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Chronic Psychological Stress and Racial Disparities in Body Mass Index Change Between Black and White Girls Aged 10–19

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Abstract

Background—One of the largest health disparities in the USA is in obesity rates between Black and White females.

Purpose—The objective of this study was to test the hypothesis that the stress–obesity link is stronger in Black females than in White females aged 10–19.

Methods—Multilevel modeling captured the dynamic of acute (1 month) and chronic (10 years) stress and body mass index (BMI; weight in kilograms divided by height in meters squared) change in the National Heart, Lung, and Blood Institute Growth and Health Study, which consists of 2,379 Black and White girls across a span of socioeconomic status. The girls were assessed longitudinally from ages 10 to 19.

Results—Higher levels of stress during the 10 years predicted significantly greater increases in BMI over time compared to lower levels of stress. This relationship was significantly stronger for Black compared to White girls.

Conclusion—Psychological stress is a modifiable risk factor that may moderate early racial disparities in BMI.

Keywords

Health disparities; Racial disparities; Stress; BMI; Obesity; Adolescence

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Conflict of Interest Statement The authors have no conflict of interest to disclose.

Introduction

Racial disparities in health persist in the USA. White populations can expect to live almost 5 years longer than Black populations, with troubling disparities in age-adjusted death rates of 1.3 times greater death rate in Black populations than White populations and 2.7 times greater death rate in Black infants than White infants [1]. In addition to disparities in mortality, significant racial disparities in obesity-related chronic diseases [2] such as cardiovascular disease [3] and diabetes [4] also exist, with Blacks suffering from these diseases at markedly higher rates than Whites. Addressing disparities in the USA has been a goal for the past 20 years of the Healthy People objectives, with Healthy People 2020 stating the most ambitious goal to date: “To achieve health equity, eliminate disparities, and improve the health of all groups” [5].

Racial disparities are striking in obesity, with Black populations suffering from significantly higher obesity prevalence rates than Whites [6]. The prevalence of obesity in Black populations is 50 % higher than in Whites, with prevalence rates of 35.7 versus 23.7 %, respectively [6]. These racial disparities are apparent even in childhood [7]. Compared to White girls, Black girls aged 2–19 have 1.99 greater odds of being above the 95th percentile of the Centers for Disease Control Growth Charts from 2000 [7]. One of the largest of these health disparities is the difference in obesity rates between Black and White female adolescents aged 12–19, with 14.7 % of White girls above the 95th percentile of age-adjusted body mass index (BMI) percentile versus 24.8 % of Black girls according to the data from the National Health and Nutrition Examination Survey from 1999–2000 [7].

Researchers have hypothesized a broad array of factors that might be causing these racial disparities in health and obesity. Hypothesized sources range from macro factors, such as neighborhood environment [8] or differential access to healthcare [9], to individual-level factors, such as inequalities in educational attainment [10], metabolic pathways [11, 12], and differences in health behaviors [13]. Many researchers in behavioral medicine focus on a biopsychosocial pathway, testing whether exposure to psychological stress and its resulting physiological response might cause or moderate these disparities [14].

The biopsychosocial model suggests that psychological stress leads to weight gain not only through behavioral pathways such as increased food consumption and sedentariness but also directly through prolonged exposure to biological stress mediators such as cortisol [15]. In two long-term studies, chronic stress predicted weight gain over many decades in adults [16, 17]. Therefore, given the bio-behavioral effects of stress on weight, we hypothesized first that perceived life stress will have cumulative effects over several years that results in greater BMI change. This study allowed us to test whether perceived stress was related to BMI cross-sectionally and, more relevant to the hypothesis, whether cumulative levels of stress over time would lead to accumulation of adiposity over a decade.

Given that ethnic minorities and in particular Black individuals experience greater psychological stress than Whites due to perceived racial discrimination and other factors (18), we were particularly interested in examining whether chronic stress effects on BMI trajectories over time differed for Black versus White females. Geronimus [19, 20]

has proposed a “weathering” hypothesis relating stress and health specifically in Black females, in which these women develop deleterious health outcomes beginning in young adulthood due to cumulative socioeconomic adversity, political marginalization, stigma, and psychological stress. As support for this framework, Geronimus and colleagues have found evidence of increased allostatic load in Black compared to White individuals aged 18–64 [21], and in this study, therefore, we hypothesize that psychological stress may moderate Black/White disparities in obesity.

Obesity and its concomitant racial disparities are emerging ever earlier in life [22]. This is a strong justification for examining factors underlying racial disparities beginning in childhood. Many studies test such hypothesized factors in cross-sectional analyses, but researchers increasingly have called for longitudinal examinations to fully understand the temporal trajectory of any potential mechanisms [23]. Some studies, such as the National Longitudinal Study of Adolescent Health, have made notable progress in understanding racial disparities in obesity by taking a longitudinal approach [24] in adolescence.

Any discussion of racial disparities in health is incomplete without addressing socioeconomic status (SES) as a potential confound. Researchers point to the intertwining of race/ethnicity and SES in studies of health disparities [25]. Some have argued that many racial disparities in health can be explained fully by SES [26], but most agree that race/ethnicity and SES contribute independently, citing examples in which racial disparities in health persist at all levels of SES [27].

In sum, the following challenges remain in eradicating racial disparities in obesity in the USA. First, we must understand the large and significant gap between Black and White women in obesity prevalence. Second, we must shift from a cross-sectional perspective to a longitudinal perspective in order to produce more rigorous predictive models of racial disparities. Finally, we must examine racial disparities controlling for socioeconomic disparities in order to understand the independent contributions of each.

Studying psychosocial stress as a potential underlying factor in the development of racial disparities in obesity within the context of these challenges is difficult. This is due, in part, to the complexity and expense associated with obtaining longitudinal psychological and health data over an extended period of time. However, such data are available in the National Heart, Lung, and Blood Institute (NHLBI) Growth and Health Study. The NHLBI Growth and Health Study was conducted in three US sites enrolling equal numbers of Black and White girls at age 10. A great advantage of this study is that the participants were followed over 10 years, and despite the fact that Black populations have on average lower SES than Whites, White and Black girls from all socioeconomic levels were recruited approximately equally [28].

In the current study, we focus on Black/White disparities in obesity prevalence beginning in childhood at age 10. We do so in a longitudinal, rather than cross-sectional, study that captures 10 years of data, and use statistical methods that model all the available data. We tie these strengths together within one study to test our hypothesis that the relationship between stress and weight from age 10 to 19 is stronger in Black girls than in White girls.

Methods

Participants

The primary aims of the original NHLBI Growth and Health Study were to investigate racial differences in psychosocial factors associated with the development of obesity from pre-adolescence through maturation between Black and White girls. Investigators recruited and enrolled girls 9 and 10 years of age from a school-based population from Richmond, CA; Cincinnati, OH; and from families enrolled in a health maintenance organization in the Washington, DC area. Between 1987 and 1988, 2,379 girls (1,213 Black and 1,166 White) were enrolled. The participants were followed for 10 years, interviewed annually, and provided information on stress and weight. The average age at baseline was 10.1 years, and the average age at the end of the study was 19.1 years. Inclusion criteria at enrollment included: within 2 weeks of age 9 or 10 years at the time of the first clinic visit, Black or White race (by self-report), living with parents/guardians with racial concordance, and having a parent/guardian willing to provide information on household demographics and consent for the girl's participation. Extensive descriptive information on this sample has been published elsewhere [28].

All measures were taken by trained and certified NHLBI Growth and Health Study staff in home visits as described below. The full protocol for the entire study is available at https://biolincc.nhlbi.nih.gov/static/studies/nghs/Protocol.pdf. Certified staff were further periodically monitored for performance to ensure that the three field sites and the ten time points were consistent in their data collection procedures. The public release data set used in this study was obtained directly from NHLBI. Full information on the quality assurance procedures are available at https://biolincc.nhlbi.nih.gov/static/studies/nghs/Summary_of_Collected_Data.pdf.

Measures

Socioeconomic Status—SES information was obtained from the parent or caregiver by trained and certified study staff in home visits and was measured in two ways. Maximum parental income was used as a four category variable (less than $5,000, $5,000–$20,000, $20,000–$40,000, and $40,000 or more) and level of education of the primary caregiver was used as a three category variable (high school or less, 1–3 years post-high school, and 4-year college degree or more). Both were measured at baseline, when the girls were age 10.

Psychological Stress—Perceived psychological stress was measured using the Perceived Stress Scale [29]. This is a validated and reliable measure of the extent to which, in the past month, respondents experienced psychological stress. It is the most widely used measure of perceived stress, and a sample item is, "In the last month, how often have you felt nervous and stressed?" answered on a 5-point Likert scale from *never* to *very often*. In nationally representative samples, the reliability of this scale ranges from Cohen's alpha of 0.78 to 0.91 [30]. This scale does not have a clinical cutoff score. Normal values from a poll of a representative US sample are available [31]. The values obtained in our sample (see Table 1) are within the normal range compared to the US population. The 14-item version of the Perceived Stress Scale was used, with wording simplified to be appropriate for use in

children by the Psychosocial Measures Subcommittee of the original NHLBI Growth and Health Study Investigator team (see protocol link above). The Perceived Stress Scale was administered by trained and certified study staff in home visits at years 2, 4, 6, 8, and 10 of the study. Perceived stress was treated as a continuous variable.

Anthropometry—Trained and certified study staff measured weight and height in home visits to the participants. Height was measured using a stadiometer. Weight was measured using a Detecto Health o Meter electronic scale. Each measurement was performed twice by the same observer, and a third measurement was made by the same observer only if the second measure differed from the first measure by more than 0.5 cm for height and 0.3 kg for weight. Although BMI percentile is appropriate for clinical use and analyses of cross-sectional BMI of children and adolescents, Berkey and Colditz [32] examined longitudinal adolescent data and demonstrated that BMI is the best measure of change in adiposity in longitudinal studies. They argue that effect estimates provided by BMI percentile have greater variability, are more difficult to interpret, and diminish power compared to BMI. Based on their recommendation, as well as the recommendation of Cole et al. [33], and because we have longitudinal data over a 10-year period, we use BMI rather than BMI percentile as the outcome measure. We use this data from the years that the Perceived Stress Scale was administered, and we treated this variable as continuous.

Pubertal Timing—Menarche was measured using a single-item question asking whether the participant had started her periods. This was treated as a dichotomous variable indicating whether a girl had reached puberty or not.

Results

Analytic Plan

Grand means, standard deviations, and ranges as well as these statistics stratified by race were calculated for descriptive statistics. Although least squares regression modeling examining post-test values controlling for baseline is the standard basic approach to longitudinal data, this approach fails to model full existing relationships when there are more than two time points. Further, longitudinal data in which more than two measurement points are nested within a participant violates assumptions (e.g., independence of observations) of traditional statistical models. In this case, we have data over 10 years, and thus standard approaches are insufficient. We therefore use multilevel modeling (also known as hierarchical linear modeling) to fully exploit the decade of measurement available in the NHLBI Growth and Health Study and capture the dynamics of BMI change from age 10–19.

Multilevel modeling uses a random effects model with restricted maximum likelihood estimation to simultaneously estimate both within-subject variance (year-to-year fluctuations in stress) and between-subject variance (such as Black girls versus White girls). This approach also allows us to understand [1] whether acute stress (within-subject stress) relates to BMI cross-sectionally at each year and [2] whether chronic stress over the entire study period relates longitudinally to BMI.

We tested the hypothesis that the stress–BMI association differs by race by estimating a multilevel growth curve model testing a three-way interaction between stress, time (i.e., growth), and race. Because our conceptual model of stress and disparities is based on cumulative stress, we considered our hypothesis supported if this chronic stress× time×race interaction was statistically significant. This final model controlled for pubertal timing (whether each girl had reached menarche or not) and parental income and education.

According to the recommendations of Raudenbush and Bryk [34] and others [35], our analysis took place in five steps, and we specified random intercept maximum likelihood estimation models. All steps had BMI as the outcome. In step 1, we built an unconditional means model with no predictor variables in it simply to understand the amount of variance in BMI partitioned into within- versus between-person variance. In step 2, we built an unconditional growth model with only time as a predictor variable to understand the extent to which within-person variation was a function of the passing of time from childhood to adulthood (growth). In step 3, we built a conditional means model to test only the direct effects of all of the predictor variables on BMI—acute and chronic stress, time, race, and the potential confounds of pubertal timing, parental income, and parental education. In step 4, we built a conditional growth model, adding two-way interaction terms to the prior model to test race×time, acute stress×time, and chronic stress×time interactions. Finally, in step 5—the critical test of our hypothesis—we built a conditional growth model and added the three-way interaction between chronic stress, time, and race. If variables were not statistically significant in any of the steps, they were dropped from all further steps. All error terms were assumed to be independently and normally distributed, with μ=0 and constant σ^2.

Descriptive Statistics

Table 1 displays descriptive information on the variables in this study. *T* tests indicated that significant disparities in BMI existed in the study, with Black girls having a higher BMI than White girls at all time points (p<0.001). This difference in BMI widens by age 19 (see Fig. 1). *T* tests indicated that there were no differences in stress between Black and White girls at each individual timepoint, with the exception of year 8, when the girls were age 17 (see Table 1). Aggregated across the entire study period, Black girls had higher BMI than White girls (Black mean=23.38, SD=5.62 versus White mean=21.22, SD=4.01; t=−10.86, p<0.001) and White girls reported more stress than Black girls (Black mean=23.83, SD=4.97 versus White mean=24.37, SD=5.64; t=2.47, p<0.05).

Step 1: Unconditional Means Model—We partitioned the between- and within-person variation in BMI across time. The estimates of the residual and intercept covariance parameters were 10.17 (SE=0.17) and 24.67 (SE=0.82), respectively. The intraclass correlation was 0.59—in other words, 41 % of the variation in BMI occurred within each girl (p<0.001) and 59 % between individuals (p<0.001).

Step 2: Unconditional Growth Model—We next examined the growth in BMI as a function of time alone to determine the extent to which the within-person variation was a function of the natural effect of growth from childhood to adulthood. Including time into the model, the residual variation dropped from 10.17 to 4.64, suggesting that growth accounts

54 % of the change in BMI over time, leaving 46 % of the variation unaccounted for. Results indicated that across all participants, BMI followed a significant linear relationship ($B_{intercept}$=19.31, SE=0.12, p<0.000; B_{time}=0.72, SE=0.01, p<0.001).

Step 3: Conditional Means Model—Next, we tested direct effects—whether acute and chronic stress and the covariates of income, education, pubertal status, natural effect of growth, and race predicted latent average BMI over the 10 years. Findings indicate that chronic stress was related to BMI over the 10 years (B=0.08, SE=0.02, p<0.001), whereas acute stress was unrelated to BMI at each time point (B=0.00, SE=0.001, p=0.18). White girls had significantly lower BMI compared to Black girls (B=−1.82, SE=0.23, p<0.001). Girls with greater parental education had significantly lower BMI compared to girls with lower parental education (B=−0.38, SE=0.16, p=0.02). Girls with higher parental income had lower BMI than girls with lower parental income (B=−0.24, SE=0.12, p=0.04). Finally, reaching puberty was related to greater BMI (B=0.96, SE=0.08, p<0.001).

Step 4: Conditional Growth Model—Next, we tested whether chronic stress and/or race predicted growth in BMI over the 10 years, including covariates. Results indicated that baseline and change over time was a function of race ($B_{time×race}$=−0.19, SE=0.02, p<0.001). Black girls had significantly greater BMI at study entry than White girls (Black girls $B_{intercept}$=20.42, SE=0.23 versus White girls $B_{intercept}$=19.53, SE=0.30). Black girls also had significantly steeper growth over time (B_{time}=0.71, SE=0.08, p<0.001) than White girls (B_{time}=0.52, SE=0.01, p<0.001). Additionally, chronic stress levels significantly predicted change over time ($B_{time×stress}$=0.01, SE=0.001, p<0.001), such that steeper growth occurred in those with higher chronic stress (B_{time}=0.68, SE=0.01, p<0.001) compared to those with lower stress (B_{time}=0.58, SE=0.01, p<0.001). High and low chronic stress are defined as one standard deviation above and below the mean of chronic stress, respectively.

Step 5: Final Model—In our final model, we tested our hypothesis by testing the three-way interaction between chronic stress, time, and race. This revealed a significant three-way interaction ($B_{time×stress×race}$=−0.01, SE=0.003, p=0.001). To interpret this interaction, we used methods recommended by Cohen, Cohen, West, and Aiken [36] by examining the interaction slopes across time in BMI at one standard deviation above and one standard deviation below the mean in chronic stress for Black and White girls. Table 2 presents these effects of chronic stress over time for each race on the intercept and slope.

As Table 2 displays, chronic stress results in BMI growth over time for both Black and White girls, indicated by the fact that higher chronic stress levels have larger slope estimates in both races. Importantly, the effect is stronger in Black girls as indicated by the larger slope estimates for Black girls regardless of the level of chronic stress. Presented another way, for high-stress Black girls, one unit increase in stress was equal to 0.8 BMI unit increases every 2 years, whereas for high-stress White girls one unit increase in stress was equal to 0.55 BMI unit increases.

Discussion

This study documents a relationship between chronic perceived stress and BMI over a decade of growth in Black and White girls from age 10 to age 19 in the NHLBI Growth and Health Study. By age 19, the average BMI for Black girls was in the “overweight” category and the average BMI for White girls was in the “normal weight” category. In BMI percentile terms, 23.2 % of Black girls had a BMI above the 95th percentile compared to 10.3 % of White girls. This disparity mirrors data from those younger than 19 years of age from the National Health and Nutrition Examination Survey [37], indicating that our sample may be somewhat representative of the USA. We found that the relationship between perceived stress and BMI was stronger in Black rather than White girls, and speculate that this may be one potential moderating factor in the observed disparities.

Our findings extend the work of prior studies on cumulative stress and weight gain [17] to the period of childhood. They also support the “weathering” hypothesis [19, 20]. Geronimus and colleagues found increased allostatic load in Black compared to White individuals aged 18–64 [21], and our results suggest that any weathering Black females experience may begin even earlier. Our findings also fall in line with broader models of health disparities. For example, Krueger and colleagues [38, 39], Williams and colleagues [27, 40, 41] and others have posited a social vulnerability model of health disparities, in which disadvantaged groups are most vulnerable to the deleterious health effects of stress and stressful environments.

That we observed these relationships in this sample of young women beginning at age 10 underscores the importance of focusing on earlier developmental stages as we try to understand the role of stress in health disparities. Obesity rates are increasing in children [42], and long-term treatments for obesity are elusive and some even argue counterproductive [43]. These two challenges clearly point to the prevention of obesity and its associated disparities as a more effective strategy to effect significant changes in long-term health.

This study adds to the idea that racial disparities in obesity are not solely due to socioeconomic status. All of the findings reported here are independent of both parental income and education, and therefore it appears that more work needs to be done to understand the pathways to and moderators of disparities that are unique to race and ethnicity. However, the relationship between race and SES in health disparities is complex [44], and our method of controlling only for baseline income and educational attainment (the data available) provided only a rough accounting for SES. Further, SES measures such as educational attainment may not be commensurate between different racial groups [45], and individual-level SES measures do not capture neighborhood- and community-level socioeconomic and health environmental factors. For example, at the same level of income and education, Black populations are less wealthy and may live in environments such as food deserts that do not promote health [46]. In terms of child populations, Acevedo-Garcia and colleagues [47] found that 76 % of Black children live under worse conditions than the worst off White children (defined as being the 25 % percent living in the highest-poverty White neighborhoods). Future investigations with available data should take into account

these macro factors to fully understand the relationship between race, SES, and obesity disparities in stress.

Stress can promote obesity through affecting food intake and through physiological pathways. In non-human animal models, stress promotes preferential intake of comfort food and highly dense calories, and this in turn appears to “comfort” or blunt the stress response [15], suggesting a functional positive feedback loop which might further induce overeating under stress. In humans, stress also predicts greater intake of comfort food [48, 49]. Chronic stress exposure can also shifts cells into fat storing mode [50]. Chronic stress also induces long-term changes in systems regulating energy balance such as insulin sensitivity, and thus it may take years for stress-induced accumulation of weight, as found in population based studies [17]. Consistent with this idea, we found that within any given year in cross-sectional analyses, stress and BMI were not related. This indicates that rather than short-term stress (stress over the last month), it appears to be chronic stress over many years that accounts for increased BMI in the long term. The divergence in these findings emphasizes the need to conduct studies with longitudinal designs so that important effects on slow-changing outcomes such as BMI that might be invisible in cross-sectional analyses can be observed.

Black and White girls reported equal levels of stress at all but 1 year, which is consistent with nationally representative samples of adults from 1983 to 2009 [30], though on average, across the study, Black girls reported less perceived stress. Despite the lower reporting by Black girls in overall stress levels, stress had a stronger relationship with weight gain in Black rather than White girls. There may be differential response to stress behaviorally, as we know that some people respond to stress with loss of appetite and some respond with eating. Jackson and colleagues, for example, have suggested that Black individuals may use eating to cope with stress at a higher rate than White individuals [51, 52]. In adults, those of higher initial BMI tend to gain weight under stress, whereas those who are lean tend to lose weight [53]. In this cohort, Black girls did start off with higher BMI and may have responded to stress with more stress eating or possibly with more insulin which would promote fat storage [15].

We note a number of limitations in this study. The NHLBI Growth and Health Study is comprised of only Black and White girls, and we cannot speak to stress–BMI relationships in boys or in other races and ethnicities. The relationship between gender, obesity, and racial disparities is multifaceted. For example, years of life lost due to obesity is significantly greater for Whites at nearly all ages than it is for Blacks [54], and this effect is particularly pronounced in women. This could be explained in part by the healthier (more subcutaneous) fat patterning that Black women have compared to White women at the same level of BMI [55, 56]. On the other hand, obesity disparities in Black and White adults [6] and children [7] are more pronounced in females than in males, despite the fact that Black/White disparities in mortality and chronic disease occur across both men and women [1]. Our findings point to stress as a possible factor accounting for this gender difference, as women in the USA report more perceived stress in probability samples than do men [30].

We were unable to test the relative importance of stressful events versus perceived stress in the stress–BMI relationship as prior studies have done [39]. Obesity and its concomitant

disparities have been documented as early as age 2 [57], and in this study we observed significantly different BMI values at baseline, and thus the investigation of stress and weight could have focused on an even earlier age than 10. Waist–hip ratio, or some other measure that accounts for central adiposity, is another interesting and important outcome that we were unable to test in this study.

The data are over 15 years old in 2012, and cohort effects may have accounted for our pattern of findings. The late 1980s to late 1990s reflect a period when obesity prevalence rates were increasing but were lower than they are in 2012 [58]. Since the start of the NHLBI Growth and Health Study, the environmental factors that promote obesity have steadily increased [59]. These so-called obesogenic environments, characterized by examples such as increased food advertising, particularly to children [60, 61], the proliferation of hyperpalatable, nutrient-poor foods [62], limited access to grocery stores [63], or decreased opportunities for physical activity [64], may have a greater impact in the obesity epidemic in 2012 than stress. Therefore, it is possible that if this study was conducted in 2012, our results may be attenuated. However, others have argued that stress-related effects on obesity are exacerbated by growing modernization [65], suggesting that our results may have been similar or even stronger.

Despite these and other limitations, the strengths of this study include examining a younger age group in a large sample with a spread of SES. We have done so using methods that fully characterize the decade of measurement available and have come one step closer in understanding one of the largest racial health disparities in the USA today.

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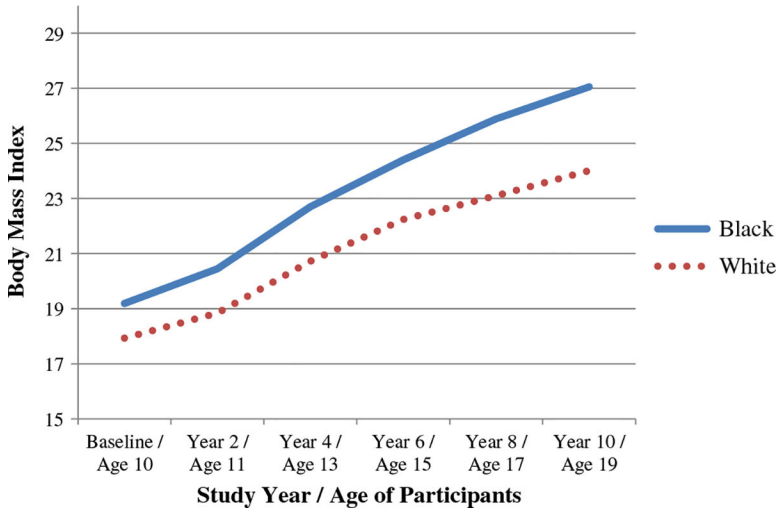

Fig. 1.
Growth in BMI in the NHLBI Growth and Health study

Table 1

Descriptive statistics for demographic, predictor, and outcome variables for baseline year and years 2, 4, 6, 8, and 10 of the National Heart, Lung, and Blood Institute Growth and Health Study

Variable	**Baseline—age 10**		**Year 2—age 11**		**Year 4—age 13**	
	Black	**White**	**Black**	**White**	**Black**	**White**
N	1,213	1,166	1,201	1,158	1,120	1,026
Income (%)						
Less than $5,000	27.9	7.8				
$5,000–$20,000	19.2	9.5				
$2,0000–$40,000	29.5	32.4				
$40,000 or more	23.3	50.3				
Education (%)						
Less than high school	31.5	20.2				
1–3 years post-high school	47.4	30.2				
4-year college degree or more	21.1	49.7				
Perceived stress						
Mean (standard deviation)			25.0a (6.7)	25.0a (7.2)	23.9a (6.9)	24.4a (7.2)
Range			0–49	0–51	2–51	3–51
Weight (kg)						
Mean (standard deviation)	39.6a (11.2)	34.3b (8.6)	46.2a (13.0)	40.5b (10.4)	57.9a (15.0)	52.0b (12.2)
Range	20.3–81.1	19.9–75.1	23.0–93.1	22.3–80.7	29.9–112.3	27.4–100.3
Height (cm)						
Mean (standard deviation)	142.9a (7.9)	139.6b (7.1)	149.5a (8.1)	145.9b (7.6)	159.5a (6.6)	158.0b (7.1)
Range	121.3–169.0	117.2–163.3	126.6–171.6	123.2–169.5	138.7–179.1	135.1–177.1
BMI						
Mean (standard deviation)	19.2a (4.2)	17.9b (3.3)	20.5a (4.7)	18.8b (3.6)	22.7a (5.4)	20.7b (4.1)
Range	12.4–35.2	11.2–34.4	12.4–39.5	12.4–36.5	13.7–42.6	13.1–39.7
Variable	**Year 6—age 15**		**Year 8—age 17**		**Year 10—age 19**	
	Black	**White**	**Black**	**White**	**Black**	**White**
N	975	834	972	914	985	958
Income (%)						
Less than $5,000						

Variable	Baseline—age 10		Year 2—age 11		Year 4—age 13	
	Black	White	Black	White	Black	White
$5,000–$20,000						
$2,0000–$40,000						
$40,000 or more						
Education (%)						
Less than high school						
1 −3 years post-high school						
4-year college degree or more						
Perceived stress						
Mean (standard deviation)	23.6a (7.3)	24.2a (8.2)	23.7a (7.7)	25.3b (8.3)	22.7a (7.5)	23.0a (8.5)
Range	0–55	2–53	2–54	4–52	0–55	3–55
Weight (kg)						
Mean (standard deviation)	64.5a (17.0)	59.5b (12.3)	69.0a (19.4)	62.7b (13.5)	72.7a (21.1)	65.6b (15.2)
Range	32.3–120.8	33.0–108.9	32.4–134.5	39.5–125.1	39.4–151.2	40.0–123.2
Height (cm)						
Mean (standard deviation)	162.4a (6.2)	163.5b (6.1)	163.1a (6.3)	164.7b (6.3)	163.8a (6.2)	165.3b (6.2)
Range	142.7–179.3	143.5–180.3	143.1–181.5	144.0–182.9	146.6–183.6	143.7–182.4
BMI						
Mean (standard deviation)	24.4a (6.1)	22.2b (4.2)	25.9a (6.9)	23.1b (4.6)	27.1a (7.5)	24.0b (5.4)
Range	15.1–46.6	13.6–42.0	15.4–51.2	15.4–47.2	15.8–55.6	15.9–47.5

Within each study timepoint, different letters indicate Black and White girls differed significantly at $p<0.001$

Table 2

Interaction estimates across time with BMI as the outcome at one standard deviation above and below the mean in chronic stress stratified by race in a conditional growth model testing the three-way interaction between chronic stress, time, and race

	Black		White	
	Intercept (SE)	**Slope (SE)**	**Intercept (SE)**	**Slope (SE)**
High chronic stress	20.38 (0.28)	0.80 (0.02)	19.81 (0.32)	0.55 (0.02)
Low chronic stress	20.43 (0.29)	0.62 (0.02)	19.23 (0.35)	0.48 (0.02)

All analyses are adjusted for acute stress, pubertal timing, parental income and education. High chronic stress is defined as one standard deviation above the mean of chronic stress, and low chronic stress is defined as one standard deviation below the mean of chronic stress. The intercept represents baseline, and the slope represents growth over time. All $p<0.001$