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Associations between ovarian hormones and emotional eating across the menstrual cycle: Do ovulatory shifts in hormones matter?

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Abstract

Objective: Elevated ovarian hormone levels are associated with increased risk for binge eating (BE) and emotional eating (EE) during the mid-luteal phase of the menstrual cycle. However, past studies have not examined whether pronounced hormonal changes that precede the mid-luteal phase (i.e., the dramatic decrease in estradiol and increase in progesterone during/after ovulation) also influence mid-luteal increases in binge-related symptoms. Past theories and studies of phenotypes strongly related to BE (e.g., depression) suggest that these pronounced hormonal changes may also contribute. This study examined this possibility in 375 female twins (aged 15-25 years) from the Michigan State University Twin Registry.

Methods: Daily ratings of EE (assessed with the Dutch Eating Behavior Questionnaire) and daily saliva samples of estradiol and progesterone were collected for 45 consecutive days.

Results: No significant associations were found between pronounced changes in estradiol or progesterone across ovulation and changes in EE scores in the mid-luteal phase. Results remained unchanged after controlling for BMI and negative affect and examining participants with clinical BE episodes or more extreme hormonal fluctuations.

Discussion: In aggregate, the current findings and past data suggest that hormone levels are more significant predictors of EE than pronounced hormonal changes across the menstrual cycle.

Keywords

emotional eating; ovarian hormones; ovulation; pronounced change; estrogen; progesterone; binge eating

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Conflicts of Interest

The authors have no conflict of interest to disclose.

Fluctuations in ovarian hormones increase risk for binge eating (BE) and emotional eating (EE**),** with the greatest risk occurring during the mid-luteal phase of the menstrual cycle when ovarian hormones are elevated (Edler, Lipson, & Keel, 2007; Klump et al., 2013, 2014). The antagonistic relationship between estrogen and progesterone is thought to underlie this observation. In the absence of progesterone, estrogen decreases food intake (Asarian & Geary, 2013), BE, and EE (Micioni Di Bonaventura et al., 2017). However, when present, progesterone inhibits estrogen's protective benefits and increases BE and EE levels (Klump et al., 2013, 2014).

Past studies showing these associations have focused on whether morning levels of a woman's hormones, relative to her mean hormone levels, predicted EE later that day. While these analyses significantly increased understanding of daily changes in hormones/EE, some suggest that *pronounced* hormonal changes (e.g., maximum – minimum hormone levels) may also predict BE phenotypes (Edler et al., 2007). If so, then perhaps women with the most pronounced ovulatory changes in ovarian hormones may be at the greatest risk of BE and EE. Ovulation is the period of maximal hormonal change during the menstrual cycle, with estradiol levels dramatically increasing then decreasing during ovulation, and progesterone levels increasing after ovulation. Given that peak rates of BE and EE occur after ovulation, pronounced changes in ovarian hormone levels during/after ovulation may also contribute to the increased risk for binge-related eating. Animal studies show that effects of hormones on behavior typically take up to 24-48+ hours to occur (Geary & Asarian, 1999; Gray & Greenwood, 1982). Because ovulation occurs approximately 3-8 days before the mid-luteal phase (Ecochard et al., 2017), pronounced ovulatory hormonal shifts may also contribute to increased BE and EE in women. While no study has examined these types of effects, studies show associations between pronounced hormonal changes and other phenotypes linked to BE (e.g., depression; Freeman, 2010; Freeman et al., 2007; Schmidt & Rubinow, 2009).

This study examined whether pronounced hormonal changes during/after ovulation predicted increased EE during the mid-luteal phase in a population-based sample of women.

Methods

Participants

Participants included 445 female twins (15-25 years old) from the *Twin Study of Hormones and Behavior Across the Menstrual Cycle* (TSHMBC; Klump et al., 2013) within the Michigan State University Twin Registry (MSUTR; see Burt & Klump, 2013; Klump & Burt, 2006 for MSUTR description). This same sample participated in our previous reports (e.g., Klump et al., 2013; Klump et al., 2014) showing associations between hormone levels and EE/BE. Twins had to meet several inclusion criteria for the TSHMBC study (e.g., no medication use – see Klump et al., 2014). Additionally, for the current study, cycles were required to demonstrate a peak in estradiol during ovulation, resulting in a final sample of 375 women (84%; 375/445). Participants remained demographically representative of the recruitment region (see Supplementary Table S1 and Klump, Keel et al., 2013). Participants

were mostly of Non-Hispanic descent (92%), with 84% identifying as White, 10% Black, 5% Multiracial, 0.5% Native American, and 0.3% Asian or Pacific Islander.

Procedures

All measures and procedures were approved by the Michigan State University Institutional Review Board. Participants provided behavioral and hormone data for 45 consecutive days. Salivary samples were collected within 30 minutes of waking using previously established methods (Edler et al., 2007; Klump, Keel, Culbert, & Edler, 2008; Klump et al., 2013) and assayed for ovarian hormones by Salimetrics, LLC using enzyme immunoassays that show excellent intra- and inter-assay coefficients of variation (see Klump et al., 2013). Participants recorded days of menstrual bleeding in a daily log book. Trained raters coded cycle phase using information about each twin's cycle length and the hormone data (see Klump et al., 2015 for method description).

Measures

The Dutch Eating Behavior Questionnaire (DEBQ; van Strien, Frihters, Bergers, & Defares, 1986) was used to assess daily levels of EE. Internal consistencies for the DEBQ EE scale were excellent in previous research (α = .93; Klump et al., 2008; Racine et al., 2012; van Strien et al., 1986) and the current sample (45-day average α = .90). The DEBQ EE scale has demonstrated concurrent validity (Wardle, 1987) and is correlated with measures of BE (*r*'s = .55–.69; Racine, Culbert, Larson, & Klump, 2009; van Strien, 2000) and palatable food intake in the laboratory (van Strien, 2000). Similar to previous research (Klump et al., 2008), the DEBQ instructions were modified with permission to ask about EE over the current day.

The Negative Affect scale from the Positive and Negative Affect Schedule (Watson, Clark, & Tellegen, 1988) assessed participants' daily negative affect. The negative affect scale exhibits excellent internal consistency and good convergent and discriminant validity (Watson et al., 1988). Internal consistency in the current study was excellent (45-day average α = .85).

Height and weight were measured using a wall-mounted ruler and digital scale during three in-person visits (i.e., beginning, halfway point, end of study). Mean BMI (weight (kilograms)/height (meters)^2) was calculated and used in analyses.

Statistical Analyses and Models

Data preparation followed methods from previous studies (Edler et al., 2007; Klump et al., 2008; Klump et al., 2013). For the repeated measures (i.e., EE, hormones, negative affect), 5-day rolling averages were calculated by anchoring each day as Day 0 and including the 2 days on either side. Rolling averages were then converted to within-person, standardized scores based upon each participant's mean/standard deviation across data collection (see Lester et al., 2003).

EE scores in the mid-luteal phase were used as the outcome variables in all analyses. Within-person hormonal change was the predictor variable and was assessed using each

women's individual hormone difference score across ovulation for estrogen (i.e., maximum during ovulation – minimum in transition to mid-luteal), and across the mid-luteal phase for progesterone (i.e., maximum during the mid-luteal phase - minimum levels during the transition to mid-luteal phase). Negative affect difference scores (calculated in the same manner as above) and BMI were included as covariates in analyses. Data were restricted to one complete cycle from the 45-day period.

Pearson correlations provided initial indications of associations between pronounced hormone changes and EE scores. Mixed linear models (MLMs) were then used to examine how the hormone difference scores predicted average EE scores in the mid-luteal phase. MLMs have been used in past work (see Klump et al., 2013, 2014) and are well-suited for these analyses because they examine predictive associations while controlling for covariates (i.e., negative affect, BMI) and non-independence of twin data. Separate models were run for the maximum/minimum scores and the difference scores due to high multicollinearity between the difference scores and maximum/minimum scores. All MLMs were conducted for estrogen only, progesterone only, and then a set of joint hormone models to examine potential estrogen × progesterone interactions.

Results

Results showed ample variability in the range of EE (raw score range = 0 - 4.01, within-person z-score range = −1.39 - 1.71) and hormone scores (i.e., 0.03 - 4.46; see Supplemental Table S1). Pearson correlations showed no significant associations between EE scores in the mid-luteal phase and any of the maximum/minimum hormone values or difference scores (r's = .001-.10; p's > .10; see Table 1). Likewise, no significant main effects of maximum/minimum hormone levels, difference scores, or significant interactions were found in the MLMs for either estrogen or progesterone alone or in the joint hormone models (all p's > .05, see Table 2).

Because effects were counter to hypotheses, post-hoc analyses were conducted to ensure that results were not unduly influenced by study design decisions or missing data. To ensure that results were not due to the exclusion of subjects missing the transition to mid-luteal phase (see Methods), models were re-run using maximum, minimum, and difference scores from other phases around ovulation (i.e., ovulation to the mid-luteal phase, rather than the transition to mid-luteal phase; follicular phase to ovulation). Again, no significant main or interaction effects were observed (all p's > .05; see Supplemental Table S2). Nonetheless, it is possible that our 5-day rolling averages over- or under-estimated mean hormone levels across phases, given varying phase lengths. All models were re-run using single day maximum/minimum values and 2- and 3-day rolling averages. Still, no significant associations were found (all p's > .05; see Supplemental Table S3).

Although past work showed similar hormone/EE associations in clinical versus non-clinical samples (Edler et al., 2007; Klump et al., 2008, 2014), effects of pronounced hormonal change may only be present in women with more severe levels of pathology. Exploratory analyses examined women (n = 20) who endorsed a lifetime history of OBEs (i.e., episodes defined by a large amount of food and a loss of control) on diagnostic interviews (see Klump

et al., 2014 for methods). Results remained unchanged (all *p*'s > .05; see Supplemental Table S4). Finally, it is possible that pronounced hormonal change/EE associations are only present in women with extreme hormonal changes. We compared emotional eating scores between women whose hormone difference scores fell in the top versus bottom third of the distribution. Results again remained unchanged (all *p*'s > .05; see Tables S5).

Discussion

This is the first study to examine the effects of pronounced hormonal change on binge-related phenotypes in women. Despite theories and findings for other phenotypes (e.g., depression; Freeman, 2010), our results showed no significant associations between EE scores and pronounced changes in estradiol or progesterone. Results remained unchanged in post-hoc analyses that examined various factors that could have influenced results (e.g., missing data, clinical status of participants, different cycle phases, more extreme hormonal fluctuations). Thus, contrary to our hypotheses, pronounced changes in ovarian hormones did not significantly predict EE scores in the mid-luteal phase of the menstrual cycle.

Together with past work, results suggest that same-day deviations from mean hormone levels examined in previous studies (Edler et al., 2007; Klump et al., 2013, 2014, 2016) may be driving the increase in EE in the mid-luteal phase. Reasons for this are unclear; although pronounced changes in estradiol and progesterone are expected components of the menstrual cycle that trigger and follow ovulation, ovulation may not increase EE risk. Conversely, large deviations from mean hormone levels in the mid-luteal phase (examined in previous studies) may reflect a riskier hormonal milieu for women because they coincide with the period in which conception may occur during which greater food intake would be desirable.

Despite the strengths of our study (e.g., large sample size, intensive daily data), limitations should be noted. A subsample of participants (8%) were eliminated from the primary analyses due to an inability to confirm ovulation. While this may have affected results, post-hoc analyses including these participants yielded similar results (see Table S2). We used a community, not clinical, sample of women. Past studies suggest no significant differences in effects across clinical and community samples (Edler et al., 2007; Klump et al., 2008; Klump et al., 2013, 2014), and post-hoc analyses suggested similar findings in women with OBEs. Nevertheless, our OBE group was small ($n = 20$), suggesting a need for larger-scale studies to replicate our results.

We relied on difference scores for measuring hormonal changes. Difference scores are limited by increased measurement error (Bereiter, 1963) that could have decreased power to detect significant effects. However, results across multiple analyses (i.e., correlations, MLMs), different samples (i.e., non-clinical versus women with OBEs), and different cycle phases (e.g., ovulation and transition to mid-luteal) all yielded non-significant results. Finally, we did not examine other factors that change across the menstrual cycle and regulate feeding behaviors (e.g., neuroactive steroids like 5α-dihydroprogesterone. 3α,5α-tetrahydroprogesterone, allopregnanolone; Monteleone et al., 2003; Pham, Porter, Svec, Eiswirth, & Svec, 2000), and we did not examine other biomarkers of hormones (e.g., urine samples; De Souza et al., 2010) that could produce more significant hormone change/EE

associations. Additional studies that examine other hormone-related factors and hormone sampling methods are needed to replicate and extend our results.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Table 1.

Pearson correlations examining associations between hormone variables, covariates, and average emotional eating scores (across the mid-luteal phase).

Hormone variables and covariates		**Correlations with Emotional Eating**	
		r	***p***
Estrogen	Maximum during Ovulation	0.10	.12
	Minimum during Transition to Mid-Luteal	0.07	.25
	Difference Score	< 0.01	.97
Progesterone	Minimum during Transition to Mid-Luteal	−0.02	.72
	Maximum during Mid-Luteal	−0.05	.39
	Difference Score	−0.03	.63
Negative Affect	Maximum during Ovulation	0.01	.88
	Maximum during Mid-Luteal	0.08	.18
	Minimum during Transition to Mid-Luteal	0.06	.30
	Difference Score Ovulation to Mid-Luteal	−0.07	.17
Body Mass Index	Mean BMI	−0.05	.38

Note: Difference score = maximum – minimum hormone; Mean BMI = average BMI across all three study visits.

Table 2.

Mixed linear models (MLMs) examining associations between emotional eating scores and minimum, maximum, and hormone difference scores.

Model	*b(SE)*	*t*	*df*	*p*
Single Hormone Estrogen Models				
Maximum/Minimum Model (n = 313)				
Intercept	0.08 (0.16)	0.49	266	.63
Min EST during Trans to Mid-Luteal	0.03 (0.04)	0.53	264	.60
Max EST during Ovulation	0.07 (0.06)	1.21	267	.23
Min Neg Aff during Trans to Mid-Luteal	0.05 (0.04)	1.26	265	.21
Max Neg Aff during Ovulation	−0.01 (0.04)	−0.23	266	.82
BMI	−0.01 (0.01)	−1.02	267	.31
Difference Score Models (n = 313)				
Intercept	0.10 (0.15)	0.68	297	.25
Difference Score EST	−0.01 (0.04)	0.15	291	.88
Difference Score Neg Aff	−0.03 (0.03)	−0.88	291	.38
BMI	<0.01 (0.01)	−0.61	296	.55
Single Hormone Progesterone Models				
Maximum/Minimum Model (n = 375)				
Intercept	0.16 (0.18)	0.89	343	.38
Min PRO during Trans to Mid-Luteal	0.02 (0.06)	0.37	343	.71
Max PRO during Mid-Luteal	−0.05 (0.06)	−0.86	343	.39
Min Neg Aff during Trans to Mid-Luteal	−0.03 (0.06)	−0.47	335	.64
Max Neg Aff during Mid-Luteal	<0.01 (<0.01)	1.02	342	.31
BMI	<0.01 (0.01)	−0.57	341	.57
Difference Score Models (n = 375)				
Intercept	0.13 (0.16)	0.80	342	.42
Difference Score PRO	−0.03 (0.04)	−0.61	342	.55
Difference Score Neg Aff	<0.01 (<0.01)	0.98	342	.33
BMI	<0.01 (0.01)	−0.52	341	.61
Joint Hormone Models				
Maximum/Minimum Models (n = 313)				
Intercept	0.08 (0.21)	0.36	290	.72
Min EST during Trans to Mid-Luteal	0.03 (0.05)	0.59	290	.55
Max EST during Ovulation	0.10 (0.06)	1.68	290	.09
Min PRO during Trans to Mid-Luteal	−0.01 (0.07)	−0.11	290	.92
Max PRO during Mid-Luteal	−0.02 (0.06)	−0.32	290	.75
Max Neg Aff during Ovulation	−0.02 (0.04)	−0.51	290	.61
Min Neg Aff during Trans to Mid-Luteal	−0.02 (0.07)	−0.25	290	.80
Max Neg Aff during Mid-Luteal	<0.01 (<0.01)	0.69	290	.49
BMI	−0.01 (0.01)	−1.05	290	.30
Difference Score Models (n = 313)				

Model	*b(SE)*	*t*	*df*	*p*
Intercept	0.13 (0.22)	0.58	284	.56
Difference Score EST	0.08 (0.14)	0.62	290	.54
Difference Score PRO	<0.01 (0.08)	0.05	291	.96
Difference Neg Aff Ovulation to Mid-Luteal	<0.01 (<0.01)	−0.37	291	.71
Difference Score EST × Difference Score PRO	−0.03 (0.06)	−0.56	291	.58
BMI	−0.01 (0.01)	−0.94	280	.35

Note: Abbreviations include: EST = estradiol, PRO = progesterone, BMI = body mass index, Max = maximum, Min = minimum, Difference score = maximum-minimum, Trans = Transition, Neg Aff = Negative Affect, SE = standard error.