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Premenstrual symptoms as a marker of ovarian hormone sensitivity in eating disorders

Sabrina L. Hardin, BA, Laura M. Thornton, PhD, Melissa A. Munn-Chernoff, PhD, Jessica H. Baker, PhD

Department of Psychiatry, University of North Carolina at Chapel Hill, Chapel Hill, North Carolina

Abstract

Objective: Research indicates a link between ovarian hormones and eating pathology, suggesting that some women with an eating disorder may be ovarian hormone sensitive. Using premenstrual symptoms (PMS) as an indirect measure of ovarian hormone sensitivity, we investigated the association between 11 PMS domains and four core eating disorder symptoms: body dissatisfaction, binge eating, purging, and restriction.

Method: Participants were young adult women ($N = 455$) who completed an online survey. PMS were assessed using the Daily Record of Severity of Problems and eating pathology with the Eating Pathology Symptoms Inventory. Pearson correlations were calculated between PMS domains and eating disorder symptoms followed by a stepwise regression to create a more refined model for each eating disorder symptom, including relevant covariates.

Results: Significant correlations between a majority of eating disorder symptoms and PMS emerged (r 's = .13–.37; $p < .01$). Backward regression revealed significant PMS domain predictors for each symptom. The final models captured a small-to-moderate amount of variance for each eating disorder symptom ($R^2 = 0.06$ – 0.25).

Discussion: Women who experience physical and psychological PMS may be at risk for eating disorder symptoms; PMS could be a marker of ovarian hormone sensitivity in women at risk for an eating disorder. Future studies should address mechanisms underlying this association.

Keywords

binge eating; eating disorders; estrogen; menstrual cycle; premenstrual symptoms

1 | INTRODUCTION

Eating disorder symptoms vary with menstrual cycle phase such that symptoms are highest during the postovulatory half of the menstrual cycle—when progesterone reaches its peak and estrogen rises in parallel—compared with the first half of the cycle (Edler, Lipson, & Keel, 2007; Lester, Keel, & Lipson, 2003; Racine et al., 2012). This mimics animal models showing that estrogen directly inhibits food intake and progesterone in the presence of

estrogen may increase food intake (Schwartz & Wade, 1981). Therefore, we have hypothesized that women with eating disorders may display differential sensitivity to *normal* changes in ovarian hormone levels (Baker, Girdler, & Bulik, 2012; Baker & Runfola, 2016). However, directly measuring hormone sensitivity requires experimental manipulation of ovarian hormones which is challenging in humans. An indirect marker of such sensitivity provides a less invasive method to evaluate this hypothesis. Premenstrual symptoms (PMS), such as irritability and breast tenderness, occur during the second half of the menstrual cycle and are due to postovulatory changes in ovarian hormones (Yonkers & Simoni, 2018). Thus, PMS may represent an indirect measure of ovarian hormone sensitivity such that greater PMS is suggestive of heightened sensitivity to normally fluctuating ovarian hormones. To further understand ovarian hormone sensitivity in eating disorders, we examined the association between PMS and eating disorder symptoms.

PMS and premenstrual dysphoric disorder (PMDD; severe form of PMS) are significantly associated with higher odds of binge-type eating disorders (Nobles et al., 2016). Additionally, the presence of PMDD in bulimia nervosa was associated with an 11-year greater duration of bulimia nervosa (Nobles et al., 2016). This may suggest that PMS and PMDD share an underlying pathophysiology with certain eating disorders. A limitation of this study, however, is that PMS and eating disorder symptoms were assessed globally: the relation between specific PMS and specific eating disorder symptoms were not evaluated. Relatedly, mood (e.g., depression) and physical (e.g., nausea) oral contraceptive side effects have been positively associated with drive for thinness and body dissatisfaction (BD) (Bird & Oinonen, 2011). However, oral contraceptives are comprised synthetic estrogen and progesterone, so it is unclear how this translates to associations with naturally occurring hormones.

The objective of this study was to investigate the association between specific domains of PMS and four core, transdiagnostic eating disorder symptoms: BD, binge eating (BE), purging, and restriction. Supporting our hypothesis of ovarian hormone sensitivity in eating disorders, we hypothesized that PMS would be significantly associated with eating disorder symptoms.

2 | METHOD

2.1 | Participants and procedure

Participants ($N = 455$) were undergraduate women (age 18 years old) from a southeastern university who completed an online survey between Fall 2016 and Fall 2017 as part of a study examining mental health in college students. Participants received course credit for participation and consented online prior to survey completion. The local Institutional Review Board approved the study.

2.2 | Measures

2.2.1 | Eating disorder symptoms—The Eating Pathology Symptoms Inventory (EPSI; Forbush et al., 2013) was used to assess eating disorder symptomatology. For the current study, the BD, BE, purging, and restriction subscales were used. The reliability

(Cronbach's alpha) of the EPSI was estimated at .90 for BD, .89 for BE, and .81 for both purging and restriction.

2.2.2 | Premenstrual symptoms—The Daily Record of Severity of Problems (DRSP) was used to assess PMS (Endicott, Nee, & Harrison, 2006). The DRSP is a 21-question self-report measure that assesses the presence of the 11 domains of PMS/PMDD outlined in the DSM-IV (American Psychiatric Association [APA], 2000): (a) depressed mood; (b) anxiety: tension, feelings keyed up/on edge; (c) affective lability: feeling suddenly sad/tearful, increased sensitivity to rejection; (d) anger: persistent anger/irritability, interpersonal conflicts; (e) decreased interest in usual activities; (f) concentration problems; (g) fatigue; (h) appetite change: change in appetite, overeating, or food cravings; (i) sleep problems: hypersomnia/insomnia; (j) feeling overwhelmed: sense of being overwhelmed or out of control; (k) physical symptoms: breast tenderness, headaches, or bloating. The DRSP was intended to be completed daily throughout the menstrual cycle to make a PMDD diagnosis. Thus, the DRSP indicates whether a specific DSM-IV PMDD domain is present and to what extent (not at all to extreme). Domains do not specifically represent subscale scores since each domain is only indicated by between 1 and 3 questions. Due to the cross-sectional study design, we modified the instructions: we asked women to report the extent to which they experience symptoms in the above domains *in the week prior to their menstrual period*. Items may capture new onset symptoms or premenstrual exacerbation of symptoms. Cronbach's alpha for the 21-item DRSP was estimated at .95.

2.2.3 | Covariates—Participant's self-reported age, race, and ethnicity. Self-reported current height and weight were used to calculate current body mass index (BMI). Three participants had implausible BMI (i.e., BMI < 6 or BMI > 54), which were recoded as missing. General symptoms of depression and anxiety over the previous week were reported with the Depression Anxiety and Stress Scales (DASS; Lovibond & Lovibond, 1995), which has strong internal consistency and validity (Antony, Cox, Enns, Bieling, & Swinson, 1998). Finally, participants reported current hormonal birth control use (yes/no).¹

2.3 | Statistical analyses

Statistical analyses were conducted with SPSS (IBM SPSS Statistics, version 25.0). Pearson correlations were used to evaluate the association between eating disorder symptoms and PMS. Due to the large number of significant correlations (defined conservatively as $p < .01$) observed (Table 1), we then completed a backward stepwise regression. The stepwise regression allowed us to obtain a more refined prediction model: it is useful when there are a limited number of independent variables that may be correlated, which are desired to be reduced to predictors that offer a unique contribution (Heinze, Wallisch, & Dunkler, 2018). The stepwise regression included all covariates and significantly correlated PMS domains ($p < .01$); the exclusion criterion was set at $p > .1$. With the exception of hormone birth control status, covariates were allowed to leave the model based on the exclusion criterion: hormone birth control status was retained in all final models. Given the appetite change domain of

¹There were no significant ($p < .05$) differences in eating disorder symptom scores based upon current hormonal birth control use. There were also limited differences in PMS domains based upon current hormonal birth control use: only fatigue and sleep problems were significantly different.

PMS included an item related to overeating, we conducted regression models with and without this domain to evaluate if associations attenuated.

3 | RESULTS

The mean age of the participants was 19 years ($SD = 1.38$ years) and mean BMI was 22.83 kg/m^2 ($SD = 4.07$ kg/m^2). The sample was 64.4% Caucasian, 11.2% African American, 16.9% Asian, 0.2% Native American, 4.2% more than one race, and 3.1% other. Seven percent of the sample reported Hispanic ethnicity. For stepwise models, race was recoded dichotomously with Caucasian women as the reference group and all other races combined for the second group whereas ethnicity was coded with Non-Hispanic women as the reference group. Forty-four percent of women were using hormonal birth control.

3.1 | Correlation analyses

BD, BE, and restriction were significantly correlated with most domains (r 's = .13–.37, $p < .01$) whereas purging was significantly correlated with six (Table 1). The within subscale correlations for the PMS (majority $r < .60$) indicated that the domains were somewhat distinct.

3.2 | Regression analyses

The PMS domains captured a small-to-moderate amount of variance for the eating disorder symptoms (Table 2). The final models for BD ($R^2 = 0.25$), BE ($R^2 = 0.24$), and restriction ($R^2 = 0.26$) had the most variance explained by the predictors, whereas the model for purging ($R^2 = 0.07$) had the least. The final stepwise model for BD included: BMI, DASS-anxiety, depressed mood, and appetite change. For BE, the final model included: BMI, DASS-anxiety, decreased interest, concentration problems, and appetite change. The final model for purging included DASS-anxiety and feeling overwhelmed, and for restriction, BMI, ethnicity, DASS-depression, DASS-anxiety, and feeling overwhelmed were retained. When appetite change was removed, only two models changed. For BD ($R^2 = 0.21$) the final model included BMI, DASS-anxiety, and affective lability. For BE ($R^2 = 0.18$), BMI, DASS-anxiety, and concentration problems were retained in the final model.

4 | DISCUSSION

We evaluated the association between 11 domains of PMS and eating disorder symptoms. Results indicated that each eating disorder symptom had a unique set of PMS domain predictors. Findings support the broader hypothesis of ovarian hormone sensitivity in eating disorders and extend this hypothesis by showing that this sensitivity is differentially expressed across various eating disorder symptoms.

Results build upon past research showing an association between PMS and eating disorder symptoms. However, our results go a step further by examining the relationship between distinct PMS domains and specific eating disorder symptoms. Results revealed the final models including PMS accounted for the most variance in BD, BE, and restriction suggesting a stronger link between these symptoms and PMS/ovarian hormones than purging. Research consistently shows postovulatory changes in ovarian hormones are

associated with BD and BE (Edler et al., 2007; Racine et al., 2012). However, fluctuations in restriction-type behaviors may be explained by negative affect and emotional eating rather than the direct effect of ovarian hormones (Racine et al., 2012). Findings of the present study further support BD and BE as sensitive to ovarian hormone change whereas only specific indicators of ovarian hormone change may be associated with restriction.

Interesting patterns emerged with specific PMS domains. The combination of appetite change and psychological PMS domains was significantly associated with BD and BE, whereas restriction and purging were limited to psychological domains. Notably, the final model for BE included an inverse association with decreased interest whereas a positive Pearson correlation was observed—this suggests a differential association between decreased interest and BE when the effects of the other independent variables are present. Finally, no PMS domains were significant across all eating disorder symptoms, indicating that aspects of ovarian hormone sensitivity may be differentially expressed across eating disorder symptoms. However, appetite change was significantly associated with BD and BE, whereas feeling overwhelmed was significantly associated with purging and restriction. When appetite change due to PMS was removed from regression models, the R^2 for BD and BE decreased slightly, suggesting that appetite change uniquely contributes to BD and BE; however, it does not entirely account for the association between PMS and BD/BE. Taken together, the association between PMS domains and eating disorder symptoms is not exclusively due to changes in food intake/appetite that may occur with the menstrual cycle.

Results must be considered in the context of limitations. First, we cannot draw conclusions about causation. Second, we used the DRSP retrospectively, which may introduce errors in recall. Relatedly, we are unable to discern whether participants are reporting new onset symptoms premenstrually, or premenstrual exacerbation of already present symptoms. However, both represent ovarian hormone sensitivity as symptoms are appearing and/or changing in relation to hormone changes. Third, our college sample may limit generalizability. However, college is a high-risk period for eating disorders, with 30% of students exhibiting behaviors indicative of an eating disorder (White, Reynolds-Malear, & Cordero, 2011); thus, the sample is enriched for our phenotype of interest. Finally, although we controlled for hormonal birth control use in models, we do not have information on the specific type of birth control used or the timeframe of use, which can have differential behavioral effects and side effects (Graham, Ramos, Bancroft, Maglaya, & Farley, 1995).

Taken together, results support our hypothesis that eating disorders may represent an ovarian hormone sensitive phenotype. Although the mechanism underlying hormone sensitivity and eating disorders remains unclear, given ovarian hormones are hypothesized to play a role in the genetic risk for eating disorders (Klump et al., 2012), there could be a shared underlying genetic etiology between eating disorders and behavioral sensitivity to hormone change. Furthermore, given this association treatments for premenstrual disorders may be efficacious for reducing/stabilizing eating disorder symptoms. For example, a treatment for PMS/PMDD is combined serotonergic antidepressant and oral contraceptive. Although oral contraceptives have been associated with increased levels of some eating disorder symptoms, alternate research suggests contraceptive use decreased symptoms for some women with bulimia nervosa (Naessén, Carlström, Byström, Pierre, & Lindén Hirschberg,

2007). This highlights the importance of personalized treatment. PMS may represent an indirect indicator of women who are sensitive to ovarian hormone change and thus, may be at increased risk for eating disorder symptoms. Future research should use a direct hormone manipulation to further understand the mechanism underlying hormone sensitivity. A deeper understanding of the association between ovarian hormones and eating disorders may lead to targeted and personalized treatments.

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DATA AVAILABILITY STATEMENT

Data are available from corresponding author upon reasonable request.

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TABLE 1

Eating disorder symptoms and PMS domains Pearson correlations

	BD	BE	Purging	Restriction	Depressed mood	Anxiety	Affective lability	Decreased interest	Concentration	Fatigue	Appetite changes	Anger	Sleep	Overwhelmed
Depressed mood	.29	.18	.20	.32										
Anxiety	.21	.19	.17	.29	.66									
Affective lability	.25	.19	.15	.23	.58	.58								
Decreased interest	.15	.13	.07	.21	.55	.51	.51							
Concentration	.20	.27	.19	.26	.54	.52	.42	.61						
Fatigue	.25	.20	.04	.23	.53	.53	.55	.61	.59					
Appetite changes	.32	.34	.14	.16	.40	.41	.54	.46	.44	.57				
Anger	.11	.09	.07	.10	.48	.51	.75	.52	.38	.49	.50			
Sleep	.22	.22	.12	.29	.51	.50	.45	.56	.57	.66	.59	.41		
Overwhelmed	.21	.18	.23	.37	.65	.55	.51	.52	.58	.45	.41	.43	.57	
Physical symptoms	.23	.20	.05	.24	.43	.47	.51	.45	.41	.56	.57	.50	.53	.45

Note: The bold terms indicate significance at the .01 level (two-tailed).

Abbreviations: BD, body dissatisfaction; BE, binge eating; PMS, premenstrual symptoms.

TABLE 2

Backward stepwise regression of eating disorder symptoms and PMS domains

	Unstandardized coefficients			Unstandardized coefficients		
	<i>B</i>	<i>SE</i>	<i>p</i> value	<i>B</i>	<i>SE</i>	<i>p</i> value
BD	Constant	-5.73	1.86	Constant	4.10	1.34
	BMI	0.49	0.07	BMI	-0.14	0.05
	Birth control	0.24	0.60	Ethnicity	1.96	0.87
	DASS-anxiety	0.23	0.05	Birth control	-0.45	0.43
	Depressed mood	0.26	0.11	DASS-depression	0.17	0.04
	Decreased interest	-0.49	0.29	DASS-anxiety	0.13	0.05
BE	Appetite changes	0.66	0.13	Sleep	0.20	0.11
	Constant	-3.74	1.68	Overwhelmed	0.26	0.12
	BMI	0.27	0.07	Constant	-0.01	0.23
	Birth control	-0.36	0.54	Birth control	-0.14	0.22
	DASS-anxiety	0.28	0.05	DASS-anxiety	0.05	0.02
	Decreased interest	-0.67	0.27	Overwhelmed	0.16	0.05
Results removing appetite change ^a	Concentration	0.89	0.28	Constant	-2.92	1.68
	Appetite changes	0.68	0.12	BMI	0.30	0.07
	Overwhelmed	-0.25	0.15	Birth control	-0.17	0.56
	Constant	-5.58	1.95	DASS-anxiety	0.26	0.05
	BMI	0.52	0.08	Concentration	0.85	0.22
	Birth control	0.39	0.61			

Note: Current hormone birth control use was forced to be retained as a predictor within all the final stepwise models.

Bold values indicate significance at *p* < .05.

Abbreviations: BD, body dissatisfaction; BE, binge eating; BMI, body mass index; DASS, Depression Anxiety and Stress Scales; PMS, premenstrual symptoms.

^aPurging and restriction results excluding appetite change are not presented because the results remain unchanged.