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## Reproductive and Appetite Hormones and Bulimic Symptoms During Midlife

Jessica H. Baker, PhD<sup>1,\*</sup>, Claire M. Peterson, PhD<sup>2</sup>, Laura M. Thornton, PhD<sup>1</sup>, Kimberly A. Brownley, PhD<sup>1</sup>, Cynthia M. Bulik, PhD<sup>1,3,4</sup>, Susan S. Girdler, PhD<sup>1</sup>, Marsha D. Marcus, PhD<sup>5</sup>, and Joyce T. Bromberger, PhD<sup>5</sup>

<sup>1</sup>Department of Psychiatry, University of North Carolina

<sup>2</sup>Division of Behavioral Medicine and Clinical Psychology, Cincinnati Children's Hospital Medical Center

<sup>3</sup>Department of Nutrition, University of North Carolina

<sup>4</sup>Department of Medical Epidemiology and Biostatistics, Karolinska Institute, Stockholm, Sweden

<sup>5</sup>Department of Psychiatry, University of Pittsburgh

### Abstract

Eating disorders and related symptoms occur during midlife; however, little is known about their etiology. It has been hypothesized that perimenopause represents a window of vulnerability for the development or exacerbation of eating disorder symptomatology because, like puberty, perimenopause is a period of reproductive hormone change. We compared symptoms of bulimia nervosa (bulimic symptomatology) assessed via mean scores on a self-report questionnaire in premenopausal and perimenopausal women. We also examined the association between hormone concentrations (reproductive/appetite) and bulimic symptomatology. No mean differences in bulimic symptomatology were observed between premenopause and perimenopause. However, there was a significant positive association between leptin and binge eating. Although no significant associations between reproductive hormones and bulimic symptomatology were observed, additional research is needed to provide definitive information. It is essential to learn more about the etiology of eating disorders and related symptomatology across the lifespan in order to develop age-relevant treatment and prevention programs.

### Keywords

bulimia; eating disorders; hormones; midlife; perimenopause; menopause transition; bulimia nervosa

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Bulimic symptomatology can occur at all ages, including midlife (Elran-Barak et al., 2015; Gagne et al., 2012; Mangweth-Matzek, Hoek, Rupp, Kemmler, Pope, et al., 2013). One study reported that 13.3% of a sample of women ages 50 and older endorsed at least one

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\*Correspondence to: Dr. Baker, Department of Psychiatry, University of North Carolina at Chapel Hill, 101 Manning Drive, CB #7160, Chapel Hill, NC 27599, jhbaker@med.unc.edu, Phone: 984-974-3794, Fax: 919-843-3950.

eating disorder symptom (Gagne, et al., 2012). For example, binge eating was reported to occur in 3% to 11% of women at midlife whereas the prevalence of purging was reported between 8% to 13.4% (Gagne, et al., 2012; Marcus, Bromberger, Wei, Brown, & Kravitz, 2007). Women in midlife, like those during adolescence and adulthood, also report significant body dissatisfaction, weight concerns, and dissatisfaction with their overall appearance. (Gagne, et al., 2012; Marcus, et al., 2007; Runfola et al., 2013). Because reproductive hormone changes during puberty play a role in the risk for bulimic symptoms in adolescent girls (Klump, 2013), it is reasonable to postulate the reproductive hormone changes that occur during perimenopause may also increase risk (Baker & Runfola, 2016). However, this area of inquiry remains largely unexplored.

Of the reproductive hormones, estrogen appears to have the most significant role in risk for bulimic symptoms (Baker, Girdler, & Bulik, 2012). In women with bulimia nervosa (BN), binge eating and purging are exacerbated during the mid-luteal and pre-menstrual phases of the menstrual cycle when estrogen levels are low (Edler, Lipson, & Keel, 2007; Lester, Keel, & Lipson, 2003). In healthy women, decreased levels of estrogen during the menstrual cycle are associated with emotional eating (i.e., eating in response to negative emotions), weight preoccupation, and body image dissatisfaction (Carr-Nangle, Johnson, Bergeron, & Nangle, 1994; Hildebrandt et al., 2014; Jappe & Gardner, 2009; Klump, Keel, Culbert, & Edler, 2008). Taken together with the role estrogen has in risk for bulimic symptoms during adolescence (Klump, 2013), the associations between estrogen levels and eating disorder symptomatology during the menstrual cycle in women with and without BN suggest that periods of hormonal fluctuation may be a vulnerable time for risk and exacerbation of bulimic symptomatology. Thus, it is expected that the unpredictable hormonal fluctuations occurring during perimenopause may also increase vulnerability for the development of bulimic symptomatology during midlife (Baker & Runfola, 2016).

To date, one study has indirectly examined the association between hormone fluctuation and eating disorder symptomatology at midlife by comparing the prevalence of combined eating disorder diagnoses in midlife women who were pre-, peri-, and post-menopause. Perimenopause involves inconsistent and rapidly fluctuating levels of estrogen, whereas post-menopause marks the complete cessation of menstruation and consistently low levels of estrogen (Santoro & Randolph, 2011). Mangweth-Matzek and colleagues (2013) found that perimenopausal women reported a significantly higher prevalence of eating disorders, determined via a self-report questionnaire, compared with premenopausal women (9% versus 2%) (Mangweth-Matzek, Hoek, Rupp, Kemmler, Pope Jr, et al., 2013). Although this study was limited by having small numbers of women with an eating disorder, midlife may be a potential time of risk for eating disorders and this risk may be associated with the hormonal fluctuations that occur with perimenopause.

Research has also shown an association between appetite hormones and eating disorders, specifically binge-eating disorder (BED) and BN. For example, adiponectin and leptin are appetite-regulatory hormones that can act as an appetite-suppressant that show associations with both such that adiponectin is decreased in adults with BED and BN, albeit associations with BN are somewhat inconsistent (Khalil & El Hachem, 2014). On the other hand, leptin is increased in adults with BED and BN (Adami, Campostano, Cella, & Scopinaro, 2002;

Monteleone, Di Lieto, Tortorella, Longobardi, & Maj, 2000). Further, in regard to leptin specifically, estrogen appears to play a role in its functionality such that estrogen increases sensitivity to the appetite suppressant effects of leptin (Asarian & Geary, 2006). Therefore, the association between reproductive hormones and binge eating-related eating disorders may be mediated via appetite hormones.

With inpatient eating disorder admissions for women ages 35 and over on the rise (Wiseman, Sunday, Klapper, Harris, & Halmi, 2001), understanding the risk and protective factors for eating disorder symptomatology in this population is of growing clinical concern. To date, the associations between estrogen and appetite hormone concentrations and eating disorder symptomatology, specifically bulimic symptoms, during midlife have not been examined. Therefore, the goals of this study were two-fold: 1) compare bulimic symptomatology in premenopausal and perimenopausal women, hypothesizing that, similar to previous research, perimenopausal women will exhibit increased bulimic symptomatology compared with premenopausal women; and 2) explore, for the first time to our knowledge, the association between reproductive (estrogen, testosterone, follicle-stimulating hormone) and appetite hormones (leptin, adiponectin) and bulimic symptomatology in women in midlife.

## Material and Methods

### Participants

Participants included a subsample of women from the multisite Study of Women's Health Across the Nation (SWAN), who participated in an ancillary mental health study (MHS), in which psychiatric information was collected. Detailed information about SWAN can be found elsewhere (Gold et al., 2000). In brief, women were eligible for the study if they were 42–52 years old, were pre- or early peri-menopausal, not using exogenous hormones, had an intact uterus, and self-identified with one of the site's designated racial/ethnic groups.

The SWAN MHS is an ancillary study of the larger parent SWAN study and included women at the Pittsburgh, Chicago, and New Jersey SWAN sites who were able to participate in a psychiatric interview within 9 months of the baseline assessment. The current sample includes only women from the Pittsburgh ( $n = 366$ ) and Chicago ( $n = 20$ ) sites who were assessed at the first annual visit and completed an eating behavior questionnaire. There were not sufficient resources at the Chicago and New Jersey sites to continue conducting psychiatric interviews after baseline on any but a small number of participants. Previous comparisons among the sites indicated the sites were similar on demographic, psychosocial, perceived health variables, and current depressive symptoms (Marcus, Bromberger, Wei, Brown, & Kravitz, 2007).

As part of the MHS, participants completed a self-report questionnaire on bulimic symptomatology during the first or second annual visit. This questionnaire was only completed by the SWAN MHS participants. We included only those women who completed the measure at their first annual visit ( $n = 386$ ). No significant differences on baseline measures of education, income, or body mass index (BMI) were observed between those women who completed the questionnaire and those who did not (Marcus, et al., 2007). However, at Pittsburgh, African American women were less likely to complete the

questionnaire than were Caucasian women, and, in Chicago, women who completed the questionnaire were less likely to report difficulty paying for basic necessities (Marcus, et al., 2007).

The current sample also includes a subset of SWAN MHS participants who had appetite hormone data available from another ancillary study. This subset of individuals were chosen based on depression status. Specifically, participants having a lifetime history of major depression on the Structured Clinical Interview for DSM-IV Disorders (SCID; (First, Spitzer, Gibbon, & Williams, 1997) were selected as cases and matched on age and ethnicity with women who had no history of major depression. Therefore, only a subset of the MHS sample (65%,  $n = 227$ ) were included in this separate research protocol and have appetite hormone data available.

Informed consent was obtained from all study participants. SWAN site Institutional Review Boards approved all study procedures and protocols. The University of North Carolina Institutional Review Board approved this study.

## Measures

**Bulimic Symptomatology**—The Bulimia Test-Revised (BULIT-R) was used to examine bulimic symptomatology (Thelen, Farmer, Wonderlich, & Smith, 1991). The BULIT-R is a 28-item self-report questionnaire that focuses on symptoms of BN in clinical and nonclinical populations. Items are scored on a 1–5 scale with higher scores reflecting increased symptomatology. For analyses, we used the BULIT-R total score, which is a summation of all items. In an earlier analysis, two additional subscales were created in order to capture specific BN symptoms including binge eating and body dissatisfaction (Marcus, et al., 2007).<sup>1</sup> Each subscale represents a summation of all items reflecting behaviors and characteristics related to binge eating or body dissatisfaction, respectively. The internal reliability of the total score ( $\alpha = 0.93$ ) and binge eating ( $\alpha = 0.91$ ) and body dissatisfaction subscales ( $\alpha = 0.83$ ) were good. All BULIT-R scales were log-transformed prior to analysis to account for a positive skew.

**Menopause status**—Menopause status was based on menstrual bleeding patterns during the previous 12 months. At study entry, women were either premenopause or early perimenopause due to eligibility requirements. At the first annual visit women were still either premenopausal or perimenopausal; none were postmenopausal. Premenopause was defined as having a menstrual period in the past 3 months with no change in regularity in the past 12 months. Perimenopause was defined as having a menstrual period in the past 3 months with change in regularity over the past 12 months or having no menstrual period within the last 3 months but some menstrual bleeding in the previous 12 months. For the current study, participants reporting surgical menopause or hormone use were excluded.

**Reproductive hormones**—Blood draws were performed following an overnight fast during days 2–5 of the follicular phase of the menstrual cycle at the first annual visit. Blood

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<sup>1</sup>The binge eating subscale included the following questions from the BULIT-R: items 1, 2, 3, 7, 8, 9, 11, 14, 15, 17, 18, 19, 24, 25, 26, and 27. The body dissatisfaction subscale included the following questions: items 4, 6, 10, 12, 20, 23, and 28.

was refrigerated prior to centrifugation 1–2 hours after the draw, and the serum was aliquoted, frozen, and batched for shipment to a central laboratory. Samples were catalogued and assayed immediately upon arrival. All assays were performed on the ACS-180 automated analyzer (Bayer Diagnostics Corporation, Tarrytown, NY) utilizing a double-antibody chemiluminescent immunoassay with a solid phase anti-IgG immunoglobulin conjugated to paramagnetic particles, anti-ligand antibody, and competitive ligand labeled with dimethylacridinium ester (DMAE).

The estrogen (E2) assay modifies the rabbit anti-E2-6 ACS-180 immunoassay to increase sensitivity, with a LLD of 1.0 pg/mL. The testosterone (T) assay modifies the rabbit polyclonal anti-T ACS-180 immunoassay, with a LLD of 2.19 ng/dL. The follicle-stimulating hormone (FSH) assay is a modification of a manual assay kit (Bayer Diagnostics) utilizing two monoclonal antibodies directed to different regions on the beta subunit, with a lower limit of detection (LLD) of 1.05 mIU/mL. Duplicate E2 assays were conducted with results reported as the arithmetic mean for each subject, with a coefficient of variation of 3–12%. All other assays were single determinations.

**Appetite hormones**—At the first annual visit, the SWAN blood collection protocol included a fasting blood draw for a specimen repository that is maintained at  $-80^{\circ}\text{C}$  until processing. Serum leptin and adiponectin were determined spectrophotometrically using commercially available immunoassay kits and run according to manufacturer's instructions. For leptin, the coefficient of variation for duplicate samples was 3.7%, and the lower limit of detection was 0.5 ng/ml. For adiponectin, the coefficient of variation was 5.4%, and the lower limit of detection was 0.78 ng/ml. As described above, both assays were completed as part of a separate research protocol at the Pittsburgh and Chicago SWAN sites and thus appetite hormone data were only available in a subset of the MHS sample. All reproductive and appetite hormone values were log-transformed prior to analysis.

**Covariates**—Age, race/ethnicity, education, and income were obtained during baseline visits. Additional covariates (i.e., BMI, history of major depression, physical activity, self-reported overall health, income, and smoking status) were obtained during the first annual visit when the BULIT-R was completed. Education was categorized as no high school degree, completed high school or GED, some college, college graduate, or more than a college degree. For income, participants were provided with income ranges (<\$10,000; \$10,000–\$19,999; \$20,000–\$34,999; \$35,000–\$49,999; \$50,000–\$74,999; \$75,000–\$99,999; \$100,000–\$149,999;  $\geq$ \$150,000) and asked to identify their appropriate range.

At each visit, height and weight, obtained in street clothes without shoes, were measured and BMI (weight [kg]/height [m<sup>2</sup>]) was calculated. A current or past history of major depression was assessed with the SCID. To examine physical activity, participants were asked whether, compared with others, their physical activity level was: 1) much less; 2) somewhat less; 3) about the same; 4) somewhat more; or 5) much more. At each visit, participants were also asked to self-report their overall health (as poor, fair, good, very good, or excellent) and whether they regularly smoked cigarettes.

## Statistical Analyses

Initially, we examined the association between the three BULIT-R scales and the aforementioned covariates of interest. Variables which had a significant association with the BULIT-R total score, binge-eating subscale, or body dissatisfaction subscale were included in respective analyses. However, the MHS assessment site (i.e., Pittsburgh or Chicago), race/ethnicity, and history of major depression (due to the oversampling of women with a history of major depression) were included as covariates in all analyses. All analyses were completed with the log transformed BULIT-R and reproductive and appetite hormone values.

Next, we completed an ANOVA, both with and without relevant covariates, to examine mean score differences in binge eating, body dissatisfaction, and the BULIT-R total score based on menopause status (pre- vs. peri-menopause) in the full sample, with premenopause women as the reference group. BULIT-R scales were included as the dependent variable and menopause status as the independent variable.

General linear models were used to evaluate the associations between bulimic symptomatology and the hormonal variables of interest. Models were conducted with the BULIT-R subscales as dependent variables and E2, T, FSH, leptin, and adiponectin as independent variables. We also included the following interactions: E2\*leptin and leptin\*adiponectin. Because the selection of participants for appetite hormone assay were based on case or control status for a history of major depression, modeling was completed in steps as we first wanted to document that the association between bulimic symptomatology and reproductive hormones was similar in the full sample and the subset of women chosen for appetite hormone assays.

First, associations were examined between bulimic symptomatology and reproductive hormones in the *full sample* ( $N = 386$ ), excluding appetite hormones as independent variables (Model 1). This allowed us to assess the association between the BULIT-R subscales and reproductive hormones in the total sample. Second, associations were examined between bulimic symptomatology and reproductive hormones in the *reduced sample* of women with appetite hormone data ( $n = 227$ ), again, excluding appetite hormones as independent variables (Model 2). This allowed us to assess the association between the BULIT-R subscales and reproductive hormones in the subset of women chosen for appetite hormone assay based on depression status—providing information as to whether there are similar associations between reproductive hormones and bulimic symptomatology in both the full and reduced samples. Last, we completed our full model of interest (Model 3), which included leptin and adiponectin as independent variables (in the reduced sample of women with appetite hormone data) and interactions of interest. Nonsignificant interactions were removed from modeling to ensure appropriate interpretation of main effects. All analyses were completed using SAS 9.2 (SAS Institute Inc, 2004) and  $p < .05$  was considered significant.



## Results

### Demographics

Sample characteristics are provided in Table 1. Thirty-two percent of the sample were African American ( $n = 125$ ), and the mean age of participants at baseline was 45 years old. At the first annual visit, 29% ( $n = 100$ ) of women were premenopausal and the mean BMI was 29.2 kg/m<sup>2</sup>. According to World Health Organization BMI classification, approximately 39% ( $n = 150$ ) of women were obese, 31.4% ( $n = 121$ ) overweight, 29% ( $n = 111$ ) in the healthy range, and 1% ( $n = 4$ ) of women were underweight. Finally, 35% ( $n = 137$ ) of the sample reported a lifetime history of major depressive disorder on the SCID. Initial analyses indicated that BMI and self-reported overall health status were significantly associated with the BULIT-R total score and the binge eating and body dissatisfaction subscales. Therefore, these covariates were included in the respective follow-up models.

No significant differences were observed between premenopausal and perimenopausal women on any of the BULIT-R subscales in the ANOVA models including and excluding relevant covariates—results were similar in both analyses. In the model excluding covariates we were able to calculate the sole effect of menopause status on bulimic symptomatology (Table 2). As can be seen, effect sizes indicated a small effect of menopause status for the BULIT-R scales.

No significant associations between E2, T, or FSH and the BULIT-R scales in either the full sample or the reduced sample were observed (Table 3; interactions were not significant and removed from the model) suggesting similar null associations in both samples. BMI was the only variable significantly associated with the BULIT-R total score, binge eating, and body dissatisfaction subscales in both models. The results were similar in the final model (model 3) including the reduced sample and including leptin and adiponectin as independent variables. However, leptin scores were significantly associated with binge eating and E2 trended towards significance with body dissatisfaction (Table 4; interactions were not significant and removed from the model).

### Discussion

Our aims were twofold: to further explore the reported association between perimenopause status and eating disorder symptomatology, specifically focusing on bulimic symptoms, and to examine, for the first time to our knowledge, the direct association between reproductive hormones, appetite hormones, and bulimic symptomatology during midlife. Contrary to our hypothesis and a previous report (Mangweth-Matzek, Hoek, Rupp, Kemmler, Pope Jr, et al., 2013), we found no significant differences in bulimic symptomatology between pre- and peri-menopausal women. Two reasons may account for differing findings. First, the two studies used differing methods to assess eating disorder symptoms. The aforementioned study focused on combined eating disorder diagnoses assessed through a self-report questionnaire whereas the current report focused on a well-validated continuous measure of bulimic symptomatology. The difference in assessment measures used could have played a role in our null findings. However, although the previous study observed significant differences in self-reported eating disorder diagnoses between pre- and peri-menopause,

that study did not find significant differences in eating disorder dimensions assessed by the Eating Disorder Inventory (Mangweth-Matzek, Hoek, Rupp, Kemmler, Pope Jr, et al., 2013). Second, the BULIT-R focuses on symptomatology most relevant for BN, a diagnosis which is less common in midlife women compared with BED or eating disorder not otherwise specified (Elran-Barak, et al., 2015; Mangweth-Matzek, Hoek, Rupp, Kemmler, Pope Jr, et al., 2013). Thus, although speculative, the differences observed in eating disorder symptomatology between pre- and peri- menopause may only be present when these symptoms reach a threshold level and/or pertain to eating disorder diagnoses only characterized by binge eating (e.g., BED). This would align with the young adult findings that show binge eating has the strongest association with estrogen fluctuation (Baker, et al., 2012).

In regression modeling, we observed similar associations in model 1 (the full sample) and model 2 (the reduced sample enriched with lifetime major depression cases and matched controls) between all independent variables, suggesting that the associations (and lack thereof) were the same in both samples. In both models, BMI was the only variable significantly associated with bulimic symptomatology. In model 3, which included all participants and all variables of interest, BMI remained significantly associated with all BULIT-R scales. However, additional patterns emerged.

Consistent with prior studies in women with BED (Monteleone, et al., 2000) but contrary to studies in young adults with BN (Bluher & Mantzoros, 2004), we observed a positive association between binge eating and leptin concentrations. Reasons for the disparate set of findings with BN may relate to several factors, including the higher prevalence of BED among midlife women (Elran-Barak, et al., 2015; Mangweth-Matzek, Hoek, Rupp, Kemmler, Pope, et al., 2013); leptin may increase over time in response to binge eating behaviors; the positive association between obesity and leptin; (Scarpace & Zhang, 2007) or increasing leptin levels across pre-, peri-, and post- menopause (Sowers et al., 2008).

A positive trend was also observed between E2 and body dissatisfaction. This is in line with Mangweth-Matzek and colleagues (2013), who observed increased feelings of fatness in perimenopausal women compared with premenopausal women. However, we did not observe increased body dissatisfaction during perimenopause. Regardless, it is conceivable that as E2 concentrations fluctuate and change during midlife, these changes may impact body fat accumulation and waist circumference (Davis et al., 2012; Wildman et al., 2012), which in turn may impact body dissatisfaction—regardless of menopause stage. Further investigation is warranted to examine this notion. Until then, our trend-level finding must be interpreted with caution.

Although our findings do not provide evidence for a strong, *direct* association among reproductive hormones, appetite hormones, and bulimic symptomatology during midlife, results must be considered preliminary and within the context of study limitations. First, our sample was somewhat small and not a true community sample. A portion of the sample was enriched with individuals selected based on lifetime major depression status. Thus, our findings may not be representative of women in midlife as a whole. Second, we did not assess current eating disorder diagnosis. As noted previously, perimenopausal women may



have a significantly higher prevalence of threshold eating disorders than premenopausal women, but because we focused on symptoms versus diagnosis this significant difference may not have been observed. Diagnostic information would have allowed us to reproduce the study by Mangweth-Matzek and colleagues (2013) and permitted the evaluation of a direct association between estrogen and eating disorder diagnosis. Finally, we only obtained one measurement of estrogen. In the young adult literature, studies have obtained multiple measurements of estrogen, exploring the impact of *changing* estrogen levels on eating disorder symptomatology. We may have observed significant associations if we were able to examine within-person change in estrogen levels as has been observed in young adult samples across the menstrual cycle.

Given the strong and consistent evidence of the role of puberty and reproductive hormones in the vulnerability for bulimic symptoms during adolescence and young adulthood, further research examining the role of hormones in the etiology of bulimic symptoms at midlife is warranted. Puberty and perimenopause may represent complementary periods of risk for bulimic symptomatology. Pre-puberty and menopause bookmark a women's reproductive cycle as hypoestrogenic states (i.e., estrogen deficiency), and puberty and perimenopause are the transitions out of or into these states, respectively. Moreover, not only are hormonal changes occurring at both stages, but both often have additional significant psychological (e.g., increased risk for depression), social (e.g., life transitions), and physical (change in adiposity, weight gain) changes. Given the reported increase of eating disorders, particularly binge eating-related disorders, in women in midlife and in the number of midlife women seeking treatment for these eating disorders, it is critical that we learn more about the etiology and risk for eating disorders and related-symptoms during this developmental stage.

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**Table 1**

Sample Characteristics for Continuous Variables at First Annual Visit

	Mean (SD)	Median	Lower Quartile	Upper Quartile	Range	N <sup>a</sup>
Age (at baseline)	45.70 (2.53)	45	44	48	42.00–52.00	386
BMI	29.24 (6.75)	28.10	24.20	33.33	17.60–55.77	384
BULIT-total score	44.05 (15.00)	39	34.00	48.00	18.00–107.00	386
BULIT-BE	21.60 (8.90)	22.00	20.00	28.00	14.00–64.00	386
BULIT-BD	9.00 (3.62)	10.00	8.00	14.00	5.00–25.00	386
E2	75.00 (77.70)	53.10	32.00	85.00	3.50–126.00	380
T	39.70 (19.50)	36.00	25.20	50.00	3.50–126.00	380
FSH	30.00 (32.14)	18.30	12.20	35.40	1.70–230.90	380
Leptin	23.72 (18.40)	18.70	12.00	31.01	0.08–117.67	227
Adiponectin	15.02 (7.50)	14.00	10.20	19.00	3.60–47.92	227

<sup>a</sup>Total available sample size at first annual visit = 386.

Abbreviations. BMI=body mass index; BULIT-total score=The Bulimia Test total score; BULIT-BE=The Bulimia Test binge eating; BULIT-BD=The Bulimia Test body dissatisfaction; E2=estrogen; T=testosterone; FSH=follicle stimulating hormone. Means and standard deviations derived from non-log transformed scores.

ANOVA Results for Log-Transformed BULIT-R Subscale Scores by Menopause Status from Full Sample with Premenopause as the Reference Group (without covariates)

**Table 2**

	N	F-value (p-value)	Premenopause Mean (SD)	Perimenopause Mean (SD)	Cohen's d
BULIT-total score	345	0.54 (.50)	42.81 (12.40)	44.50 (15.03)	0.12
BULIT-BE	344	0.56 (.46)	25.30 (8.62)	26.24 (10.20)	0.10
BULIT-BD	345	0.50 (.50)	11.34 (4.00)	11.80 (5.00)	0.10

Abbreviations. BULIT-R=The Bulimia Test Revised; BULIT-total score =The Bulimia Test total score; BULIT-BE=The Bulimia Test binge eating; BULIT-BD=The Bulimia Test body dissatisfaction. Means and standard deviations derived from non-log transformed scores.

**Table 3**

**BULIT-R General Linear Model Results of Association between BULIT-R and Reproductive Hormones**

<b>General Linear Model Results (F-value, p-value) from Full Sample (Model 1)</b>									
N	BMI	MDD history	Race	Site	Overall Health	E2	T	FSH	
BULIT-total score	376	8.10 (0.01)	2.01 (0.20)	0.25 (0.70)	0.55 (0.50)	2.10 (0.10)	1.50 (0.30)	2.61 (0.11)	0.10 (0.90)
BULIT-BE	375	8.50 (0.01)	3.00 (0.10)	0.30 (0.60)	0.50 (0.05)	2.30 (0.06)	0.45 (0.52)	2.00 (0.80)	0.05 (0.85)
BULIT-BD	376	10.13 (0.01)	0.36 (0.60)	2.01 (0.20)	0.30 (0.60)	1.00 (0.45)	1.70 (0.20)	2.60 (0.20)	0.55 (0.50)

  

<b>General Linear Model Results (F-value, p-value) from Reduced Sample<sup>a</sup> (Model 2)</b>									
BULIT-total score	222	6.00 (.03)	3.00 (.11)	0.55 (.50)	0.01 (.95)	1.20 (.40)	3.00 (.10)	0.80 (.40)	0.90 (.40)
BULIT-BE	221	5.20 (.03)	2.00 (.30)	0.22 (.65)	0.03 (.90)	1.40 (.25)	0.90 (.40)	0.70 (.42)	0.20 (.70)
BULIT-BD	222	8.51 (.01)	0.14 (.80)	3.00 (.10)	0.01 (.95)	0.40 (.90)	4.10 (.05)	1.00 (.40)	1.40 (.30)

Abbreviations. BULIT-R=The Bulimia Test BULIT-Revised; BMI=body mass index; MDD=major depressive disorder; BULIT-total score=The Bulimia Test total score; BULIT-BE=The Bulimia Test binge eating; BULIT-BD=The Bulimia Test body dissatisfaction; E2=estrogen; T=testosterone; FSH=follicle stimulating hormone. BULIT-R and reproductive hormone values log-transformed.

<sup>a</sup>the reduced sample includes only those participants that have appetite hormone information.



BULIT-R General Linear Model Results (F-value, p-value) from Full Model (Model 3) with Main Effects Only

Table 4

	N	BMI	MDD history	Site	Overall Health	Race	E2	T	FSH	Leptin	Adiponectin
BULIT-total score	222	6.00 (.02)	2.30 (.14)	0.01 (.96)	1.03 (.40)	0.60 (.44)	3.00 (.10)	1.00 (.34)	1.00 (.35)	3.00 (.10)	0.01 (.97)
BULIT-BE	221	6.00 (.02)	4.20 (.05)	0.01 (.95)	1.10 (.40)	0.32 (.60)	1.00 (.40)	0.80 (.40)	0.23 (.70)	5.00 (.03)	0.20 (.70)
BULIT-BD	222	8.42 (.01)	0.11 (.75)	0.02 (.95)	0.40 (.90)	3.00 (.10)	4.00 (.05)	1.00 (.35)	1.42 (.25)	0.13 (.80)	0.10 (.80)

Abbreviations. BULIT-R=The Bulimia Test Revised; BMI=body mass index; MDD=major depressive disorder; BULIT-total score=The Bulimia Test total score; BULIT-BE=The Bulimia Test binge eating; BULIT-BD=The Bulimia Test body dissatisfaction; E2=estrogen; T=testosterone; FSH=follicle stimulating hormone. BULIT-R and reproductive and appetite hormone values log-transformed.