EXPLORING THE ROLE OF NEGATIVE URGENCY IN THE ETIOLOGY OF BINGE EATING: GENETIC/ENVIRONMENTAL ASSOCIATIONS AND INTERACTIONS WITH OVARIAN HORMONES

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ABSTRACT

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Impulsivity has emerged as a critical personality trait contributing to individual differences in the development of binge eating, and research suggests that negative urgency (i.e., the tendency to engage in rash action in response to negative affect) is a particularly important form of impulsivity for these behaviors. However, studies investigating the extent to which genetic and/or environmental influences underlie the effects of negative urgency on binge eating are lacking. Moreover, it remains unclear whether associations between negative urgency and binge eating are simply due to the well-established role of negative affect in the development/maintenance of binge eating. Study 1 addressed these gaps by examining phenotypic and etiologic associations between trait levels of negative urgency, negative affect, and binge eating in a sample of 444 same-sex female twins from the Michigan State Twin Registry. Negative urgency was significantly associated with two well-validated measures of binge eating tendencies, even after controlling for the effects of negative affect. Genetic factors accounted for the majority (62-77%) of this phenotypic association, although a significant proportion of this genetic covariation was due to genetic influences in common with negative affect. Non-shared environmental factors accounted for a relatively smaller (23-38%) proportion of the association between negative urgency and binge eating, but these non-shared environmental effects were independent of negative affect. Findings suggest that emotion-based rash action, combined with high levels of negative affect, may increase risk for binge eating, and that this likely occurs through both genetic and environmental mechanisms.

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Study 2 considered whether, in addition to having main effects, negative urgency might interact with other well-established risk factors for binge eating. Within-person changes in estradiol and progesterone predict changes in binge eating tendencies across the menstrual cycle. However, all women have menstrual-cycle fluctuations in hormones, but few experience binge eating. Personality traits, such as negative urgency, may be critical individual difference factors that influence who will engage in emotional eating in the presence of a vulnerable hormonal environment. Self-reports of emotional eating and saliva samples for hormone measurement were collected for 45 consecutive days in adolescent and young adult females (N=239). Negative urgency and negative emotionality were measured once and were examined as moderators of hormone-emotional eating associations. Consistent with prior research, within-person changes in the interaction between estradiol and progesterone predicted emotional eating symptom changes. However, negative urgency and negative emotionality did not interact with changes in estradiol, progesterone, or the estradiol-progesterone interaction to predict changes in emotional eating across the menstrual cycle. Future research should consider additional factors, other than the two personality traits examined, that may account for individual differences in within-person associations between hormones and emotional eating.

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TABLE OF CONTENTS

LIST OF TABLES viii

LIST OF FIGURES ix

GENERAL INTRODUCTION 1

STUDY 1: EXPLORING THE RELATIONSHIP BETWEEN NEGATIVE URGENCY AND BINGE EATING: ETIOLOGIC ASSOCIATIONS AND THE ROLE OF NEGATIVE AFFECT 4

- **Methods** 8
  - **Participants** 8
  - **Measures** 9
    - **Zygosity determination** 9
    - **Binge eating** 10
    - **Negative urgency** 11
    - **Negative affect** 11
  - **Statistical Analyses** 11
    - **Phenotypic analyses** 12
    - **Etiologic analyses** 12
      - ***Twin correlations*** 12
      - ***Biometric model fitting*** 13
      - ***Model fit and selection*** 16
- **Results** 17
  - **Phenotypic Analyses** 17
  - **Etiologic Analyses** 17
    - **Twin correlations** 17
    - **Biometric model fitting** 18
- **Discussion** 20

STUDY 2: INDIVIDUAL DIFFERENCES IN THE RELATIONSHIP BETWEEN OVARIAN HORMONES AND EMOTIONAL EATING ACROSS THE MENSTRUAL CYCLE: A ROLE FOR PERSONALITY? 27

- **Methods** 31
  - **Participants** 31
  - **Procedures** 32
  - **Measures** 33
    - **Emotional eating** 33
    - **Negative urgency** 34

Negative emotionality 34
Ovarian hormones 35
Covariates 35
Negative affect 36
Body mass index 36
Statistical Analyses 36
Data preparation 36
Statistical models 37
Results 38
Discussion 39

GENERAL DISCUSSION 44

APPENDICES 47
APPENDIX A 48
APPENDIX B 56

REFERENCES 61

LIST OF TABLES

Table A1. Descriptive Statistics and Pearson Correlations 49

Table A2. Predictive Associations among Negative Urgency, Negative Affect, and Binge Eating 50

Table A3. Twin Correlations for Negative Urgency, Negative Affect, and Binge Eating 51

Table A4. Parameter Estimates and Test Statistics for the Comparison of Trivariate Cholesky Decomposition Models 52

Table A5. Genetic and Environmental Correlations and Proportions of Covariance Accounted for by Genetic and Environmental Factors 54

Table B1. The Moderating Effects of Personality on Within-Person Associations between Ovarian Hormones and Emotional Eating 55

LIST OF FIGURES

Figure A1. Path diagram for the trivariate Cholesky decomposition model 57

Figure A2. Standardized variance estimates for the additive genetic and non-shared environmental contributions to the variance within and covariance among negative affect, negative urgency, and MEBS Binge Eating 58

Figure A3. Standardized variance estimates for the additive genetic and non-shared environmental contributions to the variance within and covariance among negative affect, negative urgency, and DEBQ Emotional Eating 59

Figure B1. Interactions between estradiol and progesterone in the prediction of emotional eating in the i) negative urgency model; and ii) negative emotionality model 60

GENERAL INTRODUCTION

Eating disorders are serious psychiatric disorders that affect a considerable number of adolescent and adult women during their lifetime (Hay, Mond, Buttner, & Darby, 2008; Hudson, Hiripi, Pope, & Kessler, 2007; Machado, Machado, Gonçalves, & Hoek, 2007) and are associated with significant psychiatric and medical morbidity and mortality (Agras, 2001). One core symptom of eating disorders that cuts across virtually all diagnostic categories (with the exception of anorexia nervosa- restrictor subtype) is binge eating. Binge eating is defined as consuming an amount of food over a discrete period of time that is definitely larger than most people would consume under similar circumstances (American Psychiatric Association (APA), 2000). A sense of loss of control is also necessary for a binge episode. Importantly, subclinical and clinical forms of binge eating are prevalent in adolescent and adult community populations (prevalence rates: 4.5-18%) (Gauvin, Steiger, & Brodeur, 2009; Hay, 2003; Hudson et al., 2007). Moreover, individuals who binge eat report poorer physical and mental health and an overall reduced quality of life compared to individuals free of binge eating (Gauvin et al., 2009; Hay, 2003). For the above reasons, it is critical to understand the etiology of binge.

The current project used a set of studies to further our understanding of individual differences in risk for binge eating. Both biological and psychological risk factors for binge eating will be examined in these studies, as calls in the eating disorder field have been put forth to explore risk factors at multiple levels of analysis (Bulik, 2005; Striegel-Moore & Bulik, 2007). Building more complex etiological models can help identify women who are most at risk for the development of binge eating and may ultimately inform eating disorder prevention and intervention efforts.

The first study considered the personality trait of negative urgency as a factor predicting individual differences in risk for binge eating. Negative urgency is defined as the tendency to

engage in impulsive behavior in response to negative affect. Of the different impulsive traits identified in recent factor analytic studies of the impulsivity construct (Whiteside & Lynam, 2001), negative urgency has consistently emerged as the strongest predictor of binge eating behaviors (Fischer, Smith, & Cyders, 2008). The current study was interested in investigating etiologic factors (i.e., genetic, environmental) that underlie the robust phenotypic relationship between negative urgency and binge eating using a twin study design. In addition, given the well-established role of negative affect in binge eating, the extent to which negative affect accounts for phenotypic and etiologic associations between negative urgency and binge eating was examined. This study was the first to consider the influence of common genetic/environmental factors on relationships between negative urgency and binge eating, and results may have significant implications for understanding the personality contribution to binge eating risk.

The second study aimed to build on the first by investigating whether, in addition to having main effects, negative urgency interacts with other known risk factors for binge eating. Specifically, I examined the interaction between levels of negative urgency and a well-established set of biological risk factors for binge eating, changes in ovarian hormones across the menstrual cycle (Edler, Lipson, & Keel, 2007; Klump, Keel, Culbert, & Edler, 2008; Klump et al., in press). All normally cycling women experience menstrual-cycle fluctuations in ovarian hormones, but very few develop binge eating. Therefore, individual differences in within-person associations between changes in ovarian hormones and changes in binge eating across the menstrual cycle clearly exist. Examining such differences may help predict which women develop binge eating and which women do not. Given that negative urgency is important for binge eating, levels of negative urgency may help explain for whom changes in ovarian

hormones are most likely to lead to binge eating. Identifying women most at risk for binge eating during certain menstrual cycle phases can help determine who will benefit most from particular prevention and treatment planning strategies (i.e., tracking menstrual cycle phase to identify vulnerable hormonal periods, increasing the use of coping skills during these times).

Taken together, this set of studies examined the role of *between-subjects* differences in negative urgency as a predictor of binge eating as well as a moderator of *within-individual* associations between ovarian hormone fluctuations and binge eating across the menstrual cycle. Investigating broad mechanisms accounting for negative urgency-binge eating associations as well as interactions between negative urgency and other risk factors will serve to expand our understanding of the complex etiology of binge eating. Negative urgency is a critical individual difference factor that influences the development of binge eating; however, limited data exist that speak to exactly *how* negative urgency may be etiologically related to binge eating. Understanding the precise role of an impulsive temperament for binge eating can help advance treatments that target the tendency to act on impulse in an effort to reduce the considerable cost and suffering associated with binge eating.

STUDY 1: EXPLORING THE RELATIONSHIP BETWEEN NEGATIVE URGENCY AND BINGE EATING: ETIOLOGIC ASSOCIATIONS AND THE ROLE OF NEGATIVE AFFECT[1]

Personality traits are crucial to etiologic models of eating disorders (Lilenfeld, Wonderlich, Riso, Crosby, & Mitchell, 2006), helping to explain why some individuals develop eating disorder symptoms and others do not. Impulsivity is perhaps the most important trait to consider for binge eating and associated eating disorders. Although most individuals with eating disorders are high on negative emotionality/neuroticism, an impulsive temperament tends to differentiate patients with binge/purge behaviors from those with restrictive eating disorders (Claes, Vandereycken, & Vertommen, 2005; Rosval et al., 2006). In addition, conditions characterized by impulsivity (e.g., substance/alcohol dependence, borderline personality disorder) are often comorbid with binge/purge eating disorders (Fischer et al., 2008). Impulsivity and binge eating symptoms are positively associated in community samples of women (Fischer, Smith, & Anderson, 2003; Racine, Culbert, Larson, & Klump, 2009) and, perhaps most importantly, impulsivity appears to be a prospective risk factor for the development of bulimic symptoms (Bodell, Joiner, & Ialongo, 2012; Mikami, Hinshaw, Patterson, & Lee, 2008; Wonderlich, Connolly, & Stice, 2004).

Unfortunately, research on the role of impulsivity in binge eating has been limited by the fact that impulsivity is a broad umbrella term encompassing multiple constructs (e.g., lack of

[1]

planning, sensation seeking, lack of perseverance, affect-driven impulsivity) (Whiteside & Lynam, 2001). Pinpointing the impulsive personality trait(s) that confer greatest risk for specific phenotypes, such as binge eating, is important when considering the context and function that the impulsive behavior may serve. For example, determining whether individuals binge eat due to a need for stimulation, to distract from negative affect, or because they simply do not consider the long-term consequences of their behavior, can be important for the development of etiologic models and treatment approaches aiming to reduce binge eating.

Research using self-report measures that assess distinct impulsivity constructs has begun to accumulate. These studies convincingly suggest that negative urgency (i.e., the tendency to act rashly in response to negative affect) is the most relevant form of impulsivity for binge eating. When examined in concert with other specific impulsive traits (e.g., lack of planning, lack of perseverance, sensation seeking), negative urgency has consistently emerged as the best predictor of binge eating symptoms (Anestis, Selby, Fink, & Joiner, 2007; Anestis, Smith, Fink, & Joiner, 2009; Claes et al., 2005; Fischer & Smith, 2008). Moreover, a recent meta-analysis classified studies investigating impulsivity-bulimic symptom associations based on the type of impulsive trait examined (i.e., negative urgency, lack of planning, lack of perseverance, sensation seeking). Findings pointed to negative urgency as most important for binge eating (i.e., effect size for negative urgency = .38; effect sizes for other impulsive traits = .08-.16; Fischer et al., 2008). Individuals who tend to respond to negative affect with rash action may be at increased risk for binge eating because they may use binge eating as an attempt to regulate negative emotions (Fischer et al., 2008).

Importantly, studies thus far have only focused on phenotypic associations between negative urgency and binge eating; thus, very little is known regarding etiologic factors that

underlie negative urgency-binge eating relationships. At the level of broad mechanisms, common genetic/biological factors and/or common environmental contexts might explain the robust phenotypic association between negative urgency and binge eating. For example, it may be that the genes that predispose someone to have higher levels of negative urgency also lead to binge eating. Alternatively, certain environmental experiences (e.g., child abuse/trauma; Brodsky et al., 2001) may influence the development of an impulsive temperament, which could subsequently increase risk for binge eating (Wonderlich et al., 2001). Findings such as these could help advance etiologic models of binge eating development and ultimately inform targeted prevention and intervention programs that explicitly aim to avert risk processes.

Twin studies are especially useful for providing an initial indication of the relative contribution of genetic and environmental factors to the relationship between two variables, as they decompose the covariance into genetic and environmental components. Notably, no study to date has investigated genetic and environmental covariance between negative urgency and binge eating, and in fact, studies have not yet identified whether genetic and/or environmental factors underlie relationships between any impulsive personality trait and binge eating. Twin studies have, however, examined etiologic associations between other relevant personality traits and binge eating. Findings suggest that genetic and non-shared environmental influences contribute approximately equally to phenotypic relationships between binge eating and the traits of negative emotionality (i.e., the tendency to experience negative affect) (Klump, McGue, & Iacono, 2002) and emotional dysregulation (i.e., unstable affective responding) (Livesley, Jang, & Thordarson, 2004). Given these findings, negative urgency-binge eating associations may similarly be influenced by both genetic and non-shared environmental common factors.

One important consideration for both phenotypic and etiologic studies examining the relationship between negative urgency and binge eating is the potential role of negative affect. As a reminder, negative urgency integrates the experience of negative affect with the tendency to engage in rash action, and negative affect has been identified as a strong, proximal trigger for binge eating (Haedt-Matt & Keel, 2011). Thus, individuals high on negative urgency may be prone to binge eating simply because they frequently experience high levels of negative affect. Similarly, any significant genetic/environmental overlap between urgency and binge eating may be completely accounted for by etiologic influences on trait levels of negative affect. To my knowledge, only one study has examined the independent predictive power of negative urgency and negative affect for binge eating; this study reported that negative urgency significantly predicted binge eating over and above the effects of negative affect (Anestis et al., 2009). I am not aware of any twin studies examining etiologic overlap between negative affect and binge eating; however, I might expect significant genetic/environmental associations for these constructs based on twin study findings for negative emotionality and binge eating (see above). In sum, research at both the phenotypic and etiologic levels is needed to determine whether negative urgency is uniquely associated with binge eating, distinct from general elevations on negative affect. Findings can help shed light on the nature of the personality trait of negative urgency, more generally, as well as its specific contribution to binge eating risk.

Given the above, the aim of the current study was to investigate phenotypic and etiologic associations between trait levels of negative urgency, negative affect, and binge eating in a sample of same-sex female twins. I sought to replicate associations between negative urgency and binge eating as well as to extend previous findings by demonstrating that these relationships were present over and above the effects of negative affect. Next, I used a twin design to

investigate the extent to which negative urgency-binge eating relationships were due to common genetic and/or environmental factors and to determine what proportion of the etiologic overlap among these constructs was accounted for by genetic and environmental influences in common with negative affect.

I focused on two dimensional measures of binge eating tendencies given that the prevalence of binge episodes would be expected to be too low in our community sample for formal twin analyses. Specifically, I examined: 1) thoughts and behaviors related to binge eating using the Minnesota Eating Behaviors Survey (MEBS) Binge Eating subscale, and 2) the tendency to eat in response to negative emotions (e.g., loneliness, disappointment) using the Dutch Eating Behaviors Questionnaire (DEBQ) Emotional Eating scale. Several previous studies investigating negative urgency-binge eating associations have used the Eating Disorders Inventory Bulimia Scale, which is very similar to MEBS Binge Eating. Thus, I was able to replicate results and investigate etiologic associations using a binge eating measure previously examined in the literature. In addition, this is the first study to investigate associations between negative urgency and emotional eating, a symptom that is defined by a tendency to act in response to negative emotions. Emotional eating is an associated feature of binge eating, but it also a form of dysregulated eating that occurs more frequently in non-clinical populations. Taken together, findings may help to more broadly understand the role of negative urgency in binge eating risk.

Methods

Participants

Participants included 444 same-sex female twins (222 twin pairs; 246 monozygotic (MZ) twins; 198 dizygotic (DZ) twins) between the ages of 16 and 25 years (mean age = 18.45 years, SD = 2.18) from the Michigan State University Twin Registry (MSUTR; Klump & Burt, 2006). MSUTR twins are recruited using birth record methods previously described (Klump & Burt, 2006). Data from previous studies (Culbert, Breedlove, Burt, & Klump, 2008) and the current study indicate that MSUTR participants are demographically representative of the recruitment region (81.1% Caucasian; 15.8% African American; 1.8% Asian/Pacific Islander; 1.4% Native American; http://www.michigan.gov/cgi/0,1607,7-158-54534---,00.html).

Data for the current project are drawn from the ***Twin Study of Hormones and Behavior across the Menstrual Cycle*** (Klump et al., in press). The parent study consists of daily data collection across 45 consecutive days as well as three in-person assessment sessions at the beginning, middle, and end of the 45-day period. With regards to measures for the current study, one of the binge eating measures (i.e., Minnesota Eating Behaviors Survey; see below) and the negative urgency measure were administered on one occasion only (i.e., study intake session), whereas the second binge eating measure (i.e., emotional eating) and negative affect were assessed daily for 45 days. Given that I was interested in relationships between trait levels of negative urgency, negative affect, and binge eating, I averaged levels of emotional eating and negative affect over the 45 days.

Because the parent study focused on hormones, a number of inclusion criteria (e.g., no psychotropic or steroid medication use; no pregnancy or lactation) were necessary to capture natural hormonal variation. Importantly, comparisons between our participants and those from previous MSUTR studies without these restrictions indicated very small differences on measures of negative affect, general impulsivity, and binge eating (average d = .11, range = .01-.20),

suggesting that our participants are representative of the larger population of twins on these constructs.

Measures

Zygosity determination. Similar to other large-scale twin registries (e.g., Kendler, Heath, Neale, Kessler, & Eaves, 1992), a physical similarity questionnaire was used as the primary determinant of zygosity. This questionnaire has previously demonstrated over 95% accuracy when compared to genotyping (Lykken, Bouchard, McGue, & Tellegen, 1990). Twins, twins' guardians (for 16-17 year-old twins), and research assistants completed this questionnaire, yielding up to 9 independent ratings of physical similarity. Discrepancies were resolved by having the principal investigator (KLK) review all questionnaire responses and examine twin photographs. In addition, DNA was available for 79% of the sample and was used to validate uncertain zygosities.

Binge eating. The Minnesota Eating Behaviors Survey Binge Eating scale (MEBS; von Ranson, Klump, Iacono, & McGue, 2005)[2] measures general levels of binge eating, including contemplating binge eating (e.g., "I think a lot about overeating (eating a really large amount of food)") and engaging in binge eating behaviors (e.g. "Sometimes I eat lots and lots of food and feel like I can't stop"), via seven true/false items. Internal consistency for this subscale is adequate in the current sample (α = .71) and previous young adult samples (von Ranson et al., 2005). In addition, criterion validity has been established, as women with bulimia nervosa score

[2] The Minnesota Eating Behavior Survey (MEBS; previously known as the Minnesota Eating Disorder Inventory (M-EDI)) was adapted and reproduced by special permission of Psychological Assessment Resources, Inc., 16204 North Florida Avenue, Lutz, Florida 33549, from the Eating Disorder Inventory (collectively, EDI and EDI-2) by Garner, Olmstead, & Polivy (1983) Copyright 1983 by Psychological Assessment Resources, Inc. Further reproduction of the MEBS is prohibited without prior permission from Psychological Assessment Resources, Inc.

higher on the MEBS Binge Eating scale than unaffected control women (von Ranson et al., 2005).

The Dutch Eating Behavior Questionnaire Emotional Eating scale (DEBQ; van Strien, Frijters, Bergers, & Defares, 1986) consists of thirteen items that assess the tendency to eat in response to negative affective cues (e.g., "Did you have a desire to eat when you were discouraged?"). Items are rated on a 5-point scale from not at all to very often. Internal consistency for the DEBQ Emotional Eating scale is excellent in previous research (van Strien et al., 1986) and in the current study (α = .90). The DEBQ Emotional Eating Scale correlates with established measures of binge eating (e.g., *r*'s = .55-.69) (Racine et al., 2009; van Strien, 2000) as well as with palatable food intake (i.e., ice cream) in the laboratory (van Strien, 2000). Moreover, scores on this scale distinguish between individuals who have bulimia nervosa/binge eating, overweight individuals, and college students (Wardle, 1987).

Negative Urgency. The (Negative) Urgency, (lack of) Premeditation, (lack of) Perseverance, Sensation Seeking-Positive Urgency (UPPS-P) Impulsive Behavior Scale (Lynam, Smith, Whiteside, & Cyders, 2006) was used to assess negative urgency. The Negative Urgency scale consists of 12 items that are rated on a 4-point scale from agree strongly to disagree strongly. Internal consistency for the Negative Urgency scale was high in the current study (α = .85) and previous work (Fischer & Smith, 2008).

Negative affect. The Negative Affect scale from the Positive and Negative Affect Schedule (PANAS; Watson, Clark, & Tellegen, 1988) consists of 10 items that assess the full spectrum of daily negative emotions (e.g., fear, distress, irritability, nervousness). The degree to which each emotion was experienced was rated on a 5-point scale ranging from very slightly/not at all to extremely. This scale exhibits excellent internal consistency as well as good convergent

and discriminant validity (Watson et al., 1988), and internal consistency was excellent in the current study (α = .85).

Statistical Analyses

MEBS Binge Eating and PANAS Negative Affect scores were log-transformed prior to analyses to account for positive skew. An arctan transformation was used for DEBQ Emotional Eating given the significant leptokurtic distribution of this measure. Negative urgency was normally distributed and was not transformed.

Phenotypic analyses. Within-person, Pearson correlations were used to examine initial phenotypic associations between negative urgency, negative affect, and binge eating. Hierarchical linear models (HLM; also known as mixed linear models) were then fit to the data to examine relationships between negative urgency and binge eating while controlling for negative affect and the dyadic nature of the twin data. The non-independent data structure was accounted for by nesting a level 1 variable (individual twin) within a level 2 unit (twin pair).

Two HLMs were conducted to examine the phenotypic association between negative urgency and binge eating as well as the potential influence of negative affect on this relationship. Model 1 examined the "simple" main effect of negative urgency on binge eating. Model 2 included both negative urgency and negative affect as predictors of binge eating in order to determine whether negative urgency influences binge eating over and above any effects of negative affect. This approach has been recommended by Simmons and colleagues to directly observe the effects that covariates have on statistical results (Simmons, Nelson, & Simonsohn, 2011). Given that HLM provides unstandardized estimates of effects, I standardized all variables in order to compare effect sizes across models.

Etiologic analyses. Twin correlations and biometric model fitting were used to examine whether genetic and/or environmental influences contribute to the variance in negative urgency, negative affect, and binge eating as well as the covariation among these phenotypes.

Twin correlations. Intraclass correlations were first calculated separately for negative urgency, negative affect, and binge eating measures by zygosity (MZ vs. DZ) in order to provide an initial indication of the relative influence of genetic and environmental factors on each phenotype. Next, cross-twin cross-trait correlations (e.g., correlation between Twin 1's level of negative urgency and Twin 2's level of binge eating) were calculated to determine the extent to which phenotypic associations between negative urgency, negative affect, and binge eating are accounted for by common genes and/or common environmental factors. For both sets of correlations, additive genetic factors (A; genetic influences that add across genes) are suggested if the MZ twin correlation is approximately twice the DZ twin correlation. Non-additive genetic effects (D; interaction of genetic effects at same locus) are implied if MZ twin correlations are more than double DZ twin correlations. Shared environmental effects (C; factors that make members of a twin pair similar to one another) are inferred if MZ and DZ twin correlations are approximately equal. Finally, non-shared environmental effects (E; factors that make members of a twin pair different from one another) (and measurement error) are implied if the MZ twin correlation is less than 1.0 (for intraclass correlations) or less than the corresponding phenotypic correlation (for cross-twin cross-trait correlations).

Biometric model fitting. Trivariate, Cholesky decomposition models were used to examine the extent to which additive genetic, non-additive genetic, shared environmental, and/or non-shared environmental influences accounted for relationships among negative urgency, negative affect, and binge eating. Although independent pathway and common pathway models

are often also fitted when modeling three variables, I only examined a Cholesky model in the current study given that there were an a priori set of "directional" hypotheses (see below).

Figure 1a presents the trivariate Cholesky model with genetic, shared environmental, and non-shared environmental effects. Non-additive genetic effects are not presented in the figure, given that non-additive genetic and shared environmental effects cannot be estimated in the same model when examining only MZ and DZ twins reared together (Neale & Cardon, 1992). As shown in the figure, the trivariate model provides information regarding the magnitude of the genetic and environmental influences on each phenotype and the extent to which these influences contribute to the covariation between phenotypes. Although the ordering of the variables (i.e., first, second, third) does not affect how well the model fits the observed data, the ordering is critical for the parameter estimates that are produced. A primary aim of the current study was to determine whether genetic/environmental effects on negative urgency account for a significant proportion of the genetic/environmental influences on binge eating. In addition, I wanted to determine the extent to which etiologic influences in common with negative affect accounted for the genetic/environmental covariation between negative urgency and binge eating. Therefore, I ordered the variables in the following way: 1) negative affect, 2) negative urgency, 3) binge eating (see Figure 1). Ordering the variables in this way allowed for the variance in binge eating to be decomposed into: 1) genetic/environmental effects attributable to negative affect (a31, c31, e31; see Figure 1); 2) genetic/environmental effects attributable to negative urgency but not shared with negative affect (a32, c32, e32); and 3) residual genetic/environmental effects specific to binge eating (a33, c33, e33). The variance in negative urgency is decomposed into genetic/environmental influences overlapping with negative affect (a21, c21, e21) and those

specific to negative urgency (a22, c22, e22), whereas there is no decomposition of genetic and environmental effects on negative affect (a11, c11, e11).

Path estimates from the trivariate model can be used to produce two additional sets of indices that quantify the degree of covariation among the phenotypes: 1) genetic/environmental correlations, and 2) proportions of covariance accounted for by genetic/environmental factors. Pathways that index the genetic/environmental covariation among phenotypes can be standardized on their respective variances to produce genetic and environmental correlations. Whereas the attributable path estimates index the proportion of total variance in a phenotype (e.g., binge eating) that is accounted for by genetic/environmental influences on a second phenotype (e.g., negative urgency), genetic/environmental correlations describe the degree to which the genetic/environmental influences on negative urgency are the same as those on binge eating. Correlations are often presented in multivariate twin studies, as they range from -1 to 1 and provide an easily interpretable estimate of the genetic/environmental overlap between two phenotypes. Because they are free from measurement error, it is possible to have genetic and shared environmental correlations of 1.0 (see Kendler, Neale, Kessler, Heath, & Eaves, 1992 for an example). A genetic correlation of 1.0 would indicate, for example, that the genetic influences on one phenotype are identical to the genetic influences on a second phenotype. In contrast, a genetic correlation of 0 would suggest that genetic influences on a set of phenotypes are completely distinct. Unlike the attributable genetic and environmental estimates described above, these correlations index the degree of genetic/environmental covariation between each pair of phenotypes without removing variance associated with the other phenotype(s) in the model. Therefore, I can evaluate genetic and environmental overlap between negative urgency and binge eating without accounting for negative affect using these correlations.

The phenotypic correlations between negative affect, negative urgency, and binge eating also can be decomposed into the proportion of the association that is due to genetic factors versus environmental factors. These estimates are different from the genetic/environmental correlations in that they provide information about the relative importance of genetic and environmental factors to the *relationship* between two traits. Genetic and environmental correlations, on the other hand, index the degree to which etiologic influences on one trait correlate with those on another trait. For example, a genetic correlation could be very large, but if the heritability estimates for the two traits are low, shared genetic influences are unlikely to substantially contribute to the covariation between the traits.

Model fit and selection. Model fitting was conducted using full-information maximum likelihood raw data techniques in MX statistical software (Neale, Boker, Xie, & Maes, 2003). Raw data techniques treat missing data as missing-at-random (Little & Rubin, 1987) and allow for the retention of twin pairs in which one twin in a pair has missing data. A full ACE model and a full ADE model were both examined, based on the pattern of twin correlations (see Results). This allowed me to determine whether shared environmental or non-additive genetic parameters were more important for inclusion in the models. Nested sub-models were also fit and were compared to these full models (i.e., AE and CE models compared to ACE model; AE model compared to ADE model).[3]

Model fit comparisons were made by taking the difference in minus twice the log-likelihood (-2lnL) between the full models and the nested sub-models. Under certain regularity conditions, this comparison results in a chi-square difference test, with the degrees of freedom

[3] DE models are infrequently examined in behavior genetics studies given that the presence of non-additive genetic effects in the absence of additive genetic effects is theoretically unlikely (McGue & Christensen, 1997). Thus, DE models were not run in the current study.

(df) for this test representing the difference between the df for the full and nested models. Statistically significant chi-square values lead to the rejection of the nested model in favor of the full model. Aikake's Information Criterion (AIC; $\chi 2 - 2df$) was also used as an index of model fit. AIC measures model fit relative to model parsimony, and AIC is lowest/more negative in the best-fitting models.

Results

Phenotypic Analyses

Descriptive statistics and Pearson correlations are presented in Table 1a. The correlation between MEBS Binge Eating and DEBQ Emotional Eating was lower than expected based on previous research in community samples (*r*'s = .55-.69) (Racine et al., 2009; van Strien, 2000), but still in the moderate range (*r* = .34). Negative urgency was positively associated with both measures of binge eating (*r*'s = .26-.46), as was negative affect (*r*'s = .24-.49). Finally, the correlation between negative affect and negative urgency was moderate (*r* = .34), indicating that these are overlapping, yet distinct, constructs.

HLM results are presented in Table 2a. Negative urgency was significantly associated with both binge eating measures when only negative urgency was included in the model. Importantly, negative urgency continued to significantly predict both MEBS Binge Eating and emotional eating after including negative affect in Model 2. Thus, it appears that negative urgency is significantly associated with binge eating above and beyond the effects of negative affect. Notably, negative affect was not associated with MEBS Binge Eating in Model 2, but negative affect was a stronger predictor of DEBQ Emotional Eating than negative urgency.

Etiologic Analyses

Twin correlations. Twin intraclass and cross-twin cross-trait correlations are presented in Table 3a. Higher MZ than DZ twin correlations, and MZ twin correlations less than 1.0, indicate the presence of genetic and non-shared environmental influences, respectively, on all constructs. In addition, dominant genetic effects may be important for negative urgency and MEBS Binge Eating, given that MZ twin correlations were more than double DZ twin correlations. Finally, shared environmental factors appear to be relevant for emotional eating, as the MZ twin correlation was less than double the DZ twin correlation.

Regarding cross-twin cross-trait correlations, higher MZ than DZ correlations indicate that genetic factors likely contribute to the covariation between negative affect, negative urgency, and binge eating measures. This pattern was particularly pronounced for the association between negative urgency and MEBS Binge Eating, as MZ-DZ twin correlations were significantly different from each other for this relationship (see Table 3a). Differences between the MZ and DZ twin correlations were more modest for negative affect -MEBS Binge Eating and negative affect-emotional eating relationships, suggesting the presence of both genetic and shared environmental influences. Finally, non-shared environmental effects are implicated in the covariation of all pairs of phenotypes, given MZ cross-twin cross-trait correlations less than the corresponding phenotypic correlations (see Table 1a).

Biometric model fitting. Trivariate model fit statistics and parameter estimates for the full and nested models (i.e., ACE, ADE, AE, CE) are presented in Table 4a. Parameter estimates from the full ACE and ADE models suggested that, in general, additive genetic effects and non-shared environmental effects are most important for the phenotypes examined. These sources of variance made significant contributions to negative urgency, negative affect, and both binge eating measures, whereas shared environmental effects and non-additive genetic parameters were

non-significant across models. Confirming these impressions, model-fit comparisons indicated that the best-fitting model for all phenotypes was the AE model. The AE models did not fit significantly worse than the ACE or ADE models, according to the chi-square difference tests, and they also produced the lowest AIC values (see Table 4a).

Genetic/environmental correlations and the proportions of variance accounted for by genetic/environmental factors are presented in Table 5a. Genetic correlations between negative urgency and binge eating were large and significant for both MEBS Binge Eating (r_g = .77 (CIs: .54, .99)) and emotional eating (r_g = .52 (CIs: .25, .79)). The non-shared environmental correlation was significant between negative urgency and MEBS Binge Eating (r_e = .29 (CIs: .13, .43)) but not between negative urgency and emotional eating (r_e = .11 (CIs: -.05, .26)). The majority of the phenotypic covariation between negative urgency and binge eating measures was accounted for by genetic influences (62-77%, see Table 5a), with non-shared environmental factors contributed relatively less to these phenotypic relationships (23-38%). Taken together, genetic factors impacting negative urgency and binge eating are relatively similar, and genetic influences primarily underlie phenotypic relationships between negative urgency and binge eating.

As previously stated, genetic and non-shared environmental correlations do not take into account whether etiologic overlap is independent of negative affect, For this question, I refer to the standardized path estimates (presented in Figures 2a and 3a) which are squared to obtain estimates of attributable and unique variance (discussed in the text). As shown in Figures 2a and 3a, genetic overlap with negative affect is important to consider given that genetic influences in common with negative affect significantly contribute to the variance in negative urgency (i.e.,

14% of 35% total heritability), MEBS Binge Eating (i.e., 12% of 39% total heritability), and emotional eating (i.e., 31% of 44% total heritability). This etiologic overlap is likely to decrease the contribution of genetic/environmental influences unique to negative urgency to the variance in binge eating.

Indeed, path estimates from negative urgency to binge eating (see Figures 2a and 3a) indicate that the genetic covariance between negative urgency and binge eating is reduced after controlling for genetic influences in common with negative affect. Genetic influences unique to negative urgency significantly contributed to the total variance in MEBS Binge Eating (i.e., 12%), whereas there was virtually no unique contribution of negative urgency to emotional eating, after accounting for genetic factors that also influence negative affect. Although there was significant non-shared environmental covariance between negative urgency and MEBS Binge Eating that was completely independent of non-shared environmental factors on negative affect, these non-shared environmental influences only contributed 5% to the total variance in MEBS Binge Eating. Finally, non-shared environmental influences unique to negative urgency contributed very minimally to the total variance in emotional eating (i.e., 1%). However, non-shared environmental rather than genetic factors likely account for the phenotypic relationship between urgency and emotional eating that is independent of negative affect.

Despite significant etiologic overlap among negative affect, negative urgency, and binge eating, residual genetic/environmental variance on binge eating measures (i.e., that which is not accounted for by negative affect and negative urgency) was notable. Between 30% and 40% of the genetic variance on MEBS Binge Eating (i.e., 15% of 39% total heritability) and emotional eating (i.e., 14% of 44% total heritability) was unique, and greater than 90% (i.e., 56% of 61%

for MEBS Binge Eating; 54% of 56% for emotional eating) of non-shared environmental influences were specific to binge eating.

Discussion

This study was the first to go beyond investigating phenotypic associations between negative urgency and binge eating by examining etiologic factors that may underlie this relationship. Consistent with previous research, negative urgency was significantly associated with two separate measures of binge eating. Twin model results indicated that genetic and, to a lesser extent, non-shared environmental factors account for phenotypic relationships between negative urgency and binge eating. Moreover, the genetic factors that influence negative urgency are highly correlated with the genetic factors that influence binge eating. Taken together, findings from the current study suggest that the personality trait of negative urgency may increase risk for the development of binge eating through both genetic and environmental mechanisms.

This study was also interested in investigating the role of negative affect, a well-established risk factor for binge eating, in explaining phenotypic and etiologic relationships between negative urgency and binge eating. At a phenotypic level, negative urgency predicted both binge eating constructs over and above the effects of negative affect, indicating that phenotypic relationships between negative urgency and binge eating cannot be accounted for by negative affect. However, genetic influences on negative affect significantly contributed to the variance in negative urgency and binge eating. After controlling for genetic influences in common with negative affect, genetic factors unique to negative urgency only accounted for 0-12% of the total variance in binge eating. Therefore, genetic influences shared with negative

affect appear to contribute much of the common variance between negative urgency and binge eating.

Notably this pattern of findings does not negate the importance of the construct of negative urgency for the etiology of binge eating. Specifically, because the rash action of individuals high on negative urgency is conditional on the presence of momentary increases in negative affect, it might be expected that, after accounting for trait levels of negative affect, the remaining genetic variance in binge eating attributable to negative urgency is small. In essence, by including both negative affect and negative urgency in the same model, I may have partialled out key parts of the negative urgency construct, both generally and in relation to binge eating. Importantly, however, this provided a very strong test of our hypothesis regarding the specific role of negative urgency in binge eating. Thus, it is impressive that genetic influences unique to negative urgency significantly contributed to the variance in MEBS Binge Eating (but not emotional eating). Morever, negative affect and negative urgency together accounted for a substantial proportion of the genetic variance in binge eating. For example, of the genetic influences on MEBS Binge Eating (i.e., 39%), 12% were due to genetic influences in common with negative affect, 12% were due to genetic influences unique to negative urgency, and 15% were unique to MEBS Binge Eating. Therefore, negative urgency and negative affect, together, accounted for approximately 60% of the genetic variance in binge eating (i.e., 24% of 39% total heritability). This percent of explained genetic variance is as high or higher than what is accounted for by other risk factors for binge eating such as negative emotionality, alcohol use, and weight/shape concerns (Klump et al., 2002; Munn et al., 2010; Slane, Burt, & Klump, in press). Thus, results point to negative urgency as a significant correlate for the genetic diathesis of binge eating and suggest that individuals most at risk for binge eating may be those who

experience high levels of negative affect and who have a tendency towards emotion-based rash action.

Although negative affect and negative urgency accounted for a significant proportion of the genetic variance in binge eating, residual genetic variance was notable. Moreover, the majority of non-shared environmental influences were specific to MEBS Binge Eating and DEBQ Emotional Eating. Findings from previous research indeed suggest that, in addition to personality traits and negative affect, other psychological (e.g., dietary restraint, alcohol use) (Racine, Burt, Iacono, McGue, & Klump, 2011; Slane et al., in press), psychosocial (e.g., peer/family influences), and biological (e.g., ovarian hormones) (Klump et al., in press; Klump, Keel, Sisk, & Burt, 2010) factors appear to influence the development of binge eating. Although this study and others by our group (Klump et al., in press; Slane et al., in press) have largely focused on the main effects of these risk factors, it is likely that interactions between personality, psychological, psychosocial, and biological risk factors are relevant for the development of binge eating and may explain a larger percentage of variance than main effects alone. For example, it may be that individuals high on negative urgency are more likely to develop binge eating (versus another kind of impulsive behavior) if they are exposed to a specific trigger for eating pathology, such as attempts to restrict food intake for weight loss (Racine et al., 2011), a vulnerable hormonal milieu (Klump et al., in press), etc. Additional research is needed to elucidate these types of complex interactions and develop a more in-depth understanding of binge eating and its risk factors.

Notably, although results were generally similar for MEBS Binge Eating and DEBQ Emotional Eating, some differences emerged. In both phenotypic and etiologic analyses, negative urgency was more strongly related to MEBS Binge Eating, and negative affect was a

stronger predictor of emotional eating. Differences in the constructs represented by the two binge eating measures could be responsible for these discrepant results. Indeed, the correlation (r = .34) between these two measures supports the idea that these are related, yet distinct, constructs, and their differential relationships with negative affect and negative urgency make intuitive sense when thinking about the nature of the underlying constructs. Specifically, emotional eating directly assesses eating in response to negative affective cues, whereas items on the MEBS Binge Eating scale focus mainly on behavioral indicators of impulsive, binge eating tendencies (e.g., eating a large amount of food at once, loss of control over eating). Alternatively, differential associations may be due to measurement issues since negative urgency and MEBS Binge Eating were both assessed one time during study intake, whereas negative affect and emotional eating were assessed daily (and then averaged). Thus, stronger associations between negative urgency and MEBS Binge Eating, and negative affect and emotional eating, may reflect similarities in the measurement window rather than true differential associations. To indirectly examine this possibility, I conducted post-hoc analyses investigating associations between another study variable that was assessed daily and during the intake session (i.e., MEBS Weight Preoccupation; intake and daily scores: r = .80) and both negative urgency and negative affect. Results indicated modest-to-no differences in the magnitude of phenotypic associations (i.e., negative urgency and MEBS Weight Preoccupation scores at intake: r = .30 vs. negative urgency and daily MEBS Weight Preoccupation scores: r = .25; negative affect and MEBS Weight Preoccupation scores at intake and daily: r = .27). These findings suggest that different measurement windows are unlikely to account entirely for our differing phenotypic and etiologic associations. Nonetheless, future studies should replicate these results using multiple binge eating measures administered across the same time frame in order to understand

similarities/differences in phenotypic and etiologic associations among negative urgency, negative affect, and binge eating constructs.

Although results from the current study enhance our understanding of negative urgency-binge eating relationships, several additional limitations must be noted. First, the sample size was relatively small for a multivariate twin study, resulting in broad confidence intervals for some parameters and lower power to detect non-additive genetic and shared environmental effects. However, findings regarding significant etiologic overlap among negative affect, negative urgency, and binge eating are likely robust, as they were replicated across two measure of binge eating tendencies. Even so, additional research in larger twin samples is needed to confirm these results.

Second, I investigated binge eating in a non-clinical sample of women rather than in a clinical sample of eating disorder patients. However, research suggests that heritability estimates are very similar across clinical and community samples and that there is substantial genetic overlap for binge eating and bulimia nervosa (Bulik, Sullivan, & Kendler, 1998; Wade, Bulik, Sullivan, Neale, & Kendler, 2000). Although it is likely that findings would generalize to patients with eating disorders, future studies should directly investigate this possibility. Third, research questions were examined using two self-report measures of binge eating tendencies. Self-report measures have been criticized for overestimating the frequency of binge eating compared to interviews (Fairburn & Beglin, 1994). However, interview-based measures have also been shown to underestimate the heritability of various forms of psychopathology (Burt, 2009). Thus, there may be advantages of using both interview and self-report measures of binge eating when conducting twin studies, but further research is needed to directly compare results.

Finally, data from the current study cannot speak to causal associations and the direction of phenotypic, genetic, and environmental relationships between negative urgency and binge eating. Data suggest that impulsivity increases risk for the later development of binge eating symptoms (Bodell et al., 2012; Mikami et al., 2008; Wonderlich et al., 2004), but this has yet to be examined for negative urgency. Additional research therefore is needed to examine longitudinal relationships between negative affect, negative urgency and binge eating to confirm that negative urgency is a *prospective* genetic and/or environmental risk factor for binge eating.

STUDY 2: INDIVIDUAL DIFFERENCES IN THE REALTIONSHIP BETWEEN OVARIAN HORMONES AND EMOTIONAL EATING ACROSS THE MENSTRUAL CYCLE: A ROLE FOR PERSONALITY?[4]

Ovarian hormones are one important set of biological factors involved in the etiology of binge eating and eating disorders (Edler et al., 2007; Hildebrandt, Alfano, Tricamo, & Pfaff, 2010; Klump et al., 2008; Klump et al., in press; Racine et al., 2012). Initial evidence for these associations came from experimental animal research demonstrating that ovarian hormones regulate food intake in a variety of species. Removal of the source of ovarian hormones through bilateral ovariectomy causes increased food intake and adminstration of estradiol reverses this effect (Asarian & Geary, 2006; Kemnitz, Gibber, Lindsay, & Eisele, 1989; Tarttelin & Gorski, 1973; Varma et al., 1999). In contrast, progesterone causes increased food intake, in part, by anatagonizing the inhibitory effects of estradiol (Blaustein & Wade, 1976; Czaja, 1978; Kemnitz et al., 1989). Several recent studies suggest that ovarian hormone effects extend to models of binge eating in animals. For example, ovariectomy has been associated with increased palatable food intake (Klump et al., 2011), and the administration of a high estrogen/low progesterone treatment decreases high-fat food intake (Yu, Geary, & Corwin, 2008). In sum, animal research has been critical in demonstrating that estradiol and progesterone have direct, causal effects on food intake and most likely binge eating.

Research is also converging to suggest that ovarian hormones influence food intake and binge eating phenotypes in humans. Longitudinal studies across the menstrual cycle find that

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[4] Adapted with permission from Elsevier. The official citation that should be used in referencing this material is: Racine, S. E., Keel, P. K., Burt, S. A., Sisk, C. L., Neale, M., Boker, S., & Klump, K. L. (2013). Individual differences in the relationship between ovarian hormones and emotional eating across the menstrual cycle: A role for personality? *Eating Behaviors*, *14,* 161-166. doi: 10.1016/j.eatbeh.2013.02.007.

food intake/binge eating is highest during menstrual cycle phases characterized by high progesterone (i.e., mid-luteal phase) and lowest during phases described by high estradiol (i.e., ovulatory phase) (Barr, Janelle, & Prior, 1995; Buffenstein, Poppitt, McDevitt, & Prentice, 1995; Edler et al., 2007; Klump et al., 2008; Lester, Keel, & Lipson, 2003). Moreover, studies that have directly assayed hormone levels suggest that within-person changes in hormones drive menstrual-cycle fluctuations in binge eating, as would be predicted by animal data. Pilot studies that examined binge eating in women with bulimia nervosa (BN) and emotional eating (i.e., tendency to eat excessive amounts of food in response to negative affective cues) in women from the community suggested that these behaviors were predicted by decreases in estradiol and increases in progesterone (Edler et al., 2007; Klump et al., 2008). However, recent research with the largest sample to date has found that the effects of ovarian hormones on emotional eating symptoms are interactive, such that levels of emotional eating are highest when progesterone and estradiol are high (Klump et al., in press). This is consistent with the observation that the mid-luteal phase, a time of high progesterone and relatively high estradiol, is associated with the greatest rates of binge eating. Importantly, hormonal changes dictated by the reproductive axis appear to drive changes in dysregulated eating rather than binge eating causing hormonal changes. Supporting this point, Klump et al. (in press) did not find that changes in emotional eating predicted same-day or next-day changes in ovarian hormones levels.

The convergence of findings from experimental animal research and longitudinal studies across the menstrual cycle in women strongly suggests that changes in ovarian hormones predict binge eating. However, all normally cycling women experience changes in estradiol and progesterone across the menstrual cycle, but relatively few engage in dysregulated eating behaviors. Individual differences in within-person relationships between ovarian hormones and

binge eating/emotional eating clearly exist, and there is a need to examine factors that influence patterns of dysregulated eating across the menstrual cycle in some but not all women. Understanding individual differences can help identify women who are most vulnerable to binge eating/emotional eating during certain menstrual cycle phases, and this information can potentially be used to develop targeted prevention and intervention efforts for disordered eating symptoms.

Personality traits may represent one set of risk factors that is important for determining which individuals binge eat in response to a vulnerable hormonal environment. Personality traits are key etiologic factors for eating disorders that help to explain why some individuals develop eating disorder symptoms and others do not (Cassin & von Ranson, 2005; Lilenfeld et al., 2006). Impulsivity is perhaps the most important personality trait for binge eating and associated eating disorders. Unlike other personality traits (e.g., negative emotionality), impulsivity appears to be specifically related to binge eating versus other types of disordered eating behaviors (e.g., body dissatisfaction, dietary restraint) (Lyke & Spinella, 2004; Steiger, Leung, & Houle, 1992; Yeomans, Leitch, & Mobini, 2008) and tends to differentiate patients with binge/purge behaviors versus restrictive eating disorders (Claes et al., 2005; Rosval et al., 2006). Perhaps most importantly, data from longitudinal studies suggest that impulsivity is a risk factor contributing to the development of binge eating (Bodell et al., 2012; Mikami et al., 2008; Wonderlich et al., 2004).

Recently, the multidimensional construct of impulsivity has been decomposed into distinct traits that have been shown to be differentially related to impulsive behaviors (Smith et al., 2007; Whiteside & Lynam, 2001). One particular type of impulsivity, negative urgency (i.e., the tendency to experience strong impulses, particularly in response to negative affect), appears

to be most important for binge eating and associated eating disorders. When examined together with other impulsive traits (e.g., lack of planning, lack of perseverance, sensation seeking), negative urgency consistently emerges as the best predictor of binge eating (Anestis, Selby, & Joiner, 2007; Anestis et al., 2009; Claes et al., 2005; Fischer & Smith, 2008). Moreover, a recent meta-analysis that examined impulsivity-bulimic symptom associations according to type of impulsive trait confirmed that negative urgency confers greatest risk (Fischer et al., 2008). In sum, negative urgency is an important individual difference factor influencing the development of binge eating.

To date, no study has investigated whether personality variables critical for binge eating might moderate within-person associations between ovarian hormones and binge eating phenotypes. The primary goal of the current study was to examine whether individual differences on the trait of negative urgency may help predict who will be most likely to engage in emotional eating in the presence of a vulnerable hormonal environment. Individuals high on negative urgency have a tendency to experience strong impulses and have trouble resisting acting on their impulses (Whiteside & Lynam, 2001). The biological drive to binge eat as a result of menstrual-cycle changes in ovarian hormones may represent a strong urge that is difficult for these individuals to resist. Thus, I hypothesized that ovarian hormones will be more likely to predict within-person changes in emotional eating in individuals with high versus low trait levels of negative urgency.

As a control, I investigated whether the personality trait of negative emotionality (i.e., the tendency to chronically experience high levels of negative affective states, such as anxiety, depression, anger, etc.) may similarly moderate hormone-binge eating associations. Both negative emotionality and negative urgency involve the experience of negative affect, whereas

negative urgency includes the additional component of rash action, and it is this rash action that is hypothesized to be particularly important for the occurrence of dysregulated eating in response to a risky hormonal milieu. Examining the moderating effects of negative emotionality may help to determine whether negative urgency exhibits specificity as a moderator of hormone-binge eating associations or whether effects are more general and present for other personality traits.

Finally, consistent with previous research (Edler et al., 2007; Klump et al., 2008; Klump et al., in press), I controlled for state levels of negative affect by including daily changes in negative affect as a covariate in all models. State levels of negative affect are strong proximal predictors of binge eating/emotional eating (Haedt-Matt et al., submitted; Smyth et al., 2007), and levels of negative affect are thought to vary across the menstrual cycle (Dennerstein & Burrows, 1979; Ivey & Bardwick, 1968). Thus, I wanted to ensure that the trait-level, personality characteristics moderated the direct effects of ovarian hormones on emotional eating, independent of state levels of negative affect.[5]

Methods

Participants

Participants included 239 same-sex female twins (132 monozygotic twins; 107 dizygotic twins) between the ages of 16 and 25 years (mean age = 18.09; SD = 1.74) drawn from the Twin Study of Hormones and Behavior Across the Menstrual Cycle (Klump et al., in press) within the Michigan State University Twin Registry (MSUTR). MSUTR twins are recruited using birth record methods previously described (Klump & Burt, 2006). Data from previous studies (Culbert

[5] In order to ensure that negative affect itself did not moderate hormone-emotional eating associations, we included it as a moderator instead of a covariate in analyses. All interactions with hormones were non-significant (data not shown) and thus, negative affect was included in the final models as a covariate only.

et al., 2008) and the current study indicate that MSUTR participants are demographically representative of the recruitment region (83.3% Caucasian; 15.1% African American; 0.8% Asian/Pacific Islander; 0.8% Native American; http://www.michigan.gov/mdch).

Given the study's focus on hormones, a number of inclusion criteria were necessary to capture natural hormonal variation: 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 medication use in the past 4 weeks; 3) no pregnancy of lactation in the past 6 months; and 4) no history of genetic or medical conditions that influence hormones or appetite/weight. Importantly, comparisons between our participants and those from previous MSUTR studies without these restrictions indicated very small differences on measures of general impulsivity, negative emotionality, and binge eating (average d = .11; $d's$ = .01-.20), suggesting that our participants are representative of the larger population of twins on these constructs.

Procedures

Participants provided saliva samples and behavioral data for 45 consecutive days. Saliva samples were collected every morning within 30 minutes of waking by passively drooling into a cryovial tube until at least 1.8 ml of saliva was produced. Participants were asked to refrain from brushing their teeth, eating, drinking or smoking prior to providing saliva samples. The time the sample was taken was recorded, and participants were asked to immediately place tubes in the freezer each morning. After saliva samples were received from participants, they were stored in a -80 degree C freezer until being shipped for analysis. Behavioral questionnaires were completed each evening (after 5:00 pm) using an on-line data collection system or pre-printed scantrons (whichever the participant preferred). The timing of data collection was such that ovarian hormone measurements clearly preceded behavioral ratings each day.

In addition, participants completed three in-person visits occurring at the start of the study, mid-way through data collection (~day 23), and at the end of data collection (~day 45). Each visit included a re-assessment of study eligibility, measurement of participant's height and weight, completion of self-report questionnaires, and collection of saliva samples. Between visits, staff called/emailed participants once per week to answer questions and confirm protocol adherence. These procedures were effective at identifying individuals who were no longer eligible to participate due to missed periods, medication use, and/or pregnancy during the study (2.4%). In addition, the percentage of participants who were dropped from the study or whose data were not analyzed due to failure to collect a sufficient number of samples was minimal (2.7% for each condition). Finally, the percentage of missing data for individuals included in the current study was very low for both hormone samples ($\leq$ 3%) and behavioral data ($\leq$ 6%).

Measures

Emotional eating. The Dutch Eating Behavior Questionnaire Emotional Eating scale (DEBQ; van Strien et al., 1986) was completed each day of the 45-day collection period. The thirteen items on this scale assess eating in response to negative affective cues (e.g., "Did you have a desire to eat when you were discouraged?") and are rated on a 5-point scale from not at all to very often. Similar to previous research (Klump et al., 2008; Racine et al., 2012), the instructions for this scale were modified with permission to ask about emotional eating over the current day. Internal consistencies for the unmodified and modified versions of the DEBQ Emotional Eating scale are excellent in previous research (α = .93 and α = .98, respectively) (Klump et al., 2008; van Strien et al., 1986) and in the current study (average α = .90).

This scale was chosen as a measure of binge eating tendencies as it has exhibited robust fluctuations across the menstrual cycle in previous studies of hormone-binge eating associations

(Klump et al., 2008). Importantly, eating in response to negative emotions is thought to be a core feature of binge eating, and this scale has demonstrated validity in distinguishing between individuals with bulimia nervosa/binge eating, overweight individuals, and college students. Further, the DEBQ Emotional Eating Scale correlates with established measures of binge eating (*r*'s = .55-.69) (Racine et al., 2009; van Strien, 2000) as well as with palatable food intake (i.e., ice cream) in the laboratory (van Strien, 2000).

Negative Urgency. The (Negative) Urgency, (lack of) Premeditation, (lack of) Perseverance, Sensation Seeking-Positive Urgency (UPPS-P) Impulsive Behavior Scale (Lynam et al., 2006) was administered one-time during the study intake session and was used to assess negative urgency. The Negative Urgency scale consists of 12 items that are rated on a 4-point scale from agree strongly to disagree strongly. Internal consistency for the Negative Urgency scale was high in the current study (α = .85) and previous work (Fischer & Smith, 2008).

Negative Emotionality. The Multidimensional Personality Questionnaire-Brief Form (MPQ-BF; Patrick, Curtin, & Tellegen, 2002) was administered one-time during the study intake session and was used to assess negative emotionality. The MPQ-BF consists of three higher-order personality factors, including negative emotionality, and 11 primary trait scales (Patrick et al., 2002). Negative emotionality scores are calculated using a weighted sum of primary trait scales, and the primary trait scales that load most heavily on Negative Emotionality include Stress Reaction, Alienation, and Aggression. Consistent with previous research (Patrick et al., 2002), internal consistency estimates for these primary trait scales ranged from acceptable to good in the current study (α's = .74-.80) In addition, the Negative Emotionality factor exhibits expected convergent and discriminant relationships with scales from other personality inventories (e.g., *r* with Big Five Neuroticism = .70) (Church, 1994). Negative emotionality from

the MPQ has been examined extensively as a personality correlate of eating disorder symptoms, including binge eating, in previous research (Klump et al., 2002; Pryor & Wiederman, 1996; Stein et al., 2002).

Ovarian hormones. Saliva samples were assayed for both estradiol and progesterone. Using saliva to examine hormone concentrations has distinct advantages over other biological fluids (e.g., serum; Shirtcliff et al., 2000). Saliva sampling is less invasive, especially when repeated samples are needed, and salivary hormone levels reflect unbound hormones that provide a more accurate estimate of active estradiol and progesterone. Previous research has reported that saliva sampling is associated with greater compliance and more robust hormone-behavior associations that blood spot sampling (Edler et al., 2007).

Saliva samples were analyzed by Salimetrics, LLC (State College, PA, USA) using high-sensitivity enzyme immunoassay techniques that have been specifically designed for saliva. The estradiol assay has a lower limit of sensitivity of 0.10 pg/ml and average intra- and inter-assay coefficients of variation of 7.1% and 7.5%, respectively. Method accuracy, determined by spike recovery and linearity, are 104.2% and 99.4% for estradiol. Estradiol values from matched serum and saliva samples are highly correlated for females (r >.80). The progesterone assay has a lower limit of sensitivity of 5 pg/ml, and average intra- and inter-assay coefficients of variation of 6.2% and 7.6%, respectively. Method accuracy, determined by spike recovery and linearity, are 99.6% and 91.8% for progesterone. Progesterone values from matched serum and saliva samples show a strong linear relationship for females (r > .87). In order to conserve resources, every second saliva sample was assayed during menstrual bleeding and the early follicular phase when hormone levels are expected to be low and stable. This procedure ensured that key periods

of hormonal variation (i.e., mid-follicular through pre-menstrual phases) were captured while maximizing the number of participants assessed.

Covariates.

Negative affect. Negative affect was included as a covariate in all analyses, given the need to examine moderation of hormone-emotional eating associations independent of any menstrual cycle fluctuations in negative affect. Negative affect was assessed daily across the 45-day study period using the PANAS Negative Affect scale (Watson et al., 1988). This scale consists of 10 items that assess the full spectrum of daily negative emotions (e.g., fear, distress, irritability, nervousness). The degree to which each emotion was experienced was rated on a 5-point scale ranging from very slightly/not at all to extremely. This scale exhibits excellent internal consistency as well as good convergent and discriminant validity (Watson et al., 1988), and internal consistency was excellent in the current study (average α = .85).

Body mass index (BMI). BMI was included as a covariate in analyses given its association with binge eating (Fitzgibbon et al., 1998; Picot & Lilenfeld, 2003; Stice, Presnell, & Spangler, 2002) as well as ovarian hormone levels (Ukkola et al., 2001; Yoo et al., 1998). Height and weight were measured at each of the three study visits using a wall-mounted ruler and digital scale, respectively. BMI was calculated using the following formula: (BMI = weight (in kilograms)/height(in meters) squared). All three measurements of BMI were used, such that the first BMI was entered from the first day of the study until the time the second BMI measurement was taken, the second BMI was entered from then until the second to last day of the study, and the final BMI measurement was entered on the final day of the study.

Statistical Analyses

Data preparation. Five-day rolling averages, standardized within person, were calculated for all daily measures (i.e., estradiol, progesterone, negative affect, emotional eating), as well as for BMI. All previous studies examining menstrual cycle changes in ovarian hormones and binge eating have used within-person standardized rolling average variables. Rolling averages are preferred because they minimize random variation in behavioral data due to environmental circumstances (Gladis & Walsh, 1987) and smooth the pattern of hormone variability (Kassam et al., 1996; Waller et al., 1998). Five day-rolling averages were calculated in the following way: the level of hormone/psychological symptom on any one day (e.g., day 5) was computed as the average level of the hormone/psychological symptom for the two days before, day of, and two days after (e.g., days 3 to 7 inclusive). Five-day rolling averages were then converted into within-person standardized scores based on each participant's overall mean and standard deviation across the data collection period. Standardizing variables in this way allowed me to examine the degree to which changes in a woman's ovarian hormones, relative to her equilibrium, predict changes away from the woman's equilibrium for emotional eating across the menstrual cycle. Negative urgency and negative emotionality were standardized based on the sample mean and standard deviation. Because these personality traits were only measured once, they represent individual difference variables that may influence the strength of within-person associations between hormones and emotional eating.

Statistical models. Hierarchical linear models (HLM; also known as mixed linear models or multilevel models) were used for the current project. HLMs were ideally suited for answering this research question as they are able to examine interactive effects of predictors (i.e., hormones and personality traits) while controlling for covariates as well as for the non-independence of the data. Non-independence existed in the data set in two different ways, as I examined a sample of

twins and 45 days worth of data was collected from each individual. Therefore, I allowed residual errors for emotional eating to correlate between members of a twin pair. I also estimated a time-specific dyadic correlation that allowed twin's residual errors to correlate from day-to-day. Each time-varying predictor was included as a random effect in order to model the random slopes and the relationship between these slopes within twin pairs. There was no evidence that random slopes were correlated across twins, so an identity covariance matrix, which estimated a single variance for both twins in a pair, was used to specify these random effects.

Given the substantial correlation between negative urgency and negative emotionality in our data set (r = .60), I ran two separate HLMs to examine each personality trait as an independent moderator of hormone-emotional eating associations. All models included the main effects of estradiol, progesterone, the respective personality trait, and covariates (i.e., negative affect, BMI). In addition, the two-way interaction between estradiol and progesterone was included, given that ovarian hormones have previously demonstrated interactive effects in predicting emotional eating (Klump et al., in press). The two-way estradiol-personality trait and progesterone-personality trait interactions were included in order to test whether changes in estradiol and/or progesterone may be more strongly associated with changes in emotional eating in individuals with high vs. low levels of negative urgency/negative emotionality. Finally, the three-way interaction between estradiol, progesterone, and each personality trait was tested in order to examine whether the joint effects of estradiol, progesterone, and each personality trait predicted changes in emotional eating, over and above main effects and two-way interactions.

Results

Results from HLMs examining negative urgency and negative emotionality as moderators of ovarian hormone-emotional eating relationships are presented in Table 1b. Within-person changes in estradiol and progesterone did not exhibit significant main effects on changes in emotional eating across the menstrual cycle. However, there were significant estradiol x progesterone interactions, such that the presence of high estradiol and high progesterone was associated with greater levels of emotional eating. This finding is consistent with a previous report by our group (Klump et al., in press) that examined a smaller subset (82%) of the sample included in the current study. Results from this study are novel, however, in suggesting that the interactive effects of ovarian hormones on emotional eating are independent of individual differences on negative urgency and negative emotionality. Indeed, the nature and magnitude of this interaction (see Figure 1b) was similar to what was observed in the prior analysis (Klump et al., in press).

As shown in Table 1b, the personality traits of negative urgency and negative emotionality did not significantly interact with changes in estradiol, progesterone, or the estradiol-progesterone interaction to predict changes in emotional eating across the menstrual cycle. Therefore, contrary to hypotheses, these personality traits were not significant moderators of within-person associations between ovarian hormones and emotional eating in our sample.

Discussion

The current study was the first to examine whether personality traits might moderate within-person associations between ovarian hormones (i.e., estradiol and progesterone) and emotional eating symptoms across the menstrual cycle in a community sample of women. I hypothesized that negative urgency might be a particularly important personality trait, given

robust associations between negative urgency and binge eating previously reported (Fischer et al., 2008). In addition, individuals high on negative urgency have trouble resisting strong impulses including, possibly, a biological drive towards dysregulated eating. However, results did not support the hypothesis that negative urgency influences the strength of associations between within-person changes in ovarian hormones and emotional eating. The personality trait of negative emotionality was also not a significant moderator of hormone-emotional eating relationships. Therefore, individual differences on emotion-based rash action and chronic negative affectivity do not appear to explain why some individuals engage in emotional eating across the menstrual cycle while others do not.

In order to inform future studies examining moderators of hormone-emotional eating associations, it is important to explore potential reasons for our lack of significant hormone-negative urgency interactions. First, controlling for daily state levels of negative affect may have impacted the ability to detect moderating effects of negative urgency on hormone-emotional eating relationships. Because the rash action of individuals high on negative urgency is thought to be dependent on the presence of momentary increases in negative affect, key parts of the negative urgency construct may have been partialled out by including within-person changes in negative affect as a covariate. However, I ran post-hoc HLMs that did not control for negative affect, and interactions between ovarian hormones and negative urgency remained non-significant (data not shown). Therefore, the inclusion of negative affect as a covariate is unlikely to have meaningfully impacted our results.

Second, changes in one's propensity for impulsive action across time may be more relevant for influencing the strength of hormone-emotional eating associations than stable individual differences on the personality trait of negative urgency. According to an interactional

perspective on personality psychology (Endler & Parker, 1992), the occurrence of rash action is dependent on both a person's personality predisposition towards impulsive behavior as well as situational variables, including environmental, social, and even biological circumstances. Indeed, research has demonstrated that impulsive behavior varies in response to alcohol intake (Dougherty, Marsh-Richard, Hatzis, Nouvion, & Mathias, 2008; Dougherty, Marsh, Moeller, Chokshi, & Rosen, 2000; Finn, Justus, Mazas, & Steinmetz, 1999), manipulations of the serotonin system (Fairbanks, Melega, Jorgensen, Kaplan, & McGuire, 2001; Walderhaug et al., 2002), and phase of the menstrual cycle (Howard, Gifford, & Lumsden, 1988; Pine & Fletcher, 2011). Although most research examining fluctuations in impulsivity has used laboratory-based behavioral tasks (e.g., Go/NoGo task), a self-report measure of state impulsivity has recently been developed (Iribarren, Jiménez-Giménez, García-de Cecilia, & Rubio-Valladolid, 2011) and could be incorporated into future daily diary and ecological momentary assessment studies of dysregulated eating behavior. These research designs could be used to explicitly test whether one's immediate inclination to engage in emotion-based rash action significantly influences the likelihood of emotional eating in response to a vulnerable hormonal milieu.

Future studies are also encouraged to consider additional binge eating risk variables that may moderate within-person hormone-emotional eating associations. In the only other study of this kind, Klump et al. (submitted) investigated whether variables related to body weight regulation (i.e., BMI, dietary restraint) might influence the strength of relationships between ovarian hormones and emotional eating symptoms across the menstrual cycle. However, these two indices of body weight regulation did not significantly moderate the effects of ovarian hormones on within-person changes in emotional eating. The one exception was the presence of a trend-level interaction between dietary restraint and estradiol that appeared to explain pre-

menstrual increases in binge eating/emotional eating in women with more severe eating pathology (e.g., women with BN) (Edler et al., 2007; Lester et al., 2003).

In addition to further exploring the potential influence of restraint on hormone-emotional eating associations, particularly in clinical samples (see Klump et al., submitted), several intriguing moderator possibilities remain untested. For example, individuals with a genetic predisposition for binge eating may be particularly vulnerable towards dysregulated eating during certain phases of the menstrual cycle (Klump et al., in press). Ovarian hormones are potent regulators of genes in various neurotransmitter systems, including systems involved in food intake/binge eating (e.g., serotonin) (Bethea, Lu, Gundlah, & Streicher, 2002; Östlund, Keller, & Hurd, 2003). Menstrual cycle changes in emotional eating may reflect the influence of hormones on gene transcription. However, these effects may only be prominent in individuals who possess the risk alleles of candidate genes for binge eating phenotypes, helping to account for individual differences in the strength of hormone-binge eating associations (Klump et al., in press).

The possibility that the genomic effects of ovarian hormones account for menstrual cycle changes in binge eating phenotypes is further supported by the fact that hormone-emotional eating associations are independent of several important risk factors for dysregulated eating. Previous studies have consistently shown that relationships between estradiol, progesterone and binge eating/emotional eating are independent of BMI and daily changes in negative affect (Edler et al., 2007; Klump et al., 2008). Results from the current study further suggest that hormone effects cannot be accounted for by the personality traits of negative urgency and negative emotionality. That is, the interaction between estradiol and progesterone continued to significantly predict menstrual-cycle changes in emotional eating after controlling for trait levels

of negative urgency and negative emotionality. Taken together, ovarian hormones appear to have direct effects on binge eating tendencies that are independent of several important psychological risk factors for binge eating. Findings from this study are thus informative for continuing to build mechanistic models regarding the precise role of ovarian hormones in binge eating development/maintenance.

Strengths of this study included daily hormone and behavioral data collection across 45 days in a large sample of adolescent and young adult females that allowed for investigation of potential moderator effects. Previous studies examining hormone-binge eating associations were limited by small sample sizes and could not test whether personality traits might help explain why some, but not all, women binge eat in response to a vulnerable hormonal environment. However, this question was examined in a non-clinical sample of women from the community using emotional eating as the dependent variable, and it is unclear whether results would generalize to clinical samples of women with binge eating and related eating disorders. Identifying whether personality traits might influence associations between ovarian hormones and binge eating in clinical samples of eating disorder patients is an interesting question for future research. In addition, most participants (ages 16-25 years) were not through the peak period of risk for eating disorders, which can extend up until age 25 (Lewinsohn, Striegel-Moore, & Seeley, 2000). Nonetheless, emotional eating is present as early as childhood (Blissett, Haycraft, & Farrow, 2010) and is predictive of later binge eating (Stice et al., 2002), suggesting that the sample is likely to include a number of "at risk" individuals.

GENERAL DISCUSSION

The current project aimed to integrate research examining biological and psychological risk factors for binge eating in order to develop a more comprehensive understanding of the complex etiology of these behaviors. The focus of this set of studies was on the personality trait of negative urgency (i.e., the tendency to engage in rash action in response to negative affect), one of several personality pathways to impulsive behavior (Whiteside & Lynam, 2001). Research has accumulated to suggest that negative urgency is the most critical impulsive personality trait for binge eating, as binge eating may occur more frequently in these individuals as an attempt to regulate negative emotions (Fischer et al., 2008). Given that research to date has only focused on demonstrating phenotypic links between negative urgency and binge eating, my hope was to provide a more nuanced understanding of the role of negative urgency in binge eating risk.

This set of studies examined the main effects of negative urgency on binge eating at both phenotypic and etiologic levels as well as interactions between negative urgency and a set of well-established biological risk factors for binge eating (i.e., ovarian hormones). Findings from Study 1 suggest that negative urgency is a robust predictor of individual differences in levels of binge eating and that the majority of this phenotypic association is accounted for by common genetic factors. Therefore, a genetic predisposition towards emotion-based rash action may increase risk for the development of binge eating symptoms and may potentially account for patterns of comorbidity between binge eating and other impulsive behaviors (e.g., alcohol/drug use, risky spending). Importantly, however, negative urgency is one of many risk factors for binge eating, and it is likely that negative urgency interacts with other specific triggers for eating pathology to influence binge eating symptoms. Study 2 considered whether individual

differences on negative urgency may influence the strength of within-person associations between changes in ovarian hormones and changes in emotional eating across the menstrual cycle. Results indicated that negative urgency did not significantly moderate hormone-emotional eating associations. Instead, the interaction between estradiol and progesterone was the strongest predictor of menstrual-cycle changes in emotional eating, independent of individual differences on negative urgency. Taken together with findings from previous studies (Klump et al., submitted; Klump et al., in press), these results suggest that ovarian hormones are important predictors of within-person changes in emotional eating across the menstrual cycle in women who exhibit the full range of variation on a number of binge eating risk variables.

One important consideration when interpreting the pattern of findings across this set of studies is the level of analysis: negative urgency was a significant predictor of *between-subjects* differences in binge eating in Study 1, but negative urgency did not moderate *within-subjects* associations between hormones and emotional eating in Study 2. This implies that negative urgency may be particularly important as an etiologic factor, influencing who will develop binge eating and who will not. Thus, findings from Study 1 have implications for existing risk models that consider negative urgency to be an important personality predictor for binge eating (Combs, Pearson, & Smith, 2011; Pearson, Combs, Zapolski, & Smith, in press). Our results suggest that negative urgency should be specified as a genetic predisposing factor that may interact with a variety of biological, psychological, and environmental circumstances to lead to binge eating.

In contrast, trait-levels of negative urgency do not appear to influence the likelihood that a woman will engage in emotional eating in response to a vulnerable hormonal milieu. Rather, it may be that *changes* in a woman's propensity towards impulsive behavior are more important than stable trait differences for influencing these binge eating trajectories. Results from Study 2

as well as previous studies examining menstrual-cycle changes in emotional eating have important implications for the treatment of individuals with binge eating. Specifically, hormonal changes across the menstrual cycle should be considered as triggers for symptom exacerbation regardless of one's personality tendencies towards emotion-based rash action. Incorporating psychoeducation about menstrual cycle-symptom relationships and working on developing coping skills for additional triggers (e.g., interpersonal difficulties) during vulnerable hormonal periods may enhance treatment effectiveness across women suffering from eating pathology. In sum, research that focuses on binge eating at both the between- and within-subjects levels will provide new insights regarding etiologic and maintenance factors and, potentially, how these factors can be targeted in interventions for binge eating symptoms.

APPENDICES

APPENDIX A

Tables A1-A5 and B1

Table A1

Descriptive Statistics and Pearson Correlations

Variables	Mean (S.D)	Range	MEBS Binge Eating	DEBQ Emotional Eating	Neg. urgency	Neg. affect
Binge eating variables						
MEBS Binge Eating	1.05 (1.43)	0-7	-	-	-	-
DEBQ Emotional Eating	0.30 (.38)	0-3	.34***	-	-	-
Predictor variables						
Neg. Urgency	2.03 (.55)	1-3.67	.46***	.26***	-	-
Neg. Affect	14.74 (3.56)	10-30	.24***	.49***	.34***	-

Note. MEBS = Minnesota Eating Behaviors Survey; DEBQ = Dutch Eating Behavior Questionnaire

*** $p < .001$

Table A2

Predictive Associations among Negative Urgency, Negative Affect, and Binge Eating

Model	MEBS Binge Eating			DEBQ Emotional Eating		
	b (S.E)	*t* (df)	*p*	b (S.E)	*t* (df)	*p*
Model 1						
Neg. Urgency	.45 (.04)	10.58 (429.42)	**<.001**	.23 (.04)	5.13 (418.66)	**<.001**
Model 2						
Neg. Urgency	.43 (.05)	9.55 (419.98)	**<.001**	.10 (.04)	2.51 (420.67)	**.01**
Neg. Affect	.07 (.05)	1.44 (394.40)	.15	.42 (.04)	9.37 (406.89)	**<.001**

Note. MEBS = Minnesota Eating Behaviors Survey; DEBQ = Dutch Eating Behavior Questionnaire

Table A3

Twin Correlations for Negative Urgency, Negative Affect, and Binge Eating

Variables	MZ twins (N = 237-244)	DZ twins (N = 186-196)	Z	*p*
Intraclass correlations				
Neg. Urgency	.39***	-.03	4.48	**<.001**
Neg. Affect	.55***	.24**	3.84	**<.001**
MEBS BE	.42***	.02	4.28	**<.001**
DEBQ EE	.41***	.28***	1.50	.07
Cross-twin cross-trait correlations				
Neg. Urgency-Neg. Affect	.25***	.07	1.89	**.03**
Neg. Urgency-MEBS BE	.27***	.02	2.64	**.004**
Neg. Urgency-DEBQ EE	.17**	.08	0.93	.18
Neg. Affect-MEBS BE	.21**	.14*	0.73	.23
Neg. Affect-DEBQ EE	.35***	.29***	0.67	.25

Note. MEBS = Minnesota Eating Behaviors Survey; DEBQ = Dutch Eating Behavior Questionnaire; BE = binge eating; EE = emotional eating; MZ = monozygotic; DZ = dizygotic. Z = Fisher r-to-z transformation test of equality. p value for one-tailed test examining whether the MZ correlation is larger than the DZ correlation.
*** $p < .001$; ** $p < .01$; * $p < .05$

Table A4

Parameter Estimates and Test Statistics for the Comparison of Trivariate Cholesky Decomposition Models

Model	Standardized Parameter Estimates			Test Statistics			
	a^2	c^2 /d^2	e^2	-2lnL (df)	$\chi 2$ (df)	*p*	AIC[a]
MEBS Binge Eating (BE)							
ACE				3478.83 (1290)	-	-	-7.05
Neg. Affect	.43 (.13, .64)	.11 (0, .37)	.46 (.35, .59)				
Neg. Urgency	.34 (.15, .48)	.01 (0, .16)	.65 (.51, .79)				
MEBS BE	.37 (.15, .52)	.02 (0, .18)	.61 (.47, .77)				
CE				3499.32 (1296)	20.49 (6)	.002	1.44
Neg. Affect	-	.42 (.31, .53)	.58 (.47, .69)				
Neg. Urgency	-	.21 (.08, .33)	.79 (.67, .92)				
BE	-	.23 (.10, .35)	.77 (.65, .90)				
AE				**3039.82 (1296)**	**.78 (6)**	**.99**	**-18.32**
Neg. Affect	.55 (.42, .65)	-	.45 (.35, .58)				
Neg. Urgency	.35 (.21, .49)	-	.65 (.51, .79)				
MEBS BE	.39 (.23, .53)	-	.61 (.47, .77)				
ADE				3473.42 (1290)	-	-	-12.46
Neg. Affect	.37 (0, .63)	.19 (0, .62)	.45 (.35, .57)				
Neg. Urgency	.01 (0, .38)	.38 (0, .52)	.61 (.49, .75)				
MEBS BE	.07 (0, .41)	.37 (0, .55)	.56 (.44, .72)				
AE				**3479.56 (1296)**	**6.14 (6)**	**.41**	**-18.32**
Neg. Affect	.55 (.42, .65)	-	.45 (.35, .58)				
Neg. Urgency	.35 (.21, .49)	-	.65 (.51, .79)				
MEBS BE	.39 (.23, .53)	-	.61 (.47, .77)				
DEBQ Emotional Eating (EE)							
ACE							
Neg. Affect	.42 (.12, .63)	.12 (0, .38)	.46 (.36, .59)	3445.74 (1287)	-	-	-23.21
Neg. Urgency	.34 (.12, .47)	0 (0, .17)	.66 (.53, .81)				
DEBQ EE	.26 (.01, .53)	.17 (0, .40)	.58 (.45, .72)				

Table A4 (cont'd)

CE				3461.15 (1293)	15.41 (6)	.02	-19.90
Neg. Affect	-	.42 (.30, .52)	.58 (.48, .69)				
Neg. Urgency	-	.21 (.09, .34)	.79 (.66, .91)				
DEBQ EE	-	.35 (.23, .46)	.65 (.54, .77)				
AE				**3447.25 (1293)**	**1.51 (6)**	**.96**	**-33.80**
Neg. Affect	.55 (.42, .65)	-	.46 (.35, .58)				
Neg. Urgency	.34 (.19, .47)	-	.66 (.53, .81)				
DEBQ EE	.44 (.30, .56)	-	56 (.44, .70)				
ADE				3443.25 (1287)	-	-	-25.80
Neg. Affect	.37 (.01, .62)	.19 (0, .57)	.44 (.35, .57)				
Neg. Urgency	.02 (0, .41)	.35 (0, .49)	.63 (.50, .78)				
DEBQ EE	.38 (.02, .55)	.07 (0, .45)	.55 (.43, .69)				
AE				**3447.25 (1293)**	**4.00 (6)**	**.68**	**-33.80**
Neg. Affect	.55 (.42, .65)	-	.46 (.35, .58)				
Neg. Urgency	.34 (.19, .47)	-	.66 (.53, .81)				
DEBQ EE	.44 (.30, .56)	-	56 (.44, .70)				

Note. MEBS = Minnesota Eating Behaviors Survey; DEBQ = Dutch Eating Behavior Questionnaire; -2lnL = -2 times log likelihood; df = degrees of freedom; AIC = Aikake Information Criteria. Best-fitting model is indicated by bold type. 95% confidence intervals for variance estimates are presented in parentheses.

[a] All AICs were calculated by taking the difference in -2lnL values between a baseline, unrestricted model (i.e., a model that freely estimates variances, covariances, and means) and all other models. -2lnL(df) for baseline trivariate MEBS Binge Eating model: 3419.88 (1257); -2lnL(df) for baseline trivariate DEBQ Emotional Eating model: 3403.05 (1254)

Table A5

Genetic and Environmental Correlations and Proportions of Covariance Accounted for by Genetic and Environmental Factors

	Correlations		Proportion of covariance accounted for by:	
Variables	A	E	A	E
Neg. Affect-Neg. Urgency	63 (.41, .85)	.12 (-.04., .28)	.81	.19
Neg. Affect-Binge Eating	.55 (.31, .81)	-.03 (-.19, .14)	1.00	0
Neg. Urgency-Binge Eating	.77 (.54, .99)	.29 (.13, .43)	.62	.38
Neg. Affect-Emotional Eating	.82 (.66, .99)	.19 (.03, .34)	.82	.18
Neg. Urgency-Emotional Eating	.52 (.25, .79)	.11 (-.05, .26)	.77	.23

Note. A = additive genetic effects; E = non-shared environmental effects. 95% confidence intervals for correlations presented in parentheses.

Table B1

The Moderating Effects of Personality on Within-person Associations between Ovarian Hormones and Emotional Eating

	b (SE)	*t*	df	*p*
Negative Urgency as a Moderator				
Intercept	-.04 (.01)	-3.57	223	**<.001**
Estradiol	0 (.02)	-0.01	226	.99
Progesterone	.03 (.02)	1.44	224	.15
Estradiol*Progesterone	.03 (.01)	2.18	215	**.030**
Negative Urgency	-.006 (.01)	-0.38	218	.70
Estradiol*Negative Urgency	.01 (.02)	0.49	228	.62
Progesterone*Negative Urgency	-.006 (.02)	-0.26	228	.79
Estradiol*Progesterone*Negative Urgency	-.006 (.01)	-0.38	218	.70
Covariates				
Negative Affect	.18 (.02)	7.31	224	**<.001**
BMI	.06 (.03)	1.91	220	.06
Negative Emotionality as a Moderator				
Intercept	-.03 (.01)	-3.52	230	**.001**
Estradiol	-.001 (.02)	-0.47	229	.96
Progesterone	.03 (.02)	1.54	228	.12
Estradiol*Progesterone	.03 (.01)	2.25	223	**.026**
Negative Emotionality	-.004 (.01)	-0.42	198	.68
Estradiol*Negative Emotionality	-.004 (.02)	-0.21	223	.84
Progesterone*Negative Emotionality	-.007 (.02)	-0.33	224	.74
Estradiol*Progesterone*Negative Emotionality	.01 (.01)	0.86	221	.39
Covariates				
Negative Affect	.18 (.02)	7.35	227	**<.001**
BMI	.05 (.03)	2.24	224	.09

Note. BMI = body mass index.

APPENDIX B

Figures A1-A3 and B1

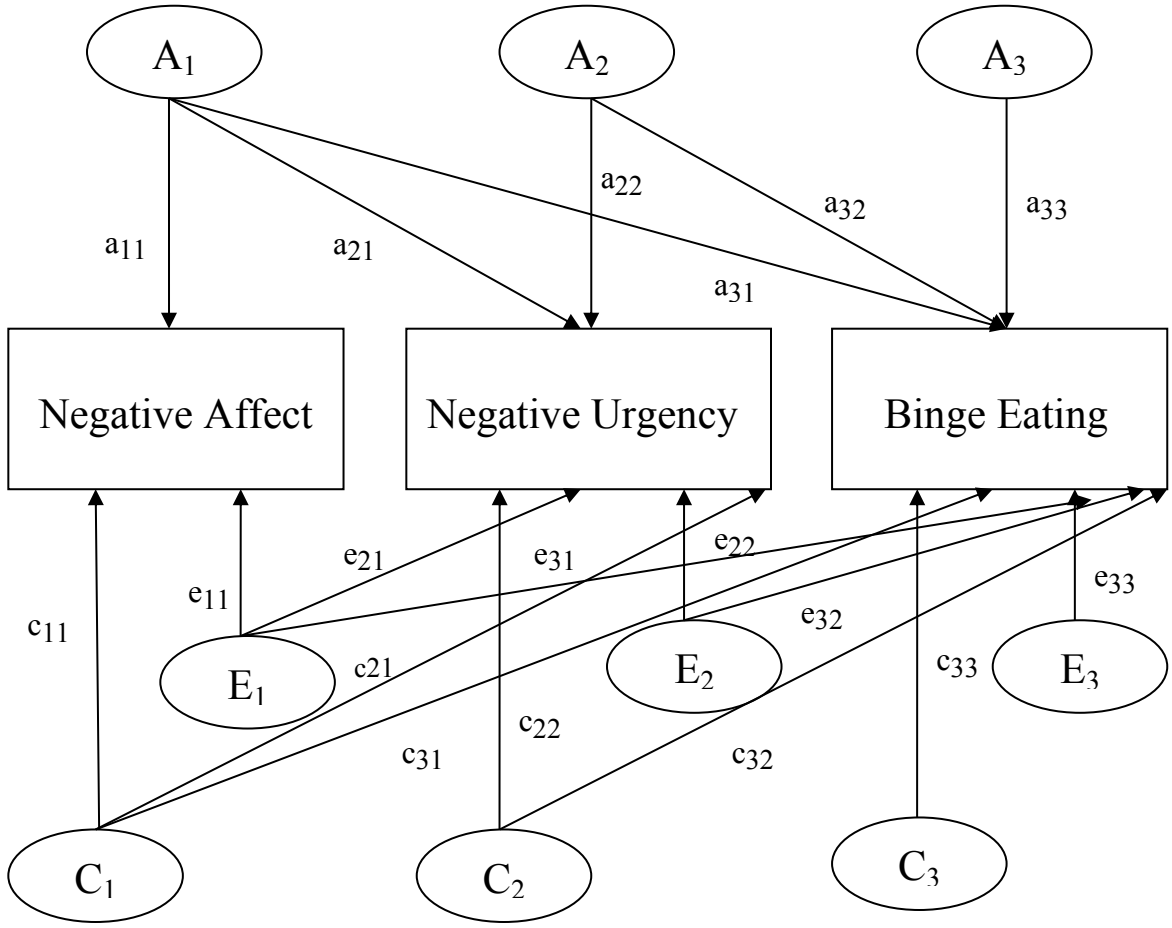


Figure A1. Path diagram for the trivariate Cholesky decomposition model.

Variance estimates are assumed to be comprised of additive genetic (A), shared environmental (C), and non-shared environmental (E) effects, including 1) genetic and environmental effects that impact negative affect, negative urgency, and binge eating (A1, C1, E1), 2) genetic and environmental influences that only contribute to negative urgency and binge eating (A2, C2, E2) and, 3) genetic and environmental influences unique to binge eating (A3, C3, E3). Pathways are represented by lowercase letters and two numbers, the first which represents the variable being influenced, and the second which reflects the latent factor

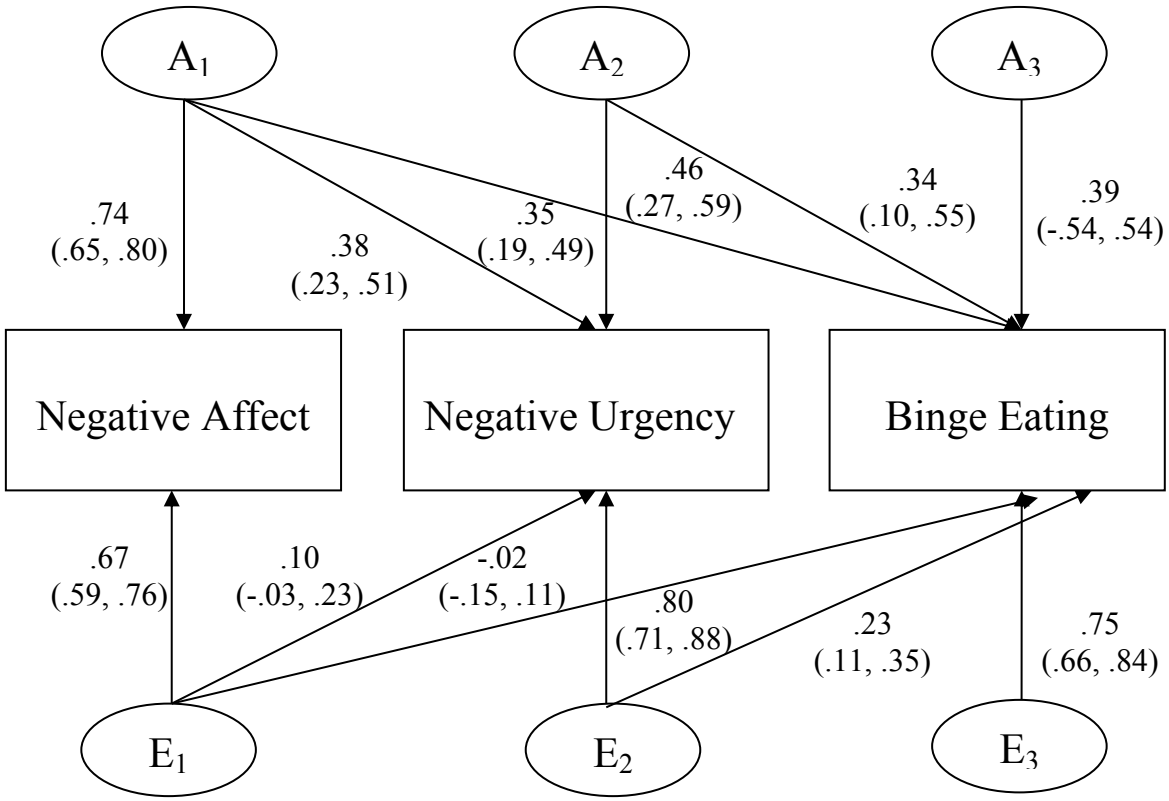


Figure A2. Standardized path estimates for the additive genetic (A) and non-shared environmental contributions (E) to the variance within and covariance among negative affect, negative urgency, and MEBS Binge Eating.

95% confidence intervals presented in parentheses. Path estimates are squared to obtain variance components, which are discussed in the text.

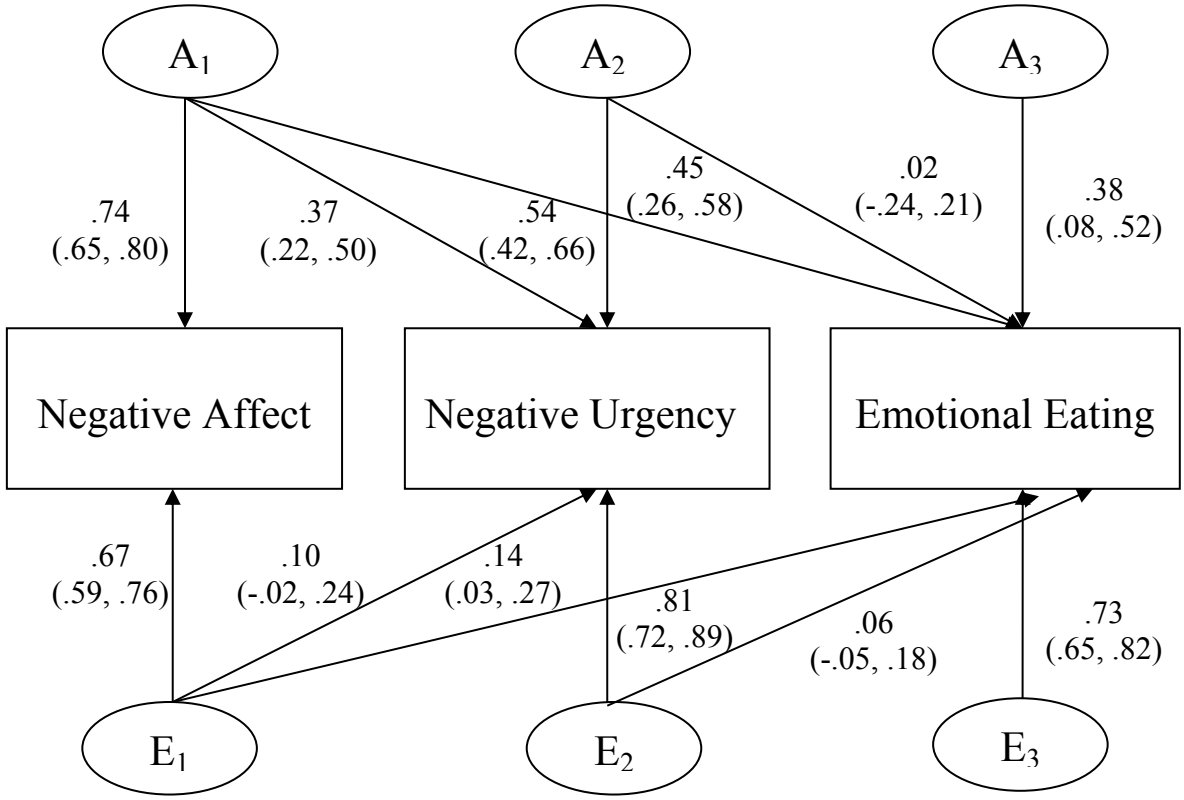
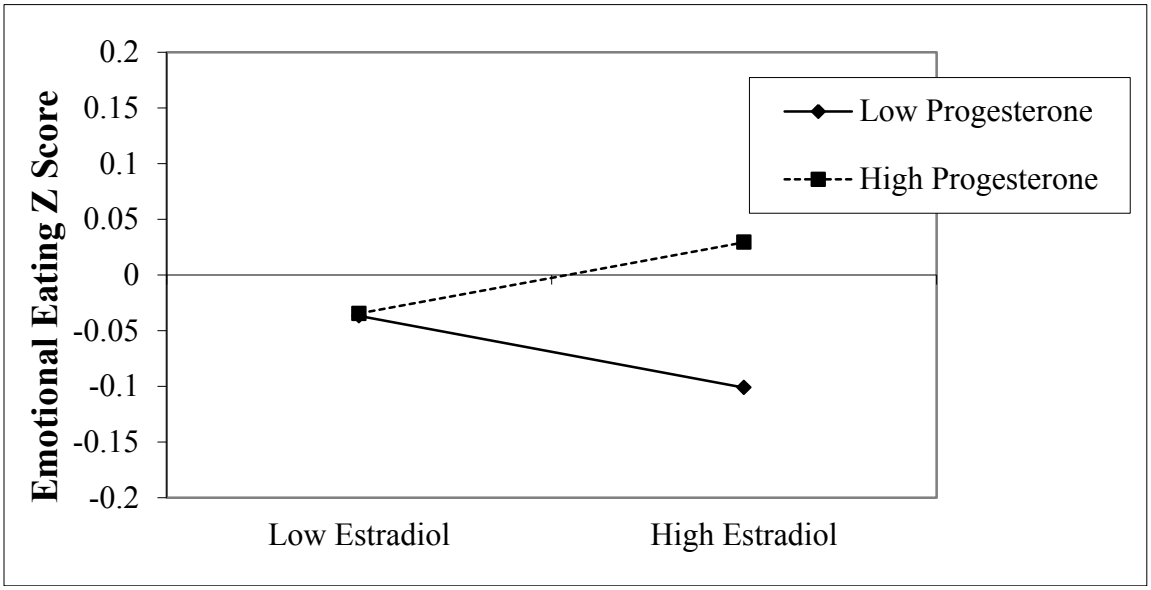


Figure A3. Standardized path estimates for the additive genetic (A) and non-shared environmental contributions (E) to the variance within and covariance among negative affect, negative urgency, and DEBQ Emotional Eating. 95% confidence intervals presented in parentheses.

Path estimates are squared to obtain variance components, which are discussed in the text.

a) <u>Estradiol x Progesterone Interaction in the Negative Urgency Model</u>



a) <u>Estradiol x Progesterone Interaction in the Negative Emotionality Model</u>

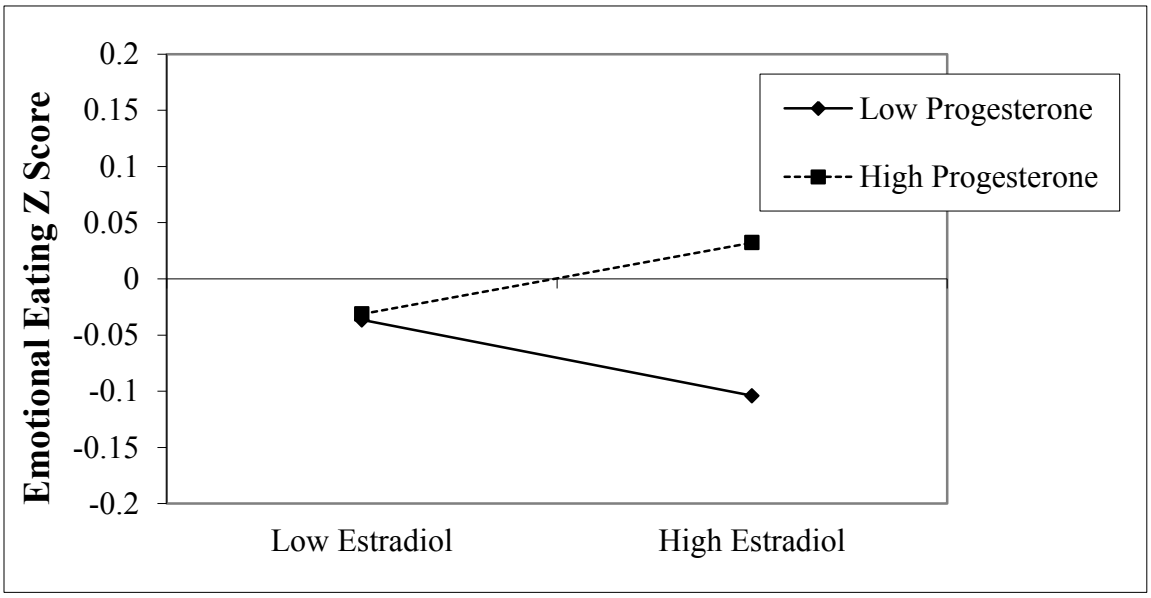


Figure B1. Interactions between Estradiol and Progesterone in the Prediction of Emotional Eating Scores in the i) Negative Urgency Model; and ii) Negative Emotionality Model. "Emotional Eating Z Score" = 5-day rolling average calculated within subjects, then averaged across participants.

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