THE ROLE OF AFFECT IN BINGE EATING PHENOTYPES: AN EXAMINATION OF INDIVIDUAL DIFFERENCES IN EMOTION EXPERIENCE AND INTERACTIONS WITH OVARIAN HORMONES

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ABSTRACT

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Ovarian hormones significantly influence dysregulated eating in females. However, most women do not develop appreciable disordered eating, suggesting that ovarian hormones may not affect all women equally. In the first study of this thesis, I examined whether individual differences in trait negative affect (NA) moderate ovarian hormone-dysregulated eating associations in 446 women who provided saliva samples for hormone measurements and ratings of NA and emotional eating daily for 45 consecutive days. Women were at greatest risk for emotional eating when they had high trait NA and experienced a hormonal milieu characterized by low estradiol or high progesterone. While effects were significant in all women, the combination of high trait NA and high progesterone was particularly risky for women with a history of clinically significant binge eating episodes. These findings provide initial evidence that affective and hormonal risk interact to promote dysregulated eating, and that effects may be amplified in women with clinically significant binge eating.

Low emotion differentiation (the tendency to experience vague affective states rather than discrete emotions) is associated with psychopathology marked by emotion regulation deficits and impulsive/maladaptive behavior. However, research examining associations between emotion differentiation and dysregulated eating is still nascent. In the second study, I therefore examined associations between several measures of emotion differentiation and binge eating phenotypes across a spectrum of severity.

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CHAPTER 1

Introduction

Ovarian hormones (i.e., estradiol, progesterone) play an important role in risk for dysregulated eating in females during puberty and adulthood (Culbert, Sisk, & Klump, 2018; Klump, Culbert, & Sisk, 2017; Mikhail, Culbert, Sisk, & Klump, 2019). At puberty, low estradiol appears to potentiate latent genetic risk for binge eating in girls (Klump et al., 2018) and increase later rates of binge eating phenotypes in female animals (Klump et al., under review). In adult women, natural fluctuations in ovarian hormones across the menstrual cycle are associated with phenotypic changes in binge eating and related behaviors, such as emotional eating (i.e., the tendency to eat in response to negative emotions, which strongly correlates with binge eating and is considered a core mechanism; Haedt-Matt & Keel, 2011; Ricca et al., 2009). Understanding these hormonal effects may therefore be critical to understanding why some women experience clinically significant dysregulated eating.

Extensive animal research suggests that estrogen protects against binge eating and related phenotypes (e.g., excessive food intake) in adult females, while progesterone antagonizes the protective effect of estrogen and increases binge eating (Asarian & Geary, 2006; Kemnitz, Gibber, Lindsay, & Eisele, 1989). Results across the menstrual cycle in women are consistent with these findings. Emotional eating tends to be lowest just before ovulation, when estradiol is at its peak, and highest during the mid-luteal phase, when progesterone is high and estradiol is moderate (Klump et al., 2013b). Correspondingly, the interaction between estradiol and progesterone is the strongest predictor of emotional eating in models with direct measures of ovarian hormones (Klump et al., 2013b). In other words, women are most susceptible to emotional eating when both estradiol and progesterone are relatively elevated, as occurs during

the mid-luteal phase of the menstrual cycle. These effects are independent of changes in negative affect across the cycle (Klump et al., 2013b), and a similar pattern is observed for binge eating frequency among women with clinically significant binge eating (Edler, Lipson, & Keel, 2007; Klump et al., 2014).

Yet while nearly all women are exposed to ovarian hormones, not all women experience binge eating or related forms of dysregulated eating. It is possible that only women who are already at elevated biological/genetic or psychosocial risk experience increased dysregulated eating during risky hormonal milieus. If so, the strength of ovarian hormone-dysregulated eating associations would be expected to vary along with individual differences in these risk factors. To date, however, few moderators of ovarian hormone-dysregulated eating associations have been identified. Women with a history of clinically significant binge eating show stronger associations between ovarian hormone levels and emotional eating than women without a history of binge eating (Klump et al., 2014), but the source of these differences is unclear. Body mass index, dietary restraint, and impulsive personality dimensions (e.g., negative urgency) do not appear to moderate hormone-emotional eating associations across the menstrual cycle (Klump et al., 2013a; Racine et al., 2013). Genetic influences on emotional eating vary across different hormonal milieus (Klump et al., 2015, 2016), indicating that individual differences in genetic risk play a role. However, specific genes or molecular pathways have yet to be identified. Additional research is therefore needed to uncover factors that predict the strength of ovarian hormone-dysregulated eating associations, which may help identify the women at greatest risk.

One promising but as yet unexplored potential moderator is the general intensity of negative affect (e.g., anger, sadness, fear) a woman experiences across situations. Women who engage in binge eating tend to experience higher negative affect and increased rates of disorders

characterized by elevated negative affect (such as depression) than other women (e.g., Welch et al., 2016; Wolff, Crosby, Roberts, & Wittrock, 2000). Similarly, women who experience higher negative affect across time report more emotional eating than those who typically experience less negative affect (Racine et al., 2013b). Negative affect also correlates with other risk factors for dysregulated eating, such as stress and emotion regulation difficulties (Bolger, DeLongis, Kessler, & Schilling, 1989; Gross, 2015). This suggests that the general intensity of negative affect a woman experiences is a significant indicator of her overall risk for dysregulated eating.

Factors that make women more generally prone to intense negative affect (e.g., difficulties with emotion regulation, neurobiological vulnerabilities) could also potentially amplify the influence of ovarian hormones on binge eating and emotional eating. Persistent elevations in negative affect may therefore serve as a proxy for a range of underlying biological and/or psychosocial vulnerabilities that could render a woman more susceptible to dysregulated eating associated with shifts in ovarian hormones. While a prior study failed to find a moderating effect of negative *emotionality* (i.e., a personality dimension encompassing emotional lability and general negative affect; Patrick, Curtin, & Tellegen, 2001) on hormone-emotional eating associations (Racine et al., 2013a), the inclusion of other traits in this construct (e.g., persecutory beliefs, antisocial tendencies) may have obscured the effect of negative affect per se.

Elucidating whether individual differences in negative affect moderate relationships between ovarian hormones and dysregulated eating could help us better understand why some women are more vulnerable to hormone effects than others. It would also significantly enhance our understanding of the role of affective experience in risk for dysregulated eating. While prior research has shown that *within-person* fluctuations in negative affect are associated with within-person changes in emotional eating and binge eating (Haedt-Matt & Keel, 2011; Haedt-Matt et

al., 2014), it is also important to understand how *between-person* differences in affective experience contribute to heightened risk across situations.

The primary aim of the current study was therefore to examine whether ovarian hormone-emotional eating associations across the menstrual cycle differ as a function of individual differences in trait negative affect. Ovarian hormone-emotional eating associations were expected to be stronger among women with higher trait negative affect. A secondary aim was to investigate whether these moderating effects are similar across the full spectrum of binge eating-related pathology. To do so, the moderating effects of trait negative affect on ovarian hormone-emotional eating associations were first compared between women with and without clinically significant binge eating. Moderation of ovarian hormone–binge eating frequency associations was then examined in the subsample of women with a history of clinically significant binge eating.

Methods

Participants

Analyses were conducted with data from 446 female twins (ages 15-24; mean age = 17.84, *SD* = 1.79) from the *Twin Study of Hormones and Behavior Across the Menstrual Cycle* (Klump et al., 2013b). These participants were recruited through the Michigan State University Twin Registry (MSUTR), which identifies twins through birth records using previously described methods (Burt & Klump, 2013, 2019; Klump & Burt, 2006). Twins had to meet the following eligibility criteria to be included in the study: 1) menstruation every 22-32 days for the past 6 months; 2) no hormonal contraceptive use in the past 3 months; 3) no psychotropic or steroid medications in the past 4 weeks; 4) no pregnancy or lactation in the past 6 months; and 5) no history of genetic or medical conditions known to influence hormone functioning or

appetite/weight (Klump et al., 2013b).

This sample was demographically representative of the recruitment region with regard to race and ethnicity (Klump et al., 2013b). The majority of participants identified as White (*n* = 368; 82.5%), with smaller numbers identifying as Black or African-American (*n* = 51; 11.4%), Asian American (*n* = 2; 0.4%), American Indian or Alaska Native (*n* = 2; 0.4%), or more than one race (*n* = 22; 4.9%). The remaining participant (*n* = 1; 0.2%) did not identify her race. In addition, 9.4% (*n* = 42) of participants across races identified as Latina. The average participant BMI was 23.69 (*SD* = 5.47, range = 15.30-47.59). With respect to zygosity, 56.5% (*n* = 252) of twins were monozygotic and 43.5% (*n* = 194) were dizygotic.

Procedure

As described by Klump et al. (2013b), participants provided daily saliva samples for ovarian hormone measurements and ratings of affect, emotional eating, and binge eating frequency for 45 consecutive days. Saliva samples were collected in the morning, while affect, emotional eating, and binge eating ratings were acquired in the evening, ensuring that hormone measures for a given day preceded behavioral ratings. Three additional in-person assessments were conducted at the beginning, midpoint, and end of data collection. At each of these assessments, saliva samples were collected, BMI was measured, and study eligibility was reconfirmed. Between assessments, staff contacted participants once per week to answer questions and confirm protocol adherence. Dropout over the course of the study (3%) and missing data (≤ 6%) were minimal, and only a small number (3%) of twins became ineligible due to pregnancy or medication use (Klump et al., 2013b).

Measures

***Emotional Eating*.** Daily emotional eating was assessed using a version of the Dutch

Eating Behavior Questionnaire (DEBQ; van Strien, Frijters, Bergers, & Defares, 1986) Emotional Eating scale modified with permission to refer to that day. Participants rated how often 13 items assessing the desire to eat in response to negative emotions (e.g., "Did you have the desire to eat when you were irritated?") were true for them on that day on a scale from 1 (not at all) to 5 (very often). This version of the DEBQ Emotional Eating scale has been used in past studies investigating ovarian hormone-dysregulated eating associations in community samples (Klump, Keel, Culbert, & Edler, 2008), and correlates strongly with traditional measures of binge eating (e.g., the Bulimia scale of the Eating Disorders Inventory) and palatable food consumption in laboratory settings (van Strien, 2000). It has also been shown to differentiate between obese women with and without binge eating disorder (Schulz & Laessle, 2010), suggesting a specific association with binge eating pathology rather than weight status or overeating.

Binge Eating. History of binge eating episodes (BEs; representing current or past clinically significant binge eating) was assessed via interview using a version of the Eating Disorders Module from the Structured Clinical Interview for DSM-IV Axis 1 Disorders (SCID-IV; First, Spitzer, Gibbon, & Williams, 1996) modified to include additional symptom probes tailored to a community sample. There was good interrater reliability for the presence of BEs (kappa = .82; Klump et al., 2014).

Twenty-seven women (6.1% of the sample) reported current or past BEs. To maximize power with a community sample and maintain consistency with prior studies (Klump et al., 2014), this includes both participants with threshold objective binge eating episodes ($n = 15$; $\geq$ 1000 calories consumed and both behavioral (e.g., eating more than intended, being unable to stop eating, going back repeatedly to find more food) and psychological (e.g., feeling sad, guilty,

or disgusted with oneself after binge eating) indicators of severe loss of control) as well as those who may have consumed somewhat fewer calories (i.e., 600-999 calories) or reported less severe loss of control during BEs (i.e., endorsed some, but not all, behavioral indicators of loss of control along with psychological indicators). This approach is consistent with evidence that loss of control over eating is clinically significant, even if the amount of food consumed falls short of "objectively large" thresholds (i.e., ≥ 1000 calories) (Forney, Haedt-Matt, & Keel, 2014). In analyses, the presence of BEs was identified using a dichotomous indicator (0 = no BEs; 1 = current or past BEs).

Participants also reported the number of times they engaged in BEs (0 to 9 or more episodes) each day during the 45 days of the study. While these data were collected for all participants, they were only analyzed in participants with a history of BEs confirmed through the SCID-IV (described above), as only these participants would be expected to engage in clinically significant binge eating over the study period. To ensure that participants provided valid reports of BEs over the course of the study, they were given a detailed definition of a BE during the first study session and quizzed on their understanding of BEs with four case examples (see Klump et al., 2014 for additional details).

***Ovarian Hormones*.** Estradiol and progesterone levels were assayed using saliva samples, which promote greater compliance and show stronger hormone-behavior associations than more invasive methods (e.g., bloodspots; Edler, Lipson, & Keel, 2007). Salivary samples were analyzed for hormone levels using specialized enzyme immunoassay kits from Salimetrics, LLC (State College, PA) that show excellent reliability (intra- and interassay coefficients of variation: estradiol = 7.1% and 7.5%; progesterone = 6.2% and 7.6%), specificity (determined by interpolating the mean optical density minus 2 SD of 10-20 replicates at the 0 pg/ml level;

estradiol = .10 pg/ml; progesterone = 5 pg/ml), and method accuracy (measured via spike recovery and linearity; estradiol = 104.2% and 99.4%; progesterone = 99.6% and 91.8%) (Klump et al., 2013b). To optimize use of resources, samples were assayed daily during key periods of hormonal change (i.e., the mid-follicular through premenstrual phases) and every other day when hormone levels were expected to be low and stable (i.e., during menstrual bleeding and the early follicular phase).

***Menstrual Phases*.** Trained raters coded menstrual phase based on estradiol and progesterone hormone plots and recorded days of menstrual bleeding. Two raters coded menstrual phases for each participant, and discrepancies were resolved at weekly meetings (see Klump et al., 2015 for more detailed descriptions of the coding methods). In analyses, phase is identified using a dichotomous indicator (0 = pre-ovulation; 1 = post-ovulation). Observations were grouped into pre- and post-ovulation rather than more finely segmented phases of the menstrual cycle to conserve power to detect interaction effects.

Negative Affect. Daily negative affect was measured using the negative emotion items from the Positive and Negative Affect Schedule (PANAS; Watson, Clark, & Tellegen, 1988). Participants rated the extent to which they experienced 10 negative emotions (distressed, upset, guilty, scared, hostile, irritable, ashamed, nervous, jittery, and afraid) each day on a scale from 1 (very slightly or not at all) to 5 (extremely). Daily negative affect composites were created by summing across negative emotion ratings on a given day.

Trait negative affect was calculated by averaging the daily negative affect composite scores for each woman over the study. This approach was used in a prior study of trait negative affect in this sample (Racine et al., 2013b) and captures differences in overall tendencies toward negative affect that exist between people. An average of 41.97 days of PANAS ratings per

participant were available to compute trait negative affect (range = 26-45 days), providing a strong indication of participants' negative affect over time.

Statistical Analyses

***Data Preparation*.** As described by Klump et al. (2013b), five-day rolling averages were calculated for both estradiol and progesterone. Rolling averages were used for ovarian hormones because they reduce "noise" introduced through intermittent hormone release (Gladis & Walsh, 1987) and help to account for potential delays in hormone effects on behavior (Eckel, 2011). Similar concerns did not apply to daily negative affect; thus, analyses used single day values (rather than rolling averages) for this covariate. Single day values were also used for the dependent variables (i.e., daily emotional eating and binge eating) to avoid inducing strong autocorrelation.

As in past studies of ovarian hormone-dysregulated eating associations (Klump et at., 2013a, 2013b, 2014; Racine et al., 2013), all time-varying measures were standardized within person (i.e., the difference between each observation and a woman's mean was divided by the standard deviation of all observations for that woman). This allowed for modeling of how changes in hormone levels from a woman's average predicted changes in emotional eating and binge eating relative to her average. By contrast, trait negative affect was standardized across all participants in the sample (for analyses involving the full sample) or a given subsample (for analyses that examined effects separately in women with and without BEs). In other words, the difference between a woman's value and the mean value for all participants in the sample/subsample was divided by the standard deviation across participants. This provided a measure of a woman's trait negative affect relative to other women.

As described by Klump et al. (2013b), in the case of missing data for PANAS or DEBQ

Emotional Eating ratings, raw scores were prorated if ≤ 10% of items were missing and marked as missing otherwise. Rolling averages were calculated if there were ≥ 3 days of data within the 5-day window and counted as missing if there were < 3 days of data. Observations from anovulatory cycles were excluded from analyses, which is in line with past studies (Klump et al., 2015, 2016) and evidence that changes in eating associated with ovarian hormone fluctuations are altered during anovulatory cycles (Asarian & Geary, 2006). Five women (1.1% of the sample) had missing data for presence of BEs on the SCID-IV, and so were excluded from analyses involving BE status.

Statistical Models. All statistical analyses were conducted using Stata version 15 (StataCorp, 2017). Mixed linear models (MLMs) were used for analyses given the non-independence of the repeated measures twin data. Models involving the full sample used maximum likelihood estimation (MLE), while models involving only the subset of women with BEs used restricted maximum likelihood (REML) to account for small sample size. Three-level models were used, with observations (level 1) nested within participants (level 2), and participants nested within twin pairs (level 3). A random slope was placed on all time-varying predictors at the participant level. Covariance structures for the random effects and residuals were determined by optimizing model fit measures (e.g., AIC, BIC) to the extent possible while maintaining convergence; final models used a diagonal covariance structure for random effects and a Toeplitz structure for residuals to account for autocorrelation (with the correlation between residuals set to 0 after a lag of five days).

This statistical modeling approach was somewhat different from that used in prior research (e.g., Klump et al., 2013b) in terms of the statistical package used (i.e., Stata versus SPSS) and different variables/parameters modeled (e.g., autocorrelation was modeled explicitly

in the current analyses; BMI was not included as a covariate given minimal changes across the study period (see Klump et al., 2013b; 2014) and minimal effects on results (see Tables 1.7-1.9 for models including BMI)).

I compared results using my updated statistical modeling approach to those previously found in the sample from Klump et al. (2013b). Ovarian hormone-emotional eating associations were broadly consistent across statistical modeling approaches – both the original method and the updated method yielded a significant or trend-level estradiol X progesterone interaction, but no significant estradiol or progesterone main effects (see Table 1.1). This suggests that the different approaches lead to similar conclusions regarding ovarian hormone-emotional eating associations, although the models used in the current paper might produce slightly more conservative estimates than models used in prior papers.

It is theoretically possible that women who generally experience more intense negative affect also experience more variability in negative affect across the cycle, confounding between- and within-person effects. Daily negative affect was therefore included as a covariate in all models to account for this possibility and maintain consistency with prior studies (Klump et al., 2013b, 2014). Note that daily within-person-standardized negative affect is by definition linearly independent from a woman's trait negative affect (i.e., the correlation between them is zero). This is true because the mean of every participant's within-person-standardized daily negative affect across the study is zero, regardless of her *absolute* average level of negative affect.

I first examined whether trait negative affect moderated menstrual phase–emotional eating associations. Menstrual phase was analyzed in addition to ovarian hormone levels to allow for comparisons with previous studies that have examined menstrual phase alone (e.g., Lester, Keel, & Lipson, 2003), or both menstrual phase and hormones (e.g., Klump et al., 2014). In these

analyses, emotional eating was predicted from menstrual phase (i.e., pre- or post-ovulation), trait negative affect, and the interaction between menstrual phase and trait negative affect.

I then examined whether trait negative affect moderated associations between fluctuations in ovarian hormones themselves (i.e., within-person-standardized estradiol and progesterone) and emotional eating. Emotional eating was initially predicted from estradiol, progesterone, trait negative affect, the estradiol X trait negative affect interaction, and the progesterone X trait negative affect interaction. Results are also reported for models including the estradiol X progesterone and three-way estradiol X progesterone X trait negative affect interactions.

Next, I examined whether the moderating effects of trait negative affect on ovarian hormone–emotional eating associations differed between women with and without BEs (i.e., a double moderation model). I approached this question in two steps. First, I modeled (three-way) interactions between menstrual phase, BE status, and trait negative affect. I then modeled three-way interactions between BE status, trait negative affect, and estradiol/progesterone. The estradiol X progesterone interaction and higher order interactions involving this term were not included in this model so that a four-way interaction was not required. If analyses in the full sample indicated a significant estradiol X progesterone X trait negative affect interaction, I modelled this interaction in women with and without BEs separately to explore whether effect sizes differed between groups. Finally, I tested whether trait negative affect moderated menstrual phase/ovarian hormone influences on daily binge eating frequency in the subsample of women with BEs only. These analyses were identical to those previously described for emotional eating in the full sample, but with daily binge eating frequency as the outcome.

Note that all models represent cross-level interactions, in which a factor that differs

between people (i.e., trait negative affect) modifies the strength of within-person associations (e.g., between ovarian hormone levels and emotional eating on a given day). In these models, the main effect of trait negative affect can be conceptualized as an "adjustment factor" on the intercept (i.e., it modifies the prediction of a woman's level of emotional eating relative to her average when her levels of estradiol, progesterone, and daily negative affect are at their average). It does *not* represent the strength of the association between trait negative affect and average level of emotional eating, which is best captured by the correlation between these variables (presented in descriptive statistics below).

Results

Descriptive Statistics

Descriptive statistics for person-level and daily variables are presented in Table 1.2. Both raw scores and z-scores are included to provide an indication of variability across the sample and (for daily variables) within each participant over the study. A range of trait negative affect was represented in the sample (mean = 14.95; range = 10.04–30.49 out of a possible range of 10–50) and nearly the full possible range of *daily* negative affect was reported (range = 10–46 out of a possible range of 10–50). The distribution of negative affect aggregated across days in this sample was comparable to that previously observed in other non-clinical samples (e.g., Watson & Clark, 1999). There was also good variability in daily emotional eating scores (range = 1–4.69 out of a possible range of 1–5). As observed in prior research, higher trait negative affect was associated with more emotional eating (r = .47, p < .001) and a greater likelihood of having current or past BEs (OR = 1.95, p = .002). In contrast, trait negative affect was unrelated to average levels of estradiol (r < .001, p = .998) or progesterone (r = -.01, p = .824). As has been observed in prior research (e.g., Klump et al., 2016, 2018), average levels of emotional eating

were also not significantly related to average estradiol (r = -.02, p = .661) or progesterone (r = -.05, p = .292) levels; however, this does not preclude *within-person* associations between fluctuations in hormone levels and fluctuations in emotional eating from day to day that have been found in previous work (e.g., Klump et at., 2013a, 2013b, 2014; Racine et al., 2013).

Moderation by Trait Negative Affect

As shown in Table 1.3, trait negative affect significantly moderated the association between menstrual phase and daily emotional eating (β = .05, p = .017), such that the post-ovulatory period was riskier for emotional eating in women with higher trait negative affect. A simple slopes analysis was conducted to examine effects in women one standard deviation above the mean, at the mean, and one standard deviation below the mean in trait negative affect. Results indicated that the change in emotional eating from pre-ovulation to post-ovulation was significant in women with high trait negative affect (β = .06, p = .026), but not in women with moderate (β = .01, p = .446) or low (β = -.03, p = .233) trait negative affect.

In the model with direct measures of ovarian hormones, trait negative affect significantly moderated associations between emotional eating and both estradiol (β = -.03, p = .007) and progesterone (β = .04, p = .003) (see Table 1.3 and Figure 1.1). The pattern of moderation indicated that higher estradiol was more protective against, and higher progesterone riskier for, emotional eating in women with higher trait negative affect. Interestingly, no main effects of hormones were observed. In this context, the presence of significant interactions without significant hormone main effects indicates that the influence of ovarian hormones on dysregulated eating depends on a woman's level of trait negative affect (rather than suggesting that hormones do not impact dysregulated eating). Indeed, simple slopes analyses indicated that progesterone was only significantly (positively) associated with emotional eating for women

high in trait negative affect (high trait negative affect: β = .05, *p* = .003; moderate trait negative affect: β = .01, *p* = .224; low trait negative affect: β = -.02, *p* = .196). Similarly, estradiol was only significantly (negatively) associated with emotional eating for women high in trait negative affect, with a trend toward the opposite effect in women with low trait negative affect (high trait negative affect: β = -.04, *p* = .032; moderate trait negative affect: β = -.004, *p* = .730; low trait negative affect: β = .03, *p* = .083). When the estradiol X progesterone X trait negative affect interaction was added to the model, it was non-significant (β = .01, *p* = .563), and the estradiol X trait negative affect and progesterone X trait negative affect interactions remained significant (see Table 1.3). These findings suggest that the strength of the interactions between trait negative affect and estradiol/progesterone do not depend on the level of the other hormone (e.g., the estradiol X trait negative affect interaction is equally strong when progesterone is high as when progesterone is low).

Note that the lack of a significant estradiol X progesterone interaction (independent of trait negative affect) in the results reported in Table 1.3 may reflect several aspects of the current study design, including use of a slightly different statistical modeling approach than previous studies (see more on this above under Statistical Methods) and the presence of higher-order interaction terms, which alter the interpretation of lower-order effects. In this case, the lack of a significant estradiol X progesterone interaction indicates that this effect was not significant in people at the mean for trait negative affect in the current sample after controlling for all other main and interaction effects in these analyses (including interactions with trait negative affect that were not included in prior studies).

BE status did not significantly moderate the estradiol X trait negative affect interaction in predicting emotional eating (β = -.01, *p* = .890 for the three-way trait negative affect X estradiol

X BE status interaction; see Table 1.4). This suggests that low estradiol is equally risky for emotional eating in women with high trait negative affect with and without a history of BEs. However, BE status *did* significantly moderate associations between both menstrual phase and emotional eating (β = .19, p = .020 for the trait negative affect X menstrual phase X BE status interaction) and progesterone levels and emotional eating (β = .14, p = .004 for the trait negative affect X progesterone X BE status interaction). While the progesterone X trait negative affect interaction was significant in both women with and without BEs, it was amplified among women with BEs (β = .03 for women without BEs and β = .16 for women with BEs; see Table 1.6 and Figure 1.2). Simple slopes analyses indicated that higher progesterone was associated with increased emotional eating in women with BEs who had high trait negative affect (β = .17, p = .003), but decreased emotional eating in women with BEs who had low trait negative affect (β = -.15, p = .017). Exploratory analyses showed that the progesterone X trait negative affect interaction remained significant if the sample was restricted to women with *current* BEs (n = 21; β = .14, p = .003) or threshold objective binge eating episodes characterized by consumption of at least 1000 calories and severe loss of control (n = 15; β = .14, p = .002). This suggests that results are robust and effects in the BE subsample were not simply due to higher negative affect among women with current BEs as compared to women with past BEs only or different effects in women with more severe BEs.

Daily negative affect was the only significant predictor of *binge eating frequency* in the subsample of women with BEs; the main effects of menstrual phase/ovarian hormones and the menstrual phase/ovarian hormone X trait negative affect interactions were not significant. However, the pattern of moderation was in the same direction as previously described for emotional eating. Specifically, the post-ovulatory period (β = .11, p = .274) and higher

progesterone levels (β = .06, p = .505) were riskier for binge eating in women with higher trait negative affect (see Table 1.5). The lack of significant effects may be due in part to reduced power (a particular concern with cross-level interaction models), as only 16 (59.3%) participants in the BE subsample reported a binge frequency of 1 or greater during the study period.

Moderation by Trait Positive Affect

To determine whether effects were specific to negative affect, analyses were repeated using a measure of trait positive affect calculated from the PANAS positive emotion items. There were no significant interactions between trait positive affect and menstrual phase or estradiol or progesterone (with or without the estradiol X progesterone interaction) in the full sample, but there was a trend-level progesterone X trait positive affect X BE status interaction (β = .11, p = .073). In the subsample of women with BEs only, there was a trend-level progesterone X trait positive affect interaction (β = .11, p = .052) that was not present in the women without BEs. However, trait positive and negative affect were positively correlated (r = .23 in the BE subsample), raising the possibility that this effect could be due to variance shared with trait negative affect. Thus, trait negative affect was regressed out of trait positive affect to examine effects unique to trait positive affect. The interaction between progesterone and residualized trait positive affect (i.e., trait positive affect independent of the variance shared with trait negative affect) in predicting emotional eating was not significant even at a trend-level in women with BEs (β = .06, p = .323). In contrast, when trait positive affect was regressed out of trait negative affect, the progesterone X residualized trait negative affect interaction remained significant in women with BEs (β = .14, p <.001).

Discussion

This study was the first to examine how affective and hormonal risk interact to influence dysregulated eating in women. The results indicate that individual differences in trait negative affect are an important moderator of hormone effects, particularly in women with a history of clinically significant binge eating. Findings may help to resolve discrepancies in prior research showing significant main effects of ovarian hormones on dysregulated eating in women with bulimia nervosa (who would be expected to have elevated negative affect) (Edler et al., 2007) but not in women from a community sample (Klump et al., 2013b). Trait positive affect did not show the same moderating effects in exploratory analyses, suggesting a unique influence of trait negative affect (which mirrors prior findings indicating stronger associations between negative affect and emotional eating at the daily level; Haedt-Matt et al., 2014). Overall, these findings advance our understanding of individual differences in ovarian hormone influences on eating behavior, as well as our ability to predict who is at greatest risk for increases in dysregulated eating during risky hormonal milieus.

In the full sample, women with high trait negative affect were at greatest risk for emotional eating when estradiol was low or progesterone was high, as occurs during the early follicular (i.e., low estradiol) and mid-luteal (i.e., high progesterone) phases of the menstrual cycle. The same overall pattern of effects was observed in women with and without BEs, though some effects were stronger in the BE subsample (see below). These findings suggest that hormonal factors may interact with risk factors for sustained high negative affect to promote increases in dysregulated eating. Importantly, women with high trait negative affect also had higher average levels of emotional eating (i.e., independent of hormone levels). Thus, women reported the highest absolute levels of emotional eating when they were high in trait negative

affect *and* experienced a risky hormonal milieu characterized by low estradiol or high progesterone. These findings are broadly consistent with results from animal studies showing that female rats engage in the highest levels of binge eating when they have undergone ovariectomy (which removes the primary source of estradiol) and have been exposed to other sources of risk for dysregulated eating (i.e., food restriction and stress) (Micioni Di Bonaventura et al., 2017).

Interestingly, while significant in women with and without BEs, the interaction between progesterone and trait negative affect in predicting emotional eating was particularly pronounced among women with a history of clinically significant binge eating. The presence of stronger hormone effects in the BE subsample in this study parallels results from a previous study that did not examine individual differences in negative affect (Klump et al., 2014), and suggests that women with a history of clinically significant binge eating may be particularly sensitive to ovarian hormone influences. While prior studies across the menstrual cycle have suggested that progesterone increases dysregulated eating by counteracting the protective effects of estradiol (Klump et al., 2013b), findings from this study are consistent with recent evidence that progesterone may also strengthen the influence of psychosocial risk factors for disordered eating (e.g., weight-based teasing; Forney et al., 2019). The combination of sustained high negative affect and a within-person peak in progesterone may therefore be a "perfect storm" for dysregulated eating in women with a history of BEs, who may also be more likely to have other sources of biological or behavioral risk (such as a history of dieting) that potentiate these effects. However, a close inspection of the progesterone X trait negative affect interaction in women with BEs (see Table 1.6 and Figure 1.2) also shows that progesterone was *negatively* associated with emotional eating in a subset of women with a history of clinically significant binge eating

but relatively *low* trait negative affect. This suggests that ovarian hormones could affect dysregulated eating differently for women whose binge eating may be primarily driven by factors other than negative affect, a finding that is consistent with prior research showing individual differences in ovarian hormone influences on binge eating in women with bulimia nervosa (Edler et al., 2007).

Mechanistically, trait negative affect may serve as a proxy for neurobiological or psychosocial risk factors that could amplify the influence of ovarian hormones on dysregulated eating. One possibility is that women who experience consistently high negative affect have underlying vulnerabilities in serotonergic or dopaminergic neural circuits that impact both mood and eating behavior, and that these vulnerabilities become more pronounced during periods of hormonal risk. Estradiol has been shown to enhance dopaminergic and serotonergic signaling (Kuhn et al., 2010; McEwen & Alves, 1999; Morissette et al., 2008), while progesterone is associated with increased neural responsivity to negative stimuli (e.g., through increased amygdala reactivity; Andreano et al., 2018). Hormonal milieus characterized by low estradiol or high progesterone may therefore potentiate existing tendencies toward negative mood or emotion dysregulation, pushing women who are already at risk "over the edge" to engage in dysregulated eating. However, other possibilities also exist; for example, high trait negative affect could indicate the presence of chronic environmental stressors rather than biological vulnerabilities innate to an individual. Research incorporating neuroimaging and psychophysiological data and/or additional self-report measures of constructs like emotion regulation and stress are therefore needed to determine the key mechanisms underlying the effects observed in this study.

Some limitations of this study should be noted. First, while a subset of women reported a history of clinically significant binge eating, this group was relatively small (n = 27), and it was

not possible to examine potential differences across eating disorder diagnoses (i.e., bulimia nervosa versus binge eating disorder). Some caution is also warranted in interpreting interaction effects in this subsample due to small sample size. Replication in larger samples of women with clinically significant binge eating is needed, though it is worth noting that the direction of effects was consistent across women with and without BEs. Second, while the sample was demographically representative of the recruitment region, it had limited representation of women from some racial/ethnic groups (e.g., Asian/Asian American and Native American women). Third, while the PANAS is a well-validated and widely-used measure of negative and positive affect, it primarily captures high arousal emotions. Alternative measures of negative/positive affect that incorporate more low arousal emotions (e.g., bored, calm) may show different patterns of moderation.

In conclusion, this study provides initial evidence that individual differences in negative affect moderate hormonal risk for dysregulated eating. High trait negative affect may not only be a risk factor for dysregulated eating in itself, but may also be associated with stronger ovarian hormone influences on binge eating-related behaviors. Among women with a history of clinically significant binge eating, the combination of sustained high negative affect and a within-person peak in progesterone may be associated with particularly high risk for dysregulated eating. These findings are important given the high comorbidity rates between disorders characterized by binge eating and those characterized by elevated negative affect (e.g. mood and anxiety disorders, Ulfvebrand, Birgegård, Norring, Högdahl, & von Hausswolff-Juhlin, 2015). Clinicians should be aware that women with persistently high negative affect (e.g., those with depression) and a current or past eating disorder characterized by binge eating may be at elevated risk for dysregulated eating during the mid-luteal phase of their menstrual cycle

(when progesterone levels are naturally higher). Additional research is needed to identify the biological and/or psychosocial factors that may underlie these effects.

APPENDIX

Table 1.1. *Ovarian hormone-emotional eating associations across statistical modeling approaches*

Results from Klump et al. (2013b)			
Variable	**β**	**SE**	***p***
Intercept	-.04	.01	.004**
Estradiol	-.002	.02	.93
Progesterone	.03	.02	.20
Estradiol X Progesterone	.04	.02	.007**
Daily NA	.19	.03	<.001***
BMI	.07	.03	.04*
Results Using the Updated Modeling Approach, Including BMI			
Variable	**β**	**SE**	***p***
Intercept	-.03	.01	.04*
Estradiol	-.01	.02	.68
Progesterone	.01	.02	.55
Estradiol X Progesterone	.03	.01	.07
Daily NA	.16	.02	<.001***
BMI	.04	.02	.09
Results Using the Updated Modeling Approach, Without BMI			
Variable	**β**	**SE**	***p***
Intercept	-.03	.02	.07
Estradiol	-.01	.02	.61
Progesterone	.01	.02	.55
Estradiol X Progesterone	.02	.01	.09
Daily NA	.16	.02	<.001***

Note: Analyses were conducted using the sample from Klump et al. (2013b) (N = 196). The updated modeling approach used single-day values for emotional eating and daily NA, and employed a Toeplitz residual structure to account for autocorrelation. NA = negative affect; BMI = within-person fluctuations in body mass index across the study. * p < .05, ** p <.01, *** p <.001.

Table 1.2. *Descriptive statistics for person-level and daily variables*

Person-Level Variables			
Variable	**Mean**	***SD***	**Range**
Trait NA – raw score	14.95	3.77	10.04–30.49
Trait NA – z-score	0	1	-1.30–4.12
Daily Variables			
Variable	**Mean**	***SD***	**Range**
Emotional eating – raw score	1.30	.46	1–4.69
Emotional eating – person-centered z-score	0	.99	-4.08–6.56
Binge eating frequency – raw score	.22	.62	0–5
Binge eating frequency – person-centered z-score	0	.99	-1.51–4.80
Estradiol – raw score	2.90	1.54	.21–47.62
Estradiol – person-centered z-score	0	.98	-3.29–3.69
Progesterone – raw score	127.77	88.72	8.42–648.63
Progesterone – person-centered z-score	0	.98	-2.60–3.02
Daily NA – raw score	14.84	5.22	10–46
Daily NA – person-centered z-score	0	.99	-2.67–5.81

Note: NA = negative affect. Person-level variables were calculated from all available information, including ratings from anovulatory cycles. In contrast, descriptive statistics for daily variables only include values from ovulatory cycles, since these were the only values included in analyses. Binge eating frequency is reported for the subset of women with binge eating episodes only.

Table 1.3. *Interactions between trait negative affect and menstrual phase (pre- vs. post-ovulation) or estradiol/progesterone in predicting emotional eating in the full sample*

Interactions with Menstrual Phase				
Variable	**β**	**SE**	***p***	**95% CI**
Intercept	.002	.01	.875	-.03, .03
Post-ovulation	.01	.02	.446	-.02, .05
Trait NA	-.02	.01	.129	-.05, .01
Post-ovulation X trait NA	.05	.02	.017*	.01, .09
Daily NA	.14	.01	<.001***	.12, .16
Two-Way Interactions with Estradiol and Progesterone Separately				
Variable	**β**	**SE**	***p***	**95% CI**
Intercept	-.02	.01	.160	-.04, .01
Estradiol	-.004	.01	.730	-.03, .02
Progesterone	.01	.01	.224	-.01, .04
Trait NA	.01	.01	.221	-.01, .04
Estradiol X trait NA	-.03	.01	.007**	-.06, -.01
Progesterone X trait NA	.04	.01	.003**	.01, .06
Daily NA	.14	.01	<.001***	.12, .17
Three-Way Interactions with Estradiol and Progesterone				
Variable	**β**	**SE**	***p***	**95% CI**
Intercept	-.02	.01	.067	-.04, .002
Estradiol	-.002	.01	.886	-.03, .02
Progesterone	.01	.01	.303	-.01, .04
Trait NA	.01	.01	.342	-.01, .03
Estradiol X progesterone	.02	.01	.120	-.004, .04
Estradiol X trait NA	-.03	.01	.009**	-.06, -.01
Progesterone X trait NA	.04	.01	.005**	.01, .06
Estradiol X progesterone X trait NA	.01	.01	.563	-.01, .03
Daily NA	.14	.01	<.001***	.12, .16

Note: NA = negative affect. * $p < .05$, ** $p < .01$, *** $p < .001$.

Table 1.4. *Interactions between trait negative affect and menstrual phase (pre- vs. post-ovulation) or estradiol/progesterone in predicting emotional eating in the full sample, moderated by BE status*

Interactions with Menstrual Phase				
Variable	**β**	**SE**	***p***	**95% CI**
Intercept	.001	.01	.928	-.03, .03
Post-ovulation	.02	.02	.405	-.02, .06
Trait NA	-.02	.02	.265	-.05, .01
BE status	.08	.08	.326	-.07, .23
Trait NA X BE status	-.09	.06	.149	-.20, .03
Post-ovulation X trait NA	.04	.02	.090	-.01, .08
Post-ovulation X BE status	-.16	.10	.125	-.37, .04
Post-ovulation X trait NA X BE status	.19	.08	.020*	.03, .35
Daily NA	.14	.01	<.001***	.12, .17
Two-Way Interactions with Estradiol and Progesterone Separately				
Variable	**β**	**SE**	***p***	**95% CI**
Intercept	-.01	.01	.199	-.04, .01
Estradiol	-.004	.01	.759	-.03, .02
Progesterone	.02	.01	.196	-.01, .04
Trait NA	.01	.01	.218	-.01, .04
BE status	-.01	.06	.859	-.12, .10
Trait NA X BE status	-.01	.05	.904	-.09, .08
Estradiol X trait NA	-.03	.01	.032*	-.06, -.003
Progesterone X trait NA	.03	.01	.043*	.001, .05
Estradiol X BE status	-.02	.06	.744	-.14, .10
Progesterone X BE status	-.12	.06	.052	-.24, .001
Estradiol X trait NA X BE status	-.01	.05	.890	-.10, .09
Progesterone X trait NA X BE status	.14	.05	.004**	.04, .23
Daily NA	.14	.01	<.001***	.12, .17

Note: NA = negative affect; BE status = the presence of current or past clinically significant binge eating episodes. * $p < .05$, ** $p < .01$, *** $p < .001$.

Table 1.5. *Interactions between trait negative affect and menstrual phase (pre- vs. post-ovulation) or estradiol/progesterone in predicting daily binge eating frequency in the BE subsample only*

Interactions with Menstrual Phase				
Variable	**β**	**SE**	***p***	**95% CI**
Intercept	-.04	.06	.491	-.17, .08
Post-ovulation	.09	.09	.304	-.08, .27
Trait NA	-.05	.07	.459	-.20, .09
Post-ovulation X trait NA	.11	.10	.274	-.09, .32
Daily NA	.17	.06	.007**	.05, .29
Two-Way Interactions with Estradiol and Progesterone Separately				
Variable	**β**	**SE**	***p***	**95% CI**
Intercept	-.03	.05	.502	-.12, .06
Estradiol	-.09	.06	.134	-.21, .03
Progesterone	.03	.08	.739	-.13, .18
Trait NA	.02	.05	.709	-.08, .12
Estradiol X trait NA	-.004	.07	.955	-.14, .13
Progesterone X trait NA	.06	.09	.505	-.12, .24
Daily NA	.15	.06	.019*	.03, .28
Three-Way Interactions with Estradiol and Progesterone				
Variable	**β**	**SE**	***p***	**95% CI**
Intercept	-.02	.05	.610	-.12, .07
Estradiol	-.10	.06	.104	-.23, .02
Progesterone	.03	.08	.755	-.13, .18
Trait NA	.01	.05	.812	-.09, .11
Estradiol X progesterone	-.04	.07	.561	-.17, .09
Estradiol X trait NA	-.01	.07	.847	-.15, .12
Progesterone X trait NA	.06	.09	.511	-.12, .24
Estradiol X progesterone X trait NA	-.004	.08	.962	-.16, .15
Daily NA	.16	.07	.020*	.02, .29

Note: NA = negative affect; BE = clinically significant binge eating episode. For these analyses, trait negative affect was standardized *within the BE subsample*; thus, effects can be interpreted as the impact of having relatively high or low trait negative affect relative to other women with BEs. * $p < .05$, ** $p < .01$, *** $p < .001$.

Table 1.6. *Interactions between trait negative affect and estradiol/progesterone in predicting emotional eating in the subsamples of women with and without BEs*

Interactions in Women with BEs				
Variable	**β**	**SE**	***p***	**95% CI**
Intercept	-.02	.04	.643	-.10, .06
Estradiol	-.05	.05	.381	-.15, .06
Progesterone	.01	.04	.823	-.08, .10
Trait NA	.01	.04	.790	-.07, .09
Estradiol X trait NA	-.04	.05	.449	-.13, .06
Progesterone X trait NA	.16	.04	<.001***	.08, .24
Daily NA	.25	.06	<.001***	.14, .36
Interactions in Women without BEs				
Variable	**β**	**SE**	***p***	**95% CI**
Intercept	-.02	.01	.185	-.04, .01
Estradiol	-.003	.01	.822	-.03, .02
Progesterone	.01	.01	.228	-.01, .04
Trait NA	.01	.01	.218	-.01, .04
Estradiol X trait NA	-.03	.01	.031*	-.05, -.003
Progesterone X trait NA	.03	.01	.044*	.001, .05
Daily NA	.13	.01	<.001***	.11, .16

Note: NA = negative affect; BE = clinically significant binge eating episode. For these analyses, trait negative affect was standardized *within each subsample*; thus, effects can be interpreted as the impact of having relatively high or low trait negative affect relative to other women with (or without) BEs. * $p < .05$, ** $p < .01$, *** $p < .001$.

Table 1.7. *Interactions between trait negative affect and menstrual phase (pre- vs. post-ovulation) or estradiol/progesterone in predicting emotional eating in the full sample, with BMI included as a covariate*

Interactions with Menstrual Phase				
Variable	**β**	**SE**	***p***	**95% CI**
Intercept	-.004	.01	.775	-.03, .02
Post-ovulation	.02	.02	.225	-.01, .06
Trait NA	-.02	.01	.105	-.05, .005
Post-ovulation X trait NA	.05	.02	.011*	.01, .09
Daily NA	.14	.01	<.001***	.12, .16
BMI	.02	.01	.110	-.01, .05
Two-Way Interactions with Estradiol and Progesterone Separately				
Variable	**β**	**SE**	***p***	**95% CI**
Intercept	-.01	.01	.184	-.03, .01
Estradiol	-.0005	.01	.967	-.02, .02
Progesterone	.02	.01	.106	-.004, .04
Trait NA	.01	.01	.242	-.01, .03
Estradiol X trait NA	-.03	.01	.016*	-.06, -.01
Progesterone X trait NA	.04	.01	.003**	.01, .06
Daily NA	.14	.01	<.001***	.12, .17
BMI	.02	.02	.215	-.01, .05
Three-Way Interactions with Estradiol and Progesterone				
Variable	**β**	**SE**	***p***	**95% CI**
Intercept	-.02	.01	.108	-.04, .004
Estradiol	.001	.01	.909	-.02, .03
Progesterone	.02	.01	.138	-.01, .04
Trait NA	.01	.01	.345	-.01, .03
Estradiol X progesterone	.01	.01	.284	-.01, .03
Estradiol X trait NA	-.03	.01	.020*	-.05, -.005
Progesterone X trait NA	.04	.01	.005**	.01, .06
Estradiol X progesterone X trait NA	.004	.01	.681	-.02, .03
Daily NA	.14	.01	<.001***	.12, .17
BMI	.02	.02	.213	-.01, .05

Note: NA = negative affect; BMI = within-person fluctuations in body mass index across the study. * $p < .05$, ** $p < .01$, *** $p < .001$.

Table 1.8. *Interactions between trait negative affect and menstrual phase (pre- vs. post-ovulation) or estradiol/progesterone in predicting emotional eating in the full sample, moderated by BE status, with BMI included as a covariate*

Interactions with Menstrual Phase				
Variable	**β**	**SE**	***p***	**95% CI**
Intercept	-.004	.01	.796	-.03, .02
Post-ovulation	.02	.02	.247	-.02, .06
Trait NA	-.02	.01	.252	-.05, .01
BE status	.05	.07	.465	-.09, .20
Trait NA X BE status	-.08	.06	.147	-.19, .03
Post-ovulation X trait NA	.04	.02	.077	-.004, .08
Post-ovulation X BE status	-.12	.10	.249	-.32, .08
Post-ovulation X trait NA X BE status	.18	.08	.023*	.02, .34
Daily NA	.14	.01	<.001***	.12, .16
BMI	.02	.01	.150	-.01, .05
Two-Way Interactions with Estradiol and Progesterone Separately				
Variable	**β**	**SE**	***p***	**95% CI**
Intercept	-.01	.01	.231	-.03, .01
Estradiol	.0002	.01	.988	-.02, .02
Progesterone	.02	.01	.087	-.003, .05
Trait NA	.01	.01	.241	-.01, .04
BE status	-.01	.05	.830	-.12, .10
Trait NA X BE status	-.004	.04	.930	-.09, .08
Estradiol X trait NA	-.03	.01	.053	-.05, .0003
Progesterone X trait NA	.03	.01	.033*	.002, .05
Estradiol X BE status	-.004	.06	.951	-.13, .12
Progesterone X BE status	-.12	.06	.059	-.24, .005
Estradiol X trait NA X BE status	-.02	.05	.702	-.11, .08
Progesterone X trait NA X BE status	.12	.05	.012*	.03, .21
Daily NA	.14	.01	<.001***	.12, .17
BMI	.02	.02	.267	-.01, .05

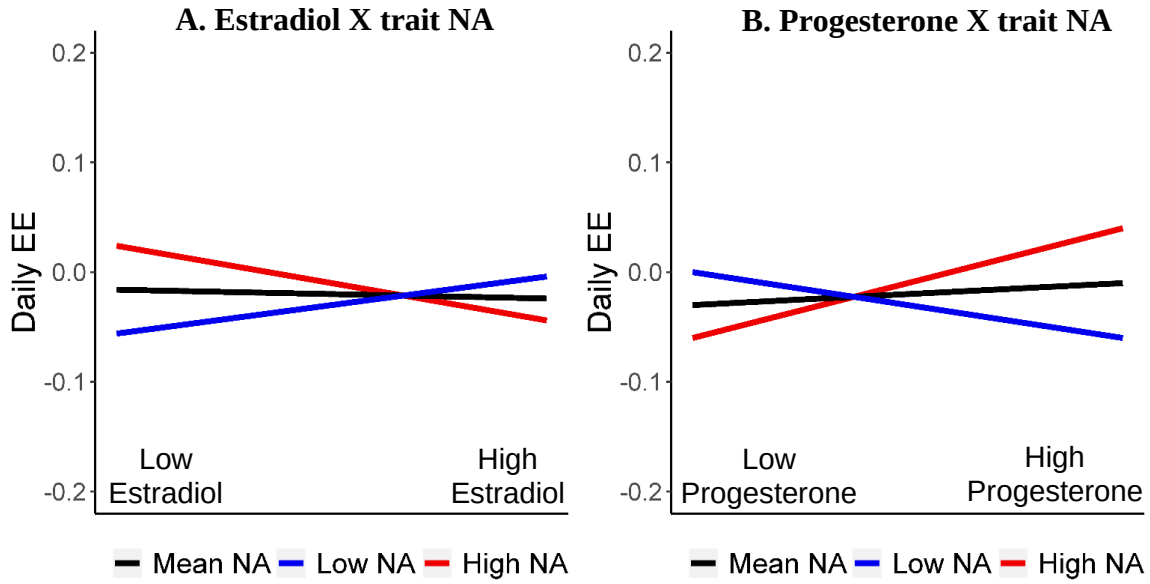
Note: NA = negative affect; BE status = the presence of current or past clinically significant binge eating episodes; BMI = within-person fluctuations in body mass index across the study.
* $p < .05$, ** $p < .01$, *** $p < .001$.

Table 1.9. *Interactions between trait negative affect and menstrual phase (pre- vs. post-ovulation) or estradiol/progesterone in predicting daily binge eating frequency in the BE subsample only, with BMI included as a covariate*

Interactions with Menstrual Phase				
Variable	**β**	**SE**	***p***	**95% CI**
Intercept	-.07	.06	.223	-.18, .04
Post-ovulation	.14	.09	.119	-.04, .31
Trait NA	-.06	.06	.319	-.19, .06
Post-ovulation X trait NA	.14	.10	.164	-.06, .33
Daily NA	.18	.06	.004**	.06, .30
BMI	-.04	.07	.537	-.18, .10
Two-Way Interactions with Estradiol and Progesterone Separately				
Variable	**β**	**SE**	***p***	**95% CI**
Intercept	-.02	.04	.621	-.09, .06
Estradiol	-.12	.06	.056	-.24, .003
Progesterone	.02	.10	.828	-.17, .21
Trait NA	.02	.04	.562	-.06, .10
Estradiol X trait NA	-.02	.06	.732	-.15, .10
Progesterone X trait NA	.06	.11	.575	-.15, .26
Daily NA	.17	.07	.014*	.03, .31
BMI	-.05	.08	.520	-.20, .10
Three-Way Interactions with Estradiol and Progesterone				
Variable	**β**	**SE**	***p***	**95% CI**
Intercept	-.02	.04	.630	-.10, .06
Estradiol	-.12	.06	.039*	-.23, -.01
Progesterone	.02	.10	.875	-.18, .21
Trait NA	.01	.04	.755	-.07, .10
Estradiol X progesterone	-.03	.07	.655	-.17, .11
Estradiol X trait NA	-.03	.06	.582	-.15, .08
Progesterone X trait NA	.06	.11	.587	-.15, .27
Estradiol X progesterone X trait NA	.002	.08	.978	-.16, .16
Daily NA	.17	.07	.017*	.03, .32
BMI	-.06	.08	.484	-.21, .10

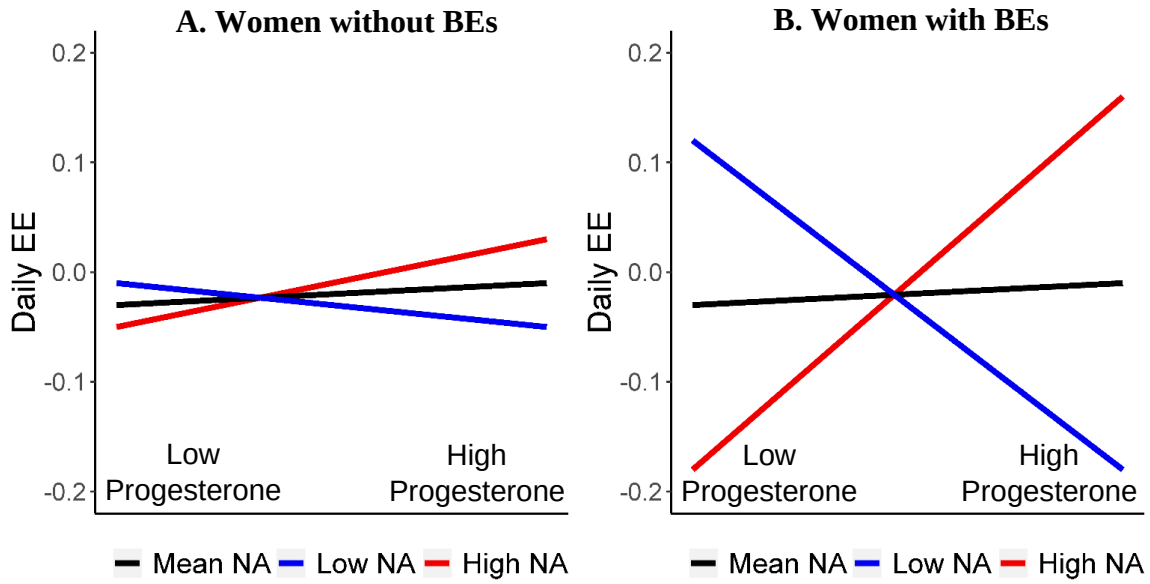
Note: NA = negative affect; BE = clinically significant binge eating episode; BMI = within-person fluctuations in body mass index across the study. For these analyses, trait negative affect was standardized *within the BE subsample*; thus, effects can be interpreted as the impact of having relatively high or low trait negative affect relative to other women with BEs. * $p < .05$, ** $p < .01$, *** $p < .001$.

Figure 1.1. *Interactions between trait negative affect and estradiol or progesterone in the full sample*



Note: EE = emotional eating; NA = trait negative affect. High/low estradiol/progesterone refer to hormone levels 1 standard deviation above or below the mean for a particular woman. High/low NA refer to trait negative affect 1 standard deviation above or below the mean across the sample.

Figure 1.2. *Interactions between trait negative affect and progesterone in women with and without BEs*



Note: EE = emotional eating; NA = trait negative affect; BE = clinically significant binge eating episode. High/low estradiol/progesterone refer to hormone levels 1 standard deviation above or below the mean for a particular woman. High/low NA refer to trait negative affect 1 standard deviation above or below the mean across the subsample (i.e., among women with or without BEs).

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CHAPTER 2

Introduction

Emotion differentiation, a person's general tendency to experience discrete emotions (e.g., angry or sad) as opposed to vague affective states (e.g., feeling "bad"), is associated with positive psychosocial functioning (Smidt & Suvak, 2015) and is reduced in individuals with several forms of psychopathology (e.g., depression, borderline personality disorder; Demiralp et al., 2012; Zaki, Coifman, Rafaeli, Berenson, & Downey, 2013). Negative (rather than positive) emotion differentiation has been most consistently linked to adaptive functioning in past studies (e.g., Barrett, Gross, Christensen, & Benvenuto, 2001; Demiralp et al., 2012; Kashdan, Ferssizidis, Collins, & Muraven, 2010; Pond et al., 2012; Zaki, Coifman, Rafaeli, Berenson, & Downey, 2013). Researchers have theorized that greater negative emotion differentiation (NED) is adaptive because it helps people more effectively tailor their responses to different kinds of affectively charged experiences (Smidt & Suvak, 2015). One clear example is emotion regulation strategy selection; while people with greater NED tend to use the same types of strategies as other people on average, they use these strategies more effectively in daily life (Kalokerinos, Erbas, Ceulemans, & Kuppens, 2019), suggesting that they may be better at picking the right strategy for a given situation. Though positive emotion differentiation (PED) has been less consistently linked to wellbeing, some studies have found that low PED is associated with greater engagement in behaviors that may feel good or provide relief in the moment, but have negative long-term consequences (e.g., restriction and use of compensatory behaviors in anorexia nervosa; Selby et al., 2014). While the mechanisms underlying these associations are not yet clear, it may be that the temporary positive feelings derived from some risky/maladaptive behaviors (e.g., purging to lose weight) are less distinct from other forms of positive affect (e.g.,

pride at getting an A in school) for people with low PED, which increases the likelihood of using maladaptive behaviors to feel good or feel better.

Both NED and PED have potential relevance for binge eating and related disorders (e.g., bulimia nervosa (BN), binge eating disorder (BED)), which are characterized by difficulty regulating negative emotions (e.g., Kenny, Singleton, & Carter, 2017; Monell, Clinton, & Birgegård, 2018) and behaviors that may be temporarily pleasurable, but ultimately detrimental (i.e., consumption of large amounts of palatable food). However, only two studies to date have examined associations between emotion differentiation and binge eating-related symptoms, each with methodological limitations. In the first study, Dixon-Gordon and colleagues (2004) found that lower PED was associated with greater binge eating urges in daily life in undergraduates with elevated borderline personality traits; however, this study only used one item to assess binge eating urges, which was examined over a single day of data collection. In the second study, lower NED was found to predict higher caloric intake in a laboratory setting in a mixed-gender undergraduate sample (Jones & Herr, 2018). Unlike many emotion differentiation studies, however, this study used hypothetical ratings of how participants *thought* they would feel in different scenarios to calculate NED rather than affect ratings in daily life, raising questions about external validity. While these studies represent an important start to examining associations between emotion differentiation and binge eating phenotypes, neither incorporated measures of clinically significant binge eating or collected emotion ratings longitudinally over an extended period in daily life to ensure emotion differentiation was measured accurately.

Given the above, the primary aim of the current study was to examine how positive and negative emotion differentiation relate to well-validated measures of binge eating and emotional eating (i.e., eating in response to negative emotions, a core mechanism and strong correlate of

binge eating; Haedt-Matt & Keel, 2011; Ricca et al., 2009) using an intensive, longitudinal study design. Affect and emotional eating ratings were collected over 45 consecutive days in a large, population-based sample of women (including a subset of women with clinically significant binge eating), and binge eating was assessed via clinical interview. It was hypothesized that women who experience greater emotional eating or have a history of clinically significant binge eating would differentiate less between different positive and negative emotions.

A secondary aim was to examine whether associations are consistent across different methods of calculating emotion differentiation. At least two methods have been used in past research, including the intraclass correlation coefficient (ICC) between ratings of negative or positive emotions across days (e.g., Dixon-Gordon et al., 2014; Jones & Herr, 2018; Kalokerinos et al., 2019; Kashdan et al., 2010; Selby et al., 2014) and the average interitem correlation between each pair of negative or positive emotions (e.g., Barrett et al., 2001; Demiralp et al., 2012; Zaki et al., 2013). While these methods have been used somewhat interchangeably, it is unclear whether they in fact show the same patterns of associations with specific forms of psychopathology, or whether they might tap different facets of emotion differentiation that relate to outcomes differently. Other measures of emotion differentiation that focus more explicitly on differences in the intensity of different emotions are also conceivable, such as the average daily variance between different positive or negative emotion ratings. Three measures of emotion differentiation (the intraclass correlation coefficient (ICC), average interitem correlation, and average daily variance – see Methods) were therefore analyzed in this study to examine whether different operationalizations of this construct show different associations with binge eating phenotypes and, if so, which specific facets are most relevant to emotional eating and binge eating.

Methods

Participants

Analyses included 482 female twins (ages 15-25; mean age = 17.86, *SD* = 1.82) from the *Twin Study of Hormones and Behavior Across the Menstrual Cycle* (see Klump et al., 2013 for additional details about this sample). Seven (1.4% of the original sample) participants were excluded due to completing less than 23 days' (i.e., 50% of the total possible number of days) worth of affect ratings. This cutoff was chosen due to concerns that fewer affect ratings might be insufficient to accurately calculate emotion differentiation variables; however, results were qualitatively unchanged if excluded participants were included in analyses.

Participants were recruited through the Michigan State University Twin Registry (MSUTR), which identifies twins through birth records using previously described methods (Burt & Klump, 2013, 2019; Klump & Burt, 2006). The sample was demographically representative of the recruitment region with regard to race and ethnicity (Klump et al., 2013). The majority of participants identified as White (*n* = 393; 81.5%), with smaller numbers identifying as Black or African American (*n* = 60; 12.4%), Asian American (*n* = 4; 1%), American Indian or Alaska Native (*n* = 2; 0.4%), or more than one race (*n* = 22; 4.6%). The remaining participant (0.2%) did not identify her race. In addition, 9.1% (*n* = 44) of participants across races identified as Latina.

Procedure

As described by Klump et al. (2013), participants provided daily ratings of positive and negative emotions and emotional eating for 45 consecutive days. Participants had their BMI measured at three in-person assessments over the study and completed a structured clinical interview assessing binge eating at the end of the study (see below). Between assessments, staff

contacted participants once per week to answer questions and confirm protocol adherence. Dropout over the course of the study (3%) and missing data (≤ 6%) were minimal (Klump et al., 2013). On average, participants provided 41.59 days' worth of negative/positive emotion ratings and 41.53 days' worth of emotional eating ratings (range = 23–45 days for all variables); participants who provided data on fewer than 45 days either discontinued participation in the study prematurely or failed to complete all scales on certain days.

Measures

***Emotion Differentiation*.** Three emotion differentiation measures were created from daily positive and negative emotion ratings from the Positive and Negative Affect Schedule (PANAS; Watson, Clark, & Tellegen, 1988). Using the PANAS, participants rated the extent to which they experienced 10 negative emotions (distressed, upset, guilty, scared, hostile, irritable, ashamed, nervous, jittery, afraid) and 10 positive emotions (interested, excited, strong, enthusiastic, proud, alert, inspired, determined, attentive, active) on a scale from 1 (very slightly or not at all) to 5 (extremely) each day. Emotion differentiation variables were then calculated as follows:

Intraclass correlation coefficient (ICC). First, the average intraclass correlation coefficient with absolute agreement (rather than consistency; see Shrout & Fleiss, 1979) was calculated for negative/positive emotion ratings across days for each participant. This method has been used in past studies of emotion differentiation in relation to disordered eating (Dixon-Gordon et al., 2014; Jones & Herr, 2018; Selby et al., 2014) and other impulsive behaviors (e.g., substance use; Kashdan et al., 2010). As in past studies (Dixon-Gordon et al., 2014; Jones & Herr, 2018; Kalokerinos et al., 2019), values were Fisher r-to-z transformed to induce a normal distribution and subtracted from 1 so that higher scores would indicate greater emotion

differentiation.

Conceptually, people who distinguish more between emotions on a single day (e.g., feel embarrassed rather than irritated) or experience specific emotions as more intense than others across time (e.g., *generally* feel more embarrassed than irritated) will have greater emotion differentiation on the ICC measure. Conversely, people whose overall affect intensity varies more from day to day (e.g., feel really bad one day, but fine the next) will have lower emotion differentiation on this measure. Thus, emotion differentiation as measured by the ICC is greatest when someone consistently experiences some emotions as more intense than others (suggesting they can distinguish between emotions), but shows little variability in how good or bad they feel from one day to the next. Note that this dependence on variability in overall affect across days distinguishes the ICC from the other measures described below.

Average interitem correlation. Second, the average interitem correlation across all pairs of negative/positive emotions was calculated for each participant. This method has been used in past studies of depression (Demiralp et al., 2012) and borderline personality disorder (Zaki et al., 2013). Pearson correlations were calculated for each pair of negative or positive emotions (e.g., hostility and shame, hostility and fear, etc.), then averaged. As with the ICC measure (and consistent with past studies; Barrett et al., 2001; Demiralp et al., 2012; Zaki et al., 2013), values were Fisher r-to-z transformed and subtracted from 1 so that higher scores would indicate greater emotion differentiation. If there was no variability in a participant's ratings of a certain emotion (e.g., it was rated "1" on every day of the study), correlations with that emotion were excluded from the final calculation because it is not possible to calculate a correlation with a constant. One participant (0.2%) only rated a single negative emotion higher than "1" on any day of the study, and so was excluded from analyses involving NED calculated using the average interitem

correlation because no pairwise correlations could be computed.

Conceptually, the average interitem correlation is based on the idea that emotions that covary little across time are more likely to be experienced as distinct. Unlike the ICC and average daily variance (described below), this measure does not take into account absolute differences in ratings of different emotions (e.g., how much more embarrassed than irritable a person feels). Thus, while the average interitem correlation is a useful measure of how emotions rise and fall together, it may omit other conceptual dimensions of emotion differentiation (i.e., differences in the absolute intensity of different emotions).

Average daily variance. Finally, the variances of each participant's negative emotion ratings and positive emotion ratings were calculated on each day, then averaged independently across the study (creating one measure of NED and one measure of PED per participant). This method was newly developed to create a measure that captured differences in the intensity of different emotions on the same day and was independent of variability in affect across days. Higher scores on this measure indicate more variability between individual emotion ratings, and thus greater emotion differentiation. Note that unlike the ICC and average interitem correlation, the average daily variance *only* captures differences in the absolute levels at which emotions are experienced on the same day, omitting how emotions are related across days.

Binge Eating Phenotypes. *Emotional eating.* Emotional eating was assessed with a version of the Emotional Eating scale from the Dutch Eating Behavior Questionnaire (DEBQ; van Strien, Frijters, Bergers, & Defares, 1986) modified with permission to refer to that day. Participants rated how often 13 items assessing the desire to eat in response to negative emotions were true for them on a scale from 1 (not at all) to 5 (very often). The DEBQ Emotional Eating scale correlates strongly with measures of binge eating (e.g., the Bulimia scale of the Eating

Disorders Inventory) and palatable food consumption in laboratory settings (van Strien, 2000), and differentiates between obese women with and without binge eating disorder (Schulz & Laessle, 2010). Internal consistency is excellent (average $\alpha = .90$ in the current study; see Klump et al., 2014). An overall measure of emotional eating was created by averaging the daily ratings for each participant over the study to allow for comparisons with person-level emotion differentiation variables.

Objective binge eating. Lifetime history of objective binge eating episodes (OBEs, representing current or past clinically significant binge eating) was assessed using a version of the Eating Disorders Module from the Structured Clinical Interview for DSM-IV Axis 1 Disorders (SCID; First, Spitzer, Gibbon, & Williams, 1996) modified to include both DSM-IV and DSM-5 (American Psychiatric Association, 1994, 2013) criteria and additional symptom probes tailored to a community sample. There was good interrater reliability for lifetime presence of OBEs (kappa = .82; Klump et al., 2014).

Twenty-nine women (6.0%) reported lifetime OBEs in this sample. To maximize power with a community sample, this includes both participants with threshold ($n = 15$; ≥ 1000 calories consumed, behavioral and psychological indicators of loss of control) and subthreshold ($n = 14$; may have consumed only 600-999 calories or reported less severe loss of control over eating) OBEs. It also includes women who may not have met the 1-2 times/week OBE frequency requirement for a DSM-IV or DSM-5 diagnosis of BED or BN. This approach is consistent with evidence that subthreshold disorders characterized by binge eating are associated with distress and functional impairment (Wilson & Sysko, 2009). In analyses, lifetime history of OBEs was identified using a dichotomous indicator (0 = no OBEs; 1 = current or past OBEs). Seven participants (1.5%) were missing OBE data on the SCID, and so were excluded from analyses

involving this measure.

Covariates. *BMI.* BMI (kilograms/meters^2) was included as a covariate to ensure that associations between emotion differentiation and binge eating phenotypes were not confounded by potential differences in weight among high/low differentiators. BMI was calculated from height and weight measured using a wall-mounted ruler and digital scale, respectively. The BMI values used in this study are an average of the measurements obtained during three in-person assessments over the 45 days of data collection.

Affect intensity. If emotion differentiation was correlated with overall affect intensity (i.e., how strongly positive/negative someone feels), then associations between emotion differentiation and binge eating phenotypes could be confounded by associations between affect intensity and binge eating/emotional eating. To examine this possibility, overall levels of negative and positive affect intensity were calculated by averaging the daily negative/positive affect ratings for each participant over the study and included as covariates in analyses.

Statistical Analyses

I first examined Pearson correlations between NED and PED and emotional eating to provide initial indications of associations without covariates. Mixed linear models (MLMs) that accounted for nesting of participants within twin pairs were then conducted for NED and PED separately, with emotional eating or presence of lifetime OBEs as the outcome. MLMs first included BMI as the only covariate. Positive and negative affect intensity were then added to examine whether NED and PED were associated with binge eating phenotypes independent of individual differences in affect intensity. Results of the MLMs are presented as standardized regression coefficients (for emotional eating) and odds ratios (for the presence of lifetime OBEs).

Results

Descriptive Statistics

Descriptive statistics are presented in Table 2.1. A wide range of emotional eating was represented (average DEBQ Emotional Eating score range = 1–4.02 out of a possible range of 1–5), indicating good variability in binge eating symptoms. Participants also varied considerably on indices of emotion differentiation. Participants' untransformed average interitem correlations ranged from -.05 to .63 for negative emotions, and from -.01 to .69 for positive emotions. Similar variability was observed for the average daily variance in emotion ratings (range = .004–3.29 for negative emotions and .04–3.26 for positive emotions out of a possible range of 0–4.44) and untransformed ICC values (range = -.15–.91 for negative emotions and -.04–.95 for positive emotions, where an ICC of 1 indicates perfect agreement between ratings of individual emotions and thus no differentiation). While some researchers discard negative ICC values, I retained them because these values can be valid when variability within groups (i.e., between different emotion ratings on the same day) is much greater than variability between groups (i.e., in overall affect levels across days) (Kenny, Mannetti, Pierro, Livi, & Kashy, 2002). Results were unchanged if these participants (n = 6) were excluded from analyses.

Pearson Correlations

Pearson correlations are presented in Table 2.2. Measures of NED and PED calculated using the same method (e.g., both calculated using ICCs) were moderately correlated with each other (rs = .23–.41, ps <.001), suggesting that NED and PED represented related but distinguishable constructs. With respect to correlations between different measures of emotion differentiation, NED/PED calculated using the ICC were highly correlated with NED/PED calculated using the average interitem correlation (rs >.80), but these measures were more

weakly correlated with NED/PED calculated using the average daily variance (*r*s = -.14 to .26). This suggests that the average daily variance between emotion ratings taps a different aspect of emotion differentiation than the ICC/average interitem correlation.

None of the PED measures were significantly associated with emotional eating but, as expected, NED measured using the ICC (r = -.17, p <.001) and the average interitem correlation (r = -.09, p = .044) were negatively correlated with emotional eating. Contrary to expectations, however, NED measured using the average daily variance was *positively* correlated with emotional eating (r = .28, p <.001). Different NED measures also showed different patterns of correlations with negative affect intensity (i.e., negative correlations for the ICC/average interitem correlation and a positive correlation for the average daily variance; see Table 2.2). Thus, inconsistent associations between NED measures and emotional eating could be due in part to confounding by negative affect intensity (especially as negative affect intensity was also significantly correlated with emotional eating; r = .46, p <.001). MLMs controlling for affect intensity examined this possibility directly (see below).

Multilevel Models

Negative Emotion Differentiation. Associations between NED and binge eating phenotypes are presented in Table 2.3. When controlling for BMI alone, lower NED measured using the ICC (OR = .54, p = .009) and average interitem correlation (OR = .65, p = .036) were associated with greater odds of lifetime OBEs. Lower NED measured using the ICC was also associated with greater emotional eating (β = -.13, p = .002). In contrast (but consistent with the Pearson correlations), *higher* NED measured using the average daily variance was associated with greater emotional eating (β = .25, p <.001); however, it was not significantly related to lifetime OBEs.

When positive and negative affect intensity were added to the models, associations between NED measured using the ICC or average interitem correlation and binge eating phenotypes were no longer significant (though there remained a trend-level association between lower NED measured using the ICC and greater odds of lifetime OBEs; see Table 2.3). However, *lower* NED measured using the average daily variance was now associated with more emotional eating ($\beta = -.31$, $p < .001$) and greater odds of lifetime OBEs (OR = .39, $p = .020$). In other words, the unique variance associated with NED measured using the average daily variance (independent of negative and positive affect intensity) was *negatively* related to binge eating phenotypes. This was likely obscured in previous models by the strong, positive correlation between this measure of NED and negative affect intensity (importantly, however, indices of multicollinearity were within acceptable limits – i.e., VIF < 4 for all predictors; O'Brien, 2007).

Positive Emotion Differentiation. Associations between PED and binge eating phenotypes are presented in Table 2.4. When controlling for BMI alone, the only significant association was between lower PED measured using the ICC and greater odds of lifetime OBEs (OR = .58, $p = .030$). After controlling for positive and negative affect intensity, this association remained significant (OR = .52, $p = .008$), and was joined by a similar association between lower PED measured using the average interitem correlation and greater odds of lifetime OBEs (OR = .64, $p = .045$). While PED measured using the average daily variance was not significantly related to lifetime OBEs, the association was in the expected direction (OR = .67, $p = .139$), and lower PED on this measure was associated with greater emotional eating ($\beta = -.15$, $p < .001$).

In summary, after controlling for potential confounds (i.e., BMI, positive and negative affect intensity), lower PED was associated with greater odds of lifetime OBEs across two measures (the ICC and average interitem correlation) and greater emotional eating on the third

(the average daily variance). Lower NED measured using the average daily variance was also associated with greater odds of lifetime OBEs and greater emotional eating. Note that the overall pattern of findings was unchanged if NED and PED were included together in the same model (see Table 2.5), except that the association between PED measured using the average interitem correlation and lifetime OBEs was only present at a trend level (OR = .67, p = .088).

Discussion

This is one of few studies examining associations between emotion differentiation and binge eating phenotypes, and the first to use data collected over several weeks in daily life, multiple well-validated measures of binge eating symptoms across different levels of severity, a large, population-based sample, and more than one measure of emotion differentiation. Overall, results provide some support for an association between lower emotion differentiation and increased risk for binge eating phenotypes. Specifically, lower PED was related to significantly greater odds of lifetime OBEs after controlling for affect intensity across two measures of emotion differentiation that have been used in past research, and lower NED was related to binge eating phenotypes on a third measure. However, differences in associations across emotion differentiation measures and between emotional eating and clinically significant objective binge eating indicate the need for further research incorporating several operationalizations of emotion differentiation and binge eating behavior.

The observed associations between lower PED and greater odds of lifetime OBEs are consistent with previous research linking binge eating urges in daily life to low PED (Dixon-Gordon et al., 2014). While speculative, it may be that individuals with low PED more readily substitute the pleasure or relief associated with palatable food intake for other sources of (perhaps less easily accessible) positive affect. In other words, for a person with low PED, the

positive feelings associated with eating highly palatable foods may feel more similar to those associated with seeing a close friend or being praised by one's supervisor, increasing the likelihood of engaging in binge eating to experience positive feelings. If the relationship between lower PED and binge eating is primarily driven by hedonic processes (i.e., desire for positive affect), this may help explain why low PED was less consistently associated with eating in response to negative emotions (i.e., emotional eating) in this study. Importantly, while binge eating is often associated with negative affect, disruptions in reward processes have also been implicated as a potentially significant mechanism (e.g., Avena & Bocarsly, 2012; Ma et al., in press; Manwaring, Green, Myerson, Strube, & Wilfley, 2011).

Lower NED was less consistently linked to binge eating phenotypes – significant associations were only observed with the average daily variance measure. While associations with this measure could potentially be spurious, consistent results across emotional eating and clinically significant binge eating suggest a meaningful effect. One possibility is that the average daily variance captures different facets of emotion differentiation than the ICC and average interitem correlation, and that these particular aspects of NED are most closely associated with binge eating. The average daily variance most directly taps differences in the experience of different emotions on the same day, rather than covariances or consistent differences in the intensity of specific emotions across days. Low NED on this measure might therefore represent a tendency to experience multiple negative emotions simultaneously (i.e., to feel angry, sad, and scared rather than just angry) more than an inability to conceptually distinguish between emotions (which may be better captured by the other NED measures that assess consistent patterns of relationships between specific emotions over time). An association between binge eating phenotypes and lower NED on the daily variance measure – but not other NED measures

– could therefore suggest that a person is more likely to experience binge eating if they tend to experience mixed negative emotions, even when those emotions are conceptually distinct. This could reflect the fact that it is easier to generate an adaptive, situation-appropriate response to one or two intense negative emotions than to multiple negative emotions experienced at a more moderate level, which may be more likely to lead to binge eating or other self-soothing behaviors. PED measured using the average daily variance may be less strongly associated with binge eating phenotypes because situations that evoke positive emotions are less likely to call for a specific change in behavior or use of emotion regulation strategies (e.g., Gross, Richards, & John, 2006), rendering the "mixed signal" of multiple positive emotions less problematic.

Differences in associations across emotion differentiation measures highlight the fact that measures are not necessarily interchangeable and each have conceptual limitations (e.g., only measuring covariances between emotions when differences in intensity may be relevant). While use of multiple emotion differentiation measures can help identify the aspects of this construct that are most relevant to a phenotype, the current measures are all indirect and require some degree of interpretation, which introduces some uncertainty about what is being measured by each. More explicit emotion differentiation measures, such as questionnaires or laboratory paradigms that assess "just-noticeable differences" between stimuli with different emotional charges (e.g., Norton, McBain, Holt, Ongur, & Chen, 2009), could play an important role in establishing convergent validity for existing measures and determining which findings most consistently replicate across ways of conceptualizing emotion differentiation. Experimental designs could also be used to determine how interventions designed to increase emotion differentiation influence different measures of this construct, and which changes correlate most strongly with changes in behavior (e.g., reduced binge eating).

Before closing, some limitations of this study should be noted. First, although the PANAS is a widely used measure of positive and negative emotions, it primarily focuses on high arousal emotions (e.g., scared, excited) – thus, results may differ if low arousal emotions (e.g., bored, calm) are included in calculations of emotion differentiation. Second, while this community sample showed ample variability in emotion differentiation and binge eating phenotypes, it is unclear whether results would fully generalize to clinical populations. For example, it is possible that associations between emotion differentiation and emotional eating could be stronger among women with current eating disorders. Third, there was low representation of women from some racial/ethnic groups (i.e., Asian American and Native American women) among participants due to the demographic composition of the recruitment region. Finally, associations observed in this study are correlational, and do not necessarily imply a causal relationship between emotion differentiation and binge eating phenotypes.

In summary, this is the first study to examine associations between multiple measures of emotion differentiation and multiple binge eating phenotypes in a large, population-based sample of women over several weeks in daily life. Overall, evidence is strongest for an association between low PED and clinically significant objective binge eating, as consistent associations were observed across two measures of emotion differentiation that have been used in prior research after controlling for affect intensity. People who tend to experience mixed negative emotions rather than one primary negative emotion may also be more likely to experience binge eating and emotional eating. Additional studies are needed to further elucidate the exact constructs captured by different emotion differentiation measures and their relevance for binge eating.

APPENDIX

Table 2.1. *Descriptive statistics*

Variable	Mean	*SD*	Range
NED-ICC	.30	.28	-.54–1.15
PED-ICC	.13	.31	-.84–1.04
NED-AC	.80	.11	.26–1.05
PED-AC	.72	.14	.15–1.01
NED-VAR	.53	.41	.004–3.29
PED-VAR	.78	.40	.04–3.26
DEBQ Emotional Eating	1.31	.40	1–4.02
Negative affect intensity	15.12	3.91	10.04–32.09
Positive affect intensity	22.89	6.32	10.39–39.90
BMI	23.71	5.47	15.30–47.59

Note: NED-ICC = negative emotion differentiation calculated using the intraclass correlation coefficient; PED-ICC = positive emotion differentiation calculated using the intraclass correlation coefficient; NED-AC = negative emotion differentiation calculated using the average interitem correlation; PED-AC = positive emotion differentiation calculated using the average interitem correlation; NED-VAR = negative emotion differentiation calculated using the average daily variance of emotion ratings; PED-VAR = positive emotion differentiation calculated using the average daily variance of emotion ratings; DEBQ Emotional Eating = average Dutch Eating Behavior Questionnaire Emotional Eating subscale score; BMI = body mass index.

Table 2.2. *Pearson correlations between emotion differentiation variables, affect intensity, emotional eating, and BMI*

	1	2	3	4	5	6	7	8	9	10
1. NED-ICC	—									
2. PED-ICC	.31***	—								
3. NED-AC	.83***	.24***	—							
4. PED-AC	.23***	.89***	.23***	—						
5. NED-VAR	-.14**	.02	-.08	.11*	—					
6. PED-VAR	-.01	.26***	.05	.19***	.41***	—				
7. Negative affect	-.31***	-.02	-.23***	.11*	.82***[a]	.26***	—			
8. Positive affect	-.06	-.12**	-.02	-.10*	.17***	.31***	.26***	—		
9. Emotional eating	-.17***	-.08	-.09*	-.01	.28***	-.03	.46***	.11*	—	
10. BMI	-.06	-.09*	-.06	-.07	-.04	-.002	-.04	-.08	.01	—

Note: NED-ICC = negative emotion differentiation calculated using the intraclass correlation coefficient; PED-ICC = positive emotion differentiation calculated using the intraclass correlation coefficient; NED-AC = negative emotion differentiation calculated using the average interitem correlation; PED-AC = positive emotion differentiation calculated using the average interitem correlation; NED-VAR = negative emotion differentiation calculated using the average daily variance of emotion ratings; PED-VAR = positive emotion differentiation calculated using the average daily variance of emotion ratings; negative and positive affect = average intensity of negative or positive affect across the study; emotional eating = average Dutch Eating Behavior Questionnaire Emotional Eating subscale score; BMI = body mass index. * p <.05, ** p <.01, *** p <.001.

[a]Note that the high correlation between NED-VAR and negative affect intensity likely reflects the fact that most participants had low to moderate negative affect intensity (as would be expected for a population-based sample), so that individuals who had greater variability in negative emotion ratings (e.g., rated some emotions "1" and other emotions "5") also tended to have higher negative affect intensity than individuals who had lower variability in negative emotion ratings (e.g., may have rated all negative emotions a "1").

Table 2.3. *Associations between binge eating phenotypes and negative emotion differentiation*

Method 1 – Intraclass Correlation Coefficient								
	DEBQ Emotional Eating				**Lifetime OBEs**			
Variable	β	SE	*p*	95% CI	OR	SE	*p*	95% CI
Intercept	.0001	.05	.998	-.10, .10	.03	.02	<.001***	.01, .09
NED	-.13	.04	.002**	-.22, -.05	.54	.13	.009**	.34, .86
BMI	.01	.05	.775	-.08, .11	1.43	.29	.079	.96, 2.14
	DEBQ Emotional Eating				**Lifetime OBEs**			
Variable	β	SE	*p*	95% CI	OR	SE	*p*	95% CI
Intercept	-.001	.04	.985	-.09, .09	.03	.02	<.001***	.01, .08
NED	-.02	.04	.665	-.10, .06	.64	.15	.051	.41, 1.002
Negative affect	.44	.04	<.001***	.35, .53	2.10	.46	.001**	1.37, 3.24
Positive affect	-.02	.04	.689	-.10, .07	.54	.15	.022*	.32, .91
BMI	.03	.04	.479	-.05, .12	1.44	.28	.061	.98, 2.10
Method 2 – Average Interitem Correlation								
	DEBQ Emotional Eating				**Lifetime OBEs**			
Variable	β	SE	*p*	95% CI	OR	SE	*p*	95% CI
Intercept	.0004	.05	.994	-.10, .10	.03	.02	<.001***	.01, .09
NED	-.06	.04	.205	-.14, .03	.65	.13	.036*	.43, .97
BMI	.02	.05	.697	-.08, .12	1.46	.30	.070	.97, 2.20
	DEBQ Emotional Eating				**Lifetime OBEs**			
Variable	β	SE	*p*	95% CI	OR	SE	*p*	95% CI
Intercept	-.002	.04	.969	-.09, .09	.03	.02	<.001***	.01, .09
NED	.03	.04	.519	-.05, .11	.77	.15	.184	.52, 1.13
Negative affect	.45	.04	<.001***	.37, .54	2.16	.47	<.001***	1.41, 3.32
Positive affect	-.02	.04	.639	-.10, .06	.57	.15	.032*	.34, .95
BMI	.03	.04	.438	-.05, .12	1.47	.29	.049*	1.002, 2.15
Method 3 – Average Daily Variance								
	DEBQ Emotional Eating				**Lifetime OBEs**			
Variable	β	SE	*p*	95% CI	OR	SE	*p*	95% CI
Intercept	-.001	.05	.985	-.10, .10	.03	.02	<.001***	.01, .09
NED	.25	.04	<.001***	.17, .34	1.27	.26	.258	.84, 1.91
BMI	.03	.05	.546	-.06, .12	1.52	.32	.046*	1.01, 2.29
	DEBQ Emotional Eating				**Lifetime OBEs**			
Variable	β	SE	*p*	95% CI	OR	SE	*p*	95% CI
Intercept	-.001	.04	.990	-.09, .08	.03	.02	<.001***	.01, .08
NED	-.31	.07	<.001***	-.45, -.17	.39	.16	.020*	.18, .86
Negative affect	.71	.07	<.001***	.57, .85	4.52	1.61	<.001***	2.25, 9.09

Table 2.3. (cont.)

Positive affect	-.03	.04	.442	-.11, .05	.48	.14	.012*	.28, .85
BMI	.03	.04	.497	-.05, .11	1.46	.28	.051	1.00, 2.14

Note: Models controlling for BMI only are presented first, followed by models controlling for BMI and positive/negative affect intensity. NED = negative emotion differentiation; DEBQ Emotional Eating = average Dutch Eating Behavior Questionnaire Emotional Eating subscale score; BMI = body mass index. * p <.05, ** p <.01, *** p <.001.

Table 2.4. *Associations between binge eating phenotypes and positive emotion differentiation*

Method 1 – Intraclass Correlation Coefficient								
	DEBQ Emotional Eating				**Lifetime OBEs**			
Variable	β	SE	*p*	95% CI	OR	SE	*p*	95% CI
Intercept	-.0001	.05	.998	-.10, .10	.02	.02	<.001***	.01, .08
PED	-.06	.04	.166	-.15, .03	.58	.15	.030*	.35, .95
BMI	.02	.05	.740	-.08, .11	1.46	.32	.079	.96, 2.24
	DEBQ Emotional Eating				**Lifetime OBEs**			
Variable	β	SE	*p*	95% CI	OR	SE	*p*	95% CI
Intercept	-.001	.04	.984	-.09, .09	.02	.01	<.001***	.01, .08
PED	-.07	.04	.109	-.15, .01	.52	.13	.008**	.32, .84
Negative affect	.45	.04	<.001***	.36, .53	2.53	.59	<.001***	1.60, 3.99
Positive affect	-.03	.04	.550	-.11, .06	.48	.13	.009**	.28, .83
BMI	.03	.04	.559	-.06, .11	1.45	.29	.060	.98, 2.14
Method 2 – Average Interitem Correlation								
	DEBQ Emotional Eating				**Lifetime OBEs**			
Variable	β	SE	*p*	95% CI	OR	SE	*p*	95% CI
Intercept	-.0001	.05	.999	-.10, .10	.02	.02	<.001***	.01, .09
PED	.003	.04	.937	-.08, .09	.76	.17	.237	.49, 1.19
BMI	.02	.05	.649	-.08, .12	1.50	.33	.063	.98, 2.29
	DEBQ Emotional Eating				**Lifetime OBEs**			
Variable	β	SE	*p*	95% CI	OR	SE	*p*	95% CI
Intercept	-.001	.04	.984	-.09, .09	.03	.01	<.001***	.01, .08
PED	-.05	.04	.228	-.13, .03	.64	.14	.045*	.41, .99
Negative affect	.45	.04	<.001***	.37, .54	2.57	.61	<.001***	1.62, 4.08
Positive affect	-.02	.04	.576	-.11, .06	.51	.14	.014*	.30, .87
BMI	.03	.04	.509	-.06, .11	1.50	.30	.041*	1.02, 2.23
Method 3 – Average Daily Variance								
	DEBQ Emotional Eating				**Lifetime OBEs**			
Variable	β	SE	*p*	95% CI	OR	SE	*p*	95% CI
Intercept	-.0001	.05	.999	-.10, .10	.03	.02	<.001***	.01, .09
PED	-.02	.04	.659	-.11, .07	.82	.20	.425	.50, 1.33
BMI	.02	.05	.646	-.08, .12	1.49	.31	.053	.99, 2.23
	DEBQ Emotional Eating				**Lifetime OBEs**			
Variable	β	SE	*p*	95% CI	OR	SE	*p*	95% CI
Intercept	-.001	.04	.986	-.09, .08	.03	.02	<.001***	.01, .09
PED	-.15	.04	<.001***	-.23, -.07	.67	.18	.139	.40, 1.14
Negative affect	.48	.04	<.001***	.39, .56	2.40	.51	<.001***	1.59, 3.63
Positive affect	.02	.04	.593	-.06, .11	.63	.16	.078	.38, 1.05

Table 2.4. (cont.)

BMI	.04	.04	.385	-.05, .12	1.47	.27	.036*	1.02, 2.12

Note: Models controlling for BMI only are presented first, followed by models controlling for BMI and positive/negative affect intensity. PED = positive emotion differentiation; DEBQ Emotional Eating = average Dutch Eating Behavior Questionnaire Emotional Eating subscale score; BMI = body mass index. * p <.05, ** p <.01, *** p <.001.

Table 2.5. *Associations between binge eating phenotypes and emotion differentiation measures, with negative and positive emotion differentiation included in the same model*

Method 1 – Intraclass Correlation Coefficient								
	DEBQ Emotional Eating				**Lifetime OBEs**			
Variable	β	SE	*p*	95% CI	OR	SE	*p*	95% CI
Intercept	-.001	.04	.984	-.09, .09	.03	.01	<.001***	.01, .08
NED	.003	.04	.947	-.08, .09	.76	.18	.243	.47, 1.21
PED	-.07	.04	.123	-.15, .02	.57	.14	.026*	.35, .94
Negative affect	.45	.04	<.001***	.36, .54	2.35	.55	<.001***	1.48, 3.73
Positive affect	-.03	.04	.548	-.11, .06	.48	.13	.009**	.28, .84
BMI	.03	.04	.557	-.06, .11	1.42	.28	.073	.97, 2.08
Method 2 – Average Interitem Correlation								
	DEBQ Emotional Eating				**Lifetime OBEs**			
Variable	β	SE	*p*	95% CI	OR	SE	*p*	95% CI
Intercept	-.002	.04	.966	-.09, .09	.03	.02	<.001***	.01, .08
NED	.04	.04	.313	-.04, .13	.85	.18	.456	.56, 1.30
PED	-.06	.04	.149	-.14, .02	.67	.16	.088	.43, 1.06
Negative affect	.47	.04	<.001***	.38, .55	2.43	.59	<.001***	1.51, 3.91
Positive affect	-.03	.04	.498	-.11, .06	.52	.14	.018*	.30, .89
BMI	.03	.04	.481	-.05, .12	1.48	.29	.047*	1.005, 2.19
Method 3 – Average Daily Variance								
	DEBQ Emotional Eating				**Lifetime OBEs**			
Variable	β	SE	*p*	95% CI	OR	SE	*p*	95% CI
Intercept	-.001	.04	.990	-.08, .08	.03	.02	<.001***	.01, .09
NED	-.25	.08	.001**	-.40, -.10	.40	.19	.048*	.16, .99
PED	-.10	.05	.032*	-.19, -.01	.95	.30	.875	.51, 1.76
Negative affect	.68	.07	<.001***	.54, .83	4.42	1.68	<.001***	2.10, 9.31
Positive affect	-.003	.04	.937	-.09, .08	.50	.16	.030*	.26, .93
BMI	.03	.04	.440	-.05, .11	1.46	.28	.050	1.00, 2.14

Note: NED = negative emotion differentiation; PED = positive emotion differentiation; DEBQ Emotional Eating = average Dutch Eating Behavior Questionnaire Emotional Eating subscale score; BMI = body mass index. * p <.05, ** p <.01, *** p <.001.

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