Cortisol, Hunger, and Desire to Binge Eat Following a Cold Stress Test in Obese Women With Binge Eating Disorder

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Objective: Increased basal cortisol levels have been found in bulimia nervosa. After stress, increased cortisol levels have been associated with increased food intake in healthy women. Therefore, we assessed cortisol, hunger, and desire to binge eat after a cold pressor test (CPT) among women with binge eating disorder (BED). **Methods:** Twenty-two obese (body mass index [BMI] = 36.7 ± 6.5 SD) females (11 non-BED, 11 BED) completed the Zung depression scale and underwent the CPT, hand submerged in ice water for 2 minutes. Over 60 minutes, periodic ratings of hunger and desire to binge eat were obtained, just before blood draws for cortisol, as well as insulin. On a separate day, participants had a 1-mg oral dexamethasone suppression test (DST). **Results:** The BED group had higher depression scores than the non-BED ($p = .04$), but depression was not a significant covariate for the cortisol response or to DST. After controlling for contraceptive use ($n = 3$), the BED group had higher basal cortisol than the non-BED group ($p = .03$), but cortisol did not differ after DST ($p = .40$). The BED group had nearly significant greater cortisol AUC after the CPT ($p = .057$) after controlling for insulin AUC and contraceptive use ($p = .057$). The BED group also had greater AUC for hunger ($p = .03$) and desire to binge eat ($p = .02$) after the CPT. **Conclusion:** These findings support our hypothesis of a hyperactive HPA-axis in BED, which may contribute to greater hunger and binge eating. **Key words:** stress responsivity, central fat, eating disorder, pain, obesity, DST.

AN = anorexia nervosa; **AUC** = area under the curve; **BED** = binge eating disorder; **BMI** = body mass index; **BN** = bulimia nervosa; **CPT** = cold pressor test; **DST** = dexamethasone suppression test; **HPA** = hypothalamic pituitary adrenal; **VAS** = visual analogue scale; **WHR** = waist-hip ratio.

INTRODUCTION

Obesity is a multidimensional disease that is increasing in prevalence, both in the United States and worldwide (1). A subset of people who are obese have binge eating disorder (BED) (2), characterized by frequent and regular intake of large amounts of food on at least 2 days/wk for 6 months, and an associated sense of loss of control over eating (3). People with BED do not engage in compensatory behaviors, unlike bulimia nervosa (BN; (4). Individuals with BED comprise about 2% of the nonpatient general community (5) and a much larger proportion, about 30% (18% to 46%) of obese people presenting for weight loss treatment. They have greater associated psychopathology, do less well in weight loss treatment, and relapse more quickly than obese non-BED (6). Given the morbidities associated with obesity and the prevalence of BED, it is important to understand the pathophysiology of this disorder.

Stress and negative affect are the most frequently cited precursors of binge eating (7). However, negative emotions have been associated with both increased and decreased food intake (8,9). Stress reactivity may distinguish stress overeaters from stress undereaters (10). Understanding predictors of stress-induced eating would therefore be useful to help reduce relapse of both obesity (11) and binge eating (12).

Cortisol secretion is a major component of the stress response (13) and could play a role in binge eating, given that exogenous glucocorticoids induce obesity by increasing food intake (14). Cortisol has been implicated as a potential mediator for increased energy intake in healthy males (14) and females (15), but this association has not been studied in obese women with BED. Koo-Loeb and colleagues (13) reported higher 24-hour urinary cortisol in women with BN after an interpersonal speech task. An exaggerated cortisol response after laboratory stress tests has been observed in women with anorexia nervosa (AN) (16), BN (13,17), and obesity (18), although Pirke et al. (19) found a blunted cortisol response. Epel and colleagues (15) recently noted that among lean women, high cortisol reactors ate significantly more food after a stress task. Stress plays a confirmed role in the onset and maintenance of BN (10) and in the initiation of binge eating episodes (12, 20). Furthermore, there is evidence that eating-disordered individuals perceive certain events as more stressful than controls (10, 21). Because cortisol is released during stress and can increase hunger and feeding behavior (14), endogenous cortisol release stimulated by stress may mediate stress-induced eating.

Excessive stimulation of the hypothalamic-pituitary-adrenal (HPA) axis may result in hypercortisolemia and diminished sensitivity to HPA negative feedback, reflected in reduced suppression of cortisol after a dexamethasone suppression test (DST). Oral administration of dexamethasone, which mimics cortisol, normally suppresses the morning cortisol rise in individuals with intact negative feedback inhibition (7). Typically, a 1-mg dose is given at 11:00 p.m., and blood is drawn the following morning at 8:00. Failure to respond to dexamethasone by conventional criteria is indicated by morning cortisol >5 μg/dl (22). Evidence of cortisol nonsuppression has been reported in AN (23,24) and BN (25), even without coexisting depression (26). Furthermore, studies in AN, BN, and the night eating syndrome (27,28) have found

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increased basal cortisol compared with controls (19). These studies (25,29) suggest that in eating-disordered individuals, hypothalamic and pituitary centers controlling adrenal activity have diminished sensitivity to negative feedback (9).

Generally, depression is positively related to basal cortisol levels (30) and to DST nonsuppression (31). Normal-weight and obese individuals who are not depressed generally have normal DST suppression (18). Only one study has examined the HPA axis in BED (22) and found normal cortisol suppression after DST, regardless of depression, although they did not measure morning basal cortisol.

Although the acute elevation of cortisol plays a protective role during stress, chronic elevations can promote insulin resistance and abdominal obesity (32,33). A large waist-hip ratio (WHR), reflecting central obesity, appears to be a health risk factor independent of obesity (34) and is related to greater HPA activation (33), associated with increased cortisol levels and vulnerability to stress (18,35). Increased cortisol has been shown to be positively related to central fat distribution in both obese and lean individuals (34,35), and morning cortisol has been shown to be positively associated with WHR and insulin (36).

In this study, we investigated the cortisol stress response to a cold pressor test (CPT) in obese women with BED. CPT has been used since the late 1930s (37) as a pain stress test (38) and has been shown to produce greater cortisol responses in patients with depression (38), AN (16), and obesity (18). Our primary hypotheses were that compared with similarly obese non-BED women, those with BED would (1) exhibit greater cortisol stress response after a CPT; (2) report more hunger and desire to binge eat after CPT; and (3) would have less cortisol suppression after DST. Our secondary hypotheses were that the BED group would have more depression and a greater WHR, which would be related to greater cortisol and insulin levels.

METHODS

Participants

Overweight women were recruited through local advertisements for participation in an outpatient study at the New York Obesity Research Center of St. Luke's/Roosevelt Hospital. During the initial telephone interview, candidates were screened to exclude those with significant health problems (including gastrointestinal, heart, kidney, liver, or Raynaud's disease [extreme peripheral sensitivity to cold], cancer, hypertension, or diabetes). Additional exclusion criteria included unstable weight (± 5%) for the past 3 months, dieting, smoking, current or past 3 months' use of most prescribed medications, especially those affecting body weight such as antidepressants and stimulants, substance abuse or dependence within the past 6 months, or previous hospitalization for psychiatric illness. Women were also excluded if pregnant or lactating but not if taking oral contraceptives (39–41). They underwent a physical examination including medical history, ECG, and blood tests to ensure good health other than obesity. The study protocol and consent form had been approved by the IRB.

The participants (n = 38) completed the Questionnaire on Eating and Weight Patterns, a validated and widely used self-report instrument to diagnose BED (5,42,43), which was then confirmed by clinical interview. They were classified as non-BED if they denied episodes of overeating and as BED if they met full DSM IV diagnostic criteria. Thirteen were classified as non-BED and 11 as BED. Fourteen were classified as subthreshold binge eaters, not meeting the full criteria for BED, and were excluded from the analysis. Two non-BED dropped out of the study due to their time limitations and did not participate in the CPT, leaving 22 women.

Procedures

Body Composition

After a 12-hour fast, participants had anthropometric measurements in the body composition lab, including circumferences of waist and hip, from which WHR was computed. Percentage of body fat was assessed by underwater weighing (Precision Biomedical Systems) to obtain water displacement, the classic method to obtain body density (44). The technicians in the body composition lab were blind to the participants' binge eating status.

Psychological Scales

After body composition, participants completed a battery of questionnaires, including the Zung Depression Rating Scale. This is a well-known reverse-scored scale that provides both a total score and a categorical rating (normal, mild, moderate, severe) of depression (45,46).

CPT

On a separate day, participants arrived at the hospital after a 12-hour fast for the experimental procedures. An IV catheter was inserted, and participants were given a fixed meal, which consisted of 600 ml of Boost (complete nutritional liquid meal, Mead Johnson), prepared 1:1 with water (2.1 J/g). Participants had blood drawn close to 8:00 a.m. (8:52 a.m. ± 1:23 SD), which provided basal cortisol, and periodically for 120 minutes for an unrelated study, and then rested for 2 hours.

Participants then underwent the CPT just after noon (12:46 p.m. ± 1:14) and immersed their nondominant hand, up to wrist level, with fingers apart and not touching sides, for a total of 2 minutes in a rectangular container of 0 °C ice water. To prevent a temperature rise, a strainer bag with ice was kept in the water, monitored with a digital thermometer. Participants were asked to rate pain in the hand, stress, hunger and desire to binge eat on visual analogue scales (VAS) from 0 (not at all) to 100 (most imaginable). Digital monitors assessed diastolic and systolic blood pressure and heart rate, and blood was drawn and assayed for cortisol and insulin. All measurements were at −10 and 0 to assess baseline measures, at 2 minutes, corresponding to hand withdrawal, and at 5, 15, 30, 45, and 60 minutes relative to initial hand submersion.

DST

On a third day, participants were requested to take a 1-mg oral dose of dexamethasone at home at 11:00 p.m. The following morning, blood was drawn at 8:00 and assayed for cortisol.

Cortisol Assay

Blood samples were kept on ice for 30 minutes to ensure clotting and then cold centrifuged for 15 minutes to obtain serum, which was stored at −70 °C until assayed. Total serum cortisol was measured by a radioimmunoassay kit (RIA from Diagnostic System Labs), a solid phase immunoassay, which detects cortisol concentrations as little as 0.2 μg/dl cortisol (intra-assay CV = 2.8; inter-assay = 4.8).

Data Analyses

Univariate ANOVA was used to compare group parametric measures. Area under the curve (AUC) was calculated for rating scores and hormone levels to sum the levels for all the time periods. Pearson correlations (r) were used for continuous dependent variables. χ^2 Was used for categorical data. Means are reported ± SD.

Data were interpolated to calculate AUC. When a value was missing between two data points, the mean of those two points was used. When a baseline value was missing, the second baseline measure was used. When a final data point was missing, the last data point was carried forward. Finally, when two or more adjacent data points were missing, which happened with only one subject, data were left as missing and not used in the analysis.

Approximately 1% of the cortisol and insulin data, 15% of the blood pressure, and 5% of the heart rate data were interpolated.

Results are presented as mean ± SD, with two-tailed $p \leq .05$ required for statistical significance. Data were analyzed using the Statistical Package for the Social Sciences (SPSS version 11.0, 2001, Chicago, IL).

RESULTS

Participant Characteristics

Participants in the two groups did not differ in age, BMI, percent body fat, or WHR, as shown in Table 1. The time of day for basal cortisol (F[1,21] = 1.0, $p = .32$) and CPT (F[1,22] = 1.3, $p = .26$) did not differ between the BED groups. Regarding the menstrual cycle, four participants were in the follicular phase and menstruating, four were in the follicular stage but not menstruating, eight were in the luteal phase, and six had no stage of cycle identified. There were no differences between groups in stage of menstrual cycle ($\chi^2 = 3.7$, $p = .30$) and no differences in morning basal cortisol (F[1,21] = 1.4, $p = .27$) or insulin levels (F[1,21] = 0.71, $p = .56$, ns) by menstrual phase. Therefore, menstrual stage was not used as a covariate in the hormone analyses. Three women (one non-BED, two BED) were taking oral contraceptives, in whom morning basal cortisol levels were higher (12.9 ± 6.8 vs. 23.8 ± 14.4) (F[1,21] = 4.7, $p = .04$), but insulin levels were similar (20.0 ± 13.4 vs. 19.4 ± 6.0; F[1,21] = 0.01, $p = .93$, ns). Therefore, contraceptive use was entered as a covariate only in the cortisol analyses.

Psychological Scales

The BED group scored significantly higher (42.3 ± 10.2) on the Zung depression scale compared with non-BED (33.3 ± 5.5; F = 6.6, $p = .02$). There were no differences between groups on the category of depression ($\chi^2 = 6.0$, $p = .11$).

Cortisol

Morning basal cortisol (μg/dl) was significantly higher in the BED (18.4 ± 10.7) compared with non-BED (10.9 ± 4.5; F = 5.4, $p = .03$), even after controlling for contraceptive use (F = 5.8, $p = .03$; Figure 1). Baseline cortisol before CPT was not significantly higher (8.9 ± 6.0 vs. 6.3 ± 4.7) in the BED group both before (F = 1.3, $p = .27$, ns) and after (F = 0.96, $p = .34$, ns) controlling for contraceptive use.

After CPT, BED individuals had a nonsignificantly greater AUC for cortisol (468.4 ± 266.0) than non-BED individuals (307.7 ± 157.4; F = 3.0, $p = .10$). After controlling for insulin, AUC for cortisol reached significance (F = 4.5, $p = .047$), but not after also controlling for contraceptive use (F = 4.1, $p = .057$; Figure 2). Depression score was not a significant covariate for cortisol AUC (F = 1.2, $p = .29$, ns), and cortisol levels did not differ between category of depression (F = 0.25, $p = .86$).

TABLE 1. Characteristics of Female Participants (mean ± SD)*

	Non-BED	BED
n	11	11
Age, y	32.5±8.5	29.±8.4
BMI	35.5±5.7	36.6±6.2
Underwater weighing (% fat)	40.1±4.1	41.7±6.4
WHR	0.87±0.10	0.83±0.05
Sagittal diameter, cm	24.4±3.3	25.5±3.6

*There were no significant differences between groups in any of the characteristics.

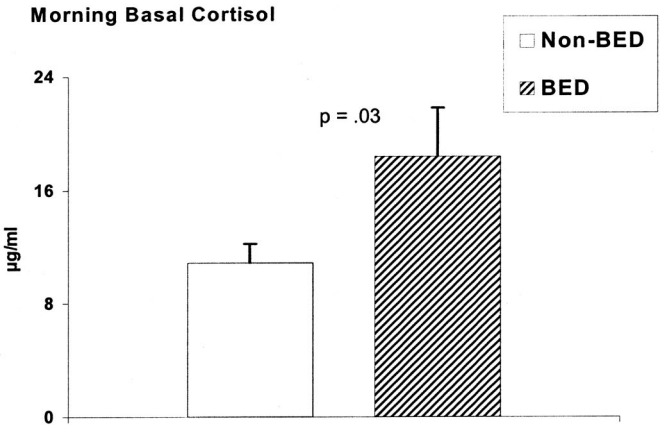

Figure 1. The BED group had higher morning fasting basal cortisol than non-BED (mean ± SE).

Insulin

There were no differences in morning basal insulin (μU/ml) in the BED (24.4 ± 15.8) compared with the non-BED (16.1 ± 17.2; F = 3.0, $p = .10$) or in baseline insulin near noon before CPT (26.9 ± 27.1 vs.13.7 ± 3.5; F = 2.6, $p = .13$, ns). After CPT, BED individuals did not have a significantly greater AUC for insulin than non-BED individuals (F = 2.4, $p = .14$).

DST

After the DST, there were no differences in cortisol levels between the non-BED and BED groups (0.58 ± 0.25 vs. 0.75 ± 0.55; F = 0.74, $p = .40$, ns). However, compared with their higher morning basal cortisol, the BED group had a greater relative reduction in cortisol after DST (F = 5.1, $p =$

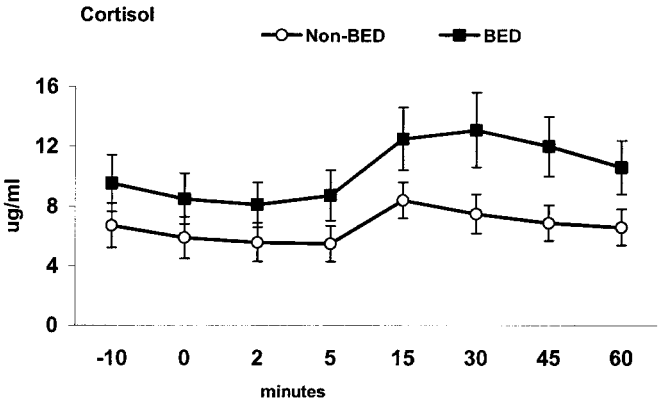

Figure 2. The BED group showed a near significant greater AUC for cortisol ($p = .057$) after controlling for insulin AUC and contraceptive use. (mean ± SE).

.03). Those using contraception had higher cortisol after DST than nonusers (1.1 ± 0.82 vs. 0.58 ± 0.29; F = 8.6, $p = .007$), and after covarying for contraceptive use, the BED groups still did not differ (F = 0.05, $p = .83$, ns). Depression (F = 0.71, $p = .41$, ns) and BMI (F = 0.70, $p = .41$, ns) were not significant covariates for cortisol after DST.

VAS Ratings

At baseline before the CPT, "desire to binge eat" (F = 5.9, $p = .03$) and stress ratings (F = 4.3, $p = .05$) were higher in the BED compared with non-BED groups; however, hunger (F = 2.1, $p = .16$, ns) and pain ratings (F = 1.0, $p = .33$, ns) were similar. AUC was significantly greater in BED than non-BED for hunger (F = 5.3, $p = .03$; Figure 3), desire to binge eat (F = 6.4, $p = .02$), stress (F = 4.4, $p = .05$) and pain (F = 5.9, $p = .02$).

Sympathetic Activity

Systolic blood pressure (F = 1.5, $p = .23$, ns), diastolic blood pressure (F = 2.3, $p = .15$, ns) and heart rate (F = 1.1, $p = .30$, ns) were similar between the groups at baseline. There were no significant differences in AUC for systolic blood pressure (F = 0.10, $p = .76$, ns), diastolic blood pressure (F = 3.8, $p = .07$), or heart rate (F = 3.0, $p = .10$) between the BED groups.

Correlations

For all subjects combined, AUC for cortisol was correlated with AUC for pain ($r = 0.46$, $p = .03$), but not with hunger or desire to binge eat. AUC for stress was correlated with AUC for desire to binge eat ($r = .67$, $p = .001$) and with AUC for pain ($r = 0.43$, $p = .05$). AUC for pain was correlated with AUC for hunger ($r = 0.45$, $p = .03$) and desire to binge eat ($r = 0.56$, $p = .006$). There were no overall significant correlations between depression, basal cortisol, cortisol AUC, insulin, or WHR. However, in the BED group only, WHR was significantly correlated with AUC for cortisol ($r = 0.81$, $p = .002$), peak cortisol response (at 15 minutes; $r = 0.80$, $p = .003$; Figure 4), but not with cortisol after DST ($r = 0.56$, $p = .07$), or morning basal cortisol ($r = 0.49$, $p = .15$, ns). After controlling for contraceptive use, the relationships in the BED group between WHR and cortisol following DST reached significance ($r = 0.69$, $p = .04$).

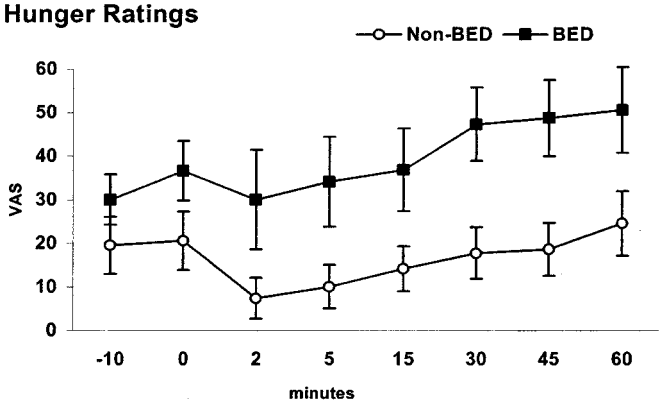

Figure 3. AUC for hunger was greater in the BED group than the non-BED ($p = .008$; mean ± SE).

DISCUSSION

This is the first report to our knowledge of higher morning basal cortisol levels in obese women with BED compared with non-BED. Additionally, we observed a greater cortisol area in the BED group during a cold stress test. Our findings in BED are similar to those in BN showing greater basal cortisol levels (19,25) and increased cortisol after stress (13,17,41). Our findings may also be consistent with a recent study, which reported increased cortisol levels after CPT in obese premenopausal women (18), perhaps due to a subset of binge eaters who were not assessed. In our study, the CPT, which is a laboratory stressor, produced a greater cortisol area in the BED group, and perceived stress and pain were significantly correlated with desires to binge eat.

Although increased pain detection thresholds have been previously observed in BED (47) and BN (48), our BED group actually reported greater pain after CPT than the non-BED. Additionally, pain and stress ratings were correlated with desire to binge eat in the BED group, which is consistent with findings that eating-disordered individuals perceive events as more painful and stressful than controls (10,21). The BED group also reported significantly greater hunger than the non-BED after CPT. These findings provide some support for the role of stress in the initiation of binge episodes (7,20) and support our hypothesized model of stress → cortisol → hunger → binge eating, where cortisol levels after stress serve as a mediator between stress and binge eating. There are multiple real-life stressors, and it maybe worthwhile to extend this work to more psychological stressors.

There is evidence for cortisol nonsuppression in AN (29) and BN (25,49), but this does not appear to be so in BED. We did not observe differences between the groups in cortisol levels after DST, and our findings are consistent with the only other study of DST in BED (22). However, because our BED group exhibited higher morning basal cortisol, they actually had a relatively larger reduction in cortisol after DST compared with the non-BED. All participants were administered a standard 1-mg dose, used in other studies of BED (22) and obesity (50). The results were the same when we entered body weight as a covariate.

As expected, those with BED had higher depression scores (51). However, depression scores did not correlate with basal cortisol levels or DST, confirming previous studies in obese women with BED (22) and normal weight women with BN (52), but in contrast with other studies of major depression (31). One likely explanation is that our obese patients were not as clinically depressed as in most studies of depression. In fact, only two of our participants scored as severely depressed on the Zung, and cortisol levels did not differ between levels of depression. Therefore, the presence of BED itself was apparently responsible for the elevated basal cortisol and AUC.

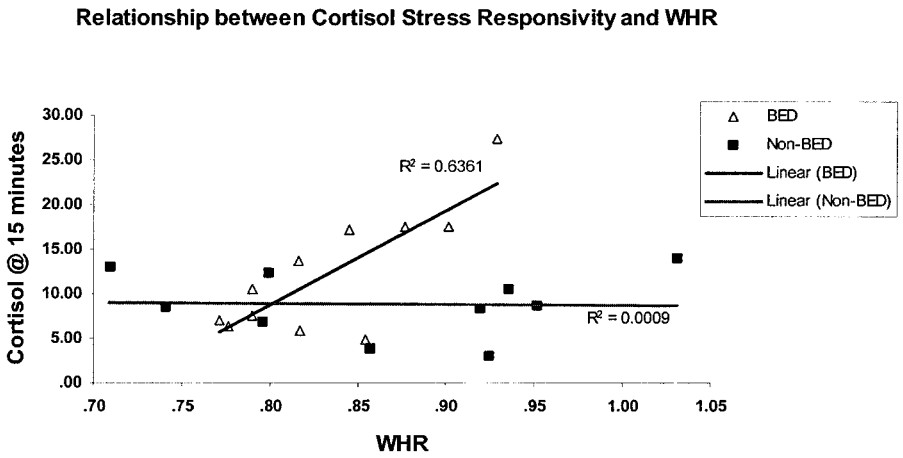

Figure 4. There was a positive and significant relationship between WHR and cortisol stress responsivity in the BED ($p = .003$) but not in the non-BED group ($p = .93$).

The BED group did not have a greater WHR than non-BED, and in contrast to several studies in obese individuals (18,53), there was no overall correlation between morning basal cortisol, insulin, or WHR. Because the BED group also reported more stress at baseline CPT and a greater AUC in response to the CPT, they could be experiencing more chronic stress. Such chronic stress may then promote abdominal obesity (32,33), reflected in cortisol levels after stress and the positive correlation between WHR in the BED group. Thus, binge eating behavior may also help explain the positive relationship between WHR and cortisol after stress (18,35,54,55).

A limitation to this study is the relatively small sample size, and the findings should be replicated with a larger sample size. In sum, we found increased basal cortisol levels in obese women with BED and a greater cortisol area after a stress test, extending the previous findings between stress and food intake in healthy and eating-disordered women to women with BED.

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