Appetite Regulating Hormones Cortisol and Peptide YY are Associated with Disordered Eating Psychopathology Independent of Body Mass Index

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Objective: Disordered eating occurs in women at both weight extremes of anorexia nervosa and obesity. Cortisol, peptide YY (PYY), leptin and ghrelin are hormones involved in appetite and feeding behavior that vary with weight and body fat. Abnormal levels of these hormones have been reported in women with anorexia nervosa, functional hypothalamic amenorrhea, and obesity. The relationship between appetite regulating hormones and disordered eating psychopathology is unknown. We therefore studied the relationship between orexigenic and anorexigenic hormones and disordered eating psychopathology in women across a range of weights.

Design: Cross-sectional study of 65 women, 18-45 yrs: 16 with anorexia nervosa, 12 normal-weight with hypothalamic amenorrhea, 17 overweight or obese, and 20 normal-weight in good health.

Methods: Two validated measures of disordered eating psychopathology, the Eating Disorders Examination-Questionnaire (EDE-Q) and Eating Disorders Inventory-2 (EDI-2) were administered. Fasting PYY, leptin, and ghrelin levels were measured; cortisol levels were pooled from serum samples obtained every 20 minutes from 8pm to 8am.

Results: Cortisol and PYY levels were positively associated with disordered eating psychopathology including restraint, eating concerns, and body image disturbance, independent of body mass index (BMI). Although leptin levels were negatively associated with disordered eating psychopathology, these relationships were not significant after controlling for BMI. Ghrelin levels were generally not associated with EDE-Q or EDI-2 scores.
**Conclusions:** Higher levels of cortisol and PYY are associated with disordered eating psychopathology independent of BMI in women across the weight spectrum, suggesting that abnormalities in appetite regulation may be associated with specific eating disorder pathologies.
Introduction

Both anorexigenic and orexigenic pathways are linked to body weight in humans. Cortisol, peptide YY (PYY), leptin and ghrelin have been implicated in the regulation of appetite and feeding behavior. Levels of these hormones differ based on body weight and body fat (1-5). Women with anorexia nervosa, a psychiatric disorder associated with low weight, self-induced starvation and amenorrhea, are known to have dysregulation of appetite mediating hormones (6-9). Overweight and obese women also have abnormal levels of appetite regulating hormones compared to normal-weight women (1, 2, 10), and despite extreme differences in weight, may also experience abnormalities in eating behaviors and weight-related cognitions (11-13). In addition, some women with anorexia nervosa (AN) have a history of obesity (14) and women with bulimia may be obese (15). It is not known whether appetite regulating hormones influence disordered eating psychopathology. Although some abnormalities in levels of appetite regulating hormones may represent expected adaptive responses to energy imbalance, others may be paradoxical. Further, several of these hormonal differences persist in those with AN despite weight recovery (7, 16). A separate line of research has demonstrated altered levels of appetite regulating hormones and increased disordered eating pathology in women with functional hypothalamic amenorrhea (17-20), suggesting that these abnormalities may exist independent of low or high weight. Taken together, these findings raise the question of whether dysregulation of hormones involved in appetite may underlie the development or maintenance of disordered eating psychopathology. To address this question, we investigated the association of four appetite regulating hormones - cortisol, PYY, leptin, and ghrelin - with measures of disordered eating psychopathology in underweight women with anorexia nervosa, normal-weight women with hypothalamic amenorrhea, overweight or obese women, and normal-weight healthy women.
We hypothesized that appetite regulating hormones would be associated with disordered eating attitudes and behaviors in women across the weight spectrum, independent of body mass index (BMI), using two well-validated measures (21, 22).

**Subjects and Methods**

*Subjects*

We studied 65 women ages 18-45: 16 with anorexia nervosa (AN), 12 normal-weight with hypothalamic amenorrhea (HA), 17 overweight or obese (OB), and 20 normal-weight in good health (HC). All subjects were recruited from the community through advertisements and referrals from healthcare providers. Patient characteristics, Hamilton Depression Rating Scale (HAM-D) (23) scores, fasting leptin levels, and pooled 12-hour overnight cortisol levels were previously reported in AN, HA and HC (24). Those who completed self-assessment measures of disordered eating psychopathology and an additional group of OB subjects are included in this analysis. EDEQ and EDI-2 scores and their relationship to hormone levels have not been previously reported in these subjects.

Subjects with AN met DSM-IV criteria, including intense fear of gaining weight, body image disturbance, weight less than 85% of ideal body weight (IBW) as determined by the 1983 Metropolitan Life tables (25), and amenorrhea for at least three consecutive months.

HA were 90 to 110% of IBW with a BMI less than 25 kg/m² and reported amenorrhea for at least three consecutive months. Exclusion criteria included polycystic ovarian syndrome, hyperprolactinemia, premature ovarian failure, and history of anorexia nervosa.
HC were at least 90% of IBW with a BMI less than 25 kg/m\(^2\) and reported regular menstrual cycles. HC reported no significant medical problems or history of amenorrhea, disordered eating, or significant anxiety or depression.

OB had a BMI of 25 to 40 kg/m\(^2\) and reported regular menstrual periods. OB reported no significant medical problems or history of amenorrhea, disordered eating, or significant anxiety or depression.

All subjects were required to have a normal TSH or free T4 to participate in the study. Exclusion criteria included diabetes mellitus, active abuse of drugs or alcohol, use of medications known to affect cortisol levels within three months (including estrogen), use of depot medroxyprogesterone within six months, and pregnancy or breastfeeding within six months of the study.

Methods

This study was approved by the Institutional Review Board of Partners Health Care, Inc. Written informed consent was obtained from all subjects prior to any procedures. All subjects were admitted to the General Clinical Research Center of Massachusetts General Hospital for an outpatient screening visit and an inpatient overnight visit.

At the screening visit, height, weight and elbow breadth were measured by research bionutritionists, blood was drawn for screening laboratory tests, and a comprehensive history and
physical exam were performed. Exercise patterns and alcohol intake were assessed. Percent ideal body weight was calculated as described above. BMI was obtained by dividing the weight in kilograms by the square of height in meters. Frame size was determined by comparing elbow breadth to race-specific norms derived from the US Health and Nutritional Examination Survey-I (25).

During the inpatient overnight visit, % IBW and BMI were reevaluated. Body composition was assessed by dual-energy X-ray absorptiometry (Hologic 4500, Hologic, Inc., Waltham, MA). This technique has a precision of 3% for fat mass (26). Nutritional intake was assessed by the 24-hour diet recall. Medical history and physical exam were performed. The EDE-Q and the EDI-2 were self-administered. Study staff administered the Hamilton Rating Scale for Depression (HAM-D) (23). An intravenous catheter was placed and subjects were allowed to acclimate to their rooms for at least two hours, followed by frequent sampling of blood every twenty minutes from 8pm to 8am. Blood draws were performed by experienced research nurses on the Clinical Research Center with identical procedures between patients and controls. Subjects were asked to fast starting at 8pm, and were allowed to sleep through the night. Fasting leptin, ghrelin, and PYY levels were obtained at 7:45 am. Overnight serum samples were pooled for average cortisol levels. HC and OB presented for their overnight visits during the follicular phase of the menstrual cycle.

**Psychological Measures**

The EDE-Q is a well-validated 36-item self-report measure assessing the severity of characteristic eating disorder psychopathology in four categories: dietary restraint, eating
concern, shape concern, and weight concern. A global score can be calculated. Behaviors such as binge eating, purging, and other compensatory activities such as excessive exercise are also evaluated (27). Normative data (mean±standard deviation) based on two studies of adult women (28, 29) are as follows: dietary restraint, 1.3±1.4 (28), 1.62±1.54 (29); eating concern, 0.76±1.06, 1.11±1.11; weight concern, 1.79±1.51, 1.97±1.56; shape concern, 2.23±1.65, 2.27±1.54; and global concern, 1.52±1.25, 1.74±1.30. The EDI-2 is a well-validated self-report measure with subscale scores assessing core eating disorder psychopathology (drive for thinness, bulimia and body dissatisfaction) and related psychological features (ineffectiveness, perfectionism, interpersonal distrust, interoceptive awareness, maturity fears, asceticism, impulse regulation and social insecurity) (22). Normative data (mean±standard error of measurement) are based on the EDI Manual norms (22), which were determined using a sample of female college students, and are as follows: drive for thinness, 5.5±2.2; bulimia, 1.2±0.9; body dissatisfaction, 12.2±3.0; ineffectiveness, 2.3±1.6; perfectionism, 6.2±2.5; interpersonal distrust, 2.0±1.3; interoceptive awareness, 3.0±2.1; maturity fears, 2.7±1.3; asceticism, 3.4±1.6; impulse regulation, 2.3±1.6; and social insecurity, 3.3±1.5. The HAM-D is a 17-item semi-structured interview that is widely used to measure depressive symptoms in clinical trials and has excellent internal consistency (23). On all of the psychological measures, higher scores indicate increased symptomatology.

Biochemical Analysis

Serum and plasma samples were stored at -80° C until analysis. Serum cortisol was measured by chemiluminescent microparticle immunoassay (Architect System, Abbot Diagnostics, Abbott Park, IL). The intra-assay coefficient of variation (CV) ranged from 2.1% to 4.8% and the total CV ranged from 3.9% to 7.7%. The sensitivity was 0.8 mcg/dL. Serum leptin levels were
measured using a radioimmunoassay (RIA) kit from LINCO Research, a division of Millipore Inc. (St. Charles, MO). The intra-assay CV ranged from 3.4% to 8.3% and the inter-assay CV ranged from 3.6% to 6.3%. The sensitivity was 0.5 ng/mL. Plasma ghrelin levels were measured using an RIA kit from LINCO research, a division of Millipore Inc. The intra-assay CV ranged from 10.0% to 14.4% and the inter-assay CV ranged from 14.7% to 16.7%. The sensitivity was 93 pg/mL. Serum total PYY levels were measured using an RIA kit from LINCO research, a division of Millipore Inc. The intra-assay CV ranged from 1.5% to 2.7% and the inter-assay CV ranged from 6.1% to 6.9%. The sensitivity was 1.4 pg/mL.

**Data Analysis**

JMP Statistical Discoveries (version 5.01; SAS Institute, Inc., Cary, NC) was used for statistical analyses. Clinical characteristics, hormone levels, and measures of disordered eating attitudes and behavior were compared using overall Analysis of Variance; variables that were significantly different were then compared by Fisher’s Least Significant Difference Test. Multiple comparisons were controlled for using Tukey-Kramer. Linear regression analyses were used to investigate the associations between hormone levels and measures of disordered eating psychopathology. Multivariate least-square analyses were constructed to control for BMI and other potential confounders. Statistical significance was defined as a two-tailed p-value < 0.05. Data are reported as mean ± S.E.M.

**Results**

*Subject Characteristics*
Subject characteristics are presented in Table 1. The groups did not differ in age. As per study design, BMI, %IBW, and %fat were lowest in AN, intermediate in HA and HC, and highest in OB. There were no differences between groups in reported 24 hour caloric intake, total fat, carbohydrate or protein content. Age of menarche did not differ between groups. By definition, HC and OB had no history of amenorrhea. The total months of amenorrhea was not significantly different in AN compared to HA. Mean level of exercise was higher in AN compared to OB and HC.

*Hormone Levels*

Hormone levels are presented in Table 1. Mean cortisol levels were elevated in AN compared to OB and HC. Mean fasting PYY levels were highest in AN, and were associated with overnight cortisol levels (r=0.37, p=0.003). This relationship was no longer statistically significant after controlling for BMI (p=0.07). Mean leptin levels were higher and mean ghrelin levels were lower in OB than all other groups.

*Measures of Disordered Eating Attitudes and Behavior*

EDE-Q and EDI-2 scores are presented in Table 2 and Figures 1 and 2. As expected, the AN group demonstrated increased eating disorder psychopathology relative to the other groups indexed by EDE-Q dietary restraint, eating concern, shape concern, and weight concern scores, and EDI-2 drive for thinness scores. In contrast, EDE-Q scores and EDI-2 core eating disorder pathology scores were within the normal ranges for women in the other three groups. Notably, however, there was a relative increase in EDE-Q dietary restraint scores in HA, and in EDI-2 body dissatisfaction in OB. Associated eating disorder behaviors measured by the EDE-Q,
including binging and purging, were normal in all groups. Global EDE-Q scores were lowest in HC, intermediate in OB and HA, and highest in AN.

With regard to the associated psychological features measured by the EDI-2, women with AN generally reported the highest scores while the scores for the other groups were within the normal range.

**Relationship Between Hormone Levels and Disordered Eating Attitudes and Behaviors**

The relationships between hormone levels and measures of disordered eating attitudes and behaviors are presented in Table 3. In terms of core disordered eating psychopathology, 12-hour overnight average cortisol levels were positively associated with all EDE-Q scores and drive for thinness on the EDI-2. The relationships between overnight cortisol levels and all EDEQ scales were independent of BMI and PYY levels. In addition, the associations between overnight cortisol levels and EDEQ scores on dietary restraint and eating concern remained significant after controlling for depressive symptoms, as assessed by the HAM-D. PYY levels were also associated with core eating disorder pathology measured by the EDE-Q and EDI-2; positive associations with EDE-Q eating concern and EDI-2 drive for thinness were independent of BMI. The relationship between PYY and EDI-2 drive for thinness remained significant after controlling for cortisol levels. The relationships between the hormones cortisol and PYY and core eating disorder psychopathology as assessed by the EDE-Q and EDI-2 that were independent of BMI were still present if the overweight subjects (n=7) were excluded from the analysis. While leptin levels were inversely associated with level of eating concern scores on the
EDE-Q, this relationship was not significant after controlling for BMI. Ghrelin levels were not associated with measures of core eating disorder pathology.

With regard to the relationship between hormone levels and the disordered eating related psychological features measured by the EDI-2, both cortisol and PYY were positively associated with several of the scale scores, while leptin levels were negatively associated with these features. After controlling for BMI and PYY, positive relationships between cortisol and interpersonal distrust remained. In addition, associations between PYY and impulse regulation and social insecurity were independent of BMI and cortisol levels. Correlations between leptin levels and these features were non-significant after controlling for BMI. Ghrelin levels were associated with scores on the maturity fears subscale of the EDI-2. However, this association was no longer significant after controlling for BMI.

**Discussion**

In this study of underweight amenorrheic women with AN, normal-weight women with hypothalamic amenorrhea, overweight or obese women, and normal-weight healthy women, overnight serum cortisol and fasting PYY levels were associated with disordered eating psychopathology independent of body mass index.

Levels of hormones involved in appetite and feeding behavior, including cortisol, PYY, leptin, and ghrelin, differ based on body weight and fat (1-5), and abnormal levels of these hormones have been reported in women with anorexia nervosa (7-9, 30) and in functional hypothalamic amenorrhea (17, 19, 20, 31), a disorder commonly associated with subclinical disordered eating
(18-20). It is unknown whether appetite regulating hormone abnormalities contribute to disordered eating symptomatology. We hypothesized that these hormones would be associated with severity of disordered eating attitudes and behaviors regardless of body weight.

Cortisol, a key component of the hypothalamic-pituitary-adrenal axis, is released in response to psychological or physical “stress” and may stimulate appetite (3). Anorexia nervosa and hypothalamic amenorrhea are both associated with elevated levels of endogenous cortisol, which is presumably stress-related (17, 32-35). An alternative possibility is that hypercortisolemia is an adaptive response to increase and mobilize energy stores in the face of decreased weight or body fat and/or increased demand. Exposure to high levels of cortisol, for example in Cushing’s syndrome or exogenous corticosteroid administration, stimulates appetite and weight gain with central fat accumulation when there is sufficient substrate. For example, when patients with AN gain weight, preferential truncal fat gain is seen (36). In contrast, characteristic symptoms of adrenal insufficiency are anorexia and weight loss. Other studies have reported an association between measures of cortisol and dietary restraint in pre- and post-menopausal women (37-39). We have shown that in comparison to healthy controls, average overnight cortisol levels were higher in women with hypothalamic amenorrhea and AN (24). In this study, across groups of women of differing weights, pooled overnight serum cortisol levels were positively associated with disordered eating attitudes and behaviors as assessed by the EDE-Q and EDI-2, independent of BMI. Furthermore, the relationships between overnight cortisol levels and dietary restraint and eating concern measures of EDE-Q core disordered eating psychopathology were independent of depressive symptoms. Therefore, hypercortisolemia may be an adaptive mechanism to starvation which further exacerbates an underlying pattern of disordered eating.
PYY is an anorexigenic polypeptide hormone secreted by the intestine in response to food intake (40). In the hypothalamus, PYY modulates appetite by binding to Y2 receptors in the arcuate nucleus, promoting anorexigenic in favor of orexigenic pathways (41). PYY levels are elevated in AN (7, 42) and HA (31), and low in obesity (1), and are inversely associated with BMI (1). In a 2003 study of 12 obese and 12 lean subjects published in the New England Journal of Medicine, Batterham et al. reported that infusion of PYY resulted in a similar reduction of appetite and caloric intake in each group (1). A study of exercising women with HA and exercising and sedentary healthy controls found that PYY levels were associated with drive for thinness as assessed by the EDI-2 (31). We demonstrated that PYY levels are correlated with characteristics of disordered eating as well as associated features, and that some of these relationships including core disordered eating psychopathology measures of EDE-Q eating concern and EDI-2 drive for thinness, remained significant after controlling for BMI. Although abnormal PYY levels may be secondary to disordered eating thinking and behavior, low levels of PYY in obesity and high levels in AN despite PYY’s function in signaling satiety argue for a possible etiologic role.

There is some evidence suggesting that PYY may be involved in hypothalamic-pituitary-adrenal axis signaling. Administration of intravenous PYY to dogs stimulates secretion of ACTH and cortisol (43, 44). Consistent with these data, we found a strong positive correlation between morning fasting PYY levels and mean overnight cortisol levels. PYY and cortisol were independently associated with different features of disordered eating as measured by the EDE-Q and EDI-2, suggesting specific effects of these hormones on disordered eating pathology.
Leptin, an anorexigenic hormone secreted by adipocytes, correlates with BMI and fat mass (5, 10). Levels are low in AN (8, 45) and HA (19, 46), and high in obesity (10). After long-term recovery from AN, leptin levels normalize (47). A recent study demonstrated an inverse association between leptin levels and drive for thinness on the EDI-2 in women with AN, but not in healthy controls (48). Monteleone et al. found no relationship between leptin levels and EDI scores in 21 women with anorexia nervosa, 32 with bulimia nervosa, 14 with binge eating disorder, and 25 controls (5). In contrast, Ehrlich et al. showed a negative correlation between leptin and drive for thinness on the EDI-2 when controlling for BMI in 57 women with AN, but not in controls (48). In our study, leptin was inversely associated with scores on the eating concern subscale of the EDE-Q and with several associated psychological features measured by the EDI-2 in the entire sample. However, these relationships were no longer significant after controlling for BMI. This may reflect leptin’s role as a marker for fat, where women with the lowest body fat (AN) have increased eating disorder pathology, rather than as a mediator of disordered eating thought and behavior.

Ghrelin, an orexigenic hormone secreted by the stomach, is elevated in AN (7, 30) and HA (20, 31), low in obesity (2), and inversely related to BMI (49). Schneider et al. reported an association between ghrelin levels and elevated scores on the Eating Attitude Test, a measure of abnormal eating thinking and behavior, in normal-weight women with hypothalamic amenorrhea (50). A correlation between ghrelin and drive for thinness subscale of the EDI-2 has been reported in a study of exercising women with hypothalamic amenorrhea and controls (31). Consistent with a
previous report (47), we found that ghrelin was associated only with the maturity fears subscale of the EDI-2, however, this relationship was no longer significant after controlling for BMI.

This study has several limitations. First, sample sizes across groups were small, which may have limited power to detect differences. Due to the cross-sectional study design, causality cannot be determined. In addition, we measured total but not acylated ghrelin levels. Acylated ghrelin is considered the most active form of ghrelin, but due to rapid degradation requires special collection techniques with a preservative for accurate assessment (56). It is possible that had we measured the active form of ghrelin, we would have identified associations with disordered eating psychopathology. Further, the EDE-Q and EDI-2 are excellent measures of disordered eating psychopathology in AN. Additional instruments may have been useful to tap into subclinical disordered eating psychopathology in HA and OB. For example, nuanced measures that more carefully assess the nature of exercise (e.g., motivation, compulsivity, intensity) may be useful in hypothalamic amenorrhea, or those that assess the psychopathology of binge eating disorder (e.g., measures of emotional eating) may be useful for individuals with overweight and obesity.

In summary, we found a strong association between overnight cortisol and fasting morning PYY levels and disordered eating attitudes and behaviors, independent of body mass index. These findings suggest that these appetite regulating hormones may be factors in disordered eating symptomatology. Further research on the role of appetite regulating hormones in the development of disordered eating will be important.
Declaration of Interest: The authors have no conflict of interest to declare.

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Figure Legends

**Figure 1. EDE-Q results.** AN (black) scored higher on the EDE-Q than HA (diagonal lines), OB (grey), and HC (white). A, p<0.0005 vs. HA; B, p<0.002 vs. OB; C, p<0.0001 vs. HC. Overall Analysis of Variance followed by Fisher’s Least Significant Difference Test, means±S.E.M.

**Figure 2. EDI-2 results.** AN (black) scored higher than the other groups (HA, diagonal lines; OB, grey; HC, white) on most subscales. A, p<0.05 vs. HA; B, p<0.004 vs. OB; C, p<0.02 vs. HC. Overall Analysis of Variance followed by Fisher’s Least Significant Difference Test, means±S.E.M.
Figure 1

- Dietary Restraint
- Eating Concern
- Shape Concern
- Weight Concern
- Global Concern

Legend:
- A, B, C
- B, C
- C
Figure 2

Core Psychopathology

Associated Psychological Features
### Table 1. Subject characteristics and hormone levels

<table>
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<tr>
<th></th>
<th>AN</th>
<th>HA</th>
<th>OB</th>
<th>HC</th>
<th>P-values</th>
<th>Overall</th>
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<tr>
<td></td>
<td>N=16</td>
<td>N=12</td>
<td>N=19</td>
<td>N=20</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age (years)</td>
<td>25.9±1.5</td>
<td>27.3±1.8</td>
<td>29.5±2.0</td>
<td>27.2±1.6</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>Body mass index (kg/m^2)</td>
<td>18.2±0.3</td>
<td>20.9±0.4</td>
<td>31.3±1.0</td>
<td>22.3±0.3</td>
<td>&lt;0.0001*</td>
<td>&lt;0.0001*</td>
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<tr>
<td>% Ideal body weight</td>
<td>79.2±1.4</td>
<td>92.9±1.4</td>
<td>132.3±4.3</td>
<td>98.0±1.4</td>
<td>&lt;0.0001*</td>
<td>&lt;0.0001*</td>
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<tr>
<td>% Fat</td>
<td>17.6±0.9</td>
<td>23.6±0.8</td>
<td>37.1±1.2</td>
<td>27.0±1.1</td>
<td>&lt;0.0001*</td>
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<tr>
<td>Caloric intake (kcal)</td>
<td>1808±149</td>
<td>1664±87</td>
<td>1713±176</td>
<td>1679±132</td>
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<td>NS</td>
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<td>Age of menarche</td>
<td>13.1±0.3</td>
<td>12.9±0.3</td>
<td>12.2±0.4</td>
<td>12.9±0.3</td>
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<td>NS</td>
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<tr>
<td>Months amenorrhea</td>
<td>62.9±16.4</td>
<td>34.9±9.6</td>
<td>0</td>
<td>0</td>
<td>NS</td>
<td>0.0004</td>
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<td>Hours exercise/week</td>
<td>8.1±1.5</td>
<td>6.0±0.9</td>
<td>2.8±0.5</td>
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<td>No. alcoholic drinks/week</td>
<td>1.6±0.5</td>
<td>2.9±0.6</td>
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<td>1.7±0.5</td>
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<tr>
<td>12-hr pooled cortisol (mcg/dl)</td>
<td>11.6±0.9</td>
<td>9.4±0.9</td>
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<td>7.6±0.4</td>
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<td>PYY (pg/ml)</td>
<td>96.6±6.8</td>
<td>75.3±4.6</td>
<td>66.4±2.7</td>
<td>78.0±6.0</td>
<td>0.02</td>
<td>0.0002*</td>
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<tr>
<td>Leptin (ng/ml)</td>
<td>2.8±0.5</td>
<td>6.3±0.8</td>
<td>23.6±2.9</td>
<td>8.7±0.7</td>
<td>0.0006</td>
<td>&lt;0.0001*</td>
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<tr>
<td>Ghrelin (pg/ml)</td>
<td>1121±100</td>
<td>1004±88</td>
<td>543±47</td>
<td>1049±108</td>
<td>NS</td>
<td>&lt;0.0001*</td>
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</table>

Overall Analysis of Variance followed by Fisher’s Least Significant Difference Test, means±S.E.M.

*p<0.05 after correcting for multiple comparisons using Tukey-Kramer.

NS, not significant.
### Table 2. Measures of disordered eating attitudes and behaviors

<table>
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<tr>
<th></th>
<th>AN</th>
<th>HA</th>
<th>OB</th>
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<th>AN v. HC</th>
<th>HA v. OB</th>
<th>HA v. HC</th>
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<td><strong>Core psychopathology</strong></td>
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<td>NS</td>
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<td>0.02*</td>
<td>0.09</td>
<td>NS</td>
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Overall Analysis of Variance followed by Fisher’s Least Significant Difference Test, means±S.E.M.

*p<0.05 after correcting for multiple comparisons using Tukey-Kramer

NS, not significant.
Table 3. Correlations between hormone levels and measures of disordered eating attitudes and behaviors

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<th>EDE-Q</th>
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<td>Overexercise/month</td>
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<td>Drive for thinness</td>
<td>0.27*</td>
<td>0.35**a</td>
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<td>0.15</td>
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Linear regression, multivariate least-square analyses
p<0.10, *p<0.05, **p<0.01, *p<0.05 after controlling for BMI