations. Finally, in men from high-HDI nations (e.g., the U.S., Australia), few studies find positive associations: most find no linear association or negative associations. Women in low-HDI and medium-HDI countries tend to show trends similar to those observed for men. In high-HDI countries, however, studies are much more likely to find negative associations in women than in men (93). This finding conforms well to what we observed among young White men and women, in whom obesity prevalence decreased in both White women and men as parental education increased.

Young Black Americans' patterns, however, were not compatible with those usually found in high-HDI countries. The gender gap observed in U.S. Black young adults more closely resembles the profile of a medium-HDI country, where obesity prevalence is usually much higher in women than in men (90, 94). Within medium-HDI and high-HDI nations, there are historical subgroups that are not fully integrated into the larger countries' social and economic systems (91, 93). McLaren classifies these "traditional subcultures" as living in countries at a lower stage of development. For instance, in her review, "American Indian and Maori subgroups were classified as having a medium HDI [Human Development Index], although the studies took place in the United States and New Zealand (both high-HDI), respectively." Similarly, Black Americans could be classified as living in a medium-HDI context: although Black Americans live in a very wealthy nation, legally sanctioned mechanisms barred them from full participation in American social and economic life until well into the twentieth century (95).

Moreover, the divergent associations between socioeconomic status and obesity observed in young U.S. 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-socioeconomic-status individuals to those of low socioeconomic status (92). This shift generally occurs in women before it occurs in men (89, 92, 96). Thus, for a time, women's obesity prevalence may be inversely associated with socioeconomic status while men's obesity prevalence may retain its positive association. Further, as obesity prevalence stabilizes or decreases among women of high socioeconomic status, it may continue to increase among men, causing the gender gap to decrease. However, over the past 35 years, the magnitude of the obesity gender disparity among U.S. Black adults has not decreased (5, 6). Further research should explore if Black Americans' obesity patterns conform to the model we observe in rapidly developing medium-HDI nations.

Future studies of differential susceptibility to obesity by sex may help to explain the gender disparity seen in U.S. Blacks, particularly those from low socioeconomic backgrounds. We hypothesize that, in obesogenic environments, women may be more physiologically susceptible to obesity then men. Genetic studies in European samples have provided evidence that obesity risk may be under different genetic regulation in women and men (97). In addition, in most countries with rapidly increasing obesity prevalence, women develop obesity at a greater rate than men (90, 94). The same is true even for U.S. Whites (a group who show little overall gender disparity) when they live in moderately food insecure households, which are thought to be particularly obesogenic (98, 99). Perhaps, in general, women are more susceptible to obesity than men, but U.S. White women manage to achieve obesity parity with their male counterparts due to intense social and economic pressures to be

thin. Indeed, at all levels of socioeconomic status, U.S. White women idealize a particularly small body size (100). This may be because in White women, more so than in any other U.S. race/gender group, thinness is highly associated with wages and family income (101).

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 lower and men higher obesity prevalence relative to other Black adults of the same gender (84). This conclusion was based largely on Zhang and Wang's analysis of the 1999-2000 NHANES dataset (102). Several aspects of that analysis differed from ours. Most notably, the 1999-2000 NHANES analysis studied adults aged 20 to 60 years old (102), while the average age of our cohort was about 21 years old; a previous study found age differences in the gender-specific relationships between obesity and socioeconomic status for Black young adults less than 30 years old in comparison to older Black adults (82). In addition, the 1999-2000 NHANES analysis used odds ratios while we used prevalence differences; they examined an adult's own educational attainment while we used parental education; their education categories [$\leq 9^{th}$ grade; 10^{th}-12^{th} grade; and college or higher] differed from ours [$<12^{th}$ grade; some college; college degree; higher than college]; and their sample size of Blacks was smaller than ours, resulting in more unstable estimates.

Our analysis is not without limitations. First, we examined a narrow age range within young adulthood; our findings may not be generalizable to older adults. Second, we did not investigate the respondent's adult socioeconomic status independent of his or her parents' educational attainment; the age range examined is a highly complex transitional period, in which it is difficult classify socioeconomic status independent of family of origin. Third, we were limited to the variables collected by the parent study. For instance, two of our variables

(family structure; number of minors in household) were time-varying, but we only examined them during adolescence; perhaps examination at a younger age or of cumulative exposure over a longer period would have given different results. Further, compared to tests of main effects in datasets of the same size, tests of modification tend to have relatively low power, which would increase the likelihood of failing to detect an association (103, 104). 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 Black females. Further, fewer Black male respondents than female respondents were retained in the young adult follow-up sample. However, Add Health's sample weights are designed to account for this differential loss-to-follow-up.

Our study offers many strengths. To our knowledge, Add Health is the only nationally representative dataset with an adequate sample size and diversity of variables suitable for this work. Further, 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 (93). Additionally, retention rates were good over the seven-year 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 in 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 socioeconomic status are more important than within-family dynamics in explaining the young adult gender difference in obesity prevalence. Future research on the causes underlying the decades-old gender difference in obesity prevalence among U.S. Blacks should especially 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.

Tables and Figures

Table 3. Characteristics of the analysis sample, adjusted for sampling design, U.S. Black and White young adults, National Longitudinal Study of Adolescent Health, 2001-02

	Black		**White**	
	females	**males**	**females**	**males**
	n=1,153	n=943	n=2,909	n=2,742
	%[1] (n)[2]	%[1] (n)[2]	%[1] (n)[2]	%[1] (n)[2]
Mean of age (yrs)	21.39	21.67	21.15	21.45
Obese	35.6 (355)	23.7 (207)	21.6 (597)	20.7 (546)
Parental education				
<HS	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 grad	17.2 (323)	21.1 (284)	27.6 (815)	27.0 (781)
Family structure				
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 non-bio	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)
Mom's age at birth (years)				
<19	13.2 (125)	12.9 (96)	5.4 (168)	6.4 (159)
19-24	37.9 (434)	40.8 (358)	35.1 (1034)	38.3 (1023)
25-34	37.9 (473)	33.5 (375)	52.8 (1518)	48.4 (1377)
35-44	8.8 (93)	8.9 (86)	5.6 (162)	5.9 (157)
≥45	2.3 (28)	4.0 (28)	1.1 (27)	1.0 (26)

# children in household				
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)
# children in full-sibship				
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				
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 weighted for oversampling and corrected for loss-to-follow up and are nationally representative.

[2]Numbers are absolute unadjusted numbers in each stratum.

Table 4. Unadjusted obesity prevalence and prevalence differences[1] for women and men stratified by race and childhood family exposures, U.S. Black and White young adults, National Longitudinal Study of Adolescent Health, 2001-02

	Black			White		
	females	**males**	**prevalence difference**	**females**	**males**	**prevalence difference**
	%	%	% points (se)[2]	%	%	% points (se)[2]
Overall	35.6	23.7	11.9 (2.4)	21.6	20.7	0.9 (1.4)
Parental education						
<HS	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 non-bio	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)
Mom's age at birth (yrs)						
<19	39.1	26.7	12.4 (9.7)	34.7	23.5	11.2 (6.0)
19-24	35.9	22.9	13.1 (4.2)	23.0	22.3	0.8 (2.3)

25-34	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	58.7	34.0	24.7 (16.7)	37.9	18.7	19.2 (14.8)
# 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)
# 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 are weighted for oversampling and corrected for loss-to-follow up and are nationally representative.

[2]percentage points and standard errors

FIGURES 1-5. Estimated multivariable-adjusted differences in obesity prevalence between women and men by race and childhood family exposures, U.S. Black and White young adults, National Longitudinal Study of Adolescent Health, 2001-2002[1,2]

[1]Error bars are 95% confidence intervals.

[2]P-values are race-specific tests of modification of the obesity prevalence difference by each exposure variable.

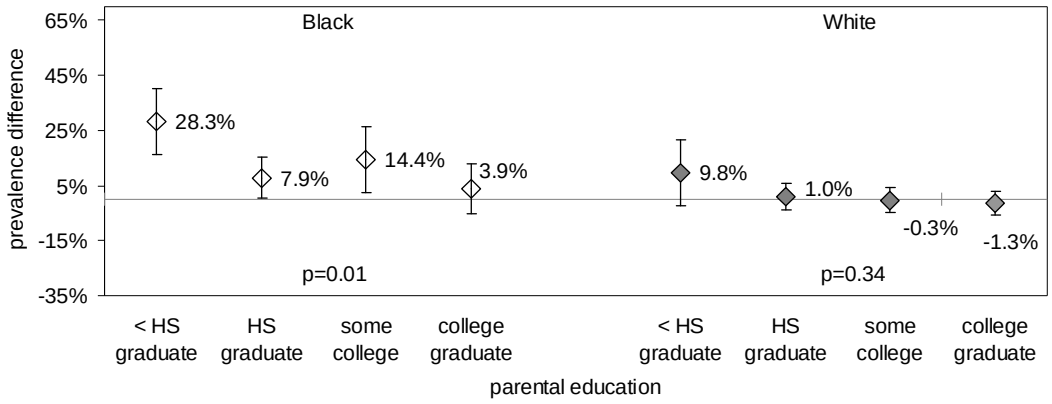

Figure 1: Gender disparity and parental education

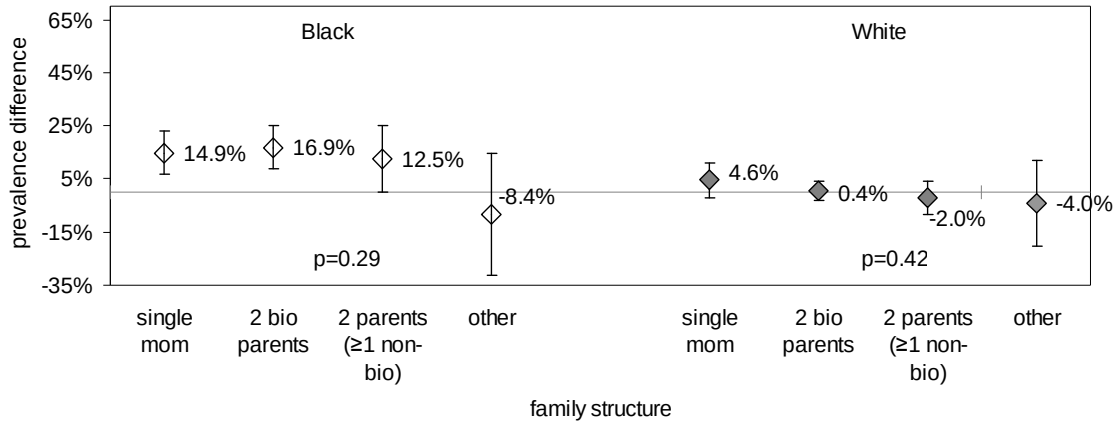

Figure 2: Gender disparity and family structure

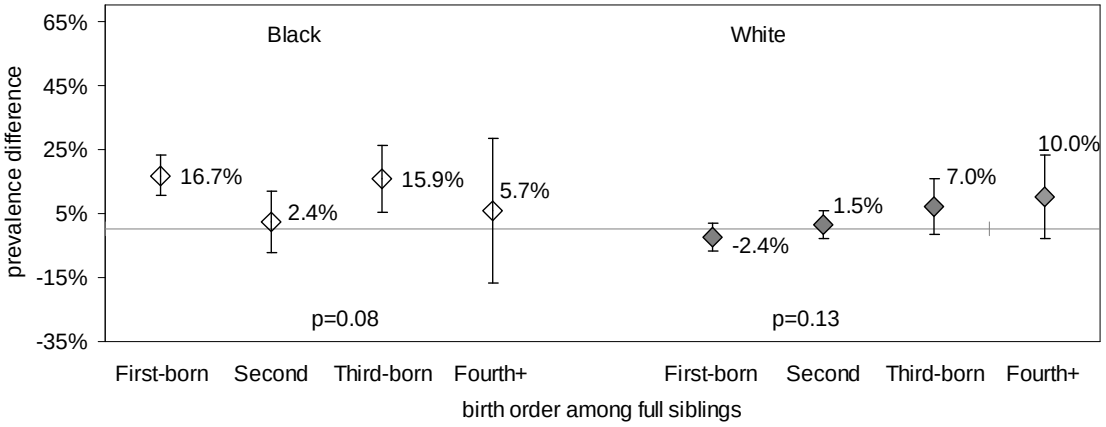

Figure 3. Gender disparity and birth order among full siblings

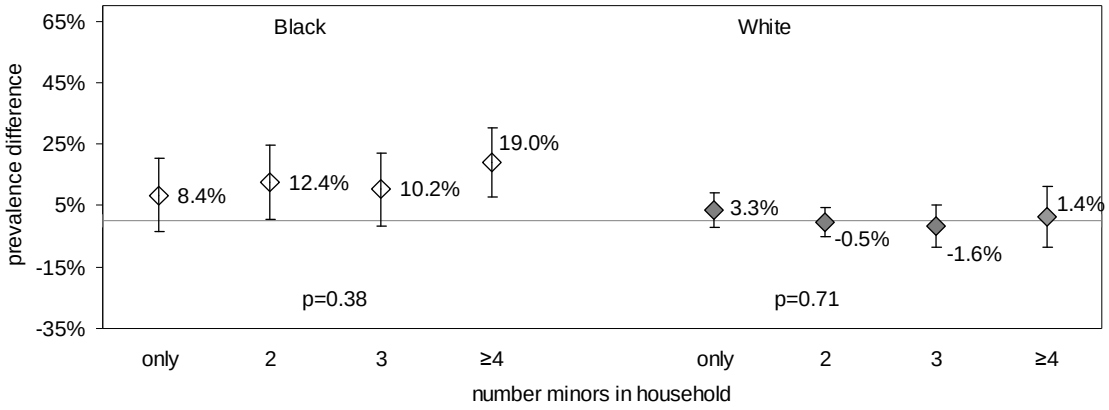

Figure 4. Gender disparity and number of minors in household

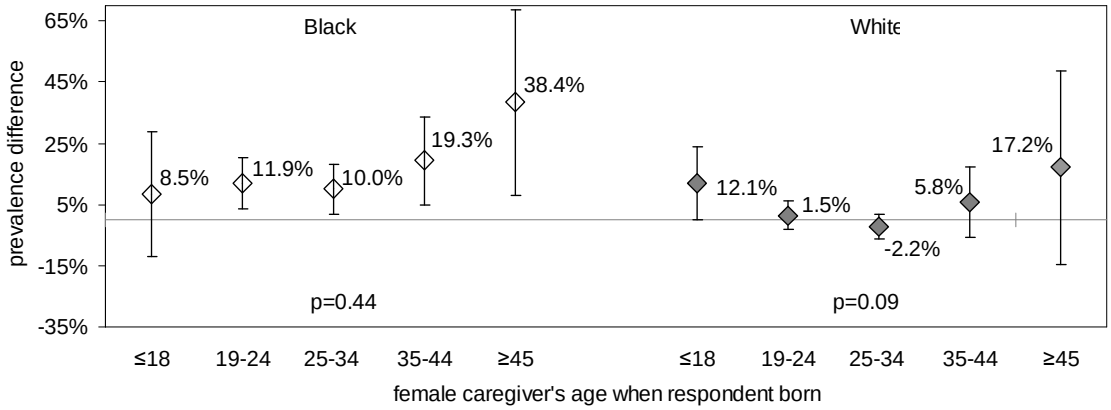

Figure 5. Gender disparity and female caregiver's age when respondent born

Table 5. Contributions of population sub-groups, defined by childhood family characteristics, to the female-male disparity in obesity prevalence in U.S. Black and White young adults, National Longitudinal Study of Adolescent Health, 2001-02

	Black			White		
		Contribution to gender gap percentage points[1] (percent)[2]			Contribution to gender gap percentage points[1] (percent)[2]	
	%[3]	unadjusted model	multivariable model	%[3]	unadjusted model	multivariable model
Total	100.0	11.9 (100)	12.5 (100)	100.0	0.9 (100)	0.4 (100)
Parental education						
<HS	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 non-biological	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 (years)						
<19	13.1	1.6 (13)	1.1 (9)	5.9	0.7 (29)	0.7 (24)
19-24	39.3	5.1 (42)	4.7 (38)	36.7	0.3 (12)	0.6 (20)

25-34	35.7	3.3 (27)	3.6 (29)	50.6	-0.5 (-24)	-1.1 (-38)
35-44	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)
# kids 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)
# 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]due to rounding, may not add up to marginal female-male prevalence difference

[2]divided by sum of absolute values of stratum-specific contributions

[3]Population weights are race-specific averages of males' and females' univariate population distributions, adjusted for Add Health's complex survey design.

Aim 2. Contributions of adolescent and parental behaviors

Abstract

Background: In the United States, Black women are at much greater risk for obesity than Black men. Differences in childhood behaviors may underlie this disparity. **Objective:** 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 obesity incidence in U.S. Black and White young adults. **Design:** Data were from the nationally representative National Longitudinal Study of Adolescent Health. Behaviors were assessed in non-immigrant Black (n=1,503) and White (n=4,452) youths in 1994-95 (aged 11-19 years) and 1995-96 (aged 12-20). Gender disparity (female obesity incidence minus male obesity incidence) was measured in 2001-02 (aged 18-26). We produced race- and gender-stratified covariate-adjusted estimates of associations between behaviors and obesity using logistic regression and then evaluated whether standardizing Black males and females to the same distributions of adolescent behaviors reduced the size of Blacks' predicted gender disparity. **Results:** Black females reported less leisure-time physical activity and lower likelihood of playing sports with either parent than did Black males. Standardizing behaviors did not reduce the estimated gender disparity in obesity incidence (non-behavior-standardized: 9.8 percentage points [95% CI: 4.5, 15.1]; fully-standardized: 10.2 percentage points [5.2, 15.2]). **Discussion:** To our knowledge, this study is the first to examine to what extent behavioral differences might account for gender

disparity in obesity prevalence in Black young adults. Male-female differences in the adolescent behaviors examined did not appear to underlie the obesity gender gap.

Introduction

In the United States (U.S.), 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 (4). 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 while only 27.9% of Black men were obese (4). In Whites, there was virtually no gender disparity: 30.7% of women were obese; 28.2% of men were obese (4).

Although the gender gap in obesity prevalence in Blacks is well-known, there has been very little research exploring the causes of this disparity. Using nationally representative data, we have shown that the gender difference in obesity prevalence was pronounced even in young adults: obesity prevalence in young Black women was 11.9 percentage points [se=2.4] greater than in young Black men (81). Although there was no gender disparity in young White adults overall, a gender gap was observed in those whose parents did not complete high school. The gender gap in these White young adults was 13.8 percentage points (se=6.1): 41.3% of females were obese versus 27.5% (81). The gap was twice as large in Black young adults whose parents did not complete high school: 45.4% of these women were obese versus 16.7% of the men (81).

The association between obesity gender disparity and the educational backgrounds of young adults' parents provides some insight but fails why males and females from similar

socioeconomic backgrounds should have dramatically different obesity risks. A deeper understanding of the causes of the disparity are needed in order to identify behavioral targets for intervention. We used data from a nationally representative prospective cohort study of U.S. adolescents followed into young adulthood to investigate behavioral factors that might contribute to gender disparities in obesity prevalence. Although we studied both Black and White young adults, our focus was on the Blacks, in whom the disparity is much larger. We examined whether behaviors during adolescence helped to explain the gender differences observed in young adults. The adolescent behaviors examined included family dinners, hours of television, playing sports with mother, playing sports with father, and bouts of physical activity.

Methods

Population: The National Longitudinal Study of Adolescent Health (Add Health)

Data were from the Add Health cohort, which began as a nationally representative survey of all U.S. public and private school students enrolled in grades 7 through 12 in 1994-1995. The survey was cluster-sampled by school and oversampled some subgroups, including Black students with a parent who had completed college or attained a professional degree. The Add Health survey focused on adolescent risk behaviors and included a wealth of behavior data.

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 re-interviewed, and height and weight were measured. For the present study, exposure variables were

collected at wave I or wave II. At wave III (2001-02), all study respondents surveyed at wave I were re-interviewed; height and weight were measured for the second time.

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 data (28)) who had at least one parent born in the United States, because the health behaviors and obesity prevalence of foreign-born Black adults are different than in U.S.-born Blacks (105, 106). Behavioral pathways leading to obesity gender disparity may differ as well.

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%); and missing measured and self-reported height or weight at 1-year or 7-year follow-up (1.3%). 3.6% were excluded because they were pregnant at either weighing (8.4% of Black women, 5.9% of White women). Self-reported height or weight was substituted for missing measured data for 1.3% of respondents at wave II and for 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.

Outcomes

The main outcome was gender disparity in young adult obesity incidence: female obesity incidence minus male obesity incidence. Incident obesity was assessed at wave III in those who were non-obese at wave II. Pre-existing wave II obesity was defined as body mass index (BMI) $\geq 95^{th}$ percentile of the age- and sex-specific CDC 2000 cutpoint or BMI > 30.0 kg/m^2 (78). Incident obesity at wave III was defined as BMI ≥ 30 kg/m^2 (79).

Exposures: family and adolescent behaviors

The five adolescent behaviors examined were self-reported by the respondent on the Add Health wave I and wave II in-home questionnaires. The behaviors were (1) frequency of dinners with a parent present (how many days in the past week a parent was present during the respondent's evening meal, average of wave I and wave II); (2) hours of television-viewing (number of hours the respondent watched television in a typical week, wave II); (3) sports participation with a biological mother or resident female caregiver (whether respondent reported playing a sport with the parent in the past month at either wave I or wave II); (4) sports participation with a biological father or resident male caregiver (whether respondent reported playing a sport with the parent in the past month at either wave I or wave II); and (5) frequency of leisure-time moderate-to-vigorous physical activity (MVPA) (MVPA score, average of wave I and wave II scores). The MVPA scores ranged from 0 to 9 and were the sums of responses to 3 questions (scored from 0 to 3) about the past week's engagement in selected activities of 5-8 metabolic equivalents (METs), i.e., "active sports," "exercise," and skating/biking. The three questions was scored according to reported frequency of the selected activities: 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 (61). 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-year or 3-year groupings. Gender, which we conceptualized as the joint expression of sex-chromosome-determined biological traits and culturally determined sex-specific roles and behaviors (86), was defined by a variable for biological sex.

Data analysis

All variables were analyzed as nominal categorical variables. All tests were adjusted for Add Health's complex sampling design and weighted to correct for loss-to-follow-up (83). Chi-square tests were used to evaluate whether the distributions of the behavior variables differed by gender in each race group.

We examined the female-male difference in obesity incidence before and after standardizing for the behavior exposure variables. "Behavior-standardized" obesity incidences were calculated separately for males and females using coefficients generated by multivariable-adjusted logistic regression. Logistic regression models were race-stratified with obesity at wave III as the dependent variable and age, parental education, and gender as covariates. In addition, interactions with gender were included for all variables due to previous evidence of gender-specific effects of obesity risk factors (28, 81, 82). We opted to use gender interactions with males and females in the same regression models rather than run gender-stratified models in order to facilitate calculation of 95% confidence intervals for the female-male incidence difference.

The coefficients obtained from the logistic regression models were used to form race- and gender-specific equations for the calculation of standardized obesity incidences. In these formulas, values of each categorical covariate and exposure variable were set to the race-specific average of the proportion of male and female respondents in that category. Thus, the proportion of respondents in each category of every variable was set to the same value for males and females of the same race group. We then calculated incidence differences as the standardized obesity incidence in females minus that in males. For instance, an obesity incidence difference of 0 represented equal projected obesity incidence for men and women. Greater than 0 indicated that women were projected to be more likely to become obese; less than 0 indicated that men were more likely to become obese. The 95% confidence intervals for the incidence differences were calculated using the delta method (83).

In addition, we calculated standardized incidence differences in Blacks and Whites whose parents did not complete high school. This was done because gender disparity in obesity prevalence in Black and White young adults is largest at low parental education.

Results

The distributions of age, parental education, and weight status before exclusion of the students who were obese in adolescence are shown in Table 6. Adolescents in all weight status categories were included in this table in order to display representative distributions of weight status in adolescence and young adulthood. Parents of Black adolescents tended to have lower educational attainment than parents of White adolescents. As expected, Black females were more likely than Black males to be obese in both adolescence and young adulthood. Among Whites, males were more likely than females to be obese in adolescence;

however, by young adulthood, the prevalence of obesity was similar in young White males and females. Over the 6-year follow-up period, 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 7 shows the distributions of behavioral characteristics in the main analysis sample, in which respondents who were obese in adolescence were excluded. In Whites and Blacks, boys reported more bouts of leisure-time physical activity than girls and were more likely to report sport with a male parent. White boys reported watching more television than White girls. In Blacks, there was no gender difference in television-viewing, but Black boys and girls as a group reported watching more television than either White boys or girls.

To assess the degree to which behavioral differences might contribute to the gender gap in obesity in Blacks, we computed obesity incidence differences that were standardized for each behavioral variable (Table 8). We compared each behavior-standardized incidence difference to the incidence difference that was standardized for only age and parental education. In Blacks, there was little suggestion that setting males and females at the same behavioral distributions resulted in a smaller gender difference in the incidence of obesity, even when all behavior variables were included at once (Table 8). If gender differences in the adolescent behaviors were important determinants of the obesity gender gap, then we would expect the “behavior-standardized” incidence differences to be smaller than the incidence differences not standardized for behavior. This was not seen in our analyses.

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.” For this group of 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 whose parents did not complete high school: 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).

Discussion

The adolescent behaviors examined here did not appear to contribute to the higher incidence of obesity in young Black women versus young Black men. If gender differences in these behaviors contributed to the young adult obesity gender gap, one would expect Black girls to eat family dinners less frequently, watch more television, be less likely to do sports with their parents, or do less leisure-time physical activity than Black boys. There were no gender differences in family dinners or television-viewing, but Black girls did engage in less leisure-time physical activity, were less likely to participate in sports with a mother, and were less likely to participate in sports with a father.

Nevertheless, setting these three physical activity-related variables to the same distributions for males and females did not make the obesity gender gap smaller. This was primarily because these variables were not strongly associated with obesity incidence over the six-year follow-up period. Chi-square tests did not find associations between adolescent physical activity score and obesity incidence for any of the four race/gender groups ($p>0.20$ in all groups). Sports participation with a father tended to be associated with lower obesity incidence in Black men (34.2% of non-obese did sports with father versus only 24.9% in

obese; p=0.09) but slightly greater obesity incidence in Black women (16.0% of non-obese did sports with father versus 19.3% in obese; p=0.48). Thus, setting males and females equal for sports participation with a father tended to produce a slightly larger rather than smaller predicted gender gap. Sports participation with a mother was weakly associated with lower obesity incidence in both Black males (p=0.55) and females (p=0.07), but sports participation similar enough in males and females (15.8% and 10.4%, respectively) that setting them equal at the average value (13.1%) did not affect the predicted gender gap much.

Our hypothesis was that the examined behavioral factors were unequally distributed in males and females, and this inequality contributed to the obesity gender gap. However, factors that are equally distributed in males and females could also exert an influence on the gender gap if the dose-response relationships between the factors and obesity differed for males and females. For instance, Whites showed no obesity gender gap, but several of the behavioral factors (e.g., higher television-viewing, less sport with father, lower physical activity score) were more common in White girls than in White boys. In Whites, gender equity in obesity is achieved at levels of behavioral variables that are unequal for males and females. It is possible, that in Blacks as well, gender equity in obesity may be achieved at behavioral levels that are unequal for males and females.

There are important limitations of our work. First, the behavioral factors we investigated were measured relatively late in childhood, in mid- to late-adolescence. Gender differences in obesity risk may be established earlier in life. Second, there is evidence that BMI classifies obesity more poorly in males than it does in females (80). Thus, our analysis may underestimate obesity incidence in Black males, thereby overestimating the magnitude of Black women's excess of obesity incidence. Further, the exposure variables were all self-

reported by the adolescent respondents and may not precisely reflect their true behaviors. For instance, the validity of self-reported measures for quantifying time spent in moderate or vigorous physical activity is fair to moderate at best (107-110). However, our short self-report questionnaire is likely to be similar in validity as more detailed ones (110). Our failure to identify adolescent behavioral factors associated with the gender gap could be due to exposure measurement error or reverse causation. Another study limitation is that we did not examine parental facilitation of sports involvement or parental modeling of sports involvement, which may be more important influences on physical activity than direct participation with the parent. Finally, reverse causation, whereby adolescents with a propensity towards obesity may have adopted behaviors perceived to be protective against obesity, may have obscured relationships between these factors and incident obesity.

To our knowledge, our study is the first to explicitly investigate child and parental behaviors that might be associated with the male-female disparity in obesity in a population-based sample of U.S. young adults Blacks. The dataset was nationally representative and had sufficient sample size and diversity to allow stratified analysis by both race and gender. In addition, because Add Health is a longitudinal study, data were collected prospectively. For most respondents, height and weight were measured, which may be especially important in investigating gender differences in weight status since reporting bias varies by gender (111). Finally, our statistical analysis reported results using a measure that assessed the gender disparity on an absolute scale (112). This is notable because measures on relative and absolute scales often differ in the context of inter-group variation in risk; most previous research on obesity in Blacks assessed risk on a relative scale. Another advantage of our measure was that it is "decomposable," which allowed us to quantify how much of the

disparity might be associated with differences in the distributions of the adolescent behaviors in males versus females" (112).

A recent paper argues that few studies have evaluated how membership in different racial groups influences men's and women's health differently (113). According to the authors, failing to look at the interaction of race and gender presents two problems: the omission "leads to overgeneralizations about the association between gender and health outcomes" and "tends to obscure important variations within racial/ethnic populations" (113). This critique applies well to obesity research, in which it is common to adjust for race or gender as a covariate rather than stratifying by both factors. Failure to jointly investigate race and gender as an obesity risk factor probably leads to overgeneralization about relationships between gender and obesity but also about race and obesity. Additionally, when the gender disparity in Blacks is obscured, factors underlying this gender inequity are not directly addressed.

While our study found behaviors that differed by gender, the behaviors were not strongly associated with obesity incidence. In fact, longitudinal studies have identified only a few behaviors that predict excess weight gain in adolescents or young adults. One factor identified in adolescent girls is a fast rate of decline in physical activity; the association is independent of baseline physical activity level (27, 114, 115). Little is known about declines in physical activity in Black adolescent males, but the dramatic rates of decline in Black females (116) indicate that sharp declines in physical activity could be associated with the obesity gender gap in young Black adults. Another factor potentially associated with the gender gap in obesity incidence is fitness level. Lower fitness is associated with weight gain in children and young adults, and Black girls and women appear to be less fit than Black

boys and men (117, 118). Other factors worthy of further investigation include breakfast-skipping (119, 120) and dieting behaviors, which are more common in females than males and may predispose adolescents to excess weight gain (121). Finally, because associations between the gender gap and these factors may be dynamic as males and females age, future work should examine the obesity gender gap over varying age ranges.

In conclusion, despite longstanding evidence of an obesity gender gap in U.S. Blacks, the behavioral factors underlying this disparity remain unclear. Because young men and women of the same ethnic group tend to live in similar neighborhoods and originate from similar socioeconomic backgrounds, differences in socioeconomic position and residential environment cannot fully explain the existence of these gender differences in young adults (113). Therefore, understanding the behavioral mechanisms underlying the gender disparity is critical for designing interventions to reduce excess obesity burden in young Black women in the U.S. Additionally, the gender disparities observed offer a supplementary check when evaluating the potential of a novel putative obesity risk factor. For instance, a risk factor that is more common in young Black females than males, especially in low-education families, might seem especially credible. Finally, relatively low obesity prevalence in young Black men from low-parental-education families argues that poorer Black families are not hopelessly obesogenic but may have unrecognized assets that can be leveraged for obesity prevention.

Tables

Table 6. Characteristics (mean or %) of the sample before exclusion of those obese in adolescence, the National Longitudinal Study of Adolescent Health, 1995-96 and 2001-02

	Black		White	
	male	female	Male	female
	n=831	n=1,018	n=2499	n=2648
Mean age, years	21.6	21.3	21.4	21.2
Parental education (%)				
<High school degree	17.0	16.1	9.3	5.1
High school graduate	32.8	41.0	30.5	30.9
Some college/vocational degree	29.4	23.4	31.0	30.4
College or professional degree	20.8	19.5	29.3	33.7
Weight status, adolescence (%)[1]				
Underweight	1.2	2.5	4.4	2.5
Normal-weight	63.9	56.0	63.7	72.8
Overweight	18.4	21.1	16.6	13.6
Obese	16.5	20.4	15.4	11.1
Weight status, young adulthood (%)[2]				
Underweight	1.3	3.2	2.6	4.4
Normal weight	48.3	35.5	45.8	55.5
Overweight	26.6	26.6	30.6	19.6
Obese	23.7	34.8	20.9	20.5

Incident obesity in young adulthood	12.3	21.2	11.1	12.2

[1]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 ≥ 5^{th} percentile or BMI ≥ 18.5) and (BMI < 85^{th} percentile and BMI < 25.0); Overweight: (BMI ≥ 85^{th} percentile or BMI ≥ 25.0) and (BMI < 95^{th} percentile and BMI < 30.0); Obesity: BMI ≥ 95^{th} percentile or BMI ≥ 30.0 (78)

[2]WHO/NIH cutpoints: BMI < 18.5, 18.5-24.9, 25.0-29.9, ≥ 30.0 (79)

Table 7. Distributions (%) of behavioral variables in those non-obese as adolescents, by race and gender, with tests for race-stratified gender differences, the National Longitudinal Study of Adolescent Health, 1994-95 and 1995-96

	Black			White		
	male n=700	female n=803	p-value[1]	male n=2140	female n=2312	p-value[1]
Dinner with parent per week			0.40			0.33
0-2.5	36.9	32.1		14.2	15.8	
3-4.5	32.2	31.4		22.0	23.7	
5-6.5	16.4	19.3		34.8	32.3	
7	14.6	17.3		29.0	28.3	
Sport with mom			0.02			0.60
in past month	15.8	10.4		17.8	18.5	
Sport with dad			<0.01			<0.01
in past month	33.1	16.7		49.7	38.7	
Bouts of MVPA per week			<0.01			<0.01
0 - 1.5	7.1	22.0		10.4	16.5	
2 - 3.5	31.1	49.6		29.3	39.4	
4 – 5	34.5	19.3		27.8	26.3	

5.5+	27.4	9.1		32.6	17.8	
Television hours per week			0.25			<0.01
0 – 4	15.4	15.2		20.5	29.7	
5 – 8	20.7	19.3		20.3	24.3	
9 – 13	9.8	14.1		17.1	15.9	
14 – 18	8.6	6.7		13.7	10.9	
19 – 21	10.2	8.7		11.2	7.2	
22+	35.4	36.0		17.2	11.9	

[1]p-values from chi-square tests of sex differences in distributions of categorical variables

Table 8. Incidence of young adults obesity among Black males and females who were non-obese as adolescents, standardized for adolescent behavioral characteristics, 2001-02

	Black			White		
Standardization variables	Incidence difference (95% CI)	Female incidence (se)	Male incidence (se)	Incidence difference (95% CI)	Female incidence (se)	Male incidence (se)
Parental education and age[1]	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[2]	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 mom[2]	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 dad[2]	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	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[2]	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[2,3]	10.2 (5.2, 15.2)	20.1 (2.2)	9.8 (1.6)	1.1 (-1.1, 3.4)	11.3 (0.9)	10.1 (0.9)

[1]Logistic model: categorical age + parental education + sex + sex* (categorical age + parental education). Ages 18 and 19 and ages 25 and 26 were collapsed into categories of 2-year increments.
[2]Other independent variables in logistic model: categorical age + parental education + sex + (sex*[categorical age]) + (sex*[parental education]). Ages 18 and 19 and ages 25 and 26 were collapsed into categories of 2-year increments.
[3] Logistic model: categorical age + parental education + dinners per week + sport with mom + sport with dad + bouts of physical activity (adolescence) + bouts of physical activity (young adult) + hours of television (adolescence) + hours of television (young adult)

+ sex + sex* (categorical age + parental education + dinners per week + sport with mom + sport with dad + bouts of physical activity [adolescence] + bouts of physical activity [young adult] + hours of television [adolescence] + hours of television [young adult])

Discussion

> "It is also important to note that the shared-environment is the result of the degree of variability of environments that were observed in the sample, and, therefore, it cannot be used to infer the possible effects of altering the environment in which we all live and that may vary only modestly among families." -- Commentary on Wardle, J., et al. 2008 (1). Musani, S.K., et al., American Journal of Clinical Nutrition 2008: 87(2): 275-76 (122).

Summary

We used innovative methodological approaches to study the gender disparity in obesity prevalence in U.S. Black young adults. With the exception of parental education, none of the childhood sociodemographic variables were strongly associated with the gender disparity. Thus, we believe that this research does not implicate differential parental social control in adolescence as a cause of the gender disparity in non-immigrant Black young adults. While 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. Male-female differences in the examined adolescent behaviors did not appear to underlie the overall obesity gender gap in Blacks nor the especially large disparity in those whose parents did not complete high school.

Methodologic considerations

In addition to the limitations and strengths discussed in the Aim 1 and Aim 2 manuscripts, below I highlight and expand on several considerations that are relevant to

future obesity gender disparity research. Our analyses indicated that processes predicting differential obesity incidence between young men and women begin before mid-adolescence. We found that being overweight in adolescence, a risk factor for incident obesity, was more common in Black girls than Black boys (see Aim 2, Table 1). Further, adolescent overweight was much more predictive of incident obesity in Black females than it was in Black males (see Table 12). While there was little gender difference in obesity incidence in those who were normal-weight in adolescence, the gender disparity in the overweight was more than 30 percentage points. Thus, most of the gender disparity in incident young adult obesity was concentrated in those who were already overweight at our study's baseline. Unfortunately, we were unable to extensively investigate behaviors leading to the gender disparity in adolescent overweight or to greater obesity risk for overweight adolescent females than males. Future obesity gender disparity research should address these questions.

In the present study, I defined obesity using nationally recognized BMI cutpoints (78, 79, 123). There is evidence that BMI is both less specific and less sensitive in classifying adiposity in males than in females (80). There is also evidence that the standard BMI cutpoints are not the most optimal ones for classifying excess adiposity in either men or women (80). In general, this measurement error may tend to bias towards the null associations between risk factors and excess weight, especially in men.

BMI's differential measurement error has implications for the study of gender disparities in obesity. In general, a high of BMI cutpoint (e.g., BMI $\geq$ 30.0) will be specific in both men and women but less sensitive in men. Therefore, an analysis using BMI $\geq$ 30 would tend to underestimate obesity incidence in Black males, thereby overestimating the magnitude of the gender disparity (female risk minus male risk) in excess adiposity. Use of a

low BMI cutpoint (e.g., BMI $\geq$ 25.0) that is fairly sensitive in both men and women but less specific in men will tend to underestimate gender disparity. Future research should examine gender differences in obesity using more accurate measures of excess adiposity; using measures, such as waist circumference, targeting other anthropometric characteristics; and performing sensitivity analyses to quantify the potential influence of outcome measurement error. For instance, in analyses in which young adult "overweight" was the outcome, adolescent physical activity was positively associated with incident overweight (25.0 $\leq$ BMI < 30.0) (see Table 9). I suspect this is because the overweight category was capturing men who were active in sports and had high lean body mass.

The analytic strengths of my study also offer guidance for future obesity disparity research. My statistical analysis evaluated the gender disparity on an absolute scale (112). Measures on relative and absolute scales often differ in the context of inter-group variation in risk. Because most previous research on obesity in Blacks assessed risk on a relative scale, quantifying racial and gender disparities on an alternate metric added to the scientific literature. The second advantage of my measure was that it was "decomposable." This feature allowed me to quantify how much of the disparity might be associated with sociodemographic characteristics or differences in the distributions of the adolescent behaviors in males versus females" (112). An additional strength of my analysis is that the modeling strategy imposed many fewer modeling constraints than most parametric analyses: my strategy allowed for heterogeneity of exposure-outcome relationships across gender and non-linearity of the associations between exposures and obesity.

Additionally, I generalized my findings back to explicit target populations, a strategy which is relevant for public health decision-making. In Blacks or Whites, the target

population of my analyses was U.S. young men and women who originated from similar childhood families. Because studies often have a harder time recruiting Black males, especially low-SES Black males, into their studies, differential selection bias by gender is often considerable in studies of U.S. Black populations. While the Black female samples may be fairly representative of the source populations, the samples of Black men are often skewed towards those with stronger social networks or of higher socioeconomic position. For instance, a recent publication concluded that there was no gender difference in the rate of obesity onset in a community sample of Black adolescents (124). However, looking at the descriptive characteristics of the sample in the paper's Table 1, one realizes that the boys included in the study were much more likely than the girls in the study to be from high-SES families. Because high-SES is positively associated with obesity onset in Black boys, this type of selection bias overestimates the prevalence of obesity in Black males and obscures gender differences in obesity prevalence. Further, even selection bias that is non-differential for males and females biases estimates of the magnitude of the gender disparity. Future research should carefully define the target population being examined and take into account bias induced by selection bias that is differentially associated with males' and females' obesity prevalence.

Another strength of my analysis is that I replicated all analyses in White young adults. Replicating analyses in a second population, one with different patterns of gender difference in exposure and outcome, leant more evidentiary weight to the main findings. Analyses of gender disparity in Whites confirmed two relationships observed in Blacks: the association of obesity gender disparity with parental education and the lack of association with adolescent behaviors.

Implications: obesity risk

Longitudinal analyses of population-based studies with measured anthropometric outcomes have identified few behaviors that predict incident obesity or excess weight gain in adolescents and young adults. Using data from the biracial CARDIA study, Lewis et al. searched for factors associated with weight gain in a sample of 18-30 year olds over 7 years of follow-up. Factors examined included intensity-weighted, questionnaire-derived physical activity; parity; cigarette-smoking; alcohol intake; and a treadmill fitness test (118). Only the fitness test strongly predicted weight gain: In Black and White men and women, lower baseline fitness and a larger 7-year decrease in fitness predicted weight gain (118). Recent evaluations of television-viewing and family dinners also failed to show large effects on excess weight gain (41, 71).

A common critique of longitudinal epidemiologic studies that find no relationship between putative obesity-related behaviors and weight gain is that the poor measurement of the behaviors obscures true associations. While self-reported measures are often poor surrogates for the underlying constructs being assessed, more accurate measures do not necessarily find stronger associations. For instance, self-reported physical activity is an imperfect surrogate for energy expenditure or minutes of moderate or vigorous physical activity (MVPA). However, several observational studies assessed precisely measured total energy expenditure, using doubly labeled water, and objectively measured minutes of MVPA, using accelerometers and heart rate monitors. The studies found that even precisely measured energy expenditure (117, 125, 126) or objectively measured physical activity (127-129) do not appear to predict excess weight gain in adolescents or adults. Similarly, the few

randomized trials for weight gain prevention in adolescents and young adults (all of which had physical activity components) were mixed in their findings (129-133). Consistently unsupportive findings across observational, clinical, and experimental settings challenge the hypothesis that higher energy expenditure or minutes of activity per se prevents future weight gain in contemporary populations of U.S. adolescents and young adults.

Contemporary epidemiology often employs a volitional behavior model of obesity onset – whereby obesity-prone individuals make conscious decisions to engage in behaviors that increase their risk of obesity – in explaining differences in obesity prevalence among individuals or groups (1). An alternative to the volitional behavior model posits that, in an obesogenic food environment, sub-conscious, non-volitional mechanisms predispose obesity-prone individuals to be in positive energy balance. The concept of energy balance may be much more relevant in explaining secular trends and inter-group differences than the current paradigm: consideration of either physical activity or energy intake assuming the other factor remains constant. Rather than framing U.S. obesity as a problem of too little energy expenditure, we might do better to articulate it as an inability to down-regulate energy intake accurately in an obesogenic food environment.

Environmental and physiologic forces that affect energy balance on a non-volitional basis may be more important than volitional decision-making in the development of obesity in general and in differential obesity incidence in young men and women specifically. Recent evidence indicates that the U.S. food environment has become so obesogenic that small differences in energy expenditure do not explain much variation in obesity incidence. In NHANES 1959-1962, height and BMI were inversely associated. However, by NHANES 2001-2004, that inverse association had weakened considerably in U.S. adults, disappearing

in males (134). While the extra energy expenditure associated with greater height probably constrained weight gain in the past, today that additional energy expenditure appears to be much less of a constraint (134). In the U.S., cheap calories are so readily available and intake so encouraged that people easily compensate for extra energy expenditure without consciously registering it. The most pressing question for obesity prevention is what factors makes the environment so obesogenic. I believe that macroeconomic pricing structures and intense food marketing are key factors contributing to the obesogenicity of the current food environment.

The next most pressing questions are why some people are good and others bad at matching intake to expenditure on a day-to-day, week-to-week basis and what behaviors could help poor matchers achieve optimal energy balance? The observational epidemiology literature suggests possible answers to the second question. Certain factors, including breakfast-skipping, restrictive dieting and eating patterns, exposure to food marketing, and low fitness levels, may exacerbate the impairment of physiological mechanisms that tend towards energy balance and appropriate compensation of intake to expenditure. Factors that may influence energy balance along psychological and neurological pathways include self-efficacy associated with adopting habits perceived as healthful; emotional health; and buffers against chronic stress (e.g., financial security, strong social networks).

Another factor that may improve regulation of energy balance is physical activity: not – as usually assessed – by increasing energy expenditure but through psychological benefits; feelings of self-efficacy in weight regulation; or greater fitness. One factor consistently associated with impaired energy balance is a decrease in physical activity. Even among very physically active runners, any decrease in miles run was associated with weight gain (115).

Even for highly active people, in a food environment as obesogenic as ours, it may be difficult for the body to down-regulate energy intake to compensate sufficiently for sudden decreases in energy expenditure.

Implications: gender disparity in obesity risk

Conceptualizing obesity as physiologically determined disregulation of energy balance offers new ways to understand the obesity gender disparity in U.S. Blacks. Some of the behaviors described above – dieting behaviors, lower fitness, rapid decreases in physical activity – are more common in women than in men. In addition, cultural norms may predispose women to positive energy balance because they spend more time preparing food and because coping mechanisms more common in men may be stigmatized more in women. Additionally, from an evolutionary biology perspective, women may be more physiologically predisposed to weight gain than are men because extra weight may be more advantageous n women than in men (135). Finally, chronic stress may disrupt the regulation of energy balance differently in women than in men. In our study, the largest excesses of female obesity in Whites and Blacks were observed in those at the lowest socioeconomic position (81). In addition, in the U.S. extreme food insecurity are associated with greater obesity in women but lower obesity in men (98). Perhaps, through non-volitional alterations in energy intake or expenditure, men and women respond differently physiologically to chronic stress.

Another puzzle is why Black and White women show much more variation in obesity prevalence by socioeconomic position than men do. It could be that women show more variation in risk factors (like those described earlier) associated with disregulation of energy balance. Specifically, the greater variation in women by socioeconomic position may be

because women are engaged in more dieting behavior, attempting to rely on cognitive control of energy balance and thus impairing physiologic regulation of energy balance. In high-SEP women, cognitive control may be a viable means of weight gain prevention. With relatively stronger cognitive skills, less chronic stress, more control of one's eating environment, more economic resources, greater control over one's time, and social support around weight control (136), continual monitoring of food intake may somewhat effective as a means of weight control. However, for women without these advantages attempts at cognitive control are more likely to be unsustainable and ultimately self-defeating. Health promotion strategies that rely on individual behavior change rather than changes to the environment often exacerbate health disparities. The emphasis on self-control and individual responsibility for weight control may be exacerbating economic disparities in obesity in U.S. women.

Conclusion

The great disparities observed between males and females from low-resource families provides further evidence that parental behavior may not play a large role in economic and racial disparities in obesity risk. A focus on the volitional behaviors of children and their parents may fail to address more salient factors influencing obesity risk in an obesogenic environment like the U.S. Additionally, obesity prevention efforts that focus on cognitive control of energy intake and expenditure may be much more effective in well-educated, high-resource populations than in populations of less well-off people, thus exacerbating economic disparities in obesity development. The large gender gap among low-education families does offers one hopeful insight: obesity is not an inevitability for poor, Black children. Insights

into the environmental, physiologic, and behavioral underpinnings of differential regulation of energy balance may be key to reducing economic-based, race-based, and gender-based population disparities in obesity prevalence.

Appendix A: Incident obesity and incident overweight, stratifying non-obese adolescents by weight status

Table 9. Incidence of young-adult overweight ($25.0 \leq BMI < 30.0$) among males and females who were underweight or normal-weight as adolescents, standardized to the same distributions of categorical behavioral variables, 2001-02

	BLACK (n=1081)			WHITE (n=3702)		
Standardization variables	Incidence difference % pts (95% CI)	Female incidence % (se)	Male incidence % (se)	Incidence difference % pts (95% CI)	Female incidence % (se)	Male incidence % (se)
Parental education and age[1]	6.4 (-1.6, 14.4)	32.4 (2.3)	26.0 (3.0)	-10.6 (-13.9, -7.3)	19.3 (1.1)	29.9 (1.3)
Dinner w/ parent / week[2]	6.1 (-1.9, 14.1)	32.3 (2.3)	26.2 (3.0)	-10.6 (-13.9, -7.3)	19.3 (1.1)	29.9 (1.3)
Sport with mom[2]	6.6 (-1.1, 14.2)	32.3 (2.4)	24.7 (2.9)	-10.6 (-13.9, -7.3)	19.3 (1.1)	29.9 (1.3)
Sport with dad[2]	5.9 (-2.2, 14.0)	32.0 (2.3)	26.2 (3.2)	-10.0 (-13.4, -6.5)	19.3 (1.1)	29.2 (1.3)
Moderate/vigorous bouts of activity/week, adolescence[2]	7.0 (-0.1, 14.9)	30.8 (2.5)	23.9 (3.0)	-9.6 (-13.3, -6.0)	19.1 (1.1)	28.7 (1.4)
Television-viewing, adolescence[2]	5.8 (-2.3, 14.0)	31.9 (2.3)	26.1 (3.0)	-10.0 (-13.3, -6.7)	19.3 (1.1)	29.2 (1.3)
All behavioral variables[2,3]	5.2 (-3.5, 13.8)	29.2 (2.6)	24.0 (3.1)	-8.7 (-12.3, -5.0)	19.0 (1.2)	27.7 (1.4)

[1]Logistic model: categorical age + parental education + sex + sex* (categorical age + parental education)

[2]Other dependent variables in logistic model: categorical age + parental education + sex + sex*(categorical age) + sex*(parental education)

[3] Logistic model: categorical age + parental education + dinners per week + sport with mom + sport with dad + bouts of physical activity (adolescence) + bouts of physical activity (young adult) + hours of television (adolescence) + hours of television (young adult) + sex + sex* (categorical age + parental education + dinners per week + sport with mom + sport with dad + bouts of

physical activity [adolescence] + bouts of physical activity [young adult] + hours of television [adolescence] + hours of television [young adult])

Table 10. Incidence of young-adult obesity (BMI ≥ 30.0) among Black males and females who were normal-weight as adolescents, standardized for categorical behavioral variables, 2001-02

	BLACK[a] n=867			WHITE n=2818		
Standardization variables	Incidence difference (95% CI)	Female incidence (se)	Male incidence (se)	Incidence difference (95% CI)	Female incidence (se)	Male incidence (se)
Parental education and age[1]	0.3 (-4.2, 4.7)	8.6 (1.9)	8.3 (1.7)	0.7 (-1.5, 3.0)	6.8 (0.8)	6.1 (0.9)
Dinner w/ parent / week[2]	0.6 (-4.0, 5.3)	8.4 (1.9)	7.8 (1.6)	0.7 (-1.5, 2.9)	6.8 (0.8)	6.1 (0.9)
Sport with mom[2]	0.3 (-4.2, 4.9)	8.4 (1.9)	8.1 (1.7)	0.1 (-2.1, 2.4)	6.2 (0.8)	6.1 (0.9)
Sport with dad[2]	0.2 (-4.3, 4.8)	8.7 (1.9)	8.5 (1.7)	0.8 (-1.5, 3.0)	6.9 (0.8)	6.1 (0.9)
Moderate/vigorous bouts of activity/week, adolescence[2]	0.0 (-5.1, 5.1)	8.5 (2.0)	8.5 (1.8)	0.5 (-1.7, 2.7)	6.3 (0.7)	5.8 (0.9)
Television-viewing, adolescence[2]	6.6 (3.0, 10.2)	8.4 (1.9)	1.8 (0.4)	1.0 (-1.2, 3.2)	6.9 (0.8)	5.9 (0.9)
All behavioral variables[2,3]	6.0 (1.8, 10.3)	7.5 (2.0)	1.5 (0.5)	0.3 (-1.8, 2.4)	5.9 (0.7)	5.6 (0.9)

[a]Because of small cell sizes, age categories 24, 25, and 26 were combined into one group in all models of Blacks.

[1]Logistic model: categorical age + parental education + sex + sex* (categorical age + parental education)

[2]Other dependent variables in logistic model: categorical age + parental education + sex + (sex*[categorical age]) + (sex*[parental education])

[3] Logistic model: categorical age + parental education + dinners per week + sport with mom + sport with dad + bouts of physical activity (adolescence) + bouts of physical activity (young adult) + hours of television (adolescence) + hours of television (young adult) + sex + sex* (categorical age + parental education + dinners per week + sport with mom + sport with dad + bouts of physical activity [adolescence] + bouts of physical activity [young adult] + hours of television [adolescence] + hours of television [young adult])

Appendix B: Other combinations of weight status transitions

Table 11. Incidence of young-adult overweight or obesity (BMI ≥ 25.0) among males and females who were underweight or normal-weight as adolescents, standardized to the same distributions of categorical behavioral variables, 2001-02

	BLACK (n=1171)			WHITE (n=3702)		
Standardization variables	Incidence difference % pts (95% CI)	Female incidence % (se)	Male incidence % (se)	Incidence difference % pts (95% CI)	Female incidence % (se)	Male incidence % (se)
Parental education and age[1]	7.1 (-1.1, 15.3)	38.6 (2.2)	31.5 (3.1)	-9.4 (-12.7, -6.1)	24.1 (1.1)	33.5 (1.3)
Dinner w/ parent / week[2]	6.9 (-1.2, 14.9)	38.6 (2.2)	31.8 (3.0)	-9.3 (-12.6, -6.1)	24.1 (1.0)	33.5 (1.3)
Sport with mom[2]	7.0 (-1.0, 14.9)	38.5 (2.2)	31.5 (3.0)	-9.5 (-12.8, -6.2)	24.0 (1.1)	33.5 (1.3)
Sport with dad[2]	6.7 (-1.6, 15.0)	38.7 (2.2)	32.0 (3.3)	-8.8 (-12.2, -5.4)	24.1 (1.1)	32.9 (1.3)
Moderate/vigorous bouts of activity/week, adolescence[2]	6.4 (-1.7, 14.5)	37.4 (2.2)	31.0 (3.2)	-8.8 (-12.3, -5.2)	23.6 (1.1)	32.4 (1.5)
Television-viewing, adolescence[2]	6.6 (-1.2, 14.5)	38.4 (2.2)	31.8 (3.0)	-8.9 (-12.1, -5.6)	24.1 (1.0)	33.0 (1.3)
All behavioral variables[2,3]	4.7 (-3.2, 12.7)	36.6 (2.3)	31.8 (3.2)	-7.9 (-11.5, -4.4)	23.7 (1.1)	31.6 (1.4)

[1]Logistic model: categorical age + parental education + sex + sex* (categorical age + parental education)

[2]Other dependent variables in logistic model: categorical age + parental education + sex + sex*(categorical age) + sex*(parental education)

[3] Logistic model: categorical age + parental education + dinners per week + sport with mom + sport with dad + bouts of physical activity (adolescence) + bouts of physical activity (young adult) + hours of television (adolescence) + hours of television (young adult) + sex + sex* (categorical age + parental education + dinners per week + sport with mom + sport with dad + bouts of physical activity [adolescence] + bouts of physical activity [young adult] + hours of television [adolescence] + hours of television [young adult])

Table 12. Incidence of young-adult obesity (BMI ≥ 30.0) among Black males and females who were overweight as adolescents, standardized for categorical behavioral variables, 2001-02

	BLACK n=332			WHITE n=750		
Standardization variables	Incidence difference (95% CI)	Female incidence (se)	Male incidence (se)	Incidence difference (95% CI)	Female incidence (se)	Male incidence (se)
Parental education and age[1]	32.6 (20.1, 45.0)	56.1 (4.5)	23.5 (4.0)	11.1 (0.2, 20.2)	47.3 (3.5)	36.1 (3.5)
Dinner w/ parent / week[2]	35.1 (23.3, 46.9)	56.3 (4.5)	21.3 (4.4)	11.5 (2.5, 10.4)	47.5 (3.5)	36.0 (3.4)
Sport with mom[2]	31.1 (18.5, 43.7)	55.0 (4.6)	23.9 (4.1)	11.4 (2.2, 20.6)	47.3 (3.5)	35.9 (3.5)
Sport with dad[2]	33.7 (21.9, 45.5)	57.8 (4.3)	24.1 (3.9)	12.0 (2.7, 21.3)	48.1 (3.6)	36.1 (3.5)
Moderate/vigorous bouts of activity/week, adolescence[2]	35.9 (23.5, 48.4)	58.3 (4.6)	22.4 (3.8)	12.3 (2.7, 21.8)	48.0 (3.6)	35.8 (3.7)
Television-viewing, adolescence[2]	35.2 (22.6, 47.7)	55.3 (4.5)	20.1 (4.2)	11.3 (2.3, 20.3)	47.2 (3.4)	35.9 (3.5)
All behavioral variables[2,3]	40.2 (28.9, 51.5)	58.7 (5.0)	18.5 (4.3)	13.7 (4.2, 23.2)	48.7 (3.5)	35.0 (3.6)

[1]Logistic model: categorical age + parental education + sex + sex* (categorical age + parental education)

[2]Other dependent variables in logistic model: categorical age + parental education + sex + (sex*[categorical age]) + (sex*[parental education])

[3] Logistic model: categorical age + parental education + dinners per week + sport with mom + sport with dad + bouts of physical activity (adolescence) + bouts of physical activity (young adult) + hours of television (adolescence) + hours of television (young adult) + sex + sex* (categorical age + parental education + dinners per week + sport with mom + sport with dad + bouts of physical activity [adolescence] + bouts of physical activity [young adult] + hours of television [adolescence] + hours of television [young adult])

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