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Menstrual Cycle Changes in Dysregulated Eating Symptoms

Kimberly S. Stevens

MENSTRUAL CYCLE CHANGES IN DYSREGULATED EATING SYMPTOMS

By

Kimberly Stevens

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Kimberly Stevens

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Kristen Culbert, Ph.D.
Examination Committee Chair

Kathryn Hausbeck Korgan, Ph.D.
Graduate College Dean

Andrew Freeman, Ph.D.
Examination Committee Member

Murray Millar, Ph.D.
Examination Committee Member

Peter Gray, Ph.D.
Graduate College Faculty Representative

ABSTRACT

Objective: Animal and human data indicate that fluctuations in ovarian hormones (e.g., estrogen and progesterone) across the estrus/menstrual cycle drive changes in eating behavior. Post-ovulatory phases (i.e., mid-luteal and pre-menstrual in women) correspond to distinct ovarian hormone profiles (i.e., low estradiol or high estradiol coupled with high progesterone) that are known to have stimulatory effects on eating behavior and have been associated with increased risk for emotional eating and binge eating. Nonetheless, other specific components of dysregulated eating (e.g., preoccupation with food, loss of control over eating) that are related to and/or prospectively predict the development of clinical binge eating episodes have yet to be examined. Determining which specific features of dysregulated eating are impacted by hormonal shifts across the menstrual cycle could aid in elucidating the etiologic effects of ovarian hormones on eating disorders. This study aimed to address this gap by examining the fluctuation of several forms of dysregulated eating in a new sample of women. **Method:** Participants were 20 normal-weight women from the community (age 18-45) who completed multiple daily measures over one menstrual cycle (~30 days). Menstrual cycle phases were determined from a range of indicators: self-report onset of menses, basal body temperature, and urine hormone test strips. Several types of dysregulated eating symptoms (e.g., loss of control over eating, external eating, emotional eating, preoccupation with food, and intense desire/cravings to eat) were assessed each evening using well-validated questionnaires that were adapted for daily reports. Daily levels of negative affect were assessed using the Positive and Negative Affect Schedule (PANAS). Height/weight measurements were taken at the baseline and final appointments and used to calculate average Body Mass Index (BMI) across the cycle. **Statistical Analyses:** Consistent with prior studies, five-day rolling averages were computed for all dysregulated

eating scores and this adjusted daily score was converted to within-person z-scores. Repeated-measures Analysis of Variance (ANOVA) and Covariance (ANCOVA) were then used to examine mean differences in levels of dysregulated eating symptoms across the four menstrual cycle phases (i.e., follicular *vs.* ovulatory *vs.* mid-luteal *vs.* pre-menstrual) and using pre-ovulatory (follicular/ovulatory) versus post-ovulatory (mid-luteal/pre-menstrual) comparisons. All cycle phase analyses adjusted for average BMI and phase levels of negative mood to ensure that changes in dysregulated eating occur above and beyond the effects of BMI and negative affect. **Results:** There was minimal evidence of cycle phase differences on most dysregulated eating symptoms. When cycle phase effects were detected, the dysregulated symptoms (e.g., external eating, intense desire to eat**,** emotional eating) appeared to be most elevated in the pre-ovulatory period (e.g., follicular phase) relative to the post-ovulatory period (i.e., midluteal or premenstrual phases) – a pattern that is opposite of prior research. Further, these phase-differences in levels of dysregulated eating were no longer significant once analyses adjusted for BMI and negative affect. **Discussion:** Findings from this study contrast with prior research that has documented post-ovulatory (midluteal and/or premenstrual phases), rather than pre-ovulatory, increases in eating behavior. While the observed pre-ovulatory increases in dysregulated eating symptoms seem to be largely accounted for by negative affect and BMI, the factors contributing to between-study replication of phase differences remain unclear. It is suspected, however, that methodological issues (e.g., lack of randomization of the start phase of the study) may have played a role. These findings highlight the importance of carefully considering several methodological issues when conducting menstrual cycle research.

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CHAPTER 1

Introduction

Eating disorders are serious mental illnesses (Klump, Bulik, Kaye, Treasure & Tyson, 2009) that are broadly characterized by disturbances in eating behavior (e.g., disruptions in appetite and food intake) and pathological cognitions regarding one's body shape and weight. Eating disorders substantially impact cognitive, emotional, and social functioning and can have dire physical health consequences, such as gastrointestinal complications, weight gain and risk for diabetes, and possible cardiac effects (Arcelus, Mitchell, Wales & Nielsen, 2011; Kimmel, Ferguson, Zerwas, Bulik & Meltzer-Brody, 2016; Mitchell 2016; Mitchell & Crow, 2010; Mitchell, Zunker, Yager & Soloman, 2016; O'Brien, Whelan, Sandler, Hall, & Weinberg, 2017). Furthermore, eating disorders have the highest mortality rates of all psychiatric disorders, elevated relapse rates, and high treatment costs (Arcelus et al., 2011; Simon, Schmidt & Pilling, 2005; Steinhausen, 2011). These serious consequences of eating disorders and the clear treatment-recovery challenges underscores their public health relevance and the need to better understand their etiology.

Notably, although the DSM-5 formally recognizes multiple types of eating disorders (e.g., anorexia nervosa, bulimia nervosa, binge eating disorder) and diagnostic distinctions may have clinical importance, several core eating disorder symptoms (e.g., binge eating, dietary restriction, body weight/shape concerns) are shared across diagnoses. This high degree of symptom overlap results in high rates of diagnostic cross-over between eating disorders (e.g., Eddy et al., 2008b; Tozzi et al., 2005). For example, 73% of patients with anorexia nervosa experience diagnostic cross-over (e.g., transition to BN diagnosis via development of binge-purge behaviors) within seven years of their initial diagnosis (Eddy et al., 2008b). The high

diagnostic cross-over, coupled with the high proportion of subthreshold or "other" cases (e.g., OSFED), have led investigators to explore whether eating pathology is best conceptualized within a categorical, dimensional, or hybrid classification system (Luo, Donnellan, Burt, & Klump, 2016). Accumulating data largely support a dimensional framework, such that symptoms occur across a spectrum of severity in the population (Luo et al., 2016). Elevated endorsement of disordered eating symptoms also prospectively predicts the development of a full-threshold eating disorder. Thus, focusing on core disordered eating symptoms (e.g., binge eating, body dissatisfaction) and assessing these symptoms on a continuum of severity will be important for elucidating the etiology of eating disorders (Culbert, Racine & Klump, 2015).

It is also notable that eating disorders and their core symptoms occur at significantly higher rates in females, as compared to males, and this female preponderance begins to emerge at puberty (Culbert, Sisk & Klump, 2018; Klump, Culbert & Sisk, 2017). Although initial research largely focused on exploring psychosocial factors (e.g., pressures for thinness) that might increase risk in females (Lilenfeld, Wonderlich, Riso, Crosby & Mitchell, 2006; Stice, Gau, Rohde & Shaw, 2017), particularly during adolescence/puberty, there has been increasing attention on the exploration of biological factors (Culbert et al., 2015). Ovarian hormones are one set of biological factors that may be particularly important (Culbert et al., 2018; Klump, et al., 2017).

Overview of Ovarian Hormones and the Menstrual Cycle

Ovarian hormones dramatically rise in females during puberty, and following the onset of menses, begin to fluctuate cyclically across the menstrual cycle. The menstrual cycle is typically 28 days long, although there are between-person differences; women who have a period (i.e., menstruation) every 25-32 days are considered to have "regular" cycles (Allen et al., 2016). The

menstrual cycle plays an integral role in female reproduction, in that the body prepares itself for possible pregnancy. Although the exact days may vary, the menstrual cycle patterns of hormone changes are generally consistent across all regularly cycling women (Allen et al., 2016).

The cycle is driven by the rise and fall of key ovarian hormones: progesterone, estrogen, follicle stimulating hormone (FSH) and luteinizing hormone (LH) (Allen et al., 2016). In a typical 28-day cycle, day 1 corresponds to the first day of bleeding and is considered the start of the follicular phase. At the beginning of the follicular phase, estrogen and progesterone both remain low (Hawkins & Matzuk, 2008) (see Figure 1). The pituitary gland releases FSH which causes follicles (each containing an egg) to rise on the ovary. In the later part of the follicular phase, one follicle will become dominant and its egg will mature; other follicles are absorbed back into the ovary. The dominant follicle produces estrogen, which results in a rise of estrogen at the end of the follicular phase (e.g., peak of estrogen occurs 1-2 days before ovulation) (see Figure 1). Higher estrogen stimulates the lining of the uterus (endometrium) to become thickened and enriched with blood (in preparation for possible pregnancy). Elevated estrogen also results in the production of gonadotropin-releasing hormone (GnRH) which triggers the pituitary gland to secrete LH. The end of the follicular phase occurs around day 12 and is marked by a sharp rise in both LH and FSH (see Figure 1). The rise in LH levels promotes ovulation, which is the release of the egg from the follicle (Hawkins & Matzuk, 2008). Ovulation typically marks the mid-point of the cycle and often occurs around day 14 (see Figure 1). The released egg enters the fallopian tube and awaits fertilization.

The second half of the cycle encompasses the luteal and premenstrual phases. The follicle closes, forming a corpus luteum, which produces progesterone and helps the uterine lining thicken even more. Estrogen also exerts a secondary post-ovulatory increase. The mid-luteal

phase is marked by the lowest estrogen to progesterone ratio during the cycle (Hawkins & Matzuk, 2008) (see Figure 1). If fertilization occurs, the corpus luteum continues to produce progesterone to prevent the uterine lining from being shed and to allow pregnancy to ensue. At the end of the luteal phase, and in the absence of fertilization, the corpus luteum degenerates, which decreases both estrogen and progesterone levels (see Figure 1). The premenstrual phase occurs on just the last days before the next cycle begins and is marked by low progesterone and estrogen levels (Allen et al., 2016). When estrogen and progesterone drop, it triggers the uterine lining to begin to shed, resulting in menstruation and the next cycle begins (see Figure 1).

Importance of Ovarian Hormones for Women's Health

Although ovarian hormones (e.g., progesterone, estrogen) undoubtedly play a critical role in women's health, there is a paucity of research evaluating the impact of the menstrual cycle or ovarian hormones on psychological and health outcomes. Moreover, there are clear health disparities between men and women; women report worse health than men despite the fact that they live longer (Gorman & Read, 2006; Verbrugge, 1985) and there are various medical conditions that show sex-differentiated prevalence rates. For example, autoimmune diseases, kidney diseases, osteoporosis, fibromyalgia, Alzheimer disease, and eating disorders all demonstrate higher prevalence rates in females than males (American Psychological Association, 2013; Regitz-Zagrosek, 2012). While various sociocultural and psychological factors (e.g., SES status, stress, engaging in health-damaging behavior; Gorman & Read, 2006) can contribute to these health disparities, it is also imperative to consider sex-specific biological factors (e.g., ovarian hormones) that could differentially impact risk. Understanding how ovarian hormones may uniquely contribute to psychological and health-related conditions that afflict women at higher rates than men is a necessary next step.

The importance of investigating sex-specific factors has been emphasized within the public health domain. Indeed, one of the two main goals of Healthy People 2010 was "to eliminate health disparities among different segments of the population," including differences that occur between genders (U.S. Department of Health and Human Services). In addition, the National Institute of Health (NIH) recently implemented a policy that requires all NIH-funded researchers to account for sex as a crucial biological variable when designing, collecting, analyzing and disseminating data and findings (Clayton & Collins, 2014). Furthermore, researchers are required to justify single-sex studies, as sex-specific studies are considered appropriate only when evaluating sex-specific phenomenon such as the menstrual cycle or prostate cancer, or in the face of acutely scare resources. This policy change reflects the recognition that a general lack of consideration of sex differences or specific processes has hindered scientific understanding and advancements. The majority of research has traditionally been conducted on males or has ignored the potential role of fluctuating ovarian hormones in women (McMurray et al., 1991). Failing to consider ovarian hormones or other sex specific processes in data collection and analyses, could result in the oversight of critical biological components that may impact health outcomes and treatment response.

Ovarian hormones are directly related to a range of health and behavioral outcomes that disproportionally affect women (e.g., eating, mood, substance use patterns, response to stress; Albert, Pruessner & Newhouse, 2015; Gorczyca et al., 2016; Joyce et al., 2018; Moos et al., 1969; Wetherill, Franklin & Allen, 2016), and estradiol and progesterone have been identified as the primary ovarian hormones at play. That is, while FSH and LH exert important physiological effects that drive reproductive function, estradiol and progesterone are the key hormonal factors relevant to behavioral/psychological outcomes. For example, low levels of estradiol and/or high

levels of progesterone have been associated with increased depression, anxiety, panic, eating disorder symptoms and reduced reaction to psychomotor stimulant drugs (Edler et al., 2007; Klump et al., 2008; Klump et al., 2013a ; Nillni, Toufexis, & Rohan, 2011; Noble, 2005; Racine et al., 2012; Terner & De Wit, 2006), whereas LH and FSH are not directly tied to behavioral outcomes – these hormones are either not associated with behavior or associations have been shown to occur through their impact on estrogen and progesterone (Gibbs, Mallinson, & De Souza, 2016; Terner & De Wit, 2006). Therefore, continued investigation of ovarian hormones, specifically estradiol and progesterone, is important given their unique relation to behavioral, psychological and health outcomes.

The proposed work will focus on understanding the role of ovarian hormones in dysregulated eating behaviors. Dysregulated eating symptoms are of clinical relevance, as they prospectively predict the development of clinical binge eating (Jacobi et al., 2004). Binge eating is a core symptom of most eating disorders (e.g., bulimia nervosa, binge eating disorder, anorexia nervosa-binge purging subtype), but can also be present in other disorders (e.g., borderline personality disorder) (American Psychiatric Association, 2013). Significant changes in appetite and food cravings, which are prominent features in women suffering from binge eating, also occur in those suffering from premenstrual dysphoric disorder (American Psychiatric Association, 2013; Epperson et al., 2012). Thus, understanding ovarian hormone influences and cycle-related changes in dysregulated eating could have relevance for a range of psychological difficulties that affect women.

Ovarian Hormones and Dysregulated Eating in Animals

The role of ovarian hormones on food intake and dysregulated eating is supported by several lines of research in animals and humans. Animal studies, conducted on a number of

species (e.g., mice, rats, monkeys), have consistently demonstrated that estrogen and progesterone impact food intake and binge eating propensity. Estrogen reduces feeding behavior, whereas progesterone exerts stimulatory effects. For example, intact rats, mice and monkeys show increased food intake or binge eating behavior during hormonal cycles (i.e., estrus cycles in rodents; menstrual cycles in primates) marked by low estradiol and/or high progesterone (e.g., Asarian & Geary, 2013; Drewett, 1974; Miocioni Di Bonaventura et al., 2017).

In addition to these cycle phase effects, immediate and sustained alterations in feeding behaviors occur following experimental manipulation of ovarian hormone exposure. Specifically, increases in the consumption of chow (Czaja & Goy, 1975) and palatable food (e.g., high in fat and sugar content; Yu, Geary & Corwin, 2008) have been found in both rats and monkeys after bilateral ovariectomy (i.e., the removal of the ovaries). Exogenous administration of estradiol reverses these effects by causing decreases in food intake (Cao et al., 2014; Czaja & Goy, 1975; Fungfuang, Terada, Komatsu, Moon & Saito, 2013; Miocioni Di Bonaventura et al., 2017; Yu, et al., 2008). Interestingly, progesterone's stimulatory effects on food intake seem to occur largely via its antagonizing effects on estrogen. Indeed, progesterone administered alone does not impact food intake; progesterone administered concurrently with exogenous estradiol has been found to increase feeding (Russ & Zucker, 1974; Wade, 1972). That is, estradiol's anorexigenic effects on feeding become attenuated when progesterone is also present (Asarian & Geary, 2006; Kemnitz, Gibber, Lindsay & Eisele, 1989; Wade, 1972).

Ovarian Hormones and Dysregulated Eating in Humans

The impact of ovarian hormones on eating behaviors in animals seem to translate to humans. Indeed, cyclical fluctuations in food intake and food cravings have been observed across the menstrual cycle in normal-cycling women. Elevated food intake occurs during the

mid-luteal phase (e.g., marked by elevated progesterone and secondary peak in estradiol) and lower levels of consumption occur before ovulation, when estrogen levels peak (Buffenstein, Poppit, McDevitt & Prentice, 1995). Similar patterns have been observed with food cravings. Increased food cravings (i.e., strong desire for high sugar and high fat foods) have been reported during both the mid-luteal phase and pre-menstrual phase (e.g., when levels of progesterone and estradiol are both low); (Bancroft, Cook & Williamson, 1988; Dye, Warner, & Bancroft, 1995; Hormes & Timko, 2011; Krishnan, Tryon, Horn, Welch, & Keim, 2016). Thus, in normally cycling women, the midluteal and premenstrual phases of the menstrual cycle appear to be most "risky" for increased eating behaviors in humans.

These cyclical effects are not confined to general food intake, but also impact pathological eating behavior. Binge eating, which is defined as consuming a large amount of food in a short period of time (< 2 hours) *and* experiencing loss of control during the eating episode, is one key form of pathological eating that has been linked to the menstrual cycle. For example, in women with bulimia nervosa, discrete episodes of binge eating are exacerbated during menstrual cycle phases characterized by low estrogen and/or high progesterone (the midluteal and premenstrual phases) (Edler et al., 2007; Gladis and Walsh, 1987; Lester, Keel & Lipson, 2003; Price, Torem & DiMarzio, 1987) These phenotypic effects have been shown to extend to non-clinical community samples of women, as emotional eating scores (e.g., eating in response to negative emotions) also peak during the midluteal phase (Klump et al., 2008; Klump et al., 2013a). Recent data has also shown that genetic effects may underlie the phenotypic exacerbation of emotional eating during post-ovulatory phases. Specifically, latent estimates of genetic influences on emotional eating are approximately two times higher in post-ovulation (heritability ~ 12-20%) than pre-ovulation phases (heritability ~35-40%; Klump et al., 2015).

These data highlight post-ovulatory increases in pathological eating behavior and suggest that these phenotypic patterns may be driven by cycle-based changes in genetic effects.

In addition to the aforementioned cycle phase effects, studies have also found *direct relationships* between ovarian hormones and dysregulated eating symptoms (i.e., binge eating and emotional eating) across the menstrual cycle in women. For example, lower levels of estradiol and higher levels of progesterone predict increases in binge eating and emotional eating in clinical and non-clinical samples of women (Edler et al., 2007; Klump et al., 2008), independent of BMI and menstrual cycle changes in negative affect. Additionally, consistent with animal data indicating that progesterone antagonizes the anorexigenic effects of estrogen, a significant interaction between progesterone and estrogen has been found. Specifically, high levels of estrogen in the presence of high levels of progesterone (i.e., a profile consistent with the mid-luteal phase of the cycle) predicted higher levels of emotional eating above and beyond BMI and negative affect (Klump et al., 2013b). These findings have been critical for confirming that menstrual cycle fluctuations in dysregulated eating are due to changes in ovarian hormones.

Subsequent analyses have provided further support for the robust and unique effects of estradiol and progesterone on binge eating and emotional eating symptoms. First, although associations between dysregulated eating and ovarian hormones are evident in clinical and non-clinical samples of women, effects have been found to be stronger in women with clinical binge episodes than those without (Klump et al., 2014). Second, ovarian hormone effects on emotional eating were found to account for menstrual cycle changes in weight preoccupation (Hildebrandt et al., 2015), implicating a unique link between ovarian hormones and pathological eating symptoms as opposed to body weight/shape symptoms. Overall, data provide strong evidence that menstrual cycle fluctuations in dysregulated eating symptoms are driven by the natural

phase-based changes in ovarian hormone levels *and* these etiologic effects are present across a spectrum of severity.

While the aforementioned findings have provided critical insights into ovarian hormone effects on pathological eating behavior in women, significant research gaps remain. The *specific features* of dysregulated eating (e.g., loss of control during eating, preoccupation with food) impacted by menstrual cycle fluctuations in ovarian hormones remains relatively unclear. Prior studies have largely focused on either binge eating episodes (comprised of a constellation of key symptoms: overeating and loss of control over eating) or emotional eating symptoms. Indeed, Klump and colleagues (2008) was the only study to examine cycle phase differences on a range of eating constructs (i.e., emotional eating, external eating, eating concerns, bulimic symptoms, disinhibition, and hunger scales) in effort to determine which symptoms may be most strongly related to changes in ovarian hormones. Among the measured symptoms, emotional eating was the only construct that significantly differed across cycle phases; it was therefore this set of findings from a small sample of women (n = 10) that then led to the subsequent and exclusive focus on this construct. Nonetheless, this prior research was restricted to the use of validated self-report measures that were available at the time, and not all forms of dysregulated eating were examined. For example, other components of dysregulated eating (e.g., preoccupation with food, loss of control over eating, intense desire/cravings to eat) that are related to and/or prospectively predict the development of clinical binge eating episodes, were not assessed. Therefore, although changes in emotional eating and binge eating have been detected within the menstrual cycle, it is important to determine if other symptoms of dysregulated eating also fluctuate across the menstrual cycle phases.

To fully elucidate the etiologic effects of ovarian hormones, it will be important to determine which specific features of dysregulated eating are impacted by hormonal shifts across the menstrual cycle. Moreover, if ovarian hormones are found to be more closely tied to certain dysregulated eating symptoms, future studies can then begin to take a more fine-tuned approach in exploring exactly *how* ovarian hormones exert their effects (e.g., via modulation of the reward system, neuropathways underlying inhibitory control, and/or altered secretion or potency of appetite-hormones) on each of the specific symptoms. In addition, future research could identify interconnections between symptoms. For example, it may be that ovarian hormones increase risk for binge eating via intermediary effects (e.g., heightening food cravings and preferences for high fat/high sweet foods) in vulnerable women; however, the lack of concurrent assessment of multiple dysregulated eating features in prior research has prevented the investigation of such processes. Concurrently measuring several types of dysregulated eating symptoms and determining the extent to which levels of these symptoms change across the menstrual cycle could serve as an important first step.

Proposed Study

This study aimed to replicate and extend upon prior research by assessing a wide range of core dysregulated eating symptoms and determining which specific features show the largest menstrual cycle fluctuations. Specifically, this project examined cycle phase differences in specific dysregulated eating features: emotional eating, external eating, preoccupation with food, intense desire/cravings to eat, binge eating, and loss of control over eating. If ovarian hormones were to influence these eating symptoms, it was expected that mean levels would be highest in post-ovulatory phases (mid-luteal and premenstrual phases) as compared to pre-ovulatory phases (follicular and ovulatory). Consistent with prior studies (e.g., Klump et al., 2013a), this study

statistically controlled for potential confounds, namely BMI and levels of negative affect. Overall, findings from this research had the potential to contribute to a growing literature and further inform etiologic models of dysregulated eating in women.

CHAPTER 2

Methods

Participants

A total of 27 regular-cycling women were enrolled in the study. Women were recruited from the community via flyers, online advertisement (e.g., facebook), and a laboratory database of previous participants who agreed to be contacted about future research opportunities. Participants were required to meet a number of inclusion/exclusion criteria related to hormone function, including: 1) normal weight status ($\geq$18.5 kg/m^2 and < 30.0 kg/m^2); 2) no hormonal, psychotropic or steroid medications within the past eight weeks; 3) no pregnancy or lactation within the past year; 4) no history of genetic or medical conditions (e.g., diabetes) known to influence hormone functioning or appetite/weight; 5) no current alcohol/substance dependence or regular smoking or tobacco use; 6) no shift work or sleep related disorder. Although study criteria also included regular menstrual cycles (i.e., every 25-32 days) for the past six months, 50% of participants were mistakenly only screened for regular cycles over the past three months and four participants were included even though their typical cycle length was > 32 days (e.g., a 33 or 34 day cycle). Eligibility screening occurred via completion of an online eligibility survey, as well as a telephone screen, prior to enrolling into the study.

Of the 27 participants that were enrolled into the study, only 20 women (ages 18-33, M = 21.99, SD = 4.40) were included in analyses. The exclusion of seven participants occurred from a variety of issues. Three participants were excluded due to lack of confirmation that ovulation occurred since estradiol and progesterone levels only follow predictable fluctuation patterns for the second-half of the cycle if ovulation occurred (Lynch et al., 2014). One participant dropped out of the study due to health complications unrelated to the study (e.g., admitted to the hospital).

One participant failed to comply with the study protocol, and two participants were excluded due to questionable data (e.g., evidence of straight-line responding on daily questionnaires). In regards to sample demographics, a quarter of participants reported being of Hispanic/Latino origin (25%). Half of the sample identified as White (50%), a quarter identified as Asian (25%), and the remaining participants identified as multiracial (20%) or Black/African American (5%).

Procedures

This study was part of a larger data collection titled, "Stress, Mood, and Eating Behavior and Attitudes across the Menstrual Cycle" (UNLV IRB Protocol # 1257624-2). Participants completed at least two in-person appointments (i.e., baseline and final assessment) and daily surveys across one menstrual cycle. The baseline assessment was scheduled within +/- three days of the start of menstruation. During the baseline appointment, study eligibility was reconfirmed, and participants were trained by study staff on all daily procedures (e.g., saliva samples, basal body temperature, urine hormone test strips, and morning and evening surveys).

Participants began completing daily procedures on day 4 of the menstrual cycle (first day of bleeding was considered day 1; see Figure 2). Online surveys were completed every morning and evening. A brief (10 minute) morning survey assessed menstrual cycle status (e.g., bleeding or spotting, basal body temperature, and uploaded image of prior day's urine strip test), whereas daily eating and mood symptoms were assessed each evening. At the final appointment, participants returned study materials and completed remaining assessments (e.g., height and weight). Participants received up to $220 for full study completion. This reimbursement is on par with other studies that have required recurrent/daily questionnaire assessments for several weeks (Engel et al., 2013; Klump et al., 2013a; Smyth et al., 2007).

Measures

Dysregulated Eating Symptoms

Several well-validated self-report scales of dysregulated eating symptoms were used in effort to replicate prior menstrual cycle findings (e.g., fluctuations in emotional eating) and to explore additional symptoms/scales not previously evaluated in menstrual cycle research. Consistent with previous menstrual cycle studies (Klump et al., 2008; Klump et al., 2013a), all scales were modified to evaluate the participant's experience "today." Furthermore, all of the included dysregulated eating measures assessed eating symptoms across a continuum of severity (i.e., from low to high), since as noted previously, data suggest that dysregulated eating is best conceptualized as dimensional in nature (Luo et al, 2016). The use of a dimensional, as opposed to categorical, approach also provided greater statistical power (Kraemer, 2007).
More detailed information about each of the dysregulated eating scales is provided below.

Emotional Eating: Emotional eating was evaluated using the Dutch Eating Behavior Questionnaire (DEBQ) Emotional Eating subscale (van Strien, 1986). The emotional eating subscale is comprised of 13 items that evaluate eating in response to various negative emotions (e.g., boredom, discouragement or loneliness). Items were rated on a scale from Never (1) to Very Often (5).

External Eating: External eating was evaluated using the DEBQ External Eating Subscale (van Strien, 1986). The external eating subscale is comprised of 10 items and assesses desire to eat and actual intake when presented with situational and environmental opportunities to eat (e.g., eating when preparing a meal). Items were rated on a scale from Never (1) to Very Often (5).

Binge Eating Symptoms: Binge eating symptoms were assessed using the Eating Pathology Symptoms Inventory (EPSI) binge eating subscale (Forbush, et al., 2013; Forbush,

Wildes, & Hunt, 2014). The EPSI binge eating subscale (Forbush, et al., 2013) includes eight items that assess symptoms that are central to binge eating, such as eating in the absence of hunger, eating a very large amount of food, and mindless eating. Items were rated on a scale from Never (0) to Very Often (4).[1]

Preoccupation with Food: Preoccupation with food was assessed using the General Food Craving Questionnaire (GFCQ; Nijs et al., 2007). The preoccupation with food subscale is comprised of six items that evaluate the inability to stop thinking about food and being consumed by thoughts of eating when food cravings arise. Items were rated from Never (1) to Always (6).

Intense Desire to Eat: Intense desire to eat was evaluated using the GFCQ (Nijs et al., 2007). The intense desire to eat subscale is comprised of three items that assess an intense desire, urge, or craving for "tasty food." Items were rated from Never (1) to Always (6).

Behavioral Symptoms of Loss of Control: Behavioral loss-of-control eating symptoms were assessed with the Loss of Control Over Eating Scale (LOCES) behavioral symptoms subscale (Latner et al., 2014). This subscale includes seven items that tap into behavioral aspects (e.g., continuing to eat past the point one wanted to stop, eating until feeling uncomfortably full or despite negative consequences, eating in the absence of hunger) of loss of control over eating. Items were rated from Never (1) to Always (5).

Cognitive Dissociation in Loss of Control: Cognitive dissociation in loss-of-control eating were assessed with the Loss of Control Over Eating Scale (LOCES) cognitive dissociation subscale (Latner et al., 2014). This subscale includes four items that reflect cognitive/dissociative

[1] From "Development and validation of the Eating Pathology Symptoms Inventory," by Forbush, K.T, et al., 2013, Psychological Assessment, 25, 859-878. Copyright c 2011 by Kelsie T. Forbush. Reproduced with permission. No further reproduction, modification, or distribution of the Eating Pathology Symptoms Inventory, derivative versions, or translated versions is permitted with advance, written permission from the copyright holder (Dr. Kelsie Forbush).

aspects (e.g., inability to concentrate on anything else but eating, feeling disconnected or experiencing dissociation while eating) of loss of control over eating. Items were rated from Never (1) to Always (5).

Euphoria Associated with Loss of Control: Euphoria associated with loss of control were assessed with the Loss of Control Over Eating Scale (LOCES) euphoria subscale (Latner et al., 2014). This subscale includes two items that capture positive-euphoric aspects (e.g., experiencing a sense of relief/release, rush, or high while eating) of loss-of-control eating. Items were rated from Never (1) to Always (5).

Overall Loss of Control Over Eating: Overall levels of loss-of-control eating were assessed with the total score from the Loss of Control Over Eating Scale (LOCES; Latner et al., 2014).This score includes all of the above items (behavioral, cognitive and euphoric aspects of loss of control) and an additional eleven items that do not distinctly load onto the above facets. Notably, the facets and the total score were evaluated as outcome variables in this study since ovarian hormones could be differentially related to the facets and the exclusive use of only the total score might obscure more specific associations. Items were rated from Never (1) to Always (5).

Psychometrics of Dysregulated Eating Scales

Since each participant completed all questionnaires across multiple days, the average (across all study days) Cronbach's alpha was calculated for each scale across participants (see Table 1). In past research and in the current study, each scale demonstrated acceptable internal consistency (Forbush, et al., 2013; Forbush, Wildes, & Hunt, 2014; Klump et al., 2008; Klump et al., 2013a; Latner et al., 2014; Nijs et al., 2007; Shope, Prow, Racine, & Culbert, 2020).

Ovulation Evaluation

Each participant's urine hormone test strip and basal body temperature data were reviewed to evaluate the presence/absence and timing of ovulation.

Ovulation Prediction - LH Levels: Previous research has shown the menstrual cycle phase lengths vary even in normally-cycling women (Cole et al., 2009; Lenton, Landgren, Sexton & Harper, 1984). In order to determine the end of the follicular phase of study participants, it was necessary to determine the timing of the pre-ovulatory LH surge. The LH surge indicates that ovulation will occur in the next 24-48 hours and signals the end of the follicular phase (see Figure 1) (Kerin, 1982). Thus, beginning on day four of bleeding, participants were asked to complete LH urine testing until the LH surge was confirmed (see Figure 2). This means that women were expected to show a series of negative daily tests, followed by a positive daily test (i.e., indicative of the LH surge), and then the resumption of negative daily tests.

Extant data indicates that the ovulation LH surge varies in configuration, amplitude and duration. This means that some women have more than one positive LH test (e.g., rise in LH lasts for more than one day) or multiple LH surges, which can arise from multiple follicular stimulation (Baerwald, Adams & Pierson, 2003). Multiple follicular stimulation occurs when the first follicle is released but does not rupture, and thus the body releases a second follicle 3-5 days later. Extant data suggest that multiple follicular stimulation occurs in 6% to 44% of women in the population (Alliende, 2002; Park, Goldsmith, Skurnick, Wojtczuk, Weiss, 2007). Based on the LH test strip results, 20% of participants (n = 4) appeared to display a hormonal profile consistent with multiple follicular stimulation – a rate that is on par with the general population. It was presumed that estrogen and progesterone levels would mimic the typical cycle prior to the first LH surge and after the second LH surge. Thus, when more than one LH surge was detected, the first surge was used to anchor the follicular phase and the second surge was used to anchor

the ovulatory phase classification and subsequent post-ovulatory phases (e.g., midluteal and premenstrual).

Ovulation Confirmation – Pdg Levels and Body Temperature: A dramatic increase in morning basal body temperature and elevated progesterone (via positive results on pregnanediol (PDG) urine test strips – a metabolite of progesterone) were used to confirm that ovulation took place (Allen et al., 2016), as progesterone does not rise in the absence of ovulation (Ecochard et al., 2013). Initially participants were provided with six Pdg test strips and were instructed to begin testing seven days after a positive LH urine test; identification of at least one positive Pdg result was used to confirm ovulation had occurred. However, as the study progressed, the apparent individual variability of Pdg results became evident. Specifically, it was difficult to determine a clear positive result (i.e., above the threshold level) from an elevation in progesterone but still a negative result (i.e., below the threshold level) and there was concern that elevation might occur outside of the prescribed six test days. Procedures were then altered to have participants complete eleven Pdg test strips.

Menstrual Cycle Phase Classification

Previous studies investigating menstrual cycle phases (without the use of hormone assays) have used a counting method that classifies cycle phases anchored around how many days have passed since bleeding. Specifically, in previous research of the menstrual cycle, the first day of bleeding and the three days prior were classified as the premenstrual phase, 6 to 9 before bleeding were considered the midluteal phase, 12 to 15 days before bleeding were considered the ovulatory phase, and days 5 to 10 after bleeding were classified as the follicular phase (Elder, Lipson & Keel, 2007; Hormes & Timko, 2011; Klump et al., 2008; Klump et al., 2013; Krishnan, Tryon, Horn, Welch, & Keim, 2016). However, researchers have been advised

that reliance on a conventional counting methodology could lead to misclassification of menstrual cycle phase. This is due to the considerable variability in phase length even in "normal" menstrual cycles; the follicular phase can last between 10-20 days and the luteal phase can last between 9-17 days (Allen et al., 2016; Cole et al., 2009).

In this study, biological markers (e.g., temperature, LH and Pdg urine test strips) were used to estimate menstrual cycle phase, and selected days were anchored around these markers in effort to try to ensure the phase captured a similar underlying hormonal profile across participants. Thus, LH surge was used as the anchor for determine the end of the follicular phase and the six days prior to the LH surge were classified as the follicular phase. The day of the LH surge and two days after were classified as the ovulatory phase. Positive (Pdg) urine test strips were used to determine the 6 days of the midluteal phase, such that days were either centered around the strongest Pdg test results (e.g., indictive of a progesterone peak) or consecutive days showed positive Pdg test results with days prior and after indicative of negative Pdg tests. The premenstrual phase included the first day of bleeding of the next cycle and the two days prior to menses onset. Figure 1 displays the hormonal changes across the menstrual cycle and Figure 2 shows the general study procedures for a typical 28-day cycle.

Covariates

Negative affect and BMI were included as covariates since these variables have been associated with ovarian hormones and/or dysregulated eating symptoms (Cohen, Sherwin & Fleming, 1987; Jones, Bennett, Olmsted, Lawson, & Rodin, 2001; Romans, Clarkson, Einstein, Petrovic, & Stewart, 2012) and have been adjusted for in prior menstrual cycle studies (Edler et al., 2007; Klump et al., 2008; Klump et al., 2013a).

Negative Mood: The negative affect subscale from the Positive and Negative Affect Schedule (PANAS) was used to assess fluctuations in negative mood (Watson et al. 1988). This measure is comprised of 22 items specifically designed to assess mood states (e.g. distress, sadness, irritability) over different time periods, including a single day. Participants reported on the extent to which they felt each emotion "today," from "very slightly or not at all" (1) to "extremely" (5). The PANAS negative affect subscale has demonstrated good internal consistency in prior research ($\alpha = .87$) and the average Cronbach's alpha across study days was also acceptable in this study ($\alpha = .83$).

Body Mass Index (BMI): Participants' height and weight measurements were collected by trained research assistants using a height rod and medical scale. Height and weight data were used to calculate BMI (body weight (kg)/ height(m) squared). The average BMI of participants (e.g., the mean of the baseline BMI and final BMI) was used in analyses.

Statistical Analyses

Data Preparation

Scales containing 10 or more items (i.e., total score from the Loss of Control Over Eating Scale, external eating and emotional eating subscales from the Dutch Eating Behavior Questionnaire, and negative affect subscale from the Positive and Negative Affect Scale) were prorated for participants missing < 10% items on a daily report. Daily scores were coded as missing for participants missing data on scales that contain less than 10 items. For all dysregulated eating scales, about 7% of data was missing. However, due to the use of five-day rolling averages, missing data was only problematic when it occurred over subsequent days. None of the 20 participants had missing data that prevented an average phase score from being calculated or that caused them to be dropped from analyses.

Menstrual Cycle Phase Comparisons

Statistical analyses for menstrual cycle phase comparisons were modeled after those used in similar studies (Edler et al., 2007; Klump et al., 2008). Specifically, five-day rolling averages were computed for all dysregulated eating scores and this adjusted daily score was converted to within-person z-scores (i.e., using each individual's overall mean and standard deviation on the measure). A within-person mean dysregulated eating score was then calculated using only the days that corresponded to each menstrual cycle phase.

Repeated-measures Analysis of Variance (ANOVAs) were used to examine mean differences in levels of various dysregulated eating symptoms between cycle phases. Cycle phase comparisons were made using a four-phase approach (follicular vs ovulatory vs mid-luteal vs premenstrual) and two-phase approach (pre-ovulatory vs post-ovulatory) to gain a comprehensive understanding of cycle-based effects and to allow for more direct comparisons between results from this study and those of prior reports (Edler et al, 2007; Klump et al., 2008). After conducting ANOVAs, repeated-measures Analysis of Covariance (ANCOVAs) were conducted to evaluate whether BMI and/or negative affect might account for cycle phase differences in dysregulated eating. BMI was entered as a covariate. Because negative affect was a daily-level variable, it could not be entered as a covariate in the ANCOVA, so daily negative affect was regressed out of the daily dysregulated eating score and the standardized residual scores were used in analyses. The average levels of negative affect was then calculated within each of the four menstrual cycle phases.

The use of standardized scores provides an indication of effect sizes and eases the interpretation of mean differences in dysregulated eating across the different cycle phases. Partial eta squared were also computed to provide the overall magnitude of mean differences between

cycle phases ($\eta^2_{partial}$ = .01, small; $\eta^2_{partial}$ = .09, medium; $\eta^2_{partial}$ = .25, large effect sizes). It was expected that the dysregulated symptoms affected by ovarian hormones would be significantly elevated in the mid-luteal and pre-menstrual phases, as compared to the follicular and ovulatory phases, and these cycle-phase effects would occur above and beyond BMI and negative affect symptoms.

Power Analysis: G-Power (version 3.1; Faul, Erdfelder, Buchner, & Lang, 2009) was used to estimate statistical power prior to the start of this study. With four assessment phases (1 from each cycle phase), a sample size of 30 women, a *p*-value of < .05, and moderate correlations among repeated measures, models would have had > 80% power to detect effects that were medium in magnitude. In addition, with two assessment phases (pre-ovulation vs post-ovulation) and the same sample size and statistical parameters, models would have had >80% power to detect effects that are medium in magnitude. Given that only 20 women were included in analyses for this study, post-hoc power analyses were conducted. These analyses indicated that this study would have had >80% to detect significant effects ($p < .05$) that were medium in magnitude.

CHAPTER 3

Results

Descriptive Statistics

Descriptive information and raw correlations between dysregulated eating symptoms are presented in Table 1 and 2. In general, dysregulated eating symptoms spanned a spectrum of severity, and the mean scores were on par with other non-clinical samples that used identical measures (Klump et al., 2008; Shope, Prows, Racine & Culbert, 2020; Stefano et al., 2016). However, most scales had a restricted range of scores in that highest categories were rarely or not endorsed. Most scales had evidence of floor effects in that the most common response was Never/Rarely. As a result, the scale scores tended to have low variability. In particular, the EPSI binge eating and LOCES cognitive dissociation subscales were excluded from analyses because of extremely low variability in scale scores. Additionally, the LOCES total score was highly correlated (r = .92) with the LOCES behavioral symptoms subscale, and these two scales also showed nearly identical correlations with all the other symptoms (LOCES total score, r's = .32 — .64; LOCES behavioral symptoms subscale, r's = .34 — .69). These correlation patterns indicate that the two scales were likely capturing redundant information, so analyses focused on the more narrow/defined construct (i.e., behavioral symptoms) and the total score was excluded from the remaining analyses. All other dysregulated eating scales showed positive correlations with each other that ranged from small to medium in magnitude (see Table 2; r's = .15 — .64).

Menstrual Cycle Phase Comparisons

Results from repeated measures ANOVAs indicated significant cycle phase differences in external eating and intense desire to eat as well as a trend for cycle phase differences in emotional eating (see Table 3). However, in contrast to hypotheses, levels of these symptoms

were highest during the follicular phase relative to the other phases, and these effects were no longer significant once BMI and negative affect were accounted for (see Table 3). In summary, my hypotheses were not supported because several dysregulated eating symptoms did not significantly differ across menstrual cycle phases or levels were higher in the follicular phase as opposed to the midluteal and/or premenstrual phases.

Pre/Post Ovulation Comparisons

Repeated measures ANOVA results from the pre-ovulatory versus post-ovulatory comparisons on dysregulated eating symptoms were largely similar to the four phase comparisons. Effects identified were opposite to hypotheses. Specifically, significantly higher levels of emotional eating and a trend for higher levels of external eating and intense desire to eat were found in the pre-ovulatory period as opposed to the post-ovulatory period (Table 4), and these pre/post ovulation differences in dysregulated eating symptoms were no longer significant after controlling for BMI and negative affect (see Table 4).

Exploratory Analyses

Given that results from this study did not replicate past research, two types of exploratory analyses were conducted to determine whether methodological issues may have impacted the pattern of observed effects. First, I aimed to evaluate whether my unexpected pattern of findings arose from between-study differences in cycle phase classification. Previous research examining dysregulated eating across the menstrual cycle used a counting method to determine phase, whereas I used biological markers (e.g., LH and Pdg urine test strips) to guide the classification of menstrual cycle phases. Thus, I recoded cycle phases using the same counting method as prior studies and conducted an exploratory repeated measures ANOVA to evaluate whether these coding differences would change the results, particularly for the symptoms/scales that have been

used in prior research (e.g., emotional eating). The pattern of results was consistent with my original findings – dysregulated eating symptoms that showed significant difference across menstrual cycle phases (i.e., emotional eating, external eating, intense desire to eat and behavioral loss of control) continued to be elevated during pre-ovulatory phases rather than during post-ovulatory phases (see Table 5). These findings suggest that the pre-ovulatory elevation in dysregulated eating is not explained by cycle phase coding differences between this study and past studies.

Second, all participants started the study at the beginning of their cycle (i.e., the fourth day of bleeding). At least some of the prior menstrual cycle studies have used continuous enrollment which means participants varied in the cycle phase they were in at the start of the study. The lack of between-person variation in the start phase of the study may have resulted in some measurement reactivity (e.g., alterations in behavior as a result of measurement). Specifically, I was concerned that a reduction of symptoms and/or reduced effort in completing items over the course of the study might have occurred as a result of daily reporting/self-monitoring. Paired samples t-test and Cohen's *d* effect sizes were therefore used to compare mean levels differences in dysregulated eating symptoms on a subset (n = 12) of participants who completed daily assessments for at least two days of their next menstrual cycle, and thus, had available data from a second follicular phase. There was some evidence that dysregulated eating symptoms were higher in the cycle 1 follicular phase than the cycle 2 follicular phase (see Table 6). Although no significant differences emerged, presumably due to small sample size, Cohen's *d* effect sizes were medium-to-large in magnitude for emotional eating ($d = .64$), external eating ($d = .53$), intense desire to eat ($d = 1.07$) and behavioral loss of control ($d = .41$).

These findings suggest that a reduction of symptoms might have occurred over the course of the study.

CHAPTER 4

Discussion

This study investigated the effects of menstrual cycle phase on a comprehensive battery of dysregulated eating symptoms and evaluated whether phase differences in dysregulated eating occur independent of BMI and/or negative affect. Overall, there was minimal evidence of cycle phase differences on dysregulated eating symptoms. Women reported higher levels of some dysregulated eating symptoms (i.e., external eating and intense desire to eat) in pre-ovulatory phases (follicular and ovulatory phases), rather than post-ovulatory phases (midluteal and premenstrual phases), and these cycle-based effects appeared to be influenced by BMI and negative affect. Indeed, cycle phase differences in levels of external eating and intense desire to eat were attenuated once BMI and negative affect were adjusted for.

Notably, this pattern of results was opposite from my hypotheses and previous menstrual cycle studies that examined dysregulated eating. Specifically, previous studies in humans have reported higher levels of food cravings, emotional eating and binge eating in post-ovulatory phases rather than pre-ovulatory phases (Elder, Lipson & Keel, 2007; Gladis and Walsh, 1987; Klump et al., 2008; Klump et al., 2013a; Lester, Keel & Lipson, 2003; Price, Torem & DiMarzio, 1987), and the relative increase in symptoms during the post-ovulatory period have remained even after adjusting for negative affect and BMI (Klump et al., 2013a). Taken together, results from this study provide evidence that dysregulated eating symptoms may fluctuate across the menstrual cycle, but the factors contributing to the changes in eating behavior in this study appear to differ from those of past studies.

The fact that cycle phase differences in external eating and intense desire to eat were attenuated once BMI and negative affect were accounted for highlights the likely importance of these “third variables.” Negative affect has been consistently linked to dysregulated eating and

this relationship is more pronounced in those with high BMI (Haedt-Matt & Keel, 2011; Zeeck, Stelzer, Linster, Joos & Hartmann, 2011). In fact, severe forms of dysregulated eating (e.g., binge eating) are often conceptualized as a maladaptive form of coping with negative affect (Whiteside et al., 2007). Negative affect has strong predictive effects on dysregulated eating behaviors and is also associated with BMI. The heightened dysregulated eating during the pre-ovulatory period (e.g., follicular phase) in women in this study could be due to concurrent elevations in negative affect and/or differences in BMI.

The elevated levels of dysregulated eating symptoms during the pre-ovulatory period was surprising given the corresponding ovarian hormonal profile (i.e., low progesterone and rise in estradiol) and well-documented ovarian hormone influences on eating behaviors. For example, food intake tends to be inversely associated with estrogen and positively associated with progesterone (Asarian & Geary, 2006). Prior studies have also directly shown that detected menstrual cycle fluctuations in dysregulated eating outcomes are due to changes in levels of estrogen and progesterone (Klump et al., 2013a; Klump et al., 2014; Miocioni Di Bonaventura et al., 2017). It is therefore unlikely that ovarian hormones contributed to the higher rates of dysregulated eating in follicular and ovulatory phases in this study.

Notably, my cycle phase findings also contrast with evolutionary theory. From an evolutionary perspective, it has been suggested that women have evolved psychological mechanisms that limit eating and cravings during late follicular and ovulatory phases in order to favor mating-related activities during the fertile period (Saad & Stenstrom, 2012). Lowering satiation would decrease food seeking, and therefore increase the time and energy available for mate seeking and mating behaviors (Fessler, 2003). The typical propensity to increase food intake during post-ovulatory phases (mid-luteal and pre-menstrual; i.e., after egg fertilization

could occur) would also be adaptive since a natural post-ovulatory drive to eat may ensure an adequate amount of food is consumed. This could subsequently increase the likelihood of a successful pregnancy or prepare the body for pregnancy at the next cycle. My results, which show an increase in dysregulated eating behaviors during peak fertile periods, do not coincide with this evolutionary perspective.

The lack of replication of prior findings, especially with scales that have been tested in multiple studies (e.g., DEBQ emotional eating scale), and inconsistency with leading theories (e.g., evolutionary explanations) is somewhat concerning. Methodological issues might have contributed to these between-study discrepancies. Although differences in menstrual cycle phase classification (e.g., the shift from counting method to biological approximations) might have played a role, results from exploratory analyses did not support this possibility. Instead, results from exploratory analyses support the possibility that measurement reactivity might have contributed to the unexpected elevation of certain dysregulated eating symptoms during the first cycle phase assessed (follicular phase).

There are two types of measurement reactivity that might explain the decrease in endorsement of dysregulated eating over the course of the study: satisficing and self-monitoring effects. Satisficing refers to limits that a respondent imposes on the amount of effort she or he is willing to apply to answer a question or set of questions (Mehl & Tamlin, 2005). It is possible that participants paid less attention to items and became less thorough as the study progressed. If so, this could contribute to higher levels of dysregulated eating symptoms endorsed during the follicular phase of cycle 1 as compared to the follicular phase of cycle 2. Higher rates of dysregulated eating during the follicular phase could also arise from self-monitoring (i.e., increased awareness, observation, and evaluation of one's behavior) and its known effects on

behavioral change, such as reductions in dysregulated eating (Collins et al., 1998; Magnan, Köblitz, McCaul, & Dillard, 2013; Simpson et al., 2005). Indeed, self-monitoring of eating behavior is a key component of leading treatments for binge eating syndromes because it aids in the reduction of binge eating episodes over time (Latner & Wilson, 2002); in this study, the recurrent reporting on eating behavior (via daily completion of morning and evening surveys) could have served as a form of self-monitoring. Satisficing and self-monitoring effects may have uniquely impacted this study since all women started data collection during their follicular phase. In studies that have used continuous enrollment (e.g., Klump et al., 2008), these types of measurement issues would have had more minimal influences on the overall findings because they would have been dispersed across different cycle phases. Future menstrual cycle studies should consider using continuous study enrollment to minimize or rule-out the effects of measurement reactivity.

An additional limitation occurred during recruitment. Despite efforts to only enroll women with normative cycles (25-32 day cycle) in this study, women with cycles that lasted beyond 32 days were included. In addition, I did not directly assess a six-month menstrual cycle history for the first ten participants. These eligibility criteria changes might have increased the probability of including participants with non-normative cycles or hormonal profiles. For example, of the 27 women who were enrolled in the study, three participants had to be excluded due to lack of confirmation that ovulation occurred. Non-normative hormone patterns could also be present in women who were included in analyses (n = 20), as only 55% (n =11) showed normal, single ovulation, based on their LH urine test strip data. An additional subset of women included in analyses (n = 9; 45% of sample) showed unexpected patterns on urine hormone test strips, such as multiple follicular stimulation (i.e., evidence of biphasic LH surges) or fluctuating

Pdg readings (e.g., test results fluctuating between negative and positive across multiple consecutive days). These participants were retained in my final sample given sample size concerns. Directly testing the hormone levels of all participants throughout the menstrual cycle is critical to more clearly identify who had normative versus non-normative cycles.

The study was designed to identify menstrual cycle phase from indirect estimates of hormone changes from urine sample test strips. While this was better methodology than most prior studies of eating pathology across the menstrual cycle, the gold standard method for assigning menstrual cycle phase is to directly measure ovarian hormones via saliva. LH urine test strips are commonly used in menstrual cycle research; however, Pdg urine test strips are new to the market and have not been widely used in research. Pdg test strips may need further development before being a reliable marker of progesterone across cycle phases (Bouchard, 2018). For example, in my study a subset of women (n = 5; 25% of the sample) had Pdg results that showed questionable between-day fluctuations, from positive to negative, during the post-ovulatory period. These fluctuations between positive and negative test results is a concern because a steady rise in progesterone would be expected in a normative hormonal profile (see Figure 1). These inconsistencies could have been due to either test strip failures or user error. These inconsistencies made it difficult to concretely determine if a woman had ovulated, and reduced confidence in the coding of cycle phases.

An additional limitation is the general low endorsement of dysregulated eating within this study sample. The mean binge eating score (M = 3.13, SD = 4.90) was substantially lower than several other studies, including community samples (M = 7.80, SD = 6.60; Coniglio et al., 2018) and college student samples (e.g., M: 8.01-9.75, SD = 5.77-6.23; Shope, Prows, Racine, Culbert, 2020), which resulted in the elimination of this scale from analyses. Lack of variability was also

evident on the LOCES cognitive dissociation scale (see Table 1). Specifically, a majority of women endorsed minimal or no binge eating or cognitive dissociation symptoms, creating floor effects within this sample. The women included in this study could be uniquely different from the general population in regard to some forms of dysregulated eating or items on these scales (i.e., EPSI binge eating subscale & LOCES cognitive dissociation) are not appropriate for daily data collection because they do not appropriately inquire about high frequency changes. Future research should utilize a larger sample size in order to increase the probability of sampling a group of women with dysregulated eating that may be more representative of the general population or specifically recruit women with dysregulated eating. Taken together, the unique lack of dysregulated eating endorsement in this sample makes it somewhat difficult to generalize these findings to a general population.

Overall, the majority of dysregulated eating symptoms did not demonstrate cycle phase effects. Dysregulated eating symptoms (intense desire to eat, external eating, emotional eating) that did show some fluctuations across the cycle were elevated in pre-ovulatory (e.g., follicular phase), rather than post-ovulatory, phases, and the cycle phase differences were diminished once covarying for BMI and negative affect. These data highlight the potential contribution of negative affect and BMI to daily reports of dysregulated eating, but the overall pattern of findings contrasts with decades of hormonal research and leading theories. Future research should individual hormonal levels (i.e., LH, estradiol, progesterone) in order to confirm cycle/hormone normality in all women and to correctly assign menstrual cycle phases. In addition, future research should enroll participants at various points in the menstrual cycle to mitigate the possible effects of measurement reactivity. Taken together, the findings from this study highlight the complications and nuances associated with studying the menstrual cycle and

highlight the importance of dedicating more attention and resources to adequately investigate this essential building block of women's health.

Table 1

Descriptive Statistics for Dysregulated Eating Symptoms

Dysregulated Eating Symptoms	Scale Range	Sample Range	*M* (*SD*)	Average α
Included Scales				
Emotional Eating	1-5	1-3	1.27 (0.38)	.87
External Eating	1-5	1-4	1.76 (0.62)	.93
Preoccupation with Food	6-32	6-21	8.87 (3.57)	.90
Intense Desire to Eat	3-18	3-18	6.89 (3.49)	.91
Behavioral Loss of Control	1-5	1-4	1.31 (0.52)	.89
Euphoria in Loss of Control	1-5	1-5	1.41 (0.81)	.77
Excluded Scales				
Binge Eating Symptoms	0-32	0-26	2.93 (3.97)	.88
Cognitive Dissociation in Loss of Control	1-5	1-3	1.14 (0.32)	.75
Total Loss of Control Symptoms	1-5	1-3	1.26 (0.40)	.96

Note: Between-subjects (n = 20) Cronbach's alpha was calculated across all days of the menstrual cycle for all participants (e.g., days 1-34 of the cycle). Mean and standard deviations were calculated using the 18 days (6 days from follicular, 3 days from ovulatory, 6 days from midluteal and 3 days from premenstrual phase) that were included in final analyses

Table 2

Correlations between Dysregulated Eating Symptoms

Dysregulated Eating Symptoms	Emotional Eating	External Eating	Preoccupation with Food	Intense Desire to Eat	Behavioral Loss of Control	Euphoria in Loss of Control
Emotional Eating	1	.59**	.57**	.39**	.64**	.35**
External Eating		1	.56**	.64**	.48**	.25**
Preoccupation with Food			1	.57**	.58**	.39**
Intense Desire to Eat				1	.32**	.15**
Behavioral Loss of Control					1	.48**
Euphoria in Loss of Control						1

Note: Scores from all days of the menstrual cycle (Days 1-34 of the study) were used to calculate intercorrelations.

**$p<.01$.

Table 3

Mean Differences in Dysregulated Eating Symptoms by Menstrual Cycle Phase.

Dysregulated Eating Symptoms	Menstrual Cycle Phase						
	Follicular *M* (*SD*)	Ovulatory *M* (*SD*)	Mid-Luteal *M* (*SD*)	Premenstrual *M* (*SD*)	*F* (3,57)	*p*	$\eta^2_{partial}$
Repeated Measures ANOVA							
Emotional Eating	0.20 (0.73)	-0.03 (0.78)	-0.45 (0.42)	-0.14 (0.93)	2.62	.06	0.12
External Eating	0.16 (0.62)	-0.10 (0.81)	-0.23 (0.62)	-0.51 (0.65)	3.13	.03	0.14
Preoccupation with Food	-0.01 (0.80)	-0.28 (0.79)	-0.03 (0.63)	-0.32 (0.71)	0.87	.46	0.04
Intense Desire to Eat	0.19 (0.67)	-0.11 (0.73)	-0.13 (0.38)	-0.66 (0.92)	4.49	.01	0.19
Behavioral Loss of Control	0.14 (0.68)	-0.09 (0.78)	-0.16 (0.81)	-0.43 (0.84)	1.50	.22	0.07
Euphoria in Loss of Control	-0.05 (0.65)	-0.22 (0.68)	-0.08 (0.76)	-0.13 (1.15)	0.13	.94	0.01
Repeated Measures ANCOVA							
Emotional Eating	0.08 (0.90)	0.10 (0.89)	-0.13 (0.53)	0.12 (0.92)	0.07	.98	0.004
External Eating	-0.10 (0.77)	0.05 (0.92)	0.02 (0.66)	0.10 (0.88)	1.20	.32	0.06
Preoccupation with Food	-0.07 (0.78)	-0.04 (0.86)	-0.04 (0.72)	0.06 (1.05)	0.60	.62	0.03
Intense Desire to Eat	0.02 (0.75)	0.06 (0.98)	-0.10 (0.52)	0.04 (0.96)	0.29	.84	0.02
Behavioral Loss of Control	-0.14 (0.75)	-0.07 (0.93)	-0.02 (0.85)	0.04 (0.97)	0.73	.54	0.04
Euphoria in Loss of Control	0.06 (0.81)	-0.07 (0.90)	-0.07 (.087)	0.05 (1.05)	0.27	.85	0.02

Note: Dysregulated eating scores are calculated from 5 day rolling averages and were within-person standardized. Repeated measures

ANCOVA results controlled for average BMI (calculated from height/weight measurements collected during baseline and final assessments) and average levels of negative affect calculated within each of the four menstrual cycle phases. Partial eta squared effect size interpretation: $\eta^2_{partial}$ = .01, small; $\eta^2_{partial}$ = .09, medium; $\eta^2_{partial}$ = .25, large effect sizes.

Table 4

Mean Differences in Dysregulated Eating in Pre-Ovulation versus Post-Ovulation.

Dysregulated Eating Symptoms	**Cycle Classification**		F (1, 19)	p	$\eta^2_{partial}$
	Pre-Ovulatory *M* (*SD*)	Post Ovulatory *M* (*SD*)			
Repeated Measures ANOVA					
Emotional Eating	0.08 (0.52)	-0.36 (0.46)	7.23	.01	0.28
External Eating	0.05 (0.57)	-0.33 (0.50)	3.36	.08	0.15
Preoccupation with Food	-0.11 (0.75)	-0.13 (0.50)	0.01	.94	0.001
Intense Desire to Eat	0.06 (0.53)	-0.31 (0.44)	3.85	.07	0.17
Behavioral Loss of Control	0.07 (0.59)	-0.25 (0.57)	1.82	.19	0.09
Euphoria in Loss of Control	-0.14 (0.49)	-0.10 (0.59)	0.02	.89	0.001
Repeated Measures ANCOVA					
Emotional Eating	0.05 (0.63)	-0.09 (0.53)	0.05	.83	0.003
External Eating	-0.06 (0.70)	0.04 (0.59)	2.51	.13	0.12
Preoccupation with Food	-0.07 (0.76)	-0.02 (0.61)	0.03	.87	0.001
Intense Desire to Eat	-0.01 (0.62)	-0.06 (0.52)	0.27	.61	0.02
Behavioral Loss of Control	-0.10 (0.68)	-0.01 (0.62)	0.20	.66	0.01
Euphoria in Loss of Control	-0.02 (0.60)	-0.04 (0.62)	0.18	.68	0.01

Note: Pre-ovulatory combined across follicular and ovulatory phases. Post ovulatory combined across midluteal and premenstrual phases. Dysregulated eating scores are calculated from 5 day rolling averages and were within-person standardized. Repeated measures ANCOVA results controlled for average BMI (calculated from height/weight measurements collected during baseline and final assessments) and average levels of negative affect calculated within each of the four menstrual cycle phases. Partial eta squared effect size interpretation: $\eta^2_{partial}$ = .01, small; $\eta^2_{partial}$ = .09, medium; $\eta^2_{partial}$ = .25, large effect sizes.

Table 5

Exploratory Analysis: Mean Differences in Dysregulated Eating Symptoms by Menstrual Cycle Phase Using Counting Classification

	Menstrual Cycle Phase						
Dysregulated Eating Symptoms	Follicular *M* (*SD*)	Ovulatory *M* (*SD*)	Mid-Luteal *M* (*SD*)	Premenstrual *M* (*SD*)	*F* (3, 57)	*p*	$\eta^2_{partial}$
Emotional Eating	0.56 (0.95)	-0.24 (0.75)	-.33 (0.43)	-0.18 (0.86)	4.35	.01	0.09
External Eating	0.61 (0.93)	0.04 (0.79)	-.20 (0.62)	-0.45 (0.65)	5.51	.002	0.23
Preoccupation with Food	0.30 (0.95)	-0.02 (0.86)	-.09 (0.64)	-0.26 (0.65)	1.40	.25	0.07
Intense Desire to Eat	0.70 (0.74)	-0.11 (0.72)	-.12 (0.45)	-0.56 (0.83)	9.07	.000	0.32
Behavioral Loss of Control	0.44 (0.94)	0.21 (0.75)	-.21 (0.79)	-0.36 (0.78)	3.20	.03	0.14
Euphoria in Loss of Control	-0.08 (0.31)	-0.14 (0.27)	-.02 (0.47)	-0.001 (0.60)	0.40	.75	0.02

Note: Counting Classification refers to estimating menstrual phase by counting the days before and after bleeding. Specifically, days 5 to 10 after the first cycle first day of bleeding (e.g., the start of the study) were classified as the follicular phase. The rest of the phases were anchored from the second cycle first day of bleeding. 12 to 15 days before second cycle bleeding were considered the ovulatory phase, 6 to 9 days before second cycle bleeding were considered the midluteal phase and the three days prior to the second cycle bleeding were classified as the premenstrual phase. Partial eta squared effect size interpretation: $\eta^2_{partial}$ = .01, small; $\eta^2_{partial}$ = .09, medium; $\eta^2_{partial}$ = .25, large effect sizes.

Table 6

Exploratory Analysis: Mean Differences in Dysregulated Eating Symptoms in the Follicular Phase of Cycle One versus Cycle Two

Scale	Follicular Phase		*t(11)*	*p*	Cohen's *d*
	Cycle 1 *M* (*SD*)	Cycle 2 *M* (*SD*)			
Emotional Eating	0.28 (.80)	-0.28 (.91)	1.32	.22	0.65
External Eating	0.07 (.58)	-0.39 (1.10)	1.06	.31	0.53
Preoccupation with Food	-0.14 (.97)	-0.33 (.99)	0.34	.74	0.19
Intense Desire to Eat	0.21 (.65)	-0.84 (1.22)	2.20	.05	1.07
Behavioral Loss of Control	0.13 (.76)	-0.23 (.96)	0.86	.41	0.41
Euphoria in Loss of Control	-0.13 (.56)	0.37 (.82)	-1.74	.11	0.82

Note: This analysis was run on a subset of participants (n = 12) who had completed measurement of dysregulated eating during at least two days of a second menstrual cycle. Cohen's *d* effect size interpretation: small, $d = .20$, medium, $d = .50$, large, $d \geq .80$.

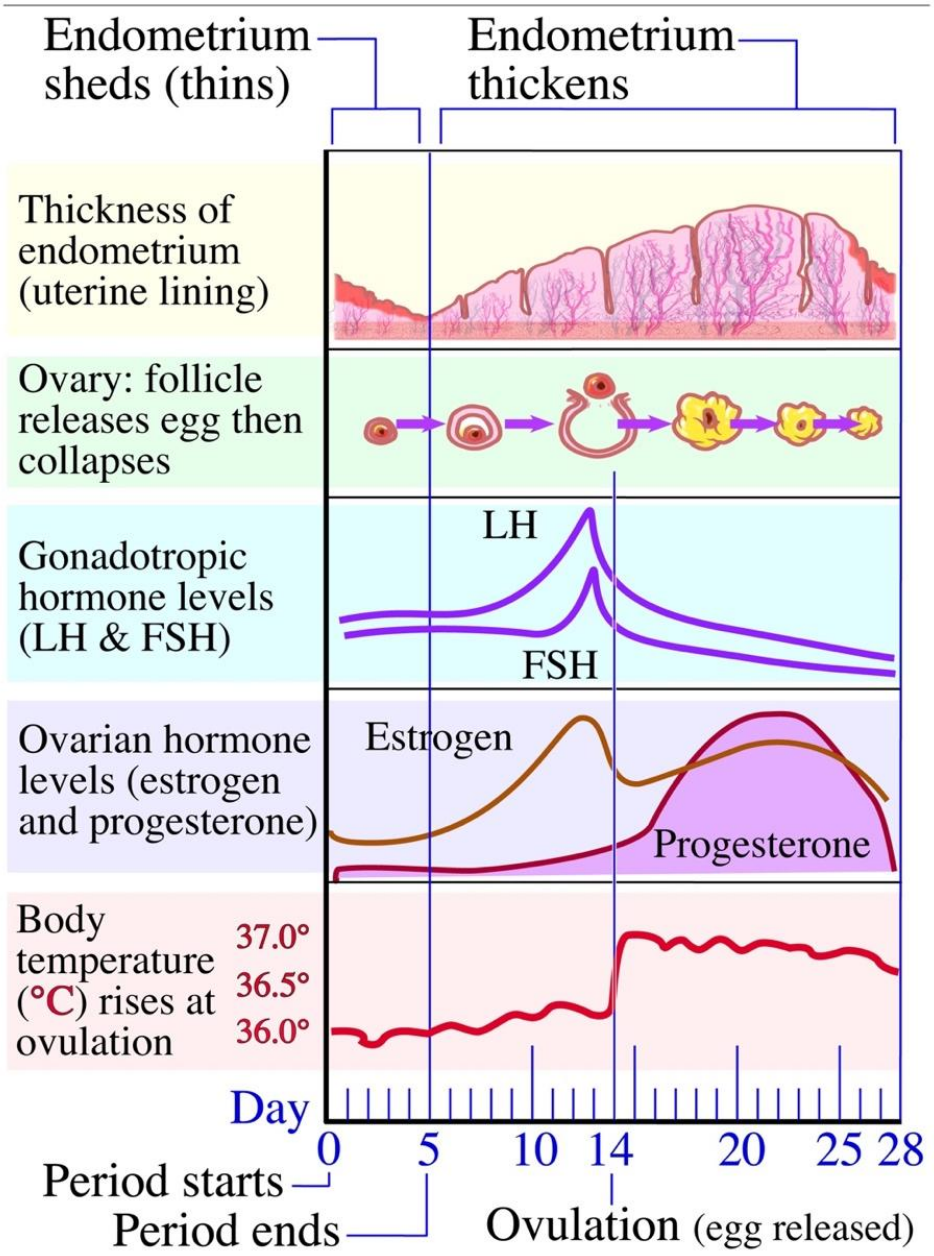

Figure 1. Physiological Changes across the Menstrual Cycle. LH = luteinizing hormone, FSH = follicle stimulating hormone. Although this image labels menstruation (i.e., period) as "Day 0", the project refers to the first day of menstruation as Day 1 of the cycle.

Days and Original Procedures in Average Menstrual Cycle

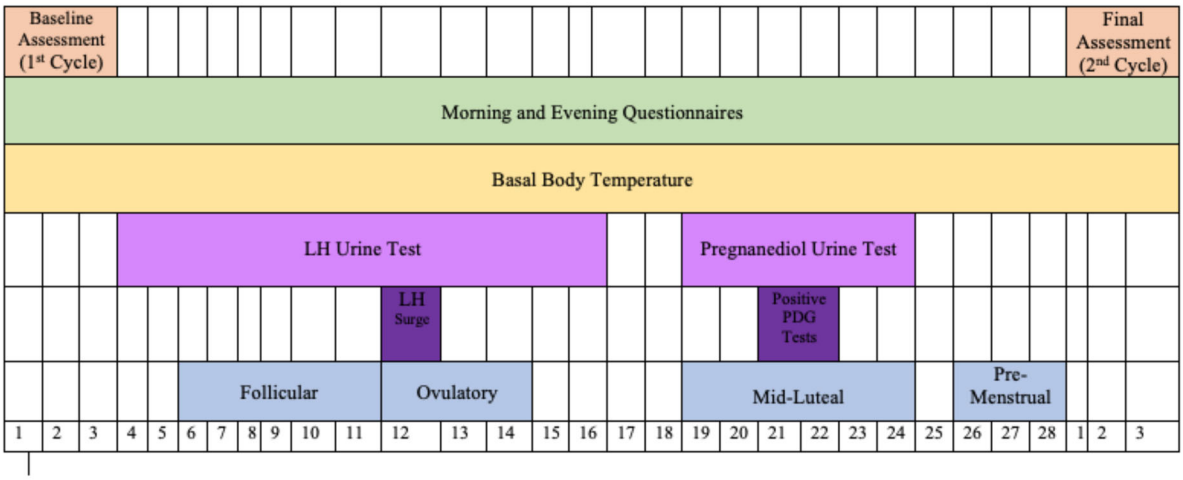

Figure 2. Original Daily Participant Procedures over the Menstrual Cycle. LH = luteinizing hormone. LH surge = positive LH urine test strip result. Although this graph depicts the phases as discrete time periods (e.g., the follicular phase as days 6-11) in reality the corresponding cycle days could vary because of cycle length and timing of events (e.g, day of LH surge). However, to facilitate phase comparison across participants, discrete time periods that correspond with distinct hormone profiles were used for each phase. In the original procedures, pregnanediol testing began 7 days after the LH surge and lasted six days.

Days and Adapted Procedures in Average Menstrual Cycle

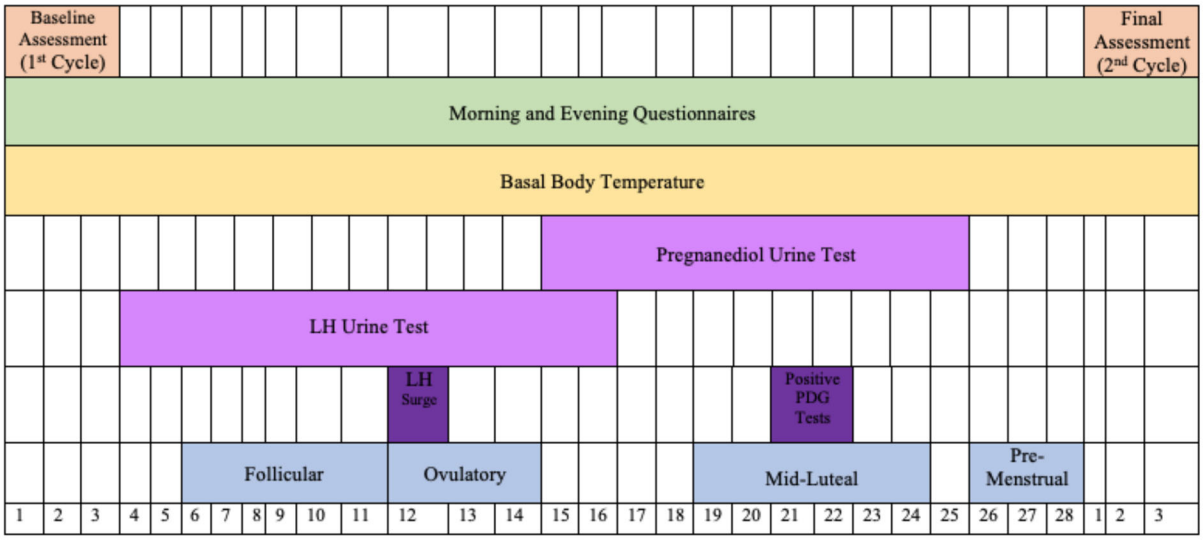

Figure 3. Adapted Daily Participant Procedures over the Menstrual Cycle. LH = luteinizing hormone. LH surge = positive LH urine test strip result. Although this graph depicts the phases as discrete time periods (e.g., the follicular phase as days 6-11), in reality the corresponding cycle days could vary because of cycle length and timing of events (e.g, day of LH surge). However, to facilitate phase comparison across participants, discrete time periods that correspond with distinct hormone profiles were used for each phase. In the modified protocol, participants tested Pdg levels for 11 days, and the midluteal phase was centered around the peak Pdg tests.

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CURRICULUM VITAE

Kimberly Stevens

Department of Psychology
University of Las Vegas, Nevada
Las Vegas, Nevada, 89154
Email: kimberlysuzannestevens@gmail.com

Education

Brigham Young University, Utah
B.S., Psychology, 2016