MANSI H. MEHTA, Ph.D. December 2019 PSYCHOLOGICAL SCIENCES

EXAMINING THE VALIDITY OF PREMENSTRUAL DYSPHORIC DISORDER (145 PP.)

Dissertation Advisor: Jeffrey A. Ciesla

Premenstrual Dysphoric Disorder (PMDD) is thought to be a cyclical affective disorder affecting some women during the premenstrual or luteal phase of the menstrual cycle. Criticisms have been raised about the incremental validity of this disorder as a distinct entity from other mood and affective problems such as Major Depressive Disorder. The purpose of the current study was to investigate the validity of a premenstrual phase specific mood disorder, and to investigate the role of two psychological constructs for women reporting premenstrual affective symptoms and women reporting general depressive symptoms. A multi-method approach to premenstrual affect assessment was used such that presence of premenstrual affective problems was retrospectively reported by participants and prospectively measured via daily diary across 30 days. Self-report measures collected at baseline assessed premenstrual affective symptoms, depressive symptoms, anxiety symptoms, rumination, and anxiety sensitivity. Daily measures of negative and positive affect and sexual behavior were also collected. Hierarchical linear modeling was utilized to examine the presence of relationship between the menstrual cycle and daily affect as well as to investigate whether anxiety sensitivity and rumination influenced daily affect across the menstrual cycle. Primary analyses indicated that women with high scores on the baseline PMDD measure experienced high levels of negative

affect irrespective of menstrual cycle phase. Women with high scores on a measure of general depressive symptoms did display a pattern of increased negative affect during the luteal phase. Exploratory analyses revealed the moderating effect of both anxiety sensitivity and rumination in predicting negative affect in the luteal phase; this effect was found in women reporting severe depressive symptoms and those reporting severe premenstrual affective symptoms. Further research is needed to clarify the presence of a premenstrual mood disorder separate from other mood disorders.

EXAMINING THE VALIDITY OF PREMENSTRUAL DYSPHORIC DISORDER

A dissertation submitted

to Kent State University in partial fulfillment

of the requirements for the

degree of Doctor of Philosophy

by

Mansi H. Mehta, M.S.

December 2019

Dissertation written by

Mansi H. Mehta

B.S., University of Michigan, 2010

M.S., Loyola University Maryland, 2012

Ph.D., Kent State University, 2019

Approved by

_____, Chair, Doctoral Dissertation Committee
Jeffrey A. Ciesla

_____, Members, Doctoral Dissertation Committee
Karin Coifman

_____
Douglas L. Delahanty

_____
Janice D. Yoder

_____
Susan Roxburgh

Accepted by

_____, Chair, Department of Psychological Sciences
Maria S. Zaragoza

_____, Dean, College of Arts and Sciences
James L. Blank

TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

ACKNOWLEDGEMENTS

First, I would like to thank my advisor, Dr. Jeffrey Ciesla, for his mentorship. I am deeply grateful for his encouragement and his belief in my ability to face the many challenges of graduate school. I would also like to express my appreciation to the members of my dissertation committee for their time and valuable feedback. Additionally, I would like to thank the Ohio Psychological Association; their funding allowed me to complete this project.

I also wish to acknowledge my colleagues from my time at Kent State and the Atlanta VA Medical Center; their support, kindness, and humor, were essential to the process of writing this dissertation. I am so grateful for their advice and more importantly, for their friendship. Further, I am thankful for the guidance and warmth shown to me by my academic and clinical supervisors throughout my graduate career. In particular, I wish to thank Dr. Rachel Grover and Dr. Christina Kraft for their wise and compassionate counsel.

Nobody has been more important to me throughout the pursuit of this project than my family members. I would like to thank my parents and sister, whose love and guidance are with me always. They are the ultimate role models. I wish to thank my husband and best friend, Alex, for his unwavering support and patience along this journey. Finally, I am grateful to Ellie and Max for providing me with unending joy even in the most difficult of times.

I. Introduction

Premenstrual dysphoric disorder (PMDD) is a diagnostic category included in the most recent *Diagnostic and Statistical Manual of Mental Disorders* (DSM-5; American Psychiatric Association [APA], 2013). The disorder is characterized by a set of emotional and physiological changes that should occur during the luteal or premenstrual phase of a woman's menstrual cycle. The emotional symptoms most commonly linked to PMDD include irritability, anxiety, depression, and affective lability (APA, 2013). Additional physiological symptoms may include weight gain, bloating, breast tenderness, insomnia, and headaches. These symptoms begin during the luteal phase, improve and remit at the onset of menstruation, and should occur during most or all menstrual cycles.

PMDD symptoms should be of sufficient severity to produce distress and functional impairment. Current estimates of the prevalence rate of PMDD range from 3% to 8%, though some studies estimate this to be lower (Di Giulio & Reissing, 2006). Thus, the prevailing view is that the disorder affects a small but sizeable subset of women. Some argue that PMDD places a significant burden on health-related quality of life, which in part may result from the chronicity of the disorder (Pearlstein & Steiner, 2008; Yang et al., 2008). The inclusion of PMDD in DSM-5 has been met with debate; despite a prevailing belief that PMDD is a valid and distinct diagnostic category, some have contested the validity of the disorder and the necessity for a formal diagnostic category (Hartlage, Breaux, & Yonkers, 2014; Wakefield, 2013).

One major criticism is that PMDD is not a distinct entity from other mood disorders. PMDD is highly comorbid with other mood and anxiety disorders, including major depressive disorder (MDD; Cohen et al., 2002; Endicott, 1994; Kim et al., 2004). A high percentage of women reporting PMDD also report multiple past episodes of other mood disorders (Cohen et al., 2002; Endicott, 1994; Kepple, Lee, Haq, Rubinow, & Schmidt, 2016). Women who currently report MDD also appear to have elevated rates of PMDD (Accortt, Kogan, & Allen, 2013). The high degree of overlap between PMDD and MDD calls the incremental validity of PMDD into question. The experience of menstrual cycle-related mood problems might be more parsimoniously attributed to an exacerbation of MDD caused by physical stressors. Given that both the menstrual cycle and pregnancy have biological causes, one might expect a strong association between premenstrual affective symptoms and post-partum depression (PPD). However, it seems that PMDD and PPD do not frequently co-occur (Accortt et al., 2013). Other studies found non-significant associations between premenstrual affective symptoms and PPD after controlling for parity, personality factors, age, and socioeconomic status (Amiel Castro, Pataky, & Ehlert, 2018). PMDD is closely related to MDD, but not closely related to other disorders linked to menstrual cycle hormones. These findings underline the concern that PMDD may not be a distinct entity from MDD, and in turn, not a valid diagnostic category.

Another criticism is centered on the nature of PMDD as a socially constructed disorder (Offman & Kleinplatz, 2004; Ussher, 2010). Shared cultural knowledge and norms may feed into and shape the discourse on premenstrual mood changes. What is considered normative for a woman's experience of the menstrual cycle is shaped as much

by popular beliefs as it is by scientific research and fact (Offman & Kleinplatz, 2004; Rodin, 1992). It is a commonly held societal belief that women should experience emotional and physical changes in the week prior to menstruation. However, premenstrual affective problems may be more influenced by the beliefs women have about this cyclical phenomenon and less reliant on lived experiences of severe distress and functional impairment that occur during the premenstrual phase. One study found that women believed the average woman experiences significantly distressing premenstrual affective and physical symptoms, and overestimated the number of women who have premenstrual physical and mood symptoms that cause clinically significant distress and impairment (Chrisler, Rose, Dutch, Sklarsky, & Grant, 2006). Additionally, women rated their own premenstrual experiences and symptoms as less distressing and intrusive than others'. There also appears to be A lack of evidence for normative, cyclical, negative mood changes during the luteal phase (Kiesner, Mendle, Eisenlohr-Moul, & Pastore, 2016; Romans, Clarkson, Einstein, Petrovic, & Stewart, 2012). Despite the inconsistency between evidence and beliefs, the societal belief regarding premenstrual negative mood continues to go unchallenged. In turn, PMDD may be conceptualized as an extension of those beliefs and not a psychological disorder (Rodin, 1992; Ussher, 2010).

Sociological and feminist criticisms of PMDD argue that inclusion of the diagnosis in the DSM-5 could be a medicalization of the normative physical and emotional processes women experience (Browne, 2015; Cosgrove & Caplan, 2004). From this perspective, people may minimize the anger and distress resulting from women's life circumstances, such as gender discrimination and trauma, attributing them

to biological processes (Browne, 2015; Rodin, 1992). Labeling these emotional responses as mental illness may allow people to ignore a woman's problems and dismiss her complaints. However, the philosophical nature of these arguments has yet to be formally examined; no empirical evidence supports these claims.

The varied nature of these criticisms underscores the wide range of concerns regarding the validity of PMDD. The disorder may be more effectively and parsimoniously conceptualized as a manifestation of MDD rather than as a unique diagnostic condition. In addition, much of the perceived covariation between affective symptoms and menstrual-cycle phase (even among women who report this covariation) may be illusory. That is, though women who self-report problems consistent with PMDD have shown heightened levels of depressive symptoms, the belief that these symptoms coincide with the luteal phase may be due to expectations rather than an accurate report of the evidence. From a feminist standpoint, labeling normative physical changes and negative emotions as a mental illness minimizes the real problems a woman may face. Despite these issues, the presence of this disorder has been widely accepted, resulting in increased research focusing on the etiology, assessment, and treatment of the disorder. Limitations of the current research, and resulting questions about the validity of PMDD, are discussed below.

Assessment of PMDD

Much of the extant literature on PMDD has methodological issues; these problems contribute to skepticism regarding the presence and validity of a menstrual-cycle-related mood disorder. Many studies of PMDD rely on a cross-sectional design, which is problematic: the criteria for this disorder require a demonstrable pattern of

significant depressive symptoms occurring across most or all of a woman's menstrual cycles. The extant research also uses a wide variety of measurement tools, statistical analyses, and diagnostic practices, without any apparent consistency between studies. Many studies used unvalidated methods; for example, one such study classified women as having PMDD by selecting the days in the luteal phase on which women reported the highest number of symptoms, and comparing those to the *average* rating score across all days in the follicular phase (Cohen et al., 2002). This format is inconsistent with the current definition of the disorder, and raises significant concern that such a practice would inflate the prevalence of PMDD because of the clear bias introduced by having two separate standards for comparing depressive symptoms across cycle phases (measuring luteal-phase distress using the days with the worst distress, versus measuring nonluteal distress as the average day). Though this is simply one example, as will be seen, such issues are common in the PMDD literature.

No consensus exists as to the best tool to measure menstrual-cycle-related mood symptoms. A review of existing instruments found that many assessment tools have not been validated or shown to be reliable (Bosman, Jung, Miloserdov, Schoevers, & aan het Rot, 2016; Haywood, Slade, & King, 2002). At least half of the known instruments rely on retrospective reports, despite DSM-5 criteria calling for prospective ratings (Accortt, Bismark, Schneider, & Allen, 2011; Bosman et al., 2016; Steiner, Macdougall, & Brown, 2003). Retrospective study designs are prone to memory-related biases on the part of respondents, including recall bias (Schwarz, 2007). Such bias might cause women to recall or amplify premenstrual affective and somatic symptoms. Indeed, prevalence rates of PMDD based on retrospective reports are higher than those based on prospective daily

charting (Gehlert, Song, Chang, & Hartlage, 2009). Retrospective reports also rely on individuals being able to accurately report about (a) their daily mood, (b) the phase of their menstrual cycle, and (c) a covariation between daily mood and menstrual-cycle phase. Because PMDD is a cyclical disorder, retrospective reports require women to give correct information about these three aspects for at least 1 month, if not more. It is common when using retrospective reports to find that instead of inferring a relationship from accurately remembered behaviors and circumstances, people more often draw on beliefs about events to reconstruct relevant behaviors (Schwarz, 2007).

Beliefs about PMDD may be based on social norms and expectations in addition to lived experiences. . There are long-held societal beliefs about the presence of negative mood and physical changes in the premenstrual phase (Chrisler & Caplan, 2002; Chrisler et al., 2006; Offman & Kleinplatz, 2004). Yet evidence may not support these beliefs of normative negative mood changes during the premenstrual phase (Kiesner et al., 2016; Romans et al., 2012). One study found women who believe in normative premenstrual mood changes were less accurate in their retrospective recall of their own premenstrual mood and physical changes (Marván & Cortés-Iniestra, 2001). Thus, retrospective reports might reflect stereotypes and beliefs, as well as actual experiences of negative mood.

Prospective daily mood ratings are an alternative and preferred method of gathering data on premenstrual mood symptoms. The use of daily diaries and mood ratings has become more popular, despite concerns about practicality and lack of use in clinical practice (Craner, Sigmon, & McGillicuddy, 2014). The DSM-5 also recommends the use of prospective daily charting of symptoms to diagnose PMDD (APA, 2013; Haywood et al., 2002; O'Brien et al., 2011). As participants complete daily diaries, they

are asked about specific symptoms and behaviors. However, knowledge of a study's purpose can increase the possibility of several types of bias, including demand characteristics and expectancy effects. Demand characteristics are a type of experimental artifact where participants form an interpretation of the experiment's purpose and subconsciously change their behavior to fit that interpretation. Women reporting on specific symptoms throughout the menstrual cycle may self-monitor mood and somatic symptoms during the premenstrual phase, potentially amplifying any underlying negative mood symptoms.

Expectancy effects may also influence participant reports: when a participant knows the experiment purpose, she may either unconsciously affect the outcome or report the expected result. Participants may expect to see negative mood changes during the premenstrual phase, and thereby misattribute underlying or unrelated mood symptoms to the menstrual cycle. In an experimental study, women who were asked to focus on negative premenstrual changes reported more of those experiences compared to a control group; women who were asked to focus on positive changes reported better premenstrual experiences (Kues, Janda, Krzikalla, Andersson, & Weise, 2018). Thus, it appears that awareness of a study's focus on premenstrual affective symptoms does alter the content and perhaps the accuracy of daily ratings of mood and symptoms (AuBuchon & Calhoun, 1985; Callaghan, Chacon, Coles, Botts, & Laraway, 2009; Gallant, Hamilton, Popiel, Morokoff, & Chakraborty, 1991). Because negative beliefs and expectations surrounding the menstrual cycle are pervasive and resistant to change, women may report a relationship between their affective problems and the menstrual cycle where one does not

exist. Women may also mistakenly attribute their affective symptoms to the menstrual cycle without considering other causes.

Regardless of the method of data collection, there is also little consensus as to how these data should be analyzed and interpreted. Though many studies assess symptoms across several cycles, analyses tend to rely only on scores from the premenstrual phase (Bosman et al., 2016). Scores during other phases, and comparisons between phases, are rarely examined. A key feature of PMDD is the increase in mood and physical symptoms that occurs specifically during the luteal phase that remit during the menstrual phase. Comparisons between a woman's symptoms across the luteal, menstrual, follicular, and ovulatory phases are necessary to determine the presence of the disorder. An absence of symptoms in the menstrual and postmenstrual phases helps distinguish between PMDD and other mood disorders. Few studies to date have used this comprehensive method as a part of their assessment of PMDD, which detracts from their conclusions about the presence of a specific premenstrual mood disorder (see Bosman et al., 2016 for review). Similarly, there is variability in which days, and the associated symptom ratings, are attributed to which part of the menstrual cycle. Some studies define the premenstrual phase as the 7 days prior to the onset of menstruation, whereas other studies defined the premenstrual phase differently (Bosman et al., 2016). This format is problematic as interindividual variability exists in the length of the menstrual cycle and its phases, as well as when symptoms are most severe (Kiesner et al., 2016). There are also several methods by which researchers can calculate significant symptom cyclicity, and a lack of consensus as to their reliability and use (Eisenlohr-Moul, et al., 2017). One prospective instrument, the Daily Record of Severity of Problems (DRSP) provides an

example of these problems (Eisenlohr-Moul et al., 2017; Endicott, Nee, & Harrison, 2006). The authors of the DRSP suggested a rating of 4, or "moderate" as the most liberal cutoff for clinically significant symptoms, yet researchers may choose to set a stricter cutoff, as desired, to account for varying study designs (Eisenlohr-Moul et al., 2017). In turn, some studies may have more lenient standards for diagnosis, whereas others are more exacting. Thus, even if women accurately report daily mood, cycle phase, and covariation between mood and phase, researchers have few guidelines for the consistent analysis and interpretation of these data.

Confirming the presence of a menstrual-cycle-related mood disorder requires careful measurement of the presence and timing of symptoms. The significant shortcomings related to defining and measuring premenstrual affective symptoms and PMDD weaken the argument that PMDD is a valid disorder distinct from MDD. These issues underscore the necessity for further research validating the condition.

Etiology of PMDD

An examination of the literature on the etiology of PMDD could help allay concerns about the validity of the disorder. The absence of etiological factors relevant to premenstrual affective symptoms and other mood disorders like MDD would bolster the argument that PMDD is a distinct disorder from MDD. The presence of etiological factors related to the menstrual cycle might also support the concept of a distinct mood disorder tied specifically to the menstrual cycle.

Reproductive hormones are one example of a potential etiological factor directly linked to the menstrual cycle. Gonadal steroids appear necessary for the presence of physical symptoms in the premenstrual phase, but it is unclear whether these hormones

cause premenstrual affective symptoms, and in turn, PMDD. Women with premenstrual affective symptoms do not seem to differ from asymptomatic women in levels of ovarian hormones (Cunningham, Yonkers, O'Brien, & Eriksson, 2009; Hantsoo & Epperson, 2015). Some support exists for the hypothesis that women with premenstrual affective symptoms are more sensitive to changes in ovarian steroid levels, though not all women with these symptoms exhibited this sensitivity (Schmidt et al., 2017). Results are inconsistent as to whether women with premenstrual affective symptoms have differing patterns of gonadal hormone release than asymptomatic women (see Halbreich, 2003, for review). One might expect to see hormonal fluctuations as a main cause of physical and affective symptoms in the premenstrual phase. Yet the evidence does not support differences in hormone patterns between asymptomatic women and those reporting premenstrual affective symptoms.

Serotonin may also play a role in the development and maintenance of premenstrual affective symptoms. The rationale for the focus on this neurotransmitter is the use of serotonergic antidepressants in treating severe premenstrual affective symptoms. Investigations of serotonergic differences between women with PMDD and asymptomatic women provide minimal support for the belief that this neurotransmitter plays a causal role in the development of premenstrual affective symptoms (Halbreich, 2003; Hantsoo & Epperson, 2015; Parry, 2001; Veeninga & Westenberg, 1992). Pharmacological challenges are often used to determine the involvement of specific receptor systems, including serotonin. This type of study also does not provide support for a specific luteal-phase serotonergic dysfunction (Parry, 2001; Yatham, 1993). Thus, a

link between serotonergic dysfunction and affective symptoms in the premenstrual phase has not been demonstrably established.

There does not appear to be strong evidence of specific biological factors that differentiate asymptomatic women and those with premenstrual affective symptoms. However, evidence suggests shared biological factors between women with premenstrual affective symptoms and women with other mood disorders. Lowered levels of allopregnanolone, one gonadal steroid, seem to be associated with symptom severity in women reporting severe premenstrual affective symptoms and women with other mood disorders, including MDD (Freeman, 2017; Schüle, Nothdurfter, & Rupprecht, 2014). This suggests a similarity in the hormones associated with affective symptoms in women reporting PMDD and women reporting other mood disorders. Some researchers also suggest that abnormalities in serotonergic functions in women with premenstrual affective symptoms may reflect an underlying vulnerability factor for dysphoric states in general, including PMDD and MDD (Halbreich, 2003). Thus, serotonergic dysfunction may contribute to affective problems throughout the menstrual cycle, and not solely during the premenstrual phase. Evidence of different patterns or effects of serotonin in women with PMDD than in women with MDD would give weight to the belief that PMDD is distinct from MDD. However, no current evidence exists to support this claim.

Several large-scale studies indicated an intergenerational component to physical and affective problems in the premenstrual phase. A high correlation emerged between mothers displaying premenstrual mood symptoms and daughters reporting similar problems (Halbreich, 2003). Other twin and familial studies place heritability estimates of premenstrual physical and affective symptoms at 33%, based on retrospective reports

(Glick, Endicott, & Nee, 1993; Halbreich, Borenstein, Pearlstein, & Kahn, 2003; Kendler, Karkowski, Corey, & Neale, 1998). Given the importance of familial factors in the etiology of premenstrual affective symptoms and major depression, familial studies that simultaneously examine both disorders would clarify whether the pattern of familial transmission justifies a distinction between the two disorders. One such study found that a family history of MDD did not increase the likelihood of a woman having premenstrual mood symptoms (Payne et al., 2009). This is one of few familial studies to date that have investigated this relationship, and replication of these results is needed. The authors highlighted their use of a single question to measure premenstrual affective symptoms as a limitation of their work, underscoring the importance of further research (Payne et al., 2009).

One recent study found a genetic difference between women with PMDD and those without, relating to an ovarian steroid-regulated gene-silencing complex (Dubey et al., 2017). The authors suggested that this cellular difference and the resulting genetic and biological pathways are responsible for the differential responses to ovarian hormones in women with PMDD. However, this study was the first to show such a difference and replication of these results is necessary. In addition, the Dubey et al. study did not provide information as to whether this difference exists between women with PMDD and women with MDD. Thus, clear conclusions cannot be drawn from these data regarding the incremental validity of PMDD separate from MDD. It is possible that this is a genetic pathway found generally in women with affective symptoms.

Psychological constructs may also play a role in causing premenstrual affective and physical symptoms (Cunningham et al., 2009; Yonkers, Pearlstein, & Rosenheck,

2003). Though many women experience physical discomfort during the menstrual cycle, the presence of physical symptoms alone is insufficient for a diagnosis of PMDD. A woman's interpretations of unpleasant physical changes may be the mechanism that leads to the distress and impairment that mark PMDD (Nillni, Rohan, Mahon, Pineles, & Zvolensky, 2013; Nillni, Rohan, & Zvolensky, 2012). This may speak to the importance of individual differences in a woman's response to physical discomfort (Kiesner et al., 2016). Although women may experience similar levels or degrees of discomfort, some women may react to these symptoms in a way that leads to distress or even impairment. Attributional style may play a role in explaining why one woman experiences distress and impairment during the premenstrual phase while another does not (Kiesner et al., 2016). Negative cognitive styles are a risk factor for PMDD, as they are for MDD (Śliwerski & Bielawska-Batorowicz, 2018). Confirmation of the presence of such etiological pathways, linked to a specific phase of the menstrual cycle, would support the validity of PMDD as a distinct diagnosis.

One construct that may link premenstrual physical symptoms to affective problems is anxiety sensitivity. Anxiety sensitivity is the fear of a wide range of somatic symptoms and the belief that these symptoms are dangerous or threatening (Reiss, Peterson, Gursky, & McNally, 1986; Taylor & Cox, 1998). Women who are more prone to this trait might be more likely to notice physical symptoms during the premenstrual phase and interpret them in a negative light, in turn leading to distress and premenstrual affective symptoms. Researchers found that women higher in anxiety sensitivity report greater numbers of premenstrual affective and physical symptoms (Craner, Sigmon, Martinson, & McGillicuddy, 2013; Nillni et al., 2013, 2012). Thus, anxiety sensitivity

may be a cognitive vulnerability to affective problems in the premenstrual phase. However, there are mixed findings as to the moderating effect of PMDD status on the relationship between anxiety sensitivity and menstrual-cycle phase. Some studies found that women with PMDD reported increased effects of anxiety sensitivity only during the premenstrual phase, whereas others found no such variation across cycle phases (Craner, Sigmon, & Young, 2016; Nillni et al., 2013; Sigmon, Whitcomb-Smith, Rohan, & Kendrew, 2004). This variation may suggest that negative interpretations of somatic symptoms, and the resulting distress, are not restricted to the premenstrual phase. However, PMDD is a cyclical disorder, and physical and affective premenstrual symptoms are to remit after the end of the luteal phase (APA, 2013). A lack of specificity during the premenstrual phase may suggest that increased anxiety sensitivity would result in greater attention to somatic problems in general, resulting in affective symptoms not restricted to the premenstrual phase. Indeed, evidence seems to show that anxiety sensitivity is linked to general affective symptoms. Anxiety sensitivity levels seem to be elevated in individuals with MDD and appears to remain that way even after depressive symptoms remit (Cox, Enns, Freeman, & Walker, 2001; Taylor, Koch, Woody, & McLean, 1996). Anxiety sensitivity may be conceived as an underlying transdiagnostic risk factor for mood and anxiety disorders, and not only for menstrual-cycle-related affective problems (Allan, Macatee, Norr, & Schmidt, 2014).

Rumination is another form of self-focused attention that may lead to premenstrual affective symptoms. When women experience physical discomfort, engaging in repetitive negative thinking about these symptoms could lead to distress and affective problems. Women reporting physical and affective symptoms during the

premenstrual phase also report significantly higher overall levels of rumination than healthy controls (Craner, Sigmon, Martinson, & McGillicuddy, 2014). Increased ruminative brooding and ruminative reflection also led to lower positive valence and reduced calmness specifically during the luteal phase (Welz et al., 2016). Evidence also indicated that higher ruminative reflection was related to increased irritability during the luteal phase (Welz et al., 2016). Other studies of women reporting premenstrual affective symptoms and PMDD showed that rumination, when combined with other cognitive vulnerabilities, highly correlated with pre- and perimenstrual distress, and predicted such distress (Craner et al., 2014; Sigmon, Schartel, Hermann, Cassel, & Thorpe, 2009). Thus, rumination seems to relate specifically to premenstrual distress and affective symptoms. However, rumination is also a well-validated risk factor for other mood and anxiety disorders, including MDD (McLaughlin & Nolen-Hoeksema, 2011; Nolen-Hoeksema, 2000; Wisco & Nolen-Hoeksema, 2008). Rumination appears to be a path leading to premenstrual affective symptoms and more general mood and affective problems. This might suggest that premenstrual affective symptoms and symptoms of depression or anxiety represent the same underlying construct. Research that delineates specific and differing pathways to premenstrual affective problems and MDD could help clarify this relationship. For example, if rumination moderated the relationship between cycle phase and affective symptoms, such that women who ruminate more also have more affective symptoms during the luteal phase, this information could help support the presence of a menstrual-cycle-related mood disorder. To date, few studies have examined and confirmed this relationship (Craner et al., 2016). Further research is needed to help understand the relationship between rumination and premenstrual affective symptoms.

A woman's use of maladaptive emotional-regulation strategies in response to negative mood in the premenstrual phase may also lead to distress and affective symptoms. To date, few studies have examined and compared emotion-regulation strategies in women with and without PMDD. Findings are mixed as to whether a positive association exists between premenstrual affective symptom severity and use of maladaptive emotion regulation strategies (Petersen et al., 2016; Reuveni et al., 2016). There are also contradictory findings as to whether women reporting higher levels of affective and physical symptoms in the premenstrual phase also report a greater use of maladaptive emotion-regulation strategies for coping with negative emotions (Eggert, Witthöft, Hiller, & Kleinstäuber, 2016; Petersen et al., 2016).

An examination of brain imaging during emotion regulation in symptomatic women with PMDD showed hypoactivity in the right dorsolateral prefrontal cortex during all tasks, which may indicate tendencies toward negative affect in general, rather than a specific emotion-regulation deficit (Petersen et al., 2018). The relationship between emotional-regulation strategies and premenstrual affective symptoms is still unclear. In contrast, the use of poorer emotion-regulation strategies has been linked to several other psychological disorders, including MDD (Joormann & Gotlib, 2010). Based on the current research, maladaptive emotional-regulation strategies may lead to affective problems during and outside of the premenstrual phase. It is also possible that use of maladaptive emotion-regulation strategies is an etiological factor that supports a distinction between MDD and PMDD. Further research may discern evidence to support either conclusion.

The main goal of reviewing the literature on the etiology of PMDD is to explore the presence of factors that would support the validity of the disorder and substantiate a distinction between PMDD and MDD. Although factors such as rumination contribute to premenstrual affective symptoms, the current research implicates many of the same risk factors that have also been implicated in other mood disorders. Additionally, it appears that few studies have examined the existence of factors that could differentiate between women with MDD and women with PMDD. These limitations underscore the need for ongoing research validating PMDD and differentiating the construct from other mood disorders.

Treatment of PMDD

Understanding the treatment of PMDD and premenstrual affective symptoms may clarify factors that help to resolve concerns about the validity of a menstrual-cycle-related mood disorder. Considering the previously mentioned comorbidity between PMDD and other mood disorders, some commonalities may exist in the treatments of these disorders. However, the existence of other treatments that are effective in treating premenstrual affective symptoms, and not effective for treating other mood disorders, may help support a distinction between PMDD and those disorders. In contrast, if premenstrual affective symptoms respond to the same treatments as other mood disorders, and in the same ways, this may suggest a more general underlying cause for these affective problems.

A review of studies investigating treatment options for women reporting PMDD showed that 49 of 55 studies focused on pharmacological interventions such as antidepressant medication, hormonal therapy, and oral contraceptives (Sepede, Sarchione, Matarazzo, Di Giannantonio, & Salerno, 2016). In part, this finding may be due to the

belief that PMDD and premenstrual affective symptoms stem from biological processes. Serotonergic antidepressants have been promoted as the first line treatment for women experiencing premenstrual affective symptoms (selective serotonin reuptake inhibitors [SSRIs]; Ismaili et al., 2016; Maharaj & Trevino, 2015). These are the same medications physicians often prescribe for other mood disorders like MDD. For women reporting premenstrual affective symptoms, these medications show significant but small effects. However, approximately 40% of women in controlled studies have an insufficient response to serotonergic antidepressants, with no clear predictors of response identified (Freeman, 2017). The exact mechanisms of the effects of SSRIs are unclear; various dosing schedules have been implemented, including continuous dosing, intermittent dosing during the luteal phase, and symptom-response dosing (Maharaj & Trevino, 2015). Additionally, some researchers showed SSRI treatment reduces premenstrual affective symptoms with a short onset of action, before individuals would have received an adequate dosage (Cunningham et al., 2009). This is concerning, particularly as it appears that the placebo response is very high for women with premenstrual affective symptoms (Eisenlohr-Moul, Girdler, Johnson, Schmidt, & Rubinow, 2017; Kleinstäuber, Witthöft, & Hiller, 2012). This placebo response may explain why SSRIs are effective in a rapid timeframe, or why intermittent and symptom-response dosing schedules are effective for premenstrual affective symptoms but not for other mood disorders. Though it is unclear as to how or why SSRI treatments are effective for some women with premenstrual affective symptoms, they remain the first option for pharmacological intervention. Thus, it appears women with premenstrual affective problems benefit from

the same antidepressant medications that are effective and most commonly used in treating other mood disorders.

Researchers have investigated psychological treatments for premenstrual affective symptoms and PMDD, though in much less detail (Freeman, 2017; Sepede et al., 2016). Studies of the effects of cognitive-behavioral treatments for premenstrual affective symptoms indicate that cognitively focused therapies are effective in reducing premenstrual affective and somatic symptoms, though they do not outperform pharmacotherapy (Kleinstäuber et al., 2012; Lustyk, Gerrish, Shaver, & Keys, 2009; Weise et al., 2019). Also some evidence has emerged that third-wave interventions targeting acceptance and mindfulness are effective in reducing premenstrual affective symptoms. Mindfulness was significantly negatively correlated with premenstrual-mood-symptom severity (Lustyk, Gerrish, Douglas, Bowen, & Marlatt, 2011). Specific mindfulness-based cognitive therapy and mindfulness-based stress-reduction intervention both appear to improve depression and anxiety symptoms and reduce overall premenstrual-affective-symptom scores (Bluth, Gaylord, Nguyen, Bunevicius, & Girdler, 2015; Panahi & Faramarzi, 2016). As with SSRI treatment, though, cognitive-behavioral and mindfulness-based interventions have been effective in the treatment of other mood disorders. Thus, it appears that the same psychological treatments are effective for premenstrual affective symptoms and for other mood disorders such as MDD.

As noted earlier, because the menstrual cycle is marked by hormonal fluctuations, it is reasonable to consider whether treatments tied to the cycle and related hormones are effective in reducing premenstrual affective symptoms. There is inconsistent support for the efficacy of oral contraceptives in reducing premenstrual affective symptoms

(Freeman, 2017). A randomized control trial comparing intermittent and continuous dosing of oral contraceptives to placebo found robust reductions in premenstrual affective symptoms in all three groups (Eisenlohr-Moul, Girdler, Johnson, et al., 2017). The placebo condition showed stronger effects than the intermittent dosing group, which is consistent with previous work demonstrating high placebo response rates in premenstrual affective symptoms (Eisenlohr-Moul, Girdler, Johnson, et al., 2017). Despite these mixed findings, oral contraceptives remain a popular treatment for premenstrual affective symptoms. Other ovarian-suppressing treatments, including gonadotropin-releasing hormone agonists and surgical interventions, do appear to reduce premenstrual affective and physical symptoms in most but not all women (Freeman, 2017; Pearlstein, 2016). Removing the menstrual cycle does seems to eliminate affective problems occurring during a certain cycle phase, providing some evidence that these affective symptoms are tied to the cycle.

There appears to be a lack of evidence that confirms specific, distinct treatments for premenstrual affective symptoms and PMDD. In addition, effective interventions for premenstrual affective symptoms seem to be the same interventions that ameliorate general affective and mood symptoms. This evidence detracts from the argument that PMDD is a distinct entity from other mood disorders, and reinforces the need to investigate the underlying validity of a menstrual-cycle-related mood disorder.

Summary of the Problem

The inclusion of PMDD in the DSM-5 has been controversial. Some criticisms focus on the validity of the disorder itself, questioning whether this diagnostic category is socially constructed or based on beliefs and expectations about normative mood

fluctuations. There exists a societal norm that women experience mood and physical problems during the premenstrual phase. Evidence appears to be supportive of physical and affective changes that occur during the premenstrual phase, with approximately 20% of women reporting such problems during the premenstrual phase of their cycle (Freeman, 2017). Psychological factors such as rumination predict premenstrual affective symptoms, and familial studies indicated an intergenerational contribution to premenstrual affective symptoms as well (Craner, Sigmon, Martinson, et al., 2014; Kendler et al., 1998; Sigmon et al., 2009). Additionally, premenstrual affective and physical symptoms remit when suppressing ovulation and the overall menstrual cycle. It appears that some women do experience physical and affective symptoms during a specific phase of their menstrual cycle, and these symptoms lead to distress and possible impairment in functioning.

However, evidence has also emerged that weakens the validity of a *distinct* menstrual-cycle-related mood disorder. PMDD and other mood disorders have high comorbidity, and a history of MDD puts women at higher risk for developing PMDD (Accortt et al., 2013; Cohen et al., 2002). As noted earlier, the etiology of PMDD overlaps with the etiology of MDD and other mood disorders. In addition, it does not appear that there are specific etiological factors tied to the menstrual cycle, such as hormones, that contribute to premenstrual affective symptoms. Many treatments shown effective for reducing premenstrual affective symptoms are also effective in treating general affective symptoms found in other mood disorders. This leads to concerns that premenstrual affective symptoms, labeled PMDD, are better accounted for by MDD. Some women with MDD may misattribute their mood problems to the menstrual cycle as

a result of societal beliefs and expectations. It may also be that during the menstrual cycle, normative physical changes and discomfort exacerbate underlying risk for MDD.

Although affective symptoms that occur during the premenstrual phase appear to be a problem for a small proportion of women, further studies are necessary to detect whether the presence of these symptoms constitutes a disorder separate from MDD. Much of the current literature has not attempted to differentiate women with PMDD from women with MDD. Extant literature also relies on methodology and study designs that undermine conclusions about the disorder. One such example is the widespread use of retrospective report, which relies on accurate reporting of symptoms, cycle phase, and a covariation between the two. This is a difficult task, possibly influenced by social norms and negative beliefs about the menstrual cycle. In addition, when women are aware that premenstrual affective and physical symptoms are the focus of a study, they may experience increased self-monitoring during the premenstrual phase, and in turn, amplify underlying affective symptoms. Thus, although women may experience affective symptoms, they may mistakenly infer a relationship between these symptoms and their menstrual cycle. Studies that minimize these and other methodological flaws could help strengthen the validity of PMDD as a unique diagnosis.

Current Study and Hypotheses

Current Study

This study investigated whether PMDD was a unique diagnosis from MDD. To investigate this question, an attempt was made to confirm the validity of a pattern of mood and affective problems during the luteal phase in women reporting premenstrual dysphoric disorder. A multi-method approach to assessing premenstrual affective

symptoms was used, such that premenstrual affective and physical symptoms were self-reported by participants and measured prospectively using daily mood questionnaires. Diagnostic status was not solely reliant upon retrospective report of a causal relationship between the menstrual cycle and mood symptoms. The study was described as an investigation of impact of mood and the menstrual cycle on sexual behavior. Thus, participants expected to answer questions about their menstrual cycle without paying special attention to premenstrual affective and physical symptoms.

The study then investigated whether differences emerged between women reporting premenstrual affective and physical symptoms and women reporting general depressive symptoms. Using the same daily ratings, I expected to see a different course of affective symptoms between women who retrospectively reported premenstrual affective and physical symptoms and women reporting general depressive symptoms. Additionally, this study investigated whether differences emerged in the psychological factors that predicted general depressive symptoms and premenstrual affective symptoms. Participants answered questions about anxiety sensitivity, rumination, and specific emotion-regulation strategies. The current study examined the relationships between these constructs, premenstrual affective symptoms, and depressive symptoms, to clarify if similarities or differences existed among those relationships.

Hypotheses

The current study explored several hypotheses aimed at clarifying the validity of PMDD and differentiating between PMDD and MDD.

Primary Hypotheses

Hypothesis 1. It was hypothesized that women who reported premenstrual affective and physical symptoms through retrospective report would display a pattern of affective and physical symptoms during the premenstrual phase on prospective daily charting. Specifically, these women would report higher levels of irritability, lower positive affect, and greater negative affect during this cycle phase, compared to nonpremenstrual phases. It was also hypothesized that these affective symptoms would lessen or remit once the menstrual cycle began. Thus, when comparing across all cycle phases, women who reported symptoms consistent with PMDD would also report the greatest levels of affective problems during the luteal phase as compared to other cycle phases.

Hypothesis 2. It was then hypothesized that women who retrospectively reported general depressive symptoms would also show a pattern of affective symptoms using prospective daily charting. This pattern would tie to the menstrual cycle such that those women would report higher levels of irritability, lower positive affect, and greater negative affect during the premenstrual phase.

Exploratory Hypotheses

It was hypothesized that women who reported premenstrual affective and physical symptoms would display a different relationship with two specific psychological factors than women who retrospectively reported general depressive symptoms. It was expected women who reported premenstrual affective and physical symptoms would also report higher levels of engagement in two specific psychological-coping strategies, which in turn would lead to increases in negative mood and affect during the premenstrual phase.

Hypothesis 3. It was hypothesized that women who reported high levels of premenstrual affective and physical symptoms would also report high levels of anxiety sensitivity.

Hypothesis 4. It was then hypothesized that for women reporting premenstrual affective and physical symptoms, anxiety sensitivity would interact with menstrual cycle phase to predict increased levels of stress and affective symptoms during the premenstrual phase. It was predicted that there would be no such interactive effect for women reporting general depressive symptoms.

Hypothesis 5. It was also hypothesized that women who reported high levels of premenstrual affective and physical symptoms would correspondingly report high levels of rumination.

Hypothesis 6. Further, it was hypothesized that for women reporting premenstrual affective and physical symptoms, greater use of rumination at baseline would interact with menstrual cycle phase to predict increased levels of stress and affective symptoms during the premenstrual phase. No such interactive effect was predicted for women reporting general depressive symptoms.

II. Method

Participants

I recruited 173 women between the ages of 18 and 45 for the study from the northeast Ohio and northwest Georgia regions. After completing a screening questionnaire, two women were deemed ineligible for further participation due to pregnancy status and 21 chose not to continue in the study. Women who provided no baseline or daily data did not significantly differ from study participants with regards to age (m = 27.42; sd = 7.98), race, marital status, sexual orientation, or birth control use.

Of the 152 women who continued in the study and completed baseline questionnaires, 145 of those women enrolled in the daily portion of the study. On average, participants completed 17.67 daily surveys (sd = 9.91); any participant who completed less than 12 daily surveys was excluded from further analysis. Another 6 participants were excluded due to a self-reported lack of menstruation. Without this information, luteal phase could not be calculated for further analysis. Data from 94 women, ages 18-44 (m = 26.97; sd = 7.48), were included in analyses.

Eligibility

Pregnant women or those planning to become pregnant in the six months following completion of initial questionnaires were excluded due to the potential disruption to their menstrual cycle during the study. Based upon the increased likelihood of dysregulation of the menstrual cycle and menopause, women over the age of 45 were excluded. Women experiencing amenorrhea for the previous one year were also excluded

from the sample due to the inability to track phases of the menstrual cycle.Of note, birth control use was *not* an exclusionary criterion for this study.

Because hormonal and nonhormonal birth-control methods do not interrupt the menstrual cycle, oral contraceptive use does not prevent women from experiencing hormonal changes that mark the various phases of the cycle. Also, there is a lack of consistent evidence suggesting the efficacy of oral contraceptives in reducing premenstrual symptoms. Additionally, approximately 62% of women of reproductive age use birth control; thus, including women using birth control may result in a more representative sample (Daniels & Abma, 2018; see Table 1).

Procedure

Based on previous research confirming the confounding effects of awareness, efforts were made to minimize participants' knowledge of the true purpose of this study. Though no deception was involved, the study was promoted as an investigation of the relationship among sexual behavior, mood, and the menstrual cycle. The additional collected data will be used to examine unrelated hypotheses.

Using recruitment materials (e.g., flyers), interested women were invited to call or e-mail study staff to complete an initial screening questionnaire to determine eligibility. Study staff assigned identification numbers to interested participants; these were used to link all data. Study staff then sent eligible women a link by e-mail to complete baseline measures. The baseline questionnaire included measures of psychological constructs, sexual functioning and behavior, and premenstrual symptoms. After completing baseline measures, participants completed the daily portion of this study. As noted earlier, though women were aware that the investigators had an interest in the menstrual cycle, attention

was not drawn specifically to premenstrual symptoms in the daily questionnaire. Women were asked to complete daily questionnaires for the following 30 days. Study staff sent daily e-mail reminders to all participants.

Participants were compensated for their time and effort. Participants who completed the full study protocol received $20 for their time. Individuals who partially completed the study protocol were reimbursed using the following system: $.50 for each daily survey completed (up to $15.00 total); and $5.00 for completing baseline questionnaires and screening.

Measures

Eligible women were invited to complete a baseline measure; this measure was composed of several questionnaires, described below.

Baseline Measures

Demographics questionnaire. Women completed a questionnaire that provided demographic information. This information included the following items: age, ethnicity, marital status, sexual orientation, and birth control use (see Appendix A).

Patient Health Questionnaire Somatic, Anxiety, and Depressive Symptom Scales. Women completed the Patient Health Questionnaire Somatic, Anxiety, and Depressive Symptom Scales as part of the baseline measure (PHQ-SADS; Kroenke, Spitzer, Williams, & Löwe, 2010). This composite measure comprises three subscales: the Patient Health Questionnaire—9, the Generalized Anxiety Disorder—7, and the Patient Health Questionnaire—15 (PHQ; see Appendix B). These subscales are intended to assess for depression, anxiety, and somatic symptoms, respectively (Kroenke et al., 2010). The total score of the PHQ-SADS is the sum of scores on each of these measures,

with higher scores indicating greater symptom severity for each instrument. Several researchers have investigated the psychometric validity and reliability of each of these measures.

The PHQ—9 can be used to make probable diagnoses of MDD. Participants are prompted to rate how frequently they experienced symptoms over the past two weeks; for example, "over the last 2 weeks, how often have you been bothered by feeling down, depressed, or hopeless." With regards to the psychometric validity of the instrument, internal consistency has been shown to be adequate, and the measure has been utilized sufficiently to warrant several meta-analyses (range = .86–.89; Kroenke et al., 2010 for review). These studies provide evidence that the PHQ—9 is equal or superior to other brief measures of depression (Henkel et al., 2004; Löwe et al., 2004; Williams, Pignone, Ramirez, & Stellato, 2002). Scores on this instrument range from 0 to 27, with cutoff scores to signify the respondent's symptom severity. Higher scores indicate more severe depression. Cronbach's alpha for this sample was acceptable at .83.

Similarly, researchers developed the GAD—7 to establish probable diagnoses of GAD (Spitzer, Kroenke, Williams, & Löwe, 2006). Scores on this measure can range from 0 to 27, with cutoff scores indicating levels of severity. Higher scores indicate more severe anxiety. Participants respond to questions such as "over the last.2 weeks, how often have you been bothered by worrying too much about different things." Studies have confirmed that internal consistency for this scale falls in the acceptable range, with Cronbach's alpha = .89 (Löwe et al., 2008). Additionally, researchers established convergent validity, as the GAD—7 highly correlates with two other anxiety scales (Kroenke et al., 2010). Internal consistency for the current scale was adequate (α = .924)

The PHQ—15 includes questions about 15 symptoms accounting for more than 90% of somatic symptoms seen in primary care (Kroenke, Spitzer, & Williams, 2002). The measure asks participants to rate how much they have been bothered by each symptom in the past month. Scores range from 0 to 30; cut points represent thresholds for varying symptom severity, with higher scores indicating more severe symptoms. Participants are asked to respond to questions such as "over the last 4 weeks, how often have you been bothered by stomach pain." Convergent validity was established via correlations with other previously validated somatization measures, as well as correlations between higher scores on the PHQ—15 and functional impairment, disability, and health care use (Kroenke et al., 2010). Internal consistency was adequate for this measure (Cronbach's alpha = .79; Interian, Allen, Gara, Escobar, & Díaz-Martínez, 2006). Cronbach's alpha for this sample was .893.

Anxiety sensitivity. The Anxiety Sensitivity Index-3 assesses an individual's fear of arousal-related symptoms (ASI-3; Taylor et al., 2007). Items are scored on a Likert scale ranging from 0 "Not at All" to 4 "Very Much." Higher scores on this measure indicate higher levels of anxiety sensitivity. The measure comprises 18 items, divided into three correlated subscales: Physical Concerns, Cognitive Concerns, and Social Concerns (see Appendix C). Each subscale comprises six items. The subscale Physical Concerns measures an individual's fear of physical harm due to anxious arousal. Items on this subscale include questions such as "when my stomach is upset, I worry that I might be seriously ill." The subscale Cognitive Concerns assesses fear of cognitive or neurological problems due to anxiety symptoms. An example item on this scale is "when my thoughts seem to speed up, I worry that I might be going crazy." The Social Concerns

subscale measures fear of social consequences due to anxious arousal. Questions on this subscale include items such as "I worry that other people will notice my anxiety." The scale's authors found moderate to high subscale correlations (range = .53–.62), and each scale met expected internal-consistency standards (range = .79–.84). This same study investigated and found support for factorial validity of the ASI-3 beyond the original ASI, as well as convergent and divergent validity for each subscale. Another study replicated these estimates of internal consistency, and found the scale to be appropriate for use in nonclinical and clinical samples (Osman et al., 2010). Internal consistency for this sample was Physical concerns, α = .834; Cognitive concerns, α = .887, and Social concerns, α = .795.

Rumination. The Ruminative Responses Scale measures an individual's tendency toward rumination, a method of coping characterized by self-reflection and repetitive negative thought (RRS; Treynor, Gonzalez, & Nolen-Hoeksema, 2003). The scale comprises two dimensions: brooding and reflection (Treynor et al., 2003). The reflection factor assesses a tendency toward introspection and cognitive problems to alleviate one's depressive symptoms. Participants rate how frequently they engage in specific ruminative responses, such as how often they "think about how hard it is to concentrate." Other researchers provided evidence supportive of this two factor structure (Whitmer & Gotlib, 2011). The RRS is a 22-item self-report instrument, with items rated on a scale from 1, denoting *almost never*, to 4, indicating *almost always* (see Appendix D). Higher scores indicate more rumination. Investigators found the internal consistency of the whole scale, as well as each factor, to be in acceptable limits (range = .72–.90; Treynor et al., 2003). This scale has been translated into several languages and validated

cross-culturally. Participants also completed this survey as part of the baseline measure, and Cronbach's alpha for the total score was acceptable at .941.

Premenstrual symptoms. Though the shortcomings of retrospective reporting have previously been discussed, one such measure was used to examine participants' perceptions of their premenstrual symptoms, allowing comparison of results of this screening to participants' daily mood ratings to examine whether beliefs about the temporal relationship between mood and the menstrual cycle match daily mood changes. The Premenstrual Symptoms Screening Tool assesses women's beliefs about the severity and impact of premenstrual symptoms (PSST; Steiner et al., 2003). The PSST is a 19-item instrument, with 14 items evaluating symptom severity and five items measuring symptom impact (Steiner et al., 2003; see Appendix E). Symptom severity questions ask how often participants experience premenstrual symptoms such as "anger/irritability" and "tearfulness/increased sensitivity to rejection." Symptom impact items include questions about the frequency with which symptoms interfere with different parts of life, including "work efficiency or productivity." Higher scores on this measure indicate more premenstrual affective and physical symptoms. The measure appears to accurately categorize women with PMDD as initial pilot testing results were in line with prospective prevalence estimates (Steiner et al., 2003). Internal consistency in this sample was acceptable (α = .906).

Daily Questionnaire

Mood. The Mood and Anxiety Symptom Questionnaire—Short Form was used to assess daily symptomatology of depression and anxiety (MASQ-SF; Watson & Walker, 1996). This instrument is based on the tripartite model, which describes these symptoms

using three dimensions: General Distress, Anhedonic Depression, and Anxious Arousal (Clark & Watson, 1991). General Distress measures general symptoms of psychological distress common to depression and anxiety. Items on this subscale include "felt sad" and "felt discouraged." Anhedonic Depression describes a lack of positive affect and low energy; examples of items on this subscale include "felt cheerful" and "felt really happy." The Anxious Arousal subscale accounts for symptoms of somatic hyperarousal. Items on this subscale include "hands were shaky" and "was short of breath." The initial form of the MASQ has been validated in the literature and shown to have adequate psychometric properties (Watson et al., 1995). Factor analysis of the MASQ confirmed a tripartite model, and the three subscales exhibited low correlations with one another, speaking to the scale's validity (Keogh & Reidy, 2000; Watson et al., 1995). Cronbach's alpha values for each subscale ranged from .88 to .95, indicating high internal consistency (Keogh & Reidy, 2000). Watson and Walker (1996) later designed a shortened form of the MASQ for ease of use. As before, the Anhedonic Depression and Anxious Arousal scales had a low correlation, and internal consistency values ranged from .87 to .94 (Watson & Walker, 1996).

The MASQ-SF consists of 62 items, narrowed from the original 90 items. Due to the daily nature of this study, the authors further eliminated 12 items (see Appendix F). Internal consistency for this sample was General Distress, α = .950; Anxious Arousal, α = .913, and Anhedonic Depression, α = .928. Participants also reported the types of stressors experienced throughout their day including stress related to academics, employment, relationships, and physical health. The MASQ-SF was chosen to measure daily mood symptoms rather than other prospective PMDD measures. Despite consensus

that the DRSP is the best prospective rating scale for PMDD, I chose not to use this measure. The DRSP items specific mention of premenstrual symptoms and the menstrual cycle may produce demand characteristics: participants might respond in ways they believe are desirable to the researcher, thereby producing erroneous results. The lack of overt reference to premenstrual symptoms, as well as the lack of attention drawn to the premenstrual phase, was purposeful. The aim was to prevent beliefs about the menstrual cycle and its relationship with mood and affective symptoms from having an undue influence on participants' daily ratings. The aim was also to minimize the effect of potential methodological issues.

In addition to the mood symptoms listed in the MASQ-SF, the daily measure also included specific irritability questions (see Appendix H). The Brief Irritability Test is a 5-item instrument designed to measure irritability in men and women (BITe; Holtzman, O'Connor, Barata, & Stewart, 2015). Items include "I have been grumpy" and " I have been feeling like I might snap." Items are scored on a Likert scale ranging from 0 "Never" to 6 "Always." Higher scores on this measure indicate more irritability. Psychometric validation of this instrument indicated that the scale items were face valid and had minimal conceptual overlap with depression, anger, and hostility, suggesting good divergent validity. Correlations between the BITe and two longer measures of irritability were high, establishing convergent validity (range =.83–.86). Cronbach's alpha was .88, indicating high internal consistency. The addition of items from other scales did not result in meaningful improvements to the BITe. Thus, though a newer scale, the BITe appears to be a valid and reliable measure of irritability. This construct was included in addition to the MASQ-SF because evidence showed that irritability may

be the main symptom of PMDD (Eriksson, 1999; Ko et al., 2013). Internal consistency was adequate in this sample (α = .948).

Affect. The Discrete Emotions Questionnaire was utilized to measure self-reported discrete emotions (DEQ; Harmon-Jones, Bastian, & Harmon-Jones, 2016). In contrast to other measures that are based on dimensional approaches to emotions, the DEQ uses a discrete-emotions model. The theory posits that individuals hold distinct attitudes to discrete emotions that predict emotion-situation selection (Harmon-Jones, Harmon-Jones, Amodio, & Gable, 2011). Thus, greater dislike of a discrete emotion may lead to increased avoidance of situations arousing that emotion. The DEQ consists of 32 items that load onto eight scales: anger (anger, made, pissed off, rage), disgust (sickened, grossed out, nausea, revulsion), fear (fear, panic, scared, terror), anxiety (worry, dread, nervous, anxiety), sadness (empty, grief, sad, lonely), desire (craving, desire, longing, wanting), relaxed (calm, chilled-out, easygoing, relaxation), and positivity (enjoyment, happy, liking, satisfaction). The scale rates items from 1 = not at all, to 7 = an extreme amount. Higher scores on each subscale indicate participants experienced more of that emotion. Internal consistency of all subscales was adequate (α > .80; Harmon-Jones et al., 2016). Additionally, studies indicated that the DEQ is sensitive to different types of emotional manipulation, and results showed that expected stimuli elevated ratings on each subscale, speaking to the validity of the measure (Harmon-Jones et al., 2016). Internal consistency for this sample was: Anger, α = .919; Disgust, α = .803; Fear, α = .882; Anxiety, α = .855; Sadness, α = .846; Desire, α = .825; Relaxation, α = .880; and Happiness, α = .897.

The final portion of the daily questionnaire consisted of items relating to sexual behavior (see Appendix F). Participants indicated their engagement in specific sexual behaviors such as kissing, sexual intercourse, and masturbation. For example, participants were asked “have you cuddled with a romantic partner today?” Participants also indicated whether they were menstruating during this portion of the questionnaire, which allowed for tracking of the phases of the menstrual cycle.

IV. Results

As the collected data contained repeated measures over time nested in individuals, multilevel linear modeling was used to analyze daily diary data and test interactions between menstrual-cycle phase and symptom report. IBM SPSS Statistics for Windows, version 25.0 (2017) was used for preliminary data analysis and Scientific Software International HLM7 for Windows was used for multilevel linear modeling (Raudenbush, Bryk, Cheong, & Congdon, 2011).

Menstrual-cycle phase was calculated from participants' reports of menstruation. Days on which participants indicated they were menstruating were coded as the menstrual phase. Prior research indicated six methodological options for determining menstrual-cycle subphase, including self-report of onset of menses (Allen et al., 2016). Using self-report, the late luteal or premenstrual phase was the 5 days prior to the start of menses (as in Endicott et al., 2006). Thus, the 5 days prior to first indicated day of menstruation were coded as the luteal phase. All other days were coded as "off" phase. The variables Menstrual, Luteal, and Off, were dummy coded such that 1 indicated phase and 0 indicated not in phase.

Correlations between Level 2 variables were also examined. Premenstrual-symptom severity did not significantly positively correlate with depression, and showed a negative and nonsignificant correlation with anxiety and somatic symptoms. Premenstrual symptoms significantly, positively correlated with rumination levels and anxiety sensitivity levels ($r = .357$, $p < .001$; $r = .445$, $p < .001$).

Power Analysis

An appropriate sample size is needed to have sufficient power to test these hypotheses. A power analysis was conducted according to guidelines suggested by Spybrook et al., (2011). A small effect size was assumed given the limited research on the relationships between these variables (Aguinis, Gottfredson, & Culpepper, 2013). According to these results, a sample size of 100 participants, each with 30 observations over time, was sufficient.

Hypothesis Testing

Preliminary Analyses

In multilevel linear models, the dependent variable is one that changes as a function of two separate processes: time-dependent processes and within-person processes. In this study, the time-dependent process, or Level 1 variable, was menstrual cycle phase, and the person-dependent factor, or Level 2 variable, was severity of premenstrual symptoms. The cycle phase was dummy coded to allow for comparisons between the luteal phase and other phases of the menstrual cycle.

The first step in the multilevel analysis was to run the fully unconditional model in HLM 7, which contained no predictors. The fully unconditional model estimated how much variance in the outcome variables was accounted for at level 2. This model was run four times, one for the main four outcome variables: the three subscales of the MASQ, and irritability. For all four models, there was a significant portion of variance accounted for at Level 2: general distress ($\chi^2(91) = 1560.46$, $p < .001$), anxious arousal ($\chi^2(91) = 3645.85$, $p < .001$), anhedonic depression ($\chi^2(91) = 1375.30$, $p < .001$), and irritability

($\chi^2(91) = 976.45$, $p < .001$). Thus, the nested nature of the data resulted in a significant portion of variance accounted for at Level 2 for all four main outcome variables.

To determine the proportion of variance accounted for at Level 2 in each of the four models, an intraclass correlation coefficient (ICC) was calculated using the following equation:

$$ICC = \frac{\tau_{00}}{\tau_{00} + \sigma^2}$$

$$\sigma^2 = \text{variability within level 1 units}$$

$$\tau_{00} = \text{variability between level 1 units}$$

For general distress, differences between individuals accounted for 46.13% of the variability in this outcome. In the fully unconditional model using anhedonic depression, differences between individuals accounted for 46.96% of the variability in anhedonic depression. The fully unconditional model using anxious arousal as the outcome resulted in an ICC of .4361, or 43.61%. For irritability, differences between individuals accounted for 48.2% of the variability.

Next, a model was run with only one Level 2 predictor to examine the main effects of that predictor on the desired outcomes. This was done first using premenstrual-symptom severity and then using depressive-symptom severity as the predictors. The main effects of premenstrual-symptoms severity and general depressive-symptom severity (see Tables 4 through 27). A significant main effect of premenstrual-symptom severity emerged on general distress such that when women reported higher levels of premenstrual affective and physical symptoms, they also reported higher levels of general distress across all phases ($\beta_{01} = 3.54$, $p < .001$). The main effect of premenstrual-symptom severity on anxious arousal was also significant, with higher levels of

premenstrual symptoms predicting higher levels of anxious arousal (β_{01} = 1.47, p < .001). This main effect was also significant when irritability was used as the outcome variable, with more severe premenstrual symptoms predicting higher levels of irritability (β_{01} = .193, p = .009). No such significant effect emerged for anhedonic depression (β_{01} = -1.87, p = .051). The main effect of premenstrual-symptom severity was also significant in predicting anger (β_{01} = -.692, p = .016). This main effect was significant when disgust was the outcome variable (β_{01} = 0.609, p = .004).

A significant main effect emerged of premenstrual symptoms on anxiety, as well as on sadness (β_{01} = 1.209, p < .001; β_{01} = 0.928, p = .002). The main effect of premenstrual-symptom severity was also significant for desire (β_{01} = 1.135, p < .001). This main effect was not significant when the DEQ subscale of fear was entered as the outcome variable (β_{01} = 0.465, p = .067). The main effect was also not significant for relaxation or happiness (β_{01} = -0.101, p = .757; β_{01} = -0.062, p = .862). When women reported higher levels of premenstrual affective and physical symptoms, they also reported higher levels of general distress, anxious arousal, anger, disgust, anxiety, sadness, and desire, irrespective of menstrual-cycle phase.

The main effect of depressive-symptom severity on general distress was not significant (β_{01} = .198, p = .34). The main effect of depressive symptom severity was also not significant in predicting anhedonic depression or anxious arousal (β_{01} = .089, p = .661; β_{01} = -.006, p = .945). When irritability was used as the outcome variable, the main effect of depressive symptoms was also not significant (β_{01} = .013, p = .3961). The main effect of depressive-symptom severity was not significant for any of the eight DEQ subscales.

Primary Analyses

The first hypothesis stated that the interaction between cycle phase and premenstrual symptoms would predict daily affective symptoms such that the effect of cycle phase on affective symptoms would be highest when women reported more severe premenstrual affective and physical symptoms. A series of hierarchical linear regressions was used to test whether the relationship between two Level 1 variables—affect and cycle phase—changed as a function of a Level 2 variable, premenstrual-symptom severity. This moderation was tested using a cross-level interaction (Raudenbush & Bryk, 2002).

To test this hypothesis, a series of moderation analyses was conducted using premenstrual-symptom severity, measured by the PSST, as a moderator of the relationship between cycle phase and affective symptoms. The dependent variables were affective symptoms, measured by the three subscales of the MASQ-SF, the eight DEQ subscales, and the BITe measure. A separate analysis was conducted for each separate subtype of affective symptoms: General Distress, Anhedonic Depression, Anxious Arousal, Anger, Disgust, Fear, Anxiety, Sadness, Desire, Relaxation, Happiness, and Irritability (see Tables 28 through 39).

First, all variables were entered into HLM 7, and identified as either Level 1 or Level 2 variables. All data were linked using previously assigned identification numbers. The dependent variable of General Distress subscale was then entered as the outcome variable. The independent variable of luteal cycle phase was grand-mean centered and entered into the Level 1 model, represented by the following equation:

$$GenDistress_{ti} = \pi_{0i} + \pi_{1i}(Luteal) + r_{ij}$$

The moderator variable of premenstrual-symptom severity was also grand-mean centered and entered into the Level 2 model, represented by the following equations:

$$\pi_{0i} = \beta_{00} + \beta_{01}(PSST) + \mu_{0i}$$

$$\pi_{1i} = \beta_{10} + \beta_{11}(PSST)$$

The interaction term is then calculated as the Level 2 equations are substituted into the Level 1 model, represented by the following equation:

$$GenDistress_{ij} = \beta_{00} + \beta_{10}(Luteal) + \beta_{01}(PSST) + \beta_{11}(PSST * Luteal) + u_{0j} + r_{ij}$$

u_{0j}represents the random effect varying across participants.

β_{00} represents the intercept, or average score for the dependent variable, general distress.

β_{10} represents the slope for the cycle-phase predictor.

β_{01} represents the slope for the diagnostic-status predictor.

β_{11} represents the slope for the interaction term between diagnostic status and cycle phase.

r_{ij} represents the residual, or random error, associated with participant j's i'th general-distress score.

The software created the interaction term between premenstrual-symptom severity and luteal-cycle phase, which was then entered as a predictor into this equation. A statistically significant coefficient for the interaction term indicates moderation. Similar equations were created for the remaining outcome variables.

The first hypothesis predicted that premenstrual-symptom severity would interact with cycle phase to predict affective symptoms. When general distress was entered as the outcome variable, the interaction was not significant (β_{11} = 0.578, p = .168). When anhedonic depression was entered as the outcome variable, this interaction was

significant (β_{11} = 0.911, p = .031). This effect was not in the expected direction, such that when women reported more severe premenstrual symptoms, they also reported more positive emotions during the luteal phase. When the anxious arousal subscale was the outcome variable, the interaction between luteal phase and premenstrual symptoms was not significant (β_{11} = -0.09, p = .556). Using irritability as the outcome variable, the phase and symptom interaction was not significant (β_{11} = -0.012, p = .737). Two significant interactions emerged when predicting the DEQ subscales, specifically the happiness subscale (β_{11} = 0.477, p = .004) and the relaxation subscale (β_{11} = 0.428, p = .009). Women reporting higher levels of premenstrual symptoms appeared happier, more relaxed, and had more positive emotions during the luteal phase. The interaction between premenstrual-symptom severity and luteal phase was not significant in predicting the remaining six DEQ subscales of Anger, Disgust, Fear, Anxiety, Sadness, and Desire.

The second hypothesis stated that there would be an interaction would emerge between depressive symptoms and cycle phase in predicting daily affect. It was hypothesized that cycle phase would have larger effects on daily affect for women with overall higher levels of depressive symptoms. The same moderation analysis discussed above were repeated with depressive symptoms as the independent variable. Premenstrual, or luteal, phase continued to serve as the moderator variable. Again, affective symptoms, specifically the three MASQ subscales, irritability, and eight DEQ subscales, served as the dependent variables. Using general distress as the first outcome variable, a significant interaction emerged between depressive symptoms and luteal phase (β_{11} = 0.236, p = .007). Those women reporting higher levels of depressive symptoms also reported greater general distress specifically during the luteal phase. No significant

interaction arose between depressive symptomatology and luteal phase in predicting anhedonic depression (β_{11} = -.057, p = 0.514). This interaction was also not significant in predicting anxious arousal or irritability (β_{11} = 0.054, p = .105; β_{11} = 0.013, p = .07). The interaction between depressive symptoms and the luteal phase was significant when using the DEQ subscale of Fear (β_{11} = 0.056, p = .016). Higher levels of general depressive symptoms were associated with higher levels of fear during the luteal phase, relative to the other cycle phases. However, a significant interaction between depressive symptoms and luteal phase did not emerge when the remaining seven DEQ subscales of Anger, Disgust, Anxiety, Sadness, Desire, Relaxation, and Happiness, were the dependent variables.

Exploratory Subgroup Analyses

The following analyses were used to examine the hypothesized relationships between psychological constructs and daily affect in women who reported high levels of depressive symptoms and women who reported high levels of premenstrual affective symptoms. These analyses were performed, first, looking only at women who specifically reported elevations in premenstrual symptoms, and then repeated by examining women who specifically reported elevations in depressive symptoms.

The PSST was used to measure premenstrual-symptom severity with total scores of 3 or higher, considered indicative of severe premenstrual symptoms. Using this cutoff, 20 women were included in the premenstrual-symptom subgroup analyses. Symptoms of depression were measured using the PHQ-9; this measure uses cut points to categorize scores. Women whose scores fell into the categories of moderately severe or severe on

the PHQ-9 were included in the depressive-symptom subgroup analyses. Using this cutoff, 13 women were included in the depressive symptom subgroup analyses.

Two separate *t*-tests were conducted to test the third hypothesis. This hypothesis stated that women who reported more severe premenstrual symptoms would report higher levels of anxiety sensitivity than women reporting minimal symptoms. It was also hypothesized that women who reported more severe depressive symptoms would also report higher levels of anxiety sensitivity than women reporting minimal symptoms. An independent-samples *t*-test was conducted to compare mean anxiety-sensitivity scores for those reporting severe premenstrual symptoms and those reporting minimal symptoms (see Table 52). Another independent-samples *t*-test was conducted to compare mean anxiety-sensitivity scores between women reporting higher levels of general depressive symptoms and women reporting minimal symptoms (see Table 53). Women reporting more severe premenstrual symptoms had higher anxiety-sensitivity scores (M = 35.15, SD = 15.78) than did those women reporting minimal symptoms (M = 19.67, SD = 12.26; $t(91) = 4.691$, $p < .001$). Women reporting higher levels of general depressive symptoms did not have significantly different anxiety-sensitivity scores (M = 23.77, SD = 12.36) than women reporting minimal symptoms (M = 23.04, SD = 14.90; $t(90) = .168$, $p = .867$).

The fourth hypothesis stated that in the subgroup of women reporting more severe premenstrual symptoms, an interaction would emerge between anxiety sensitivity and menstrual cycle phase to predict affective-symptom severity. As anxiety sensitivity is a Level 2 variable and affective symptoms and cycle phase are time dependent, a multilevel model was used to test the cross-level interaction. Another series of moderation analyses

was utilized to test this; this procedure followed the process for testing the cross-level interactions discussed above.

For women reporting more severe premenstrual symptoms, anxiety sensitivity interacted with luteal phase to predict higher levels of general distress (β_{11} = 0.289, p = .01). This interaction was not significant when predicting anxious arousal or anhedonic depression (β_{11} = .081, p = .088; β_{11} = -0.114, p = .241). The interaction between luteal phase and anxiety sensitivity was also nonsignificant when predicting irritability (β_{11} = .017; p = .055). I also examined the phase and anxiety-sensitivity interaction for the DEQ subscales; this interaction was significant when predicting anger (β_{11} = 0.102, p = .007). The interaction was also significant when the Anxiety subscale was the outcome variables (β_{11} = 0.089; p = .032). This interaction was not significant when the remaining six DEQ subscales of Disgust, Fear, Sadness, Desire, Relaxation, or Happiness, were the outcome variables. Women with more severe premenstrual symptoms and higher levels of anxiety sensitivity experienced more general distress, anger, and anxiety, specifically during the luteal phase.

An additional set of moderation analyses were performed to examine the relationships between anxiety sensitivity and menstrual-cycle phases in the subgroup of women reporting higher levels of depressive symptoms. Anxiety sensitivity was a Level 2 variable and a cross-level interaction was used to test for moderation using the previously described series of moderation analyses. A statistically significant coefficient for the interaction term indicated moderation.

For women reporting more severe depressive symptoms, the interaction between anxiety sensitivity and luteal phase significantly predicted the DEQ subscale of Disgust

(β_{11} = 0.077, p = .025). Women who reported severe depressive symptoms and high levels of anxiety sensitivity experienced more disgust during the luteal phase relative to the other cycle phases. However, this interaction between anxiety sensitivity and luteal phase was not significant in predicting other measures of negative affect, including irritability, anger, fear, anxiety, sadness, or the MASQ subscales of general distress and anxious arousal. The interaction between anxiety-sensitivity level and luteal phase was also not significant in predicting changes in positive affect, measured by the DEQ subscales of Desire, Happiness, or Relaxation, or the MASQ subscale of Anhedonic Depression.

The fifth hypothesis stated that women with more severe premenstrual symptoms would also report higher levels of rumination than women reporting minimal symptoms. It was also hypothesized that women reporting higher levels of general depressive symptoms would experience higher levels of rumination than women with minimal symptoms. Separate *t*-tests were conducted to test these hypotheses. An independent-samples *t*-test was conducted to compare mean rumination levels for those reporting severe premenstrual symptoms and those reporting minimal symptoms. Another independent-samples *t*-test was conducted to compare mean rumination scores between women reporting higher levels of general depressive symptoms and women reporting minimal symptoms. Women reporting more severe premenstrual symptoms had higher rumination scores (M = 58.25, SD = 17.84) than did those women reporting minimal symptoms (M = 49.77, SD = 13.06; $t(92) = 2.372$, p = .020). Women reporting higher levels of general depressive symptoms did not have significantly different rumination

scores (M = 50.92, SD = 17.77) than did those women reporting minimal symptoms (M = 51.99, SD = 13.88; $t(91)$ = -.246, p =.806).

Hypothesis 6 predicted that the subgroup of women reporting higher levels of premenstrual symptoms would experience an interaction between rumination and menstrual-cycle phase in predicting affective-symptom severity. As previously described, rumination was a Level 2 variable, and a cross-level interaction was used to test for moderation through the previously described series of moderation analyses. A statistically significant coefficient for the interaction term indicated moderation. These analyses were performed using the aforementioned subsample of women reporting severe premenstrual symptoms.

In the subgroup of women reporting more severe premenstrual symptoms, the interaction between rumination and luteal phase significantly predicted general distress (β_{11} = 0.375, p < .001). This interaction was also significant when predicting anxious arousal (β_{11} = 0.115, p = .002). Both effects were in the expected direction; thus, women with severe premenstrual symptoms and higher levels of rumination experienced more general distress and anxious arousal during the luteal phase compared to the other cycle phases. The luteal phase and rumination interaction was also significant in predicting anhedonic depression (β_{11} = -0.275, p < .001). This interaction was in the expected direction, as the rumination and phase interaction predicted lower levels of positive emotions during the luteal phase for women with severe premenstrual symptoms. For these same women, phase and rumination also interacted to predict higher levels of irritability (β_{11} = 0.021, p = .003). Women with high levels of premenstrual symptoms who also ruminate more are also more irritable during the luteal phase. This interaction

also significantly predicted anger (β_{11} = 0.113, p < .001). When disgust was entered as the dependent variable, the interaction term was significant (β_{11} = 0.066, p = .003). The interaction term was also significant when fear was the dependent variable (β_{11} = 0.054, p = .027). The luteal phase and rumination interaction was significant when predicting anxiety and when predicting sadness (β_{11} = 0.086, p = .008; β_{11} = 0.075, p = .007). Women with more severe premenstrual symptoms who also ruminate more experienced elevations in disgust, anger, fear, anxiety, and sadness, specifically during the luteal phase. The phase and rumination interaction was also significant when predicting happiness (β_{11} = -0.077, p = .006); women reported less happiness during the luteal phase. The interaction between rumination and the luteal phase was not significant when the DEQ subscales of Desire or Relaxation were the outcome variables.

An additional set of moderation analyses were performed to examine the relationships between rumination and menstrual-cycle phases in the subgroup of women reporting higher levels of depressive symptoms. Rumination was considered a Level 2 variable and a cross-level interaction was used to test for moderation using the previously described series of moderation analyses. A statistically significant coefficient for the interaction term indicated moderation.

For women reporting more severe depressive symptoms, the interaction between rumination and luteal phase significantly predicted general distress (β_{11} = 0.3122, p = .002). Rumination also interacted with luteal phase to predict higher levels of anhedonic depression (β_{11} = -0.203, p =.042). In this subset of the sample, a significant interaction emerged between rumination and phase in predicting anger during the luteal phase (β_{11} = 0.069, p =.02). The interaction was also significant in predicting disgust

(β_{11} = 0.060, p =.008). The rumination and phase interaction was nonsignificant when anxious arousal was the dependent variable (β_{11} = 0.068, p =.091). This interaction was also not significant when predicting irritability (β_{11} = 0.014, p =.084). This interaction was also not significant when the DEQ subscales of Fear, Anxiety, Sadness, Desire, Relaxation, or Happiness, were the outcome variables. Women who reported more depressive symptoms and higher levels of rumination experienced higher levels of general distress, anger, and disgust, specifically during the luteal phase. These women also experienced fewer positive emotions during the luteal phase relative to the other cycle phases.

V. Discussion

PMDD is believed to be a cyclical pattern of mood and affective symptoms associated with the premenstrual phase of the menstrual cycle. The characterization of this disorder as a valid entity distinct from other depressive disorders, such as MDD, has been met with criticism. Support for a separate, menstrual-cycle-specific mood disorder could be drawn from evidence demonstrating replicable differences in biology, symptomatology, course, or psychosocial antecedents of PMDD and MDD. In the current study, an attempt was made first to validate the presence of a pattern of mood and affective problems specifically during the luteal phase in women who reported having severe premenstrual problems. This process was followed by an examination of potential differences between women reporting severe premenstrual symptoms and women reporting more general depressive symptoms. In the same daily mood ratings, different patterns of affective symptoms were expected for women with premenstrual symptoms and for women reporting more general depressive symptoms. Finally, two potential etiological factors were hypothesized to predict high levels of premenstrual affective symptoms.

Based on a preliminary examination of the main effects of self-reported PMDD symptoms, data from this study demonstrated that higher levels of self-reported PMDD symptoms were also associated with elevated levels of negative affect, regardless of menstrual-cycle phase. In comparison, the evidence did not support a general association between higher levels of self-reported PMDD symptoms and positive affect. Thus,

women reporting elevations on a measure of PMDD symptoms appeared to have general difficulties with negative affect unrelated to the menstrual cycle.

Results from this study also indicated that women who reported higher levels of PMDD symptoms did display a cyclical pattern of affective changes. Notably, the pattern of changes contradicted the cyclical pattern defining PMDD. For example, during the luteal phase, women reporting higher scores on a measure of PMDD did not report higher levels of negative affect; instead, higher levels of self-reported PMDD were associated with increased positive affect. In other words, women who scored higher on a measure of PMDD did not show corresponding evidence of premenstrual elevations in negative affect on daily diary measures.

Though these findings were not initially hypothesized, the lack of evidence relating to cyclical changes in negative affect is consistent with some current literature that does not support cyclical negative mood changes in the premenstrual phase (Romans et al., 2012, 2013). Although this pattern of menstrual-cycle related changes in positive affect was unexpected and contrary to the theoretical construct of PMDD, the extant literature provides limited support for premenstrual changes in positive affect (King & Ussher, 2013; Kues et al., 2018; Welz et al., 2016). The pattern of results found in this study may provide evidence to support criticisms of the incremental validity of a specific premenstrual mood disorder.

One potential explanation for these contradictory findings is that current measures of premenstrual affective changes and PMDD are not valid or reliable. It is possible that current assessment tools do not accurately measure the construct of PMDD, in turn allowing for women who are experiencing general affective changes to be miscategorized

as having symptoms only during the luteal phase. This could explain why some women who retrospectively report symptoms of PMDD did not display evidence of negative affect specifically during the luteal phase but did display *overall* negative affect. Indeed, there have been several criticisms aimed at the most frequently used assessments of PMDD (Accortt et al., 2011; Bosman et al., 2016; Haywood et al., 2002; Steiner et al., 2003). It is possible that the hypothesized effects did not arise as expected due to the lack of reliable and valid measurement tools available to measure this construct. Further studies focusing on the assessment of menstrual-cycle-related affective and physical symptoms would have scientific and clinical benefits. More accurate measurement of these symptoms could improve future research in this area.

Another explanation for this unanticipated effect is that women may expect to see a relationship between general depressive symptoms and their menstrual cycle, in part due to the influence of societal expectations of the premenstrual phase. The menstrual cycle may be a natural target when seeking causes of affective problems or symptoms of depression due to the prevalent negative connotations of menstruation. It is possible that women who are already experiencing problems with mood have culturally bound expectations of the premenstrual period, and in retrospect, selectively attend to confirmatory information that maintains this belief. Women expect to see a relationship between their mood problems and their menstrual cycle, even when no such relationship exists. This notion supports other research that calls into question a normative, cyclical pattern of premenstrual distress and mood dysregulation, consistent with feminist theories that PMDD is a medicalization of normative physical processes (Browne, 2015; Cosgrove & Caplan, 2004; Romans et al., 2012). Thus, due to inaccurate societal beliefs,

women may expect to experience premenstrual mood changes and mistakenly attribute distress to this process. Additionally, a belief that the menstrual cycle and premenstrual phase are inherently negative experiences may shape women's expectations such that they minimize or ignore positive changes. Despite extant literature indicative of the presence of positive changes associated with the menstrual cycle, women may be primed to notice negative affect but not changes in positive affect. In turn, women do not attribute these changes in positive affect to the menstrual cycle or the premenstrual phase.

The effect of menstrual-cycle phase on patterns of daily affect was also examined for women reporting higher levels of depressive symptoms in general. Unexpectedly, women who reported high levels of general depressive symptoms did show a pattern of changes in negative affect related to the menstrual cycle. During the luteal phase, women reporting elevated levels of general depressive symptoms experienced increases in negative affect. Thus, women who self-reported high levels of general depressive symptoms also reported a cyclical pattern of increased negative affect during the luteal phase on daily affect measures.

In sum, data from retrospective self-report was inconsistent with patterns shown on measures of daily affect. It was originally hypothesized that women with high scores on a measure of PMDD would show a pattern of daily negative affect consisting of (a) minimal to no elevations prior to the luteal phase, (b) a clinically significant increase during the luteal phase, and (c) a return to baseline levels after the luteal phase. The blue line in Figure 1 illustrates this hypothetical curve. However, for women who self-reported higher levels of PMDD symptoms, the observed pattern of daily affect contradicted retrospective report. Additionally, some women did display a cyclical pattern of negative

affect that peaked during the luteal phase. However, this pattern was found for women who reported significant symptoms of general depression and not PMDD.

It is possible that some women who experience depressive symptomatology also experienced increases in those symptoms during the luteal phase of their cycle. These women may not have identified themselves as having a menstrual-cycle-related mood disorder, possibly because their symptoms were not restricted to the premenstrual phase. This is consistent with previous research where the menstrual cycle has been implicated as a physical stressor associated with a worsening of a variety of psychological disorders (Hartlage, Brandenburg, & Kravitz, 2004; Kornstein, 2010; Kornstein et al., 2005; Pinkerton, Guico-Pabia, & Taylor, 2010). The black line in Figure 1 illustrates a pattern of premenstrual worsening of underlying depressive symptoms. Although this outcome suggests the presence of a relationship between the menstrual cycle and affective problems, it is not consistent with the current conceptualization of PMDD as demonstrated by the blue line in Figure 1. As depicted by this theoretical curve, there is a lack of clinically significant depressive symptoms until the luteal phase, at which time depressive symptoms increase; these symptoms resolve after the end of the luteal phase. The absence of clinically significant symptoms outside of the luteal phase is central to the construct. Therefore, as PMDD is marked by affective symptoms which are "not merely an exacerbation of the symptoms of another disorder, such as major depressive disorder" (APA, 2013, p. 175), the increase of underlying negative affect during the luteal phase is not indicative of PMDD, but rather a premenstrual worsening of another disorder. Indeed, the present investigation did not find the presence of cyclical, premenstrual phase

specific, affective changes that occur separately from the experience of more general affective problems.

Though the initial goal of this study was to investigate potential phenomenological differences between PMDD and MDD, measures of rumination and anxiety sensitivity were included to assess potential etiological factors. Although the hypothesized patterns of PMDD did not emerge, relationships among daily affect, anxiety sensitivity, and rumination, were still examined. In the current literature, both anxiety sensitivity and rumination have been previously implicated as etiological factors for PMDD (Sigmon, Dorhofer, Rohan, & Boulard, 2000; Sigmon, Rohan, Boulard, Dorhofer, & Whitcomb, 2000). The menstrual reactivity hypothesis posited that some women respond using cognitive strategies such as rumination, as well as actual experiences of physical symptoms and cultural expectations, to interpret bodily changes in a particularly negative manner. This may lead to distress and increased negative affect during the premenstrual phase.

In this study, high scores on a measure of PMDD were positively associated with rumination and with anxiety sensitivity. These results are consistent with current literature, which is indicative of a significant positive relationship between PMDD symptoms and anxiety sensitivity, as well as PMDD symptoms and rumination (Craner et al., 2013; Craner, Sigmon, Martinson, et al., 2014; Sigmon, Dorhofer, et al., 2000). In contrast to previous research, high levels of self-reported general depressive symptoms were not significantly associated with either rumination or anxiety sensitivity in this study (e.g., Allan et al., 2014; McLaughlin & Nolen-Hoeksema, 2011; Naragon-Gainey, 2010; Nolen-Hoeksema, 2000; Olthuis, Watt, & Stewart, 2014). These unexpected results

should be interpreted with caution in light of the discrepancy with extant literature, and should be reexamined in a larger, more representative sample, to see if they can be replicated.

It was further hypothesized that anxiety sensitivity and rumination would each moderate the relationship between cycle phase and daily affect. In the subset of women reporting high levels of PMDD symptoms, results supported this hypothesis. A relationship emerged between cycle phase and high levels of anxiety sensitivity in predicting cyclical increases in negative affect during the luteal phase. This effect also arose when examining rumination; high levels of rumination predicted increased negative affect during the luteal phase. However, a similar patterns of results emerged when examining these associations in the subset of participants reporting moderate to severe symptoms of general depression. For women reporting higher levels of general depressive symptoms, higher levels of anxiety sensitivity and rumination again predicted cyclical increases in negative affect during the luteal phase.

These results suggest that rumination and anxiety sensitivity are each important cognitive vulnerability factors that contribute to women's experiences of negative affect throughout the menstrual cycle. These findings are consistent with extant literature indicative of interactive effects among anxiety sensitivity, rumination, and menstrual-cycle phase for women reporting PMDD symptoms (Craner, Sigmon, Martinson, et al., 2014; Nillni et al., 2013). It is not yet clear whether these constructs are underlying vulnerability factors for affective symptoms, or whether women use these cognitive strategies to respond to symptoms that are already present.

Importantly, this study failed to find evidence of different effects of anxiety sensitivity and rumination between those reporting PMDD and those reporting general depressive symptoms. Because PMDD and MDD are phenomenologically similar, it is possible that the two disorders overlap in these specific etiological factors. Anxiety sensitivity and rumination were specifically chosen based on evidence from current literature, but many other psychological constructs that could help clarify a distinction between PMDD and other mood disorders. It is possible that etiological differences between MDD and PMDD, whether biological or psychological, have not yet been discovered. It is also possible that anxiety sensitivity and rumination contributed to similar patterns of symptoms because PMDD and MDD are not distinct constructs. Evidence from other studies suggests that the same negative cognitive styles are present for those reporting symptoms of PMDD and for those reporting symptoms of MDD (Śliwerski & Bielawska-Batorowicz, 2018). Findings of similar effects of both constructs in both groups may be a result of the significant overlap between the two groups. These results did not provide evidence that justifies the discriminant validity of a premenstrual mood disorder. Given these findings, as well as the lack of evidence for menstrual-cycle-related mood changes unrelated to underlying depressive symptoms, the experience of menstrual-cycle-related mood problems *may* be more parsimoniously attributed to a cyclical worsening of preexisting depressive symptoms and not to a separate premenstrual disorder.

If PMDD is not a distinct construct from MDD, it is possible that women who are experiencing significant affective problems are being misdiagnosed and in turn, not receiving appropriate treatment for their symptoms. The current conceptualization of a

menstrual-cycle-related mood disorder may lead to an underestimation of the breadth and severity of women's affective problems. The presence and impact of symptoms outside of the luteal phase may be discounted by providers. Additionally, attributing the cause of affective symptoms to the menstrual cycle may lead to poorer or ineffective treatment, in turn, prolonging women's symptoms. For example, a focus on a relationship between the menstrual cycle and negative affect might also result in a lack of consideration of the other factors that can have a distinct impact on mood.

In contrast, if future evidence is supportive of a distinction between PMDD and MDD, this may underscore the clinical relevance of the menstrual cycle and the premenstrual phase to women's mental health. Evidence of this distinction and the impact of reproductive functions on mental health could help to legitimize the concerns of women who have been suffering from significant mood problems. Understanding the relationship between reproductive and mental health could also be beneficial for mental health care across a woman's lifespan.

Additionally, if PMDD and MDD are not the same disorder, the current evidence may highlight the importance of developing better methods for detecting and differentiating symptoms of PMDD from those of other disorders. Improving upon the ability to identify PMDD will allow for better treatment of women who suffer from premenstrual affective problems. Assessments that combine reliability, validity, and efficiency can then be developed so those women with premenstrual and other affective problems can receive adequate and appropriate mental health care.

Limitations

Some limitations of the current research should be noted. Some of the results from this study, such as the patterns of premenstrual mood exacerbations, are in keeping with current literature. Others results were inconsistent with current research and with prevailing theory regarding PMDD. Thus, these results should be interpreted with caution and reexamined in a larger sample to see if they can be replicated.

Other limitations of this study include the lack of precise measurements for each participant's menstrual-cycle phases. Though I measured cycle phase according to current methodological standards, actual ovarian hormone levels were unavailable. Thus, measures of cycle phase may not be accurate for each participant; future studies could include hormone measures for a more exact measurement. It could also be beneficial to assess mood changes over two or more cycles to be able to assess the stability of mood fluctuations across phases and to measure premenstrual affective and physical symptoms prospectively. Although these improvements may prove beneficial, they may not be practical or feasible for researchers. Additionally, these methods may not be ecologically valid, given current research on assessment of PMDD in clinical practice (Craner, Sigmon, & McGillicuddy, 2014).

Another limitation could be the chosen age range of the sample (18–44 years). Although all women who participated were premenopausal, hormone profiles, and thus, experiences of affective symptoms, may have varied by age. Additionally, though the statistical models in this study were based on more than 2,200 observations, due to the longitudinal design of this study, data came from only 94 women. Future investigations should include larger samples, as the small sample size may have resulted in limited

statistical power to detect some meaningful differences. A larger and more representative sample would also enhance the generalizability of findings. Further, exploratory analyses were examined only after dichotomizing the variables measuring PMDD symptoms and MDD symptoms. Dichotomizing this data might have resulted in a loss of information and a loss in power to detect significant differences. The resulting sample sizes for each group (n = 13 and 20, respectively) might also have contributed to reduced statistical power.

Finally, each hypothesis in this study was tested using multiple dependent variables as the outcome. The use of multiple tests increases the likelihood of committing a Type I error; this may be referred to as alpha inflation. A Type I error, or a false positive, occurs if the null is rejected when the null is true. Thus, alpha inflation may lead to an increased probability of a false positive result. However, evidence from this study did not support the main hypotheses; the null hypothesis was not rejected despite the use of multiple dependent variables. The lack of evidence for rejecting the null indicates that concerns committing a Type I error may not be relevant to these analyses. Due to the exploratory nature of the remaining hypotheses, the criterion for alpha was not adjusted.

Conclusion

The results of this study did not show evidence in support of the current conceptualization of PMDD as a disorder separate from MDD. Indeed, the hypothesized differences in the course of the disorder and etiology did not emerge. However, results suggested that some women do experience exacerbations of underlying affective symptoms that temporally relate to their menstrual cycles. In addition, the presence of specific cognitive vulnerabilities may play a role in some women's experiences of these

exacerbations. Future research on the differences between PMDD and other mood and anxiety disorders could help provide further clarity regarding the presence of a distinct, menstrual-cycle-related psychiatric disorder.

REFERENCES

Accortt, E. E., Bismark, A., Schneider, T. R., & Allen, J. J. (2011). Diagnosing premenstrual dysphoric disorder: The reliability of a structured clinical interview. *Archives of Women's Mental Health, 14,* 265–267. doi:10.1007/s00737-011-0209-3

Accortt, E. E., Kogan, A. V., & Allen, J. J. (2013). Personal history of major depression may put women at risk for premenstrual dysphoric symptomatology. *Journal of Affective Disorders, 150,* 1234–1237. doi:10.1016/j.jad.2013.05.091

Aguinis, H., Gottfredson, R. K., & Culpepper, S. A. (2013). Best-practice recommendations for estimating cross-level interaction effects using multilevel modeling. *Journal of Management, 39,* 1490–1528. doi:10.1177/0149206313478188

Allan, N. P., Macatee, R. J., Norr, A. M., & Schmidt, N. B. (2014). Direct and interactive effects of distress tolerance and anxiety sensitivity on generalized anxiety and depression. *Cognitive Therapy and Research, 38,* 530–540. doi:10.1007/s10608-014-9623-y

Allen, A. M., McRae-Clark, A. L., Carlson, S., Saladin, M. E., Gray, K. M., Wetherington, C. L., … Allen, S. S. (2016). Determining menstrual phase in human biobehavioral research: A review with recommendations. *Experimental and Clinical Psychopharmacology, 24,* 1–11. doi:10.1037/pha0000057

American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). Arlington, VA: Author.

Amiel Castro, R. T., Pataky, E. A., & Ehlert, U. (2018). Associations between premenstrual syndrome and postpartum depression: A systematic literature review. *Biological Psychology,* Art. 107612. doi:10.1016.j.biopsycho.2018.10 .014

AuBuchon, P. G., & Calhoun, K. S. (1985). Menstrual cycle symptomatology: The role of social expectancy and experimental demand characteristics. *Psychosomatic Medicine, 47,* 35–45. doi:10.1097/00006842-198501000-00004

Bluth, K., Gaylord, S., Nguyen, K., Bunevicius, A., & Girdler, S. (2015). Mindfulness-based stress reduction as a promising intervention for amelioration of premenstrual dysphoric disorder symptoms. *Mindfulness, 6,* 1292–1302. doi:10 .1007/s12671-015-0397-4

Bosman, R. C., Jung, S. E., Miloserdov, K., Schoevers, R. A., & aan het Rot, M. (2016). Daily symptom ratings for studying premenstrual dysphoric disorder: A review. *Journal of Affective Disorders, 189,* 43–53. doi:10.1016/j.jad.2015.08.063

Browne, T. K. (2015). Is premenstrual dysphoric disorder really a disorder? *Journal of Bioethical Inquiry, 12*(2), 313–330. doi:10.1007/s11673-014-9567-7

Callaghan, G. M., Chacon, C., Coles, C., Botts, J., & Laraway, S. (2009). An empirical evaluation of the diagnostic criteria for premenstrual dysphoric disorder: Problems with sex specificity and validity. *Women & Therapy, 32,* 1–21. doi:10 .1080/02703140802402396

Chrisler, J. C., & Caplan, P. (2002). The strange case of Dr. Jekyll and Ms. Hyde: How PMS became a cultural phenomenon and a psychiatric disorder. *Annual Review of Sex Research, 13,* 274–306. doi:10.1080/10532528.2002.10559807

Chrisler, J. C., Rose, J. G., Dutch, S. E., Sklarsky, K. G., & Grant, M. C. (2006). The PMS illusion: Social cognition maintains social construction. *Sex Roles, 54,* 371–376. doi:10.1007/s11199-006-9005-3

Clark, L. A., & Watson, D. (1991). Tripartite model of anxiety and depression: Psychometric evidence and taxonomic implications. *Journal of Abnormal Psychology, 100,* 316–336. doi:10.1037/0021-843X.100.3.316

Cohen, L. S., Soares, C. N., Otto, M. W., Sweeney, B. H., Liberman, R. F., & Harlow, B. L. (2002). Prevalence and predictors of premenstrual dysphoric disorder (PMDD) in older premenopausal women: The Harvard Study of Moods and Cycles. *Journal of Affective Disorders, 70,* 125–132. doi:10.1016/S0165-0327(01)00458-X

Cosgrove, L., & Caplan, P. J. (2004). Medicalizing menstrual distress. In P. J. Caplan & L. Cosgrove (Eds.), *Bias in psychiatric diagnosis.*(pp. 221–230). Lanham, MD, US: Jason Aronson.

Cox, B. J., Enns, M. W., Freeman, P., & Walker, J. R. (2001). Anxiety sensitivity and major depression: Examination of affective state dependence. *Behaviour Research and Therapy, 39,* 1349–1356. doi:10.1016/S0005-7967(00)00106-6

Craner, J., Sigmon, S., Martinson, A., & McGillicuddy, M. (2013). Perceptions of health and somatic sensations in women reporting premenstrual syndrome and premenstrual dysphoric disorder. *Journal of Nervous and Mental Disease, 201,* 780–785. doi:10.1097/NMD.0b013e3182a213f1

Craner, J. R., Sigmon, S. T., Martinson, A. A., & McGillicuddy, M. L. (2014). Premenstrual disorders and rumination. *Journal of Clinical Psychology, 70,* 32–47. doi:10.1002/jclp.22007

Craner, J. R., Sigmon, S. T., & McGillicuddy, M. L. (2014). Does a disconnect occur between research and practice for premenstrual dysphoric disorder (PMDD) diagnostic procedures? *Women's Health, 54,* 232–244. doi:10.1080/03630242 .2014.883658

Craner, J. R., Sigmon, S. T., & Young, M. A. (2016). Self-focused attention and symptoms across menstrual cycle phases in women with and without premenstrual disorders. *Cognitive Therapy and Research, 40,* 118–127. doi:10 .1007/s10608-015-9721-5

Cunningham, J., Yonkers, K. A., O'Brien, S., & Eriksson, E. (2009). Update on research and treatment of premenstrual dysphoric disorder. *Harvard Review of Psychiatry, 17,* 120–137. doi:10.1080/10673220902891836

Daniels, K., & Abma, J. C. (2018). *Current contraceptive status among women aged 15–49: United States, 2015–2017* (NCHS Data Brief, no 327). Hyattsville, MD, US: National Center for Health Statistics.

Di Giulio, G., & Reissing, E. D. (2006). Premenstrual dysphoric disorder: Prevalence, diagnostic considerations, and controversies. *Journal of Psychosomatic Obstetrics & Gynecology, 27,* 201–210. doi:10.1080/01674820600747269

Dubey, N., Hoffman, J. F., Schuebel, K., Yuan, Q., Martinez, P. E., Nieman, L. K., … Goldman, D. (2017). The ESC/E(Z) complex, an effector of response to ovarian steroids, manifests an intrinsic difference in cells from women with premenstrual dysphoric disorder. *Molecular Psychiatry, 22,* 1172–1184. doi:10.1038/mp.2016.229

Eggert, L., Witthöft, M., Hiller, W., & Kleinstäuber, M. (2016). Emotion regulation in women with premenstrual syndrome (PMS): Explicit and implicit assessments. *Cognitive Therapy and Research, 40,* 747–763. doi:10.1007/s10608-016-9788-7

Eisenlohr-Moul, T. A., Girdler, S. S., Johnson, J. L., Schmidt, P. J., & Rubinow, D. R. (2017). Treatment of premenstrual dysphoria with continuous versus intermittent dosing of oral contraceptives: Results of a three-arm randomized controlled trial. *Depression and Anxiety, 34,* 908–917. doi:10.1002/da.22673

Eisenlohr-Moul, T. A., Girdler, S. S., Schmalenberger, K. M., Dawson, D. N., Surana, P., Johnson, J. L., & Rubinow, D. R. (2017). Toward the Reliable diagnosis of dsm-5 premenstrual dysphoric disorder: The Carolina Premenstrual Assessment Scoring System (C-PASS). *American Journal of Psychiatry, 174,* 51–59. doi:10.1176/appi.ajp.2016.15121510

Endicott, J. (1994). Differential diagnoses and comorbidity. In J. H. Gold & S. K. Severino (Eds.), *Premenstrual dysphorias: Myths and realities.* (pp. 3–17). Arlington, VA, US: American Psychiatric Association.

Endicott, J., Nee, J., & Harrison, W. (2006). Daily record of severity of problems (DRSP): Reliability and validity. *Archives of Women's Mental Health, 9,* 41–49. doi:10.1007/s00737-005-0103-y

Eriksson, E. (1999). Serotonin reuptake inhibitors for the treatment of premenstrual dysphoria. *International Clinical Psychopharmacology, 14*(Suppl 2), S27–S33. Retrieved from https://journals.lww.com/intclinpsychopharm/pages/default.aspx

Freeman, E. W. (2017). Premenstrual dysphoric disorder. In R. J. DeRubeis & D. R. Strunk (Eds.), *The Oxford Handbook of Mood Disorders* (p. 238). Oxford, England: Oxford University Press. doi:10.1093/oxfordhb/9780199973965.001 .0001

Gallant, S. J., Hamilton, J. A., Popiel, D. A., Morokoff, P. J., & Chakraborty, P. K. (1991). Daily moods and symptoms: Effects of awareness of study focus, gender, menstrual-cycle phase, and day of the week. *Health Psychology, 10,* 180–189. doi:10.1037/0278-6133.10.3.180

Gehlert, S., Song, I. H., Chang, C. H., & Hartlage, S. A. (2009). The prevalence of premenstrual dysphoric disorder in a randomly selected group of urban and rural women. *Psychological Medicine, 39,* 129–136. doi:10.1017/S003329170800322X

Glick, H., Endicott, J., & Nee, J. (1993). Premenstrual changes: Are they familial? *Acta Psychiatrica Scandinavica, 88,* 149–155. doi:0.1111/j.1600-0447.1993.tb03430.x

Halbreich, U. (2003). The etiology, biology, and evolving pathology of premenstrual syndromes. *Psychoneuroendocrinology, 28,* 55–99. doi:10.1016/S0306-4530 (03)00097-0

Halbreich, U., Borenstein, J., Pearlstein, T., & Kahn, L. S. (2003). The prevalence, impairment, impact, and burden of premenstrual dysphoric disorder (PMS/PMDD). *Psychoneuroendocrinology, 28,* 1–23. doi:10.1016/s0306-4530(03)00098-2

Hantsoo, L., & Epperson, C. N. (2015). Premenstrual dysphoric disorder: Epidemiology and treatment. *Current Psychiatry Reports, 17,* 87. doi:10.1007/s11920-015-0628-3

Harmon-Jones, C., Bastian, B., & Harmon-Jones, E. (2016). The Discrete Emotions Questionnaire: A new tool for measuring state self-reported emotions. *PLoS ONE, 11,* e0159915. doi:10.1371/journal.pone.0159915

Harmon-Jones, E., Harmon-Jones, C., Amodio, D. M., & Gable, P. A. (2011). Attitudes toward emotions. *Journal of Personality and Social Psychology, 101,* 1332–1350. doi:10.1037/a0024951

Hartlage, S. A., Brandenburg, D. L., & Kravitz, H. M. (2004). Premenstrual exacerbation of depressive disorders in a community-based sample in the United States. *Psychosomatic Medicine, 66,* 698–706. doi:10.1097/01.psy.0000138131.92408.b9

Hartlage, S. A., Breaux, C. A., & Yonkers, K. A. (2014). Addressing concerns about the inclusion of premenstrual dysphoric disorder in DSM-5. *Journal of Clinical Psychiatry, 75,* 70–76. doi:10.4088/JCP.13cs08368

Haywood, A., Slade, P., & King, H. (2002). Assessing the assessment measures for menstrual cycle symptoms: A guide for researchers and clinicians. *Journal of Psychosomatic Research, 52,* 223–237. doi:10.1016/S0022-3999(02)00297-0

Henkel, V., Mergl, R., Kohnen, R., Allgaier, A.-K., Möller, H.-J., & Hegerl, U. (2004). Use of brief depression screening tools in primary care: Consideration of heterogeneity in performance in different patient group. *General Hospital Psychiatry, 26,* 190–198. doi:10.1016/j.genhosppsych.2004.02.003

Holtzman, S., O'Connor, B. P., Barata, P. C., & Stewart, D. E. (2015). The Brief Irritability Test (BITe): A measure of irritability for use among men and women. *Assessment, 22,* 101–115. doi:10.1177/1073191114533814

IBM Corp. (2017). *IBM SPSS statistics for windows, version 25.0.* Armonk, NY: IBM Corp.

Interian, A., Allen, L. A., Gara, M. A., Escobar, J. I., & Díaz-Martínez, A. M. (2006). Somatic complaints in primary care: Further examining the validity of the Patient Health Questionnaire (PHQ-15). *Psychosomatics, 47*, 392–398. doi:10.1176/appi .psy.47.5.392

Ismaili, E., Walsh, S., O'Brien, P. M. S., Bäckström, T., Brown, C., Dennerstein, L., … Yonkers, K. (2016). Fourth consensus of the International Society for Premenstrual Disorders (ISPMD): Auditable standards for diagnosis and management of premenstrual disorder. *Archives of Women's Mental Health, 19,* 953–958. doi:10.1007/s00737-016-0631-7

Joormann, J., & Gotlib, I. H. (2010). Emotion regulation in depression: Relation to cognitive inhibition. *Cognition and Emotion, 24,* 281–298. doi:10.1080 /02699930903407948

Kendler, K. S., Karkowski, L. M., Corey, L. A., & Neale, M. C. (1998). Longitudinal population-based twin study of retrospectively reported premenstrual symptoms and lifetime major depression. *American Journal of Psychiatry, 155,* 1234–1240. doi:10.1176/1jp.155.9.1234

Keogh, E., & Reidy, J. (2000). Exploring the factor structure of the Mood and Anxiety Symptom Questionnaire (MASQ). *Journal of Personality Assessment, 74,* 106–125. doi:10.1207/S15327752JPA740108

Kepple, A. L., Lee, E. E., Haq, N., Rubinow, D. R., & Schmidt, P. J. (2016). History of postpartum depression in a clinic-based sample of women with premenstrual dysphoric disorder. *Journal of Clinical Psychiatry, 77,* e415–420. doi:10.4088/JCP.15m09779

Kiesner, J., Mendle, J., Eisenlohr-Moul, T. A., & Pastore, M. (2016). Cyclical symptom change across the menstrual cycle. *Clinical Psychological Science, 4,* 882–894. doi:10.1177/2167702616635031

Kim, D. R., Gyulai, L., Freeman, E. W., Morrison, M. F., Baldassano, C., & Dube, B. (2004). Premenstrual dysphoric disorder and psychiatric co-morbidity. *Archives of Women's Mental Health, 7,* 37–47. doi:10.1007/s00737-003-0027-3

King, M., & Ussher, J. M. (2013). It's not all bad: Women's construction and lived experience of positive premenstrual change. *Feminism & Psychology, 23,* 399–417. doi:10.1177/0959353512440351

Kleinstäuber, M., Witthöft, M., & Hiller, W. (2012). Cognitive-behavioral and pharmacological interventions for premenstrual syndrome or premenstrual dysphoric disorder: A meta-analysis. *Journal of Clinical Psychology in Medical Settings, 19,* 308–319. doi:10.1007/s10880-012-9299-y

Ko, C.-H., Long, C.-Y., Chen, S.-Y., Chen, I. J., Huang, T.-H., & Yen, J.-Y. (2013). Depression, irritability, and anxiety in women with premenstrual dysphoric disorder. *International Journal of Psychiatry in Medicine, 46,* 39–55. doi:10.2190/PM.46.1.d

Kornstein, S. G. (2010). Gender issues and DSM-V. *Archives of Women's Mental Health, 13,* 11–13. doi:10.1007/s00737-009-0113-2

Kornstein, S. G., Harvey, A. T., Rush, A. J., Wisniewski, S. R., Trivedi, M. H., Svikis, D. S., … Harley, R. (2005). Self-reported premenstrual exacerbation of depressive symptoms in patients seeking treatment for major depression. *Psychological Medicine, 35,* 683–692. doi:10.1017/S0033291704004106

Kroenke, K., Spitzer, R. L., & Williams, J. B. W. (2002). The PHQ-15: Validity of a new measure for evaluating the severity of somatic symptoms. *Psychosomatic Medicine, 64,* 258–266. doi:10.1097/00006842-200203000-00008

Kroenke, K., Spitzer, R. L., Williams, J. B., & Löwe, B. (2010). The Patient Health Questionnaire Somatic, Anxiety, and Depressive Symptom Scales: A systematic review. *General Hospital Psychiatry, 32,* 345–359. doi:10.1016/j.genhosppsych.2010.03.006

Kues, J. N., Janda, C., Krzikalla, C., Andersson, G., & Weise, C. (2018). The effect of manipulated information about premenstrual changes on the report of positive and negative premenstrual changes. *Women & Health, 58,* 16–37. doi:10.1080 /03630242.2016.1263274

Löwe, B., Decker, O., Müller, S., Brähler, E., Schellberg, D., Herzog, W., & Herzberg, P. Y. (2008). Validation and standardization of the Generalized Anxiety Disorder Screener (GAD-7) in the general population. *Medical Care, 46,* 266–274. doi:10 .1097/MLR.0b013e318160d093

Löwe, B., Spitzer, R. L., Gräfe, K., Kroenke, K., Quenter, A., Zipfel, S., … Herzog, W. (2004). Comparative validity of three screening questionnaires for DSM-IV depressive disorders and physicians' diagnoses. *Journal of Affective Disorders, 78,* 131–140. doi:10.1016/S0165-0327(02)00237-9

Lustyk, M. K. B., Gerrish, W. G., Douglas, H., Bowen, S., & Marlatt, G. A. (2011). Relationships among premenstrual symptom reports, menstrual attitudes, and mindfulness. *Mindfulness, 2,* 37–48. doi:10.1007/s12671-011-0041-x

Lustyk, M. K., Gerrish, W. G., Shaver, S., & Keys, S. L. (2009). Cognitive-behavioral therapy for premenstrual syndrome and premenstrual dysphoric disorder: A systematic review. *Archives of Women's Mental Health, 12,* 85–96. doi:10.1007 /s00737-009-0052-y

Maharaj, S., & Trevino, K. (2015). A comprehensive review of treatment options for premenstrual syndrome and premenstrual dysphoric disorder. *Journal of Psychiatric Practice, 21,* 334–350. doi:10.1097/PRA.0000000000000099

Marván, M. L., & Cortés-Iniestra, S. (2001). Women's beliefs about the prevalence of premenstrual syndrome and biases in recall of premenstrual changes. *Health Psychology, 20,* 276–280. doi:10.1037/0278-6133.20.4.276

McLaughlin, K. A., & Nolen-Hoeksema, S. (2011). Rumination as a transdiagnostic factor in depression and anxiety. *Behavior Research and Therapy, 49,* 186–193. doi:10.1016/j.brat.2010.12.006

Naragon-Gainey, K. (2010). Meta-analysis of the relations of anxiety sensitivity to the depressive and anxiety disorders. *Psychological Bulletin, 136,* 128–150. doi:10 .1037/a0018055

Nillni, Y. I., Rohan, K. J., Mahon, J. N., Pineles, S. L., & Zvolensky, M. J. (2013). The role of anxiety sensitivity in the experience of menstrual-related symptoms reported via daily diary. *Psychiatry Research, 210,* 564–569. doi:10.1016/j .psychres.2013.07.016

Nillni, Y. I., Rohan, K. J., & Zvolensky, M. J. (2012). The role of menstrual cycle phase and anxiety sensitivity in catastrophic misinterpretation of physical symptoms during a CO(2) challenge. *Archives of Women's Mental Health, 15,* 413–422. doi: 10.1007/s00737-012-0302-2

Nolen-Hoeksema, S. (2000). The role of rumination in depressive disorders and mixed anxiety/depressive symptoms. *Journal of Abnormal Psychology, 109,* 504–511. doi:10.1037/0021-843X.109.3.504

O'Brien, P. M., Backstrom, T., Brown, C., Dennerstein, L., Endicott, J., Epperson, C. N., … Yonkers, K. (2011). Towards a consensus on diagnostic criteria, measurement and trial design of the premenstrual disorders: The ISPMD Montreal consensus. *Archives of Women's Mental Health, 14,* 13–21. doi:10.1007/s00737-010-0201-3

Offman, A., & Kleinplatz, P. J. (2004). Does PMDD belong in the DSM? Challenging the medicalization of women's bodies. *Canadian Journal of Human Sexuality, 13,* 17–27. Retrieved from https://utorontopress.com/ca/the-canadian-journal-of-human-sexuality

Olthuis, J. V., Watt, M. C., & Stewart, S. H. (2014). Anxiety Sensitivity Index (ASI-3) subscales predict unique variance in anxiety and depressive symptoms. *Journal of Anxiety Disorders, 28,* 115–124. doi:10.1016/j.janxdis.2013.04.009

Osman, A., Gutierrez, P. M., Smith, K., Fang, Q., Lozano, G., & Devine, A. (2010). The Anxiety Sensitivity Index-3: Analyses of dimensions, reliability estimates, and correlates in nonclinical samples. *Journal of Personality Assessment, 92,* 45–52. doi:10.1080/00223890903379332

Panahi, F., & Faramarzi, M. (2016). The effects of mindfulness-based cognitive therapy on depression and anxiety in women with premenstrual syndrome. *Depression Research and Treatment, 2016,* 9816481. doi:10.1155/2016/9816481

Parry, B. L. (2001). The role of central serotonergic dysfunction in the aetiology of premenstrual dysphoric disorder. *CNS Drugs, 15*(4), 277–285. doi:10.2165/00023210-200115040-00003

Payne, J. L., Klein, S. R., Zamoiski, R. B., Zandi, P. P., Bienvenu, O. J., MacKinnon, D. F., … Potash, J. B. (2009). Premenstrual mood symptoms: Study of familiality and personality correlates in mood disorder pedigrees. *Archives of Women's Mental Health, 12,* 27–34. doi:10.1007/s00737-008-0043-4

Pearlstein, T. (2016). Treatment of premenstrual dysphoric disorder: Therapeutic challenges. *Expert Review of Clinical Pharmacology, 9,* 493–496. doi:10.1586/17512433.2016.1142371

Pearlstein, T., & Steiner, M. (2008). Premenstrual dysphoric disorder: Burden of illness and treatment update. *Journal of Psychiatry & Neuroscience, 33,* 291–301. doi:10.1176/appi.focus.10.1.90

Petersen, N., Ghahremani, D. G., Rapkin, A. J., Berman, S. M., Liang, L., & London, E. D. (2018). Brain activation during emotion regulation in women with premenstrual dysphoric disorder. *Psychological Medicine, 48,* 1795–1802. doi:10.1017/S0033291717003270

Petersen, N., London, E. D., Liang, L., Ghahremani, D. G., Gerards, R., Goldman, L., & Rapkin, A. J. (2016). Emotion regulation in women with premenstrual dysphoric disorder. *Archives of Women's Mental Health, 19,* 891–898. doi:10.1007/s00737-016-0634-4

Pinkerton, J. V., Guico-Pabia, C. J., & Taylor, H. S. (2010). Menstrual cycle-related exacerbation of disease. *American Journal of Obstetrics and Gynecology, 202,* 221–231. doi:10.1016/j.ajog.2009.07.061

Raudenbush, S. W., & Bryk, A. S. (2002). *Hierarchical linear models: Applications and data analysis methods* (2nd ed.). Thousand Oaks, CA, US: Sage.

Raudenbush, S. W., Bryk, A. S., Cheong, Y. F., & Congdon, R. (2011). *HLM 7 for Windows* [Computer software]. Skokie, IL, CA: Scientific Software International.

Reiss, S., Peterson, R. A., Gursky, D. M., & McNally, R. J. (1986). Anxiety sensitivity, anxiety frequency and the predictions of fearfulness. *Behaviour Research and Therapy, 24,* 1–8. doi:10.1016/0005-7967(86)90143-9

Reuveni, I., Dan, R., Segman, R., Evron, R., Laufer, S., Goelman, G., … Canetti, L. (2016). Emotional regulation difficulties and premenstrual symptoms among Israeli students. *Archives of Women's Mental Health, 19,* 1063–1070. doi:10.1007 /s00737-016-0656-y

Rodin, M. (1992). The social construction of premenstrual syndrome. *Social Science & Medicine, 35,* 49–56. doi:10.1016/0277-9536(92)90118-A

Romans, S., Clarkson, R., Einstein, G., Petrovic, M., & Stewart, D. (2012). Mood and the menstrual cycle: A review of prospective data studies. *Gender Medicine, 9,* 361–384. doi:10.1016/j.genm.2012.07.003

Romans, S. E., Kreindler, D., Asllani, E., Einstein, G., Laredo, S., Levitt, A., … Stewart, D. E. (2013). Mood and the menstrual cycle. *Psychotherapy and Psychosomatics, 82,* 53–60. doi:10.1159/000339370

Schmidt, P. J., Martinez, P. E., Nieman, L. K., Koziol, D. E., Thompson, K. D., Schenkel, L., … Rubinow, D. R. (2017). Premenstrual dysphoric disorder symptoms following ovarian suppression: triggered by change in ovarian steroid levels but not continuous stable levels. *American Journal of Psychiatry, 174,* 980–989. doi: 10.1176/appi.ajp.2017.16101113

Schüle, C., Nothdurfter, C., & Rupprecht, R. (2014). The role of allopregnanolone in depression and anxiety. *Progress in Neurobiology, 113,* 79–87. doi:10.1016/j .pneurobio.2013.09.003

Schwarz, N. (2007). Retrospective and concurrent self-reports: The rationale for real-time data capture. In A. A. Stone, S. Shiffman, A. A. Atienza, & L. Nebeling (Eds.), *The science of real-time data capture: Self-reports in health research* (p. 11–26). Oxford, England: Oxford University Press. doi:10.1201/9781584888901.ch2

Sepede, G., Sarchione, F., Matarazzo, I., Di Giannantonio, M., & Salerno, R. M. (2016). Premenstrual dysphoric disorder without comorbid psychiatric conditions: A systematic review of therapeutic options. *Clinical Neuropharmacology, 39,* 241–261. doi:10.1097/WNF.0000000000000173

Sigmon, S. T., Dorhofer, D. M., Rohan, K. J., & Boulard, N. E. (2000). The impact of anxiety, sensitivity, bodily expectations, and cultural beliefs on menstrual symptom reporting: A test of the menstrual reactivity hypothesis. *Journal of Anxiety Disorders, 14,* 615–633. doi:10.1016/S0887-6185(00)00054-2

Sigmon, S. T., Rohan, K. J., Boulard, N. E., Dorhofer, D. M., & Whitcomb, S. R. (2000). Menstrual reactivity: The role of gender-specificity, anxiety sensitivity, and somatic concerns in self-reported menstrual distress. *Sex Roles, 43,* 143–161. doi:10.1023/A:1007036012911

Sigmon, S. T., Schartel, J. G., Hermann, B. A., Cassel, A. G., & Thorpe, G. L. (2009). The relationship between premenstrual distress and anxiety sensitivity: The mediating role of rumination. *Journal of Rational-Emotive & Cognitive-Behavior Therapy, 27,* 188–200. doi:10.1007/s10942-009-0100-6

Sigmon, S. T., Whitcomb-Smith, S. R., Rohan, K. J., & Kendrew, J. J. (2004). The role of anxiety level, coping styles, and cycle phase in menstrual distress. *Journal of Anxiety Disorders, 18,* 177≠191. doi:10.1016/s0887-6185(02)00243-8

Śliwerski, A., & Bielawska-Batorowicz, E. (2018). Negative cognitive styles as risk factors for the occurrence of PMS and PMDD. *Journal of Reproductive and Infant Psychology, 37,* 322–337. doi:10.1080/02646838.2018.1543943

Spitzer, R. L., Kroenke, K., Williams, J. B. W., & Löwe, B. (2006). A brief measure for assessing generalized anxiety disorder: The GAD-7. *Archives of Internal Medicine, 166,* 1092–1097. doi:10.1001/archinte.166.10.1092

Spybrook, J., Bloom, H., Congdon, R., Hill, C., Martinez, A., & Raudenbush, S. W. (2011). *Optimal design plus empirical evidence: Documentation for the "Optimal Design" Software Version.* Retrieved from http://hlmsoft.net/od/od-manual -20111016-v300.pdf

Steiner, M., Macdougall, M., & Brown, E. (2003). The premenstrual symptoms screening tool (PSST) for clinicians. *Archives of Women's Mental Health, 6,* 203–209. doi: 10.1007/s00737-003-0018-4

Taylor, S., & Cox, B. J. (1998). Anxiety sensitivity: Multiple dimensions and hierarchic structure. *Behaviour Research and Therapy, 36,* 37–51. doi:10.1016/S0005-7967 (97)00071-5

Taylor, S., Koch, W. J., Woody, S., & McLean, P. (1996). Anxiety sensitivity and depression: How are they related? *Journal of Abnormal Psychology, 105,* 474–479. doi:10.1037/0021-843X.105.3.474

Taylor, S., Zvolensky, M. J., Cox, B. J., Deacon, B., Heimberg, R. G., Ledley, D. R., … Cardenas, S. J. (2007). Robust dimensions of anxiety sensitivity: Development and initial validation of the Anxiety Sensitivity Index-3. *Psychological Assessment, 19,* 176–188. doi:10.1037/1040-3590.19.2.176

Treynor, W., Gonzalez, R., & Nolen-Hoeksema, S. (2003). Rumination reconsidered: A psychometric analysis. *Cognitive Therapy and Research, 27,* 247–259. doi:10 .1023/A:1023910315561

Ussher, J. M. (2010). Are we medicalizing women's misery? A critical review of women's higher rates of reported depression. *Feminism & Psychology, 20,* 9–35. doi:10.1177/0959353509350213

Veeninga, A. T., & Westenberg, H. G. (1992). Serotonergic function and late luteal phase dysphoric disorder. *Psychopharmacology, 108,* 153–158. doi:10.1007 /BF02245301

Wakefield, J. C. (2013). DSM-5: An overview of changes and controversies. *Clinical Social Work Journal, 41,* 139–154. doi:10.1007/s10615-013-0445-2

Watson, D., & Walker, L. M. (1996). The long-term stability and predictive validity of trait measures of affect. *Journal of Personality and Social Psychology, 70,* 567–577. doi:10.1037/0022-3514.70.3.567

Watson, D., Weber, K., Assenheimer, J. S., Clark, L. A., Strauss, M. E., & McCormick, R. A. (1995). Testing a tripartite model: I. Evaluating the convergent and discriminant validity of anxiety and depression symptom scales. *Journal of Abnormal Psychology, 104,* 3–14. doi:10.1037/0021-843X.104.1.3

Weise, C., Kaiser, G., Janda, C., Kues, J. N., Andersson, G., Strahler, J., & Kleinstäuber, M. (2019). Internet-based cognitive-behavioural intervention for women with premenstrual dysphoric disorder: A randomized controlled trial. *Psychotherapy and Psychosomatics, 88,* 16–29. doi:10.1159/000496237

Welz, A., Huffziger, S., Reinhard, I., Alpers, G. W., Ebner-Priemer, U., & Kuehner, C. (2016). Anxiety and rumination moderate menstrual cycle effects on mood in daily life. *Women Health, 56,* 540–560. doi:10.1080/03630242.2015.1101739

Whitmer, A., & Gotlib, I. H. (2011). Brooding and reflection reconsidered: A factor analytic examination of rumination in currently depressed, formerly depressed, and never depressed individuals. *Cognitive Therapy and Research, 35,* 99–107. doi:10.1007/s10608-011-9361-3

Williams, J. W., Jr., Pignone, M., Ramirez, G., & Stellato, C. P. (2002). Identifying depression in primary care: A literature synthesis of case-finding instruments. *General Hospital Psychiatry, 24,* 225–237. 10.1016/S0163-8343(02)00195-0

Wisco, B. E., & Nolen-Hoeksema, S. (2008). Ruminative response style. In K. S. Dobson & D. J. A. Dozois (Ed.), *Risk factors in depression* (pp. 221–236). San Diego, CA, US: Elsevier Academic Press.

Yang, M., Wallenstein, G., Hagan, M., Guo, A., Chang, J., & Kornstein, S. (2008). Burden of premenstrual dysphoric disorder on health-related quality of life. *Journal of Women's Health, 17,* 113–121. doi:10.1089/jwh.2007.0417

Yatham, L. N. (1993). Is 5HT1A receptor subsensitivity a trait marker for late luteal phase dysphoric disorder? A pilot study. *Canadian Journal of Psychiatry, 38,* 662–664. doi:10.1177/070674379303801007

Yonkers, K. A., Pearlstein, T., & Rosenheck, R. A. (2003). Premenstrual disorders: Bridging research and clinical reality. *Archives of Women's Mental Health, 6,* 287–292. doi:10.1007/s00737-003-0026-4

Table 1

Sample Demographic Characteristics

Demographic	*M(SD)* or %
Age, M(SD)	26.97 years (7.48)
Race	Race
Caucasian	72.3
African American	11.7
Asian	13.8
Hispanic	2.1
Other	2.1
Marital Status	
Single	62.4
Married	32.3
Divorced or Separated	5.4
Sexual Orientation	
Heterosexual	79.8
Bisexual	17.0
Other	2.1
Prefer not to say	1.1
Hormonal birth control use	
Yes	44.7
No	55.3
Other birth control use	
None	75.5
Condoms	16.0
Implant	1.1
IUD	3.3
Patches	2.1
Rhythm method	1.1
Vasectomy	1.1

Table 2

Pearson Correlation Matrix Between Premenstrual and Other Psychiatric Symptoms

	PMS Symptoms	Depression	Anxiety	Somatic symptoms	Rumination	Anxiety sensitivity
PMS symptoms	1.00	.024	-.039	-.021	.357*	.445*
Depression		1.00	.802*	.649*	.048*	-.033
Anxiety			1.00	.641*	-.063*	-.080
Somatic symptoms				1.00	-.105*	-.091
Rumination					1.00	.510*
Anxiety sensitivity						1.00

Note. PMS = premenstrual, *$p < .05$; **$p < .01$.

Table 3

Pearson Correlation Matrix BETWEEN Psychiatric Symptoms and Daily Mood and Affect Ratings

	PMS SYMPTOMS	Depression	Irritability	General Distress	Anhedonic Depression	Anxious Arousal	Anger	Disgust	Fear	Anxiety	Sadness	Desire	Relaxation	Happiness
PMS symptoms	1.00													
Depression	.024	1.00												
Irritability	.207**	.091**	1.00											
General distress	.293**	.063**	.707**	1.00										
Anhedonic depression	-.155**	.057**	-.395**	-.490**	1.00									
Anxious arousal	.298**	-.010	.556**	.727**	-.217**	1.00								
Anger	.175**	.074**	.784**	.674**	-.296**	.604**	1.00							
Disgust	.233**	.032	.509**	.598**.	-.168**	.736**	.607**	1.00						
Fear	.130**	.090**	.554**	.699**	-.191**	.669**	.677**	.651**	1.00					
Anxiety	.304**	.114**	.633**	.791**	-.373**	.552**	.603**	.524**	.692**	1.00				
Sadness	.251**	.050*	.659**	.820**	-.452**	.570**	.684**	.575**	.662**	.701**	1.00			
Desire	.289**	.110**	.291**	.358**	.168**	.397**	.317**	.440**	.362**	.352**	.401**	1.00		
Relaxation	-.022**	.001	-.246**	-.259**	.718**	-.046**	-.151*	.015	-.048*	-.222**	-.201**	.335**	1.00	
Happiness	-.010**	.072**	-.224**	-.264**	.830**	-.039	-.128**	.024	-.040	-.207**	-.225**	.390**	.797**	1.00

Note. PMS = premenstrual, *$p < .01$.

Table 4

Multilevel Model Results Predicting General Distress From Depressive Symptom Severity Using Full Maximum Likelihood Estimation (N = 94)

	Final estimation of fixed effects				
	Coefficient	*SE*	*t*-ratio	Appx *d.f.*	*p*-value
For Intercept 1, π_0					
Intercept 2, β_{00}	33.755	1.193	28.289	90	< .001
Depressive symptoms, β_{01}	0.191	0.200	0.954	90	.343

Note. * $p < .05$

Table 5

Multilevel Model Results Predicting Anhedonic Depression From Depressive Symptom Severity Using Full Maximum Likelihood Estimation (N = 94)

	Final estimation of fixed effects				
	Coefficient	*SE*	*t*-ratio	Appx *d.f.*	*p*-value
For Intercept 1, π_0					
Intercept 2, β_{00}	55.884	1.196	46.738	89	< .001
Depressive symptoms, β_{01}	0.088	0.200	0.440	89	.661

Note. * $p < .05$.

Table 6

Multilevel Model Results Predicting Anxious Arousal From Depressive Symptom Severity Using Full Maximum Likelihood Estimation (N = 94)

	Final estimation of fixed effects				
	Coefficient	*SE*	*t*-ratio	Appx *d.f.*	*p*-value
For Intercept 1, π_0					
Intercept 2, β_{00}	13.817	0.520	26.554	89	< .001
Depressive symptoms, β_{01}	-0.006	0.087	-0.069	89	.945

Note. * $p < .05$.

Table 7

Multilevel Model Results Predicting Irritability From Depressive Symptom Severity Using Full Maximum Likelihood Estimation (N = 94)

	Final estimation of fixed effects				
	Coefficient	*SE*	*t*-ratio	Appx *d.f.*	*p*-value
For Intercept 1, π_0					
Intercept 2, β_{00}	1.972	0.095	20.838	89	< .001
Depressive symptoms, β_{01}	0.013	0.016	0.836	89	0.405

Note. * $p < .05$.

Table 8

Multilevel Model Results Predicting Anger From Depressive Symptom Severity Using Full Maximum Likelihood Estimation (N = 94)

	Final estimation of fixed effects				
	Coefficient	*SE*	*t*-ratio	Appx *d.f.*	*p*-value
For Intercept 1, π_0					
Intercept 2, β_{00}	6.733	0.361	18.651	90	< .001
Depressive symptoms, β_{01}	0.039	0.061	0.655	90	.514

Note. * $p < .05$.

Table 9

Multilevel Model Results Predicting Disgust From Depressive Symptom Severity Using Full Maximum Likelihood Estimation (N = 94)

	Final estimation of fixed effects				
	Coefficient	*SE*	*t*-ratio	Appx *d.f.*	*p*-value
For Intercept 1, π_0					
Intercept 2, β00	5.916	0.270	21.905	90	< .001
Depressive symptoms, β01	0.019	0.045	0.421	90	0.675

Note. * $p < .05$.

Table 10

Multilevel Model Results Predicting Fear From Depressive Symptom Severity Using Full Maximum Likelihood Estimation (N = 94)

	Final estimation of fixed effects				
	Coefficient	*SE*	*t*-ratio	Appx *d.f.*	*p*-value
For Intercept 1, π_0					
Intercept 2, β_{00}	6.045	0.314	19.255	90	< .001
Depressive Symptoms, β_{01}	0.061	0.053	1.168	90	0.246

Note. * $p < .05$.

Table 11

Multilevel Model Results Predicting Anxiety From Depressive Symptom Severity Using Full Maximum Likelihood Estimation (N = 94)

	Final estimation of fixed effects				
	Coefficient	*SE*	*t*-ratio	Appx *d.f.*	*p*-value
For Intercept 1, π_0					
Intercept 2, β_{00}	8.918	0.369	24.162	90	< .001
Depressive symptoms, β_{01}	0.096	0.062	1.553	90	0.124

Note. * $p < .05$.

Table 12

Multilevel Model Results Predicting Sadness From Depressive Symptom Severity Using Full Maximum Likelihood Estimation (N = 94)

	Final estimation of fixed effects				
	Coefficient	*SE*	*t*-ratio	Appx *d.f.*	*p*-value
For Intercept 1, π_0					
Intercept 2, β_{00}	7.667	0.374	20.482	90	< .001
Depressive symptoms, β_{01}	0.042	0.063	0.669	90	0.505

Note. * $p < .05$.

Table 13

Multilevel Model Results Predicting Desire From Depressive Symptom Severity Using Full Maximum Likelihood Estimation (N = 94)

	Final estimation of fixed effects				
	Coefficient	*SE*	*t*-ratio	Appx *d.f.*	*p*-value
For Intercept 1, π_0					
Intercept 2, β_{00}	8.336	0.413	20.184	90	< .001
Depressive symptoms, β_{01}	0.064	0.069	0.918	90	.361

Note. * $p < .05$.

Table 14

Multilevel Model Results Predicting Relaxation From Depressive Symptom Severity Using Full Maximum Likelihood Estimation (N = 94)

	Final estimation of fixed effects				
	Coefficient	*SE*	*t*-ratio	Appx *d.f.*	*p*-value
For Intercept 1, π_0					
Intercept 2, β_{00}	10.680	0.403	26.502	90	< .001
Depressive symptoms, β_{01}	-0.006	0.068	-0.089	90	0.929

Note. * $p < .05$.

Table 15

Multilevel Model Results Predicting Happiness From Depressive Symptom Severity Using Full Maximum Likelihood Estimation (N = 94)

	Final estimation of fixed effects				
	Coefficient	*SE*	*t*-ratio	Appx *d.f.*	*p*-value
For Intercept 1, π_0					
Intercept 2, β_{00}	11.036	0.443	24.890	90	< .001
Depressive symptoms, β_{01}	0.036	0.074	0.486	90	0.628

Note. * $p < .05$.

Table 16

Multilevel Model Results Predicting General Distress From Premenstrual Symptom Severity Using Full Maximum Likelihood Estimation (N = 94)

	Final estimation of fixed effects				
	Coefficient	*SE*	*t*-ratio	Appx *d.f.*	*p*-value
For Intercept 1, π_0					
Intercept 2, β_{00}	33.752	1.106	30.504	90	< .001
Premenstrual symptoms, β_{01}	3.541	0.894	3.960	90	< .001*

Note. * $p < .05$.

Table 17

Multilevel Model Results Predicting Anhedonic Depression From Premenstrual Symptom Severity Using Full Maximum Likelihood Estimation (N = 94)

	Final estimation of fixed effects				
	Coefficient	*SE*	*t*-ratio	Appx *d.f.*	*p*-value
For Intercept 1, π_0					
Intercept 2, β_{00}	56.034	1.148	48.824	90	< .001
Premenstrual symptoms, β_{01}	-1.852	0.928	-1.996	90	0.049*

Note. * p < .05.

Table 18

Multilevel Model Results Predicting Anxious Arousal From Premenstrual Symptom Severity Using Full Maximum Likelihood Estimation (N = 94)

	Final estimation of fixed effects				
	Coefficient	*SE*	*t*-ratio	Appx *d.f.*	*p*-value
For Intercept 1, π_0					
Intercept 2, β_{00}	13.771	0.472	29.148	90	< .001
Premenstrual symptoms, β_{01}	1.472	0.382	3.856	90	< .001*

Note. * p < .05.

Table 19

Multilevel Model Results Predicting Irritability From Premenstrual Symptom Severity Using Full Maximum Likelihood Estimation (N = 94)

	Final estimation of fixed effects				
	Coefficient	*SE*	*t*-ratio	Appx *d.f.*	*p*-value
For Intercept 1, π_0					
Intercept 2, β_{00}	1.962	0.089	21.889	90	< .001
Premenstrual symptoms, β_{01}	0.193	0.072	2.670	90	.009*

Note. * p < .05.

Table 20

Multilevel Model Results Predicting Anger From Premenstrual Symptom Severity Using Full Maximum Likelihood Estimation (N = 94)

	Final estimation of fixed effects				
	Coefficient	*SE*	*t*-ratio	Appx *d.f.*	*p*-value
For Intercept 1, π_0					
Intercept 2, β_{00}	6.733	0.351	19.210	90	< .001
Depressive symptoms, β_{01}	0.692	0.283	2.444	90	.016*

Note. * *p* < .05.

Table 21

Multilevel Model Results Predicting Disgust From Premenstrual Symptom Severity Using Full Maximum Likelihood Estimation (N = 94)

	Final estimation of fixed effects				
	Coefficient	*SE*	*t*-ratio	Appx *d.f.*	*p*-value
For Intercept 1, π_0					
Intercept 2, β_{00}	5.915	0.258	22.897	90	< .001
Depressive symptoms, β_{01}	0.609	0.209	2.916	90	.004*

Note. * *p* < .05.

Table 22

Multilevel Model Results Predicting Fear From Premenstrual Symptom Severity Using Full Maximum Likelihood Estimation (N = 94)

	Final estimation of fixed effects				
	Coefficient	*SE*	*t*-ratio	Appx *d.f.*	*p*-value
For Intercept 1, π_0					
Intercept 2, β_{00}	6.045	0.311	19.466	90	< .001
Depressive symptoms, β_{01}	0.465	0.251	1.854	90	.067

Note. * $p < .05$.

Table 23

Multilevel Model Results Predicting Anxiety From Premenstrual Symptom Severity Using Full Maximum Likelihood Estimation (N = 94)

	Final estimation of fixed effects				
	Coefficient	*SE*	*t*-ratio	Appx *d.f.*	*p*-value
For Intercept 1, π_0					
Intercept 2, β_{00}	8.917	0.339	26.302	90	< .001
Depressive symptoms, β_{01}	1.209	0.274	4.413	90	< .001*

Note. * $p < .05$.

Table 24

Multilevel Model Results Predicting Sadness From Premenstrual Symptom Severity Using Full Maximum Likelihood Estimation (N = 94)

	Final estimation of fixed effects				
	Coefficient	*SE*	*t*-ratio	Appx *d.f.*	*p*-value
For Intercept 1, π_0					
Intercept 2, β_{00}	7.666	0.355	21.589	90	< .001
Depressive symptoms, β_{01}	0.928	0.287	3.235	90	.002*

Note. * $p < .05$.

Table 25

Multilevel Model Results Predicting Desire From Premenstrual Symptom Severity Using Full Maximum Likelihood Estimation (N = 94)

	Final estimation of fixed effects				
	Coefficient	*SE*	*t*-ratio	Appx *d.f.*	*p*-value
For Intercept 1, π_0					
Intercept 2, β_{00}	8.336	0.388	21.501	90	< .001
Depressive symptoms, β_{01}	1.135	0.313	3.621	90	< .001*

Note. * $p < .05$.

Table 26

Multilevel Model Results Predicting Relaxation From Premenstrual Symptom Severity Using Full Maximum Likelihood Estimation (N = 94)

	Final estimation of fixed effects				
	Coefficient	*SE*	*t*-ratio	Appx *d.f.*	*p*-value
For Intercept 1, π_0					
Intercept 2, β_{00}	10.681	0.403	26.515	90	< .001
Depressive symptoms, β_{01}	-0.101	0.326	0.310	90	.757

Note. * $p < .05$.

Table 27

Multilevel Model Results Predicting Happiness From Premenstrual Symptom Severity Using Full Maximum Likelihood Estimation (N = 94)

	Final estimation of fixed effects				
	Coefficient	*SE*	*t*-ratio	Appx *d.f.*	*p*-value
For Intercept 1, π_0					
Intercept 2, β_{00}	11.036	0.444	24.861	90	< .001
Depressive symptoms, β_{01}	-0.062	0.359	-0.174	90	.862

Note. * $p < .05$.

Table 28

Multilevel Model Results Predicting General Distress From Luteal Phase and Premenstrual Symptom Severity Interaction Using Full Maximum Likelihood Estimation (N = 94)

	Final estimation of fixed effects				
	Coefficient	*SE*	*t*-ratio	Appx *d.f.*	*p*-value
For Intercept 1, π_0					
Intercept 2, β_{00}	33.749989	1.108	30.458	90	< .001
Premenstrual symptoms, β_{01}	3.539483	0.896	3.952	90	< .001
For luteal slope π_1					
Intercept 2, β_{10}	1.022	0.520	1.964	2106	.050
Premenstrual symptoms, β_{11}	0.578	0.419	1.380	2106	.168

Note. * $p < .05$.

Table 29

Multilevel Model Results Predicting Anhedonic Depression From Luteal Phase and Premenstrual Symptom Severity Interaction Using Full Maximum Likelihood Estimation (N = 94)

	Final estimation of fixed effects				
	Coefficient	*SE*	*t*-ratio	Appx *d.f.*	*p*-value
For Intercept 1, π_0					
Intercept 2, β_{00}	56.033502	1.147	48.836	90	< .001
Premenstrual symptoms, β_{01}	-1.853387	0.927	-1.999	90	.049
For luteal slope π_1					
Intercept 2, β_{10}	-0.014	0.526	-0.027	2106	.978
Premenstrual symptoms, β_{11}	0.918	0.423	2.155	2106	.031*

Note. * $p < .05$.

Table 30

Multilevel Model Results Predicting Anxious Arousal From Luteal Phase and Premenstrual Symptom Severity Interaction Using Full Maximum Likelihood Estimation (N = 94)

	Final estimation of fixed effects				
	Coefficient	*SE*	*t*-ratio	Appx *d.f.*	*p*-value
For Intercept 1, π_0					
Intercept 2, β_{00}	13.771	0.472	29.146	90	< .001
Premenstrual symptoms, β_{01}	1.472	0.382	3.856	90	< .001
For luteal slope π_1					
Intercept 2, β_{10}	0.275	0.201	1.372	2106	.170
Premenstrual symptoms, β_{11}	-0.095	0.161	-0.588	2106	.556

Note. * $p < .05$.

Table 31

Multilevel Model Results Predicting Irritability From Luteal Phase and Premenstrual Symptom Severity Interaction Using Full Maximum Likelihood Estimation (N = 94)

	Final estimation of fixed effects				
	Coefficient	*SE*	*t*-ratio	Appx *d.f.*	*p*-value
For Intercept 1, π_0					
Intercept 2, β_{00}	1.962	0.089	21.881	90	< .001
Premenstrual symptoms, β_{01}	0.193	0.072	2.670	90	.009
For luteal slope π_1					
Intercept 2, β_{10}	0.071	0.044	1.614	2108	.107
Premenstrual symptoms, β_{11}	-0.012	0.035	-0.335	2108	.737

Note. * $p < .05$.

Table 32

Multilevel Model Results Predicting Anger From Luteal Phase and Premenstrual Symptom Severity Interaction Using Full Maximum Likelihood Estimation (N = 94)

	Final estimation of fixed effects				
	Coefficient	*SE*	*t*-ratio	Appx *d.f.*	*p*-value
For Intercept 1, π_0					
Intercept 2, β_{00}	6.732	0.347	19.160	90	< .001
Premenstrual symptoms, β_{01}	0.692	0.284	2.437	90	.017
For luteal slope π_1					
Intercept 2, β_{10}	0.429	0.179	2.416	2113	.016
Premenstrual symptoms, β_{11}	0.219	0.143	1.526	2113	.127

Note. * $p < .05$.

Table 33

Multilevel Model Results Predicting Disgust From Luteal Phase and Premenstrual Symptom Severity Interaction Using Full Maximum Likelihood Estimation (N = 94)

	Final estimation of fixed effects				
	Coefficient	*SE*	*t*-ratio	Appx *d.f.*	*p*-value
For Intercept 1, π_0					
Intercept 2, β_{00}	5.916	0.259	22.873	90	< .001
Premenstrual symptoms, β_{01}	0.609	0.209	2.912	90	.005
For luteal slope π_1					
Intercept 2, β_{10}	-0.030	0.122	-0.246	2113	.806
Premenstrual symptoms, β_{11}	0.125	0.099	1.273	2113	.203

Note. * $p < .05$.

Table 34

Multilevel Model Results Predicting Fear From Luteal Phase and Premenstrual Symptom Severity Interaction Using Full Maximum Likelihood Estimation (N = 94)

	Final estimation of fixed effects				
	Coefficient	*SE*	*t*-ratio	Appx *d.f.*	*p*-value
For Intercept 1, π_0					
Intercept 2, β_{00}	6.045	0.311	19.459	90	< .001
Premenstrual symptoms, β_{01}	0.465	0.251	1.854	90	.067
For luteal slope π_1					
Intercept 2, β_{10}	0.122	0.139	0.879	2113	.379
Premenstrual symptoms, β_{11}	0.008	0.112	0.075	2113	.941

Note. * $p < .05$.

Table 35

Multilevel Model Results Predicting Anxiety From Luteal Phase and Premenstrual Symptom Severity Interaction Using Full Maximum Likelihood Estimation (N = 94)

	Final estimation of fixed effects				
	Coefficient	*SE*	*t*-ratio	Appx *d.f.*	*p*-value
For Intercept 1, π_0					
Intercept 2, β_{00}	8.917	0.339	26.298	90	< .001
Premenstrual symptoms, β_{01}	1.208	0.274	4.411	90	< .001
For luteal slope π_1					
Intercept 2, β_{10}	-0.070	0.914	-0.363	2113	.716
Premenstrual symptoms, β_{11}	0.051	0.157	0.324	2113	.746

Note. * $p < .05$.

Table 36

Multilevel Model Results Predicting Sadness From Luteal Phase and Premenstrual Symptom Severity Interaction Using Full Maximum Likelihood Estimation (N = 94)

	Final estimation of fixed effects				
	Coefficient	*SE*	*t*-ratio	Appx *d.f.*	*p*-value
For Intercept 1, π_0					
Intercept 2, β_{00}	7.666	0.355	21.570	90	< .001
Premenstrual symptoms, β_{01}	0.928	0.287	3.230	90	.002
For luteal slope π_1					
Intercept 2, β_{10}	0.011	0.173	0.064	2112	.949
Premenstrual symptoms, β_{11}	0.168	0.139	1.200	2112	.230

Note. * $p < .05$.

Table 37

Multilevel Model Results Predicting Desire From Luteal Phase and Premenstrual Symptom Severity Interaction Using Full Maximum Likelihood Estimation (N = 94)

	Final estimation of fixed effects				
	Coefficient	*SE*	*t*-ratio	Appx *d.f.*	*p*-value
For Intercept 1, π_0					
Intercept 2, β_{00}	8.336	0.387	21.500	90	< .001
Premenstrual symptoms, β_{01}	1.134	0.313	3.620	90	< .001
For luteal slope π_1					
Intercept 2, β_{10}	-0.141	0.154	-0.915	2112	0.360
Premenstrual symptoms, β_{11}	0.129	0.124	1.044	2112	0.296

Note. * $p < .05$.

Table 38

Multilevel Model Results Predicting Relaxation From Luteal Phase and Premenstrual Symptom Severity Interaction Using Full Maximum Likelihood Estimation (N = 94)

	Final estimation of fixed effects				
	Coefficient	*SE*	*t*-ratio	Appx *d.f.*	*p*-value
For Intercept 1, π_0					
Intercept 2, β_{00}	10.681	0.403	26.535	90	< .001
Premenstrual symptoms, β_{01}	-0.102	0.325	-0.313	90	.755
For luteal slope π_1					
Intercept 2, β_{10}	-0.179	0.201	-0.889	2113	.374
Premenstrual symptoms, β_{11}	0.428	0.163	2.625	2113	.009*

Note. * $p < .05$.

Table 39

Multilevel Model Results Predicting Happiness From Luteal Phase and Premenstrual Symptom Severity Interaction Using Full Maximum Likelihood Estimation (N = 94)

	Final estimation of fixed effects				
	Coefficient	*SE*	*t*-ratio	Appx *d.f.*	*p*-value
For Intercept 1, π_0					
Intercept 2, β_{00}	11.036	0.444	24.871	90	< .001
Premenstrual symptoms, β_{01}	-0.063	0.359	-0.177	90	.860
For luteal slope π_1					
Intercept 2, β_{10}	0.046	0.208	0.223	2114	.824
Premenstrual symptoms, β_{11}	0.477	0.168	2.845	2114	.004*

Note. * $p < .05$.

Table 40

Multilevel Model Results Predicting General Distress From Luteal Phase and Depressive Symptom Severity Interaction Using Full Maximum Likelihood Estimation (N = 94)

	Final estimation of fixed effects				
	Coefficient	*SE*	*t*-ratio	Appx *d.f.*	*p*-value
For Intercept 1, π_0					
Intercept 2, β_{00}	33.760	1.191	28.345	90	< .001
Depressive symptoms, β_{01}	0.191	0.199	0.959	90	.340
For luteal slope π_1					
Intercept 2, β_{10}	1.057	0.519	2.032	2106	.042
Depressive symptoms, β_{11}	0.5236	0.087	2.713	2106	.007*

Note. * $p < .05$.

Table 41

Multilevel Model Results Predicting Anhedonic Depression From Luteal Phase and Depressive Symptom Severity Interaction Using Full Maximum Likelihood Estimation (N = 94)

	Final estimation of fixed effects				
	Coefficient	*SE*	*t*-ratio	Appx *d.f.*	*p*-value
For Intercept 1, π_0					
Intercept 2, β_{00}	56.032	1.172	47.817	90	< .001
Depressive symptoms, β_{01}	0.067	0.196	0.341	90	.734
For luteal slope π_1					
Intercept 2, β_{10}	-0.012	0.527	-0.022	2106	.982
Depressive symptoms, β_{11}	-0.057	0.088	-0.652	2106	.514

Note. * $p < .05$.

Table 42

Multilevel Model Results Predicting Anxious Arousal From Luteal Phase and Depressive Symptom Severity Interaction Using Full Maximum Likelihood Estimation (N = 94)

	Final estimation of fixed effects				
	Coefficient	*SE*	*t*-ratio	Appx *d.f.*	*p*-value
For Intercept 1, π_0					
Intercept 2, β_{00}	13.773	0.509	27.018	90	< .001
Depressive symptoms, β_{01}	-0.005	0.085	-0.063	90	.950
For luteal slope π_1					
Intercept 2, β_{10}	0.282	0.201	1.405	2106	.160
Depressive symptoms, β_{11}	0.054	0.034	1.621	2106	.105

Note. * $p < .05$.

Table 43

Multilevel Model Results Predicting Irritability From Luteal Phase and Depressive Symptom Severity Interaction Using Full Maximum Likelihood Estimation (N = 94)

	Final estimation of fixed effects				
	Coefficient	*SE*	*t*-ratio	Appx *d.f.*	*p*-value
For Intercept 1, π_0					
Intercept 2, β_{00}	1.962	0.093	21.170	90	< .001
Depressive symptoms, β_{01}	0.013	0.016	0.857	90	.394
For luteal slope π_1					
Intercept 2, β_{10}	0.072	0.044	1.649	2108	.099
Depressive symptoms, β_{11}	0.013	0.007	1.813	2108	.070

Note. * $p < .05$.

Table 44

Multilevel Model Results Predicting Anger From Luteal Phase and Depressive Symptom Severity Interaction Using Full Maximum Likelihood Estimation (N = 94)

	Final estimation of fixed effects				
	Coefficient	*SE*	*t*-ratio	Appx *d.f.*	*p*-value
For Intercept 1, π_0					
Intercept 2, β_{00}	6.733	0.361	18.641	90	< .001
Depressive symptoms, β_{01}	0.039	0.061	0.660	90	.511
For luteal slope π_1					
Intercept 2, β_{10}	0.436	0.178	2.447	2113	.014
Depressive symptoms, β_{11}	0.036	0.029	1.205	2113	.228

Note. * $p < .05$.

Table 45

Multilevel Model Results Predicting Disgust From Luteal Phase and Depressive Symptom Severity Interaction Using Full Maximum Likelihood Estimation (N = 94)

	Final estimation of fixed effects				
	Coefficient	*SE*	*t*-ratio	Appx *d.f.*	*p*-value
For Intercept 1, π_0					
Intercept 2, β_{00}	5.917	0.269	21.940	90	< .001
Depressive symptoms, β_{01}	0.019	0.045	0.419	90	.676
For luteal slope π_1					
Intercept 2, β_{10}	-0.024	0.122	-0.201	2113	.840
Depressive symptoms, β_{11}	0.038	0.020	1.852	2113	.064

Note. * $p < .05$.

Table 46

Multilevel Model Results Predicting Anxiety From Luteal Phase and Depressive Symptom Severity Interaction Using Full Maximum Likelihood Estimation (N = 94)

	Final estimation of fixed effects				
	Coefficient	*SE*	*t*-ratio	Appx *d.f.*	*p*-value
For Intercept 1, π_0					
Intercept 2, β_{00}	8.920	0.369	24.204	90	< .001
Depressive symptoms, β_{01}	0.096	0.062	1.552	90	.124
For luteal slope π_1					
Intercept 2, β_{10}	-0.061	0.194	-0.317	2113	0.751
Depressive symptoms, β_{11}	0.057	0.032	1.711	2113	.077

Note. * $p < .05$.

Table 47

Multilevel Model Results Predicting Fear From Luteal Phase and Depressive Symptom Severity Interaction Using Full Maximum Likelihood Estimation (N = 94)

	Final estimation of fixed effects				
	Coefficient	*SE*	*t*-ratio	Appx *d.f.*	*p*-value
For Intercept 1, π_0					
Intercept 2, β_{00}	6.047	0.314	19.287	90	< .001
Depressive symptoms, β_{01}	0.061	0.053	1.170	90	.245
For luteal slope π_1					
Intercept 2, β_{10}	0.129	0.139	0.933	2113	.351
Depressive symptoms, β_{11}	0.056	0.023	2.413	2113	.016*

Note. * $p < .05$.

Table 48

Multilevel Model Results Predicting Sadness From Luteal Phase and Depressive Symptom Severity Interaction Using Full Maximum Likelihood Estimation (N = 94)

	Final estimation of fixed effects				
	Coefficient	*SE*	*t*-ratio	Appx *d.f.*	*p*-value
For Intercept 1, π_0					
Intercept 2, β_{00}	7.668	0.374	20.500	90	< .001
Depressive symptoms, β_{01}	0.042	0.063	0.668	90	.506
For luteal slope π_1					
Intercept 2, β_{10}	0.017	0.173	0.098	2112	.922
Depressive symptoms, β_{11}	0.039	0.029	0.029	2112	.177

Note. * $p < .05$.

Table 49

Multilevel Model Results Predicting Desire From Luteal Phase and Depressive Symptom Severity Interaction Using Full Maximum Likelihood Estimation (N = 94)

	Final estimation of fixed effects				
	Coefficient	*SE*	*t*-ratio	Appx *d.f.*	*p*-value
For Intercept 1, π_0					
Intercept 2, β_{00}	8.338	0.413	20.202	90	< .001
Depressive symptoms, β_{01}	0.063	0.069	0.915	90	.362
For luteal slope π_1					
Intercept 2, β_{10}	-0.134	0.154	-0.877	2112	0.381
Depressive symptoms, β_{11}	0.038	0.026	1.467	2112	.143

Note. * $p < .05$.

Table 50

Multilevel Model Results Predicting Relaxation From Luteal Phase and Depressive Symptom Severity Interaction Using Full Maximum Likelihood Estimation (N = 94)

	Final estimation of fixed effects				
	Coefficient	*SE*	*t*-ratio	Appx *d.f.*	*p*-value
For Intercept 1, π_0					
Intercept 2, β_{00}	10.682	0.403	26.505	90	< .001
Depressive symptoms, β_{01}	-0.006	0.068	-0.092	90	.927
For luteal slope π_1					
Intercept 2, β_{10}	-0.174	.202	-0.861	2113	.389
Depressive symptoms, β_{11}	0.031	0.034	0.918	2113	.359

Note. * $p < .05$.

Table 51

Multilevel Model Results Predicting Happiness From Luteal Phase and Depressive Symptom Severity Interaction Using Full Maximum Likelihood Estimation (N = 94)

	Final estimation of fixed effects				
	Coefficient	*SE*	*t*-ratio	Appx *d.f.*	*p*-value
For Intercept 1, π_0					
Intercept 2, β_{00}	11.038	0.444	24.888	90	< .001
Depressive symptoms, β_{01}	0.036	0.074	0.485	90	.629
For luteal slope π_1					
Intercept 2, β_{10}	00.054.282	0.208	0.261	2114	.794
Depressive symptoms, β_{11}	0.049	0.035	1.418	2114	.156

Note. * $p < .05$.

Table 52

Independent Samples Test of Mean Differences in Anxiety Sensitivity for High and Low Levels of Premenstrual Symptoms

		Levene's test for equality of variances		*t*-test for equality of means			
		F	Sig.	*t*	*df*	Sig. (2-tailed)	Mean difference
ASITot	Equal variances assumed	1.781	.185	4.691*	91	.000	15.47877

Note. *$p < .05$

Table 53

Independent Samples Test of Mean Differences in Anxiety Sensitivity for High and Low Levels of Depressive Symptoms

		Levene's test for equality of variances		*t*-test for equality of means			
		F	Sig.	*t*	*df*	Sig. (2-tailed)	Mean difference
ASITot	Equal variances assumed	.328	.569	.168	90	.867	.73126

Note. * $p < .05$.

Table 54

Multilevel Model Results Predicting General Distress From Luteal Phase and Anxiety Sensitivity Interaction in women with Severe Premenstrual Symptoms Using Full Maximum Likelihood Estimation (N = 94)

	Final estimation of fixed effects				
	Coefficient	*SE*	*t*-ratio	Appx *d.f.*	*p*-value
For Intercept 1, π_0					
Intercept 2, β_{00}	42.123	2.977	14.149	18	< .001
Anxiety sensitivity, β_{01}	0.490	0.194	2.525	18	.021
For luteal slope π_1					
Intercept 2, β_{10}	3.058	1.570	1.948	458	.052
Anxiety sensitivity, β_{11}	0.289	2.571	2.571	458	.010*

Note. * $p < .05$.

Table 55

Multilevel Model Results Predicting Anhedonic Depression From Luteal Phase and Anxiety Sensitivity Interaction in women with Severe Premenstrual Symptoms Using Full Maximum Likelihood Estimation (N = 94)

	Final estimation of fixed effects				
	Coefficient	*SE*	*t*-ratio	Appx *d.f.*	*p*-value
For Intercept 1, π_0					
Intercept 2, β_{00}	51.644	2.631	19.632	18	< .001
Anxiety sensitivity, β_{01}	-0.263	0.172	-1.535	18	.142
For luteal slope π_1					
Intercept 2, β_{10}	1.854	1.355	1.368	458	.172
Anxiety sensitivity, β_{11}	-0.114	0.097	-1.174	458	0.241

Note. * $p < .05$.

Table 56

Multilevel Model Results Predicting Anxious Arousal From Luteal Phase and Anxiety Sensitivity Interaction in women with Severe Premenstrual Symptoms Using Full Maximum Likelihood Estimation (N = 20)

	Final estimation of fixed effects				
	Coefficient	*SE*	*t*-ratio	Appx *d.f.*	*p*-value
For Intercept 1, π_0					
Intercept 2, β_{00}	17.253	1.329	12.975	18	< .001
Anxiety sensitivity, β_{01}	0.231	0.087	2.661	18	.016
For luteal slope π_1					
Intercept 2, β_{10}	0.236	0.661	0.357	458	.721
Anxiety sensitivity, β_{11}	0.081	0.047	1.712	458	.088

Note. * $p < .05$.

Table 57

Multilevel Model Results Predicting Irritability From Luteal Phase and Anxiety Sensitivity Interaction in women with Severe Premenstrual Symptoms Using Full Maximum Likelihood Estimation (N = 20)

	Final estimation of fixed effects				
	Coefficient	*SE*	*t*-ratio	Appx *d.f.*	*p*-value
For Intercept 1, π_0					
Intercept 2, β_{00}	2.419	0.241	10.055	18	< .001
Anxiety sensitivity, β_{01}	0.024	0.016	1.502	18	.150
For luteal slope π_1					
Intercept 2, β_{10}	0.083	0.123	0.675	459	.500
Anxiety sensitivity, β_{11}	0.017	0.009	1.923	459	.055

Note. * $p < .05$.

Table 58

Multilevel Model Results Predicting Anger From Luteal Phase and Anxiety Sensitivity Interaction in women with Severe Premenstrual Symptoms Using Full Maximum Likelihood Estimation (N = 20)

	Final estimation of fixed effects				
	Coefficient	*SE*	*t*-ratio	Appx *d.f.*	*p*-value
For Intercept 1, π_0					
Intercept 2, β_{00}	8.379	0.963	8.698	18	< .001
Anxiety sensitivity, β_{01}	0.141	0.063	2.248	18	.0037
For luteal slope π_1					
Intercept 2, β_{10}	1.183	0.529	2.236	459	.026
Anxiety sensitivity, β_{11}	0.102	0.038	2.689	459	.007*

Note. * $p < .05$.

Table 59

Multilevel Model Results Predicting Disgust From Luteal Phase and Anxiety Sensitivity Interaction in women with Severe Premenstrual Symptoms Using Full Maximum Likelihood Estimation (N = 20)

	Final estimation of fixed effects				
	Coefficient	*SE*	*t*-ratio	Appx *d.f.*	*p*-value
For Intercept 1, π_0					
Intercept 2, β_{00}	7.357	0.691	10.647	18	< .001
Anxiety sensitivity, β_{01}	0.082	0.045	1.823	18	.085
For luteal slope π_1					
Intercept 2, β_{10}	0.385	0.397	0.970	459	.333
Anxiety sensitivity, β_{11}	0.054	0.028	1.905	459	.057

Note. * $p < .05$.

Table 60

Multilevel Model Results Predicting Anxiety From Luteal Phase and Anxiety Sensitivity Interaction in women with Severe Premenstrual Symptoms Using Full Maximum Likelihood Estimation (N = 20)

	Final estimation of fixed effects				
	Coefficient	*SE*	*t*-ratio	Appx *d.f.*	*p*-value
For Intercept 1, π_0					
Intercept 2, β_{00}	11.767	0.919	12.808	18	< .001
Anxiety sensitivity, β_{01}	0.055	0.059	0.921	18	.369
For luteal slope π_1					
Intercept 2, β_{10}	0.255	0.576	0.443	459	.658
Anxiety sensitivity, β_{11}	0.088	0.041	2.149	459	.032*

Note. * $p < .05$.

Table 61

Multilevel Model Results Predicting Sadness From Luteal Phase and Anxiety Sensitivity Interaction in women with Severe Premenstrual Symptoms Using Full Maximum Likelihood Estimation (N = 20)

	Final estimation of fixed effects				
	Coefficient	*SE*	*t*-ratio	Appx *d.f.*	*p*-value
For Intercept 1, π_0					
Intercept 2, β_{00}	9.861	0.959	10.279	18	< .001
Anxiety sensitivity, β_{01}	0.113	0.063	1.811	18	.087
For luteal slope π_1					
Intercept 2, β_{10}	0.574	0.492	1.167	459	.244
Anxiety sensitivity, β_{11}	0.071	0.035	2.019	459	.044*

Note. * $p < .05$.

Table 62

Multilevel Model Results Predicting Fear From Luteal Phase and Anxiety Sensitivity Interaction in women with Severe Premenstrual Symptoms Using Full Maximum Likelihood Estimation (N = 20)

	Final estimation of fixed effects				
	Coefficient	*SE*	*t*-ratio	Appx *d.f.*	*p*-value
For Intercept 1, π_0					
Intercept 2, β_{00}	7.148	0.861	8.303	18	< .001
Anxiety sensitivity, β_{01}	0.118	0.056	2.098	18	.050
For luteal slope π_1					
Intercept 2, β_{10}	0.172	0.433	0.397	459	.692
Anxiety sensitivity, β_{11}	0.014	0.031	0.458	459	.647

Note. * $p < .05$.

Table 63

Multilevel Model Results Predicting Desire From Luteal Phase and Anxiety Sensitivity Interaction in women with Severe Premenstrual Symptoms Using Full Maximum Likelihood Estimation (N = 20)

	Final estimation of fixed effects				
	Coefficient	*SE*	*t*-ratio	Appx *d.f.*	*p*-value
For Intercept 1, π_0					
Intercept 2, β_{00}	11.004	1.25	8.799	18	< .001
Anxiety sensitivity, β_{01}	0.023	0.081	0.287	18	.777
For luteal slope π_1					
Intercept 2, β_{10}	0.252	0.481	0.523	459	.601
Anxiety sensitivity, β_{11}	0.036	0.034	1.056	459	.292

Note. * $p < .05$.

Table 64

Multilevel Model Results Predicting Relaxation From Luteal Phase and Anxiety Sensitivity Interaction in women with Severe Premenstrual Symptoms Using Full Maximum Likelihood Estimation (N = 20)

	Final estimation of fixed effects				
	Coefficient	*SE*	*t*-ratio	Appx *d.f.*	*p*-value
For Intercept 1, π_0					
Intercept 2, β_{00}	10.427	0.776	13.441	18	< .001
Anxiety sensitivity, β_{01}	-0.061	0.051	-1.196	18	.247
For luteal slope π_1					
Intercept 2, β_{10}	0.705	0.541	1.304	459	.193
Anxiety sensitivity, β_{11}	-0.051	0.039	-1.328	459	.185

Note. * $p < .05$.

Table 65

Multilevel Model Results Predicting Happiness From Luteal Phase and Anxiety Sensitivity Interaction in women with Severe Premenstrual Symptoms Using Full Maximum Likelihood Estimation (N = 20)

	Final estimation of fixed effects				
	Coefficient	*SE*	*t*-ratio	Appx *d.f.*	*p*-value
For Intercept 1, π_0					
Intercept 2, β_{00}	10.878	0.932	11.672	18	< .001
Anxiety sensitivity, β_{01}	-0.070	0.061	-1.153	18	.264
For luteal slope π_1					
Intercept 2, β_{10}	1.137	0.500	2.274	459	.023
Anxiety sensitivity, β_{11}	-0.012	0.036	-0.344	459	.731

Note. * $p < .05$.

Table 66

Multilevel Model Results Predicting General Distress From Luteal Phase and Anxiety Sensitivity Interaction in women with Severe Depressive Symptoms Using Full Maximum Likelihood Estimation (N = 13)

	Final estimation of fixed effects				
	Coefficient	*SE*	*t*-ratio	Appx *d.f.*	*p*-value
For Intercept 1, π_0					
Intercept 2, β_{00}	36.886	2.362	15.615	11	< .001
Anxiety sensitivity, β_{01}	0.477	0.199	2.389	11	.036
For luteal slope π_1					
Intercept 2, β_{10}	4.024	1.773	2.269	310	.024
Anxiety sensitivity, β_{11}	0.200	0.155	1.290	310	.198

Note. * $p < .05$.

Table 67

Multilevel Model Results Predicting Anhedonic Depression From Luteal Phase and Anxiety Sensitivity Interaction in women with Severe Depressive Symptoms Using Full Maximum Likelihood Estimation (N = 13)

	Final estimation of fixed effects				
	Coefficient	*SE*	*t*-ratio	Appx *d.f.*	*p*-value
For Intercept 1, π_0					
Intercept 2, β_{00}	57.445	2.463	23.325	11	< .001
Anxiety sensitivity, β_{01}	-0.527	0.208	-2.533	11	.028
For luteal slope π_1					
Intercept 2, β_{10}	-1.187	1.731	-0.686	10	.493
Anxiety sensitivity, β_{11}	-0.128	0.151	-0.845	310	.399

Note. * $p < .05$.

Table 68

Multilevel Model Results Predicting Anxious Arousal From Luteal Phase and Anxiety Sensitivity Interaction in women with Severe Depressive Symptoms Using Full Maximum Likelihood Estimation (N = 13)

	Final estimation of fixed effects				
	Coefficient	*SE*	*t*-ratio	Appx *d.f.*	*p*-value
For Intercept 1, π_0					
Intercept 2, β_{00}	14.091	0.846	16.660	11	< .001
Anxiety sensitivity, β_{01}	0.162	0.072	2.258	11	.045
For luteal slope π_1					
Intercept 2, β_{10}	0.296	0.695	0.426	310	.671
Anxiety sensitivity, β_{11}	0.058	0.061	0.949	310	.344

Note. * $p < .05$.

Table 69

Multilevel Model Results Predicting Irritability From Luteal Phase and Anxiety Sensitivity Interaction in women with Severe Depressive Symptoms Using Full Maximum Likelihood Estimation (N = 13)

	Final estimation of fixed effects				
	Coefficient	*SE*	*t*-ratio	Appx *d.f.*	*p*-value
For Intercept 1, π_0					
Intercept 2, β_{00}	1.978	0.166	11.944	11	< .001
Anxiety sensitivity, β_{01}	0.021	0.014	1.522	11	.156
For luteal slope π_1					
Intercept 2, β_{10}	0.335	0.137	2.488	310	.015
Anxiety sensitivity, β_{11}	0.009	0.012	0.733	310	.464

Note. * $p < .05$.

Table 70

Multilevel Model Results Predicting Anger From Luteal Phase and Anxiety Sensitivity Interaction in women with Severe Depressive Symptoms Using Full Maximum Likelihood Estimation (N = 13)

	Final estimation of fixed effects				
	Coefficient	*SE*	*t*-ratio	Appx *d.f.*	*p*-value
For Intercept 1, π_0					
Intercept 2, β_{00}	6.589	0.712	9.251	11	< .001
Anxiety sensitivity, β_{01}	0.039	0.060	0.644	11	.533
For luteal slope π_1					
Intercept 2, β_{10}	1.176	0.516	2.281	310	.023
Anxiety sensitivity, β_{11}	0.055	0.045	1.211	310	.227

Note. * $p < .05$.

Table 71

Multilevel Model Results Predicting Disgust From Luteal Phase and Anxiety Sensitivity Interaction in women with Severe Depressive Symptoms Using Full Maximum Likelihood Estimation (N = 13)

	Final estimation of fixed effects				
	Coefficient	*SE*	*t*-ratio	Appx *d.f.*	*p*-value
For Intercept 1, π_0					
Intercept 2, β_{00}	6.177	0.707	8.744	11	< .001
Anxiety sensitivity, β_{01}	0.062	0.059	1.043	11	.319
For luteal slope π_1					
Intercept 2, β_{10}	0.087	0.390	0.223	310	.824
Anxiety sensitivity, β_{11}	0.077	0.034	2.252	310	.025*

Note. * $p < .05$.

Table 72

Multilevel Model Results Predicting Fear From Luteal Phase and Anxiety Sensitivity Interaction in women with Severe Depressive Symptoms Using Full Maximum Likelihood Estimation (N = 13)

	Final estimation of fixed effects				
	Coefficient	*SE*	*t*-ratio	Appx *d.f.*	*p*-value
For Intercept 1, π_0					
Intercept 2, β_{00}	6.319	0.385	16.428	11	< .001
Anxiety sensitivity, β_{01}	0.039	0.033	1.200	11	.256
For luteal slope π_1					
Intercept 2, β_{10}	0.533	0.469	1.136	310	.257
Anxiety sensitivity, β_{11}	0.011	0.041	0.278	310	.781

Note. * $p < .05$.

Table 73

Multilevel Model Results Predicting Anxiety From Luteal Phase and Anxiety Sensitivity Interaction in women with Severe Depressive Symptoms Using Full Maximum Likelihood Estimation (N = 13)

	Final estimation of fixed effects				
	Coefficient	*SE*	*t*-ratio	Appx *d.f.*	*p*-value
For Intercept 1, π_0					
Intercept 2, β_{00}	10.486	0.788	13.301	11	< .001
Anxiety sensitivity, β_{01}	0.109	0.067	1.639	11	.130
For luteal slope π_1					
Intercept 2, β_{10}	0.895	0.638	1.403	310	.162
Anxiety sensitivity, β_{11}	-0.017	0.056	-0.307	310	.759

Note. * $p < .05$.

Table 74

Multilevel Model Results Predicting Sadness From Luteal Phase and Anxiety Sensitivity Interaction in women with Severe Depressive Symptoms Using Full Maximum Likelihood Estimation (N = 13)

	Final estimation of fixed effects				
	Coefficient	*SE*	*t*-ratio	Appx *d.f.*	*p*-value
For Intercept 1, π_0					
Intercept 2, β_{00}	8.235	0.565	14.571	11	< .001
Anxiety sensitivity, β_{01}	0.088	0.048	1.830	11	.094
For luteal slope π_1					
Intercept 2, β_{10}	0.514	0.532	0.967	310	.334
Anxiety sensitivity, β_{11}	0.028	0.047	0.600	310	.549

Note. * $p < .05$.

Table 75

Multilevel Model Results Predicting Desire From Luteal Phase and Anxiety Sensitivity Interaction in women with Severe Depressive Symptoms Using Full Maximum Likelihood Estimation (N = 13)

	Final estimation of fixed effects				
	Coefficient	*SE*	*t*-ratio	Appx *d.f.*	*p*-value
For Intercept 1, π_0					
Intercept 2, β_{00}	10.041	0.930	10.797	11	< .001
Anxiety sensitivity, β_{01}	0.005	0.079	0.059	11	.954
For luteal slope π_1					
Intercept 2, β_{10}	0.271	0.576	0.470	310	.639
Anxiety sensitivity, β_{11}	0.049	0.050	0.980	310	.328

Note. * $p < .05$.

Table 76

Multilevel Model Results Predicting Relaxation From Luteal Phase and Anxiety Sensitivity Interaction in women with Severe Depressive Symptoms Using Full Maximum Likelihood Estimation (N = 13)

	Final estimation of fixed effects				
	Coefficient	*SE*	*t*-ratio	Appx *d.f.*	*p*-value
For Intercept 1, π_0					
Intercept 2, β_{00}	10.769	0.964	11.169	11	< .001
Anxiety sensitivity, β_{01}	-0.167	0.081	-2.057	11	.064
For luteal slope π_1					
Intercept 2, β_{10}	-0.031	0.608	-0.051	310	.959
Anxiety sensitivity, β_{11}	-0.057	0.053	-1.071	310	.285

Note. * $p < .05$.

Table 77

Multilevel Model Results Predicting Happiness From Luteal Phase and Anxiety Sensitivity Interaction in women with Severe Depressive Symptoms Using Full Maximum Likelihood Estimation (N = 13)

	Final estimation of fixed effects				
	Coefficient	*SE*	*t*-ratio	Appx *d.f.*	*p*-value
For Intercept 1, π_0					
Intercept 2, β_{00}	11.866	0.946	12.537	11	< .001
Anxiety sensitivity, β_{01}	-0.139	0.080	-1.736	11	.111
For luteal slope π_1					
Intercept 2, β_{10}	0.334	0.684	0.488	310	.626
Anxiety sensitivity, β_{11}	-0.017	0.059	-0.275	210	.783

Note. * $p < .05$.

Table 78

Independent Samples Test of Mean Differences in Rumination for High and Low Levels of Premenstrual Symptoms

		Levene's test for equality of variances		*t*-test for equality of means			
		F	Sig.	*t*	*df*	Sig. (2-tailed)	Mean difference
Rumination	Equal variances assumed	2.509	.117	2.372	92	.020	8.47973

Note. *$p < .05$

Table 79

Independent Samples Test of Mean Differences in Rumination for High and Low Levels of Depressive Symptoms

		Levene's test for equality of variances		*t*-test for equality of means			
		F	Sig.	*t*	*df*	Sig. (2-tailed)	Mean difference
Rumination	Equal variances assumed	.821	.367	-.246	91	.806	-1.06442

Note. *$p < .05$

Table 80

Multilevel Model Results Predicting General Distress From Luteal Phase and Rumination Interaction in women with Severe Premenstrual Symptoms Using Full Maximum Likelihood Estimation (N = 20)

	Final estimation of fixed effects				
	Coefficient	*SE*	*t*-ratio	Appx *d.f.*	*p*-value
For Intercept 1, π_0					
Intercept 2, β_{00}	42.006	3.244	12.959	18	< .001
Rumination, β_{01}	0.278	0.186	1.491	18	.153
For luteal slope π_1					
Intercept 2, β_{10}	2.729	1.531	1.783	458	.075
Rumination, β_{11}	0.375	0.087	4.289	458	< .001*

Note. * $p < .05$.

Table 81

Multilevel Model Results Predicting Anhedonic Depression From Luteal Phase and Rumination Interaction in women with Severe Premenstrual Symptoms Using Full Maximum Likelihood Estimation (N = 20)

	Final estimation of fixed effects				
	Coefficient	*SE*	*t*-ratio	Appx *d.f.*	*p*-value
For Intercept 1, π_0					
Intercept 2, β_{00}	51.709	2.504	20.647	18	< .001
Rumination, β_{01}	-0.299	0.144	-2.077	18	.052
For luteal slope π_1					
Intercept 2, β_{10}	1.879	1.319	1.424	458	.155
Rumination, β_{11}	-0.275	0.075	-3.647	458	< .001*

Note. * $p < .05$.

Table 82

Multilevel Model Results Predicting Anxious Arousal From Luteal Phase and Rumination Interaction in women with Severe Premenstrual Symptoms Using Full Maximum Likelihood Estimation (N = 20)

	Final estimation of fixed effects				
	Coefficient	*SE*	*t*-ratio	Appx *d.f.*	*p*-value
For Intercept 1, π_0					
Intercept 2, β_{00}	17.207	1.559	11.036	18	< .001
Rumination, β_{01}	0.023	0.089	0.255	18	.802
For luteal slope π_1					
Intercept 2, β_{10}	0.164	0.648	0.253	458	.800
Rumination, β_{11}	0.115	0.037	3.091	458	.002*

Note. * $p < .05$.

Table 83

Multilevel Model Results Predicting Irritability From Luteal Phase and Rumination Interaction in women with Severe Premenstrual Symptoms Using Full Maximum Likelihood Estimation (N = 20)

	Final estimation of fixed effects				
	Coefficient	*SE*	*t*-ratio	Appx *d.f.*	*p*-value
For Intercept 1, π_0					
Intercept 2, β_{00}	2.413	0.246	9.800	18	< .001
Premenstrual symptoms, β_{01}	0.0154	0.0142	1.081	18	.294
For luteal slope π_1					
Intercept 2, β_{10}	0.062	0.121	0.513	459	.608
Premenstrual symptoms, β_{11}	0.021	0.007	2.976	459	.003*

Note. * $p < .05$.

Table 84

Multilevel Model Results Predicting Anger From Luteal Phase and Rumination Interaction in women with Severe Premenstrual Symptoms Using Full Maximum Likelihood Estimation (N = 20)

	Final estimation of fixed effects				
	Coefficient	*SE*	*t*-ratio	Appx *d.f.*	*p*-value
For Intercept 1, π_0					
Intercept 2, β_{00}	8.339	1.060	7.866	18	< .001
Rumination, β_{01}	0.050	0.061	0.082	18	.423
For luteal slope π_1					
Intercept 2, β_{10}	1.053	0.519	2.031	459	.043
Rumination, β_{11}	0.113	0.029	3.817	459	< .001*

Note. * $p < .05$.

Table 85

Multilevel Model Results Predicting Disgust From Luteal Phase and Rumination Interaction in women with Severe Premenstrual Symptoms Using Full Maximum Likelihood Estimation (N = 20)

	Final estimation of fixed effects				
	Coefficient	*SE*	*t*-ratio	Appx *d.f.*	*p*-value
For Intercept 1, π_0					
Intercept 2, β_{00}	7.328	0.742	9.872	18	< .001
Premenstrual symptoms, β_{01}	-0.016	0.043	-0.376	18	.711
For luteal slope π_1					
Intercept 2, β_{10}	0.325	0.390	0.832	459	.406
Premenstrual symptoms, β_{11}	0.067	0.022	2.985	459	.003*

Note. * $p < .05$.

Table 86

Multilevel Model Results Predicting Fear From Luteal Phase and Rumination Interaction in women with Severe Premenstrual Symptoms Using Full Maximum Likelihood Estimation (N = 20)

	Final estimation of fixed effects				
	Coefficient	*SE*	*t*-ratio	Appx *d.f.*	*p*-value
For Intercept 1, π_0					
Intercept 2, β_{00}	7.129	0.944	7.551	18	< .001
Premenstrual symptoms, β_{01}	0.043	0.054	0.799	18	.434
For luteal slope π_1					
Intercept 2, β_{10}	0.192	0.425	0.452	459	.651
Premenstrual symptoms, β_{11}	0.054	0.024	2.222	459	.027*

Note. * $p < .05$.

Table 87

Multilevel Model Results Predicting Anxiety From Luteal Phase and Rumination Interaction in women with Severe Premenstrual Symptoms Using Full Maximum Likelihood Estimation (N = 20)

	Final estimation of fixed effects				
	Coefficient	*SE*	*t*-ratio	Appx *d.f.*	*p*-value
For Intercept 1, π_0					
Intercept 2, β_{00}	11.739	0.923	12.719	18	< .001
Premenstrual symptoms, β_{01}	0.029	0.053	0.551	18	.588
For luteal slope π_1					
Intercept 2, β_{10}	0.125	0.568	0.221	459	.825
Premenstrual symptoms, β_{11}	0.086	0.032	2.657	459	.008*

Note. * $p < .05$.

Table 88

Multilevel Model Results Predicting Sadness From Luteal Phase and Rumination Interaction in women with Severe Premenstrual Symptoms Using Full Maximum Likelihood Estimation (N = 20)

	Final estimation of fixed effects				
	Coefficient	*SE*	*t*-ratio	Appx *d.f.*	*p*-value
For Intercept 1, π_0					
Intercept 2, β_{00}	9.835	0.997	9.859	18	< .001
Premenstrual symptoms, β_{01}	0.068	0.057	1.186	18	.251
For luteal slope π_1					
Intercept 2, β_{10}	00.474.125	0.484	0.981	45	.327
Premenstrual symptoms, β_{11}	0.075	0.028	2.715	459	.007*

Note. * $p < .05$.

Table 89

Multilevel Model Results Predicting Desire From Luteal Phase and Rumination Interaction in women with Severe Premenstrual Symptoms Using Full Maximum Likelihood Estimation (N = 20)

	Final estimation of fixed effects				
	Coefficient	*SE*	*t*-ratio	Appx *d.f.*	*p*-value
For Intercept 1, π_0					
Intercept 2, β_{00}	11.000	1.244	8.841	18	< .001
Premenstrual symptoms, β_{01}	0.029	0.072	0.413	18	.684
For luteal slope π_1					
Intercept 2, β_{10}	0.169	0.475	0.355	459	.723
Premenstrual symptoms, β_{11}	0.005	0.027	0.187	459	.852

Note. * $p < .05$.

Table 90

Multilevel Model Results Predicting Relaxation From Luteal Phase and Rumination Interaction in women with Severe Premenstrual Symptoms Using Full Maximum Likelihood Estimation (N = 20)

	Final estimation of fixed effects				
	Coefficient	*SE*	*t*-ratio	Appx *d.f.*	*p*-value
For Intercept 1, π_0					
Intercept 2, β_{00}	10.444	0.773	13.518	18	< .001
Premenstrual symptoms, β_{01}	-0.055	0.044	-1.240	18	.231
For luteal slope π_1					
Intercept 2, β_{10}	00.781.125	0.533	1.465	459	.144
Premenstrual symptoms, β_{11}	-0.055	0.030	-1.800	459	.073

Note. * $p < .05$.

Table 91

Multilevel Model Results Predicting Happiness From Luteal Phase and Rumination Interaction in women with Severe Premenstrual Symptoms Using Full Maximum Likelihood Estimation (N = 20)

	Final estimation of fixed effects				
	Coefficient	*SE*	*t*-ratio	Appx *d.f.*	*p*-value
For Intercept 1, π_0					
Intercept 2, β_{00}	10.895	0.917	11.879	18	< .001
Premenstrual symptoms, β_{01}	-0.073	0.057	-1.381	18	.184
For luteal slope π_1					
Intercept 2, β_{10}	1.097	0.489	2.242	459	.025
Premenstrual symptoms, β_{11}	-0.077	0.028	-2.744	459	.006*

Note. * $p < .05$.

Table 92

Multilevel Model Results Predicting General Distress From Luteal Phase and Rumination Interaction in women with Severe Depressive Symptoms Using Full Maximum Likelihood Estimation (N = 13)

	Final estimation of fixed effects				
	Coefficient	*SE*	*t*-ratio	Appx *d.f.*	*p*-value
For Intercept 1, π_0					
Intercept 2, β_{00}	36.869	2.580	14.288	11	< .001
Rumination, β_{01}	0.253	0.151	1.673	11	.123
For luteal slope π_1					
Intercept 2, β_{10}	4.023	1.749	2.300	310	.022
Rumination, β_{11}	0.312	0.101	3.092	310	.002*

Note. * $p < .05$.

Table 93

Multilevel Model Results Predicting Anhedonic Depression From Luteal Phase and Rumination Interaction in women with Severe Depressive Symptoms Using Full Maximum Likelihood Estimation (N = 13)

	Final estimation of fixed effects				
	Coefficient	*SE*	*t*-ratio	Appx *d.f.*	*p*-value
For Intercept 1, π_0					
Intercept 2, β_{00}	57.452	2.852	20.141	11	< .001
Rumination, β_{01}	-0.230	0.167	-1.376	11	.196
For luteal slope π_1					
Intercept 2, β_{10}	-1.191	1.718	-0.693	310	.489
Rumination, β_{11}	-0.203	0.099	-2.045	310	.042*

Note. * $p < .05$.

Table 94

Multilevel Model Results Predicting Anxious Arousal From Luteal Phase and Rumination Interaction in women with Severe Depressive Symptoms Using Full Maximum Likelihood Estimation (N = 13)

	Final estimation of fixed effects				
	Coefficient	*SE*	*t*-ratio	Appx *d.f.*	*p*-value
For Intercept 1, π_0					
Intercept 2, β_{00}	14.088	1.017	13.854	11	< .001
Rumination, β_{01}	0.012	0.060	0.195	11	.849
For luteal slope π_1					
Intercept 2, β_{10}	0.321	0.692	0.464	310	.643
Rumination, β_{11}	0.068	0.039	1.697	310	.091

Note. * $p < .05$.

Table 95

Multilevel Model Results Predicting Irritability From Luteal Phase and Rumination Interaction in women with Severe Depressive Symptoms Using Full Maximum Likelihood Estimation (N = 13)

	Final estimation of fixed effects				
	Coefficient	*SE*	*t*-ratio	Appx *d.f.*	*p*-value
For Intercept 1, π_0					
Intercept 2, β_{00}	1.977	0.177	11.138	11	< .001
Rumination, β_{01}	0.008	0.010	0.757	11	.465
For luteal slope π_1					
Intercept 2, β_{10}	0.336	0.136	2.474	310	.014
Rumination, β_{11}	0.014	0.008	1.731	310	.084

Note. * $p < .05$.

Table 96

Multilevel Model Results Predicting Anger From Luteal Phase and Rumination Interaction in women with Severe Depressive Symptoms Using Full Maximum Likelihood Estimation (N = 13)

	Final estimation of fixed effects				
	Coefficient	*SE*	*t*-ratio	Appx *d.f.*	*p*-value
For Intercept 1, π_0					
Intercept 2, β_{00}	6.583	0.725	9.084	11	< .001
Rumination, β_{01}	-0.009	0.042	-0.235	11	.819
For luteal slope π_1					
Intercept 2, β_{10}	1.188	0.512	2.323	310	.021
Rumination, β_{11}	0.069	0.030	2.319	310	.021*

Note. * $p < .05$.

Table 97

Multilevel Model Results Predicting Disgust From Luteal Phase and Rumination Interaction in women with Severe Depressive Symptoms Using Full Maximum Likelihood Estimation (N = 13)

	Final estimation of fixed effects				
	Coefficient	*SE*	*t*-ratio	Appx *d.f.*	*p*-value
For Intercept 1, π_0					
Intercept 2, β_{00}	6.179	0.729	8.467	11	< .001
Rumination, β_{01}	-0.023	0.043	-0.541	11	.599
For luteal slope π_1					
Intercept 2, β_{10}	0.120	0.388	0.309	310	.757
Rumination, β_{11}	0.060	0.022	2.676	310	.008*

Note. * $p < .05$.

Table 98

Multilevel Model Results Predicting Fear From Luteal Phase and Rumination Interaction in women with Severe Depressive Symptoms Using Full Maximum Likelihood Estimation (N = 13)

	Final estimation of fixed effects				
	Coefficient	*SE*	*t*-ratio	Appx *d.f.*	*p*-value
For Intercept 1, π_0					
Intercept 2, β_{00}	6.319	0.406	15.575	11	< .001
Rumination, β_{01}	0.007	0.024	0.313	11	.760
For luteal slope π_1					
Intercept 2, β_{10}	0.541	0.468	1.157	310	.248
Rumination, β_{11}	0.019	0.027	0.706	310	.481

Note. * $p < .05$.

Table 99

Multilevel Model Results Predicting Anxiety From Luteal Phase and Rumination Interaction in women with Severe Depressive Symptoms Using Full Maximum Likelihood Estimation (N = 13)

	Final estimation of fixed effects				
	Coefficient	*SE*	*t*-ratio	Appx *d.f.*	*p*-value
For Intercept 1, π_0					
Intercept 2, β_{00}	10.472	0.809	12.944	11	< .001
Rumination, β_{01}	0.066	0.047	1.384	11	.194
For luteal slope π_1					
Intercept 2, β_{10}	0.863	0.635	1.1359	310	.175
Rumination, β_{11}	0.044	0.037	1.200	310	.231

Note. * $p < .05$.

Table 100

Multilevel Model Results Predicting Sadness From Luteal Phase and Rumination Interaction in women with Severe Depressive Symptoms Using Full Maximum Likelihood Estimation (N = 13)

	Final estimation of fixed effects				
	Coefficient	*SE*	*t*-ratio	Appx *d.f.*	*p*-value
For Intercept 1, π_0					
Intercept 2, β_{00}	8.236	0.578	14.239	11	< .001
Rumination, β_{01}	0.055	0.034	1.620	11	.133
For luteal slope π_1					
Intercept 2, β_{10}	0.521	0.530	0.983	310	.327
Rumination, β_{11}	0.036	0.031	1.185	310	.237

Note. * $p < .05$.

Table 101

Multilevel Model Results Predicting Desire From Luteal Phase and Rumination Interaction in women with Severe Depressive Symptoms Using Full Maximum Likelihood Estimation (N = 13)

	Final estimation of fixed effects				
	Coefficient	*SE*	*t*-ratio	Appx *d.f.*	*p*-value
For Intercept 1, π_0					
Intercept 2, β_{00}	10.058	0.913	11.016	11	< .001
Rumination, β_{01}	0.037	0.054	0.692	11	.503
For luteal slope π_1					
Intercept 2, β_{10}	0.310	0.576	0.539	310	.591
Rumination, β_{11}	-0.011	0.033	-0.327	310	.744

Note. * $p < .05$.

Table 102

Multilevel Model Results Predicting Relaxation From Luteal Phase and Rumination Interaction in women with Severe Depressive Symptoms Using Full Maximum Likelihood Estimation (N = 13)

	Final estimation of fixed effects				
	Coefficient	*SE*	*t*-ratio	Appx *d.f.*	*p*-value
For Intercept 1, π_0					
Intercept 2, β_{00}	10.762	1.076	10.001	11	< .001
Rumination, β_{01}	-0.069	0.063	-1.094	11	.297
For luteal slope π_1					
Intercept 2, β_{10}	-0.061	0.607	-0.101	310	.920
Rumination, β_{11}	-0.038	0.035	-1.092	310	.276

Note. * $p < .05$.

Table 103

Multilevel Model Results Predicting Happiness From Luteal Phase and Rumination Interaction in women with Severe Depressive Symptoms Using Full Maximum Likelihood Estimation (N = 13)

	Final estimation of fixed effects				
	Coefficient	*SE*	*t*-ratio	Appx *d.f.*	*p*-value
For Intercept 1, π_0					
Intercept 2, β_{00}	11.873	1.039	11.426	11	< .001
Rumination, β_{01}	-0.048	0.061	-0.787	11	.448
For luteal slope π_1					
Intercept 2, β_{10}	0.341	0.682	0.500	310	.618
Rumination, β_{11}	-0.050	0.039	-1.282	310	.201

Note. * $p < .05$.

APPENDIX A: DEMOGRAPHICS SHEET

Age: _____ years old

Race/Ethnicity (please check all that apply):

- Caucasian / White
- African-American / Black
- Hispanic / Latino
- American Indian / Alaska Native
- Asian or Asian American
- Hawaiian or Other Pacific Islander

Marital Status:

- Single, never married
- Married
- Divorced or separated
- Widowed

Sexual Orientation:

- Heterosexual
- Bisexual
- Homosexual
- Other (please specify): _____

Have you experienced a menstrual cycle in the last 12 months?

- Yes
- No

Are you taking hormonal birth control?

- Yes
- No

Do you use any other form of birth control?

- ☐ Yes (please specify): _____
- ☐ No

Do you currently suffer from any of the following health conditions (check all that apply):

- ☐ Diabetes
- ☐ Hypertension (high blood pressure)
- ☐ High cholesterol
- ☐ Arthritis
- ☐ Migraines
- ☐ Heart disease
- ☐ Breast cancer
- ☐ Ovarian cancer
- ☐ Cervical cancer
- ☐ Other cancer (please specify): _____
- ☐ Human Papillomavirus (HPV)
- ☐ Polycystic ovary syndrome (PCOS)
- ☐ Ovarian cysts
- ☐ Pelvic Inflammatory Disease (PID)
- ☐ Menstrual irregularities
- ☐ Urinary tract infections
- ☐ Bacterial vaginosis
- ☐ Vaginitis
- ☐ Vulvodynia
- ☐ Uterine fibroids
- ☐ Pelvic floor disorders
- ☐ Yeast infections
- ☐ Endometriosis
- ☐ Osteoporosis
- ☐ Thyroid disease
- ☐ Irritable Bowel Syndrome (IBS)
- ☐ Crohn's Disease
- ☐ Fibromyalgia
- ☐ Other (please specify): _____

APPENDIX B: PATIENT HEALTH QUESTIONNAIRE SOMATIC, ANXIETY, AND DEPRESSIVE SYMPTOM SCALES

During the last 4 weeks, how much have you been bothered by any of the following problems?	Not bothered	Bothered a little	Bothered a lot
1. Stomach pain	0	1	2
2. Back pain	0	1	2
3. Pain in your arms, legs, or joints (knees, hips, etc.)	0	1	2
4. Feeling tired or having little energy	0	1	2
5. Trouble falling asleep or staying asleep, or sleeping too much	0	1	2
6. Menstrual cramps or other problems with your periods	0	1	2
7. Pain or problems during sexual intercourse	0	1	2
8. Headaches	0	1	2
9. Chest pain	0	1	2
10. Dizziness	0	1	2
11. Fainting spells	0	1	2
12. Feeling your heart pound or race	0	1	2
13. Shortness of breath	0	1	2
14. Constipation, loose bowels, or diarrhea	0	1	2
15. Nausea, gas, or indigestion	0	1	2

Over the last 2 weeks, have you felt bothered by any of these things?	Not at all	Several Days	More than half the days	Nearly Every day
1. Feeling nervous, anxious, or on edge?	0	1	2	3
2. Not being able to stop or control worrying?	0	1	2	3
3. Worrying too much about different things?	0	1	2	3
4. Trouble relaxing?	0	1	2	3
5. Being so restless that it is hard to sit still?	0	1	2	3
6. Becoming easily annoyed or irritable?	0	1	2	3
7. Feeling afraid as if something awful might happen?	0	1	2	3

If you checked off any problems, how difficult have these problems made it for you to do your work, take care of the things at home, or get along with other people?

Not difficult at all	Somewhat difficult	Very difficult	Extremely difficult
☐	☐	☐	☐

Over the last 2 weeks, how often have you been bothered by any of the following problems?	Not at all	Several Days	More than half the days	Nearly Every day
1. Little interest or pleasure in doing things	0	1	2	3
2. Feeling down, depressed, or hopeless	0	1	2	3
3. Trouble falling asleep or sleeping too much	0	1	2	3
4. Feeling tired or having little energy	0	1	2	3
5. Poor appetite or overeating	0	1	2	3
6. Feeling bad about yourself- or that you are a failure or have let yourself or family down	0	1	2	3
7. Trouble concentrating on things, such as reading the newspaper or watching television	0	1	2	3
8. Moving or speaking so slowly that other people could have noticed. Or the opposite-being so fidgety or restless that you have been moving around a lot more than usual	0	1	2	3
9. Thoughts that you would be better off dead, or of hurting yourself in some way	0	1	2	3

If you checked off any problems, how difficult have these problems made it for you to do your work, take care of the things at home, or get along with other people?

Not difficult at all	Somewhat difficult	Very difficult	Extremely difficult
☐	☐	☐	☐

If you are experiencing any distress because of these questions, or have any concerns about the study, please contact the principle investigator, Dr. Jeffrey A. Ciesla at (330) 672-1192, or SAMCstudy@gmail.com

APPENDIX C: ANXIETY SENSITIVITY INDEX—3

Enter the number from the scale below that best describes how typical or characteristic each of the 16 items is of ***you***, putting the number next to the item. You should make your ratings in terms of how much you agree or disagree with the statement as a ***general*** description of yourself.

0 1 2 3 4

very little a little some much very much

1. It is important for me not to appear nervous.
2. When I cannot keep my mind on a task, I worry that I might be going crazy.
3. It scares me when my heart beats rapidly.
4. When my stomach is upset, I worry that I might be seriously ill.
5. It scares me when I am unable to keep my mind on a task.
6. When I tremble in the presence of others, I fear what people might think of me.
7. When my chest feels tight, I get scared that I won't be able to breathe properly.
8. When I feel pain in my chest, I worry that I'm going to have a heart attack.
9. I worry that other people will notice my anxiety.
10. When I feel "spacey" or spaced out I worry that I may be mentally ill.
11. It scares me when I blush in front of people.
12. When I notice my heart skipping a beat, I worry that there is something seriously wrong with me.

13. When I begin to sweat in a social situation, I fear people will think negatively of me.
14. When my thoughts seem to speed up, I worry that I might be going crazy.
15. When my throat feels tight, I worry that I could choke to death.
16. When I have trouble thinking clearly, I worry that there is something wrong with me.
17. I think it would be horrible for me to faint in public.
18. When my mind goes blank, I worry there is something terribly wrong with me.

APPENDIX D: RUMINATIVE RESPONSE SCALE

People think and do many different things when they feel depressed. Please read each of the items below and indicate whether you almost never, sometimes, often, or almost always think or do each one when you feel down, sad, or depressed. Please indicate what you *generally* do, not what you think you should do.

0	1	2	3	4
almost never		sometimes	often	almost always

1. ____ Think about how alone you feel
2. ____ Think "I won't be able to do my job if I don't snap out of this"
3. ____ Think about your feelings of fatigue and achiness
4. ____ Think about how hard it is to concentrate
5. ____ Think "What am I doing to deserve this?"
6. ____ Think about how passive and unmotivated you feel.
7. ____ Analyze recent events to try to understand why you are depressed
8. ____ Think about how you don't seem to feel anything anymore
9. ____ Think "Why can't I get going?"
10. ____ Think "Why do I always react this way?"
11. ____ Go away by yourself and think about why you feel this way
12. ____ Write down what you are thinking about and analyze it
13. ____ Think about a recent situation, wishing it had gone better
14. ____ Think "I won't be able to concentrate if I keep feeling this way."
15. ____ Think "Why do I have problems other people don't have?"
16. ____ Think "Why can't I handle things better?"
17. ____ Think about how sad you feel.
18. ____ Think about all your shortcomings, failings, faults, mistakes
19. ____ Think about how you don't feel up to doing anything
20. ____ Analyze your personality to try to understand why you are depressed
21. ____ Go someplace alone to think about your feelings
22. ____ Think about how angry you are with yourself

APPENDIX E: PREMENSTRUAL SYMPTOMS SCREENING TOOL

(please mark an "X" in the appropriate box)

Do you experience some or any of the following premenstrual symptoms which *start before* your period and *stop* within a few days of bleeding?

Symptom	Not at all	Mild	Moderate	Severe
1. Anger/irritability				
2. Anxiety/tension				
3. Tearful/Increased sensitivity to rejection				
4. Depressed mood/hopelessness				
5. Decreased interest in work activities				
6. Decreased interest in home activities				
7. Decreased interest in social activities				
8. Difficulty concentrating				
9. Fatigue/lack of energy				
10. Overeating/food cravings				
11. Insomnia				
12. Hypersomnia (needing more sleep)				
13. Feeling overwhelmed or out of control				
14. Physical symptoms: breast tenderness, headaches, joint/muscle pain, bloating, weight gain				

Have your symptoms, as listed above, interfered with:

	Not at all	Mild	Moderate	Severe
A. Your work efficiency or productivity				
B. Your relationships with coworkers				
C. Your relationships with your family				
D. Your social life activities				
E. Your home responsibilities				

APPENDIX F: DAILY QUESTIONNAIRE

Below is a list of feelings, sensations, problems, and experiences that people sometimes have. Read each item and then mark the appropriate choice in the space next to that item. Use the choice that best describes how much you have felt or experienced things this way today. Use this scale when answering:

1	2	3	4	5
not at all	a little bit	moderately	quite a bit	extremely

_____1. Felt sad
_____2. Startled easily
_____3. Felt cheerful
_____4. Felt afraid
_____5. Felt discouraged
_____6. Hands were shaky
_____7. Felt optimistic
_____8. Felt worthless
_____9. Felt really happy
_____10. Felt nervous
_____11. Felt depressed
_____12. Was short of breath
_____13. Felt uneasy
_____14. Was proud of myself
_____15. Felt faint
_____16. Felt unattractive
_____17. Had hot or cold spells
_____18. Felt like a failure
_____ 19. Felt like I was having a lot of fun
_____ 20. Blamed myself for a lot of things
_____ 21. Felt withdrawn from other people
_____ 22. Felt keyed up, “on edge”
_____ 23. Felt like I had a lot of energy
_____ 24. Was trembling or shaking
_____ 25. Felt inferior to others
_____ 26. Felt like crying
_____ 27. Was unable to relax
_____ 28. Felt really slowed down
_____ 29. Was disappointed in myself
_____ 30. Felt nauseous
_____ 31. Felt hopeless

_____ 32. Felt dizzy or lightheaded
_____ 33. Felt sluggish or tired
_____ 34. Felt really “up” or lively
_____ 35. Looked forward to things with enjoyment
_____ 36. Felt pessimistic about the future
_____ 37. Had a very dry mouth
_____ 38. Felt like I had a lot of interesting things to do
_____ 39. Felt like I had accomplished a lot
_____ 40. Felt like it took extra effort to get started
_____ 41. Felt like nothing was very enjoyable
_____ 42. Heart was racing or pounding
_____ 43. Felt like I had a lot to look forward to
_____ 44. Felt numbness or tingling in my body
_____ 45. Felt tense or “high-strung”
_____ 46. Felt hopeful about the future
_____ 47. Felt like there wasn’t anything interesting or fun to do
_____ 48. Seemed to move quickly and easily
_____ 49. Muscles were tense or sore
_____ 50. Felt really good about myself

Are you experiencing any stress related to school today? For example: failing an important exam

Are you experiencing any stress related to work today? For example: losing your job

Are you experiencing any stress related to significant others today? For example: breaking up with a romantic partner

Are you experiencing any other stress today? If yes, please explain below:

Are you experiencing any physical illnesses, injuries, or discomforts? For example: headache, cold, cramps, sore throat, etc.

Please indicate how often you have felt or behaved in the following ways today:

1	2	3	4	5	6
Never	Rarely	Sometimes	Often	Very Often	Always

_____ 1. I have been grumpy

_____ 2. I have been feeling like I might snap

_____ 3. Other people have been getting on my nerves

_____ 4. Things have been bothering me more than they normally do

_____ 5. I have been feeling irritable

The following questions ask about sexual behaviors in which you may and or may not have been a part of today

have you engaged in passionate kissing (i.e., using tongue) today?

have you cuddled with a romantic partner today?

have you engaged in vaginal intercourse today?

have you engaged in anal intercourse today?

have you engaged in self-stimulation (masturbation) today?

have you <u>performed</u> oral sex on another person today?

did you <u>receive</u> oral sex from another person today?

did you watch/view/read erotic material (e.g., videos, pictures, stories, etc.) today?

did you perform hand sex on a partner today?

did a partner perform hand sex on you today?

If you are experiencing any distress because of these questions, or have any concerns about the study, please contact the principle investigator, Dr. Jeffrey A. Ciesla at (330) 672-1192, or SAMCstudy@gmail.com

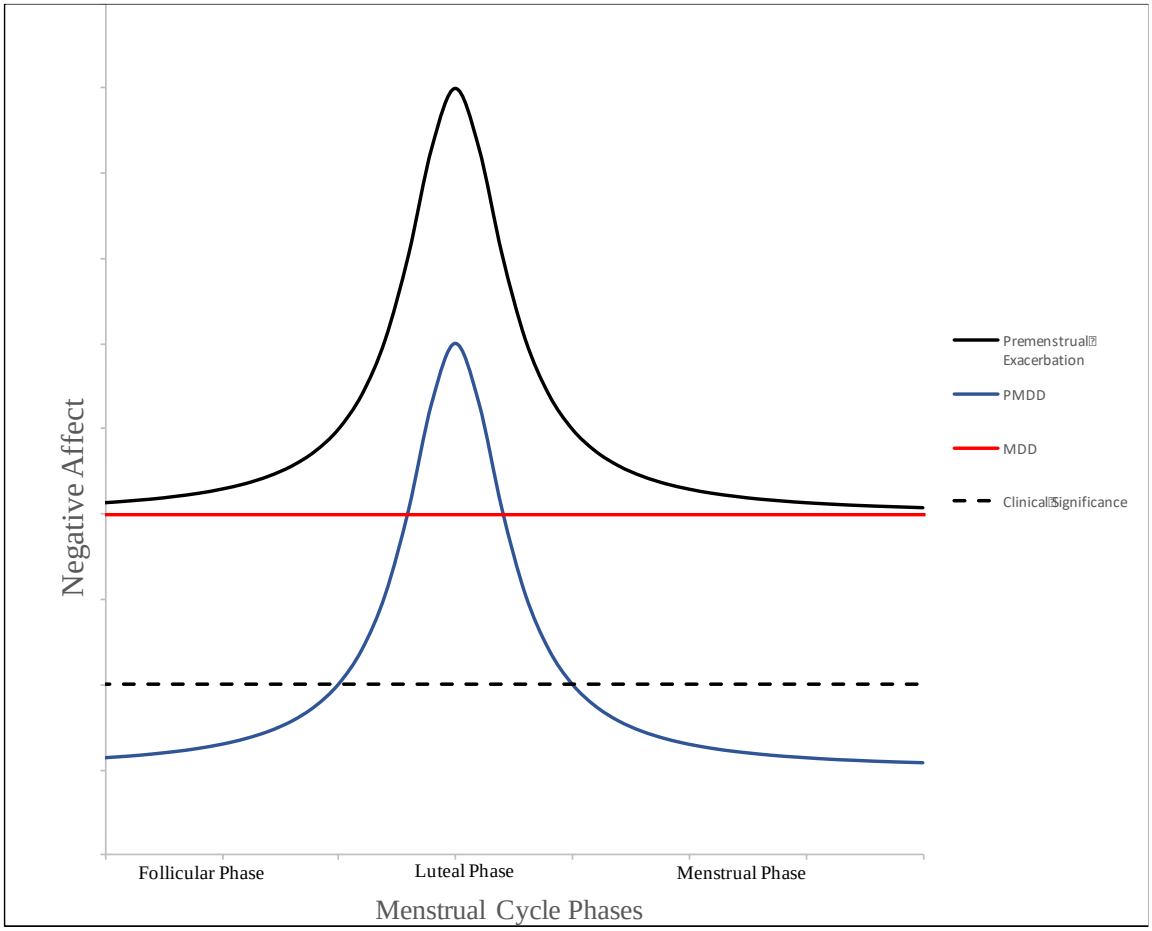

Figure 1. Theoretical patterns of negative affect across the menstrual cycle.