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Ethnic differences in eating disorder prevalence, risk factors, and predictive effects of risk factors among young women

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Abstract

Findings regarding ethnic differences in eating disorder diagnoses and risk factors have been mixed. This study evaluated whether there are ethnic differences in eating disorder prevalence, risk factors, and the predictive relations of the risk factors to future eating disorder onset. We used a large sample of young women followed longitudinally over three years to increase sensitivity to detect differences and to provide the first test of ethnic differences in the relation of risk factors to future onset of eating disorders. Females with body image concerns (N = 1,177) were recruited from high schools and colleges for trials of a body acceptance eating disorder prevention program. They completed surveys and interviews at baseline and at 1-, 6-, 12-, 24-, and 36-month follow-up. Significant differences between ethnic groups were found for two of the 13 baseline risk factors: thin-ideal internalization and body mass index. No significant differences in later onset rates among ethnic groups were found. There were also no reliable ethnic differences in the relation of risk factors for future eating disorder onset. These findings suggest that eating disorders affect ethnic minorities as much as Whites and that there are more overlapping risk factors shared among various ethnic groups than differences.

Keywords

ethnicity; eating disorders; risk factors; prevalence

Eating disorders affect over 13% of adolescent and adult women (Allen, Byrne, Oddy, & Crosby, 2013; Stice, Marti, & Rohde, 2013) and are associated with a range of negative health outcomes. Anorexia nervosa and bulimia nervosa combined is the 12th leading cause of disability-adjusted life years among 15-19-year-old females (Hoek, 2016). Anorexia

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nervosa and bulimia nervosa pose a heightened risk for suicide (Franko & Keel, 2006) and are linked with osteoporosis, gastrointestinal, cardiovascular, and endocrine system pathologies (Crow, 2005; Klein & Walsh, 2003; Mehler & Krantz, 2003). Binge eating disorder is associated with psychiatric comorbidity and severe obesity (Haines & Neumark-Sztainer, 2006; Hudson, Hiripi, Pope, & Kessler, 2007; Wade, Bergin, Tiggemann, Bulik, & Fairburn, 2006) in addition to medical conditions such as type 2 diabetes, metabolic syndrome, sleep disorders, asthma, menstrual and gastrointestinal issues, and cardiovascular disease that are not accounted solely by weight gain (Mitchell et al., 2015; Olguin, Fuentes, Gabler, Guerdjikova, Keck & McElroy, 2017). Given the negative consequences of eating disorders, it is vital to understand the prevalence of eating disorders and risk factors for all ethnic groups. This is important because the portion of ethnic minorities in the US is increasing (Passel & Cohn, 2008). Early eating disorder research investigating the role of ethnicity proposed that White women are at increased risk compared with ethnic minorities. However, more recent studies show this may not be the case, although findings are inconsistent (Wildes & Forbush, 2015).

Some studies have found that White adults have a higher lifelong prevalence for eating disorders including anorexia nervosa (Udo & Grilo, 2018), bulimia nervosa (Striegel-Moore, Dohm, Kraemer, Taylor, Daniels, Crawford, & Schreiber 2003), and binge eating disorder (Udo & Grilo, 2018) compared to other ethnic groups. However, many studies have found no significant ethnic differences in the prevalence of anorexia nervosa (Marques et al., 2011; Swanson, Scott, Crow, Le Grange, Swendsen & Merikangas, 2011), bulimia nervosa (Udo & Grilo, 2018) or binge eating disorder (Lee-Winn, Mendelson, & Mojtabai, 2014; Marques et al., 2011; Swanson et al., 2011) among female adolescents and women. Another study did not detect ethnic differences in the prevalence of any DSM-5 eating disorders (Solmi, Hotopf, Hatch, Treasure, & Micali, 2016).

There have also been mixed findings regarding ethnic differences in risk factors. There is early evidence that White women have a higher subscription to the thin-ideal and body dissatisfaction than African American and Latina women (O'Neill, 2003; Roberts, Cash, Feingold, & Johnson, 2006; Warren, Gleaves, Cepeda-Benito, Fernandez, & Rodriguez, 2005). This is particularly true when comparing body dissatisfaction between Whites and African Americans; the latter consistently report lower body dissatisfaction and more positive body image than the former (Grabe & Hyde, 2006; Wildes, Emery, & Simons, 2001). Although some studies have found that ethnic minorities are less susceptible to eating disorders than Whites, some research suggests much greater similarities in eating disorders and body image concerns among ethnic groups than differences. For example, a meta-analysis found no differences in body dissatisfaction between Asian Americans and Whites (Grabe & Hyde, 2006), which is in line with results from studies that have tested for ethnic differences in eating disorder symptoms and risk factors (Gluck & Geliebter, 2002; Gordon, Castro, Sitnikov, & Holm-Denoma, 2010; Shaw, Ramirez, Trost, Randall, & Stice, 2004). Some studies have found ethnic differences in BMI among women (Franko et al., 2012; Quick & Byrd-Bredbenner, 2014; Striegel-Moore et al., 2003), which can predict eating disorder severity (Berner, Shaw, Witt, & Lowe, 2013).

One potential explanation for these mixed findings is that most studies had small samples, limiting the ability to detect reliable differences between ethnic groups. Another potential reason is the differential influence of the thin-ideal and the Western media on ethnic minority groups. Previous studies have found that ethnic minorities may have certain cultural factors that protect them from body dissatisfaction and eating disorders. For example, many East Asian societies traditionally preferred a larger body size because a slender body was associated with poverty and poor health (Jung & Forbes, 2007). Similarly, among some Hispanic and African American communities, a larger, voluptuous body size is considered more beautiful, and ethnic minority young women tend to endorse larger ideal body size than Whites (Gordon et al., 2010). Ethnic minority's health and dieting behaviors are influenced by whether such behaviors are considered in-group behaviors (Oyserman, Fryberg & Yoder, 2012). Experimental data shows that African and Hispanic Americans ranked dieting and watching one's weight to be racially incongruent behaviors but their ratings of health behaviors changed in response to the manipulation of their racial-ethnic identity (Oyserman et al., 2012). These findings suggest that a stronger identification with certain ethnic minority identity and culture may protect females from developing body dissatisfaction, endorsing the thin-ideal, and engaging in eating disorder behaviors.

Despite the mixed findings, Western media and the drive for thinness that accompanies it, can affect the rate and expression of eating disorders among different cultural communities. Cross-cultural studies have found that exposure to Western media in Asian and Pacific Islander cultures is linked with increased body image and eating disturbances among female adolescents (Becker, Burwell, Herzog, Hamburg, & Gilman, 2002; Watters, 2010). The industrialization and Westernization of many countries have led to changes in body ideals where female adolescents and women have become more concerned with gaining weight, and the ideal body type has evolved from a fuller figure to a thinner one (Gunewardene, Huon & Zheng, 2001; Watters, 2010; Wildes et al., 2001). Thus, one plausible explanation for the similarities in the prevalence and risk factors for the onset of eating disorders is the widespread social-cultural pressure for thinness in Western media.

The Current Study

As noted, some of the mixed findings in ethnic differences in eating disorder prevalence and risk factors may have been due to the small sample sizes used in past studies. Thus, to increase sensitivity to detect potential differences, we combined three studies (N = 1,177) to examine ethnic differences using a large sample of high-risk individuals. All the studies used in-person diagnostic interviews and assessed binge eating disorder, purging disorder, and subthreshold eating disorders. The present sample completed annual eating disorder diagnostic interviews over a 3-year follow-up, providing a unique opportunity to test for ethnic differences in the relation of baseline risk factors to future onset of eating disorders for the first time. We examined the predictive effects of overeating, fasting, excessive exercise, functional impairment, and mental health service usage on eating disorders based on evidence that these factors predict future onset of eating disorders (see Stice, Gau, Rohde & Shaw, 2017).

Method

Participants

We combined data from one efficacy trial (Trial 1; Stice, Marti, Spoor, Presnell, & Shaw, 2008) and two effectiveness trials (Trial 2; Stice, Rohde, Shaw, & Gau, 2011; Trial 3; Stice, Rohde, Butryn, Shaw, & Marti, 2015) for the present study. We excluded 5% of participants who did not report race or identified as “mixed heritage” and another 2% of the sample who reported Native American ethnicity because this cell size was too small for adequate power. The resulting 1,177 participants (*M* age = 18.6, *SD* = 4.3) were composed of 5% African American, 11% Asian American/Pacific Islander, 12% Hispanic American and 72% White Americans.

Mailings and fliers were used to recruit female high school (Trial 1 and 2) and college students (Trial 1 and 3) for health prevention groups. Informed consent was obtained from participants and parents if minors. Participants were asked to report body image concerns during a phone screen as the trials evaluated selective prevention programs for young women at risk for eating disorders. We excluded less than 2% of the participants who were initially screened out using DSM-IV criteria for meeting full-threshold anorexia nervosa and bulimia nervosa, but not individuals endorsing binge eating disorder, purging disorder, or subthreshold levels of any of these eating disorders at baseline. Further, DSM-5 lowered the thresholds for many eating disorders, meaning that some participants with subthreshold eating disorders met criteria for a threshold eating disorder per DSM-5 (Allen et al., 2013; see below). Trial 1 participants were randomized to the *Body Project* of the *Healthy Weight* eating disorder prevention programs, an expressive writing group, or an assessment-only control condition. Trial 2 and 3 participants were randomized to the *Body Project* or an educational brochure control condition. All the participants, including those randomized to the prevention programs or the control conditions, were included in this study. Participants completed surveys and interviews at baseline and at 1-, 6-, 12-, 24-, and 36-month follow-up (see Stice et al. 2008, 2011, 2015 for additional details). The appropriate Institutional Review Boards approved all the procedures and measures.

Measures

Eating pathology.—The semi-structured Eating Disorder Diagnostic Interview (EDDI: Stice et al., 2008) assessed eating disorder symptoms over the past 3-months at baseline. At each assessment, eating disorder symptoms since the previous interview were assessed on a month-by-month basis. We used DSM-5 criteria for eating disorders, as operationalized in Stice, Marti, and Rhode (2013). We focused on subthreshold anorexia nervosa, rather than atypical anorexia nervosa, because we did not assess weight history, which meant we were unable to reliably identify people who had lost a substantial amount of weight that is necessary for a diagnosis of atypical anorexia nervosa. *Overeating* included counts of eating rapidly, eating until uncomfortably full, eating large amounts when not hungry, eating alone due to embarrassment, feeling depressed or guilty after overeating, and feeling upset that you could not control overeating. *Fasting* reflected the number of times per week in the past three months that two meals in a row were skipped to prevent weight gain. *Excessive exercise* reflected the number of times per week in the past three months excessive exercise

was used to counteract the effects of overeating. EDDI eating disorder diagnoses have shown 1-week test-retest reliability (α = .79) and inter-rater agreement (α = .75) for DSM-5 eating disorders and sensitivity to prevention program effects [see Stice et al., 2008 (42% non-White participants); Stice, Butryn, et al., 2013 (42% non-White participants); Stice, Marti, et al., 2013 (32% non-White participants)]. Fasting and excessive exercise were normalized with a log transformation.

Thin-ideal internalization.—The Ideal-Body Stereotype Scale-Revised assessed thin-ideal internalization (Stice et al., 2008). It has shown internal consistency (α = .91), 2-week test-retest reliability (r = .80), predictive validity for bulimic symptom, and sensitivity to intervention effects (see Stice et al., 2008). Response options were on a 5-point scale (1 = *strongly disagree, 5 = strongly agree*) and a mean score was computed with higher scores indicating greater levels of this variable, as was the case for the remaining variables.

Body dissatisfaction.—Items from the Satisfaction and Dissatisfaction with Body Parts Scale (Berscheid, Walster, & Bohrnstedt, 1973) assessed dissatisfaction with nine body parts. It has shown internal consistency (α = .94), 3-week test-retest reliability (r = .90), predictive validity for bulimic symptoms, and sensitivity to intervention effects (Stice et al., 2008). Response options were on a 5-point scale (1 = *extremely dissatisfied*, 5 = *extremely satisfied*), but items were reversed coded before computing a mean score.

Dieting.—The Dutch Restrained Eating Scale (van Strien, Frijters, van Staveren, Defares, & Deurenberg, 1986) assesses the frequency of various dieting behaviors. It has shown internal consistency (α = .95), 2-week test-retest reliability (r = .82), convergent validity with self-reported caloric intake (but not objectively measured intake), predictive validity for bulimic symptom, and sensitivity to intervention effects (Stice, Sysko, Roberto, & Allison, 2010; Stice et al., 2008; van Strien et al., 1986). Response options were on a 5-point scale (1 = *strongly disagree*, 5 = *strongly agree*).

Healthy eating.—We used the Healthy Eating Scale we developed to assess self-reported healthy eating behaviors (sample item: *I have reduced the amount of fat in my diet*). It has shown internally consistency (α = .82), 1-month test-retest reliability (r = .63), and sensitivity to intervention effects (Stice, Presnell, Gau, & Shaw, 2007; 39% non-White participants). Response options were on a 5-point scale (1 = *strongly disagree*, 5 = *strongly agree*).

Thinness expectancy.—The Thinness Expectancy Scale (Hohlstein, Smith, & Atlas, 1998) was used to assess expected social and psychological benefits from achieving thinness. This scale has shown internal consistency (α = .86) and predictive validity for the onset of body dissatisfaction (Stice & Whitenton, 2002; 32% non-White participants). Response options were on a 5-point scale (1 = *strongly disagree*, 5 = *strongly agree*).

Denial of costs of pursuing the thin-ideal.—We created five items assessing denial of costs associated with pursuing the thin-ideal (sample item: *The risks associated with severe dieting are overrated*). It has shown internal consistency (α = .82), 1-week test-retest reliability (r = .87), and predictive validity for eating disorders (Stice, Rohde, Gau, & Shaw,

2012; 40% non-White participants). Response options were on a 5-point scale (1 = *strongly disagree,* 5 = *strongly agree*).

Functional impairment.—Impairment in the family, peer group, romantic, and school domains were measured with 17 items from the Social Adjustment Scale-Self Report for Youth (Weissman, Orvaschel, & Padian, 1980). The original scale has shown convergent validity with clinician and collateral ratings (M r = .72) and sensitivity to treatment effects (Weissman & Bothwell, 1976). The 17-item version has shown internal consistency (α = .77) and 1-week test-retest reliability (r = .83; Stice et al., 2008). Response options were on a 5-point scale (1 = *never,* 5 = *always).*

Mental health care utilization.—Use of mental health services was assessed as the frequency of mental health visits. Receipt of care in the last 6-months was coded “1” and no health care coded “0”. This item showed 1-year test-retest reliability (r = .89) and sensitivity to intervention effects [Stice, Shaw, Burton, & Wade, 2006 (42% non-White participant); Stice, Butryn, et al., 2013],

Negative affect.—Different measures of negative affect were used for each trial and were standardized with a mean of zero and a standard deviation of one. In Trial 1, negative affect was assessed with the sadness, guilt, and fear/anxiety subscales from the Positive Affect and Negative Affect Scale-Revised scale (PANAS-X; Watson & Clark, 1992). It has shown internal consistency (α = .95), 3-week test-retest reliability (r = .78), predictive validity for bulimic symptoms, and sensitivity to intervention effects (Stice et al., 2006). In Trial 2, negative affect was assessed with the Center for Epidemiologic Studies Depression Scale (CES-D; Radioff, 1977). It has shown internal consistency (α = .74 - .91), reliability (2- to 8-week test-retest r = .51 - .59), and convergent validity with clinician ratings of depressive symptoms (M r = .88; Andrews, Lewinsohn, Hops, & Roberts, 1993; Roberts, Lewinsohn, & Seeley, 1991; α = .94 at T1). For Trial 3, negative affect was assessed with the 21-item Beck Depression Inventory (Beck, Steer, & Garbin, 1988). It has shown internal consistency (α = .73-.95), 1-week test-retest reliability (r = .60-,90), convergent validity with clinician ratings of depressive symptoms, and sensitivity to intervention effects (M r = .75; Beck et al., 1988; Stice et al., 2015).

Body mass.—The BMI (kg/m2; Pietrobelli, Faith, Allison, Gallagher, Chiumello, & Heymsfield, 1998) was used as a baseline predictor of eating disorder onset, in addition to diagnosing anorexia nervosa. Height was measured to the nearest millimeter using portable stadiometers. Weight was assessed to the nearest 0.1 kg using digital scales. Height and weight were measured twice at each assessment and averaged to reduce errors. BMI has shown predictive validity with direct measures of body fat among adolescents (Mei, Grummer-Strawn, Pietrobelli, Goulding, Goran, & Dietz, 2002). Age- and sex-adjusted BMI centiles (Faith, Saelens, Wilfley, & Allison, 2001) were used to determine whether participants were underweight for anorexia nervosa.

Statistical Methods

We used chi-square tests to examine study prevalence of eating disorders and incidence of eating disorder onset over follow-up. To determine ethnic group differences for baseline risk factors, ANOVA with follow-up Tukey's tests were estimated with SAS PROC GLM (SAS Institute Inc., 2011). Each risk factor was tested in a separate model. Cohen's d, computed as the mean difference between groups divided by the pooled standard deviation, were provided as effect size indices using the convention .2, .5, and .8 for small, medium, and large.

Cox proportional hazard models estimated with SAS PHREG (SAS Institute Inc., 2011) tested which ethnic contrasts predict the onset of eating disorders. To isolate onset effects from intervention effects, dummy coded vectors reflecting condition were included as covariates and we confirmed there were no significant intervention by ethnicity interactions. Participants who met criteria for an eating disorder at baseline were excluded from the Cox models that predicted future onset of eating disorders. Threshold and subthreshold categories were combined to increase sensitivity. The Breslow (1974) method was used to handle tied event times in the hazard models. Graphs of the Kaplan-Meier estimates of the survivor function were used to evaluate the proportional hazards assumption. Hazard ratios and 95% confidence intervals are provided as effect size indices following conventions of 1.48, 2.48, and 4.28 for small, medium, and large for hazard ratios greater than 1.0; and .68, .40, and .23 for small, medium, and large for hazard ratios less than 1.0 (Lipsey & Wilson, 2001). Missing onset data were accounted for with right-censoring in the hazard models and 20 imputed data sets were used to account for missing risk factor data in ANCOVA models. Sequential regression multiple imputation (SRMI; van Burren, 2007) was used to impute data sets using the IVEware software V0.2 (Raghunathan, Solenberger & Van Hoewyk, 2002).

Before modeling, we conducted power analyses to determine which group contrasts had a power of .80 to detect medium or greater effects based on observed cell sizes and alpha set at .05. For group differences in baseline risk factors, we had sufficient power to detect medium or larger effects for all contrasts with White, Hispanic American, Asian American, and African American groups. For prediction of onset of an eating disorder, we had sufficient power to detect medium ($HR = 2.48$) or larger effects for contrasts of Whites with each ethnic group when predicting bulimia nervosa and binge eating disorder. We had sufficient power to detect large effects ($HR = 4.28$) for contrasts of Whites with each ethnic group when predicting anorexia nervosa and purging disorder. We report ethnic group differences in baseline risk factors for all pairwise contrasts and group differences in onset of eating disorders for contrasts between Whites with all other ethnic groups due to the limited sensitivity for the latter analyses. To protect against making a Type I error, a Bonferroni correction to the critical p-value was made for determining significance for group differences in baseline risk factors ($p < .0006$) and the onset of an eating disorder ($p < .004$).

Results

Preliminary Analysis

The average age at the baseline assessment was 18.6 years (SD = 4.30) and 72% reported at least one of their parents had a bachelor or advanced degree. Attrition for diagnostic data was 2% at posttest, 6% at 6-months, 7% at 1-year, 12% at 2-year, and 10% at 3-year follow-up. All participants completed the baseline assessment and 99% completed at least one follow-up (M = 5.3 of 6 assessments, SD = 0.9). The number of assessments completed did not significantly differ across ethnic groups (χ^2[15,1177] = 23.6,p = .07).

Prevalence

Table 1 presents the prevalence and incidence of full-syndrome and subthreshold eating disorders. Prevalence reflects the number of participants who met criteria at baseline or at any assessment during follow-up. Incidence reflects participants who showed onset during the follow-up period, excluding those who met criteria at baseline. Due to small cell sizes (less than five cases), ethnic groups were not compared on prevalence rates of individual disorders. However, the prevalence of any threshold or subthreshold eating disorder (19.8% White, 19.9% Hispanic American, 20.7% African American, and 21.5% Asian American) were compared and no significant differences were found (χ^2[3,1177] = 0.23,p = .97).

Baseline Risk Factors

Correlations between baseline risk factors are shown in Table 2. The average correlation among risk factors was r = .18 (range = −.02 - .54). Statistically significant ethnic differences were found for two of the 13 baseline risk factors: thin-ideal internalization and body mass index (see Table 3 and 4). First, Asian Americans had significantly higher thin-ideal internalization than African Americans (d = .75) and White Americans (d = .44). Second, African Americans had significantly greater BMI than Asian Americans (d = .65). No significant ethnic group differences were found for thinness expectancy, denial of costs of pursuing the thin-ideal, body dissatisfaction, healthy eating, dieting, negative affect, overeating, fasting, excessive exercise, functional impairment or mental health care use.

Onset of Eating Disorders

No statistically significant difference in eating disorders onset among ethnic groups was found (see Table 5). We also tested whether ethnicity moderated the relation of risk factors to the future onset of eating disorder. The main effect of each baseline risk factor was added, separately, to the onset models described above along with the interaction between baseline risk factor and ethnic group contrasts (White vs. African American, White vs. Asian American, White vs. Hispanic American) resulting in 156 tests. No significant moderating effects were found.

Discussion

The main objective of this study was to examine whether there are ethnic differences in the prevalence, risk factors, and the relation of risk factors to future onset of eating disorders. We found no significant differences in the overall prevalence of subthreshold and threshold

eating disorders across ethnic groups. We found two of the 13 eating disorders risk factors showed significant differences between ethnic groups. We found no statistically significant differences in eating disorders onset among ethnic groups. Given the limited significant differences we found, our findings overall suggest that there are more commonalities than differences contributing to the risk and development of eating disorders across ethnic groups. This supports prior studies showing more similarities than differences in eating disorders among ethnic groups (Quick & Byrd-Bredbenner, 2014; Shaw et al., 2004; Solmi et al., 2016). This overall pattern of findings may imply that some of the ethnic differences reported in earlier studies were due to chance, as many of those studies did not correct for multiple testing.

Although the present study found more similarities between ethnic groups, several significant differences were found after correcting for multiple testing. We will discuss whether these results are consistent and supported by prior findings. Our results indicated that Asian Americans had significantly higher thin-ideal internalization than White and African American females despite having significantly lower BMI compared to African Americans. Ethnic differences between Asian Americans and Whites have been somewhat mixed; an earlier metaanalysis found Asian American females reporting slightly higher dieting, weight and dieting concerns, body dissatisfaction, and eating disorder symptoms than Whites (Wildes et al., 2001). However, Grabe and Hyde (2006)’s later meta-analysis found no ethnic differences between Asian American and White females for body dissatisfaction.

Asian Americans typically weigh less than White and other ethnic minority females (Jung & Forbes, 2007; Wildes et al., 2001). Thus the level of thin-ideal internalization we found among Asian American females is noteworthy. Because cross-cultural studies have found that exposure to Western media in Asian and Pacific Islander cultures is associated with increased body image and eating disturbances (Watters, 2010), Asian Americans’ adherence to the Western appearance ideal may account for both the elevation and the similarities in risk factors and onset of eating disorders found between Asian Americans and other ethnic groups.

The present results also revealed that African Americans had significantly lower thin-idealization than Asian American females, and as previously discussed, had significantly higher BMI than Asian Americans. A lower thin-idealization is protective against eating disorders, but a higher BMI can predict greater eating disorder severity (Berner et al., 2013; Stice et al., 2008). Past studies show that African American women tend to endorse larger body sizes as ideal and to hold body standards less rooted in the thin-ideal (Flowers, Levesque, & Fischer, 2012; Gordon et al., 2010; Overstreet, Quinn, & Agocha, 2010). They also tend to rank behaviors such as dieting and watching one’s weight to be less congruent with their racial identity (Oyserman et al., 2012). Although African Americans may have cultural factors that protect them from eating disorders, our study did not show that they have a lower incidence and prevalence rates of eating disorders compared to other ethnic groups. This suggests that African Americans are at a similar risk of developing eating disorders, and an important target group for eating disorder prevention. Lastly, prevention efforts focused on combating the thin-ideal have been found to be similarly effective for

young female African Americans compared to other ethnic groups in preventing eating disorders (Rodriguez, Marchand, Ng, & Stice, 2008; Stice, Marti, & Cheng, 2014).

Surprisingly, we found no significant ethnic differences for Hispanic females. It is also noteworthy that we did not find White females to have elevated risks and onset of eating disorders compared to ethnic minorities on most of the variables we examined. Prior research has found that young Hispanic females tend to have larger ideal body size than White females (Gordon et al., 2010). But more acculturated and non-bilingual Hispanic Americans have higher anti-fat attitudes compared to those who are less assimilated to mainstream American culture (Pepper & Ruiz, 2007). These findings again imply that cultural identity may protect certain ethnic minority females from developing body dissatisfaction or eating disorders (Warren et al., 2005). Thus, it is possible that we found limited significant ethnic differences among our participants in general, and for Hispanic females in particular, because our ethnic minority participants are more acculturated and pursuing the Western standards of body ideals at a similar level as our White female participants. Qualitatively different findings might have emerged if we had studied less acculturated ethnic minority females.

Overall, these findings deviate from early notions suggesting that eating disorders are largely a White, upper-middle class, female disorder in Western countries (Striegel-Moore & Bulik, 2007). A plausible explanation for similarities in risk factors and onset of eating disorders is the widespread social-cultural pressure for thinness in Western countries. The thin-ideal is very pervasive, and various groups face pressures from the media, family members and peers to be thin (Striegel-Moore & Bulik, 2007; Shaw et al., 2004). It is also important to note that previous research was conducted more with clinical samples from treatment centers. This may have produced biased estimates of eating disorders onset because ethnic minorities are less likely to seek, receive or be recommended for treatment, reflecting a service inequity that might be rooted in economic factors, stigma differences, or ethnic stereotypes (Becker, Franko, Speck & Herzog, 2003; Cheng, 2015; Marques et al., 2011; Oysermann et al, 2012).

Limitations and Future Directions

Study limitations included having a smaller number of ethnic minority groups compared to White females. This may explain the limited ethnic differences and the potential for missing clinically significant effect sizes for these smaller groups. Our sample participants may not be representative of the larger population given the inclusion criteria of the prevention trials where we excluded participants who met criteria for eating disorders and the possible higher stigma and reluctance to admit body image concerns among ethnic minority groups. Recruiting larger and more representative samples of ethnic minorities are important for future research. Because eating disorder symptoms were self-reported retrospectively on a month-by-month basis, participants may have had a poorer recall when they reported symptoms over 12-month windows. Also, as acculturation and endorsement of Western ideals might explain the similarities across ethnic groups, a limitation is that we did not include a measure of acculturation. Future studies on ethnic differences in eating disorders risk factors and prevalence should include acculturation measures. Lastly, ethnic difference (or the lack of) does not equate to cultural differences; it is important to examine cultural

differences in eating disorders in future studies as culture can influence people's subjective experience, expression, and interpretation of mental illness.

Despite these limitations, the present study has important contributions and implications for eating disorders prevention and treatment programs. First, this study updated the current literature by using current DSM-5 criteria for eating disorders. It also had increased sensitivity due to the large sample size and use of in-person diagnostic interview. Further, this is the first study to test for ethnic differences in the relation of risk factors to future eating disorder onset. Our findings suggest that there are similarities in eating disorder risk factors and predictive effects for young women in the US.

A recent meta-analysis showed a medium effect where culturally adapted interventions perform better than unadapted versions of the same intervention ($g = .52$) although the effect size of culturally adapted versus the same unadapted intervention did not significantly differ from the effect size of culturally adapted versus control condition (Hall, Ibaraki, Huang, Marti & Stice, 2016). This result was based on only nine studies thus future research should directly test whether prevention programs are equally effective for various ethnic groups or if culturally tailoring an intervention would yield better effects than an unadapted version. The present findings suggest that it may be helpful to focus such research on Asian Americans who were a high-risk group. Given the expectation that the percent of individuals from ethnic minority groups will increase in the US (Passel & Cohn, 2008), understanding cultural differences in the etiology of eating disorders and developing culturally tailored prevention efforts are important in addressing public health issues such as eating disorders.

References

Allen KL, Byrne SM, Oddy WH, & Crosby RD (2013). DSM–IV–TR and DSM-5 eating disorders in adolescents: Prevalence, stability, and psychosocial correlates in a population-based sample of male and female adolescents. Journal of Abnormal Psychology, 122, 720. [PubMed: 24016012]

Andrews JA, Lewinsohn PM, Hops H, & Roberts RE (1993). Psychometric properties of scales for the measurement of psychological variables associated with depression in adolescence. Psychological Reports, 73, 1019–1046. [PubMed: 8302975]

Beck AT, Steer RA, & Garbin MG (1988). Psychometric properties of the Beck Depression Inventory: Twenty-five years of evaluation. Clinical Psychology Review, 8, 77–100.

Becker AE, Burwell RA, Herzog DB, Hamburg P, & Gilman SE (2002). Eating behaviours and attitudes following prolonged exposure to television among ethnic Fijian adolescent girls. The British Journal of Psychiatry, 180, 509–514. doi: 10.1192/bjp.180.6.509 [PubMed: 12042229]

Becker AE, Franko DL, Speck A, & Herzog DB (2003). Ethnicity and differential access to care for eating disorder symptoms. International Journal of Eating Disorders, 33, 205–212. [PubMed: 12616587]

Berner LA, Shaw JA, Witt AA, & Lowe MR (2013). The relation of weight suppression and body mass index to symptomatology and treatment response in anorexia nervosa. Journal of Abnormal Psychology, 122, 694–708. [PubMed: 24016010]

Berscheid E, Walster E, & Bohmstedt G (1973). The happy American body: A survey report. Psychology today, 76, 119–131.

Breslow N (1974). Covariance analysis of censored survival data. Biometrics, 30, 89–99. [PubMed: 4813387]

Cheng ZH (2015). Asian Americans and Whites' stigma levels in response to biological and social explanations of depression. Social Psychiatry and Psychiatric Epidemiology, 50, 767–776. [PubMed: 25539590]

Crow S (2005). Medical complications of eating disorders In Wonderlich S, Mitchell J, de Zwaan M, & Steiger H (Eds.), Eating disorders review, Part I (pp. 127–36). Abingdon, UK: Radcliffe Publishing Ltd.

Faith MS, Saelens BE, Wilfley DE, & Allison DB (2001). Behavioral treatment of childhood and adolescent overweight: Current status, challenges, and future directions In Thompson JK & Smolak L, (Eds.). Body image, eating disorders, and overweight in youth: Assessment, prevention, and treatment (pp. 313–340). Washington, DC: American Psychological Association

Fernandez S, & Pritchard M (2012). Relationships between self-esteem, media influence and drive for thinness. Eating Behaviors, 13, 321–325. [PubMed: 23121782]

Flowers KC, Levesque MJ, & Fischer S (2012). The relationship between maladaptive eating behaviors and racial identity among African American women in college. Journal of Black Psychology, 38, 290–312. doi: 10.1177/0095798411416459

Franko D, & Keel P (2006). Suicidality in eating disorders: Occurrence, correlates, and clinical implications. Clinical Psychology Review, 26, 769–82. [PubMed: 16875766]

Franko DL, Thompson-Brenner H, Thompson DR, Boisseau CL, Davis A, Forbush KT, … & Devlin MJ (2012). Racial/ethnic differences in adults in randomized clinical trials of binge eating disorder. Journal of Consulting and Clinical Psychology, 80, 186. [PubMed: 22201327]

Gluck ME, & Geliebter A (2002). Racial/ethnic differences in body image and eating behaviors. Eating Behaviors, 3, 143. [PubMed: 15001011]

Gordon KH, Castro Y, Sitnikov L, & Holm-Denoma JM (2010). Cultural body shape ideals and eating disorder symptoms among White, Latina, and Black college women. Cultural Diversity and Ethnic Minority Psychology, 16, 135–143. doi:10.1037/a0018671 [PubMed: 20438151]

Grabe S, & Hyde JS (2005). Ethnicity and body Dissatisfaction among women in the United States: A meta-analysis. Psychological Bulletin, 132, 622–640.

Gunewardene A, Huon GF, & Zheng R (2001). Exposure to Westernization and dieting: A cross-cultural study. International Journal of Eating Disorders, 29, 289–293. doi: 10.1002/eat.1020 [PubMed: 11262507]

Hall GCN, Ibaraki AY, Huang ER, Marti CN, & Stice E (2016). A meta-analysis of cultural adaptations of psychological interventions. Behavior Therapy, 47, 993–1014. [PubMed: 27993346]

Haines J, & Neumark-Sztainer D (2006). Prevention of obesity and eating disorders: A consideration of shared risk factors. Health Education Research, 21, 770–782. [PubMed: 16963727]

Hoek H (2016). Review of the worldwide epidemiology of eating disorders. Current Opinion in Psychiatry, 29, 336–339. doi:10.1097/YC0.0000000000000282 [PubMed: 27608181]

Hohlstein LA, Smith GT, & Atlas JG (1998). An application of expectancy theory to eating disorders: Development and validation of measures of eating and dieting expectancies. Psychological Assessment, 10, 49–58.

Hudson J, Hiripi E, Pope H, & Kessler R (2007). The prevalence and correlates of eating disorders in the National Comorbidity Survey Replication. Biological Psychiatry, 61, 348–58. doi: 10.1016/j.biopsych.2006.03.040 [PubMed: 16815322]

Jung J, & Forbes GB (2007). Body dissatisfaction and disordered eating among college women in China, South Korea, and the United States: Contrasting predictions from sociocultural and feminist theories. Psychology of Women Quarterly, 31, 381–393.

Klein D & Walsh B (2003). Eating disorders. International Review of Psychiatry, 15, 205–216. [PubMed: 15276960]

Lee-Winn A, Mendelson T, & Mojtabai R (2014). Racial/ethnic disparities in Binge Eating Disorder prevalence, symptom presentation, and help-seeking among Asian Americans and Non-Latino Whites. American Journal of Public Health, 104, 1263–1265. [PubMed: 24832409]

Lipsey MW, & Wilson DB (2001). Practical meta-analysis. Thousand Oaks, CA: Sage.

Marques L, Alegria M, Becker A, Chen C, Fang A, Chosak A, & Diniz J (2011). Comparative prevalence, correlates of impairment, and service utilization for eating disorders across US ethnic groups: Implications for reducing ethnic disparities in health care access for eating disorders. International Journal of Eating Disorders, 44, 412–420. [PubMed: 20665700]

Mehler P & Krantz M (2003). Anorexia nervosa medical issues. Journal of Women's Health, 12, 331–340.

Mei Z, Grummer-Strawn LM, Pietrobelli A, Goulding A, Goran MI, & Dietz WH (2002). Validity of body mass index compared with other body-composition screening indexes for the assessment of body fatness in children and adolescents. The American Journal of Clinical Nutrition, 75, 978–985 [PubMed: 12036802]

Mitchell JE, King WC, Pories W, Wolfe B, Flum DR, Spaniolas K, … & Engel S (2015). Binge eating disorder and medical comorbidities in bariatric surgery candidates. International Journal of Eating Disorders, 48, 471–476. [PubMed: 25778499]

Olguin P, Fuentes M, Gabler G, Guerdjikova AI, Keck PE, & McElroy SL (2017). Medical comorbidity of binge eating disorder. Eating and Weight Disorders-Studies on Anorexia, Bulimia and Obesity, 22, 13–26.

O'Neill S (2003). African American women and eating disturbances: A meta-analysis. Journal of Black Psychology, 29, 3–16.

Overstreet NM, Quinn DM, & Agocha VB (2010). Beyond thinness: The influence of a curvaceous body ideal on body dissatisfaction in Black and White women. Sex Roles, 63, 91–103. doi: 10.1007/s11199-010-9792-4

Oyserman D, Fryberg SA, & Yoder N (2007). Identity-based motivation and health. Journal of Personality and Social Psychology, 93, 1011. [PubMed: 18072851]

Passel J, & Cohn D (2008). US population projections: 2005–2050. Washington, DC: Pew Hispanic Center.

Pepper AC & Ruiz SY (2007). Acculturation's influence on antifat attitudes, body image and eating behaviors. Eating Disorders, 15, 427–447. doi:10.1080/10640260701667912 [PubMed: 17987451]

Quick V & Byrd-Bredbenner C (2014). Disordered eating, socio-cultural media influencers, body image, and psychological factors among a racially/ethnically diverse population of college women. Eating Behaviors, 15, 37–41. [PubMed: 24411747]

Radloff LS (1977). The CES-D scale: A self-report depression scale for research in the general population. Applied Psychological Measurement, 1, 385–401.

Raghunathan TE, Solenberger PW, & Van Hoewyk J (2002). IVEware: Imputation and variance estimation software. Ann Arbor, MI: Survey Methodology Program, Survey Research Center, Institute for Social Research, University of Michigan.

Roberts A, Cash T, Feingold A, & Johnson B (2006). Are Black-White differences in females' body dissatisfaction decreasing? A meta-analytic review. Journal of Consulting and Clinical Psychology, 74, 1121–1131. doi: 10.1037/0022-006X.74.6.1121 [PubMed: 17154741]

Roberts RE, Lewinsohn PM, & Seeley JR (1991). Screening for adolescent depression: A comparison of depression scales. Journal of the American Academy of Child and Adolescent Psychiatry, 305, 8–66.

Rodriguez R, Marchand E, Ng J, & Stice E (2008). Effects of a cognitive-dissonance-based eating disorder prevention program are similar for Asian American, Hispanic, and White participants. International Journal of Eating Disorders, 41, 618–625. [PubMed: 18528871]

SAS Institute Inc. (2011). Base SAS® 9.3 Procedures Guide: Statistical Procedures. Cary, NC: SAS Institute Inc.

Shaw H, Ramirez L, Trost A, Randall P, & Stice E (2004). Body image and eating disturbances across ethnic groups: More similarities than differences. Psychology of Addictive Behaviors, 18, 12–18. [PubMed: 15008681]

Solmi F, Hotopf M, Hatch., Treasure J, & Micali N (2016). Eating disorders in a multiethnic inner-city UK sample: Prevalence, comorbidity and service use. Social Psychiatry and Psychiatric Epidemiology, 51, 369–381. [PubMed: 26631229]

Stice E, Butryn ML, Rohde P, Shaw H, & Marti CN (2013). An effectiveness trial of a new enhanced dissonance eating disorder prevention program among female college students. Behaviour Research and Therapy, 51, 862–871. doi: 10.1016/j.brat.2013.10.003 [PubMed: 24189570]

Stice E, Gau JM, Rohde P, & Shaw H (2017). Risk factors that predict future onset of each DSM–5 eating disorder: Predictive specificity in high-risk adolescent females. Journal of Abnormal Psychology, 126, 38. [PubMed: 27709979]

Stice E, Marti CN, & Cheng ZH (2014). Effectiveness of a dissonance-based eating disorder prevention program for ethnic groups in two randomized controlled trials. Behaviour Research and Therapy, 55, 54–64. [PubMed: 24655465]

Stice E, Marti N, & Rohde P (2013). Prevalence, incidence, impairment, and course of the proposed DSM-5 eating disorder diagnoses in an 8-year prospective community study of young women. Journal of Abnormal Psychology, 122, 445–457. doi: 10.1037/a0030679 [PubMed: 23148784]

Stice E, Marti CN, Spoor S, Presnell K, & Shaw H (2008). Dissonance and Healthy Weight eating disorder prevention programs: Long-term effects from a randomized efficacy trial. Journal of Consulting and Clinical Psychology, 76, 329–340. [PubMed: 18377128]

Stice E, Presnell K, Gau J, & Shaw H (2007). Testing mediators of intervention effects in randomized controlled trials: An evaluation of two eating disorder prevention programs. Journal of Consulting and Clinical Psychology, 75, 20–32. [PubMed: 17295560]

Stice E, Rohde P, Butryn ML, Shaw H, & Marti CN (2015). Effectiveness trial of a selective dissonance-based eating disorder prevention program with female college students: Effects at 2- and 3-year follow-up. Behaviour Research & Therapy, 71, 20–26. doi: 10.1016/j.brat.2015.05.012 [PubMed: 26056749]

Stice E, Rohde P, Gau J, & Shaw H (2012). Effect of a dissonance-based prevention program on risk for eating disorder onset in the context of eating disorder risk factors. Prevention Science, 13, 129–139. doi: 10.1007/s11121-011-0251-4 [PubMed: 21975593]

Stice E, Rohde P, Shaw H, & Gau J (2011). An effectiveness trial of a selected dissonance-based eating disorder prevention program for female high school students: Long-term effects. Journal of Consulting and Clinical Psychology, 79, 500–508. [PubMed: 21707136]

Stice E, Shaw EL, Burton E, & Wade E (2006). Dissonance and Healthy Weight eating disorder prevention programs: A randomized efficacy trial. Journal of Consulting and Clinical Psychology, 74, 263–275. [PubMed: 16649871]

Stice E, Sysko R, Roberto CA, & Allison S (2010). Are dietary restraint scales valid measures of dietary restriction? Additional objective behavioral and biological data suggest not. Appetite, 54, 331–339. [PubMed: 20006662]

Stice E, & Whitenton K (2002). Risk factors for body dissatisfaction in adolescent girls: A longitudinal investigation. Developmental Psychology, 38, 669–678. [PubMed: 12220046]

Striegel-Moore RH & Bulik C (2007). Risk factors for eating disorders. American Psychologist, 62, 181–198. [PubMed: 17469897]

Striegel-Moore RH, Dohm F, Kraemer H, Taylor C, Daniels S, Crawford P, & Schreiber G (2003). Eating disorders in White and Black women. American Journal of Psychiatry, 160, 1326–1331. [PubMed: 12832249]

Swanson S, Scott J, Crow M, Le Grange D, Swendsen J, Merikangas K (2011). Prevalence and correlates of eating disorders in adolescents. Archives of General Psychiatry, 68, 714–723. [PubMed: 21383252]

Udo T, & Grilo CM (2018). Prevalence and Correlates of DSM-5–Defined Eating Disorders in a Nationally Representative Sample of US Adults. Biological Psychiatry, 84, 345–354. [PubMed: 29859631]

van Burren S (2007). Multiple imputation of discrete and continuous data by fully conditional specification. Statistical Methods in Medical Research, 16, 219–242. [PubMed: 17621469]

van Strien T, Frijters JR, van Staveren WA, Defares PB, & Deurenberg P (1986). The predictive validity of the Dutch Restrained Eating Scale. International Journal of Eating Disorders, 5, 747–755.

Wade TD, Bergin JL, Tiggemann M, Bulik CM, & Fairburn CG (2006). Prevalence and long-term course of lifetime eating disorders in an adult Australian twin cohort. Australian and New Zealand Journal of Psychiatry, 40, 121–128. [PubMed: 16476129]

Warren C, Gleaves D, Cepeda-Benito A, Fernandez M, & Rodriguez R (2005). Ethnicity as a protective factor against internalization of a thin ideal and body dissatisfaction. International Journal of Eating Disorders, 37, 241–249. doi: 10.1002/eat.20102 [PubMed: 15822090]

Watson D, & Clark LA (1992). On traits and temperament: General and specific factors of emotional experience and their relation to the Five-Factor Model. Journal of Personality, 60, 441–476. [PubMed: 1635050]

Watters E (2010). Crazy like us: The globalization of the American psyche. New York, NY: Simon and Schuster.

Weissman MM, Orvaschel H, & Padian N (1980). Children's symptom and social functioning self-report scales. Comparison of mothers' and children's reports. The Journal of Nervous and Mental Disease, 168, 736–740. [PubMed: 7452212]

Weissman MM, & Bothwell S (1976). Assessment of social adjustment by patient selfreport. Archives of General Psychiatry, 33, 1111–1115. [PubMed: 962494]

Wildes J, Emery R, & Simons A (2001). The roles of ethnicity and culture in the development of eating disturbance and body dissatisfaction: A meta-analytic review. Clinical Psychology Review, 2, 521–551.

Wildes J & Forbush K (2015). Ethnicity as a risk factor for eating disorders In Levine M & Smolak L (Eds.), The Wiley-Blackwell handbook of eating disorders (pp. 324–333). Oxford, UK: Wiley.

Highlights

- No ethnic differences were found in the prevalence of eating disorders (EDs)
- We found ethnic differences for 2 of 13 ED risk factors
- No ethnic differences in the relation of risk factors for future ED onset
- We overall found more shared ED risk factors and rates than ethnic differences

Table 1

Rates of DSM-5 Eating Disorders

	White (*n*=848)		Hispanic American (*n*=141)		Asian American (*n*=130)		African American (*n*=58)	
	n	**%**	**n**	**%**	**n**	**%**	**n**	**%**
Study Prevalence								
Anorexia nervosa	6	0.7	2	1.4	1	0.8	0	0.0
Subthreshold anorexia nervosa	13	1.5	4	2.8	5	3.8	1	1.7
Bulimia nervosa	50	5.9	11	7.8	8	6.2	2	3.4
Subthreshold bulimia nervosa	76	9.0	11	7.8	12	9.2	8	13.8
Binge eating disorder	46	5.4	5	3.5	10	7.7	4	6.9
Subthreshold binge eating disorder	50	5.9	6	4.3	10	7.7	0	0.0
Purging disorder	31	3.7	12	8.5	2	1.5	2	3.4
Cumulative 3-year Incidence								
Anorexia nervosa	4	0.5	2	1.4	1	0.8	0	0.0
Subthreshold anorexia nervosa	11	1.3	2	1.4	5	3.8	1	1.7
Bulimia nervosa	31	3.7	6	4.3	6	4.6	0	0.0
Subthreshold bulimia nervosa	58	6.8	9	6.4	9	6.9	5	8.6
Binge eating disorder	33	3.9	5	3.5	6	4.6	3	5.2
Subthreshold binge eating disorder	44	5.2	6	4.3	6	4.6	0	0.0
Purging disorder	25	2.9	9	6.4	1	0.8	1	1.7

Note. Eating disorder classifications are not mutually exclusive across disorders. Study prevalence reflects the number of participants who met criteria at baseline and those who showed onset during the follow-up period. Cumulative incidence reflects participants who showed onset during the follow-up period, excluding those who met criteria at baseline.

Table 2

Pearson Correlations for Baseline Risk Factors

		1	2	3	4	5	6	7	8	9	10	11	12	13
1.	Thin ideal internalization	1.00												
2.	Thinness expectancy	.31	1.00											
3.	Denial costs	.15	.42	1.00										
4.	Body dissatisfaction	.26	.48	.23	1.00									
5.	Healthy eating	.08	.13	.07	.09	1.00								
6.	Dieting	.30	.36	.29	.37	.46	1.00							
7.	Negative affect	.24	.33	.25	.42	.07	.30	1.00						
8.	Overeating	.17	.22	.13	.26	.08	.28	.27	1.00					
9.	Fasting	.11	.19	.24	.18	.07	.26	.20	.13	1.00				
10.	Excessive exercise	.11	.15	.19	.17	.12	.29	.29	.13	.27	1.00			
11.	Functional impairment	.07	.21	.15	.30	−.01	.15	.54	.24	.19	.11	1.00		
12.	Mental health care use	.03	.13	.08	.16	.08	.10	.27	.16	.10	−.02	.19	1.00	
13.	Body Mass Index	−.02	.21	.03	.36	.10	.12	.07	.04	.01	.01	.01	.04	1.00
	Mean	3.70	3.24	2.26	3.40	2.88	2.66	0.00	1.01	1.03	1.08	2.32	0.23	24.0
	SD	0.52	0.81	0.71	0.79	0.83	0.90	1.00	2.01	3.52	3.25	0.53	0.42	4.98

Notes. All correlations, means, and standard deviations are averaged across 20 imputed data sets. Non-log transformed values are reported for fasting and excessive exercise.

Table 3

Baseline Risk Factors by Racial/Ethnic Groups

		White		Hispanic American		Asian American		African American	
		Mean	**SD**	**Mean**	**SD**	**Mean**	**SD**	**Mean**	**SD**
1.	Thin ideal internalization	3.67	0.50	3.77	0.59	3.89	0.47	3.50	0.62
2.	Thinness expectancy	3.23	0.77	3.31	0.82	3.22	0.79	2.99	0.89
3.	Denial costs	2.23	0.70	2.38	0.75	2.31	0.63	2.31	0.74
4.	Body dissatisfaction	3.39	0.77	3.55	0.88	3.42	0.74	3.15	0.88
5.	Healthy eating	2.91	0.83	2.78	0.83	2.86	0.78	2.70	0.88
6.	Dieting	2.63	0.90	2.73	0.93	2.74	0.86	2.67	0.89
7.	Negative affect	−0.06	0.95	0.19	1.15	−0.03	0.97	0.01	1.20
8.	Overeating	0.96	1.99	1.19	2.11	1.18	2.13	0.93	1.68
9.	Fasting	1.02	3.60	1.28	3.93	0.48	1.78	1.41	3.24
10.	Excessive exercise	1.08	3.42	1.04	3.07	1.12	2.70	1.26	2.58
11.	Functional impairment	2.33	0.54	2.40	0.50	2.25	0.48	2.29	0.60
12.	Mental health care use	0.25	0.43	0.15	0.36	0.16	0.37	0.22	0.42
13.	Body Mass Index	23.81	4.62	24.83	5.93	22.50	4.14	25.74	6.54

Notes. Means and standard deviations are averaged across 20 imputed datasets. Non-log transformed values are reported for fasting and excessive exercise.

Table 4

Effect Size Estimates (d) for the Mean Comparisons between Ethnic Groups

	W vs. HA	W vs. AA	W vs. AF	HA vs. AA	HA vs. AF	AA vs. AF
1. Thin ideal internalization	−0.19	**−0.44**	0.33	−0.22	0.45	**0.75**
2. Thinness expectancy	−0.10	0.01	0.31	0.11	0.38	0.28
3. Denial costs	−0.21	−0.12	−0.11	0.10	0.09	0.00
4. Body dissatisfaction	−0.20	−0.04	0.31	0.16	0.45	0.34
5. Healthy eating	0.16	0.06	0.25	−0.10	0.09	0.20
6. Dieting	−0.11	−0.12	−0.04	−0.01	0.07	0.08
7. Negative affect	−0.25	−0.03	−0.07	0.21	0.15	−0.04
8. Overeating	−0.11	−0.11	0.02	0.00	0.13	0.12
9. Fasting	−0.14	0.18	−0.20	0.32	−0.06	−0.41
10. Excessive exercise	−0.03	−0.10	−0.16	−0.07	−0.13	−0.06
11. Functional impairment	−0.13	0.15	0.07	0.31	0.21	−0.08
12. Mental health care use	0.24	0.21	0.07	−0.03	−0.19	−0.16
13. Body Mass Index	−0.21	0.29	−0.41	0.45	−0.15	**−0.65**

Notes. Bolded effect size comparisons are significantly different at a Bonferroni adjusted $p < .0006$ based on analysis of variance models with post-hoc Tukey tests. W= White; HA= Hispanic American; AA= Asian American; AF= African American

Table 5

Results from Cox Regression Models Predicting Onset of Future Eating Disorder.

	Wald χ^2	*p*-value	*HR*	[CI_{95}]
Anorexia nervosa				
White vs. Hispanic American	0.94	.332	0.57	[0.19-1.77]
White vs. Asian American	2.86	.091	0.43	[0.16-1.14]
White vs. African American	<0.01	.971	0.96	[0.13-7.41]
Bulimia nervosa				
White vs. Hispanic American	0.01	.911	0.96	[0.45-2.04]
White vs. Asian American	0.07	.786	1.11	[0.52-2.37]
White vs. African American	0.05	.820	1.15	[0.47-1.70]
Binge eating disorder				
White vs. Hispanic American	0.03	.863	1.30	[0.07-24.81]
White vs. Asian American	0.97	.324	1.54	[0.66-3.61]
White vs. African American	0.15	.696	1.26	[0.39-4.08]
Purging disorder				
White vs. Hispanic American	4.19	.041	0.45	[0.21-0.97]
White vs. Asian American	2.51	.113	5.04	[0.68-37.35]
White vs. African American	0.51	.477	2.07	[0.28-15.43]

Notes. For each comparison, White is the reference group. HR = hazard ratio, CI = confidence interval.