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Longitudinal Stability of Disordered-Eating Symptoms From Age 12 to 40 in Black and White Women

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Abstract

The purpose of the current study was to test the longitudinal association between disordered eating symptoms (body dissatisfaction, drive for thinness and bulimia) in adolescence (ages 12, 14, 16, 18, 19) and adulthood (age 40) in a sample of 883 white and Black women. We also investigated moderation by race. Adolescent symptoms at each time point significantly predicted adulthood symptoms for the body dissatisfaction and drive for thinness subscales, for both Black and white women. Bulimia symptoms in adolescence predicted symptoms in adulthood; however, the effect was largely driven by white women. Although moderation was non-significant, among white women, bulimia symptoms at all adolescent time points predicted adulthood bulimia, but among Black women, only symptoms at ages 18 and 19 were predictive of adulthood bulimia. Results

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Conceptualization: J. Parker, J. Levinson, J. Hunger, and A.J. Tomiyama; Methodology: J. Parker, J. Levinson, C. Enders, and A.J. Tomiyama; Software: C. Enders; Formal Analysis: J. Parker and C. Enders; Investigation: B. Laraia, E. Epel, and A.J. Tomiyama; Writing – original draft: J. Parker, J. Levinson, C. Enders, and A.J. Tomiyama; Writing – review & editing: J. Parker, J. Levinson, J. Hunger, C. Enders, B. Laraia, E. Epel and A.J. Tomiyama; Supervision: J. Hunger, C. Enders, B. Laraia, E. Epel, A.J. Tomiyama; Funding acquisition: J. Parker, B. Laraia, E. Epel, A.J. Tomiyama;

Conflicts of Interest

The authors declare that there were no conflicts of interest with respect to the authorship or the publication of this article.

Pre-registration

All analyses were preregistered at <https://osf.io/6dws2/>.

Reporting

We report how we determined our sample size, all data exclusions, all manipulations, and all measures in the study.

Ethical Approval

All research was conducted in compliance with the American Psychological Association ethical standards for the treatment of human subjects. The present secondary data analyses did not meet the definition of human subjects research per UCLA's Office of Human Research Protection Program and were accordingly granted an Institutional Review Board Exemption (IRB #13-000876).

suggest that both Black and white women are susceptible to disordered eating and that symptoms emerging in adolescence can potentially follow women into midlife.

Keywords

Body dissatisfaction; bulimia; Black women; disordered eating; longitudinal

Introduction

Eating disorders and their symptoms are associated with myriad mental and physical health consequences, including cardiovascular disease (Casiero & Frishman, 2006), neurological deficits (Johnson et al., 2002), suicidal ideation (Neumark-Sztainer et al., 1998), substance use (Crow et al., 2006), and pre-mature mortality (Keel et al., 2003). Symptoms of eating disorders often begin in adolescence, concurrent with puberty, during which young girls become increasingly vigilant of their changing body shape and size (Kelch-Oliver & Ancis, 2011; Voelker et al., 2015). Meta-analyses indicate that while lifetime prevalence of clinical eating disorders is only 8% among women, over 60% of adolescent girls present with sub-clinical disordered eating behaviors and cognitions (Bartholdy et al., 2017). Accordingly, studies demonstrate that the onset of symptoms occurs long before a clinical diagnosis, and may persist throughout the lifespan (McClelland et al., 2020; Stice et al., 2011; Stice & Van Ryzin, 2019). For example, one study of elementary- and middle school-aged children illustrated short-term stability of symptoms—children who reported restricting or bingeing food in fifth grade were about 3 and 6 times more likely to report those same behaviors, respectively, one year later (Combs et al., 2013). Kotler et al. (2001) provide evidence for longer-term stability of eating disorder symptoms—girls that exhibited disordered eating symptoms in early adolescence had approximately 2–3 times greater odds of experiencing those symptoms 17 years later. Nonetheless, questions remain regarding how stable disordered eating symptoms are across longer periods of time, or if a specific age of onset in adolescence is particularly influential for stability of symptoms into adulthood.

Moreover, recent dialogue has elucidated the extent to which racial and ethnic minorities remain underrepresented in disordered eating research (Egbert et al., 2022; Sonnevile & Lipson, 2018)—a meta-analysis demonstrates that less than half of studies report race/ethnicity data, and of those that do, white participants make up 70% of research samples. Although Black women make up less than 5% of the research population (Egbert et al., 2022), previous research suggests that they demonstrate patterns of disordered eating distinct from white women. Indeed, eating disorders and embodiment occur in a sociocultural context, one that specifically includes racial and cultural influences on eating, beauty, body shape, and body size (Watson et al., 2019). For example, perhaps due to cultural norms and differences in idealized body shape and size (White & Grilo, 2005), Black women may be less vulnerable to thin standards of beauty, instead favoring a fuller-figured body (Capodilupo & Kim, 2014; Rucker & Cash, 1992). Several studies assert that Black female college students report fewer body image concerns (Quick & Byrd-Bredbenner, 2014) and less body image distortion than white students (Gluck & Geliebter, 2002); however, from middle school through young adulthood, Black girls report a steeper increase in body

dissatisfaction over time than their white counterparts (Bucchianeri et al., 2013). Potential differences in bulimic symptomology for white and Black women also exist. While some studies assert that white women report more bulimic symptoms than Black women (Perez & Joiner, 2003; Striegel-Moore et al., 2003), across ages 11–14, Black girls score higher than white girls on Bulimia subscale of the Eating Disorder Inventory (Striegel-Moore, Schreiber, et al., 2000). Thus, although the fuller-figured, Afrocentric idealized body shape and size may be protective against body dissatisfaction and disordered eating to an extent, Black women and girls are not entirely immune from the dominant, white-centric cultural influences that idealize thinness and the associated patterns of eating behaviors women may engage in to achieve thinner silhouettes (Williamson, 1998).

Observed discrepancies in incidence and prevalence of disordered eating and body dissatisfaction between racial and ethnic groups may also exist because they have been primarily assessed through a white and Western lens (Wildes et al., 2001). Lower levels of body dissatisfaction and disordered eating reported among Black women may have more to do with overreliance on white-centric conceptualizations of beauty and the inability of existing measures to account for racially-salient influences on disordered eating and notions of beauty (Cassidy et al., 2015; Lowy et al., 2021). Disordered eating behaviors and cognitions among Black and white women are informed by distinct conceptualizations of femininity, with both racial and gender identities contributing to differences in how women think and feel about their bodies (Lovejoy, 2001). This is best contextualized through the lens of objectification theory, which holds that in an oppressive society, women are often cultured to view their bodies through the lens of the oppressor (Fredrickson & Roberts, 1997; Schaefer et al., 2018). Although for White women, sexism undoubtedly influences their eating behaviors and how they feel about their bodies, Black women also contend with racial discrimination. The multiplicative effects of racism and sexism on disordered eating and embodiment have not been extensively investigated (Watson et al., 2012, 2019). Disordered eating may not only be a means to control body shape and size, but also a coping strategy and a locus of control in oppressive social contexts (Kempa & Thomas, 2000). Thus, in addition to examining disordered eating trends across longer timespans, specific care should be taken to examine the patterns of disordered eating symptoms in different racial groups and specifically, examine how the trajectory of these symptoms may change over time, differentially.

The Current Study

It is imperative to understand the stability of eating behaviors in childhood and adolescence as they are established risk factors for disordered eating symptoms and clinical disorders in adulthood, alongside adverse health outcomes. Although some studies demonstrate stability of disordered eating symptoms across childhood and adolescence, longer-term effects in middle-adulthood are still unknown. To our knowledge, the current analysis is the longest test of the association between disordered eating behavior and cognitions in adolescence (ages 12–19) and those same behaviors and cognitions in adulthood (age 40). Moreover, the present study focuses on a diverse sample of girls to provide insight into the trajectories of eating disorder symptoms in adolescent and adult Black women. We hypothesize that disordered eating symptoms across ages 12 to 19 will predict disordered eating symptoms

at age 40. We expect that the above relationship will be moderated by race for Drive for Thinness and Bulimia such that the longitudinal association between disordered eating in adolescence and adulthood will be stronger in white women compared to Black women as Black women are potentially less susceptible to the Western thin ideal (Ordaz et al., 2018; Rucker & Cash, 1992; Striegel-Moore et al., 2003). Without a clear consensus from existing literature on body dissatisfaction in Black and white women, race moderation for this subscale was treated as exploratory (i.e., we did not propose a directional hypothesis).

Methods

Author positionality

We and others (e.g., (Cole, 2020; Roberts et al., 2020) acknowledge the importance of asserting positionality, or defining the individual lenses through which we as researchers approach a given study or program of research, as our identities may influence how we conceptualize, report, and write about research findings. Eating disorders literature has been disproportionately focused on and conducted by cisgender white women with far fewer studies acknowledging the prevalence of these behaviors among minority women or approaching this field of work through a culturally-salient lens (Halbeisen et al., 2022). Thus, we feel it is important to offer transparency through which this group of authors approach the present study. The first author identifies as a millennial, upper middle-class, cisgender, Black woman and is a psychology doctoral student who studies body image, eating behaviors and health in Black women. The second author identifies a middle-class, cisgender, white female doctoral student in psychology who studies contributors to and consequences of weight stigma and disordered eating behaviors and cognitions in adolescence and throughout the lifespan. The third author identifies as a white, upper middle-class, cisgender gay man, who is trained as a social/health psychologist with particular expertise on the health effects of stigma and discrimination. The fourth author identifies as a white male quantitative psychologist, whose research expertise centers on missing data methodologies. The fifth author is a senior researcher who is a cisgender, white, upper middle-class female with expertise in the measurement and analysis of dietary intake, food security, and eating behaviors. The sixth author is a mid-life, upper middle-class, white woman with a background in health psychology and health disparities. The senior author identifies as an Asian American, upper middle-class, cisgender woman who is a social health psychologist with interests in weight stigma, body image, stress, and eating behavior.

Study Design

Data for the present study were collected as part of the National Heart Lung and Blood Institute's Growth and Health Study, a longitudinal cohort study spanning adolescence (ages 10–19) and adulthood (age 40) in a diverse sample of women. The primary goal of the study was to identify psychosocial factors associated with the development of obesity, including disordered eating behaviors, cognitions, and pathology. The study began in 1987, enrolling Black and white girls between the ages of 9 and 10, across three primary recruitment sites in Richmond, California, Washington, D.C., and Cincinnati, Ohio. The women included in the age 40 follow-up timepoint were from the Richmond, CA site only. Additional details about

the study and participant cohort have been extensively described elsewhere (The National Heart, Lung, and Blood Institute Growth and Health Study & Research Group, 1992).

Participants

Baseline enrollment period for the study spanned from 1987 to 1989, in which investigators enrolled 2,379 girls (1,213 Black and 1,166 white). Inclusion criteria were current age within 2 weeks younger or older than 9 or 10 years old, respectively; self-reported race of White or Black; and living with a parent or guardian of the same self-reported race who was willing to consent on behalf of the child and provide demographic information about the household and family. Across ten annual time points (approximately ages 10 – 19), data were collected via in-home visits, in which a trained study staff member administered psychosocial and demographic questionnaires and collected anthropometric measurements.

Of the 2,379 girls who completed the initial clinical visit and the subset of 883 girls who were from the Richmond, CA site, 624 of those women completed a follow up visit at approximately age 40. After consent for the follow-up study, psychosocial data on stress and eating behaviors was collected via mailed survey. Study staff then scheduled an in-home visit to obtain anthropometric measurements.

We initially pre-registered a sample size of $N=624$; however, this decision would have assumed that the data missing from 259 women who dropped out between adolescent and follow-up data was due to a haphazard missing completely at random process. To avoid this assumption, we ran analyses on both sets of data (i.e., with and without the 259 women included rendering samples sizes of $N=883$ and $N=624$, respectively) and determined that the analyses yielded identical inferences. Thus, the analyses presented below correspond to the full sample of $N=883$, which leverages the more realistic, conditionally missing at random assumption. The parallel analyses for the smaller sample are presented in Supplementary Materials.

Measures

The focal predictors and outcomes in adolescence and adulthood were the Body Dissatisfaction, Drive for Thinness, and Bulimia subscales of the Eating Disorder Inventory, version 1 and 3, respectively (Garner, 2004; Garner et al., 1983). Studies investigating the psychometric properties of the various EDI subscales suggest that all three are valid, reliable, temporally stable, and internally consistent in both Black and white women (Bardone-Cone & Boyd, 2007; Kelly et al., 2012; Rothstein et al., 2017; Wilfley et al., 1996). The Drive for Thinness and Bulimia subscales appear to be factor invariant between Black and white women (Rothstein et al., 2017); however, more recent work suggests that the Body Dissatisfaction subscale captures some but not all of Black women's body image concerns (Parker et al., 2022). Although more culturally-salient measures may more holistically represent Black women's body image concerns (Wilfred & Lundgren, 2021), the EDI-BD subscale nonetheless is reliable and valid for measuring the intended body image concerns (Kashubeck-West et al., 2013). In the present study, this measure can be thought of as indexing the discrepancy between Black women's perceived and idealized body, which reliably predicts their EDI-body dissatisfaction scores (Gordon et al., 2010).

Eating Disorder Inventory-1—The focal predictors in adolescence were the Body Dissatisfaction, Drive for Thinness, and the Bulimia subscales of the Eating Disorder Inventory, Version 1 (Garner et al., 1983). The EDI-1 was administered at ages 12, 14, 16, 18, 19, resulting in 5 measurement timepoints for each subscale (measurement timepoints were dummy coded to account for unequal intervals). A brief overview of each subscale is as follows: The Body Dissatisfaction subscale contains nine items that represent preoccupation with one's own shape and size (e.g., "I feel satisfied with the shape of my body," "I think that my thighs are too big," "I like the shape of my behind."); the Drive for Thinness subscale contains seven items that reflect excessive concern with dieting and weight and a pursuit of a thinner figure (e.g., "I feel very guilty after I eat too much," "I care too much about my weight," "I think about being thinner a lot."); the Bulimia subscale contains seven items that index tendency for uncontrollable eating (bingeing) and associated compensatory behaviors (e.g., "I eat when I am upset," "Sometimes I have started eating too much and could not stop," "I think about trying to throw up to lose weight."). Individual items in each subscale are rated on a six-point scale, corresponding to "always," "usually," "often," "sometimes," "rarely," or "never." The first three response options on the scale are coded 3, 2 and 1, respectively, while the latter three options all receive a score of 0. The resultant score ranges for the Drive for Thinness, Body Dissatisfaction, and Bulimia subscales are 0 – 21, 0 – 27, and 0 – 21, respectively.

Eating Disorder Inventory-3—At age 40, participants again completed the Eating Disorder Inventory, albeit an updated version of the questionnaire (EDI-3; Garner, 2004). The three subscales were scored on the same six-point rating scale; however, responses were coded on a 0 to 4 scale. "Always," "usually," "often," and "sometimes," received respective scores of 4, 3, 2 and 1 while both "rarely" and "never" received scores of 0. The Body Dissatisfaction subscale contained ten items with a score range of 0 – 40 (Cronbach's alpha = 0.86 overall; 0.88 and 0.83 for white and Black women, respectively), the Drive for Thinness subscale contained seven items with a score range of 0 – 28 (Cronbach's alpha = 0.86 overall; 0.89 and 0.83 for white and Black women, respectively), and the Bulimia subscale contained eight items with a score range of 0 – 32 (Cronbach's alpha = 0.86 overall; 0.90 and 0.81 for white and Black women, respectively). Despite minor changes in scoring, subscales index the same disordered eating behaviors.

We additionally conducted confirmatory factor analyses using our own data to test configural and measurement invariance for each EDI-3 subscale in adulthood across Black and white women (Gregorich, 2006). The bulimia subscale demonstrated sufficient configural and measurement invariance across race (comparative fit index [CFI] = 0.95, root mean square error of approximation [RMSEA] = 0.10 [0.09, 0.11], χ^2 difference (9) = 53.75, $p < 0.001$). The body dissatisfaction and drive for thinness subscales had sufficient configural invariance as measured by the CFI but not the RMSEA (CFI = 0.95, 1.00 and RMSEA = 0.10 [0.09, 0.11], RMSEA = 0.008 [0.00, 0.05], respectively). Neither subscale demonstrated metric invariance (χ^2 difference (6) = 18.61, $p = 0.005$ and χ^2 difference (7) = 10.31, $p = 0.17$, respectively). This pattern is largely consistent with previous studies.

Socioeconomic status—Parental income and education were included as covariates (dummy coded) in all analyses and were collected at baseline, when participants were first enrolled in the study in adolescence (age 10). We elected to use a measure of SES in adolescence as opposed to an equivalent measure in adulthood because adolescent SES could plausibly influence both our adolescent and adulthood variables, whereas adult SES could not retrospectively cause adolescent outcomes. Parental income was measured on a four-point categorical scale defined as: (1) less than \$5,000, (2) \$5,000–\$20,000, (3) \$20,000–\$40,000, and (4) \$40,000 or more. Parental education was defined as the highest level of education obtained by the participant’s primary caregiver on a three-point categorical scale: (1) high school or less, (2) 1–3 years post-high school, and (3) 4-year college degree or more.

Analytic Plan

Model Construction—We fit three multiple linear regression models (one per EDI subscale) to examine the relationship between disordered eating symptoms in adolescence and disordered eating symptoms at age 40, to test moderation by race, and to examine the pattern of age-specific effects. Missing data was addressed using a Bayesian model-based imputation procedure. Further details are provided in Supplementary Materials.

Outlier detection was performed in accordance with the pre-registration. For each model, potential influential points were identified by visualizing a plot of model residuals vs. leverage, examining the DFBETAS for each participant, and estimating models with and without any potential influential point(s). In addition, each model was run with and without the inclusion of cases with standardized residuals greater than an absolute value of 3 (i.e., ± 3 standard deviations). Inference was determined to be robust to inclusion/exclusion of data points.

Hypotheses—To test whether disordered eating symptoms throughout adolescence predicted disordered eating symptoms at age 40 (Hypothesis 1), we examined the two-way interaction terms between disordered eating symptoms and age (dummy coded). To test whether the age-specific associations were moderated by race (Hypothesis 2), we additionally included three-way interaction terms between race, disordered eating symptoms, and age.

The three-way interactions reflected whether the age-specific influence of disordered eating (i.e., the two-way interaction) differed by race. If the omnibus test of moderation (i.e., the test of significance for the four three-way interaction terms between race, disordered eating, and the dummy coded age variables) for Hypothesis 2 was significant, we used contrasts to examine the pattern of age-specific effects within each racial group. If the omnibus test of the three-way interaction terms was non-significant, the terms were dropped from the model (along with the two-way interaction terms between race and age, and race and disordered eating), and we examined the lower-order two-way interactions between disordered eating and age, averaged across race. Given that literature examining disordered eating behaviors longitudinally among Black women is limited and the call for more inclusive eating disorders research (Egbert et al., 2022; Lowy et al., 2021; Mikhail & Klump, 2021; Rodgers

et al., 2018), we diverge from our pre-registration to report the race- and age-specific effects (i.e., the age-specific effects for both white and Black women), regardless of significance of the three-way interaction terms. To control Type I error, we used the False Discovery Rate approach (Benjamini & Hochberg, 1995) with a false positive rate set at 5% per model, as stated in our pre-registration.

Preregistration—All analyses were preregistered at <https://osf.io/6dws2/>.

Results

Table 1 presents the descriptive statistics for Black ($n = 455$) and white women ($n = 428$), stratified by age. Means observed for the three EDI-1 and -3 subscales largely appeared to be consistent with published community norms, based on age- and race-specific patterns (Clausen et al., 2011; Gordon et al., 2010; Kelly et al., 2012; Klemchuk et al., 1990; Nyman-Carlsson et al., 2015; Rancourt et al., 2022; Rosen et al., 1988; Rothstein et al., 2017). Specific details on comparable norms for each subscale, stratified by version, and with age- and race-specific reference groups, can be found in Supplementary Materials. We also report on the percentage of participants who scored above a zero on each measure, stratified by race, subscale, and age (Table S1). Additionally, the Eating Disorder Risk Composite (EDRC) score—a global measure of eating concerns—was calculated at age 40 using the EDI-3 and is presented in Supplementary Materials (Garner et al., 1983); Table S2). Since the NHLBI group did not collect clinical diagnoses, we present the EDRC scores and clinical qualitative ranges with both Anorexia Nervosa and Bulimia Nervosa as the reference group. Over 30% and 50% of our sample of Black and white women, respectively, appeared to be at typical or elevated clinical risk for Anorexia Nervosa. Approximately 2% of Black women and 8% of white women had typical or elevated risk for Bulimia Nervosa.

Body Dissatisfaction

The association between body dissatisfaction in adolescence and adulthood was not moderated by race ($F(4, 276.88) = 1.22, p = .43$), nor was there evidence for moderation by race at any time point (Table S3). As such, the three-way interaction was dropped from the model and we examined the two-way interaction between age and body dissatisfaction averaged across race, which was significant ($F(4, 410.03) = 3.75, p = .01$).

Table 2 presents the results for the model predicting the association between adolescent and adulthood body dissatisfaction, averaged across racial groups. The estimate for the β_1 coefficient represents the conditional effect of body dissatisfaction at age 12 on body dissatisfaction at age 40, averaged across Black and white women. At age 12, body dissatisfaction significantly predicted adulthood body dissatisfaction, such that increases in body dissatisfaction at age 12 predict greater body dissatisfaction at age 40, controlling for parental income and education ($\beta_1 = 0.32, p < .001$). To determine the remaining age-specific associations, we estimated the conditional effects at each time point, which are presented at the bottom of Table 2. At ages 14, 16, 18, and 19, increases in body dissatisfaction in adolescence predicted later increases in body dissatisfaction in adulthood (all p 's $< .001$). Note that the effects were noticeably stronger at ages 18 and 19.

Whereas the conditional effects in Table 2 represent the average association at each time point (e.g., is the association between adolescent and adulthood body dissatisfaction significant across Black and white women?), we also wanted to determine whether the conditional effects for each racial group were significant, at each age (e.g., is the association between body dissatisfaction in adolescence in adulthood significant for Black women? Is it significant for white women?). Thus, as exploratory analyses, we additionally estimated these conditional effects stratified by racial group, to examine the pattern of age-specific effects for white and Black women separately (Table S4). At each age, body dissatisfaction in adolescence significantly predicted body dissatisfaction in adulthood, for both Black and white women (all p 's < .01). The observed effects were similar in size and magnitude, which is consistent with the absence of a significant three-way interaction in which the association between age and body dissatisfaction was moderated by race.

Drive for Thinness

The overall association between drive for thinness in adolescence and adulthood was not moderated by race ($F(4, 373.34) = 0.16, p = .96$) and there was no evidence of moderation by race at any time point (Table S5). Thus, the three-way interaction terms were dropped from the model, and it was subsequently re-estimated to examine the age-specific associations, averaged across racial groups. The two-way interaction between age and drive for thinness was significant ($F(4, 256.06) = 3.73, p = .01$).

Table 3 presents the age-specific associations averaged across Black and white women. The estimate for the β_1 coefficient represents the conditional effect of drive for thinness at age 12 on drive for thinness at age 40, averaged across Black and white women. At age 12, drive for thinness in adolescence significantly predicted drive for thinness in adulthood, such that increases in drive for thinness in adolescence predicted greater drive for thinness at age 40, controlling for parental income and education ($\beta_1 = 0.33, p < .001$). To determine the remaining age-specific associations, we estimated the conditional effects at each time point, which are presented at the bottom of Table 3—at ages 14, 16, 18 and 19, drive for thinness in adolescence remained a significant predictor of adulthood drive for thinness, controlling for covariates, such that increases in drive for thinness in adolescence were associated with greater drive for thinness in adulthood (all p 's < .001).

Again, as exploratory analyses, we calculated the conditional effects stratified by racial group to examine the pattern of age-specific effects within each racial group (Table S6). For both Black and white women, drive for thinness across ages 12, 14, 16, 18 and 19 predicted drive for thinness at age 40, such that drive for thinness in adolescence was positively associated with greater drive for thinness in adulthood (all p 's < .01). Effects were again quite similar between racial groups, which is consistent with the absence of a three-way interaction effect where race moderated the two-way association between age and drive for thinness.

Bulimia

The omnibus test of moderation by race was non-significant for the bulimia subscale ($F(4, 278.81) = 0.49, p = .78$). Although age-specific trends for moderation were evident (i.e., at

ages 16 and 19), these effects did not survive correction for multiple comparisons (Table S7). After dropping the three-way interaction terms between race, bulimia symptoms, and age from the model, the remaining two-way interaction between bulimia symptoms and age was significant ($F(4, 324.33) = 15.39, p < .001$).

Table 4 presents the results for the model predicting adulthood bulimia symptoms from adolescent bulimia symptoms, averaged across racial groups. Results indicated that at all ages, bulimia symptoms in adolescence predicted bulimia symptoms in adulthood, with greater adolescent symptoms positively predicting symptoms in adulthood (Table 4). The β_1 coefficient represents the conditional effect of bulimia symptoms at age 12 on bulimia at age 40, averaged across Black and white women. To determine the remaining age-specific associations, we estimated the conditional effects at each age, which are presented at the bottom of Table 4. At age 12, the conditional effect was only marginally significant ($p = .05$), whereas for ages 14, 16, 18 and 19, the effect was statistically significant.

Finally, as exploratory analyses, we examined the age-specific effects for white and Black women separately, at each time point. For white women, bulimia symptoms at all adolescent timepoints predicted bulimia symptoms in adulthood; however, for Black women, only bulimia symptoms at ages 18 and 19 significantly predicted adulthood bulimia symptoms (Table S8).

Discussion

To our knowledge, this 28-year study represents the largest time gap in which the longitudinal stability of disordered eating behaviors has been examined. Moreover, this investigation was conducted in a racially-diverse sample of 455 Black and 428 white women, advancing research equity in a field where some estimates show only 5% of participants have been Black women. We hypothesized that adolescent disordered eating behaviors and cognitions would predict the presence of these same behaviors in adulthood. Further, based on previous literature, we hypothesized that these trends would be distinct between white and Black women. To some extent, our results were in line with the hypotheses: for body dissatisfaction and drive for thinness, adolescent disordered eating behaviors and cognitions predicted adulthood behaviors and cognitions for both white and Black women, and this effect was significant at all time points. When examining this trend for the bulimia subscale, the same trend appears to occur—when averaged across racial groups, bulimia symptoms at all adolescent time points predicted bulimia symptoms in adulthood—however, closer inspection of the race-specific effects reveals that this effect is largely driven by white women. Statistical moderation by race was not evident for any of the three disordered eating behaviors.

Although effects for body dissatisfaction between white and Black women did not differ significantly, it is important to acknowledge that the estimates obtained for Black women may not be adequately capturing all dimensions of body satisfaction. Studies suggest that Black women conceptualize body image differently from their white counterparts (Davis et al., 2010), and indeed, the EDI-body dissatisfaction (EDI-BD) subscale does not adequately capture all body image concerns for Black women (Kashubeck-West et al., 2013). Using

confirmatory factory analyses, Kashubeck-West and colleagues (2013) found that while the EDI-BD subscale was reliable and valid among Black women, the measure does not index racially-salient body image concerns for Black women (e.g., hair, lips, skin color). Indeed, along with others, our own work demonstrates that Black women have unique body image concerns, such as skin color, that are related to their disordered eating behaviors (Capodilupo & Kim, 2014; Parker et al., 2022). Given the downstream health consequences of body dissatisfaction, future research should measure and examine body image through a lens that acknowledges the sociocultural context in which women evaluate their bodies (Mucherah & Frazier, 2013).

Previously, it was widely accepted that Black women were protected from thin idealization due to cultural norms that favor fuller figured bodies (Williamson, 1998). Although it is reasonable, and there is evidence to suggest that Black women do not value thinness as highly as white women do (e.g., Gluck & Geliebter, 2002), this does not suggest that Black women are then immune from the mainstream societal norms that, throughout history, have sought to negate their physical qualities as representative of beauty (Hughes, 2021). Our results support this idea: First, no significant differences in drive for thinness emerged between Black and white women and for both groups drive for thinness in adolescence influences adulthood drive for thinness; however, examining the magnitude of these estimates suggests that Black women may value thinness to a lesser degree.

Unlike research on the body dissatisfaction subscale of the EDI, evidence suggests that the bulimia subscale functions as intended among Black women (Bardone-Cone & Boyd, 2007). However, research estimating the prevalence of bulimia among white vs. Black women has been conflicting—for example, some studies estimate a higher prevalence of bulimia diagnoses among white women (Striegel-Moore et al., 2003), others find equivalent prevalence when comparing bulimia symptoms (Cachelin et al., 2000). Despite this, pervasive stereotypes of bulimia being a white, affluent, thin woman's disease have prevailed, resulting in lower rates of treatment and fewer resources for those who fall outside of that stereotype (Sonneville & Lipson, 2018). Deeper exploration of the etiology and symptomology of bulimia among Black women is particularly important in the context of Binge Eating Disorder, which only recently received DSM diagnostic recognition (American Psychiatric Association, 2013), and has substantial overlap with bulimia. Binge Eating Disorder, in which black women experience high rates (Goode et al., 2020; Striegel-Moore, Wilfley, et al., 2000; Taylor et al., 2013), is distinct only from bulimia in the use of compensatory behaviors (e.g., purging). Since our data did not capture actual eating disorder diagnosis, the endorsement of bulimia symptoms likely has overlap with Binge Eating Disorder. With white and Black women idealizing different body shapes and sizes, bulimic behaviors among white women may coincide with the drive for thinness, but for Black women, perhaps these symptoms could also be related to binge eating or the idealization of a curvier, hourglass figure (Wilfred & Lundgren, 2021).

Interestingly, the relationship between adolescent and adulthood bulimia appeared to emerge at a later age for Black women (ages 18 and 19), than for white women. From a theoretical standpoint, there are myriad psychological, cultural, and familial factors that may explain the apparent later onset (Brytek-Matera & Czepczor, 2017). In the domain of

cultural and psychological influence, it may be that as Black girls enter adulthood, they experience greater pressures toward acculturation, leading to idealization of thinner figures and subsequent engagement in bulimic behaviors to achieve said silhouette. There may also be familial, socioeconomic influence on the age-specific presence of bulimic behaviors for Black girls. For example, due to residential segregation and economic disenfranchisement, Black women are the most likely to live in high density homes, with a greater number of family members living under the same roof (Chambers et al., 2010). The resulting home density may prevent Black girls from being able to secretly engage in some common bulimic behaviors, such as purging, while living at home. Thus, future eating disorders intervention research may benefit from considering age-specific and race-specific influences on the onset of bulimic behaviors.

Limitations

Several limitations of the current study should be considered when interpreting the results. From a methodological standpoint, we acknowledge constraints specifically related to our use of the EDI subscale measures. While age 40, we had sufficient item-level data to calculate psychometrics for the EDI-3 stratified by subscale and race, we did not have item-level data for the EDI-1 in adolescence to include psychometrics for this particular measure. The absence of item-level data in adolescence unfortunately also precludes examination of how the EDI factor structure, psychometric properties, and change patterns across time may vary between Black and white women in our sample. Given that these measures were specifically developed in and for white women, it would be useful for future longitudinal studies to investigate whether this measure retains equivalent validity, reliability, and stability in diverse groups over time. Of note, the Cronbach's alpha for all three EDI-3 subscales was lower for Black women compared to white women in our sample and only the bulimia subscale demonstrated configural and measurement invariance. The body dissatisfaction and drive for thinness subscales showed evidence for configural invariance only. This pattern of results is largely consistent with previous studies and for this reason, we suggest that greater confidence should be put in the analyses stratified by race, rather than those that compare the EDI measures. The disordered eating literature may benefit from updated measures that better capture these behaviors across diverse groups, which may include the addition of questions to examine culturally-salient disordered eating behaviors.

Although some studies indeed demonstrate the validity of the EDI in racial and ethnic minorities, others question whether it fully captures the same constructs in Black and white women (Kashubeck-West et al., 2013). Moreover, the bulimia subscale of the EDI does not disentangle overlapping symptomology with Binge Eating Disorder, which accounts for the largest burden of disease among all eating disorders (Santomauro et al., 2021) and across racial groups (Striegel-Moore, Wilfley, et al., 2000). With regard to demographic methodology, a categorical assessment of race did not allow for more nuanced examination of how race as a multidimensional, socially constructed category influences eating behaviors, given studies that suggest the importance of capturing the influence of racial identity rather than dichotomized racial categories (Roth, 2016). In fact, in the introduction to a recently released guidebook for eating disorder practitioners, titled *Treating*

Black Women with Eating Disorders: A Clinician's Guide, authors Small and Fuller (2020) specifically highlight how heterogeneity *within* Black women's experiences may play a key role in presentation and etiology of disordered eating.

Finally, the absence of data between age 19 and 40 did not allow us to examine how the magnitude of the association between disordered eating symptoms in young adulthood (e.g., 20s and 30s) and adulthood may differ from the patterns we observed in adolescence. Moreover, we did not assess clinical diagnostic status to determine whether the presence of these symptoms is related to threshold-level psychopathology; however, using the EDI-3 EDRC score, over 30% of our sample appeared to be at typical or elevated clinical risk for Anorexia Nervosa. Less than 10% of the sample reached typical or elevated clinical risk for Bulimia Nervosa (Table S2).

Conclusion

Starting in adolescence, disordered eating cognitions and behaviors appear to exert a lifelong influence, remaining stable over the course of 28 years. While research has focused disproportionately on these symptom presentations among white women, our results reveal that Black women, too, experience body dissatisfaction, drive for thinness, and bulimia disordered eating symptoms. These findings affirm more recent assertions that Black women are not immune from body image and eating disturbances, highlighting the adulthood risk associated with disordered eating behaviors for both white and Black girls. Further, in the absence of moderation by race, these results suggest that the present subscales of disordered eating manifest similarly among white and Black women, though future studies should aim to determine the degree to which racially-salient disordered eating and body image concerns are adequately captured with existing measures.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Data, materials, code, and online resources

Analysis scripts and de-identified data specific to the present study are available on the OSF page. In addition, general study data for NGHS Wave I – III (adolescent data, ages 10–19) is available per NIH guidelines at <https://biolincc.nhlbi.nih.gov/studies/nghs/> along with the

full study protocol, a summary of all data collected, and additional details on the study population and biospecimen collection.

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Table 1

Descriptive statistics for participants, stratified by race, across all time points

	Age 10	Age 12	Age 14	Age 16	Age 18	Age 19	Age 40
Black women (n = 455)							
Parental Education (%)							
High school	26.03%						
1–3 years post-high school	54.40%						
4-year college degree	19.57%						
Parental income (%)							
< \$5,000	33.77%						
\$5,000 – \$20,000	22.81%						
\$20,000 – \$40,000	23.84%						
\$40,000	16.58%						
Body Dissatisfaction		6.29 (6.40)	6.23 (6.44)	6.20 (6.45)	7.36 (7.21)	7.87 (7.17)	19.25 (8.68)
Drive for Thinness		4.49 (5.08)	3.63 (4.97)	3.62 (4.98)	3.56 (4.91)	4.10 (5.17)	8.33 (6.86)
Bulimia		2.41 (3.78)	1.96 (3.28)	1.27 (2.38)	0.92 (2.03)	0.92 (2.12)	2.04 (3.94)
White women (n= 428)							
Parental Education (%)							
High school	20.92%						
1–3 years post-high school	39.57%						
4-year college degree	39.51%						
Parental income (%)							
< \$5,000	7.55%						
\$5,000 – \$20,000	12.15%						
\$20,000 – \$40,000	34.49%						
\$40,000	45.80%						
Body Dissatisfaction		7.47 (7.42)	8.52 (7.37)	8.50 (7.39)	10.36 (8.36)	10.60 (8.14)	22.89 (9.12)
Drive for Thinness		4.85 (5.81)	4.92 (5.89)	4.90 (5.92)	5.49 (6.12)	5.80 (6.10)	10.45 (7.71)
Bulimia		1.39 (2.95)	1.37 (2.81)	1.09 (2.58)	0.95 (2.48)	0.91 (2.35)	3.24 (4.59)

Values presented in the table for parental income and education are the percentage of the sample of Black or white women represented in that category. Values presented for Body Dissatisfaction, Drive for Thinness and Bulimia are means and (standard deviations).

Table 2

Body Dissatisfaction Model Results without moderation (Hypothesis 1)

Parameter	Estimate	SE	<i>p</i>
β_0	21.20	0.69	< .001
β_1 (<i>BODY DIS.</i>)	0.32	0.05	< .001
Age			
(<i>AGE 12</i>)	Reference group		
β_2 (<i>AGE 14</i>)	-0.31	0.63	.73
β_3 (<i>AGE 16</i>)	-0.36	0.68	.71
β_4 (<i>AGE 18</i>)	-1.86	0.64	.01
β_5 (<i>AGE 19</i>)	-2.38	0.65	.001
Race			
(WHITE)	Reference group		
β_6 (BLACK)	-2.80	0.32	< .001
Parental Education			
(<i>HIGH SCHOOL</i>)	Reference group		
β_7 (<i>1-3 YRS POST-HIGH SCHOOL</i>)	-1.54	0.45	.003
β_8 (<i>4-YR COLLEGE DEGREE</i>)	-2.50	0.50	< .001
Parental Income			
(< \$5,000)	Reference group		
β_9 (\$5,000 – \$20,000)	1.23	0.58	.07
β_{10} (\$20,000 – \$40,000)	-0.21	0.52	.75
β_{11} (<i>\$40,000</i>)	1.01	0.50	.08
Body Dissatisfaction × Age			
(<i>BODY DIS. × AGE 12</i>)	Reference group		
β_{12} (<i>BODY DIS. × AGE 14</i>)	0.02	0.07	.81
β_{13} (<i>BODY DIS. × AGE 16</i>)	0.03	0.07	.75
β_{14} (<i>BODY DIS. × AGE 18</i>)	0.15	0.07	.05
β_{15} (<i>BODY DIS. × AGE 19</i>)	0.19	0.06	.01
Conditional Effects by Age			
<i>BODY DIS. @ AGE = 14</i>	0.34	0.05	< .001
<i>BODY DIS. @ AGE = 16</i>	0.35	0.05	< .001
<i>BODY DIS. @ AGE = 18</i>	0.47	0.04	< .001
<i>BODY DIS. @ AGE = 19</i>	0.51	0.04	< .001

Table 3

Drive for Thinness Model Results without moderation (Hypothesis 1)

Parameter	Estimate	SE	<i>p</i>
β_0	9.19	0.52	< .001
β_1 (<i>DRIVE FOR THIN.</i>)	0.33	0.06	< .001
Age			
(<i>AGE 12</i>)	Reference group		
β_2 (<i>AGE 14</i>)	-0.22	0.51	.75
β_3 (<i>AGE 16</i>)	-0.22	0.49	.75
β_4 (<i>AGE 18</i>)	-0.96	0.52	.13
β_5 (<i>AGE 19</i>)	-1.20	0.49	.03
Race			
(WHITE)	Reference group		
β_6 (BLACK)	-1.92	0.27	< .001
Parental Education			
(<i>HIGH SCHOOL</i>)	Reference group		
β_7 (<i>1-3 YRS POST-HIGH SCHOOL</i>)	0.73	0.31	.04
β_8 (<i>4-YR COLLEGE DEGREE</i>)	-0.49	0.35	.29
Parental Income			
(< \$5,000)	Reference group		
β_9 (\$5,000 – \$20,000)	-0.27	0.41	.68
β_{10} (\$20,000 – \$40,000)	-1.12	0.39	.01
β_{11} (<i>\$40,000</i>)	-0.59	0.39	.23
Drive for Thinness × Age			
(<i>DRIVE FOR THIN. × AGE 12</i>)	Reference group		
β_{12} (<i>DRIVE FOR THIN. × AGE 14</i>)	0.07	0.08	.60
β_{13} (<i>DRIVE FOR THIN. × AGE 16</i>)	0.07	0.09	.60
β_{14} (<i>DRIVE FOR THIN. × AGE 18</i>)	0.22	0.08	.02
β_{15} (<i>DRIVE FOR THIN. × AGE 19</i>)	0.24	0.07	.002
Conditional Effects by Age			
<i>DRIVE FOR THIN. @ AGE = 14</i>	0.40	0.05	< .001
<i>DRIVE FOR THIN. @ AGE = 16</i>	0.40	0.06	< .001
<i>DRIVE FOR THIN. @ AGE = 18</i>	0.55	0.05	< .001
<i>DRIVE FOR THIN. @ AGE = 19</i>	0.57	0.05	< .001

Table 4**Bulimia Model Results without moderation (Hypothesis 1)**

Parameter	Estimate	SE	<i>p</i>
β_0	3.40	0.31	< .001
β_1 (<i>BULIMIA</i>)	0.12	0.05	.05
Age			
(<i>AGE 12</i>)	Reference group		
β_2 (<i>AGE 14</i>)	-0.08	0.28	.78
β_3 (<i>AGE 16</i>)	-0.15	0.28	.68
β_4 (<i>AGE 18</i>)	-0.42	0.26	.24
β_5 (<i>AGE 19</i>)	-0.30	0.28	.39
Race			
(WHITE)	Reference group		
β_6 (BLACK)	-1.52	0.17	< .001
Parental Education			
(<i>HIGH SCHOOL</i>)	Reference group		
β_7 (<i>1-3 YRS POST-HIGH SCHOOL</i>)	0.25	0.21	.37
β_8 (<i>4-YR COLLEGE DEGREE</i>)	-0.33	0.25	.31
Parental Income			
(< \$5,000)	Reference group		
β_9 (\$5,000 – \$20,000)	0.15	0.28	.68
β_{10} (\$20,000 – \$40,000)	-0.53	0.26	.10
β_{11} (<i>\$40,000</i>)	-0.37	0.27	.30
Bulimia × Age			
(<i>BULIMIA × AGE 12</i>)	Reference group		
β_{12} (<i>BULIMIA × AGE 14</i>)	0.05	0.07	.63
β_{13} (<i>BULIMIA × AGE 16</i>)	0.20	0.09	.09
β_{14} (<i>BULIMIA × AGE 18</i>)	0.59	0.09	< .001
β_{15} (<i>BULIMIA × AGE 19</i>)	0.48	0.09	< .001
Conditional Effects by Age			
<i>BULIMIA @ AGE = 14</i>	0.17	0.05	.01
<i>BULIMIA @ AGE = 16</i>	0.32	0.07	.01
<i>BULIMIA @ AGE = 18</i>	0.72	0.08	< .001
<i>BULIMIA @ AGE = 19</i>	0.60	0.08	< .001