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Expression of Borderline Personality Disorder Symptoms across the Ovulatory Cycle: A Multilevel Investigation

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EXPRESSION OF BORDERLINE PERSONALITY DISORDER SYMPTOMS
ACROSS THE OVULATORY CYCLE: A MULTILEVEL INVESTIGATION

DISSERTATION

A dissertation submitted in partial fulfillment of the
requirements for the degree of Doctor of Philosophy
in the College of Agriculture at the University of Kentucky

By
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Lexington, Kentucky

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Lexington, Kentucky

2013

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ABSTRACT OF DISSERTATION

EXPRESSION OF BORDERLINE PERSONALITY DISORDER SYMPTOMS ACROSS THE OVULATORY CYCLE: A MULTILEVEL INVESTIGATION

Borderline Personality Disorder (BPD) is a disabling condition characterized by chronic emotion dysregulation and behavioral impulsivity. Prospective studies that test proposed mechanisms of within-person change in BPD hold the key to improving symptom predictability and control in this disorder. A small body of evidence suggests that fluctuations in estradiol such as those occurring naturally at ovulation during the monthly female reproductive cycle may increase symptoms in women with BPD (DeSoto et al., 2003). Furthermore, there is preliminary evidence that both self-esteem and feelings of social rejection are highest at ovulation, when estradiol peaks (Durante and Hill, 2009; Eisenlohr-Moul et al., under review). Such feelings have been reliably linked to increases in BPD-related behavior in all individuals (e.g., Twenge et al., 2002). The purpose of this dissertation was to test a cyclical vulnerability model for women with BPD in which ovulatory estradiol shifts are associated with reductions in felt social acceptance, which in turn are associated with increased BPD symptom expression. 40 women, sampled to achieve a flat distribution of BPD symptoms, completed 28 daily diaries online, as well as four 1-hour weekly visits to the laboratory to complete longer assessments and provide saliva samples, which were assayed for estradiol. In addition, participants underwent the Structured Clinical Interview for the Diagnosis of BPD at the end of the study. Results of multilevel models revealed the opposite of the predicted effects of within-person changes in estradiol and their interaction with trait BPD. The data suggest a pattern in which women high in trait BPD show increases in felt acceptance and reductions in BPD symptom expression at higher levels of conception probability and higher-than-usual levels of estradiol. Women low in trait BPD show the opposite pattern in some cases. Several alternative moderators were tested, and results suggest that some risk factors for BPD (e.g., Neuroticism, Sexual Abuse) interact with high *trait* levels of estradiol to predict greater symptoms. Both average levels of estradiol and monthly fluctuations in estradiol may have relevance for women with BPD. It is recommended that future studies utilize clinical samples and additional physiological measures to

further elucidate the mechanisms through which estradiol exerts clinically-relevant change.

KEY WORDS: Borderline Personality Disorder, 17-Beta Estradiol, Menstrual Cycle, Ovulation

Tory Anne Eisenlohr-Moul

April 24, 2013

EXPRESSION OF BORDERLINE PERSONALITY DISORDER SYMPTOMS
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Chapter One: Introduction

Borderline Personality Disorder

Individuals with Borderline Personality Disorder (BPD) suffer from a distinctive combination of particularly disabling psychological and behavioral symptoms (Skodol et al., 2002). Extreme emotion dysregulation, harmful impulsive behaviors, identity disturbance, chronic feelings of emptiness, repeated parasuicidal and suicidal behaviors, and chaotic interpersonal relationships are all common features of BPD. Roughly 10% of outpatients and 20% of inpatients meet criteria for BPD, and epidemiological studies suggest that roughly 6% of the U.S. population will meet criteria for BPD at some point in their lives (DSM-IV-TR, 2000; Swartz et al., 1990; Widiger & Weissman, 1991; Grant et al., 2008). Though the disorder is equally prevalent in men and women, more women than men receive the diagnosis, and BPD is associated with greater distress and functional impairment in women (Grant et al., 2008).

For many years, BPD was considered untreatable by psychiatrists and psychologists alike. Historically, individuals with BPD have been perceived by clinicians as manipulative, hostile, and unable to benefit from treatment due to their erratically shifting emotions, behavior, and attitudes—especially toward clinicians and therapy. However, the recent development of cognitive and behavioral interventions such as Dialectical Behavior Therapy (DBT; Linehan, 1993), which have shown promise in the treatment of BPD, has led clinical scientists to rethink the assumption that individuals with BPD either do not wish to change or are unable to change. This has led to an increased interest in the study of potential mechanisms of treatment-related improvement among those who suffer from this condition.

Though BPD and its response to treatment are better understood today than when treatments such as DBT were initially developed, the nature and causes of natural day-to-day variability in BPD symptom expression remain poorly understood, especially on a physiological level. A clearer understanding of the physiological underpinnings of emotional, cognitive, and behavioral variability in BPD would be of great use to those interested in improving and streamlining both psychosocial and physiological treatments for the disorder. Existing treatments for BPD focus on the development of skills aimed at understanding, acknowledging, and responding to emotions, thoughts, and physical sensations in more helpful ways. Such cognitive behavioral treatments are effective for reducing dysfunctional, harmful behavior patterns; they are also costly and more time consuming than normal outpatient psychotherapy. A better understanding of the physiological causes of symptom variability in BPD could aid in the development of synergistic approaches that utilize both psychosocial and biological treatments. Therefore, the present project sought to address this gap in the scientific literature by exploring whether the female monthly reproductive cycle is a reliable source of variability in day-to-day and week-to-week expression of BPD symptoms.

The Reproductive Cycle and BPD Symptoms: Could Hormones Play a Role?

Given the higher rates of diagnosis and diagnosis-related distress and impairment among women with BPD, it has been suggested that monthly fluctuations in reproductive hormones associated with the reproductive (or ovulatory) cycle may play a role in symptom expression. A small literature, reviewed below, specifically examines the links between estradiol and BPD symptoms. Although progesterone and other hormones may play important roles in predicting these outcomes, the present project focuses primarily

on the association of ovulation and associated changes in estradiol on symptom expression.

Cyclical and Hormonal Effects on BPD Symptoms. A small body of research links shifts in estradiol, a steroid reproductive hormone that rises before and falls after ovulation, to increased expression of BPD symptoms. Across the female lifespan, BPD symptoms are greatest during adolescence and perimenopause—developmental transitions characterized by rapid changes in estradiol (Bardenstein & McGlashen, 1988; Stone, 1992). Additionally, differential prevalence of BPD in men and women has been identified during these same developmental transitions, suggesting that shifts in estradiol may be associated with changes in BPD symptom expression (Bardenstein & McGlashen, 1988).

The most convincing evidence for a causal link between estradiol and BPD symptoms comes from a series of three studies conducted by DeSoto, Geary, Hoard, Sheldon, and Cooper (2003). In the first study, 226 undergraduate women completed the Borderline subscale of the Personality Assessment Inventory (PAI-BOR; Morey, 1991) and reported the number of days since the beginning of their most recent menstrual period. Individuals assessed on days 5 through 10 of their cycle, when estradiol is rising, reported higher levels of BPD symptoms. In the second study, 57 women provided four weekly saliva samples and PAI-BOR assessments. Greater overall variability in estradiol across the cycle was associated with higher overall PAI-BOR scores. In the third study, 24 women about to begin estrogen-containing hormonal contraceptives and 29 control participants completed eight weekly PAI-BOR measurements (four measurements prior to starting birth control and four measurements after starting birth control). Women with

above average levels of BPD symptoms prior to starting the pill evidenced a significant increase in symptoms after starting the pill, whereas BPD symptoms did not change significantly in women with low pre-existing symptoms or in women not starting the pill. Additionally, results of a laboratory study of 52 women suggest that even short-term changes in estradiol occurring during a laboratory session are associated with higher scores on the PAI-BOR (Evardone, Alexander, & Morey, 2008). Taken together, these results provide a groundwork of evidence that changes in estradiol at ovulation may have implications for the expression of BPD symptoms. Furthermore, the finding that shifts in estradiol may be problematic specifically for women with high trait levels of BPD symptoms is consistent with recent evidence that women may differ in their emotional and behavioral responses to hormones across the reproductive cycle (Kiesner, 2011). Finally, no study has examined the effects of both average levels of estradiol (between-person effects on *trait* BPD symptoms) and fluctuations in estradiol (within-person effects on *state* BPD symptoms).

Cyclical Effects on Felt Social Acceptance. Evidence from the social and evolutionary psychological literature also indicates that ovulation—and, by extension, fluctuations in estradiol—is associated with decreased self-esteem (Hill & Durante, 2009), decreased felt acceptance (Eisenlohr-Moul et al., under review), and increased implicit motivation to affiliate closely with others (Schultheiss, Dargel, & Rohde, 2003). Such changes may indicate a downward shift in feelings of social acceptance at ovulation (Leary, Haupt, Strausser, & Chokel, 1998). While such patterns may be benign or even adaptive in normal women, fluctuations in levels of self-esteem, felt acceptance, and

motivations to pursue relationships may play a role in within-person changes in BPD symptoms across the reproductive cycle.

A New Cyclical Vulnerability Theory of BPD in Women

Sociometer theory (Leary et al., 1998) argues that self-esteem serves as an indicator of social acceptance (i.e., a “sociometer”). Leary (2004) points out that the “sociometer” can sometimes be miscalibrated; one type of miscalibration occurs when the sociometer is “set low.” Individuals with a sociometer that is set low tend to perceive less social acceptance than others are objectively communicating, thereby experiencing chronically low self-esteem. If, in some women, ovulatory estradiol release leads to exaggerated reductions in self-esteem (i.e., decreased felt acceptance), this may create strong monthly patterns of change in attitudes towards oneself and others. This appears to describe BPD symptoms quite well; individuals with BPD suffer from excessive variability in attitudes toward themselves (self-esteem; Tolpin, Gunthert, Cohen, & O’Neill, 2004) and others (Russell, Moskowitz, Zuroff, Sookman, & Paris, 2007) that may be expected to arise when a sociometer is recalibrated drastically twice each month (i.e., prior to and after ovulation).

I propose a new cyclical vulnerability theory of within-woman change in BPD that predicts exaggerated downward shifts in sociometric calibration and increased symptoms as a function of ovulatory increases in estradiol in women with high trait levels of BPD symptoms. As stated before, some evidence suggests that feelings of self-esteem and social acceptance decrease at ovulation in all women (Durante & Hill, 2009; Eisenlohr-Moul et al., under review). Such feelings of low social acceptance are 1) inherent in many of the symptoms of BPD and 2) a reliable risk factor for behavioral

dysregulation of the kind typically seen in BPD (impulsivity and risk-taking: Twenge, Catanese, & Baumeister, 2002; anger and aggression: Sampson & Laub, 1990; Twenge et al., 2007; identity disturbance: Richman et al., under review; chronic feelings of emptiness: Twenge, Catanese, & Baumeister, 2003). Also critically, no study has simultaneously examined the impact of both trait levels of felt acceptance (e.g., rejection sensitivity) and within-person changes in felt acceptance on expression of BPD symptoms. The present study will seek to comprehensively test this full cyclical vulnerability theory on multiple levels using multiple methods.

FFM Personality Abnormalities: Alternative Moderators of Cyclical Vulnerability

BPD is an extremely heterogeneous disorder; there are 256 ways to meet criteria for BPD using the DSM-IV-TR diagnostic criteria (Ellis, Abrams, & Abrams, 2008). Furthermore, BPD symptoms overlap strongly with a wide variety of other psychological disorders such as anxiety disorders, other personality disorders, mood disorders, and substance use disorders (Lenzenweger, Lane, Loranger, & Kessler, 2007). In an attempt to more clearly capture the underlying personality extremity that characterizes BPD and other personality disorders, recent models of personality disorders posit a dimensional model of personality disorders based on the Five Factor Model (FFM) of Personality (Widiger & Mullins-Sweatt, 2009). Of relevance for the present study, individuals with BPD are characterized by extremely high levels of all aspects of Neuroticism, low levels of some aspects of Agreeableness and Conscientiousness, and high levels of some aspects of Openness to Experience. Such traits may be the “active ingredients” in moderating the effect of cycle variables on BPD symptoms. *If “trait” levels of BPD do indeed modulate the impact of ovulatory estradiol shifts on BPD symptom expression, it would be*

theoretically important to determine which aspect(s) of extreme FFM personality functioning are responsible for this reactivity to the cycle.

A Special Case of Linehan's Biosocial Model of BPD: Aspects of Childhood Maltreatment as Alternative Moderators of Cyclical Vulnerability

Linehan's biosocial model of BPD (Linehan, 1993) is "a biological theory of emotion regulation" positing that symptoms of BPD emerge as a result of a complex interaction between (1) a physiological predisposition toward emotion dysregulation, and (2) an invalidating childhood environment. The physiological predisposition is conceptualized as whichever physiological conditions produce a pattern of extreme emotional reactions, sensitivity to such emotional reactions, and abnormally prolonged emotional reactions (i.e., slow return to baseline). The cyclical vulnerability model proposed above assumes that, at least for some women, higher-than-usual estradiol may *temporarily* increase one's physiological predisposition toward emotion dysregulation. In this way, the cyclical vulnerability model is a kind of special case of the biosocial theory in which *state* levels of physiological variables (i.e., fertility, estradiol) are thought to boost a physiological predisposition to emotion dysregulation, creating a greater sensitivity to internal and external social rejection cues.

The second part of the biosocial model has to do with childhood environments that invalidate and devalue the child's desires and emotions rather than instructing the child in or modeling useful skills for managing emotions, thoughts, and behavior. Linehan argues that such invalidating environments teach the child to respond to their own emotional responses in unhelpful ways (e.g., chronic emotional suppression) that tend to have the paradoxical effects of further dysregulating emotions and behavior

among these individuals. In discussing the myriad forms that such invalidating environments can take, Linehan emphasizes both the subjectivity of the experience of emotional invalidation and the idea that overt childhood emotional, physical, and sexual abuse exemplify invalidating environments. It is possible that some aspects of childhood maltreatment that interact with trait physiological vulnerability to emotion dysregulation could also act as potent moderators of the *transient* ovulatory changes in physiologically-based emotional vulnerability hypothesized here. *In the present study, therefore, if “trait” levels of BPD do indeed modulate responses to estradiol and other ovulatory processes, I will explore the possibility that various aspects of childhood maltreatment serve as a central predisposing factors that interact with ovulatory physiological changes to produce changes in BPD symptoms.*

Alternative Mechanisms of Cycle-Related Change in BPD Symptoms

The cyclical vulnerability theory presented here predicts that ovulatory effects on the emotional and behavioral symptoms of BPD across the cycle occur due to decreases in felt acceptance. More proximally, however, these changes may also be due to ovulatory changes in various aspects of impulsivity or global self-control. In the context of this theory, it may be helpful to determine whether cycle-related changes in BPD symptoms are also mediated by ovulatory effects on one of these interrelated variables with relevance for BPD: global self-regulatory ability, emotion-related urges, problems with thinking ahead (or premeditation), problems persisting in the face of difficulty, or desire to seek out new, potentially dangerous sensations. *Therefore, in addition to testing felt acceptance as the primary mediator in the cyclical vulnerability model, I will also test these more proximal mediators of cyclical effects on BPD symptoms.*

Global Self-Control as an Alternative Mediator

Self-control refers to the application of executive cognitive functions in the service of long-term goals. Although some evidence suggests that self-control and the executive functions that underlie self-control may be heterogeneous (Duckworth & Kern, 2011), questionnaires measuring a unitary construct of self-control perform quite well in predicting a wide variety of BPD-related distress and functional outcomes (de Ridder, Lensvelt-Mulders, Finkenauer, Stok, & Baumeister, 2012). Therefore, the present study operationalizes self-control as successful application of executive functions that produce adaptive behavioral responses consistent with long-term goals in a wide variety of situations (Tangney, Baumeister, and Boone, 2004). This will also be the first study to simultaneously examine both between-person and within-person effects of global self-control on the expression of BPD symptoms.

Aspects of Impulsivity as Alternative Mediators

The UPPS-P model of impulsivity describes five aspects of impulsivity (Whiteside and Lynam, 2001): *Negative Urgency*, or the tendency to experience increased urges and to engage in related rash action under conditions of negative emotion; *Positive Urgency*, or the tendency to experience increased urges and to engage in related rash action under conditions of positive emotion; *Lack of Premeditation*, or the tendency to fail to think over the consequences of an action before engaging in that action; *Lack of Perseverance*, or difficulty persisting on tasks that may be difficult or boring; and *Sensation Seeking*, or the tendency to seek out and be open to engaging in stimulating, novel experiences that may or may not be dangerous. Previous evidence links trait levels of BPD symptoms as measured using the Personality Assessment

Inventory Borderline subscale (PAI-BOR; Morey, 1991) to higher trait levels of Negative Urgency, Positive Urgency, and Lack of Premeditation (Tragesser & Robinson, 2009). However, as with self-control, no study has examined the potentially unique roles of between-person differences and within-person changes in the UPPS-P facets on BPD symptoms.

The Present Study: Aims and Hypotheses

Since the establishment of tentative links between ovulatory estradiol shifts and BPD symptoms a decade ago (DeSoto et al, 2003), little or no work has examined the psychological mechanisms through which estradiol influences BPD symptoms. Prospective studies that test proposed mechanisms of within-person change in BPD hold the key to improving symptom predictability and control in this disorder. The present study aimed to test a proposed cyclical vulnerability model of BPD: women, and especially those with higher trait levels of BPD, respond to ovulatory estradiol shifts with exaggerated downward shifts in the calibration of the sociometer (decreased feelings of acceptance), which in turn leads to increases in BPD symptom expression. The present study tested this model using a 2-pronged repeated measures design.

It was hypothesized that;

1. In all participants, ovulation (as measured by both deviations in estradiol at the weekly level and conception probability at the daily level) would be associated with greater expression of BPD symptoms as measured by daily and weekly measures of BPD symptoms.
2. Trait levels of BPD symptoms (as measured by the average of weekly scores on a measure of BPD symptoms and number of SCID-II items endorsed in a diagnostic

interview) would moderate the effect of ovulation (as measured using weekly deviations in estradiol and daily conception probability) on expression of BPD symptoms. If these results are significant, further models will replace the Trait BPD variable with each of the five domains of the FFM and with aspects of childhood maltreatment in order to elucidate the underlying factor(s) conferring risk for ovulatory changes in BPD.

3. All significant effects of both deviations in estradiol and conception probability on symptoms of BPD would be mediated by decreases in felt acceptance at the same measurement level. Specifically, on days or weeks when estradiol or conception probability is high, felt social acceptance will be low and will mediate the association between estradiol deviations or conception probability and symptoms of BPD. Decreased feelings of social acceptance were also expected to mediate the interactive effect of estradiol and trait BPD on daily BPD symptoms.

Further, weekly changes in aspects of impulsivity and self-control were expected to mediate the association between weekly estradiol deviations and increased BPD symptoms, and also to mediate the interactive effect of estradiol deviations and trait BPD on weekly symptoms.

Chapter Two: Method

Overview and Study Design

The present study consisted of a two-pronged repeated measures approach to understanding within-woman change in BPD across the reproductive cycle. 40 women, sampled to achieve a flat distribution of trait BPD scores, were invited and incentivized to provide up to 28 days of online daily diary reports of ovulatory cycle status, felt acceptance, and BPD symptoms. Self-harm and suicidality items were omitted from daily diary assessments in order to reduce the risk of participant distress. In addition, they attended 4 weekly laboratory sessions at which they completed more comprehensive repeated assessments of felt acceptance, BPD symptoms, impulsivity, and self-control. During a fifth and final visit, participants underwent a structured clinical interview for diagnosis of BPD and completed trait measures of FFM personality traits and aspects of childhood maltreatment.

Participants

Participants were 40 naturally-cycling undergraduate women between the ages of 18 and 30 who were fulfilling research participation requirements for an introductory undergraduate psychology course. Participants were recruited so as to achieve a “flat” distribution; 10 participants had low average PAI-BOR scores ($T < 50$), 10 had high average scores ($50 < T < 60$), 10 had above average scores ($60 < T < 70$), and 10 will have high scores ($T > 70$). Participants were not allowed to participate if they reported 1) currently using hormonal birth control, 2) currently using any “as needed” psychiatric medication (e.g., benzodiazepines), 3) not speaking English fluently, 3) having reproductive cycles typically lasting fewer than 25 days or greater than 35 days, or 4)

having a history of psychosis other than brief periods of dissociation. Final demographic characteristics of the sample can be found in Table 1.

Procedure

Screening and Recruitment

Initial screening was completed in introductory psychology classes. During a department-wide screening session, women completed the PAI-BOR and a measure of inclusion criteria. 10 eligible women were recruited via telephone from each of the four symptom ranges. Interested participants were asked to schedule 4 repeating weekly timeslots at the same day and time each week.

Because individuals with high levels of trait BPD symptoms were expected to have greater-than-average difficulty complying with laboratory attendance expectations and daily diary protocols, monetary incentives were offered to increase the rate of daily diary and weekly session compliance. In addition to course credit for the completion of laboratory visits, individuals were paid \$25 if they completed at least 75% of the 28 daily diaries. In addition, in order to improve retention, weekly laboratory visits lasted only 50 minutes, and daily diaries were pre-tested to ensure that the average time to complete the diaries was roughly 5 minutes.

Weekly Laboratory Protocol

Participants came to the lab once a week for 5 weeks at the same day and time (4 assessments and 1 interview/debriefing session). Reminder emails were sent two days in advance of each session, reminding the participant of the location, date, and time of their next session as well as requesting that participants refrain from chewing gum, drinking caffeine or alcohol, or taking nonprescription medications for 12 hours prior to sessions

Table 1

Demographic Sample Characteristics (N = 40)

Variable	Mean	SD	%
Age	18.66	1.38	
Body Mass Index (BMI)	23.20	3.62	
Race			
Caucasian			73.2
African American			9.8
Hispanic			9.8
Asian American			14.6
Other			2.4
Religion			
Christian - Protestant			39
Christian - Catholic			34.1
Islam			2.4
Judaism			2.4
Other			2.4
None/Atheist/Agnostic			19.7

Note. SD = standard deviation.

on visit days (instructions related to saliva sample; see Estradiol section below). Nearly all missed sessions were rescheduled and completed within 3 days of the missed appointments; in the few cases where this was not possible ($n = 6$ sessions), the participant returned to the lab for the next scheduled session and added an additional week to their participation to compensate for the missed session.

Upon arrival in the laboratory for weekly sessions, the participant was met by either the principal investigator or an undergraduate research assistant and taken to a private room. At the first session, participants completed an informed consent form, were reminded of the exclusion criteria, and were oriented to the online diary system. Next, at each session, participants were reminded of the free mental health services available to them as students at the university and reminded that they could always call emergency services if they felt that they were not safe due to parasuicidal or suicidal symptoms. Participants were then asked to turn off their cell phones. Next, they were instructed in the provision of the saliva sample and entered information about any relevant control information into the computer (e.g., use of caffeine, medication, etc.). After that, participants were given 8 minutes to provide the saliva sample. Finally, after the experimenter assured the individual that all answers were confidential, participants completed all weekly measures on a computer in a randomized order. At the fifth session, the principal investigator administered the SCID-II for BPD diagnosis and debriefed the participant. Following the fifth session, participants were sent a check in the mail for \$25 if they had completed at least 75% of the online diaries that they received.

Follow-Up Risk Assessment Protocol

Weekly measurements included self-harm and suicidality items. Responses to these items were screened daily by the principal investigator, and follow-up telephone risk assessments were conducted where endorsement level indicated significant risk of harm to the participant. Follow-up telephone risk assessments consisted of a call to the participant in which the principal investigator briefly screened the participant for current risk of life-threatening self-harm or suicidal behavior (n = 23). In each case, risk assessments were carried out to ensure that the participant was not actively suicidal. Though in some cases participants endorsed suicidal planning (n = 3), no participants endorsed current intent to engage in self-injury or suicidal behavior. Each call consisted of a brief screening followed by encouraging the client to (1) seek out the free mental health care available to them as students at the university, and (2) go to the local emergency room if necessary. Though some participants reported thoughts about suicidal planning (n = 6; e.g., “I think about the fact that if I cut myself in the right way, I could die), no participants reported current suicidal intent (e.g., “I think about how I could do it, but I never would and I have no intent to die”). Therefore, these telephone risk assessments did not result in any emergency hospitalization of clients. No clients reported seeking out psychological or psychiatric services during the study; however, several participants noted their intention to do so after completion of the study.

Daily Diary Protocol

As described above, participants were oriented to the online diary system during the first laboratory session. They provided their email address, which was then added to the mailing list to receive the daily diaries for 28 days. Participants received a daily

email with a link to the URL for that day's online diary for the completion of de-identified daily assessments. During the first session, participants were instructed to fill out the daily diary between 5pm-2am, and that diaries completed outside of this time frame would not be accepted.

Weekly Measures

Items administered repeatedly at each of the four weekly sessions are discussed below. Means, standard deviations, and aggregated internal consistencies for estradiol and measures of BPD are discussed and compared in the descriptive analysis section of the Results. Means, standard deviations, and aggregated internal consistencies for weekly mediators are presented in the text below.

17 β -Estradiol

Participants were instructed to salivate by passive drool into a polypropylene vial (Salimetrics; State College, PA). During each session, participants recorded use of the following in the past 24 hours: nicotine, caffeine, over-the-counter drugs, prescription drugs, and illicit drugs. No participants reported prescription or illicit drug use. At some assessment points, participants reported having smoked cigarettes (13 assessment points), having more than 1 caffeinated beverage (2 assessment points), or having used over-the-counter drugs (7 assessment points) in the past 12 hours.¹ Participants passively drooled into a polypropylene vial through a straw until 1.8 mL of saliva had been collected.

Samples were immediately frozen at -20° C in a locked room. Later, they were

¹ All hypotheses concerning estradiol were originally tested controlling for the use of these substances; however, use of these substances did not significantly impact estradiol or any outcome, and inclusion of these controls in models did not change model outcomes in any substantive way. Therefore, they were not included in models presented in the results section.

transferred to the University of Kentucky General Clinical Research Center for 17β -Estradiol assay using ELISA kits (Salimetrics). Intra-assay coefficient of variation for estradiol was 1.6%; inter-assay coefficient of variation was 2.2%. The standard curves were of expected shape and slope for 17β -Estradiol.

Weekly BPD Symptoms

Personality Assessment Inventory - Borderline Subscale (PAI-BOR; Morey, 1991).

The PAI-BOR is a 24-item measure of BPD symptoms, including 4 subscales measuring affective instability (example item: “my mood could shift quite suddenly”), identity problems (example item: “my attitude about myself changed a lot”), negative relationships (example item: “my relationships have been stormy”), and self-harm (example item: “I was a reckless person). Notably, the self-harm subscale actually measures tendency toward impulsive behaviors rather than physical self-harm. The PAI-BOR is the most well-studied measure of borderline personality disorder symptoms, and has been used widely in both research and clinical settings to predict BPD diagnosis (Morey, 1991; Stein, Pinsker-Aspen, & Hilsenroth, 2007). Participants were asked to rate the extent to which each statement described them *in the past week* on a scale from 0 (False, Not True at All) to 4 (Very True).

Borderline Symptom List - 23 (BSL-23; Bohus et al., 2007). The BSL-23 is a 23-item shortened version of a 95-item measure of BPD symptoms based on the SCID-II DSM-IV diagnosis of BPD. In the initial validation sample, scores on the full and shortened versions of the BSL were significantly greater among individuals with a SCID-II diagnosis of BPD than among those with Axis I diagnosis (e.g., mood or anxiety disorders) and among healthy controls. In another validation sample of individuals with a

diagnosis of BPD, scores the BSL reduced significantly in response to Dialectical Behavior Therapy; this evidence that BSL scores change with treatment makes it a particularly appropriate measure of change for the present study. Example items include “I felt helpless”, “my mood rapidly cycled in terms of anxiety, anger, and depression”, “I was afraid of losing control”, and “I didn’t believe in my right to live.” Participants were asked to rate the extent to which each statement described them *in the past week* on a scale from 0 (Not at all) to 4 (Very much). A simple comparison of the face validity of the items on this scale to items on the PAI-BOR led to the conclusion that the items on this scale may be more appropriate for measuring the upper, more extreme range of BPD symptoms. Distributions of the two scales confirmed this; scores on the BSL-23 appeared more positively skewed than scores on the PAI-BOR, suggesting that the BSL-23 may be capturing rarer, more extreme symptoms of BPD. In further support of this hypothesis, the correlations of number of SCID-II criteria met in the present study with the PAI-BOR and the BSL-23 revealed a significantly stronger correlation of SCID-II number of criteria with the BSL-23 than with the PAI-BOR or the MSI-BPD (see descriptive section of the Results).

McLean Screening Instrument for BPD (MSI-BPD; Zanarini et al., 2003). The MSI-BPD uses 10 dichotomous (yes or no) items to measure the nine DSM-IV BPD criteria. Example items include, “Have you been distrustful of other people?”, “Have you been extremely moody?”, and “Have you deliberately hurt yourself physically (e.g., punched yourself, cut yourself, burned yourself)? How about made a suicide attempt?” In several studies, scores on the MSI-BPD were positively associated with other measures

of BPD symptoms (Gardner & Qualter, 2009), and predicted actual SCID-II diagnosis of BPD (Zanarini et al., 2003).

Weekly Feelings of Social Acceptance

Weekly feelings of social acceptance were measured using the Social Evaluation scale of the State Self-Esteem Scale (SSES; Heatherton & Polivy, 1991). The Social Evaluation subscale uses 7 items to measure the degree to which an individual feels that they are valued highly by the members of their social group. Social stress tasks and social shame inductions reliably produce temporary reductions in scores on this subscale of the SSES, suggesting that it taps into relatively temporary levels of felt acceptance (Gruenewald, Kemeny, Aziz, & Fahey, 2004). Example items include, “I feel inferior to others at this moment” (reverse scored) and “I feel that others respect and admire me.” Participants rated the extent to which they felt each item described their thoughts *in the past week* on a scale from 1 (Not at all) to 5 (Extremely). In the present study, individuals’ average scores on this scale functioned adequately with a mean of 5.37, a standard deviation of .88, and an internal consistency of $\alpha = .87$.

Weekly Self-Control

Self-control was measured using the Brief Self-Control Scale (BSCS; Tangney, Baumeister, & Boone, 2004). The BSCS is a 13-item questionnaire designed to measure one’s trait capacity for self-control. Example items include, “I am good at resisting temptation”, “I am lazy” (reverse-scored), and “People would say that I have iron self-discipline.” Participants were asked to rate the extent to which each item had been true of them in the past week on a scale from 1 (“Not at All”) to 5 (“Very Much). Higher scores on the self-control scale are associated with higher grade point average and positive

psychological adjustment (Tagney, Baumeister, & Boone, 2004). In the present study, individuals' average scores on the SCS functioned adequately, with a mean of 3.42, a standard deviation of .05, and an internal consistency of $\alpha = .87$.

Weekly Impulsivity

Weekly impulsivity was measured using the UPPS-P Impulsivity Scale (Whiteside and Lynam, 2001). The UPPS-P impulsivity scale measures five distinct pathways to impulsive behavior: negative urgency, or the tendency to act rashly in the face of negative emotion, positive urgency, or the tendency to act rashly in the presence of positive emotion, lack of perseverance, or the lack of ability to persist in the face of boredom, lack of premeditation, or the tendency not to think through actions, and sensation seeking, or the tendency to engage in novel, high-sensation behaviors. Example items include, "This week, it was hard for me to resist acting on my feelings" (Negative Urgency), "This week when I was happy, I tended to do things that could cause problems in my life" (Positive Urgency), "This week, my thinking was careful and purposeful" (Lack of Premeditation), "This week, I finished what I started" (Lack of Perseverance, and "This week, I sought out new experiences and sensations" (Sensation Seeking). Participants were asked to rate the extent to which each item had been true for them in the past week on a scale from 1 (Not at All) to 4 (Very much). In a variety of studies, facets of the UPPS-P have predicted a variety of impulsivity-related outcomes such as substance use and abuse (Verdejo-Garcia, Bechara, Recknor, & Perez-Garcia, 2007), problem gambling (Cyders & Smith, 2008), eating disorder symptoms (Fischer, Anderson, & Smith, 2004), and aggression (Derefinko, DeWall, Metze, Walsh, & Lynam, 2011). In the present study, individuals' average scores on the UPPS-P functioned

adequately, including Negative Urgency (Mean: 3.30, SD: .05, $\alpha = .83$), Positive Urgency (Mean: 3.15, SD: .04, $\alpha = .73$), Lack of Premeditation (Mean: 3.15, SD: .06, $\alpha = .85$), Lack of Perseverance (Mean: 3.13, SD: .05, $\alpha = .84$), and Sensation Seeking (Mean: 2.76, SD: .06, $\alpha = .78$).

Daily Measures

Conception Probability

Daily, participants reported the start date of their most recent menstrual period. This allowed for the calculation of cycle day and associated conception probabilities (Wilcox et al., 2001) for estimating fertility/ovulation.

Daily BPD Symptoms

Daily symptoms of BPD were measured using a modified version of the Personality Assessment Inventory - Borderline Subscale (see weekly measures above; PAI-BOR; Morey, 1991) in which the one item referencing physical self-harm (“When I was upset, I typically did something to hurt myself”) was omitted. This item was omitted to reduce the risk of daily priming and distress. Participants were asked to rate the extent to which each statement described them *in the past 24 hours* on a scale from 0 (False, not true at all) to 4 (Very true). Descriptive statistics and internal consistencies on the PAI-BOR at the daily level were as follows: Affective Instability (Mean: .59, SD: .02, $\alpha = .84$), Identity Instability (Mean: .65, SD: .02, $\alpha = .70$), Negative Relationships (Mean: .73, SD: .02, $\alpha = .74$), and Self-Harm (Mean: .38, SD: .01, $\alpha = .73$).

Daily Felt Acceptance

Daily levels of felt acceptance were measured using a scale created for this study. Participants were asked to rate the following items indicating the extent to which she had experienced each item *in the past 24 hours*: “Accepted”, “Included”, “Rejected”, “Excluded”, “Lonely”, and “Abandoned” (last four items reverse-scored). Response options ranged from 1 (“Very Slightly or Not at All”) to 5 (“Extremely”). Descriptive statistics and internal consistency for the average of daily felt acceptance are as follows: Mean = 4.05, SD = 1.10, $\alpha = .91$. In addition, person averages of this composite variable were negatively associated with person averages of weekly measures of Social Evaluation subscale of the State Self-Esteem Scale ($r = .84, p < .0001$).

Between-Person Measures

Five Factor Model (FFM) Personality

The Five Factor Model Rating Form (FFMRF) is a 30-item instrument that asks participants to rate themselves on the 30 facets of the Five Factor Model of personality (FFM; Mullins-Sweatt, Jamerson, Samuel, Olson, & Widiger, 2006). Each item is rated on a scale from 1 (“Extremely Low”) and 5 (“Extremely High”). Individual facets were combined to construct the FFM domains: Neuroticism, Extraversion, Openness to Experience, Agreeableness, and Conscientiousness. This short measure of the five factor model of personality has been validated for use in student samples, and correlates well with longer measures of FFM personality such as the NEO-PI-R (Costa & McCrae, 1992; Mullins-Sweatt et al., 2006).

Childhood Maltreatment

The Childhood Trauma Questionnaire (CTQ) is a 28-item retrospective measure of various types of maltreatment in childhood, and includes subscales measuring Emotional Abuse (example item: “Someone in my family yelled and screamed at me”), Emotional Neglect (“I felt like there was someone in my family who wanted me to be a success”—reverse-scored), Physical Abuse (“Someone in my family hit me so hard that it left me with bruises or marks”), Physical Neglect (“I had to wear dirty clothes”), and Sexual Abuse (“Someone threatened to hurt me or tell lies about me unless I did something sexual with them”) (Bernstein et al., 2003). Each item begins with the phrase, “When I was growing up,” and is rated on a scale from 1 (“Never True”) to 5 (“Rarely True”). The CTQ also includes a 3-item validity scale designed to detect underreporting of maltreatment; subscale scores from individuals scoring above the cutoff on this validity scale are invalid and not included in analyses ($n = 0$ in the present study). In diverse samples, the CTQ correlates well with therapist and peer reports of an individual’s childhood maltreatment, as well as independent evidence of childhood maltreatment in the domain specified (Bernstein et al., 2003).

DSM-IV Diagnosis of BPD

The BPD module of the Structured Clinical Interview for Diagnosis—II (SCID-II; First, 1997) was administered to determine diagnostic status and the number of DSM-IV BPD criteria met. During the final session, the principal investigator ($n = 30$) and another master’s level clinician with experience completing the SCID-II ($n = 10$) led each participant through the BPD module of the SCID-II. After completion of the study, transcripts of participant responses to interview prompts were independently scored

(without knowledge of the original clinician's ratings) by the clinician who did not complete the interview. Agreement was perfect for all items.

Data Analytic Plan

Power Analysis

The current study is powered on the results of the post-hoc moderation analyses from a preliminary study (Eisenlohr-Moul et al., under review). Power was estimated by (1) using formulae provided by Snijders and Bosker (1999) to determine design effects due to the intraclass correlation (ICC) and the resulting effective N, and (2) calculating smallest detectable effect size based upon the effective N, 80% power, and an alpha level of .05. Results indicated that the current study would have 80% power to detect small effects ($r = .10$) with 711 lower-level units, yielding an effective N of 515 after accounting for average ICCs for daily felt acceptance and BPD-related outcomes found in the original study. In order to obtain an effective N of this size in the current study, I would need only 25 women (i.e., 25 multiplied by 28 is equal to 728 daily measurements). However, in order to allow for missed diaries and to ensure that the sample size will be large enough to detect week-level effects, data will be collected from 40 women (i.e., 40 multiplied by 4 weekly visits is equal to 160 weekly measurements; effective N estimated to be 115). This results in 80% power to detect medium ($r = .25$) effects of estradiol at the weekly level, and small effects ($r = .12$) of conception probability (ovulation) at the daily level.

Multilevel Models

Data were analyzed using multilevel models in SAS PROC MIXED and SAS PROC GLIMMIX with daily diaries or laboratory visits at Level 1 and people at Level 2.

Multilevel models utilize all available data with no listwise deletion. In daily and weekly models, level 1 predictors were centered to isolate the within-person component. For example, each person at each laboratory visit had two estradiol variables: (1) the person's mean levels of estradiol across all visits (the same across all visits within an individual), and (2) the person's deviation from their own mean score at the current laboratory visit. The latter deviation reflects fluctuations in estradiol relative to the person's own mean level and was the relevant, visit-level predictor. Prior to conducting analyses, all variables were screened for distributional normality; in most cases, the distribution was positively skewed to such an extent that linear transformations were not successful in approximating normality. Given the relatively low base rate of BPD in the general population, such distributional characteristics are unsurprising. Preliminary inspection of the properties of these distributions suggested that most followed a Poisson or zero-inflated Poisson distribution. Furthermore, comparisons of the model fit for various alternative multilevel models (zero-inflated Poisson, negative binomial, and Poisson) suggested that a Poisson distribution resulted in the best fit. In these cases, therefore, multilevel Poisson models were utilized. All continuous between-person predictors were standardized.

Model Fitting and Random Effects

For each model, I tested the significance of changes in $-2 \log$ likelihood (or, in the case of Poisson models, -2 restricted log pseudo-likelihood) in a stepwise manner, comparing: (1) a model with no predictors and a random intercept, (2) a model adding all relevant predictors for testing hypotheses as fixed effects, and (3) a model adding random effects for the relevant within-person predictor (e.g., deviations in estradiol). Significant differences between each model and the best-fitting previous model are clearly labeled in

each table. Random effects were only retained in further (e.g., mediation/moderation) models where the improvement in model fit was significant with their inclusion. When moderation hypotheses were tested, results of best-fitting models up to that point were transposed to new tables for ease of model comparison.

Specific Hypothesis Tests

The primary hypothesis was that increases in conception probability and estradiol would be associated with increased expression of BPD symptoms. At the daily level, this hypothesis was tested in multilevel Poisson regression models with daily assessments at level one and women at level two; daily scores on the PAI-BOR were regressed on conception probability. At the weekly level, the hypothesis was tested in multilevel Poisson regression models with weekly assessments at level one and women at level two; weekly scores on the PAI-BOR, the BSL-23, and the MSI-BPD were regressed on average estradiol and weekly deviation in estradiol.

Moderation by Trait BPD Symptoms. The second hypothesis concerned the moderation of these effects by trait levels of BPD symptoms as measured by (1) the average of weekly PAI-BOR total score and (2) the number of SCID-II criteria met in the diagnostic interview. At the daily level, I tested this hypothesis in two multilevel Poisson regression models: (1) regressing daily PAI-BOR scores on conception probability, average weekly PAI-BOR total score, and their interaction, and (2) regressing daily PAI-BOR scores on conception probability, number of SCID-II criteria met during the diagnostic interview, and their interaction. At the weekly level, I tested this hypothesis in two multilevel Poisson regression models for each of the three measures of BPD symptoms (PAI-BOR, BSL-23, and MSI-BPD): (1) regressing weekly symptoms on

average estradiol and weekly deviation in estradiol, average weekly PAI-BOR total score, and the interactions of average weekly PAI-BOR total score with both average estradiol and weekly deviation in estradiol, and (2) regressing weekly symptoms on average estradiol and weekly deviation in estradiol, number of SCID-II criteria met during the diagnostic interview, and the interactions of number of SCID-II criteria with both average estradiol and weekly deviation in estradiol. Because trait BPD as defined here is a heterogeneous construct, further moderation analyses were also conducted in which aspects of FFM personality and different types of childhood maltreatment were substituted as moderators of the effects of estradiol.

Mediation by Felt Acceptance, Impulsivity, and Self-Control. The third hypothesis concerned the mediation of conception probability and estradiol effects on symptoms by felt acceptance, multiple facets of impulsivity, and self-control. A primary step in testing mediation is to test for effects of the focal predictors on outcomes of interest; this was accomplished during tests of hypothesis 1 and 2. A second step is to test the effects of these focal predictors on the mediators. A third step is to test the impact of the mediator on the outcomes of interest. Finally, given significant findings in these three tests, a final test used estimates and standard errors for the A and B paths to generate a 95% confidence interval for the indirect effect of the focal predictor (e.g., weekly deviations in estradiol) on the outcome of interest (e.g., BPD symptoms) via the mediator (e.g., felt acceptance) using the RMediation program (Tofighi & MacKinnon, 2011). Where moderation hypotheses were supported, mediational analyses were also conducted to determine whether felt acceptance, UPPS impulsivity, or self-control mediated these

moderation effects. Models included in testing the second and third steps discussed above will now be described in detail for each of the three proposed mediators.

The first proposed mediator was felt acceptance. At the daily level, this was tested in two models: (1) regressing daily felt acceptance on conception probability, and (2) regressing daily PAI-BOR scores on average levels of felt acceptance and daily deviations in felt acceptance. At the weekly level, this was tested in similar models: (1) regressing weekly scores on the Social Evaluation subscale of the SSE scale on average estradiol and weekly deviations in estradiol, and (2) regressing weekly BPD symptoms (on the PAI-BOR, the BSL-23, and the MSI) on average scores on the Social Evaluation subscale of the SSE scale and weekly deviations in scores on the Social Evaluation subscale of the SSE scale. Where moderation models were significant, tests of mediated moderation were also carried out. The second proposed mediator was impulsivity. At the weekly level, this was tested in the following models: (1) regressing weekly scores on each subscale of the UPPS-P on average estradiol and weekly deviations in estradiol, and (2) regressing weekly BPD symptoms (on the PAI-BOR, the BSL-23, and the MSI-BPD) on average scores on each subscale of the UPPS-P and weekly deviations in scores on each subscale of the UPPS-P. The third proposed mediator was self-control. At the weekly level, this was tested in the following models: (1) regressing weekly scores on the BSCS on average estradiol and weekly deviations in estradiol, and (2) regressing weekly BPD symptoms (on the PAI-BOR, the BSL-23, and the MSI) on average of all weekly scores on the BSCS and weekly deviations in scores on the BSCS.

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Chapter Three: Results

Observations at the Daily and Weekly Level

At the daily level, there were 729 observations, indicating an average of 18.22 diaries completed per participant. At the weekly level, there were no missed lab sessions that were unable to be rescheduled; therefore, the maximum number of 160 data points were collected. This means that all 40 participants provided data at exactly four lab visits.

Descriptive and Correlational Analyses

Before carrying out hypothesis tests, I examined the zero-order correlations between a woman's average levels of estradiol, a woman's average scores on daily and weekly BPD measures, and between-person variables (e.g., SCID criteria met, FFM personality domains, and types of childhood maltreatment). Zero-order correlations, means, standard deviations, and internal consistencies for these variables can be found in Table 2. Unexpectedly, higher average levels of estradiol were significantly negatively correlated with Extraversion. Additional post-hoc correlations of average estradiol with the five facet items for Extraversion on the FFM-RF revealed that this significant association was accounted for by significant negative correlations between average estradiol and the warmth ($r = -.36, p = .01$) and positive emotions ($r = -.32, p = .02$) facets. In addition, average levels of estradiol were associated with higher levels of both physical and emotional abuse; these correlations were also not anticipated, and the reasons for these associations are unclear. Average levels of estradiol were uncorrelated with any measure of BPD.

Table 2

Zero-order correlations among between-person measures, average estradiol, and average daily and weekly measures of BPD

Variable	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16
1 Average Estradiol	-															
2 Neuroticism	.12	-														
3 Agreeableness	-.02	-.24	-													
4 Conscientiousness	.11	-.19	.07	-												
5 Extraversion	-.30*	-.23	.21	.21	-											
6 Openness	.03	.12	.33*	-.05	.27*	-										
7 Emotional Abuse	.25*	.27*	-.14	.05	-.02	.14	-									
8 Emotional Neglect	.02	.32*	-.21	-.13	-.07	.15	.83*	-								
9 Physical Abuse	.26*	.22	-.17	.10	-.14	.09	.81*	.70*	-							
10 Physical Neglect	.13	.34*	-.09	-.10	-.04	.28*	.70*	.78*	.78*	-						
11 Sexual Abuse	.04	.18	-.26*	.11	.0001	.01	.52*	.39*	.55*	.61*	-					
12 SCID-II Criteria Met	-.13	.41*	-.10	-.18	.06	.15	.38*	.42*	.18	.26*	.13	-				
13 Avg PAI-BOR (D)	-.08	.59*	-.29*	-.29*	-.04	.21	.21	.34*	.06	.19	.006	.64*	-			
14 Avg PAI-BOR (W)	-.002	.59*	-.31*	-.26*	-.08	.17	.33*	.38*	.17	.26*	.25*	.66*	.95*	-		
15 Avg BSL-23 (W)	.07	.59*	-.34*	-.10	-.09	.13	.54*	.62*	.42*	.47*	.26*	.78*	.82*	.88*	-	
16 Avg MSI (W)	-.10	.49*	-.31*	-.19	-.03	.23	.22	.40*	.15	.25*	.03	.67*	.88*	.87*	.87*	-
Mean	3.39	2.40	3.55	3.57	3.71	3.90	.13	1.55	1.26	1.22	1.28	1.32	.59	.70	.48	2.08
(SD)	(.08)	(.12)	(.11)	(.09)	(.12)	(.10)	(.37)	(.15)	(.11)	(.09)	(.14)	(.33)	(.06)	(.04)	(.47)	(2.26)
Cronbach's α	-	.75	.75	.71	.77	.81	.85	.93	.86	.81	.95	-	.89	.91	.93	.77

Note. * $p < .05$. Estradiol units are pg/mL.

Consistent with dimensional FFM personality models of BPD (Widiger & Mullins-Sweatt, 2009), Neuroticism was positively correlated with all measures of BPD. Agreeableness was negatively correlated with all measures of BPD except the SCID-II interview, and Conscientiousness was negatively associated with two of the five measures of BPD; however, Openness was not associated with any measure of BPD. Neuroticism was also positively correlated with higher scores on some facets of the CTQ (Emotional Abuse, Emotional Neglect, and Physical Neglect). Consistent with Linehan's biosocial theory of BPD, subscales of the CTQ were generally but inconsistently associated with higher BPD features; Emotional Abuse, Emotional Neglect, Physical Neglect, and Sexual Abuse showed the most consistent positive associations with BPD scores. Notably, scores on the Sexual Abuse subscale were both positively associated with higher average scores on the weekly PAI-BOR and BSL-23 and negatively correlated with Agreeableness. Intercorrelations among average daily, average weekly, and number of SCID-II criteria met were all significant in expected directions, suggesting good convergent validity among these various ways of measuring BPD (all r 's between .69 and .95). All subscales of the CTQ were also highly intercorrelated in expected directions. Internal consistencies were all acceptable to excellent.

Examination of the SCID-II criteria met variable revealed that, on average, individuals endorsed a low number (1.33) of BPD criteria during the interview. Notably, though all of the other measures of BPD were significantly correlated with number of SCID-II criteria met, significance testing for the differences between correlations revealed that the correlation between the BSL-23 and number of SCID-II criteria met ($r = .78$) was significantly greater than the correlations between number of SCID-II criteria

met and average weekly PAI-BOR ($t(37) = -2.38, p = .002$), average daily PAI-BOR ($t(37) = -2.25, p = .03$), and the MSI-BPD ($t(37) = -2.09, p = .04$). Therefore, although all of the self-report measures were highly positively correlated with BPD symptoms as measured using the SCID-II diagnostic interview, the BSL-23 may be considered a stronger indicator of interview-confirmed BPD symptoms in the present sample.

Testing Hypothesis 1: Ovulation, Estradiol, and BPD Symptom Expression

Daily Analyses. It was predicted that higher levels of conception probability would predict greater expression of BPD symptoms at the daily level. Results of multilevel Poisson regression models predicting each of the four PAI-BOR subscales and the PAI-BOR total scale from conception probability are presented in Table 3.

Results of these models do not support Hypothesis 1; conception probability did not significantly predict any PAI-BOR subscale or the total scale. However, comparing Model 2 (a model with a fixed effect of conception probability) to Model 3 (a model with a random effect specified for conception probability) revealed significant improvement in model fit when predicting each of the PAI-BOR subscales and the total. This indicates that the influence of conception probability (ovulation) on BPD symptoms differs between individuals (i.e., there is a moderator).

Weekly Analyses. It was also predicted that increases in estradiol (which are typically associated with ovulation) would predict greater expression of BPD symptoms at the weekly level. Results of models predicting each of the four PAI-BOR subscales and total score, the MSI-BPD, and BSL-23 from average estradiol and weekly deviations in estradiol are presented in Table 4.

Table 3

Multilevel Poisson Regression Models Predicting Daily PAI-BOR Scores from Daily Conception Probability Values

Parameter	Model 1 (null)	Model 2 (fixed CP slope)	Model 3 (random CP slope)
<u>Dependent Variable: PAI-BOR Total Score</u>			
		Fixed Effects	
Intercept	-.62* (.10)	-.62* (.10)	-.62* (.10)
Conception Probability		.17 (.59)	.20 (.98)
		Random Parameters	
Intercept	.38* (.09)	.37* (.09)	.39* (.09)
Conception Probability			18.25* (7.30)
Residual	.13* (.006)	.13* (.007)	.12* (.006)
-2 Restricted Log Pseudo-likelihood	1348.35	1224.61†	1185.02†
<u>Dependent Variable: PAI-BOR Affective Instability Subscale</u>			
		Fixed Effects	
Intercept	-.68* (.12)	-.68* (.13)	-.67* (.13)
Conception Probability		.13 (.93)	-.24 (1.29)
		Random Parameters	
Intercept	.59* (.15)	.60* (.15)	.60* (.15)
Conception Probability			20.71* (12.12)
Residual	.33* (.01)	.13* (.93)	.31* (.01)
-2 Restricted Log Pseudo-likelihood	2117.42	1915.66†	1903.81†
<u>Dependent Variable: PAI-BOR Identity Disturbance Subscale</u>			
		Fixed Effects	
Intercept	-.55* (.10)	-.53* (.10)	-.53* (.11)
Conception Probability		-.56 (.65)	-.71 (.99)
		Random Parameters	
Intercept	.42* (.10)	.43* (.10)	.45* (.11)
Conception Probability			15.48* (7.39)
Residual	.17* (.009)	.17* (.009)	.16* (.009)
-2 Restricted Log Pseudo-likelihood	1521.40	1376.87†	1357.97†

Table Continued on Next Page

Dependent Variable: PAI-BOR Negative Relationships Subscale			
		Fixed Effects	
Intercept	-.48* (.10)	-.49* (.11)	-.50* (.11)
Conception Probability		.60 (.68)	.73 (1.04)
		Random Parameters	
Intercept	.44* (.10)	.44* (.11)	.45* (.11)
Conception Probability			16.59* (8.81)
Residual	.22* (.01)	.23* (.01)	.21* (.01)
-2 Restricted Log Pseudo-likelihood	1622.09	1487.02†	1472.61†
Dependent Variable: PAI-BOR Self-Harm Subscale			
		Fixed Effects	
Intercept	-1.13* (.15)	-1.11* (.15)	-1.10* (.15)
Conception Probability		.70 (1.17)	.36 (.83)
		Random Parameters	
Intercept	.87* (.24)	.77* (.22)	.77* (.23)
Conception Probability			48.07* (23.80)
Residual	.34* (.01)	.34* (.01)	.31* (.01)
-2 Restricted Log Pseudo-likelihood	2551.02	2264.19†	2242.93†

Note. Standard errors are in parentheses. CP = Daily Conception Probability.

* $p < .05$.

†Change in -2 Restricted Log Pseudo-likelihood over the previous model is significant at $p < .05$.

Table 4

Multilevel Poisson Regression Models Predicting Weekly BPD Symptoms from Average Levels of Estradiol and Weekly Deviations in Estradiol

Parameter	Model 1 (null)	Model 2 (fixed deviation slope)	Model 3 (random deviation slope)
<u>Dependent Variable: PAI-BOR Total Score</u>			
		Fixed Effects	
Intercept	-.50* (.10)	-.50* (.10)	-.50* (.10)
Average Weekly Estradiol Deviations in Estradiol		-.02 (.10) .001 (.04)	-.02 (.09) .001 (.05)
		Random Parameters	
Intercept	.39* (.09)	.40* (.10)	.40* (.10)
Deviations in Estradiol			.03 (.06)
Residual	.08* (.01)	.08* (.01)	.08* (.02)
-2 Restricted Log Pseudo-likelihood	240.55	239.27	240.27
<u>Dependent Variable: PAI-BOR Affective Instability Subscale</u>			
		Fixed Effects	
Intercept	-.60* (.15)	-.61* (.15)	-.61* (.15)
Average Weekly Estradiol Deviations in Estradiol		-.10 (.15) -.05 (.06)	-.10 (.14) -.05 (.06)
		Random Parameters	
Intercept	.83* (.23)	.85* (.23)	.85* (.23)
Deviations in Estradiol			.04 (.03)
Residual	.16* (.02)	.16* (.02)	.16* (.02)
-2 Restricted Log Pseudo-likelihood	370.17	370.09	369.89
<u>Dependent Variable: PAI-BOR Identity Disturbance Subscale</u>			
		Fixed Effects	
Intercept	-.30* (.11)	-.30* (.11)	-.30* (.11)
Average Weekly Estradiol Deviations in Estradiol		-.03 (.11) -.00069 (.05)	-.04 (.11) .01 (.06)
		Random Parameters	
Intercept	.45* (.11)	.46* (.11)	.47* (.11)
Deviations in Estradiol			.02 (.04)
Residual	.12* (.01)	.12* (.01)	.11* (.01)
-2 Restricted Log Pseudo-likelihood	274.93	273.58	273.18

Table Continued on Next Page

<u>Dependent Variable: PAI-BOR Negative Relationships Subscale</u>			
		Fixed Effects	
Intercept	- .41* (.12)	- .42 (.12)	- .42 (.12)
Average Weekly Estradiol		- .01 (.10)	- .01 (.10)
<i>Deviations in Estradiol</i>		.03 (.15)	.03 (.15)
		Random Parameters	
Intercept	.53* (.14)	.55* (.16)	.55* (.16)
Deviations in Estradiol			.01 (.07)
Residual	.16* (.02)	.17* (.02)	.17* (.02)
-2 Restricted Log Pseudo-likelihood	332.16	332.10	330.77
<u>Dependent Variable: PAI-BOR Self-Harm Subscale</u>			
		Fixed Effects	
Intercept	-1.03* (.11)	-1.03* (.11)	-1.04* (.11)
Average Weekly Estradiol		.01 (.11)	.01 (.11)
<i>Deviations in Estradiol</i>		.02 (.09)	.02 (.09)
		Random Parameters	
Intercept	.35* (.12)	.37* (.12)	.37* (.12)
Deviations in Estradiol			.002 (.05)
Residual	.18* (.02)	.18* (.02)	.18* (.02)
-2 Restricted Log Pseudo-likelihood	407.23	411.88	411.86
<u>Dependent Variable: Borderline Symptom Checklist—23 (BSL-23)</u>			
		Fixed Effects	
Intercept	2.05* (.16)	2.04* (.17)	2.04* (.18)
Average Weekly Estradiol		.003 (.17)	.003 (.16)
<i>Deviations in Estradiol</i>		-.11* (.03)	-.11* (.02)
		Random Parameters	
Intercept	1.06* (.29)	1.11* (.30)	1.11* (.30)
Deviations in Estradiol			.004 (.06)
Residual	2.34* (.31)	2.26* (.30)	2.26* (.30)
-2 Restricted Log Pseudo-likelihood	379.72	371.22	378.22

Table Continued on Next Page

Dependent Variable: McLean Screening Instrument for BPD (MSI-BPD)			
		Fixed Effects	
Intercept	.17 (.22)	.17 (.22)	.17 (.22)
Average Weekly Estradiol		-.03 (.22)	-.03 (.22)
<i>Deviations in Estradiol</i>		-.08 (.07)	-.08 (.07)
		Random Parameters	
Intercept	1.80* (.54)	1.86* (.56)	1.87* (.56)
Deviations in Estradiol			-.03 (.22)
Residual	.68* (.09)	.69* (.09)	-.08 (.07)
-2 Restricted Log Pseudo-likelihood	481.04	480.91	480.02

Note. Standard errors are in parentheses. A dashed line indicates that the model did not converge, likely due to nonsignificance of random effects included.

* $p < .05$.

†Change in -2 Restricted Log Pseudo-likelihood over the previous model is significant at $p < .05$.

For each of the models except the one predicting the BSL-23, were no significant fixed or random effects of either average levels of estradiol or weekly deviations in estradiol. There was a significant fixed effect of deviations in estradiol such that higher-than-usual levels of estradiol were associated with lower scores on the BSL-23.

Testing Hypothesis 2: Moderation of Cycle Effects by Trait BPD

Daily Analyses. It was predicted that the effect of daily conception probability values on expression of BPD symptoms as measured by the PAI-BOR would be moderated by (1) average weekly values of the total PAI-BOR scale and (2) number of BPD criteria met during the SCID-II diagnostic interview. Specifically, it was hypothesized that women with generally higher levels of BPD symptoms (as measured by average of the four extended weekly assessments *or* number of BPD criteria met during the SCID-II interview) would show a stronger positive association between daily conception probability values and BPD symptoms. Results of models predicting each of the four PAI-BOR subscales and total scale from conception probability, average of weekly PAI-BOR total score assessments, and their interaction are presented in Table 5. In each case, Model 3 information was transplanted from Table 3 for ease of comparing Model 3 (in which a random effect of conception probability was identified) to Model 4, in which the moderator is included.

Though models including the moderator provided significantly better model fit than a model including only a random effect of conception probability, only one of these models provided evidence that trait BPD moderates the impact of daily conception probability values on daily BPD symptoms. There was a significant interactive effect of average daily total PAI-BOR scores and conception probability in the opposite of the

Table 5

Multilevel Poisson Regression Models Predicting Daily PAI-BOR Scores from the Interaction of Daily Conception Probability Values and Average of Weekly PAI-BOR Assessments (Trait BPD)

Parameter	Model 3 (random CP slope)	Model 4 (with moderator)
<u>Dependent Variable: Daily PAI-BOR Total Score</u>		
Fixed Effects		
Intercept	-.62* (.10)	-.65* (.04)
Conception Probability	.20 (.98)	.49 (.64)
Avg of Weekly PAI-BOR		.56* (.03)
<i>Avg of Weekly PAI-BOR*CP</i>		-.89* (.07)
Random Parameters		
Intercept	.39* (.09)	.03* (.01)
Conception Probability	18.25* (7.30)	16.48* (6.67)
Residual	.12* (.006)	.13* (.007)
-2 Restricted Log Pseudo-likelihood	1185.02	1146.20†
<u>Dependent Variable: Daily PAI-BOR Affective Instability Subscale</u>		
Fixed Effects		
Intercept	-.67* (.13)	-.70* (.07)
Conception Probability	-.24* (1.29)	-.43 (1.33)
Avg of Weekly PAI-BOR		.62* (.18)
<i>Avg of Weekly PAI-BOR*CP</i>		-.17 (.88)
Random Parameters		
Intercept	.60* (.15)	.15* (.05)
Conception Probability	20.71* (12.12)	19.12* (11.02)
Residual	.31* (.01)	.31* (.01)
-2 Restricted Log Pseudo-likelihood	1903.81	1849.49†
<u>Dependent Variable: Daily PAI-BOR Identity Disturbance Subscale</u>		
Fixed Effects		
Intercept	-.53* (.11)	-.56* (.06)
Conception Probability	-.71 (.99)	-.54 (.99)
Avg of Weekly PAI-BOR		.57* (.05)
<i>Avg of Weekly PAI-BOR*CP</i>		-1.11 (.89)
Random Parameters		
Intercept	.45* (.11)	.10* (.03)
Conception Probability	15.48* (7.39)	13.93* (6.82)
Residual	.16* (.009)	.16* (.009)
-2 Restricted Log Pseudo-likelihood	1357.97	1307.95†

Table Continued on Next Page

<u>Dependent Variable: Daily PAI-BOR Negative Relationships Subscale</u>		
	Fixed Effects	
Intercept	-.50* (.11)	-.52* (.06)
Conception Probability	.73 (1.04)	.97 (1.00)
Avg of Weekly PAI-BOR		.57* (.06)
<i>Avg of Weekly PAI-BOR*CP</i>		-1.16 (.91)
	Random Parameters	
Intercept	.45* (.11)	.11* (.03)
Conception Probability	16.59* (8.81)	12.79* (7.38)
Residual	.21* (.01)	.21* (.01)
-2 Restricted Log Pseudo-likelihood	1472.61	1424.62†
<u>Dependent Variable: Daily PAI-BOR Self-Harm Subscale</u>		
	Fixed Effects	
Intercept	-.62* (.10)	-1.12* (.12)
Conception Probability	.20 (.98)	.14 (1.77)
Avg of Weekly PAI-BOR		.55* (.11)
<i>Avg of Weekly PAI-BOR*CP</i>		-.25 (.87)
	Random Parameters	
Intercept	.77* (.23)	.43* (.13)
Conception Probability	48.07* (23.80)	46.84* (23.18)
Residual	.31* (.01)	.31* (.01)
-2 Restricted Log Pseudo-likelihood	2242.93	2216.45†

Note. Standard errors are in parentheses. CP = Daily Conception Probability.
 * $p < .05$.
 †Change in -2 Restricted Log Pseudo-likelihood over the previous model is significant at $p < .05$.

predicted direction: women with *lower* (-1 standard deviation) trait BPD symptoms showed increased daily BPD symptoms on days when conception probability was higher ($\gamma_{\text{LOWTRAITBPD*CP}} = .58, SE = .44, t(697) = 3.53, p = .0004$), whereas there was no effect of conception probability in women with *higher* (+1 standard deviation) trait levels of BPD ($\gamma_{\text{HIGHTRAITBPD*CP}} = -.05, SE = .26, t(697) = -.19, p = .84$). A graph depicting the interaction can be found in Figure 1.

Next, models were executed in which number of SCID-II criteria met was substituted for Average Weekly PAI-BOR scores as an alternative measure of trait BPD symptoms. Results of these models are presented in Table 6. The results from the random slope models (Model 3) were again transplanted from Table 3 for ease of model comparison. As with the majority of the PAI-BOR moderation models, the SCID-II criteria moderation models provided significantly better model fit than a model including only a random effect of conception probability; however, these models provided no evidence that trait BPD influences the impact of daily conception probability values on daily BPD symptoms.

Weekly Analyses. It was also predicted that the effect of weekly deviations in estradiol on BPD symptoms as measured by the PAI-BOR, the BSL-23, and the MSI-BPD would be moderated by (1) average weekly values of the total PAI-BOR scale and (2) number of criteria met on the SCID-II BPD module. Again, it was hypothesized that women with generally higher levels of BPD symptoms (as measured by average of the four extended weekly assessments *and* number of BPD criteria met during the SCID-II interview) would show stronger positive associations between weekly fluctuations in estradiol and BPD symptoms. Results of models predicting each of the four PAI-BOR

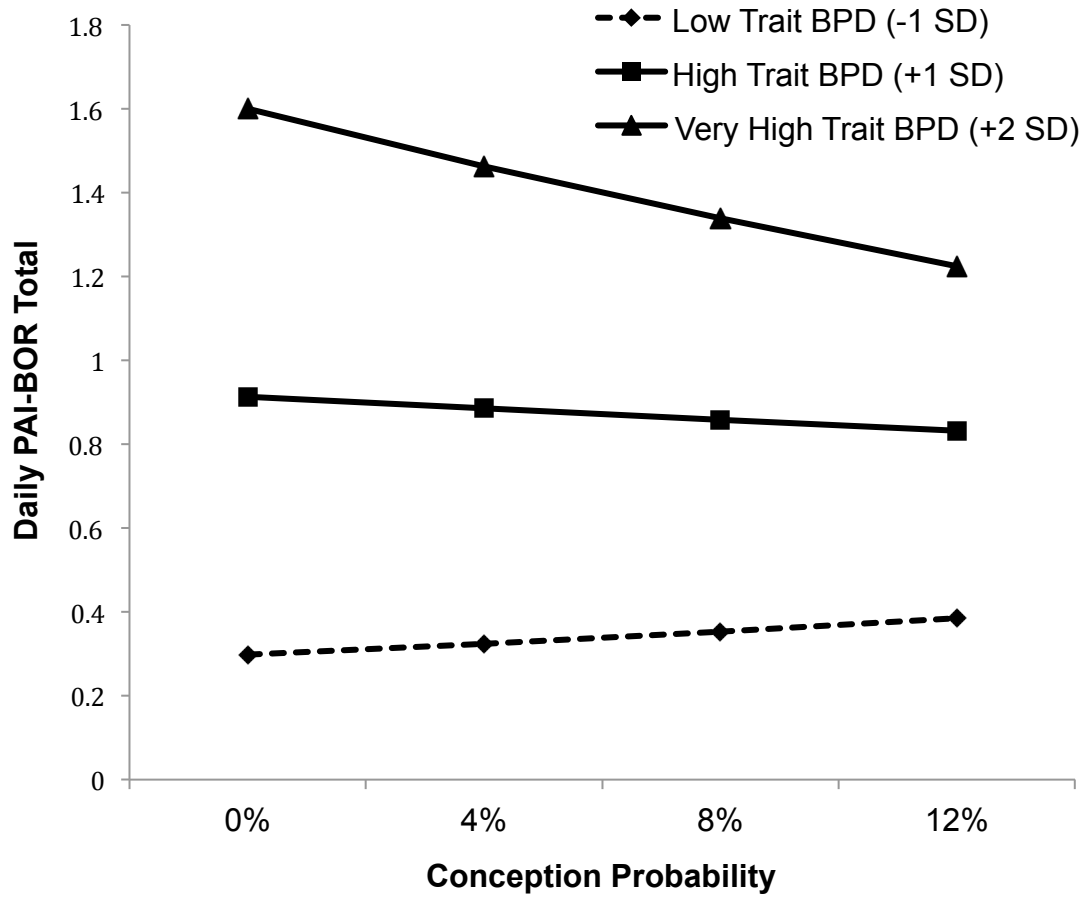


Figure 1. A graph of the interaction between trait BPD symptoms and daily conception probability predicting daily PAI-BOR total score.

Table 6

Multilevel Poisson Regression Models Predicting Daily PAI-BOR Scores from the Interaction of Daily Conception Probability Values and Number of SCID-II BPD Criteria Met (Trait BPD)

Parameter	Model 3 (random CP slope)	Model 4 (with moderator)
<u>Dependent Variable: PAI-BOR Total Score</u>		
Fixed Effects		
Intercept	-.62* (.10)	-.65*(.08)
Conception Probability	.20 (.98)	.24 (.99)
SCID-II BPD Criteria Met		.39* (.08)
<i>SCID-II BPD Criteria Met*CP</i>		-.56 (.89)
Random Parameters		
Intercept	.39* (.09)	.24* (.06)
Conception Probability	18.25* (7.30)	18.23* (7.22)
Residual	.12* (.006)	.12* (.006)
-2 Restricted Log Pseudo-likelihood	1185.02	1164.11†
<u>Dependent Variable: PAI-BOR Affective Instability Subscale</u>		
Fixed Effects		
Intercept	-.67* (.13)	-.70* (.11)
Conception Probability	-.24 (1.29)	-.47 (1.34)
SCID-II BPD Criteria Met		.46* (.10)
<i>SCID-II BPD Criteria Met*CP</i>		.10 (1.17)
Random Parameters		
Intercept	.60* (.15)	.38* (.10)
Conception Probability	20.71* (12.12)	21.46* (12.34)
Residual	.31* (.01)	.31* (.01)
-2 Restricted Log Pseudo-likelihood	1903.81	1883.70†
<u>Dependent Variable: PAI-BOR Identity Disturbance Subscale</u>		
Fixed Effects		
Intercept	-.53* (.11)	-.56* (.09)
Conception Probability	-.71 (.99)	-.54 (1.01)
SCID-II BPD Criteria Met		.40* (.09)
<i>SCID-II BPD Criteria Met*CP</i>		-.51 (.91)
Random Parameters		
Intercept	.45* (.11)	.30* (.07)
Conception Probability	15.48* (7.39)	15.86* (7.34)
Residual	.16* (.009)	.16* (.009)
-2 Restricted Log Pseudo-likelihood	1357.97	1336.18†

Table Continued on Next Page

Dependent Variable: Negative Relationships Subscale

	Fixed Effects	
Intercept	-.50* (.11)	-.52* (.09)
Conception Probability	.73 (1.04)	.77 (1.06)
SCID-II BPD Criteria Met		.39* (.09)
<i>SCID-II BPD Criteria Met*CP</i>		-.53 (.95)
	Random Parameters	
Intercept	.45* (.11)	.31* (.08)
Conception Probability	16.59* (8.81)	16.88* (8.85)
Residual	.21* (.01)	.21* (.01)
-2 Restricted Log Pseudo-likelihood	1472.61	1457.30†

Dependent Variable: Self-Harm Subscale

	Fixed Effects	
Intercept	-.62* (.10)	-1.14* (.14)
Conception Probability	.20 (.98)	.40 (1.75)
SCID-II BPD Criteria Met		.33* (.14)
<i>SCID-II BPD Criteria Met*CP</i>		-1.32 (1.60)
	Random Parameters	
Intercept	.77* (.23)	.71* (.21)
Conception Probability	48.07* (23.80)	47.84* (23.67)
Residual	.31* (.01)	.31* (.01)
-2 Restricted Log Pseudo-likelihood	2242.93	2234.05†

Note. Standard errors are in parentheses. CP = Daily Conception Probability.

* $p < .05$.

†Change in -2 Restricted Log Pseudo-likelihood over the previous model is significant at $p < .05$.

subscales and the PAI-BOR total scale, the BSL-23, and the MSI-BPD from average estradiol, deviations in estradiol, average of weekly PAI-BOR total score assessments, and the interactions of trait BPD and each estradiol variable are presented in Table 7. In each case, the null model (Model 1) was transplanted from Table 4 for ease of comparison with Model 4, in which the trait BPD moderator is included.

For each outcome, model fit was significantly improved with the inclusion of the fixed effects. Results presented in Table 7 reveal several significant interactive effects of trait BPD and fluctuations in estradiol, though the interactive effects are in the opposite of the predicted direction. In the model predicting the total score of the PAI-BOR, women with *lower* levels of trait BPD showed a positive association between higher-than-usual levels of estradiol and BPD symptoms ($\gamma_{\text{LOWTRAITBPD*ESTRADIOLDEVIATION}} = .14$, $SE = .08$, $t(117) = 2.70$, $p = .007$), while higher-than-usual levels in estradiol were associated with lower BPD symptoms among women with *higher* levels of trait BPD ($\gamma_{\text{HIGHTRAITBPD*ESTRADIOLDEVIATION}} = -.13$, $SE = .03$, $t(117) = 4.33$, $p < .0001$). A graph of the interaction can be found in Figure 2.

Results for several of the other scales tended to follow a similar pattern of results in which BPD symptoms tended to be reduced at higher-than-usual estradiol among those with high trait levels of BPD but tended to be increased with higher-than-usual estradiol among those with low trait levels of BPD symptoms (see Figure 3-5 for graphs depicting significant interactions). In the model predicting Identity Disturbance, there was a significant association between higher-than-usual estradiol and decreased symptoms among women *high* in trait BPD ($\gamma_{\text{HIGHTRAITBPD*ESTRADIOLDEVIATION}} = -.18$, $SE = .06$, $t(117) = 1.99$, $p = .04$), but there was not a significant association between fluctuations in

Table 7

Multilevel Poisson Regression Models Predicting Weekly BPD Symptoms from the Interaction of Average of Weekly PAI-BOR Assessments (Trait BPD) with Average Estradiol and Weekly Deviations in Estradiol

Parameter	Model 1 (null)	Model 4 (with moderator)
<u>Dependent Variable: PAI-BOR Total Score</u>		
Fixed Effects		
Intercept	-.50* (.10)	-.51* (.03)
Avg Estradiol		-.01 (.03)
Weekly Deviation in Estradiol		.03 (.05)
Avg of Weekly PAI-BOR		.53* (.02)
Avg of Weekly PAI-BOR*Avg Estradiol		.003 (.02)
<i>Avg of Weekly PAI-BOR*Estradiol Deviation</i>		-.10* (.05)
Random Parameters		
Intercept	.39* (.09)	.001 (.007)
Weekly Deviation in Estradiol		
Residual	.08* (.01)	.08* (.01)
-2 Restricted Log Pseudo-likelihood	240.55	161.70†
<u>Dependent Variable: PAI-BOR Affective Instability Subscale</u>		
Fixed Effects		
Intercept	-.60* (.15)	-.62* (.08)
Avg Estradiol		-.09 (.08)
Weekly Deviation in Estradiol		.001 (.07)
Avg of Weekly PAI-BOR		.69* (.07)
Avg of Weekly PAI-BOR*Avg Estradiol		.02 (.07)
<i>Avg of Weekly PAI-BOR*Estradiol Deviation</i>		-.13* (.03)
Random Parameters		
Intercept	.83* (.23)	.16* (.06)
Weekly Deviation in Estradiol		
Residual	.16* (.02)	.16* (.02)
-2 Restricted Log Pseudo-likelihood	370.17	328.42†

Table Continued on Next Page

Dependent Variable: PAI-BOR Identity Disturbance Subscale

	Fixed Effects	
Intercept	-.30* (.11)	-.33* (.05)
Avg Estradiol		-.03 (.05)
Weekly Deviation in Estradiol		.04 (.05)
Avg of Weekly PAI-BOR		.58* (.04)
Avg of Weekly PAI-BOR*Avg Estradiol		.01 (.04)
<i>Avg of Weekly PAI-BOR*Estradiol Deviation</i>		-.13* (.05)
	Random Parameters	
Intercept	.45* (.11)	.05* (.02)
Weekly Deviation in Estradiol		
Residual	.12* (.01)	.12* (.01)
-2 Restricted Log Pseudo-likelihood	274.93	223.86†

Dependent Variable: PAI-BOR Negative Relationships Subscale

	Fixed Effects	
Intercept	-.41* (.12)	-.42* (.06)
Avg Estradiol		-.01 (.06)
Weekly Deviation in Estradiol		.06 (.06)
Avg of Weekly PAI-BOR		.57* (.05)
Avg of Weekly PAI-BOR*Avg Estradiol		.04 (.05)
<i>Avg of Weekly PAI-BOR*Estradiol Deviation</i>		-.07 (.07)
	Random Parameters	
Intercept	.53* (.14)	.07* (.03)
Weekly Deviation in Estradiol		
Residual	.16* (.02)	.16* (.02)
-2 Restricted Log Pseudo-likelihood	332.16	288.91†

Dependent Variable: PAI-BOR Self-Harm Subscale

	Fixed Effects	
Intercept	-1.03* (.11)	-1.05* (.09)
Avg Estradiol		.06 (.09)
Weekly Deviation in Estradiol		.04 (.09)
Avg of Weekly PAI-BOR		.39* (.08)
Avg of Weekly PAI-BOR*Avg Estradiol		-.15* (.06)
<i>Avg of Weekly PAI-BOR*Estradiol Deviation</i>		-.10 (.10)
	Random Parameters	
Intercept	.35* (.12)	.17* (.08)
Weekly Deviation in Estradiol		
Residual	.18* (.02)	.19* (.02)
-2 Restricted Log Pseudo-likelihood	407.23	402.96†

Table Continued on Next Page

Dependent Variable: Borderline Symptom Checklist—23 (BSL-23)		
	Fixed Effects	
Intercept	2.05* (.16)	2.02* (.10)
Avg Estradiol		.03 (.10)
Weekly Deviation in Estradiol		-.07 (.06)
Avg of Weekly PAI-BOR		.77* (.09)
Avg of Weekly PAI-BOR*Avg Estradiol		-.03 (.09)
<i>Avg of Weekly PAI-BOR*Estradiol Deviation</i>		-.13* (.05)
	Random Parameters	
Intercept	1.06* (.29)	.32* (.10)
Weekly Deviation in Estradiol		
Residual	2.34* (.31)	2.24* (.30)
-2 Restricted Log Pseudo-likelihood	379.72	342.77†
Dependent Variable: McLean Screening Instrument for BPD (MSI-BPD)		
	Fixed Effects	
Intercept	.17 (.22)	.17 (.14)
Avg Estradiol		-.02 (.14)
Weekly Deviation in Estradiol		-.05 (.09)
Avg of Weekly PAI-BOR		.96* (.13)
Avg of Weekly PAI-BOR*Avg Estradiol		-.11 (.12)
<i>Avg of Weekly PAI-BOR*Estradiol Deviation</i>		-.06 (.10)
	Random Parameters	
Intercept	1.80* (.54)	.56* (.20)
Weekly Deviation in Estradiol		
Residual	.68* (.09)	.72* (.09)
-2 Restricted Log Pseudo-likelihood	481.04	450.59†

Note. Standard errors are in parentheses.

* $p < .05$.

†Change in -2 Restricted Log Pseudo-likelihood over the previous model is significant at $p < .05$.

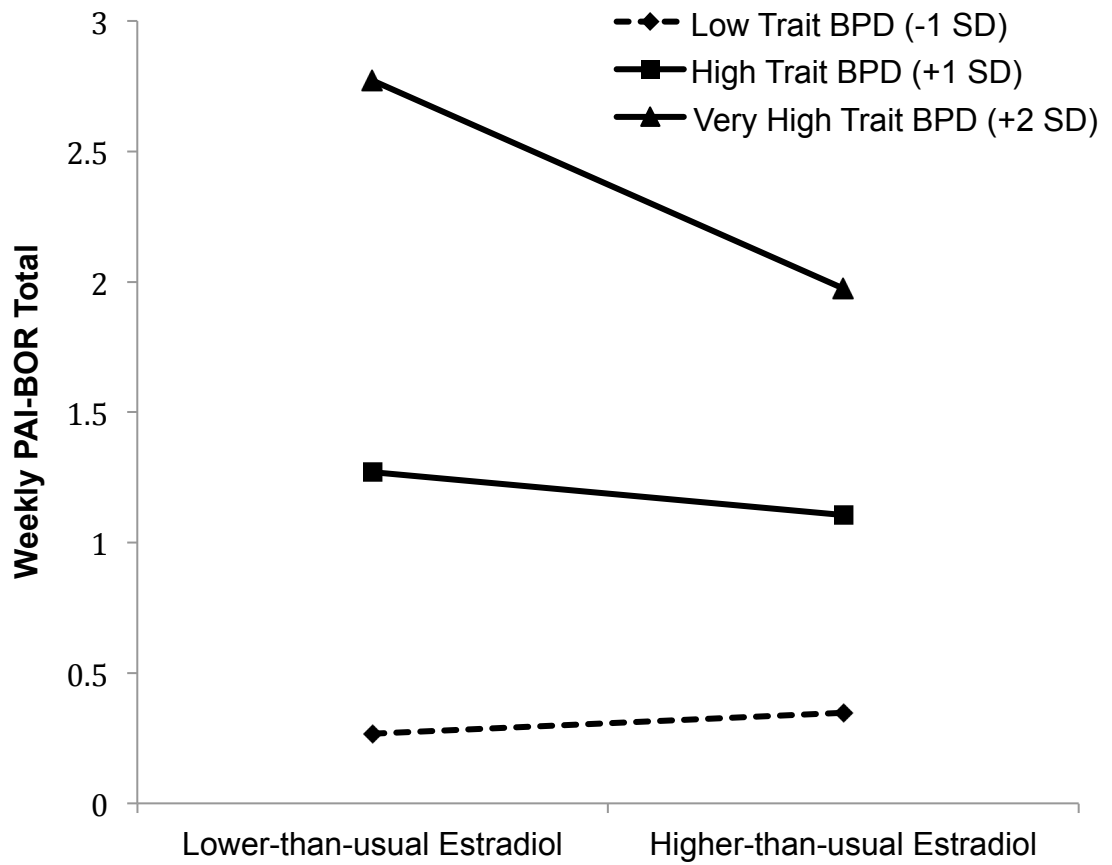


Figure 2. A graph of the interaction between trait BPD symptoms and *deviation* in estradiol predicting weekly PAI-BOR total score.

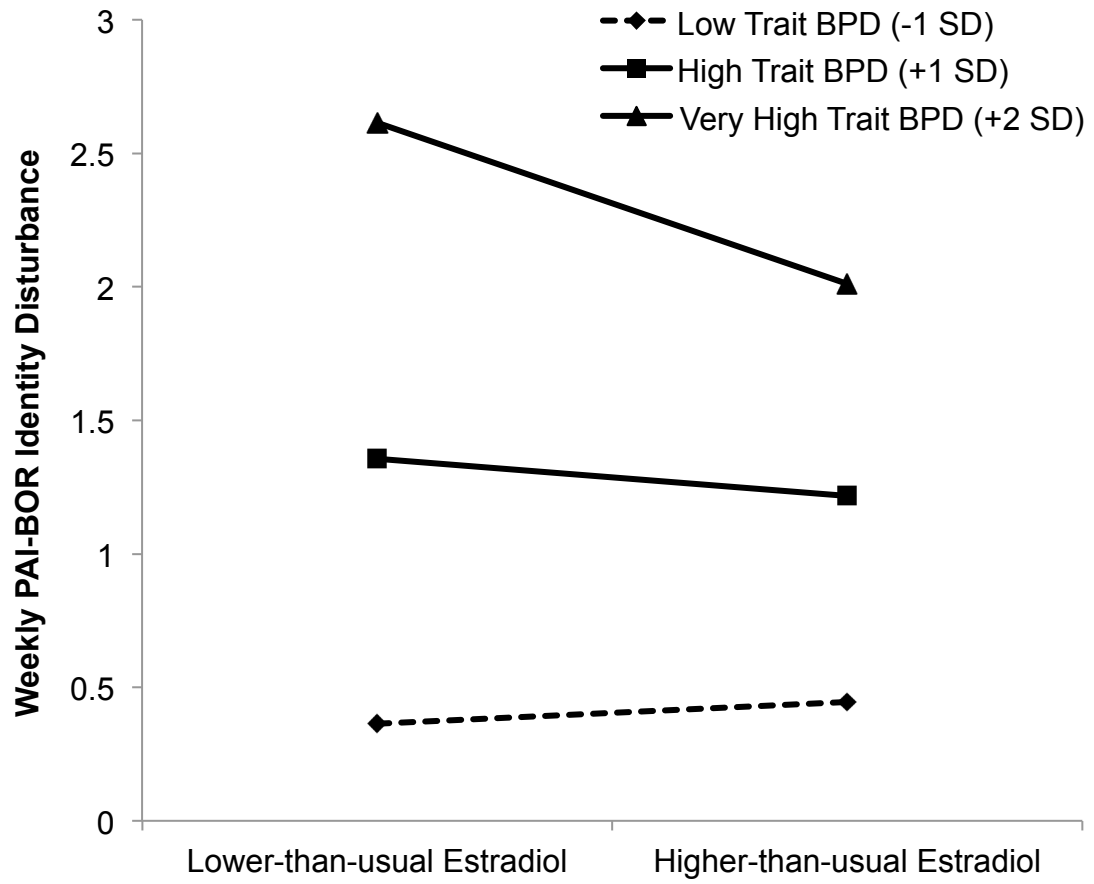


Figure 3. A graph of the interaction between trait BPD symptoms and *deviation* in estradiol predicting weekly PAI-BOR Identity Disturbance subscale score.

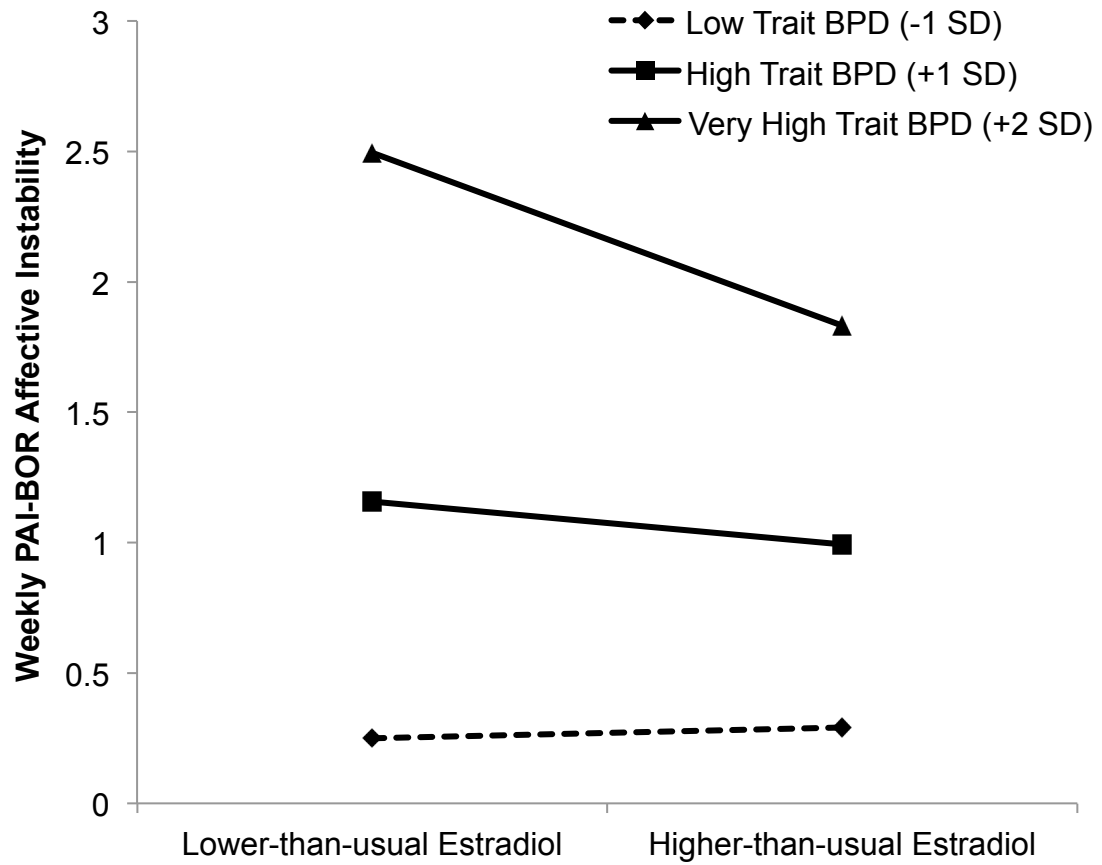


Figure 4. A graph of the interaction between trait BPD symptoms and *deviation* in estradiol predicting weekly PAI-BOR Affective Instability subscale score.

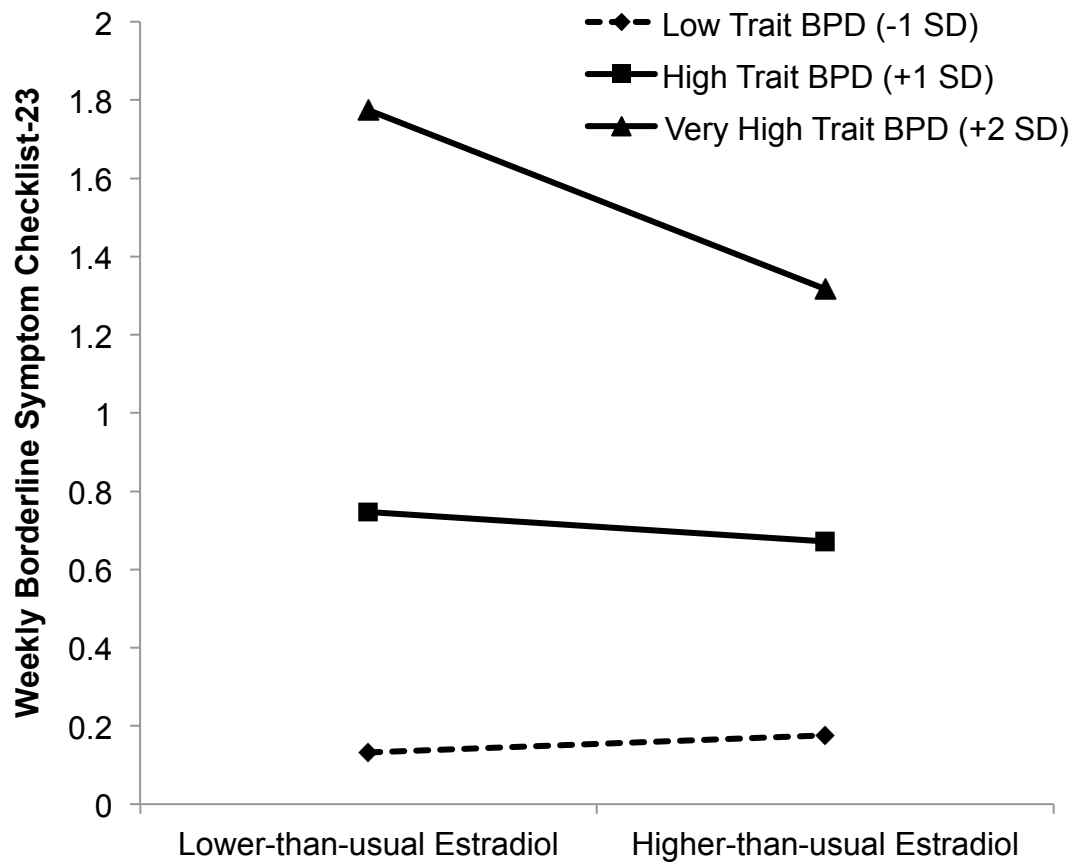


Figure 5. A graph of the interaction between trait BPD symptoms and *deviation* in estradiol predicting weekly BSL-23 score.

estradiol and symptom expression among women *low* in trait BPD symptoms ($\gamma_{\text{LOWTRAITBPD*ESTRADIOLDEVIATION}} = .08, SE = .06, t(117) = 1.39, p = .16$). In the model predicting the Affective Instability subscale, there was a significant effect of higher-than-usual estradiol predicting lower Affective Instability among women *higher* in trait BPD ($\gamma_{\text{HIGHTRAITBPD*ESTRADIOLDEVIATION}} = -.13, SE = .04, t(117) = -3.25, p = .0007$), but there was no significant effect of higher-than-usual estradiol on Affective Instability among women with *lower* trait BPD ($\gamma_{\text{LOWTRAITBPD*ESTRADIOLDEVIATION}} = .13, SE = .11, t(117) = 1.18, p = .29$). There was also a significant interaction between average scores on the PAI-BOR total and fluctuations in estradiol predicting scores on the BSL-23. Among women *higher* in trait BPD, higher-than-usual levels of estradiol were associated with decreased BPD symptoms ($\gamma_{\text{HIGHTRAITBPD*ESTRADIOLDEVIATION}} = -.21, SE = .07, t(117) = -2.72, p = .003$). However, among women *lower* in trait BPD, higher-than-usual estradiol was not associated with BPD symptom expression ($\gamma_{\text{LOWTRAITBPD*ESTRADIOLDEVIATION}} = .06, SE = .11, t(117) = .54, p = .58$). Finally, in the model predicting the Self-Harm subscale, there was no significant interaction between trait BPD and fluctuations in estradiol; however, there was a significant interaction between trait BPD and *average levels* of estradiol such that higher average levels of estradiol were associated with greater symptoms among women with *lower* trait BPD ($\gamma_{\text{LOWTRAITBPD*HIGHAVGESTRADIOL}} = .21, SE = .10, t(117) = 2.10, p = .01$), but was not significant among women with *higher* trait BPD ($\gamma_{\text{HIGHTRAITBPD*HIGHAVGESTRADIOL}} = -.09, SE = .11, t(117) = -.81, p = .40$). A graph of the interaction predicting Self-Harm can be found in Figure 6.

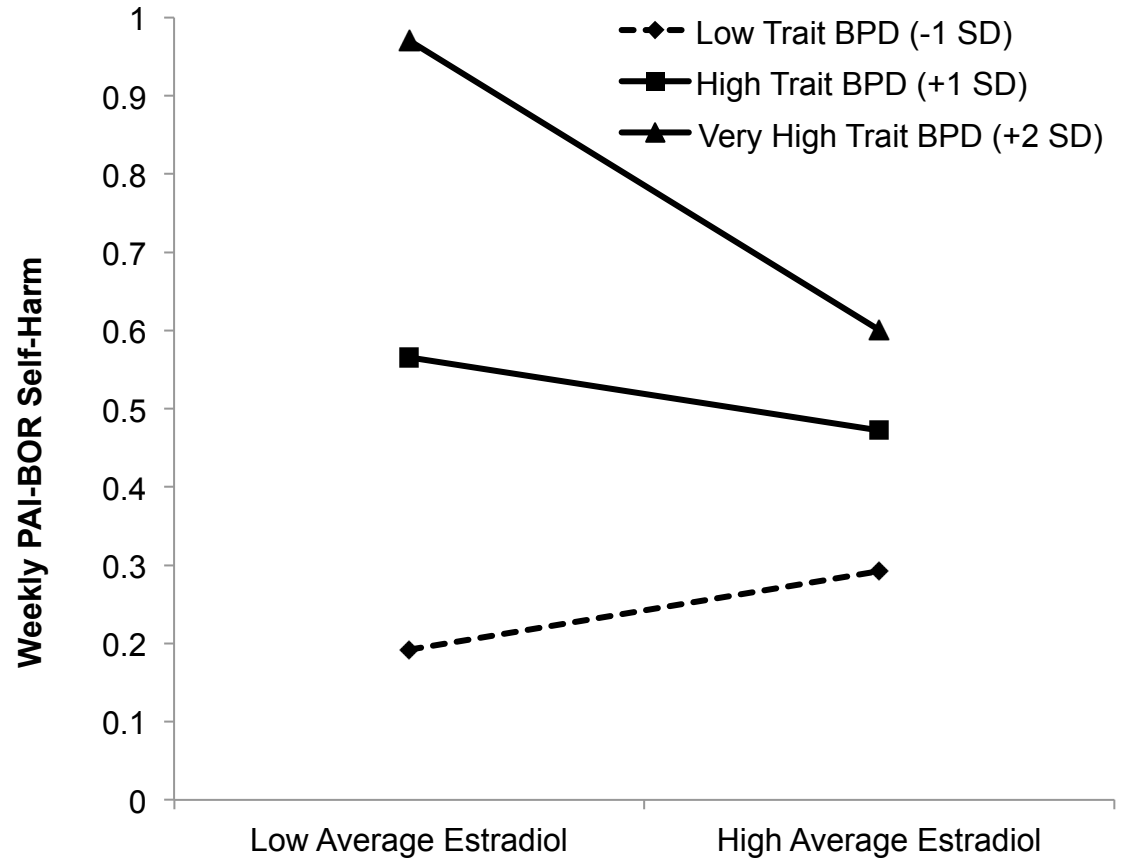


Figure 6. A graph of the interaction between trait BPD symptoms and *average* estradiol predicting weekly PAI-BOR Self-Harm subscale score.

Results of models substituting number of SCID-II criteria met for average weekly scores on the PAI-BOR total scale are presented in Table 8. None of the interactions of trait BPD with average estradiol or fluctuations in estradiol were statistically significant.

Testing Hypothesis 3a: Mediation by Felt Social Acceptance

It was predicted that increases in daily conception probability would be associated with lower daily felt acceptance, and that these effects would be stronger among women high in trait BPD features. Previous analyses suggest that conception probability and estradiol may not exert robust main effects on BPD features (i.e., the C path of the mediation model may not be significant); however, tests of whether increases in conception probability and estradiol are associated with decreased felt acceptance (Path A in the mediation model) and whether changes in felt acceptance were associated with daily BPD symptom expression (Path B in the mediation model) were nevertheless carried out. In addition, because the interactive effect of weekly deviations in estradiol and trait levels of BPD features on weekly BPD features were in some cases significant, similar models predicting felt acceptance (in addition to full mediation testing) were carried out as well using averages of weekly PAI-BOR as the moderator.

Path A at the Daily Level: Does daily conception probability exert a main or moderated effect on daily felt acceptance? Results of multilevel models regressing daily felt acceptance on conception probability are presented in Table 9. Because the distribution of daily felt rejection followed the inverse of a Poisson distribution (i.e., most people reported feeling high levels of acceptance on most days) and could not be transformed to normality using a linear transformation, Poisson models predicting reverse-scored acceptance (i.e., rejection), were utilized; however, the signs (i.e., -/+)

Table 8

Multilevel Poisson Regression Models Predicting Weekly BPD Symptoms from the Interaction of Number of SCID-II Criteria Met (Trait BPD) with Average Estradiol and Weekly Deviations in Estradiol

Parameter	Model 1 (null)	Model 4 (with moderator)
<u>Dependent Variable: PAI-BOR Total Score</u>		
Fixed Effects		
Intercept	-.50* (.10)	-.52* (.07)
Avg Estradiol		-.02 (.08)
Weekly Deviation in Estradiol		.001 (.04)
Number of SCID-II Criteria Met		.42* (.07)
Number of SCID-II Criteria Met*Avg Estradiol		.09 (.06)
<i>Number of SCID-II Criteria Met*Estradiol Deviation</i>		.03 (.05)
Random Parameters		
Intercept	.39* (.09)	.20* (.05)
Weekly Deviation in Estradiol		
Residual	.08* (.01)	.08* (.01)
-2 Restricted Log Pseudo-likelihood	240.55	232.89†
<u>Dependent Variable: PAI-BOR Affective Instability Subscale</u>		
Fixed Effects		
Intercept	-.60* (.15)	-.61* (.12)
Avg Estradiol		-.11 (.12)
Weekly Deviation in Estradiol		-.05 (.06)
Number of SCID-II Criteria Met		.55* (.12)
Number of SCID-II Criteria Met*Avg Estradiol		.16 (.12)
<i>Number of SCID-II Criteria Met*Estradiol Deviation</i>		.03 (.07)
Random Parameters		
Intercept	.83* (.23)	.49* (.15)
Weekly Deviation in Estradiol		
Residual	.16* (.02)	.16* (.02)
-2 Restricted Log Pseudo-likelihood	370.17	361.50†

Table Continued on Next Page

Dependent Variable: PAI-BOR Identity Disturbance Subscale		
	Fixed Effects	
Intercept	-0.30* (.11)	-.32 (.08)
Avg Estradiol		-.03 (.08)
Weekly Deviation in Estradiol		.001 (.05)
Number of SCID-II Criteria Met		.48 (.08)
Number of SCID-II Criteria Met*Avg Estradiol		.08 (.06)
<i>Number of SCID-II Criteria Met*Estradiol Deviation</i>		-.01 (.05)
	Random Parameters	
Intercept	.45* (.11)	.20* (.06)
Weekly Deviation in Estradiol		
Residual	.12* (.01)	.12* (.01)
-2 Restricted Log Pseudo-likelihood	274.93	261.14†

Dependent Variable: PAI-BOR Negative Relationships Subscale		
	Fixed Effects	
Intercept	-0.41* (.12)	-.29* (.09)
Avg Estradiol		-.03 (.10)
Weekly Deviation in Estradiol		.04 (.15)
Number of SCID-II Criteria Met		.33* (.08)
Number of SCID-II Criteria Met*Avg Estradiol		.11 (.08)
<i>Number of SCID-II Criteria Met*Estradiol Deviation</i>		.005 (.18)
	Random Parameters	
Intercept	.53* (.14)	.37* (.11)
Weekly Deviation in Estradiol		
Residual	.16* (.02)	.16* (.02)
-2 Restricted Log Pseudo-likelihood	332.16	329.44†

Dependent Variable: PAI-BOR Self-Harm Subscale		
	Fixed Effects	
Intercept	-1.03* (.11)	-1.04* (.11)
Avg Estradiol		.05 (.11)
Weekly Deviation in Estradiol		.04 (.11)
Number of SCID-II Criteria Met		.22* (.11)
Number of SCID-II Criteria Met*Avg Estradiol		.02 (.09)
<i>Number of SCID-II Criteria Met*Estradiol Deviation</i>		.11 (.10)
	Random Parameters	
Intercept	.35* (.12)	.35* (.12)
Weekly Deviation in Estradiol		
Residual	.18* (.02)	.18* (.02)
-2 Restricted Log Pseudo-likelihood	407.23	406.88

Table Continued on Next Page

Dependent Variable: Borderline Symptom Checklist—23 (BSL-23)		
	Fixed Effects	
Intercept	2.05* (.16)	2.02* (.14)
Avg Estradiol		.03 (.14)
Weekly Deviation in Estradiol		-.11* (.05)
Number of SCID-II Criteria Met		.62* (.14)
Number of SCID-II Criteria Met*Avg Estradiol		.09 (.11)
<i>Number of SCID-II Criteria Met*Estradiol Deviation</i>		-.05 (.07)
	Random Parameters	
Intercept	1.06* (.29)	.68* (.20)
Weekly Deviation in Estradiol		
Residual	2.34* (.31)	2.26* (.30)
-2 Restricted Log Pseudo-likelihood	379.72	366.35†

Dependent Variable: McLean Screening Instrument for BPD (MSI-BPD)		
	Fixed Effects	
Intercept	.17 (.22)	.13 (.19)
Avg Estradiol		.007 (.20)
Weekly Deviation in Estradiol		-.08 (.07)
Number of SCID-II Criteria Met		.76* (.19)
Number of SCID-II Criteria Met*Avg Estradiol		.02 (.15)
<i>Number of SCID-II Criteria Met*Estradiol Deviation</i>		.04 (.08)
	Random Parameters	
Intercept	1.80* (.54)	1.26* (.41)
Weekly Deviation in Estradiol		
Residual	.68* (.09)	.70* (.09)
-2 Restricted Log Pseudo-likelihood	481.04	473.71†

Note. Standard errors are in parentheses.

* $p < .05$.

†Change in -2 Restricted Log Pseudo-likelihood over the previous model is significant at $p < .05$.

Table 9

Multilevel Poisson Regression Models Predicting Daily Felt Acceptance from Daily Conception Probability Values

Parameter	Model 1 (null)	Model 2 (fixed CP slope)	Model 3 (random CP slope)
<u>Dependent Variable: Daily Felt Acceptance</u>			
		Fixed Effects	
Intercept	-.13* (.05)	-.53* (.06)	-.54* (.06)
Conception Probability		.27 (.40)	.65 (.73)
		Random Parameters	
Intercept	-.13* (.03)	-.13* (.03)	-.14* (.03)
Conception Probability			-11.98* (4.33)
Residual	-.17* (.009)	-.18* (.09)	-.16* (.008)
-2 Restricted Log Pseudo-likelihood	652.33	603.67†	548.99†

Note. Standard errors are in parentheses. CP = Daily Conception Probability.

* $p < .05$.

†Change in -2 Restricted Log Pseudo-likelihood over the previous model is significant at $p < .05$.

were reversed so that the estimates presented in Table 9 reflect the prediction of daily felt acceptance rather than felt rejection.

Results presented in Table 9 indicate that, though the fixed effect of conception probability was not significant for daily felt acceptance, there was a significant random effect of conception probability on daily felt acceptance. This indicates that the influence of conception probability values on daily felt acceptance differed between individuals. Therefore, I next carried out moderation analyses to determine whether the average of weekly PAI-BOR total score assessments or (2) the number of SCID-II BPD criteria met moderated the effect of conception probability. Results of those models are presented in Table 10 and Table 11. As before, estimates from previous best-fitting models were transposed into these tables for ease of model comparison.

Results revealed a significant interactive effect of trait BPD and conception probability predicting daily felt acceptance in the opposite of the direction predicted. Women with *higher* levels of trait BPD showed increases in daily felt acceptance when conception probability was higher ($\gamma_{\text{HIGHTRAITBPD*CP}} = .70, SE = .20, t(686) = 3.50, p = .0006$), while women with *lower* levels of trait BPD showed no association between conception probability and felt acceptance ($\gamma_{\text{LOWTRAITBPD*CP}} = .41, SE = .63, t(686) = .65, p = .51$). A graph depicting the interaction can be found in Figure 7. In contrast, number of SCID-II BPD criteria met was not a significant moderator of the effect of conception probability.

Path A at the Weekly Level: Do changes in weekly felt acceptance impact weekly BPD symptom expression? Next, I tested the same hypothesis at the weekly level. Results of models regressing weekly scores on the Social Evaluation subscale of the State

Table 10

Multilevel Poisson Regression Models Predicting Daily Felt Acceptance from the Interaction of Daily Conception Probability Values and Average of Weekly PAI-BOR Assessments (Trait BPD)

Parameter	Model 3 (random CP slope)	Model 4 (with moderator)
<u>Dependent Variable: Daily Felt Acceptance</u>		
Fixed Effects		
Intercept	-.54* (.06)	-.52* (.04)
Conception Probability	.65 (.73)	.61 (.73)
Avg of Weekly PAI-BOR		-.29* (.04)
<i>Avg of Weekly PAI-BOR*CP</i>		<i>1.02* (.10)</i>
Random Parameters		
Intercept	-.14* (.03)	-.05* (.01)
Conception Probability	-11.98* (4.33)	-12.21* (4.47)
Residual	-.16* (.008)	-.15* (.008)
-2 Restricted Log Pseudo-likelihood	548.99	517.22†

Note. Standard errors are in parentheses. CP = Daily Conception Probability.

* $p < .05$.

†Change in -2 Restricted Log Pseudo-likelihood over the previous model is significant at $p < .05$.

Table 11

Multilevel Poisson Regression Models Predicting Daily Felt Acceptance from the Interaction of Daily Conception Probability Values and Number of SCID-II BPD Criteria Met (Trait BPD)

Parameter	Model 3 (random CP slope)	Model 4 (with moderator)
<u>Dependent Variable: Daily Felt Acceptance</u>		
Fixed Effects		
Intercept	.54* (.06)	-.52* (.05)
Conception Probability	-.65 (.73)	.52 (.74)
SCID-II BPD Criteria Met		-.20* (.05)
<i>SCID-II BPD Criteria Met*CP</i>		.61 (.70)
Random Parameters		
Intercept	.14* (.03)	.10* (.02)
Conception Probability	11.98* (4.33)	12.40* (4.42)
Residual	.16* (.008)	.15* (.008)
-2 Restricted Log Pseudo-likelihood	548.99	531.54†

Note. Standard errors are in parentheses. CP = Daily Conception Probability.

* $p < .05$.

†Change in -2 Restricted Log Pseudo-likelihood over the previous model is significant at $p < .05$.

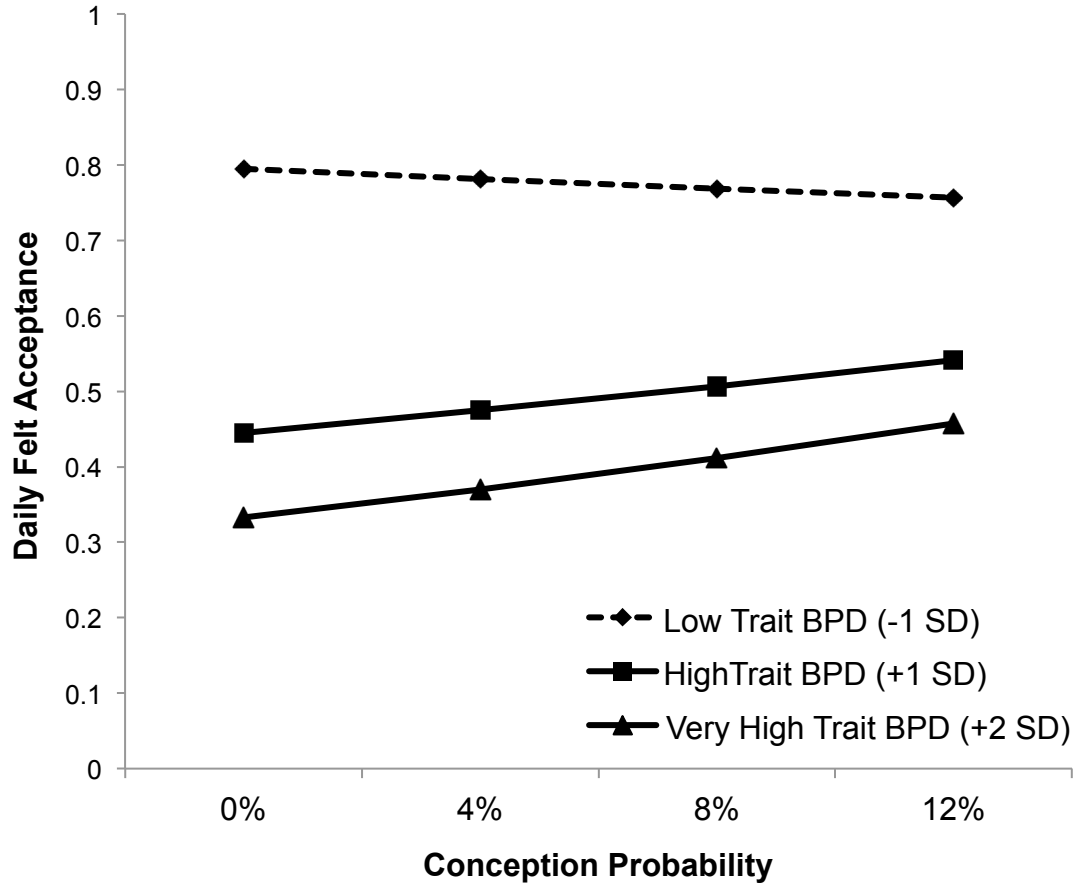


Figure 7. A graph of the interaction between trait BPD symptoms and daily conception probability predicting daily felt acceptance.

Self-Esteem Scale on average estradiol and weekly deviations in estradiol are presented in Table 12. There were no significant effects of deviations in estradiol on felt acceptance. However, there was a significant improvement in model fit with the inclusion of a random effect of deviations in estradiol, indicating that the effect of deviations in estradiol on felt social acceptance differed between individuals.

Therefore, I next examined whether the effect of deviations in estradiol on weekly felt acceptance was moderated by trait BPD symptoms as measured using the average of weekly PAI-BOR total assessments. Results of this model can be found in Table 13. Inclusion of the moderator significantly improved model fit, and the interaction was significant in the opposite of the predicted direction. Among women *higher* in trait BPD, higher-than-usual levels of estradiol were associated with higher felt acceptance ($\gamma_{\text{HIGHTRAITBPD*ESTRADIOLDEVIATION}} = .21, SE = .08, t(117) = 2.71, p = .007$). Among women *lower* in trait BPD, higher-than-usual levels of estradiol were associated with lower felt acceptance ($\gamma_{\text{LOWTRAITBPD*ESTRADIOLDEVIATION}} = -.17, SE = .08, t(117) = -2.12, p = .03$). A graph depicting this interaction can be found in Figure 8.

Path B at the Daily Level: Do changes in daily felt acceptance impact daily BPD symptom expression? Next, I tested whether average levels of and daily changes in felt acceptance impact expression of BPD symptoms. Results of models regressing average levels of daily felt acceptance and daily deviations from one's own average levels of felt acceptance on each subscale and the total score of the daily PAI-BOR are presented in Table 14.

For each subscale and the PAI-BOR total score, model fit was significantly improved with the addition of the felt acceptance predictors as fixed effects. Model fit

Table 12

Multilevel Regression Models Predicting Weekly Felt Acceptance from Average Estradiol and Weekly Deviations in Estradiol

Parameter	Model 1 (null)	Model 2 (fixed deviation slope)	Model 3 (random deviation slope)
Dependent Variable: Weekly Felt Acceptance (Social Evaluation subscale of the SSES)			
		Fixed Effects	
Intercept	5.37* (.12)	5.37* (.12)	5.37* (.12)
Average Estradiol		.06 (.13)	.09 (.13)
<i>Weekly Deviations in Estradiol</i>		.02 (.05)	.0002 (.05)
		Random Parameters	
Intercept	.58* (.14)	.58* (.13)	.58* (.14)
Weekly Deviations in Estradiol			.001 (.09)
Residual	.18* (.02)	.18* (.02)	.18* (.02)
-2 log likelihood	292.9	292.1	289.4†

Note. Standard errors are in parentheses.

* $p < .05$.

†Change in -2 log likelihood over the previous model is significant at $p < .05$.

Table 13

Multilevel Regression Models Predicting Weekly Felt Acceptance from the Interaction of Average Weekly PAI-BOR Total Score with Average Estradiol and Deviations in Estradiol

Parameter	Model 1 (null)	Model 4 (with moderator)
<u>Dependent Variable: Weekly Felt Acceptance (Social Evaluation subscale of the SSES)</u>		
Fixed Effects		
Intercept	5.37* (.12)	5.39* (.10)
Average Estradiol		.04 (.11)
Weekly Deviations in Estradiol		.03 (.05)
Avg Weekly PAI-BOR Total		-.40* (.09)
Avg PAI-BOR*Avg Estradiol		.15 (.10)
<i>Avg PAI-BOR*Estradiol Deviation</i>		.16* (.05)
Random Parameters		
Intercept	.58* (.14)	.40* (.09)
Weekly Deviations in Estradiol		
Residual	.18* (.02)	.18* (.02)
-2 log likelihood	292.9	272.3†

Note. Standard errors are in parentheses.

* $p < .05$.

†Change in -2 log likelihood over the previous model is significant at $p < .05$.

Table 14

Multilevel Poisson Regression Models Predicting Daily PAI-BOR Scores from Average Levels of Daily Felt Acceptance and Daily Deviations in Felt Acceptance

Parameter	Model 1 (null)	Model 2 (fixed deviation slope)	Model 3 (random deviation slope)
<u>Dependent Variable: PAI-BOR Total Score</u>			
		Fixed Effects	
Intercept	-.62* (.10)	.70* (.06)	.70* (.06)
Average Daily Felt Acceptance <i>Deviations in Felt Acceptance</i>		-.45* (.06) -.34* (.01)	-.45* (.06) -.36* (.03)
		Random Parameters	
Intercept	.38* (.09)	.15* (.03)	.15* (.03)
Deviations in Felt Acceptance			.01 (.01)
Residual	.13* (.006)	.08* (.004)	.08* (.004)
-2 Restricted Log Pseudo-likelihood	1348.35	1015.14†	1009.87†
<u>Dependent Variable: PAI-BOR Affective Instability Subscale</u>			
		Fixed Effects	
Intercept	-.68* (.12)	.70* (.09)	.78* (.09)
Average Daily Felt Acceptance <i>Deviations in Felt Acceptance</i>		-.49* (.06) -.41* (.04)	-.50* (.08) -.45* (.04)
		Random Parameters	
Intercept	.59* (.15)	.31* (.08)	.31* (.08)
Deviations in Felt Acceptance			.01 (.01)
Residual	.33* (.01)	.01* (.01)	.25* (.01)
-2 Restricted Log Pseudo-likelihood	2117.42	2048.48†	1938.95†
<u>Dependent Variable: PAI-BOR Identity Disturbance Subscale</u>			
		Fixed Effects	
Intercept	-.55* (.10)	.62* (.07)	.62* (.06)
Average Daily Felt Acceptance <i>Deviations in Felt Acceptance</i>		-.47* (.06) -.30* (.02)	-.47* (.06) -.34* (.03)
		Random Parameters	
Intercept	.42* (.10)	.17* (.04)	.17* (.04)
Deviations in Felt Acceptance			.01 (.02)
Residual	.17* (.009)	.13* (.007)	.13* (.007)
-2 Restricted Log Pseudo-likelihood	1521.40	1322.46†	1324.32

Table Continued on Next Page

Dependent Variable: PAI-BOR Negative Relationships Subscale			
		Fixed Effects	
Intercept	-.48* (.10)	.55* (.07)	.55* (.07)
Average Daily Felt Acceptance		-.47* (.07)	-.47* (.07)
<i>Deviations in Felt Acceptance</i>		-.31* (.02)	-.35* (.05)
		Random Parameters	
Intercept	.44* (.10)	.19* (.04)	.19* (.04)
Deviations in Felt Acceptance			.04 (.03)
Residual	.22* (.01)	.18* (.009)	.17* (.009)
-2 Restricted Log Pseudo-likelihood	1622.09	1450.27†	1447.83†
Dependent Variable: PAI-BOR Self-Harm Subscale			
		Fixed Effects	
Intercept	-1.13* (.15)	1.21* (.14)	1.20* (.14)
Average Daily Felt Acceptance		-.38* (.13)	-.38* (.13)
<i>Deviations in Felt Acceptance</i>		-.31* (.04)	-.29* (.05)
		Random Parameters	
Intercept	.87* (.24)	.74* (.21)	.74* (.21)
Deviations in Felt Acceptance			.01 (.01)
Residual	.34* (.01)	.32* (.01)	.32* (.01)
-2 Restricted Log Pseudo-likelihood	2551.02	2521.59†	2520.04

Note. Standard errors are in parentheses. CP = Daily Conception Probability.

* $p < .05$.

†Change in -2 Restricted Log Pseudo-likelihood over the previous model is significant at $p < .05$.

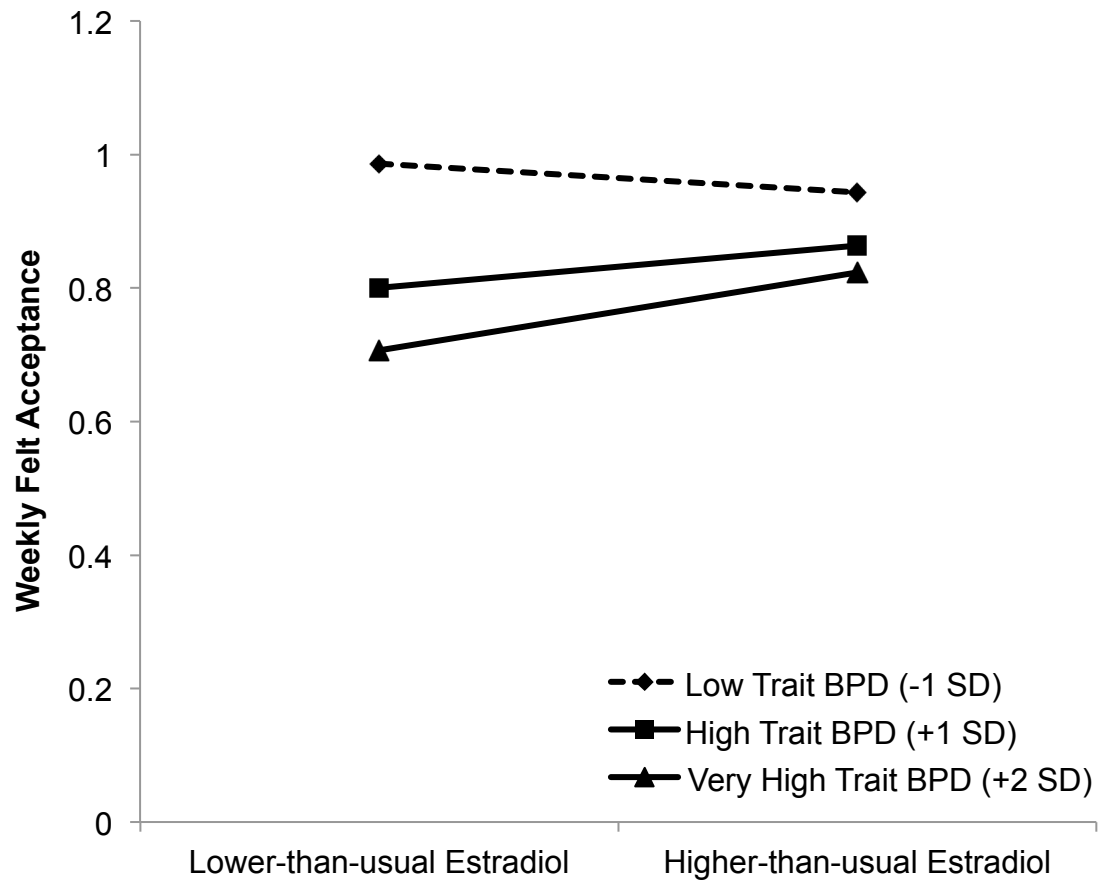


Figure 8. A graph of the interaction between trait BPD symptoms and deviation in estradiol predicting weekly felt acceptance.

improved significantly with the addition of random effects of daily deviations in felt acceptance in all cases except the Self-Harm subscale and the Identity Disturbance subscale; however, the random effect of deviations in felt acceptance was not significant for any of the subscales or the total, suggesting that the effects of changes in felt acceptance may be relatively similar across women. Results indicate that, for each daily PAI-BOR subscale and the total score, both average levels of daily felt acceptance and higher-than-usual daily levels of felt acceptance were associated with lower expression of BPD symptoms.

Path B at the Weekly Level: Do changes in weekly felt acceptance impact weekly BPD symptom expression? Next, I tested whether average levels of and daily changes in felt acceptance impact expression of BPD symptoms at the weekly level. Results of models regressing average levels of weekly felt acceptance (measured using the Social Evaluation subscale of the State Self-Esteem Scale) and weekly deviations in felt acceptance on each subscale and the total score of the daily PAI-BOR, the BSL-23, and the MSI-BPD are presented in Table 15. For each dependent variable, model fit was significantly improved with the inclusion of fixed effects of average weekly levels of felt acceptance and weekly fluctuations in felt acceptance; however, model fit was not improved significantly with the inclusion of random effect of weekly fluctuations in felt acceptance, suggesting once again that the impact of felt acceptance on BPD symptoms is similar across individuals. In every case, both higher average levels of felt acceptance and higher-than-usual weekly felt acceptance were associated with lower levels of BPD symptoms.

Table 15

Multilevel Poisson Regression Models Predicting Weekly BPD Symptoms from Average Levels of Weekly Felt Acceptance and Weekly Deviations in Felt Acceptance

Parameter	Model 1 (null)	Model 2 (fixed deviation slope)	Model 3 (random deviation slope)
<u>Dependent Variable: PAI-BOR Total Score</u>			
		Fixed Effects	
Intercept	-.50 (.10)	-.51* (.08)	-.51* (.08)
Average Weekly Felt Acceptance <i>Deviations in Felt Acceptance</i>		-.36* (.08) -.27* (.06)	-.36* (.08) -.26* (.08)
		Random Parameters	
Intercept	.39* (.09)	.27* (.07)	.27* (.07)
Deviations in Felt Acceptance			.04 (.04)
Residual	.08* (.01)	.07* (.009)	.06* (.009)
-2 Restricted Log Pseudo-likelihood	240.55	220.48†	218.38
<u>Dependent Variable: PAI-BOR Affective Instability Subscale</u>			
		Fixed Effects	
Intercept	-.60 (.15)	-.63* (.14)	-.64* (.14)
Average Weekly Felt Acceptance <i>Deviations in Felt Acceptance</i>		-.42* (.14) -.38* (.08)	-.42* (.14) -.39* (.11)
		Random Parameters	
Intercept	.83* (.23)	.70* (.20)	.72* (.20)
Deviations in Felt Acceptance			.07 (.07)
Residual	.16* (.02)	.14* (.01)	.13* (.01)
-2 Restricted Log Pseudo-likelihood	370.17	358.78†	358.85
<u>Dependent Variable: PAI-BOR Identity Disturbance Subscale</u>			
		Fixed Effects	
Intercept	-.30 (.11)	-.31* (.09)	-.31* (.09)
Average Weekly Felt Acceptance <i>Deviations in Felt Acceptance</i>		-.42* (.09) -.24* (.06)	-.42* (.09) -.23* (.07)
		Random Parameters	
Intercept	.45* (.11)	.29* (.07)	.29* (.07)
Deviations in Felt Acceptance			.02 (.03)
Residual	.12* (.01)	.11* (.01)	.11* (.01)
-2 Restricted Log Pseudo-likelihood	274.93	258.58†	256.62

Table Continued on Next Page

Dependent Variable: PAI-BOR Negative Relationships Subscale			
		Fixed Effects	
Intercept	-.41* (.12)	-.42* (.10)	-.42* (.10)
Average Weekly Felt Acceptance		-.42* (.10)	-.42* (.12)
<i>Deviations in Felt Acceptance</i>		-.22* (.08)	-.23* (.08)
		Random Parameters	
Intercept	.53* (.14)	.36* (.10)	.36* (.11)
Deviations in Felt Acceptance			.001 (.10)
Residual	.16* (.02)	.16* (.02)	.16* (.02)
-2 Restricted Log Pseudo-likelihood	332.16	320.31†	322.34
Dependent Variable: PAI-BOR Self-Harm Subscale			
		Fixed Effects	
Intercept	-1.03 (.11)	-1.04* (.10)	-1.06* (.10)
Average Weekly Felt Acceptance		-.19* (.06)	-.18* (.06)
<i>Deviations in Felt Acceptance</i>		-.23* (.13)	-.23* (.13)
		Random Parameters	
Intercept	.35* (.12)	.33* (.11)	.35* (.12)
Deviations in Felt Acceptance			.24* (.23)
Residual	.18* (.02)	.18* (.02)	.16* (.02)
-2 Restricted Log Pseudo-likelihood	407.23	405.74†	406.88
Dependent Variable: McLean Screening Instrument for BPD (MSI-BPD)			
		Fixed Effects	
Intercept	.17 (.22)	.15 (.18)	.12 (.19)
Average Weekly Felt Acceptance		-.85* (.19)	-.87* (.20)
<i>Deviations in Felt Acceptance</i>		-.30* (.10)	-.27* (.14)
		Random Parameters	
Intercept	1.80* (.54)	1.16* (.36)	1.22* (.38)
Deviations in Felt Acceptance			.12 (.11)
Residual	.68* (.09)	.67* (.09)	.61* (.08)
-2 Restricted Log Pseudo-likelihood	481.04	470.40†	471.91
Dependent Variable: Borderline Symptom Checklist—23 (BSL-23)			
		Fixed Effects	
Intercept	2.05 (.16)	1.97*	1.97*
Average Weekly Felt Acceptance		-.85* (.13)	-.85* (.13)
<i>Deviations in Felt Acceptance</i>		-.48* (.11)	-.48* (.11)
		Random Parameters	
Intercept	1.06* (.29)	.54* (.14)	.56* (.15)
Deviations in Felt Acceptance		.15* (.08)	.15* (.08)
Residual	2.34* (.31)	1.36* (.19)	1.35* (.19)
-2 Restricted Log Pseudo-likelihood	379.72	322.00†	336.62

Note. Standard errors are in parentheses.

* $p < .05$.

†Change in -2 Restricted Log Pseudo-likelihood over the previous model is significant at $p < .05$.

Tests of Indirect Effects. Figures 9-12 depict mediation models with felt acceptance as the mediator. First, I used the RMediation program (Tofighi & MacKinnon, 2011) to estimate 95% confidence intervals for the indirect effects of the interaction of trait BPD and deviations in estradiol on weekly BPD symptoms through weekly felt acceptance. The 95% confidence intervals did not include zero for the indirect effects of Trait BPD x Deviations in Estradiol on the total score of the PAI-BOR (95% CI: -.01 to -.08), the Affective Instability subscale of the PAI-BOR (95% CI: -.03 to -.12), the Identity Disturbance subscale of the PAI-BOR (95% CI: -.02 to -.07), or the BSL-23 (95% CI: -.03 to -.14) via weekly felt acceptance. Furthermore, when average and weekly deviations in felt acceptance were included in the models predicting BPD symptoms from the interaction of Trait BPD with deviations in estradiol, none of the interactive effects of Trait BPD X Deviations in Estradiol remained significant (Predicting the PAI-BOR total score: $\gamma_{\text{TRAITBPD*ESTRADIOLDEVIATION}} = -.05, SE = .05, t(117) = -1.05, p = .29$; Predicting the PAI-BOR Affective Instability subscale: $\gamma_{\text{TRAITBPD*ESTRADIOLDEVIATION}} = -.05, SE = .07, t(117) = -.70, p = .48$; Predicting the PAI-BOR Identity Disturbance subscale: $\gamma_{\text{TRAITBPD*ESTRADIOLDEVIATION}} = -.08, SE = .05, t(117) = -1.49, p = .13$; Predicting the BSL-23: $\gamma_{\text{TRAITBPD*ESTRADIOLDEVIATION}} = -.03, SE = .05, t(117) = -.59, p = .55$). These results indicate that the interactive effects of Trait BPD and deviations in estradiol on BPD symptom expression are partially attributable to changes in felt acceptance.

Testing Hypothesis 3b and 3c: Mediation by Impulsivity and Self-Control

It was hypothesized that increases in estradiol would be associated with greater impulsivity and poorer self-control, that these effects would be stronger among women high in trait BPD features, and that such effects would mediate the main or moderated

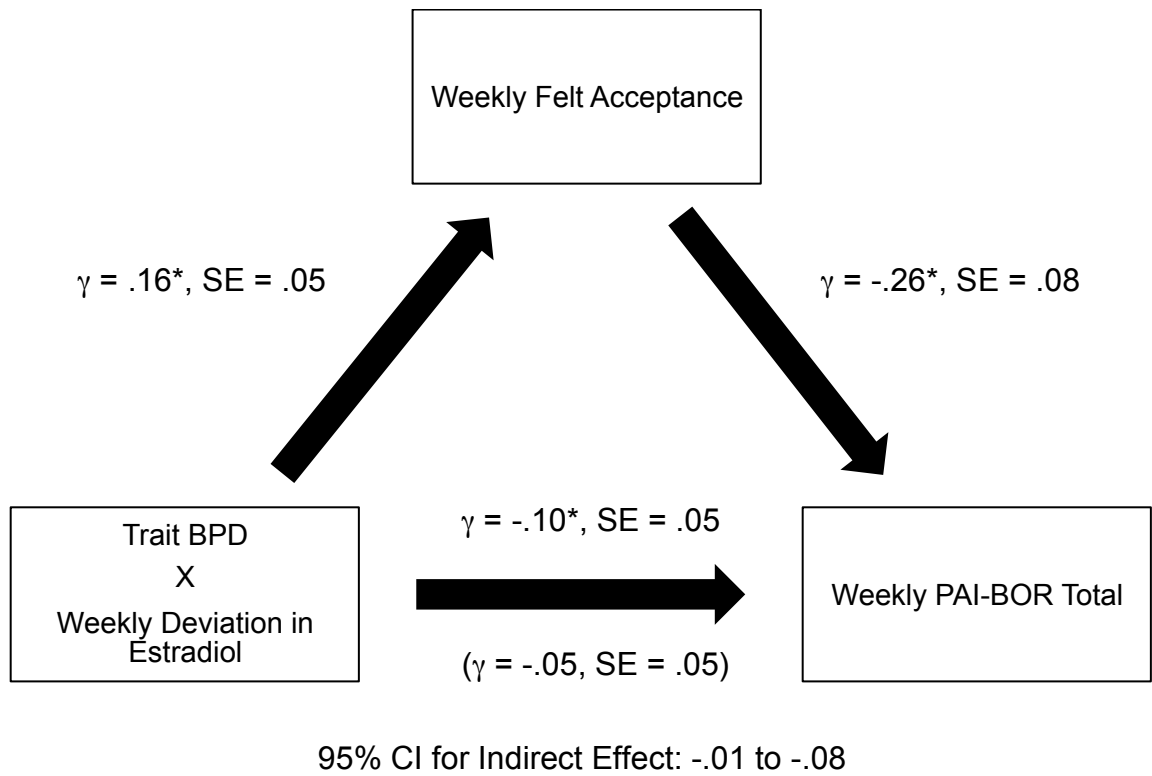
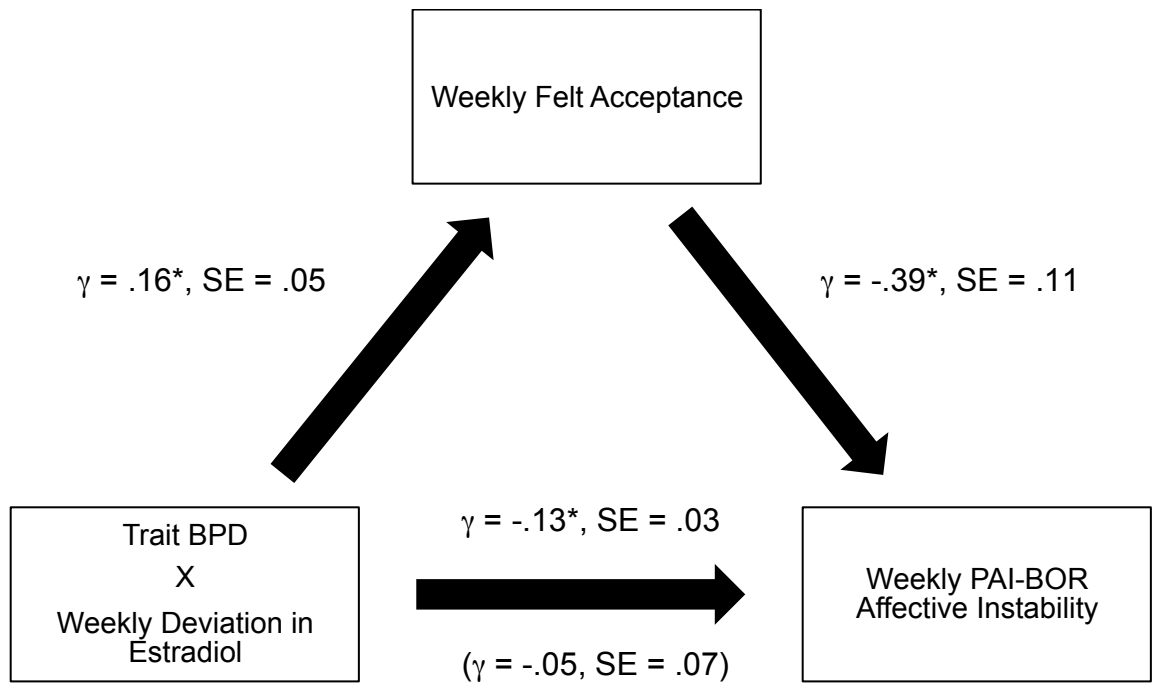


Figure 9. A depiction of the indirect effect of the interactive effect of trait BPD and weekly *deviation* in estradiol on weekly PAI-BOR total score via weekly felt acceptance.



95% CI for Indirect Effect: -.03 to -.12

Figure 10. A depiction of the indirect effect of the interactive effect of trait BPD and weekly *deviation* in estradiol on weekly PAI-BOR Affective Instability scores via weekly felt acceptance.

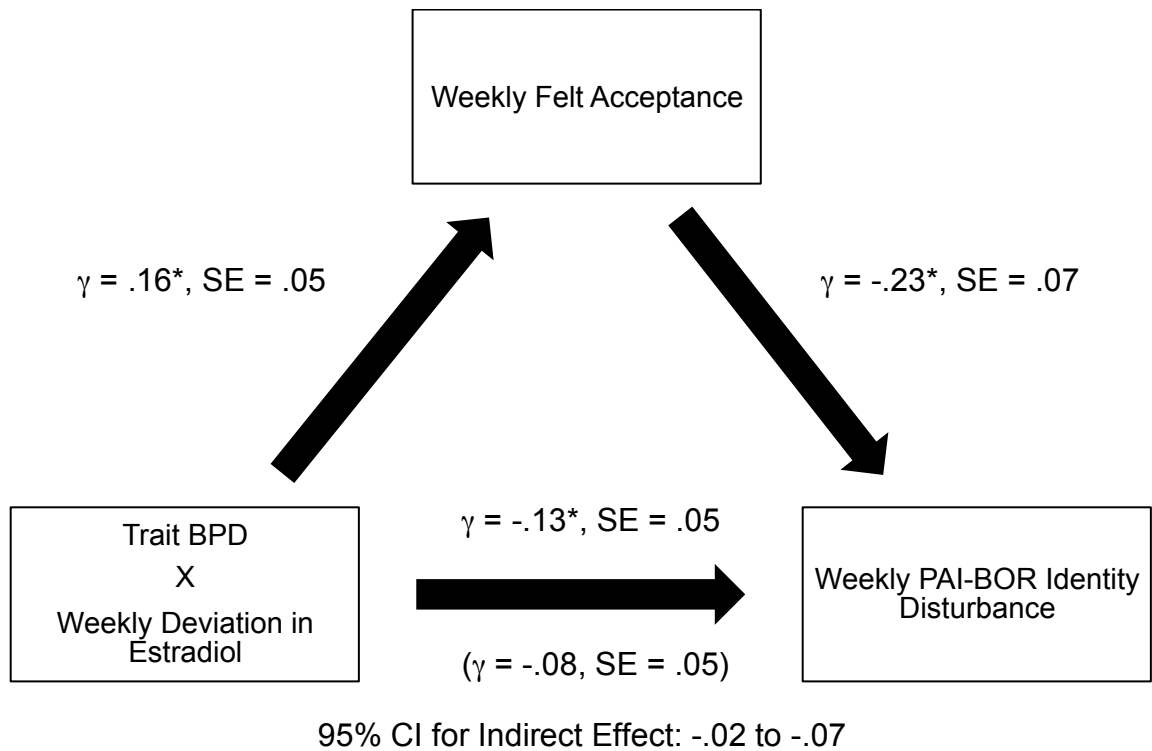
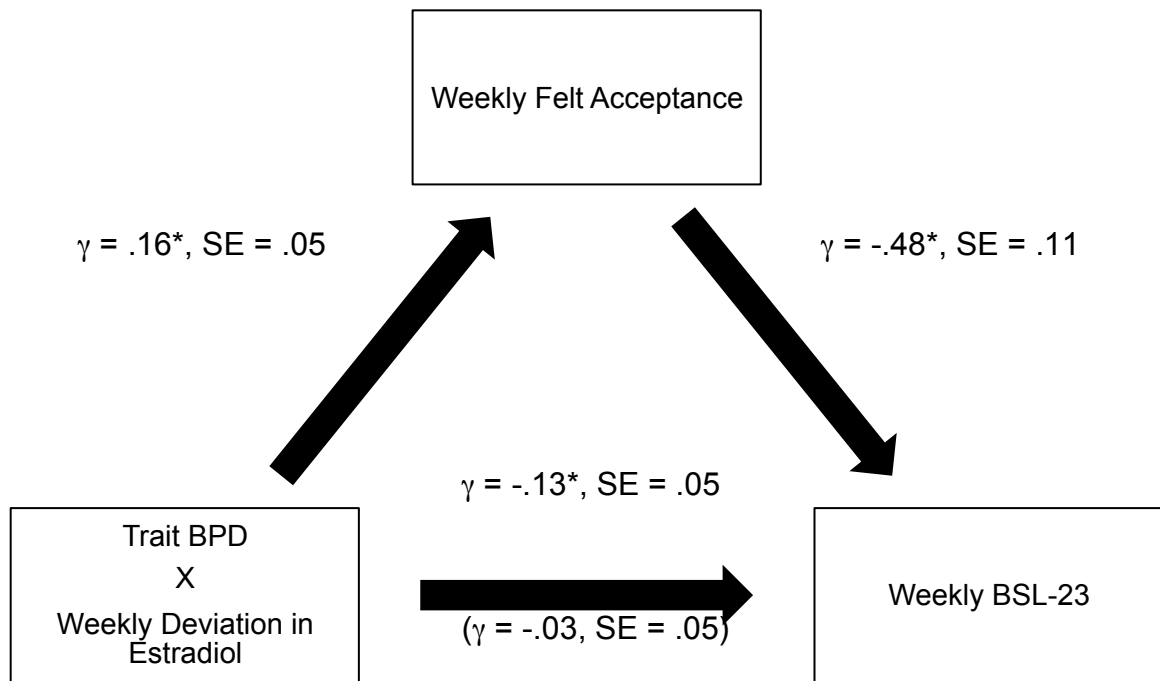


Figure 11. A depiction of the indirect effect of the interactive effect of trait BPD and weekly *deviation* in estradiol on weekly PAI-BOR Identity Disturbance scores via weekly felt acceptance.



95% CI for Indirect Effect: -.03 to -.14

Figure 12. A depiction of the indirect effect of the interactive effect of trait BPD and weekly deviation in estradiol on weekly BSL-23 scores via weekly felt acceptance.

effects of deviations in estradiol on weekly BPD features. Previous analyses suggest that weekly deviations in estradiol do not exert a main effect on BPD features (i.e., there is not a significant C path in the mediation model); however, tests of whether increases in estradiol are associated with poorer self-control and greater impulsivity (Path A in the mediation model) and whether changes in self-control and impulsivity are associated with weekly BPD symptom expression (Path B in the mediation model) were nevertheless carried out. In addition, because the interactive effect of weekly deviations in estradiol and trait levels of BPD features on weekly BPD features were in some cases significant, similar models predicting impulsivity and self-control (in addition to full mediation testing) will be carried out as well using averages of weekly PAI-BOR as the moderator. Because self-control and impulsivity were measured only at the weekly level, this hypothesis was tested only at that level.

Path A at the Weekly Level: Do changes in weekly self-control impact weekly BPD symptom expression? First, I regressed weekly scores on self-control and impulsivity on average estradiol and weekly deviations in estradiol are presented in Table 16. Higher-than-usual levels of estradiol were associated with higher Self-Control, lower Negative Urgency, lower Positive Urgency, and lower Lack of Perseverance. In each case, model fit was significantly improved with the inclusion of a random effect of deviations in estradiol, indicating the presence of a moderator. Therefore, I next examined models predicting self-control and aspects of impulsivity from the interaction of the average of weekly PAI-BOR total scores and deviations in estradiol. Results of these models can be found in Table 17. The results of Model 3 (including a random effect of deviations in estradiol) were transferred into the table for ease of model comparison.

Table 16

Multilevel Regression Models Predicting Weekly Self-Control and Impulsivity from Average Estradiol and Weekly Deviations in Estradiol

Parameter	Model 1 (null)	Model 2 (fixed deviation slope)	Model 3 (random deviation slope)
<u>Dependent Variable: Weekly Self-Control</u>			
		Fixed Effects	
Intercept	3.42* (.10)	3.41* (.09)	3.41* (.09)
Average Estradiol		.07 (.09)	.06 (.09)
<i>Weekly Deviations in Estradiol</i>		.09+ (.06)	.09* (.03)
		Random Parameters	
Intercept	.37* (.09)	.36* (.09)	.37* (.09)
Weekly Deviations in Estradiol			.02+ (.01)
Residual	.14* (.01)	.14* (.01)	.13* (.01)
-2 log likelihood	246.7	242.6†	238.8†
<u>Dependent Variable: Weekly UPPS-P Negative Urgency (Poisson model)</u>			
		Fixed Effects	
Intercept	.48* (.05)	.48* (.05)	.48* (.05)
Average Estradiol		-.05 (.05)	-.05 (.05)
<i>Weekly Deviations in Estradiol</i>		-.05* (.01)	-.05* (.01)
		Random Parameters	
Intercept	.10* (.02)	.10* (.02)	.10* (.02)
Weekly Deviations in Estradiol			.002* (.001)
Residual	.07* (.009)	.07* (.009)	.07* (.01)
-2 log likelihood	57.05	55.34†	50.24†
<u>Dependent Variable: Weekly UPPS-P Positive Urgency</u>			
		Fixed Effects	
Intercept	1.84* (.07)	1.84* (.07)	1.84* (.07)
Average Estradiol		-.15* (.06)	-.14* (.06)
<i>Weekly Deviations in Estradiol</i>		-.10* (.02)	-.11* (.03)
		Random Parameters	
Intercept	.20* (.05)	.17* (.04)	.17* (.04)
Weekly Deviations in Estradiol			.01 (.02)
Residual	.13* (.01)	.12* (.01)	.12* (.01)
-2 log likelihood	210.1	201.70†	191.23†
<u>Dependent Variable: Weekly UPPS-P Lack of Premeditation</u>			
		Fixed Effects	
Intercept	1.84* (.08)	1.84* (.08)	1.84* (.08)
Average Estradiol		-.08 (.08)	-.06 (.07)
<i>Weekly Deviations in Estradiol</i>		-.02 (.03)	-.02 (.03)
		Random Parameters	
Intercept	.26* (.06)	.26* (.06)	.28* (.07)
Weekly Deviations in Estradiol			.001 (.10)
Residual	.14* (.01)	.14* (.01)	.14* (.01)
-2 log likelihood	231.20	227.3†	220.4†

Table Continued on Next Page

Dependent Variable: Weekly UPPS-P Lack of Perseverance			
		Fixed Effects	
Intercept	1.85* (.08)	1.85* (.08)	1.85* (.08)
Average Estradiol		-.03 (.08)	-.04 (.07)
<i>Weekly Deviations in Estradiol</i>		-.14* (.06)	-.15* (.06)
		Random Parameters	
Intercept	.23* (.06)	.23* (.06)	.23* (.06)
Weekly Deviations in Estradiol			.03 (.03)
Residual	.17* (.02)	.16* (.02)	.14* (.02)
-2 log likelihood	250	242.4†	235.10†
Dependent Variable: Weekly Sensation Seeking			
		Fixed Effects	
Intercept	2.24* (.10)	2.24* (.10)	2.24* (.10)
Average Estradiol		-.03 (.10)	-.05 (.10)
<i>Weekly Deviations in Estradiol</i>		-.07 (.07)	-.07 (.06)
		Random Parameters	
Intercept	.36* (.09)	.36* (.09)	.36* (.09)
Weekly Deviations in Estradiol			.03+ (.03)
Residual	.17* (.02)	.17* (.02)	.15* (.02)
-2 log likelihood	269	266.1†	262.1†

Note. Standard errors are in parentheses.

* $p < .05$.

+ $p < .15$.

†Change in -2 log likelihood over the previous model is significant at $p < .05$.

Table 17

Multilevel Regression Models Predicting Weekly Self-Control and Impulsivity from the Interaction of Average Weekly PAI-BOR Total Score with Average Estradiol and Weekly Deviations in Estradiol

Parameter	Model 3 (random deviation slope)	Model 4 (with moderator)
<u>Dependent Variable: Weekly Self-Control</u>		
Fixed Effects		
Intercept	3.41* (.09)	3.43* (.06)
Average Estradiol	.06 (.09)	.06 (.05)
Weekly Deviations in Estradiol	.09+ (.05)	.09+ (.05)
Avg Weekly PAI-BOR Total		-.44* (.05)
Avg PAI-BOR* Avg Estradiol		.13* (.04)
<i>Avg PAI-BOR* Estradiol Deviation</i>		.18* (.06)
Random Parameters		
Intercept	.37* (.09)	.16* (.04)
Weekly Deviations in Estradiol	.02+ (.01)	.01 (.02)
Residual	.13* (.01)	.13* (.01)
-2 log likelihood	239.8	202.9†
<u>Dependent Variable: Weekly UPPS-P Negative Urgency</u>		
Fixed Effects		
Intercept	.48* (.05)	.47* (.02)
Average Estradiol	-.05 (.05)	-.04+ (.02)
Weekly Deviations in Estradiol	-.05* (.02)	-.05* (.02)
Avg Weekly PAI-BOR Total		.27* (.02)
Avg PAI-BOR* Avg Estradiol		-.03* (.01)
<i>Avg PAI-BOR* Estradiol Deviation</i>		-.06* (.03)
Random Parameters		
Intercept	.10* (.02)	.02* (.008)
Weekly Deviations in Estradiol	.002* (.009)	.0001 (.09)
Residual	.07* (.01)	.07* (.009)
-2 log likelihood	62.51	27.70†
<u>Dependent Variable: Weekly UPPS-P Positive Urgency</u>		
Fixed Effects		
Intercept	1.84* (.07)	1.84* (.06)
Average Estradiol	-.14* (.06)	-.15* (.05)
Weekly Deviations in Estradiol	-.11* (.03)	-.10* (.03)
Avg Weekly PAI-BOR Total		.21* (.05)
Avg PAI-BOR* Avg Estradiol		-.03 (.03)
<i>Avg PAI-BOR* Estradiol Deviation</i>		-.11* (.04)
Random Parameters		
Intercept	.17* (.04)	.13* (.03)
Weekly Deviations in Estradiol	.001 (.10)	.0001 (.11)
Residual	.12* (.01)	.12* (.01)
-2 log likelihood	201.6	186.00†

Table Continued on Next Page

Dependent Variable: Weekly UPPS-P Lack of Premeditation

	Fixed Effects	
Intercept	1.84* (.08)	1.83* (.07)
Average Estradiol	-.06 (.07)	-.07 (.06)
Weekly Deviations in Estradiol	-.02 (.03)	-.04 (.03)
Avg Weekly PAI-BOR Total		.25* (.06)
Avg PAI-BOR* Avg Estradiol		-.12* (.05)
<i>Avg PAI-BOR* Estradiol Deviation</i>		-.09* (.04)
	Random Parameters	
Intercept	.28* (.07)	.18* (.05)
Weekly Deviations in Estradiol	.001 (.10)	.0001 (.10)
Residual	.14* (.01)	.13* (.01)
-2 log likelihood	220.4	208.1†

Dependent Variable: Weekly Lack of Perseverance

	Fixed Effects	
Intercept	1.85* (.08)	1.85* (.07)
Average Estradiol	-.04 (.07)	-.03 (.07)
Weekly Deviations in Estradiol	-.15* (.06)	-.16* (.04)
Avg Weekly PAI-BOR Total		.23* (.07)
Avg PAI-BOR* Avg Estradiol		-.04 (.08)
<i>Avg PAI-BOR* Estradiol Deviation</i>		-.22* (.05)
	Random Parameters	
Intercept	.23* (.06)	.18* (.04)
Weekly Deviations in Estradiol	.03 (.03)	.0001 (.09)
Residual	.14* (.02)	.14* (.01)
-2 log likelihood	235.10	218.01†

Dependent Variable: Weekly Sensation Seeking

	Fixed Effects	
Intercept	2.24* (.10)	2.23* (.09)
Average Estradiol	-.05 (.10)	-.04 (.09)
Weekly Deviations in Estradiol	-.07 (.06)	-.06 (.06)
Avg Weekly PAI-BOR Total		.14 (.08)
Avg PAI-BOR* Avg Estradiol		-.14 (.10)
<i>Avg PAI-BOR* Estradiol Deviation</i>		.04 (.07)
	Random Parameters	
Intercept	.36* (.09)	.33* (.08)
Weekly Deviations in Estradiol	.03+ (.03)	.03 (.03)
Residual	.15* (.02)	.15* (.01)
-2 log likelihood	262.1	257.7†

Note. Standard errors are in parentheses.

* $p < .05$.

+ $p < .15$.

†Change in -2 log likelihood over the previous model is significant at $p < .05$.

In the model predicting self-control, there was a significant interaction between trait BPD and deviations in estradiol such that higher-than-usual levels of estradiol were associated with higher self-control among women *higher* (+1 standard deviation) in trait BPD ($\gamma_{\text{HIGHTRAITBPD*ESTRADIOLDEVIATION}} = .28, SE = .09, t(117) = 2.96, p = .003$) but were not associated with self-control among women *lower* (-1 standard deviation) in trait BPD ($\gamma_{\text{LOWTRAITBPD*ESTRADIOLDEVIATION}} = -.09, SE = .07, t(117) = -1.21, p = .23$). A graph of this interaction can be found in Figure 13. Unexpectedly, there was also a significant interaction between trait BPD and average levels of estradiol such that higher average levels of estradiol were associated with higher levels of self-control among women *higher* in trait BPD ($\gamma_{\text{HIGHTRAITBPD*ESTRADIOLDEVIATION}} = .19, SE = .05, t(37) = 3.34, p = .001$), but was not associated with self-control among women *lower* in trait BPD ($\gamma_{\text{LOWTRAITBPD*ESTRADIOLDEVIATION}} = -.06, SE = .07, t(37) = -.88, p = .38$). A graph of this interaction can be found in Figure 14.

In the model predicting Negative Urgency, there was a significant interaction between trait BPD and deviations in estradiol such that higher-than-usual levels of estradiol were associated with lower Negative Urgency among women *higher* in trait BPD ($\gamma_{\text{HIGHTRAITBPD*ESTRADIOLDEVIATION}} = -.11, SE = .04, t(117) = -2.75, p = .006$) but were not associated with Negative Urgency among women *lower* in trait BPD ($\gamma_{\text{LOWTRAITBPD*ESTRADIOLDEVIATION}} = .01, SE = .04, t(117) = .24, p = .80$). A graph of this interaction can be found in Figure 15. Once again, there was also an unexpected interaction between trait BPD and average levels of estradiol such that higher average levels of estradiol were associated with lower levels of Negative Urgency among women

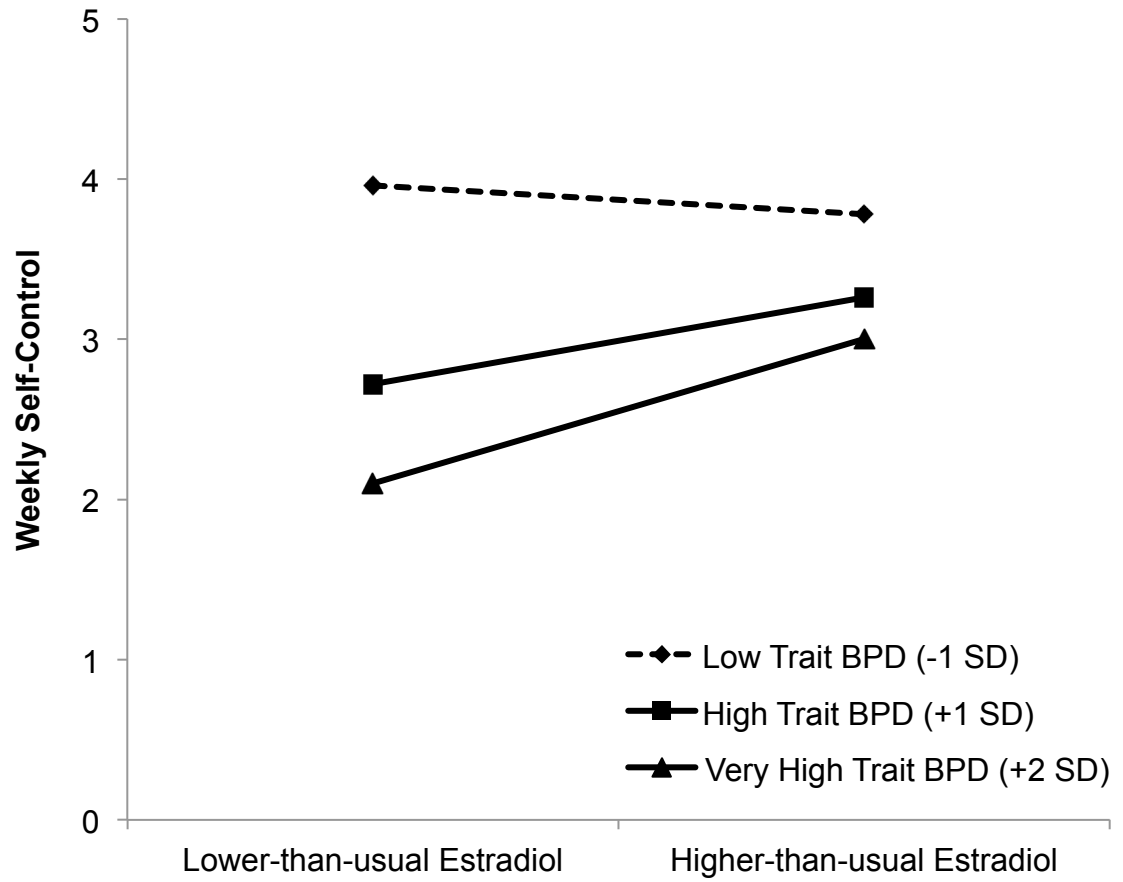


Figure 13. A graph of the interaction between trait BPD symptoms and *deviation* in estradiol predicting weekly self-control.

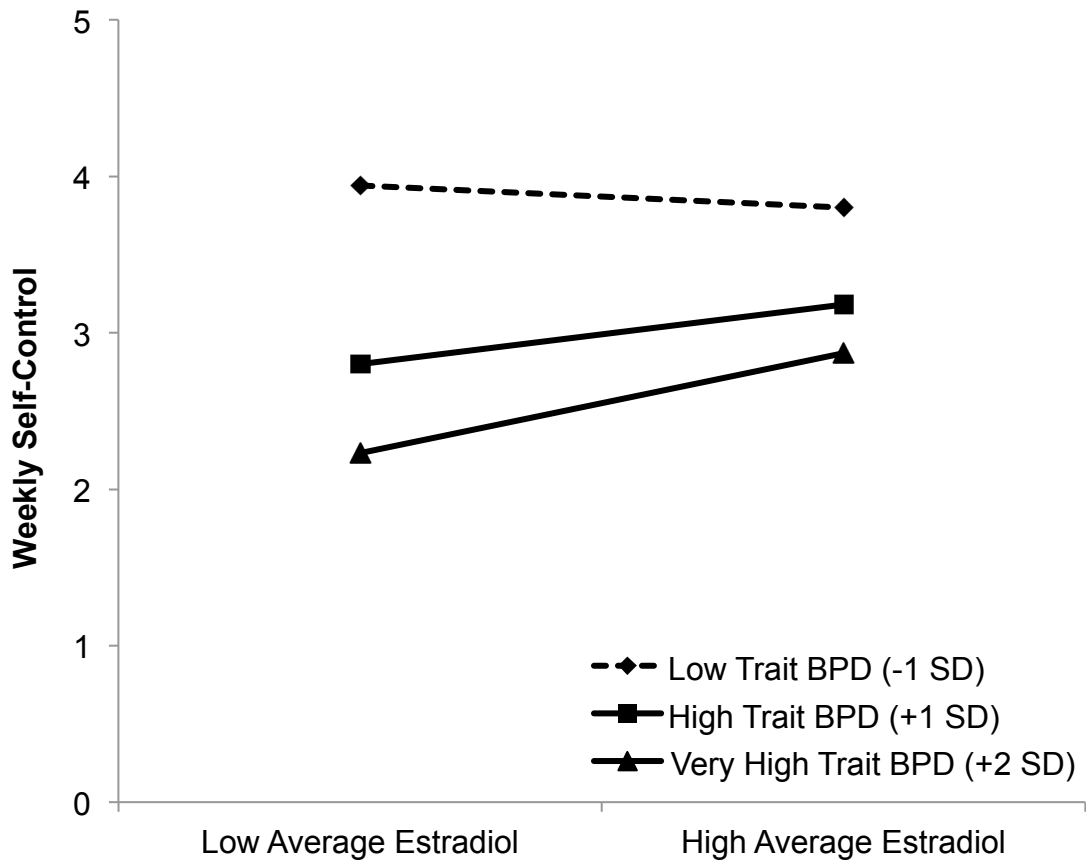


Figure 14. A graph of the interaction between trait BPD symptoms and *Average* estradiol predicting weekly self-control.

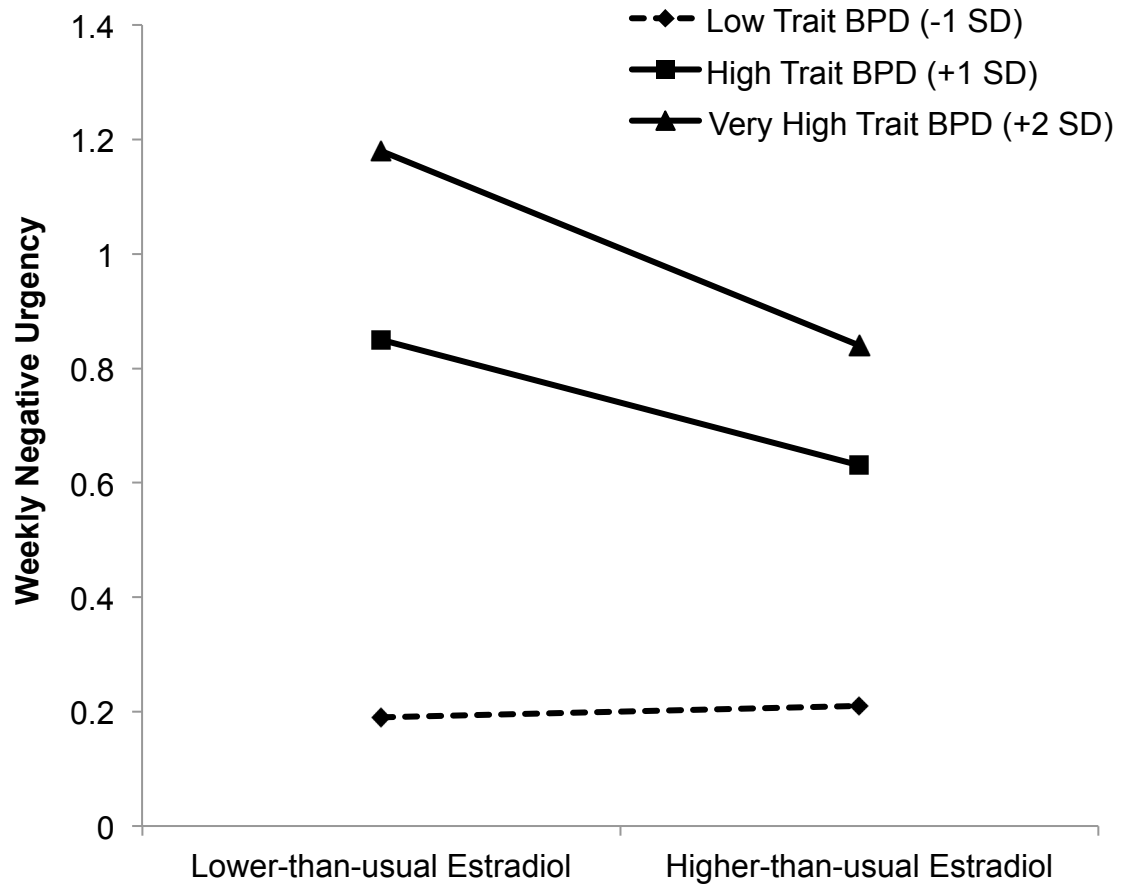


Figure 15. A graph of the interaction between trait BPD symptoms and deviation in estradiol predicting weekly UPPS-P negative urgency.

higher in trait BPD ($\gamma_{\text{HIGHTRAITBPD}*\text{AVERAGEESTRADIOL}} = -.07, SE = .03, t(37) = -2.10, p = .04$) but were not associated with levels of Negative Urgency among women *lower* in trait BPD ($\gamma_{\text{LOWTRAITBPD}*\text{AVERAGEESTRADIOL}} = -.003, SE = .04, t(37) = -.09, p = .93$). A graph of this interaction can be found in Figure 16.

In the model predicting Positive Urgency, there was a significant interaction between trait BPD and deviations in estradiol such that higher-than-usual levels of estradiol were associated with lower weekly levels of Positive Urgency among women *higher* in trait BPD ($\gamma_{\text{HIGHTRAITBPD}*\text{ESTRADIOLDEVIATION}} = -.20, SE = .07, t(117) = -2.90, p = .004$) but were not associated with Positive Urgency among women *lower* in trait BPD ($\gamma_{\text{LOWTRAITBPD}*\text{ESTRADIOLDEVIATION}} = .01, SE = .04, t(117) = .22, p = .82$). A graph of the interaction can be found in Figure 17.

In the model predicting Lack of Perseverance, there was a significant interaction between trait BPD and deviations in estradiol such that higher-than-usual levels of estradiol were associated with lower weekly Lack of Perseverance among women *higher* in trait BPD ($\gamma_{\text{HIGHTRAITBPD}*\text{ESTRADIOLDEVIATION}} = -.39, SE = .06, t(117) = -6.45, p < .0001$) but was not significantly associated with Lack of Perseverance among women *lower* in trait BPD ($\gamma_{\text{LOWTRAITBPD}*\text{ESTRADIOLDEVIATION}} = .06, SE = .07, t(117) = .88, p = .38$). The interaction is depicted in Figure 18.

Finally, in the model predicting Lack of Premeditation, there was a significant interaction between trait BPD and deviations in estradiol such that higher-than-usual levels of estradiol were associated with lower weekly Lack of Premeditation among women *higher* in trait BPD ($\gamma_{\text{HIGHTRAITBPD}*\text{ESTRADIOLDEVIATION}} = -.14, SE = .06, t(117) = -1.99, p = .04$) but was not significantly associated with Lack of Premeditation among

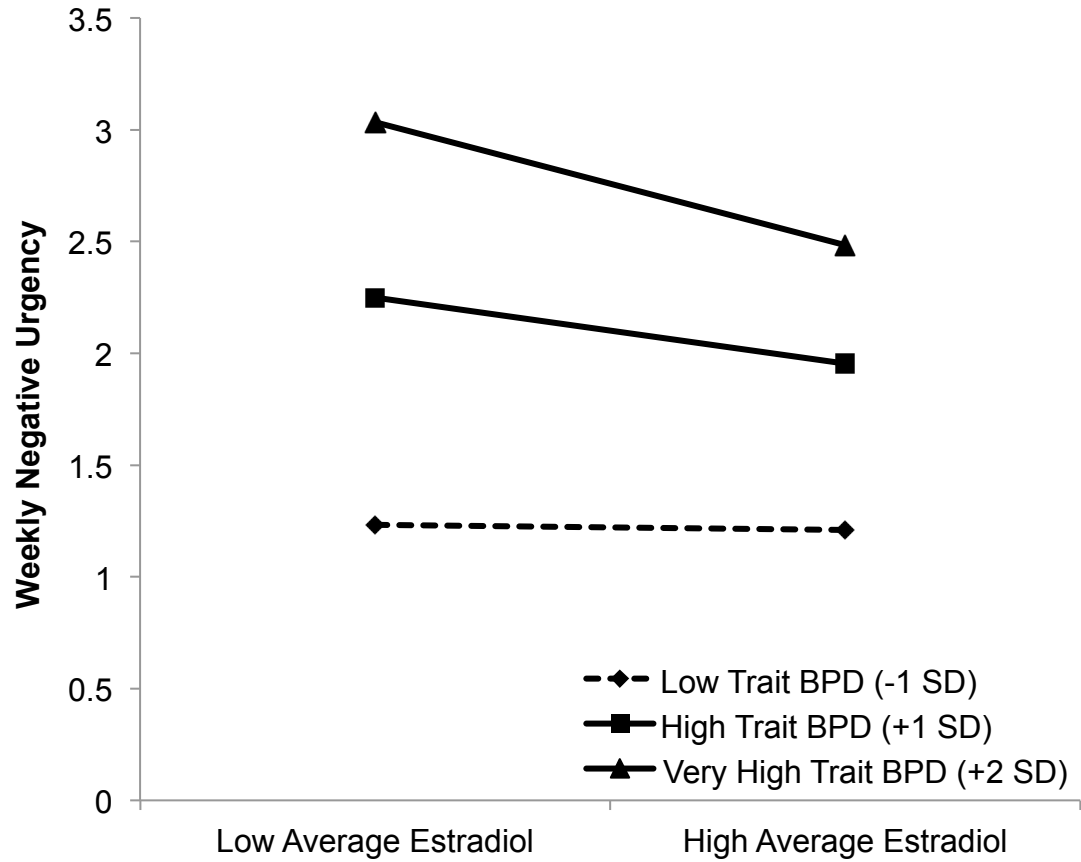


Figure 16. A graph of the interaction between trait BPD symptoms and average estradiol predicting weekly UPPS-P negative urgency.

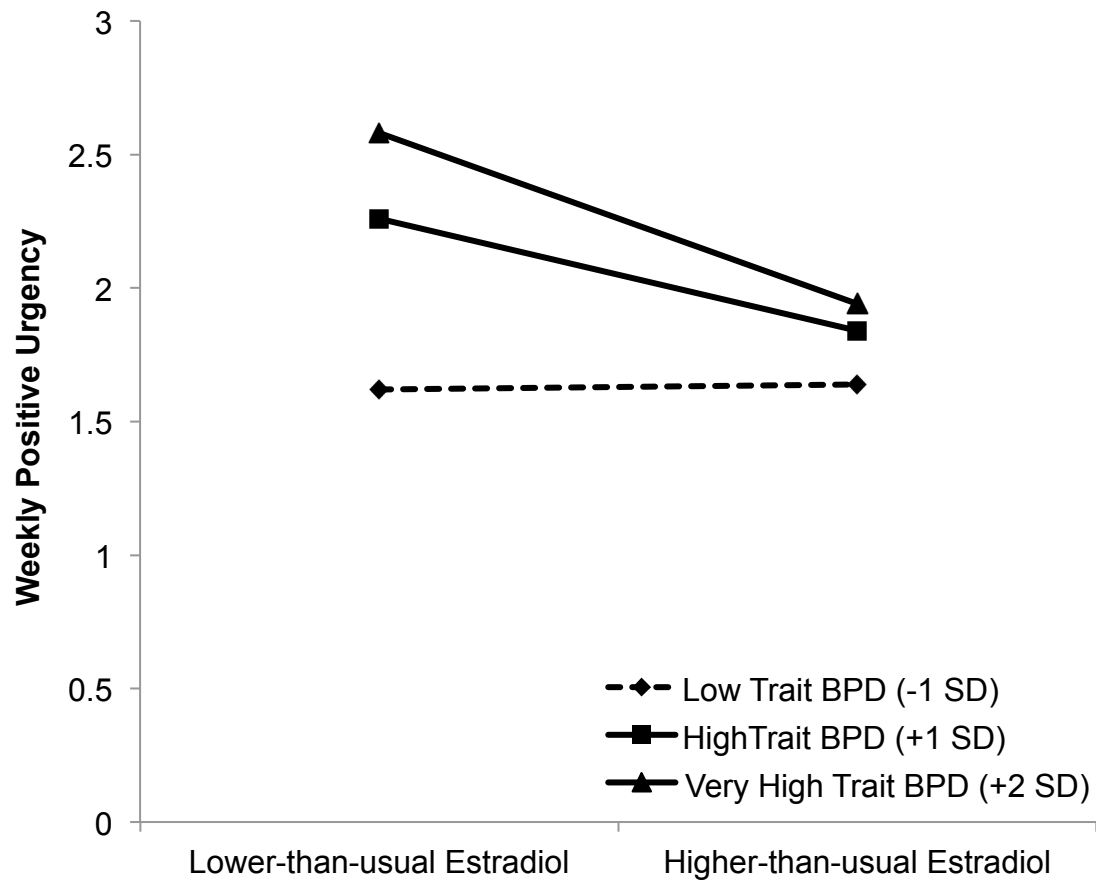


Figure 17. A graph of the interaction between trait BPD symptoms and *deviation* in estradiol predicting weekly UPPS-P positive urgency.

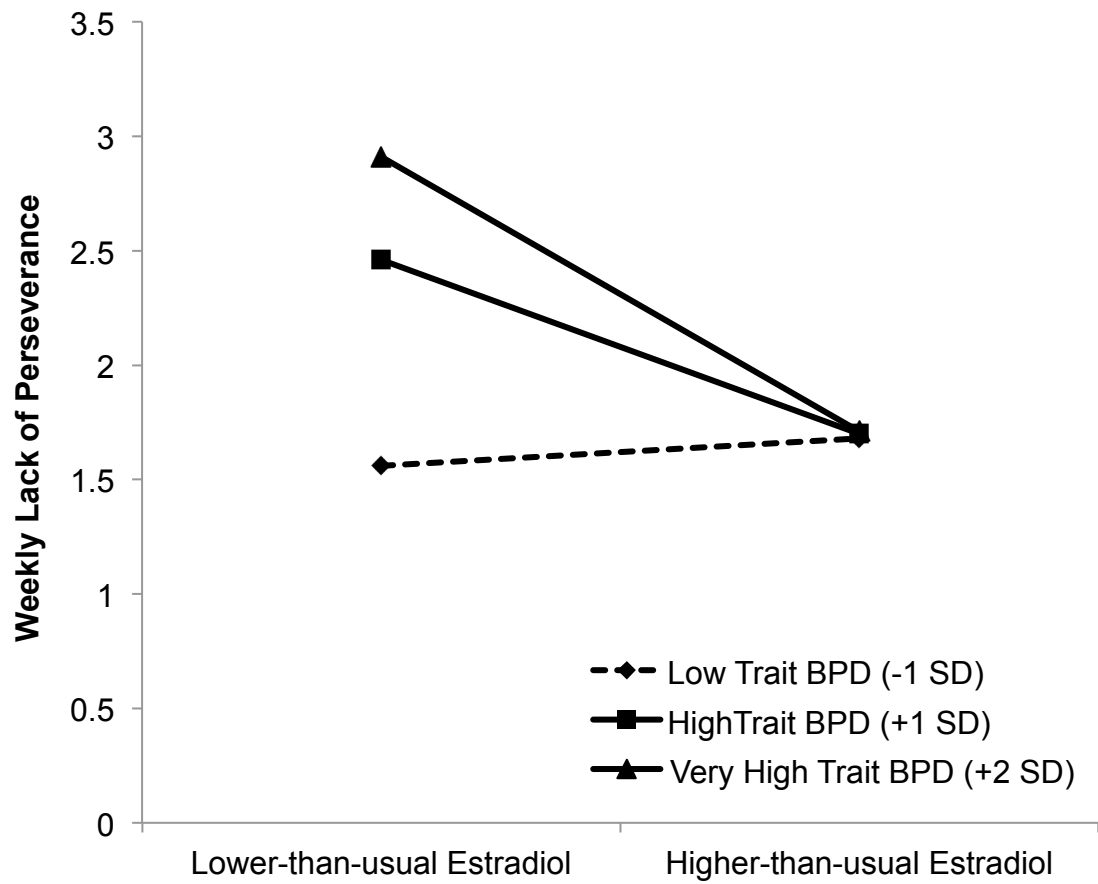


Figure 18. A graph of the interaction between trait BPD symptoms and *deviation* in estradiol predicting weekly UPPS-P lack of perseverance.

women *lower* in trait BPD ($\gamma_{\text{LOWTRAITBPD*ESTRADIOLDEVIATION}} = .05, SE = .05, t(117) = .96, p = .33$). See Figure 19 for a graph of the interaction. Once again, there was a significant unexpected interaction between average levels of estradiol and trait BPD such that higher average levels of estradiol were associated with lower Lack of Premeditation among women with *higher* levels of trait BPD ($\gamma_{\text{HIGHTRAITBPD*AVERAGEESTRADIOL}} = -.19, SE = .06, t(37) = -2.87, p = .006$) but were not associated with Lack of Premeditation among women with *lower* levels of trait BPD ($\gamma_{\text{LOWTRAITBPD*AVERAGEESTRADIOL}} = .05, SE = .10, t(37) = .49, p = .62$). See Figure 20 for a graph of this interaction.

Path B at the Weekly Level: Do changes in weekly self-control and impulsivity impact weekly BPD symptom expression? Next, I tested whether average levels of and weekly changes in self-control and impulsivity impact expression of BPD symptoms. Results of models regressing BPD symptoms on average levels of and deviations in self-control and UPPS-P impulsivity on BPD symptom expression are presented in Tables 18-23 and discussed in the following sections.

Self-Control. In models predicting BPD symptoms from average and weekly deviations in self-control, models including fixed effects of deviations in self-control significantly improved model fit. However, models including random effects of deviations in self-control did not significantly improve model fit, suggesting that the impact of changes in self-control on BPD symptoms is similar across individuals. In each case, both average levels of self-control and higher-than-usual levels of self-control were associated with lower BPD symptom expression.

Negative Urgency. In models predicting BPD symptoms from average and weekly deviations in Negative Urgency, model fit improved significantly with the inclusion of

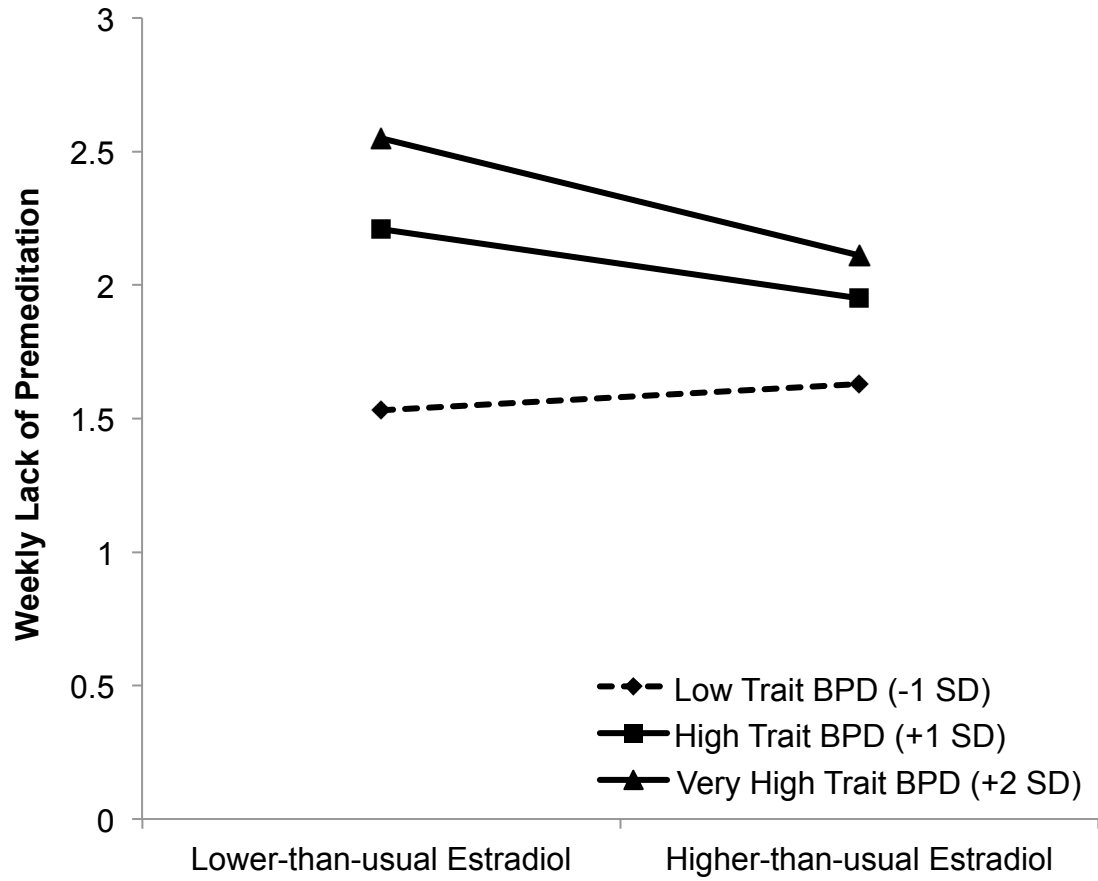


Figure 19. A graph of the interaction between trait BPD symptoms and *deviation* in estradiol predicting weekly UPPS-P lack of premeditation.

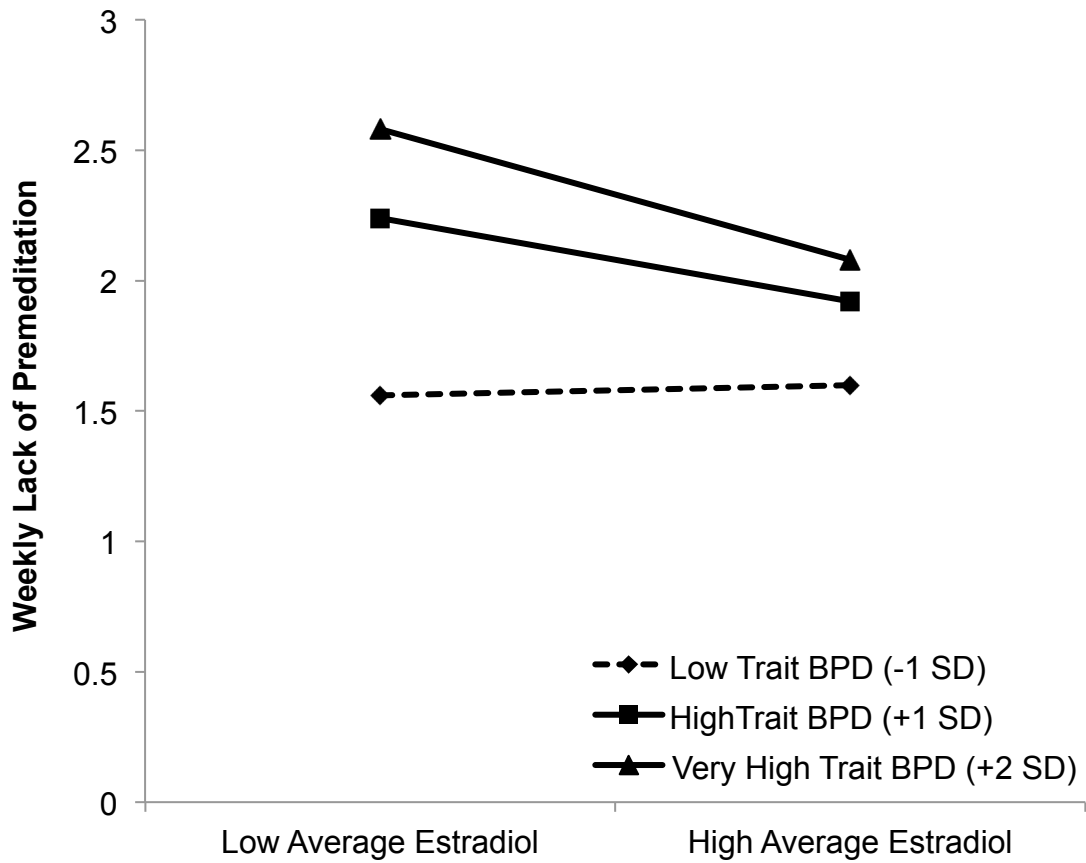


Figure 20. A graph of the interaction between trait BPD symptoms and *average* estradiol predicting weekly UPPS-P lack of premeditation.

Table 18

Multilevel Poisson Regression Models Predicting Weekly BPD Symptoms from Average Levels of Weekly Self-Control and Weekly Deviations in Self-Control

Parameter	Model 1 (null)	Model 2 (fixed deviation slope)	Model 3 (random deviation slope)
<u>Dependent Variable: PAI-BOR Total Score</u>			
		Fixed Effects	
Intercept	-.50 (.10)	-.51* (.07)	-.52* (.07)
Average Self-Control		-.45* (.07)	-.45* (.07)
<i>Deviations in Self-Control</i>		-.30* (.06)	-.30* (.06)
		Random Parameters	
Intercept	.39* (.09)	.18* (.05)	.18* (.05)
Deviations in Self-Control			.001 (.01)
Residual	.08* (.01)	.06* (.009)	.06* (.009)
-2 Restricted Log Pseudo-likelihood	240.55	202.85†	203.59
<u>Dependent Variable: PAI-BOR Affective Instability Subscale</u>			
		Fixed Effects	
Intercept	-.60 (.15)	-.62* (.11)	-.62* (.11)
Average Self-Control		-.62* (.11)	-.62* (.11)
<i>Deviations in Self-Control</i>		-.36* (.09)	-.36* (.09)
		Random Parameters	
Intercept	.83* (.23)	.43* (.13)	.42* (.13)
Deviations in Self-Control			.0001 (.03)
Residual	.16* (.02)	.15* (.02)	.15* (.02)
-2 Restricted Log Pseudo-likelihood	370.17	342.56†	342.24
<u>Dependent Variable: PAI-BOR Identity Disturbance Subscale</u>			
		Fixed Effects	
Intercept	-.30 (.11)	-.31* (.09)	-.31* (.09)
Average Self-Control		-.40* (.09)	-.40* (.09)
<i>Deviations in Self-Control</i>		-.25* (.07)	-.27* (.07)
		Random Parameters	
Intercept	.45* (.11)	.29* (.07)	.29* (.07)
Deviations in Self-Control			.02 (.03)
Residual	.12* (.01)	.11* (.01)	.10* (.01)
-2 Restricted Log Pseudo-likelihood	274.93	254.46†	254.59

Table Continued on Next Page

Dependent Variable: PAI-BOR Negative Relationships Subscale			
		Fixed Effects	
Intercept	-.41* (.12)	-.42* (.09)	-.42* (.09)
Average Self-Control		-.45* (.09)	-.45* (.10)
<i>Deviations in Self-Control</i>		-.25* (.09)	-.25* (.09)
		Random Parameters	
Intercept	.53* (.14)	.32* (.09)	.32* (.09)
Deviations in Self-Control			.0001 (.04)
Residual	.16* (.02)	.16* (.09)	.16* (.02)
-2 Restricted Log Pseudo-likelihood	332.16	316.38†	315.89
Dependent Variable: PAI-BOR Self-Harm Subscale			
		Fixed Effects	
Intercept	-1.03* (.11)	-1.05* (.09)	-1.05* (.09)
Average Self-Control		-.39* (.09)	-.39* (.09)
<i>Deviations in Self-Control</i>		-.41* (.14)	-.41* (.14)
		Random Parameters	
Intercept	.35* (.12)	.20* (.08)	.20* (.08)
Deviations in Self-Control			.0001 (.04)
Residual	.18* (.02)	.17* (.02)	.17* (.02)
-2 Restricted Log Pseudo-likelihood	407.23	391.96†	391.01
Dependent Variable: McLean Screening Instrument for BPD (MSI-BPD)			
		Fixed Effects	
Intercept	.17 (.22)	.14 (.18)	.13 (.18)
Average Self-Control		-.85* (.18)	-.86* (.18)
<i>Deviations in Self-Control</i>		-.40* (.11)	-.45* (.11)
		Random Parameters	
Intercept	1.80* (.54)	1.09* (.34)	1.12* (.35)
Deviations in Self-Control			.02 (.03)
Residual	.68* (.09)	.64* (.08)	.62* (.08)
-2 Restricted Log Pseudo-likelihood	481.04	462.90†	462.42
Dependent Variable: Borderline Symptom Checklist—23 (BSL-23)			
		Fixed Effects	
Intercept	2.05 (.16)	2.01* (.14)	2.00* (.14)
Average Self-Control		-.63* (.14)	-.63* (.14)
<i>Deviations in Self-Control</i>		-.40* (.08)	-.41* (.08)
		Random Parameters	
Intercept	1.06* (.29)	.75* (.20)	.75* (.20)
Deviations in Self-Control			.009 (.03)
Residual	2.34* (.31)	1.91* (.25)	1.86* (.30)
-2 Restricted Log Pseudo-likelihood	379.72	350.31†	350.41

Note. Standard errors are in parentheses.

* $p < .05$.

†Change in -2 Restricted Log Pseudo-likelihood over the previous model is significant at $p < .05$.

Table 19

Multilevel Poisson Regression Models Predicting Weekly BPD Symptoms from Average Levels of Weekly UPPS-P Negative Urgency and Weekly Deviations in UPPS-P Negative Urgency

Parameter	Model 1 (null)	Model 2 (fixed deviation slope)	Model 3 (random deviation slope)
<u>Dependent Variable: PAI-BOR Total Score</u>			
		Fixed Effects	
Intercept	-.50 (.10)	-.52* (.06)	-.52* (.06)
Average Negative Urgency		.49* (.06)	.50* (.06)
<i>Deviations in Negative Urgency</i>		.36* (.06)	.40* (.08)
		Random Parameters	
Intercept	.39* (.09)	.13* (.03)	.13* (.03)
Deviations in Negative Urgency			.03 (.02)
Residual	.08* (.01)	.06* (.008)	.05* (.007)
-2 Restricted Log Pseudo-likelihood	240.55	180.48†	172.80†
<u>Dependent Variable: PAI-BOR Affective Instability Subscale</u>			
		Fixed Effects	
Intercept	-.60 (.15)	-.63* (.10)	-.65* (.10)
Average Negative Urgency		.64* (.10)	.65* (.10)
<i>Deviations in Negative Urgency</i>		.51* (.09)	.58* (.12)
		Random Parameters	
Intercept	.83* (.23)	.37* (.11)	.40* (.12)
Deviations in Negative Urgency			.09 (.07)
Residual	.16* (.02)	.13* (.01)	.12* (.01)
-2 Restricted Log Pseudo-likelihood	370.17	322.53†	322.48
<u>Dependent Variable: PAI-BOR Identity Disturbance Subscale</u>			
		Fixed Effects	
Intercept	-.30 (.11)	-.32* (.07)	-.32* (.07)
Average Negative Urgency		.52* (.06)	.53* (.07)
<i>Deviations in Negative Urgency</i>		.31* (.06)	.39* (.08)
		Random Parameters	
Intercept	.45* (.11)	.16* (.04)	.16* (.04)
Deviations in Negative Urgency			.03 (.03)
Residual	.12* (.01)	.10* (.01)	.09* (.01)
-2 Restricted Log Pseudo-likelihood	274.93	226.11†	222.81†
<u>Dependent Variable: PAI-BOR Negative Relationships Subscale</u>			
		Fixed Effects	
Intercept	-.41* (.12)	-.43* (.08)	-.43* (.09)
Average Negative Urgency		.50* (.08)	.50* (.08)
<i>Deviations in Negative Urgency</i>		.36* (.09)	.39* (.11)
		Random Parameters	
Intercept	.53* (.14)	.26* (.08)	.26* (.08)
Deviations in Negative Urgency			.03 (.07)
Residual	.16* (.02)	.15* (.02)	.14* (.02)
-2 Restricted Log Pseudo-likelihood	332.16	302.14†	303.40

Table Continued on Next Page

Dependent Variable: PAI-BOR Self-Harm Subscale			
		Fixed Effects	
Intercept	-1.03* (.11)	-1.04* (.09)	-1.04* (.09)
Average Negative Urgency		.29* (.09)	.29* (.09)
<i>Deviations in Negative Urgency</i>		.20* (.10)	.20* (.10)
		Random Parameters	
Intercept	.35* (.12)	.26* (.10)	.26* (.10)
Deviations in Negative Urgency			.01 (.08)
Residual	.18* (.02)	.18* (.02)	.18* (.02)
-2 Restricted Log Pseudo-likelihood	407.23	403.45†	403.59
Dependent Variable: McLean Screening Instrument for BPD (MSI-BPD)			
		Fixed Effects	
Intercept	.17 (.22)	.10 (.17)	.06* (.18)
Average Negative Urgency		.93* (.17)	.95* (.17)
<i>Deviations in Negative Urgency</i>		.59* (.10)	.70* (.16)
		Random Parameters	
Intercept	1.80* (.54)	1.01* (.32)	1.11* (.35)
Deviations in Negative Urgency			.24 (.20)
Residual	.68* (.09)	.55* (.07)	.48* (.16)
-2 Restricted Log Pseudo-likelihood	481.04	444.98†	451
Dependent Variable: Borderline Symptom Checklist—23 (BSL-23)			
		Fixed Effects	
Intercept	2.05 (.16)	2.00* (.13)	1.99* (.13)
Average Negative Urgency		.70* (.13)	.71* (.13)
<i>Deviations in Negative Urgency</i>		.47* (.07)	.53* (.11)
		Random Parameters	
Intercept	1.06* (.29)	.61* (.17)	.64* (.18)
Deviations in Negative Urgency			.09+ (.06)
Residual	2.34* (.31)	1.74* (.23)	1.53* (.21)
-2 Restricted Log Pseudo-likelihood	379.72	332.24†	329.89†

Note. Standard errors are in parentheses. CP = Daily Conception Probability.

* $p < .05$.

+ $p < .15$.

†Change in -2 Restricted Log Pseudo-likelihood over the previous model is significant at $p < .05$.

Table 20

Multilevel Poisson Regression Models Predicting Weekly BPD Symptoms from Average Levels of Weekly UPPS-P Positive Urgency and Weekly Deviations in UPPS-P Positive Urgency

Parameter	Model 1 (null)	Model 2 (fixed deviation slope)	Model 3 (random deviation slope)
<u>Dependent Variable: PAI-BOR Total Score</u>			
		Fixed Effects	
Intercept	-.50 (.10)	-.51* (.09)	-.51* (.09)
Average Positive Urgency		.29* (.09)	.29* (.09)
<i>Deviations in Positive Urgency</i>		.14* (.07)	.15+ (.10)
		Random Parameters	
Intercept	.39* (.09)	.31* (.08)	.31* (.08)
Deviations in Positive Urgency			.03 (.08)
Residual	.08* (.01)	.07* (.01)	.07* (.01)
-2 Restricted Log Pseudo-likelihood	240.55	235†	235.19
<u>Dependent Variable: PAI-BOR Affective Instability Subscale</u>			
		Fixed Effects	
Intercept	-.60 (.15)	-.61* (.15)	-.61* (.14)
Average Positive Urgency		.36* (.14)	.36* (.14)
<i>Deviations in Positive Urgency</i>		.13 (.11)	.13 (.11)
		Random Parameters	
Intercept	.83* (.23)	.71* (.20)	.71* (.20)
Deviations in Positive Urgency			.0001 (.08)
Residual	.16* (.02)	.16* (.02)	.16* (.02)
-2 Restricted Log Pseudo-likelihood	370.17	367.28†	367.28
<u>Dependent Variable: PAI-BOR Identity Disturbance Subscale</u>			
		Fixed Effects	
Intercept	-.30 (.11)	-.30* (.10)	-.31* (.10)
Average Positive Urgency		.25* (.10)	.25* (.10)
<i>Deviations in Positive Urgency</i>		.12 (.09)	.15 (.11)
		Random Parameters	
Intercept	.45* (.11)	.39* (.10)	.40* (.10)
Deviations in Positive Urgency			.11 (.13)
Residual	.12* (.01)	.12* (.01)	.11 (.01)
-2 Restricted Log Pseudo-likelihood	274.93	271.76†	273.56
<u>Dependent Variable: PAI-BOR Negative Relationships Subscale</u>			
		Fixed Effects	
Intercept	-.41* (.12)	-.42* (.11)	-.42* (.11)
Average Positive Urgency		.31* (.11)	.31* (.11)
<i>Deviations in Positive Urgency</i>		.17* (.09)	.17* (.09)
		Random Parameters	
Intercept	.53* (.14)	.45* (.12)	.45* (.13)
Deviations in Positive Urgency			.0001 (.07)
Residual	.16* (.02)	.16* (.02)	.16* (.02)
-2 Restricted Log Pseudo-likelihood	332.16	328.31†	328.31

Table Continued on Next Page

Dependent Variable: PAI-BOR Self-Harm Subscale			
		Fixed Effects	
Intercept	-1.03* (.11)	-1.03* (.10)	-1.05* (.10)
Average Positive Urgency		.26* (.10)	.26* (.10)
<i>Deviations in Positive Urgency</i>		.17 (.25)	.20 (.19)
		Random Parameters	
Intercept	.35* (.12)	.27* (.10)	.29* (.11)
<i>Deviations in Positive Urgency</i>			.25 (.29)
Residual	.18* (.02)	.18* (.02)	.17* (.02)
-2 Restricted Log Pseudo-likelihood	407.23	405.26†	406.49
Dependent Variable: McLean Screening Instrument for BPD (MSI-BPD)			
		Fixed Effects	
Intercept	.17 (.22)	.15 (.21)	.15 (.21)
Average Positive Urgency		.54* (.21)	.54* (.21)
<i>Deviations in Positive Urgency</i>		.29* (.13)	.29* (.12)
		Random Parameters	
Intercept	1.80* (.54)	1.58* (.48)	1.58* (.48)
<i>Deviations in Positive Urgency</i>			.0001 (.04)
Residual	.68* (.09)	.67* (.09)	.67* (.10)
-2 Restricted Log Pseudo-likelihood	481.04	478.24†	478.20
Dependent Variable: Borderline Symptom Checklist—23 (BSL-23)			
		Fixed Effects	
Intercept	2.05 (.16)	2.02* (.16)	2.00* (.16)
Average Positive Urgency		.38* (.16)	.39* (.16)
<i>Deviations in Positive Urgency</i>		.23* (.10)	.23 (.17)
		Random Parameters	
Intercept	1.06* (.29)	.99* (.22)	1.05* (.28)
<i>Deviations in Positive Urgency</i>			.39 (.22)
Residual	2.34* (.31)	2.24* (.29)	1.80* (.27)
-2 Restricted Log Pseudo-likelihood	379.72	354.81†	375.27

Note. Standard errors are in parentheses.

* $p < .05$.

† $p < .15$.

†Change in -2 Restricted Log Pseudo-likelihood over the previous model is significant at $p < .05$.

Table 21

Multilevel Poisson Regression Models Predicting Weekly BPD Symptoms from Average Levels of Weekly UPPS-P Lack of Premeditation and Weekly Deviations in UPPS-P Lack of Premeditation

Parameter	Model 1 (null)	Model 2 (fixed deviation slope)	Model 3 (random deviation slope)
<u>Dependent Variable: PAI-BOR Total Score</u>			
		Fixed Effects	
Intercept	-.50 (.10)	-.52* (.09)	-.52* (.09)
Average Lack of Premeditation <i>Deviations in Lack of Premeditation</i>		.25* (.09)	.25* (.09)
		.34* (.06)	.35* (.07)
		Random Parameters	
Intercept	.39* (.09)	.33* (.08)	.34* (.08)
Deviations in Lack of Premeditation			.01 (.03)
Residual	.08* (.01)	.06* (.008)	.06* (.008)
-2 Restricted Log Pseudo-likelihood	240.55	216.89†	216.50
<u>Dependent Variable: PAI-BOR Affective Instability Subscale</u>			
		Fixed Effects	
Intercept	-.60* (.15)	-.64* (.14)	-.65* (.14)
Average Lack of Premeditation <i>Deviations in Lack of Premeditation</i>		.32* (.14)	.32* (.14)
		.52* (.09)	.54* (.13)
		Random Parameters	
Intercept	.83* (.23)	.78 (.22)	.81* (.23)
Deviations in Lack of Premeditation			.15 (.14)
Residual	.16* (.02)	.13* (.01)	.12* (.01)
-2 Restricted Log Pseudo-likelihood	370.17	351.33†	353.86
<u>Dependent Variable: PAI-BOR Identity Disturbance Subscale</u>			
		Fixed Effects	
Intercept	-.30 (.11)	-.31* (.10)	-.31* (.10)
Average Lack of Premeditation <i>Deviations in Lack of Premeditation</i>		.21* (.10)	.21* (.10)
		.24* (.07)	.26* (.08)
		Random Parameters	
Intercept	.45* (.11)	.41* (.10)	.41* (.10)
Deviations in Lack of Premeditation			.01 (.03)
Residual	.12* (.01)	.11* (.01)	.11* (.01)
-2 Restricted Log Pseudo-likelihood	274.93	267.26†	267.11
<u>Dependent Variable: PAI-BOR Negative Relationships Subscale</u>			
		Fixed Effects	
Intercept	-.41* (.12)	-.42* (.11)	-.42* (.11)
Average Lack of Premeditation <i>Deviations in Lack of Premeditation</i>		.22* (.11)	.21* (.11)
		.29* (.09)	.28* (.09)
		Random Parameters	
Intercept	.53* (.14)	.49* (.13)	.48* (.11)
Deviations in Lack of Premeditation			
Residual	.16* (.02)	.15* (.02)	.15* (.01)
-2 Restricted Log Pseudo-likelihood	332.16	327.50†	326.70

Table Continued on Next Page

Dependent Variable: PAI-BOR Self-Harm Subscale			
		Fixed Effects	
Intercept	-1.03* (.11)	-1.04* (.09)	-1.06* (.09)
Average Lack of Premeditation		.32* (.09)	.32* (.09)
<i>Deviations in Lack of Premeditation</i>		.39* (.14)	.41* (.18)
		Random Parameters	
Intercept	.35* (.12)	.23* (.09)	.25* (.10)
Deviations in Lack of Premeditation			.22 (.30)
Residual	.18* (.02)	.18* (.02)	.16* (.02)
-2 Restricted Log Pseudo-likelihood	407.23	399.10†	398.67
Dependent Variable: McLean Screening Instrument for BPD (MSI-BPD)			
		Fixed Effects	
Intercept	.17 (.22)	.14 (.22)	.10 (.22)
Average Lack of Premeditation		.42* (.21)	.42 (.22)
<i>Deviations in Lack of Premeditation</i>		.41* (.11)	.45* (.18)
		Random Parameters	
Intercept	1.80* (.54)	1.73* (.52)	1.83* (.55)
Deviations in Lack of Premeditation			.30 (.26)
Residual	.68* (.09)	.63* (.08)	.57* (.09)
-2 Restricted Log Pseudo-likelihood	481.04	476.74†	483.59
Dependent Variable: Borderline Symptom Checklist—23 (BSL-23)			
		Fixed Effects	
Intercept	2.05 (.16)	2.01* (.16)	1.98* (.17)
Average Lack of Premeditation		.25* (.12)	.24* (.12)
<i>Deviations in Lack of Premeditation</i>		.43* (.08)	.43* (.14)
		Random Parameters	
Intercept	1.06* (.29)	1.08* (.29)	1.17* (.31)
Deviations in Lack of Premeditation			.26 (.15)
Residual	2.34* (.31)	1.94* (.26)	1.48* (.22)
-2 Restricted Log Pseudo-likelihood	379.72	364.74†	364.70

Note. Standard errors are in parentheses.

* $p < .05$.

†Change in -2 Restricted Log Pseudo-likelihood over the previous model is significant at $p < .05$.

Table 22

Multilevel Poisson Regression Models Predicting Weekly BPD Symptoms from Average Levels of Weekly UPPS-P Lack of Perseverance and Weekly Deviations in UPPS-P Lack of Perseverance

Parameter	Model 1 (null)	Model 2 (fixed deviation slope)	Model 3 (random deviation slope)
<u>Dependent Variable: PAI-BOR Total Score</u>			
		Fixed Effects	
Intercept	-.50 (.10)	-.51* (.09)	-.51* (.09)
Average Lack of Perseverance <i>Deviations in Lack of Perseverance</i>		.28* (.09) .26* (.06)	.28* (.09) .25* (.08)
		Random Parameters	
Intercept	.39* (.09)	.32* (.08)	.32* (.08)
Deviations in Lack of Perseverance			.04 (.04)
Residual	.08* (.01)	.07* (.009)	.06* (.009)
-2 Restricted Log Pseudo-likelihood	240.55	226.66†	225.66
<u>Dependent Variable: PAI-BOR Affective Instability Subscale</u>			
		Fixed Effects	
Intercept	-.60 (.15)	-.63* (.13)	-.64* (.14)
Average Lack of Perseverance <i>Deviations in Lack of Perseverance</i>		.44* (.13) .43* (.09)	.45* (.14) .43* (.12)
		Random Parameters	
Intercept	.83* (.23)	.69* (.19)	.72* (.20)
Deviations in Lack of Perseverance			.11 (.09)
Residual	.16* (.02)	.14* (.01)	.13* (.01)
-2 Restricted Log Pseudo-likelihood	370.17	353.09†	354.71
<u>Dependent Variable: PAI-BOR Identity Disturbance Subscale</u>			
		Fixed Effects	
Intercept	-.30 (.11)	-.31* (.10)	-.31* (.10)
Average Lack of Perseverance <i>Deviations in Lack of Perseverance</i>		.20* (.10) .28* (.07)	.20* (.10) .28* (.07)
		Random Parameters	
Intercept	.45* (.11)	.42* (.10)	.42* (.10)
Deviations in Lack of Perseverance			.0001 (.03)
Residual	.12* (.01)	.11* (.01)	.11* (.01)
-2 Restricted Log Pseudo-likelihood	274.93	267.37†	267.30
<u>Dependent Variable: PAI-BOR Negative Relationships Subscale</u>			
		Fixed Effects	
Intercept	-.41* (.12)	-.42* (.11)	-.42* (.11)
Average Lack of Perseverance <i>Deviations in Lack of Perseverance</i>		.23* (.11) .21* (.10)	.23* (.11) .22* (.11)
		Random Parameters	
Intercept	.53* (.14)	.49* (.13)	.49* (.14)
Deviations in Lack of Perseverance			.03 (.07)
Residual	.16* (.02)	.16* (.02)	.15* (.02)
-2 Restricted Log Pseudo-likelihood	332.16	322.42†	330.69

Table Continued on Next Page

Dependent Variable: PAI-BOR Self-Harm Subscale			
		Fixed Effects	
Intercept	-1.03* (.11)	-1.03* (.09)	-1.06* (.09)
Average Lack of Perseverance		.32* (.09)	.32* (.09)
<i>Deviations in Lack of Perseverance</i>		.06 (.13)	-.03 (.21)
		Random Parameters	
Intercept	.35* (.12)	.24* (.09)	.27* (.10)
<i>Deviations in Lack of Perseverance</i>			.52 (.36)
Residual	.18* (.02)	.18* (.02)	.15* (.02)
-2 Restricted Log Pseudo-likelihood	407.23	400.24†	403.53
Dependent Variable: McLean Screening Instrument for BPD (MSI-BPD)			
		Fixed Effects	
Intercept	.17 (.22)	.13 (.21)	.13 (.21)
Average Lack of Perseverance		.50* (.21)	.51* (.21)
<i>Deviations in Lack of Perseverance</i>		.49* (.12)	.46* (.14)
		Random Parameters	
Intercept	1.80* (.54)	1.69* (.51)	1.71* (.51)
<i>Deviations in Lack of Perseverance</i>			.06 (.11)
Residual	.68* (.09)	.64* (.08)	.62* (.08)
-2 Restricted Log Pseudo-likelihood	481.04	478.44†	479.57
Dependent Variable: Borderline Symptom Checklist—23 (BSL-23)			
		Fixed Effects	
Intercept	2.05 (.16)	1.98* (.17)	1.98* (.17)
Average Lack of Perseverance		.40* (.17)	.41* (.17)
<i>Deviations in Lack of Perseverance</i>		.59* (.08)	.53* (.11)
		Random Parameters	
Intercept	1.06* (.29)	1.09* (.29)	1.10* (.29)
<i>Deviations in Lack of Perseverance</i>			.09 (.08)
Residual	2.34* (.31)	1.58* (.21)	1.45* (.21)
-2 Restricted Log Pseudo-likelihood	379.72	345.95†	345.14

Note. Standard errors are in parentheses. CP = Daily Conception Probability.

* $p < .05$.

†Change in -2 Restricted Log Pseudo-likelihood over the previous model is significant at $p < .05$.

Table 23

Multilevel Poisson Regression Models Predicting Weekly BPD Symptoms from Average Levels of Weekly UPPS-P Sensation Seeking and Weekly Deviations in UPPS-P Sensation Seeking

Parameter	Model 1 (null)	Model 2 (fixed deviation slope)	Model 3 (random deviation slope)
<u>Dependent Variable: PAI-BOR Total Score</u>			
		Fixed Effects	
Intercept	-.50 (.10)	-.50* (.10)	-.51* (.10)
Average Sensation Seeking		.11 (.10)	.11 (.10)
<i>Deviations in Sensation Seeking</i>		-.03 (.06)	-.03 (.08)
		Random Parameters	
Intercept	.39* (.09)	.39* (.09)	.39* (.09)
Deviations in Sensation Seeking			.04 (.04)
Residual	.08* (.01)	.08* (.01)	.07* (.01)
-2 Restricted Log Pseudo-likelihood	240.55	239.37	240.15
<u>Dependent Variable: PAI-BOR Affective Instability Subscale</u>			
		Fixed Effects	
Intercept	-.60 (.15)	-.61* (.15)	-.62* (.15)
Average Sensation Seeking		.06 (.15)	.06 (.15)
<i>Deviations in Sensation Seeking</i>		-.007 (.09)	-.02 (.12)
		Random Parameters	
Intercept	.83* (.23)	.85* (.24)	.88* (.24)
Deviations in Sensation Seeking			.13 (.11)
Residual	.16* (.02)	.16* (.02)	.14* (.01)
-2 Restricted Log Pseudo-likelihood	370.17	369.08	370.03
<u>Dependent Variable: PAI-BOR Identity Disturbance Subscale</u>			
		Fixed Effects	
Intercept	-.30 (.11)	-.30* (.10)	-.31* (.10)
Average Sensation Seeking		.15 (.10)	.15 (.10)
<i>Deviations in Sensation Seeking</i>		-.10 (.07)	-.10 (.08)
		Random Parameters	
Intercept	.45* (.11)	.44* (.11)	.44* (.11)
Deviations in Sensation Seeking			.02 (.04)
Residual	.12* (.01)	.12* (.01)	.11* (.01)
-2 Restricted Log Pseudo-likelihood	274.93	274.29	273.24
<u>Dependent Variable: PAI-BOR Negative Relationships Subscale</u>			
		Fixed Effects	
Intercept	-.41* (.12)	-.42* (.12)	-.42* (.12)
Average Sensation Seeking		.14 (.11)	.14 (.12)
<i>Deviations in Sensation Seeking</i>		.01 (.09)	.006 (.10)
		Random Parameters	
Intercept	.53* (.14)	.52* (.14)	.53* (.14)
Deviations in Sensation Seeking			.04 (.07)
Residual	.16* (.02)	.16* (.02)	.16 (.02)
-2 Restricted Log Pseudo-likelihood	332.16	329.15	331.99

Table Continued on Next Page

Dependent Variable: PAI-BOR Self-Harm Subscale			
		Fixed Effects	
Intercept	-1.03* (.11)	-1.03* (.11)	-1.06* (.11)
Average Sensation Seeking		.04 (.11)	.04 (.11)
<i>Deviations in Sensation Seeking</i>		-.01 (.14)	.08 (.19)
		Random Parameters	
Intercept	.35* (.12)	.36* (.12)	.39* (.13)
Deviations in Sensation Seeking			.34 (.27)
Residual	.18* (.02)	.18* (.02)	.16* (.02)
-2 Restricted Log Pseudo-likelihood	407.23	406.88	412.39
Dependent Variable: McLean Screening Instrument for BPD (MSI-BPD)			
		Fixed Effects	
Intercept	.17 (.22)	.14 (.21)	.12 (.22)
Average Sensation Seeking		.47* (.21)	.48* (.21)
<i>Deviations in Sensation Seeking</i>		-.04 (.11)	.02 (.14)
		Random Parameters	
Intercept	1.80* (.54)	1.67* (.50)	1.72* (.52)
Deviations in Sensation Seeking			.12 (.12)
Residual	.68* (.09)	.68* (.09)	.64* (.09)
-2 Restricted Log Pseudo-likelihood	481.04	479.09†	480.16
Dependent Variable: Borderline Symptom Checklist—23 (BSL-23)			
		Fixed Effects	
Intercept	2.05 (.16)	2.03* (.16)	1.99* (.17)
Average Sensation Seeking		.25* (.12)	.27* (.12)
<i>Deviations in Sensation Seeking</i>		.07 (.09)	.10 (.14)
		Random Parameters	
Intercept	1.06* (.29)	1.03* (.28)	1.12* (.30)
Deviations in Sensation Seeking			.25* (.12)
Residual	2.34* (.31)	2.32* (.32)	1.70* (.25)
-2 Restricted Log Pseudo-likelihood	379.72	371.19†	378.42

Note. Standard errors are in parentheses.

* $p < .05$.

†Change in -2 Restricted Log Pseudo-likelihood over the previous model is significant at $p < .05$.

fixed effects of average Negative Urgency and deviations in Negative Urgency. Further, in the case of models predicting the PAI-BOR total score, the PAI-BOR Identity Disturbance subscale, and the BSL-23, model fit improved further with the addition of random effects of deviations in Negative Urgency, indicating that the impact of changes in impulsivity on BPD symptoms may differ across individuals. In each case, both higher average levels of Negative Urgency and higher-than-usual Negative Urgency were associated with increased expression of BPD symptoms.

Positive Urgency. In models predicting BPD symptoms from average levels of and weekly deviations in Positive Urgency, model fit improved significantly with the inclusion of fixed effects of average and weekly deviations in Positive Urgency. They were not further improved by the addition of random effects of deviations in Positive Urgency, suggesting that the impact of fluctuations in Positive Urgency on BPD symptoms are similar across individuals. In the models predicting the PAI-BOR total score, the Negative Relationships subscale of the PAI-BOR, the MSI-BPD, and the BSL-23, both average levels of Positive Urgency and deviations in Positive Urgency were associated with higher expression of BPD symptoms. In the models predicting the PAI-BOR Self-Harm subscale, the PAI-BOR Identity Disturbance subscale, and the PAI-BOR Affective Instability subscale, only higher average levels of Positive Urgency were associated with higher BPD symptom expression.

Lack of Premeditation. In models predicting BPD symptoms from average and weekly deviations in Lack of Premeditation, model fit was improved significantly by the inclusion of fixed effects of average and weekly deviations in Lack of Premeditation, but was not further improved by the inclusion of the random effect of weekly deviations in

Lack of Premeditation, indicating that the impact of changes in lack of premeditation on BPD symptoms was similar across individuals. In each model, both higher average levels of Lack of Premeditation and higher-than-usual Lack of Premeditation were associated with greater BPD symptom expression.

Lack of Perseverance. In models predicting BPD symptoms from average and weekly deviations in Lack of Perseverance, model fit was improved significantly by the inclusion of the fixed effects of average and weekly deviations in Lack of Perseverance, but was not further improved by the inclusion of random effects of deviations in Lack of Perseverance, indicating that the impact of changes in Lack of Perseverance on BPD symptoms were similar across individuals. In each case except the model predicting the PAI-BOR Self-Harm subscale, both higher average levels of Lack of Perseverance and higher-than-usual Lack of Perseverance were associated with greater expression of BPD symptoms. In the model predicting the PAI-BOR Self-Harm subscale, only average levels of Lack of Perseverance were associated with higher BPD symptom expression.

Sensation Seeking. In models predicting BPD symptoms from average and weekly deviations in Sensation Seeking, model fit was only improved by the inclusion of fixed effects of average Sensation Seeking and weekly deviations in Sensation Seeking in the case of the MSI-BPD and the BSL-23, and was never further improved by the inclusion of random effects of deviations in sensation seeking, suggesting that the impact of changes in Sensation Seeking on BPD symptoms was similar (i.e., null) across individuals. In both of those models, there was a significant effect of average Sensation Seeking on BPD symptoms. There were no significant effects of weekly deviations in Sensation Seeking on BPD symptom expression.

Tests of Indirect Effects. Next, I once again used the RMediation program (Tofighi & MacKinnon, 2011), to create a 95% confidence intervals for the indirect effects of the interaction of trait BPD and deviations in estradiol on weekly BPD symptoms through all weekly self-control and impulsivity variables that: (1) were significantly predicted by the interaction between Trait BPD and deviations in estradiol (e.g., Trait BPD X Deviations in estradiol predict self-control), and (2) significantly predicted BPD symptoms (e.g., deviations in self-control predict weekly scores on the PAI-BOR).

Self-Control. Figures 21-24 depict mediation models with self-control as the mediator. The 95% confidence intervals did not include zero for the indirect effects of Trait BPD x Deviations in Estradiol via self-control on the PAI-BOR total scale (95% CI: -.01 to -.10), the Affective Instability subscale of the PAI-BOR (95% CI: -.02 to -.13), the Identity Disturbance subscale of the PAI-BOR (95% CI: -.01 to -.09), or the BSL-23 (95% CI: -.03 to -.14) via weekly self-control. Furthermore, when average and weekly deviations in self-control were included in the model predicting BPD from the interaction of Trait BPD with estradiol, the interactive effects of Trait BPD X Deviations in Estradiol were no longer significant (Predicting the PAI-BOR total score:

$\gamma_{\text{TRAITBPD*ESTRADIOLDEVIATION}} = -.04, SE = .05, t(117) = -.86, p = .39$; Predicting the PAI-

BOR Affective Instability subscale: $\gamma_{\text{TRAITBPD*ESTRADIOLDEVIATION}} = -.06, SE = .08, t(117) = -.79, p = .43$; Predicting the PAI-BOR Identity Disturbance subscale:

$\gamma_{\text{TRAITBPD*ESTRADIOLDEVIATION}} = -.07, SE = .05, t(117) = -1.33, p = .18$; Predicting the BSL-23: $\gamma_{\text{TRAITBPD*ESTRADIOLDEVIATION}} = -.05, SE = .07, t(117) = -.76, p = .45$). These results

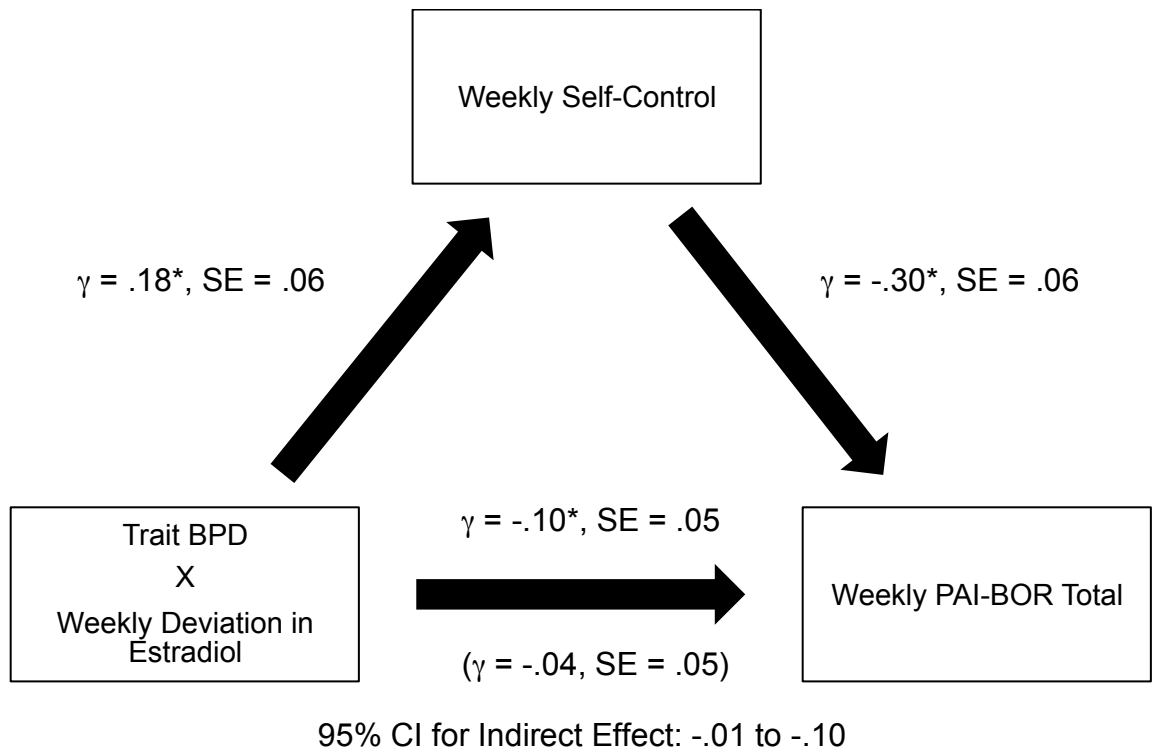


Figure 21. A depiction of the indirect effect of the interactive effect of trait BPD and weekly *deviation* in estradiol on weekly PAI-BOR total score via weekly self-control.

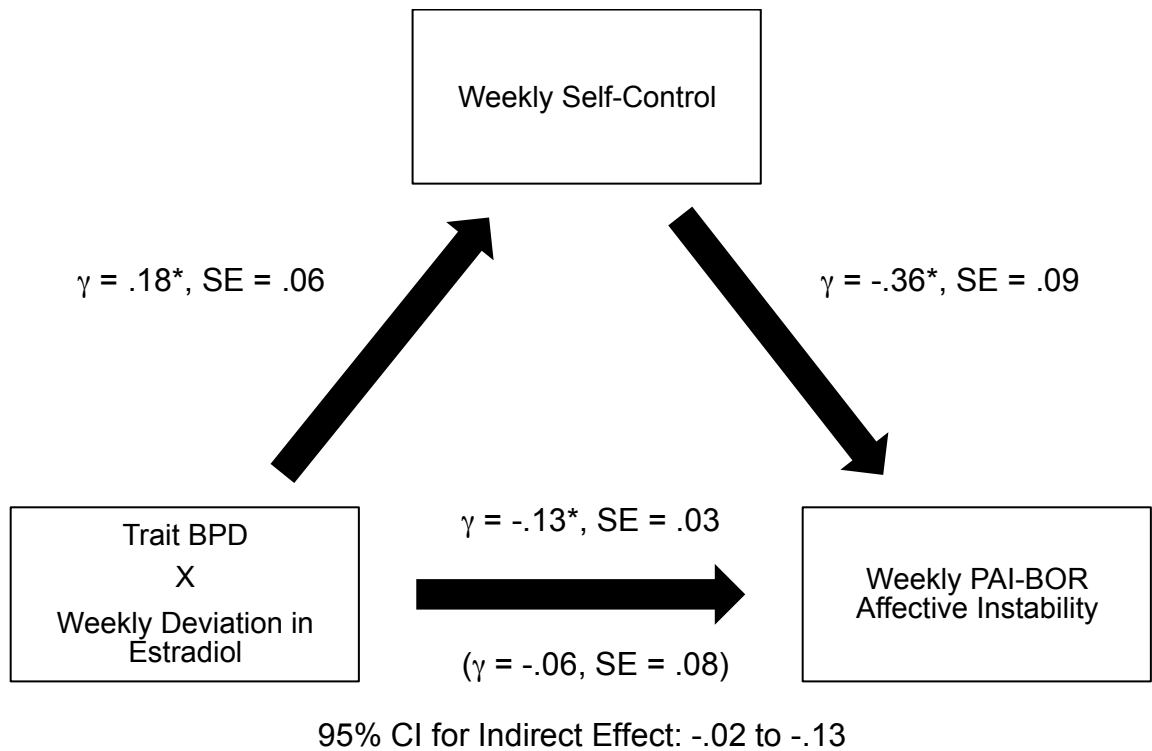


Figure 22. A depiction of the indirect effect of the interactive effect of trait BPD and weekly *deviation* in estradiol on weekly PAI-BOR Affective Instability score via weekly self-control.

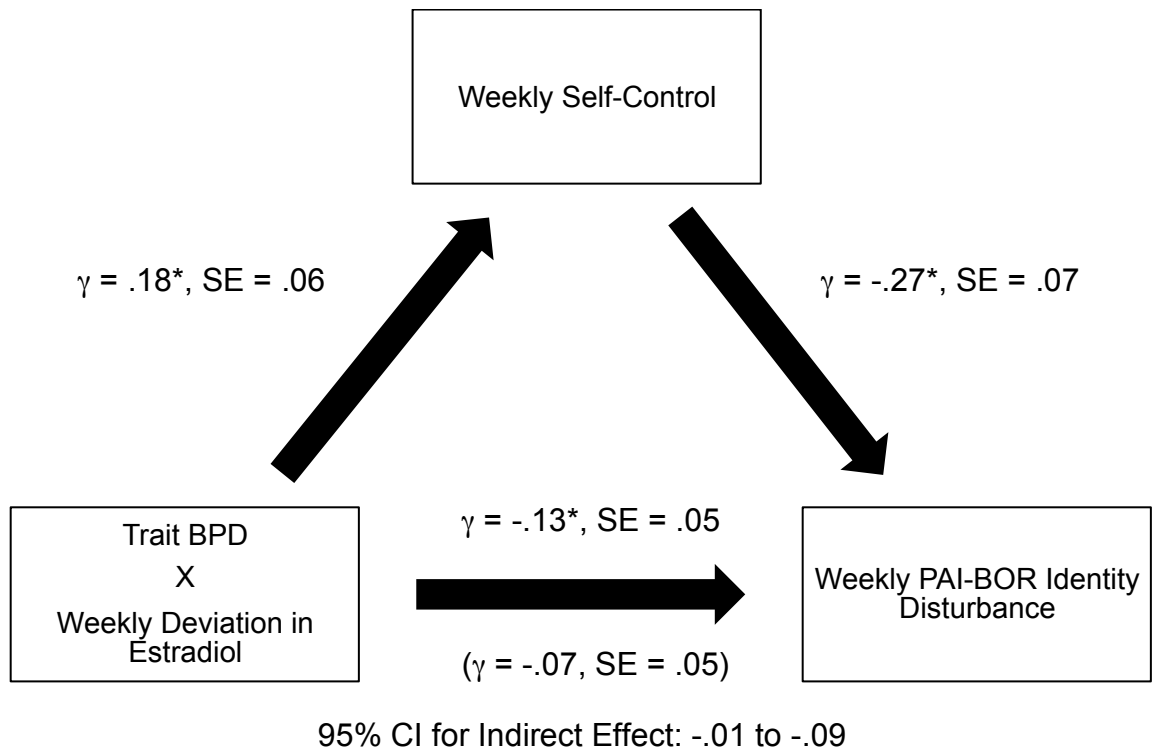


Figure 23. A depiction of the indirect effect of the interactive effect of trait BPD and weekly *deviation* in estradiol on weekly PAI-BOR Identity Disturbance score via weekly self-control.

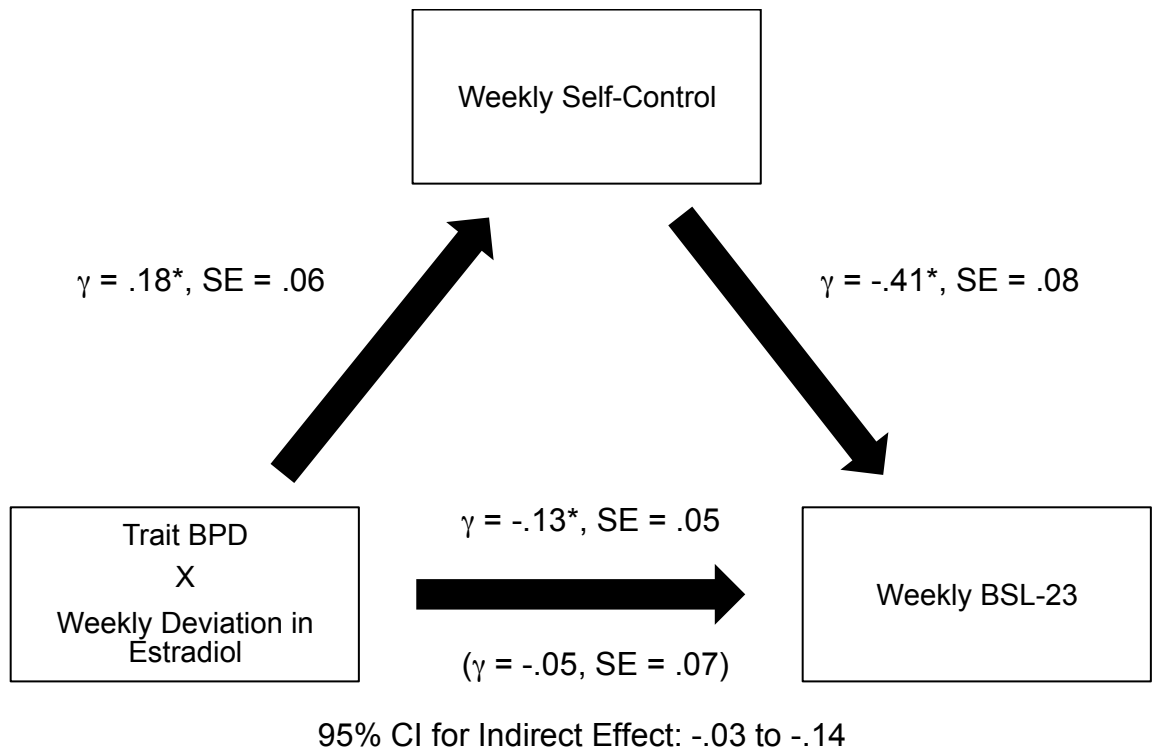
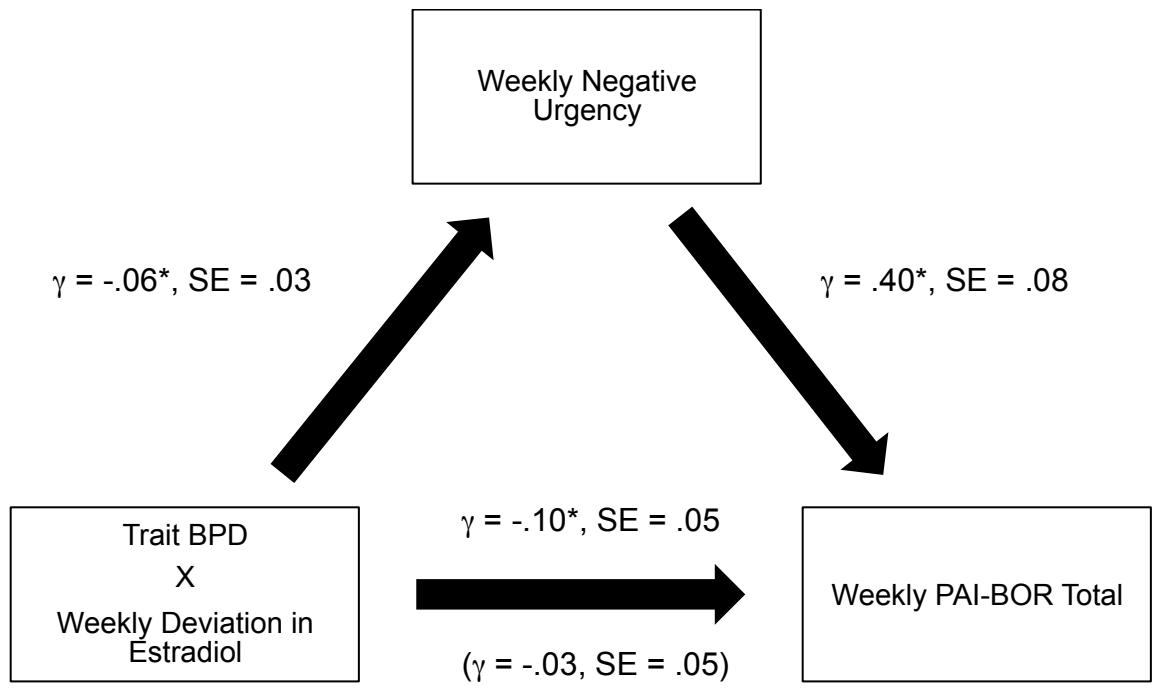


Figure 24. A depiction of the indirect effect of the interactive effect of trait BPD and weekly *deviation* in estradiol on weekly BSL-23 score via weekly self-control.

indicate that the interactive effect of trait BPD and deviations estradiol on BPD symptom expression is partially attributable to changes in self-control.

Negative Urgency. Figures 25-28 depict mediation models with Negative Urgency. The 95% confidence intervals did not include zero for the indirect effects of Trait BPD x Deviations in Estradiol via Negative Urgency on the PAI-BOR total scale (95% CI: -.01 to -.04), the Affective Instability subscale (95% CI: -.001 to -.06), the Identity Disturbance subscale (95% CI: -.01 to -.09), or the BSL-23 (95% CI: -.004 to -.06) via Negative Urgency. When average and weekly deviations in Negative Urgency were included in the model predicting BPD from the interaction of Trait BPD with estradiol, the interactive effects of Trait BPD X Deviations in Estradiol were no longer significant (Predicting PAI-BOR total: $\gamma_{\text{TRAITBPD*ESTRADIOLDEVIATION}} = -.03$, $SE = .05$, $t(117) = -.71$, $p = .47$; Predicting Affective Instability subscale: $\gamma_{\text{TRAITBPD*ESTRADIOLDEVIATION}} = -.03$, $SE = .07$, $t(117) = -.42$, $p = .67$; Predicting Identity Disturbance subscale: $\gamma_{\text{TRAITBPD*ESTRADIOLDEVIATION}} = -.06$, $SE = .05$, $t(117) = -1.13$, $p = .26$; Predicting BSL-23: $\gamma_{\text{TRAITBPD*ESTRADIOLDEVIATION}} = -.02$, $SE = .07$, $t(117) = -.42$, $p = .67$). Therefore, the interactive effect of trait BPD and deviations estradiol on BPD symptoms is partially attributable to changes in Negative Urgency.

Positive Urgency. Figure 29 depicts the significant mediation model with Positive Urgency. The 95% confidence interval did not include zero for the indirect effect of Trait BPD x Deviation in Estradiol via Positive Urgency on BSL-23 (-.01 to -.06). When average and weekly deviation in Positive Urgency were included in the model predicting



95% CI for Indirect Effect: -.01 to -.04

Figure 25. A depiction of the indirect effect of the interactive effect of trait BPD and weekly *deviation* in estradiol on weekly PAI-BOR total score via weekly UPPS-P Negative Urgency.

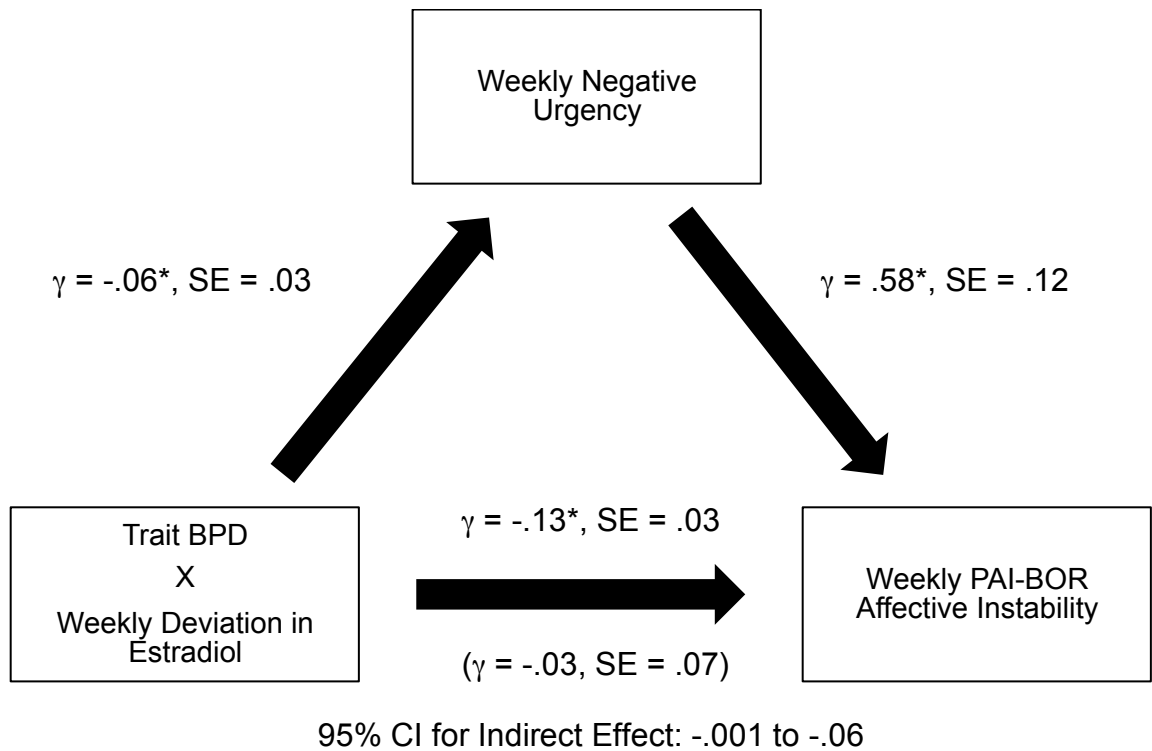


Figure 26. A depiction of the indirect effect of the interactive effect of trait BPD and weekly *deviation* in estradiol on weekly PAI-BOR Affective Instability score via weekly UPPS-P Negative Urgency.

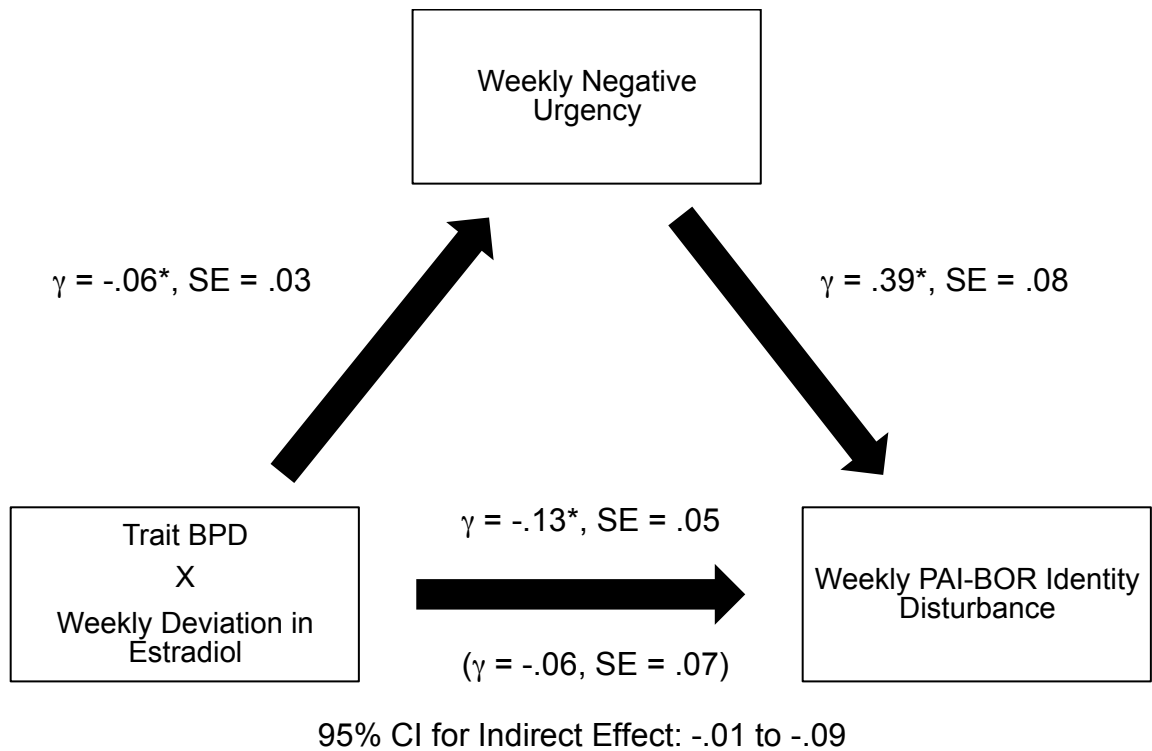


Figure 27. A depiction of the indirect effect of the interactive effect of trait BPD and weekly *deviation* in estradiol on weekly PAI-BOR Identity Disturbance score via weekly UPPS-P Negative Urgency.

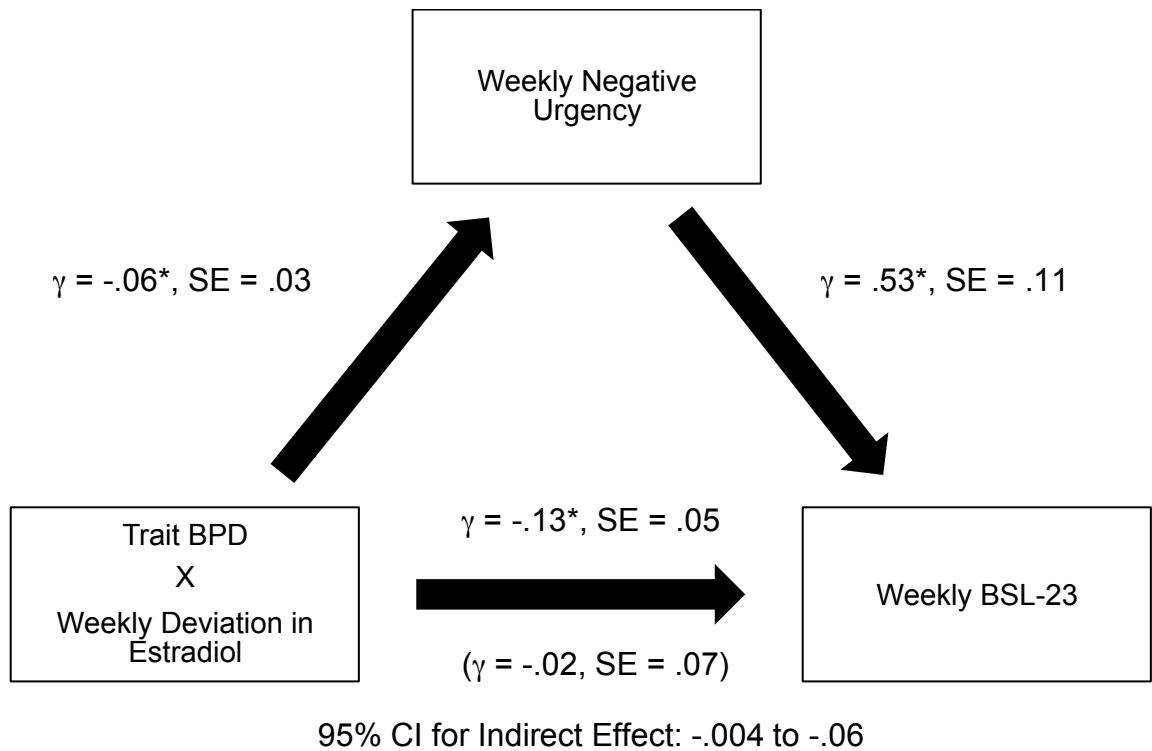


Figure 28. A depiction of the indirect effect of the interactive effect of trait BPD and weekly *deviation* in estradiol on weekly BSL-23 score via weekly UPPS-P Negative Urgency.

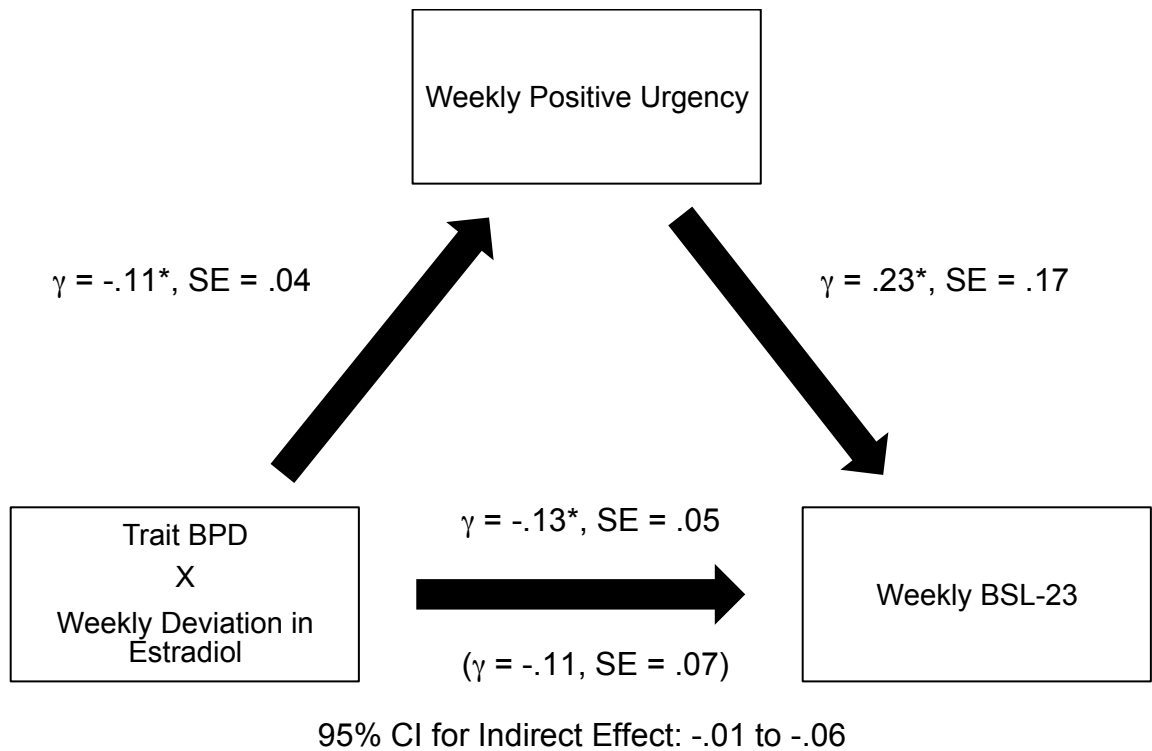


Figure 29. A depiction of the indirect effect of the interactive effect of trait BPD and weekly *deviation* in estradiol on weekly BSL-23 score via weekly UPPS-P Positive Urgency.

symptoms from the interaction of Trait BPD with estradiol, the interactive effects of Trait BPD X Deviations in Estradiol were no longer significant for the BSL-23 only (Predicting the BSL-23: $\gamma_{\text{TRAITBPD*ESTRADIOLDEVIATION}} = -.11$, $SE = .07$, $t(117) = -1.53$, $p = .12$). These results indicate that the interactive effect of trait BPD and deviations estradiol on expression of more extreme BPD symptoms are partially attributable to changes in Positive Urgency.

Lack of Perseverance. Figures 30-33 depict mediation models with Lack of Perseverance as the mediator. The 95% confidence intervals did not include zero for the indirect effects of Trait BPD x Deviations in Estradiol via Lack of Perseverance on the PAI-BOR total scale (95% CI: -.04 to -.10), the Affective Instability subscale of the PAI-BOR (95% CI: -.04 to -.16), the Identity Disturbance subscale of the PAI-BOR (95% CI: -.02 to -.11), or the BSL-23 (95% CI: -.07 to -.20) via Lack of Perseverance. Furthermore, when average and weekly deviations in Lack of Perseverance were included in the model predicting BPD from the interaction of Trait BPD with estradiol, the interactive effects of Trait BPD X Deviations in Estradiol were no longer significant (Predicting the PAI-BOR total score: $\gamma_{\text{TRAITBPD*ESTRADIOLDEVIATION}} = -.03$ $SE = .05$, $t(117) = -.74$, $p = .45$; Predicting the PAI-BOR Affective Instability subscale: $\gamma_{\text{TRAITBPD*ESTRADIOLDEVIATION}} = -.03$, $SE = .07$, $t(117) = -.50$, $p = .61$; Predicting the PAI-BOR Identity Disturbance subscale: $\gamma_{\text{TRAITBPD*ESTRADIOLDEVIATION}} = -.06$, $SE = .06$, $t(117) = -1.05$, $p = .29$; Predicting the BSL-23: $\gamma_{\text{TRAITBPD*ESTRADIOLDEVIATION}} = -.01$, $SE = .06$, $t(117) = -.23$, $p = .82$). These results indicate that the interactive effect of trait BPD and deviations estradiol on BPD symptom expression is partially attributable to changes in Lack of Perseverance.

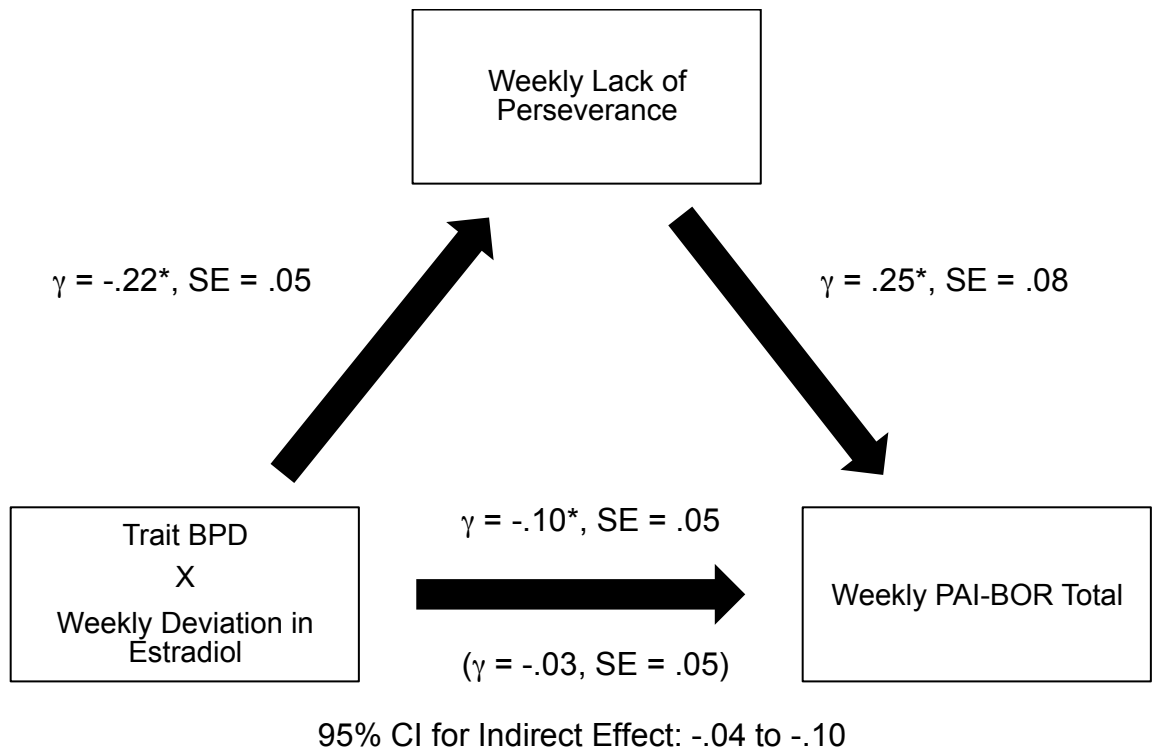


Figure 30. A depiction of the indirect effect of the interactive effect of trait BPD and weekly *deviation* in estradiol on weekly PAI-BOR total score via weekly UPPS-P Lack of Perseverance.

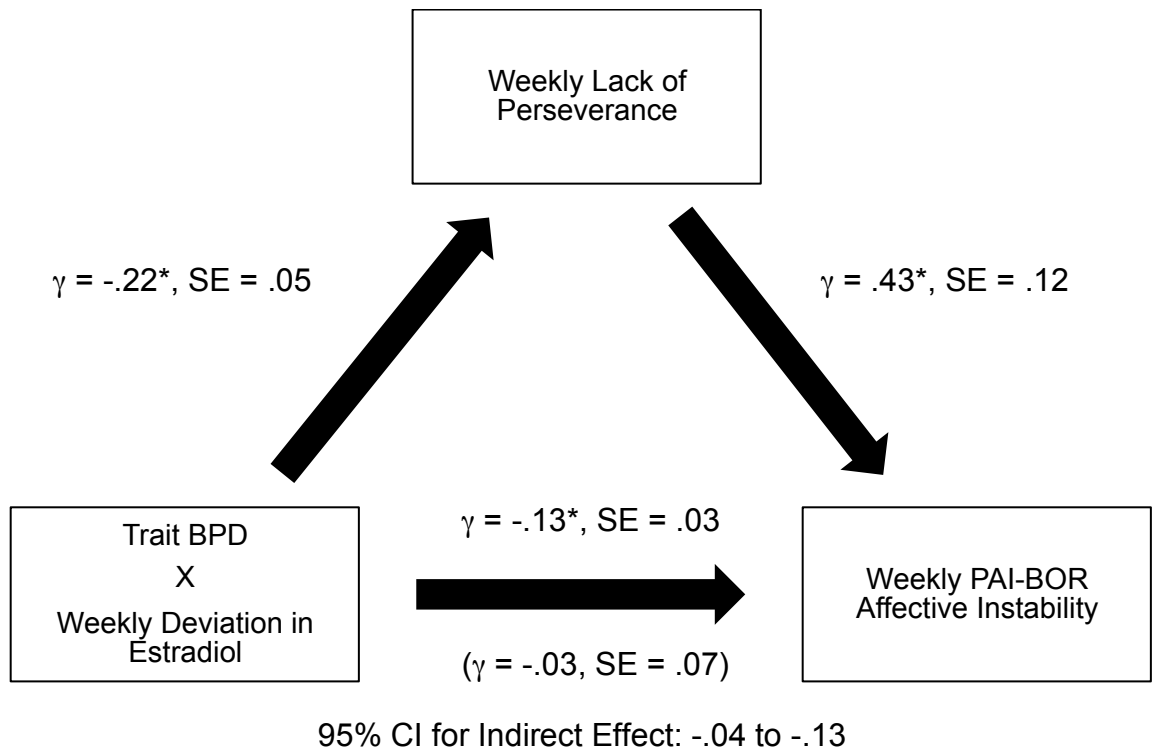


Figure 31. A depiction of the indirect effect of the interactive effect of trait BPD and weekly *deviation* in estradiol on weekly PAI-BOR Affective Instability score via weekly UPPS-P Lack of Perseverance.

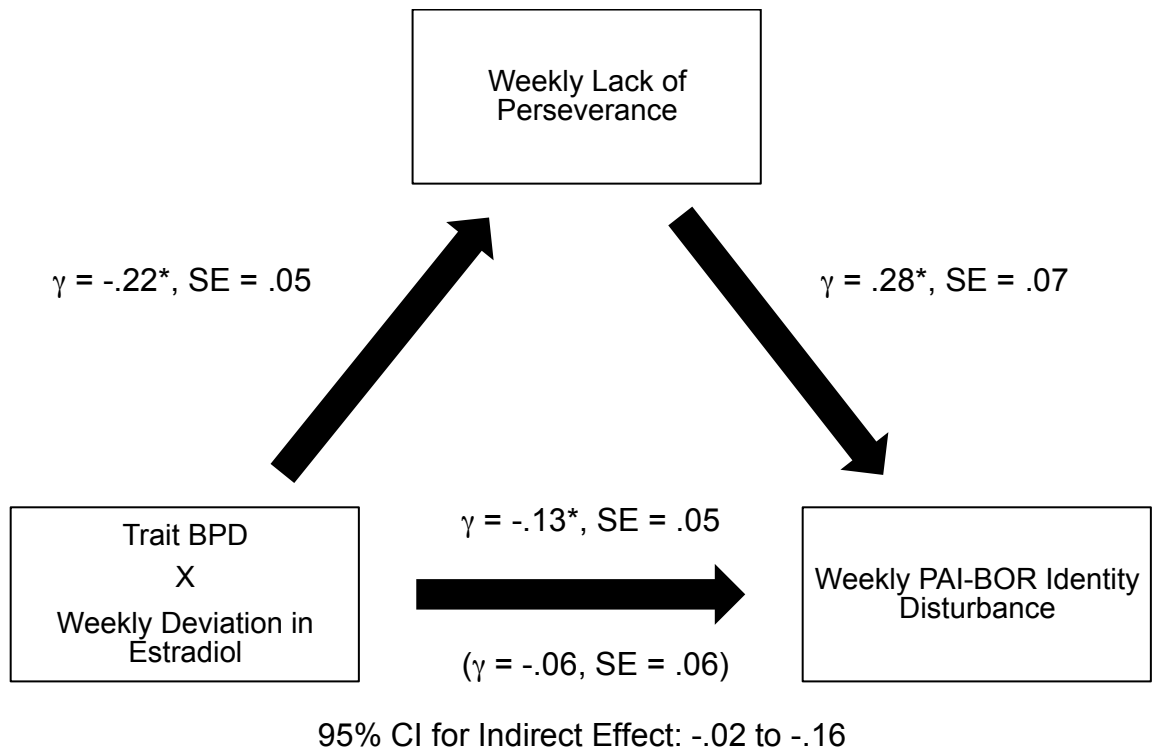


Figure 32. A depiction of the indirect effect of the interactive effect of trait BPD and weekly *deviation* in estradiol on weekly PAI-BOR Identity Disturbance score via weekly UPPS-P Lack of Perseverance.

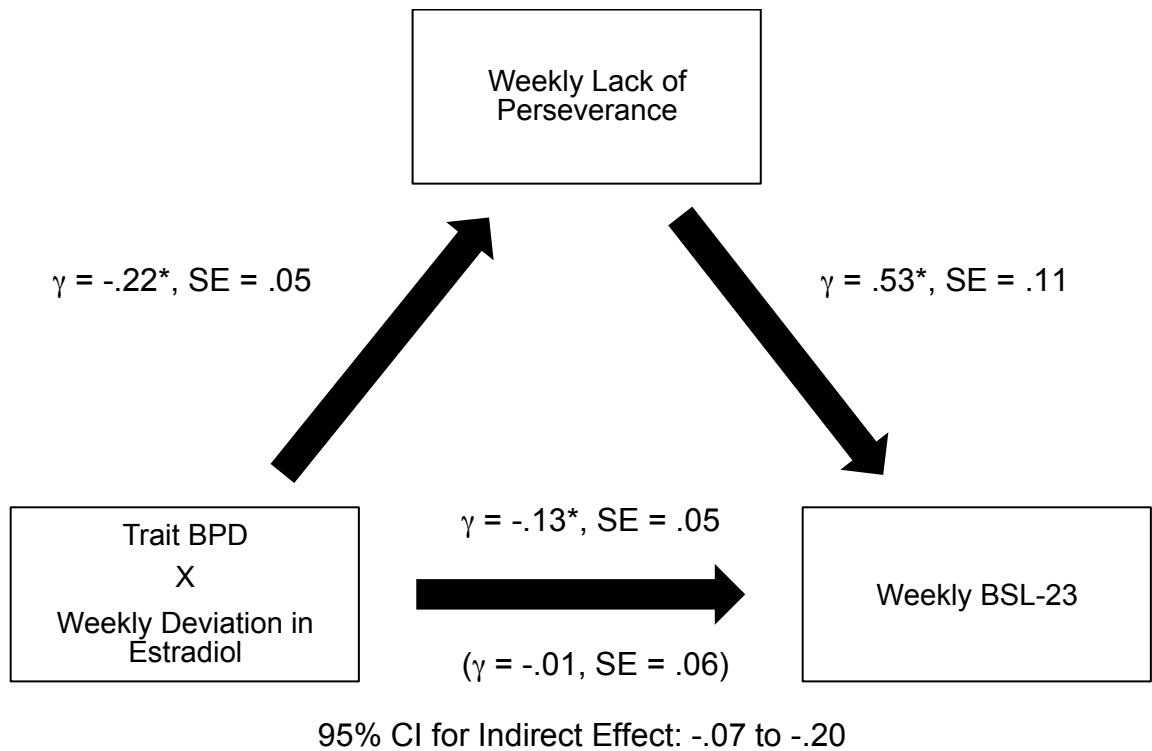


Figure 33. A depiction of the indirect effect of the interactive effect of trait BPD and weekly *deviation* in estradiol on weekly BSL-23 score via weekly UPPS-P Lack of Perseverance.

Lack of Premeditation. Figures 34-37 depict mediation models with Lack of Premeditation as the mediator. The 95% confidence intervals did not include zero for the indirect effects of Trait BPD x Deviations in Estradiol via Lack of Premeditation on the PAI-BOR total scale (95% CI: -.004 to -.06), the Affective Instability subscale of the PAI-BOR (95% CI: -.001 to -.06), the Identity Disturbance subscale of the PAI-BOR (95% CI: -.01 to -.07), or the BSL-23 (95% CI: -.01 to -.07) via Lack of Premeditation. Furthermore, when average and weekly deviations in Lack of Premeditation were included in the model predicting BPD from the interaction of Trait BPD with estradiol, the interactive effects of Trait BPD X Deviations in Estradiol were no longer significant (Predicting the PAI-BOR total score: $\gamma_{\text{TRAITBPD*ESTRADIOLDEVIATION}} = -.04, SE = .05, t(117) = -.91, p = .36$; Predicting the PAI-BOR Affective Instability subscale: $\gamma_{\text{TRAITBPD*ESTRADIOLDEVIATION}} = -.04, SE = .07, t(117) = -.52, p = .60$; Predicting the PAI-BOR Identity Disturbance subscale: $\gamma_{\text{TRAITBPD*ESTRADIOLDEVIATION}} = -.09, SE = .05, t(117) = -1.54, p = .12$; Predicting the BSL-23: $\gamma_{\text{TRAITBPD*ESTRADIOLDEVIATION}} = -.05, SE = .07, t(117) = -.76, p = .44$). These results indicate that the interactive effect of trait BPD and deviations estradiol on BPD symptom expression is partially attributable to changes in Lack of Premeditation.

Testing Alternative Moderators: Dismantling Trait BPD Moderation Effects Using FFM Personality and Childhood Maltreatment

A final set of analyses sought to further explore the finding that “trait BPD” (as measured using average scores on a heterogeneous measure of BPD symptoms) moderates the effect of within-person changes in estradiol on BPD symptom expression. I wanted to determine whether extreme levels of FFM personality domain scores (in

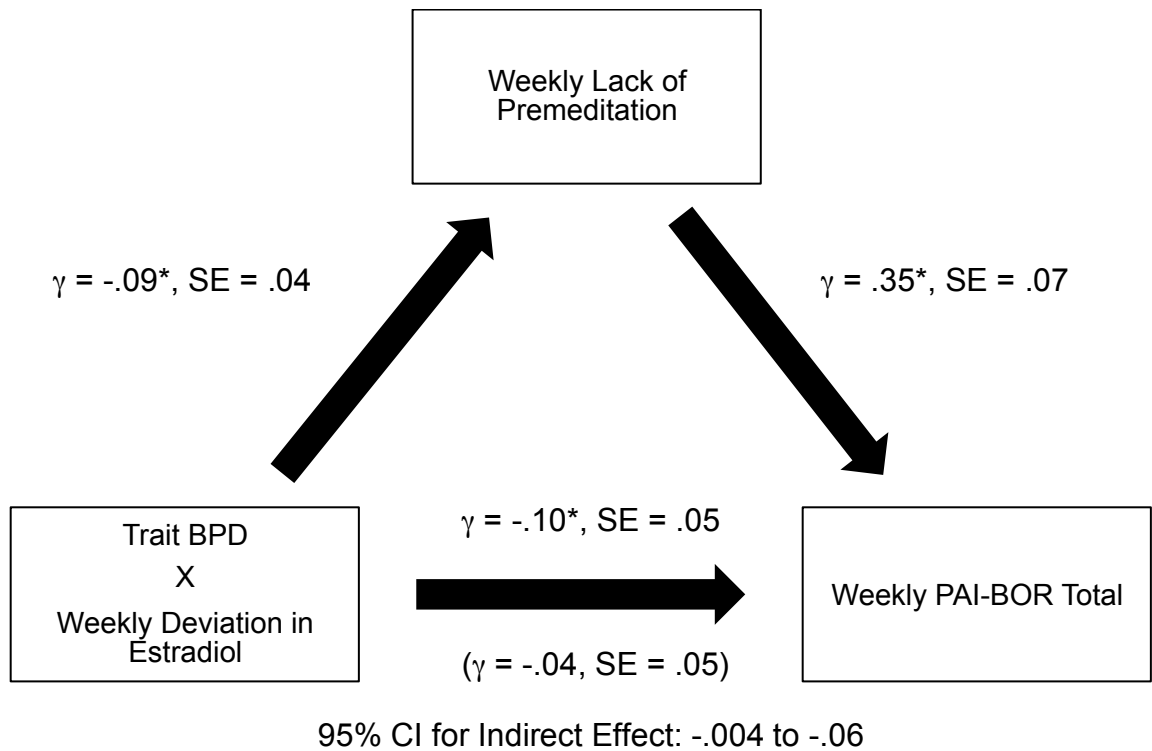


Figure 34. A depiction of the indirect effect of the interactive effect of trait BPD and weekly *deviation* in estradiol on weekly PAI-BOR total score via weekly UPPS-P Lack of Premeditation.

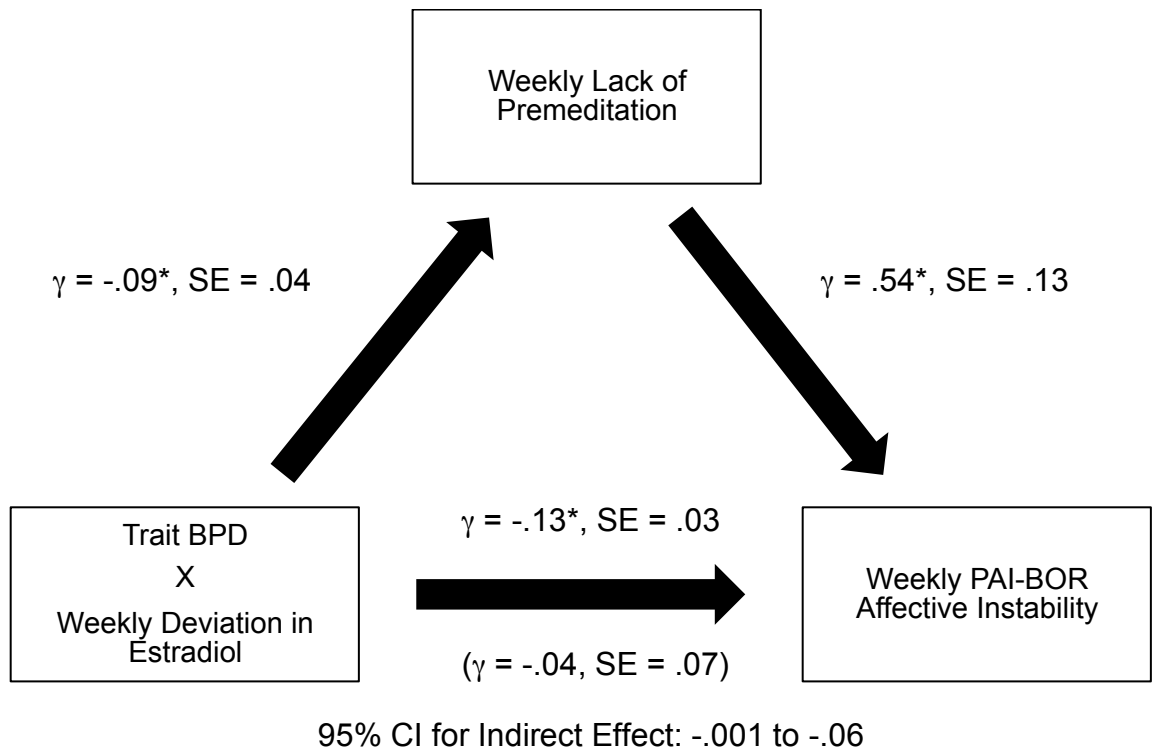


Figure 35. A depiction of the indirect effect of the interactive effect of trait BPD and weekly *deviation* in estradiol on weekly PAI-BOR Affective Instability score via weekly UPPS-P Lack of Premeditation.

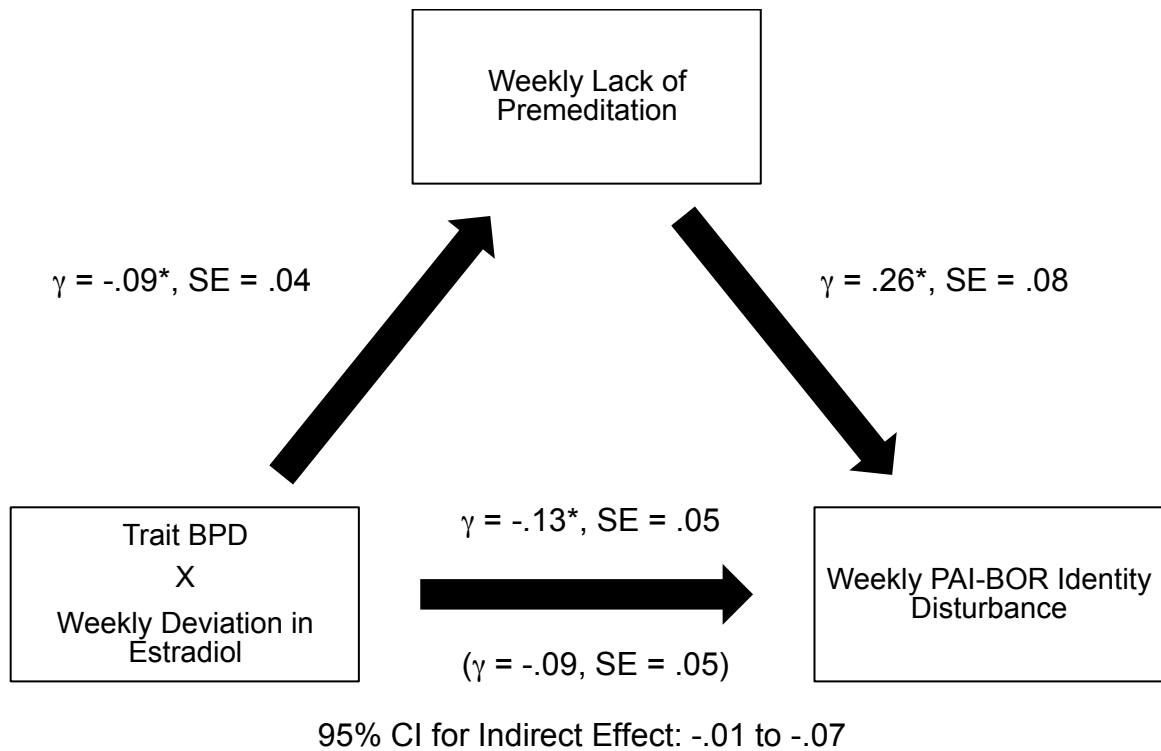


Figure 36. A depiction of the indirect effect of the interactive effect of trait BPD and weekly *deviation* in estradiol on weekly PAI-BOR Identity Disturbance score via weekly UPPS-P Lack of Premeditation.

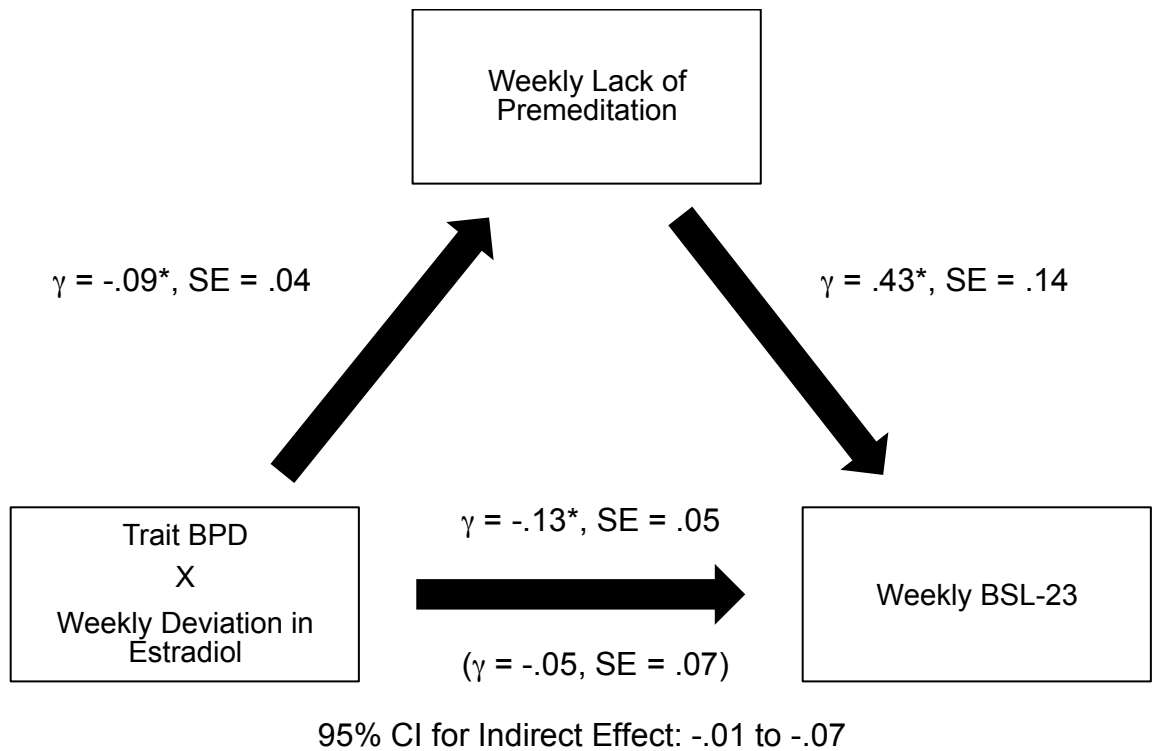


Figure 37. A depiction of the indirect effect of the interactive effect of trait BPD and weekly *deviation* in estradiol on weekly BSL-23 score via weekly UPPS-P Lack of Premeditation.

accordance with the FFM theory of BPD) or different types of childhood maltreatment (in accordance with the biosocial theory of BPD) would perform similarly to “trait BPD” as moderators of the effects of weekly deviations in estradiol on BPD symptom expression.

FFM Personality Domains as Alternative Moderators. First, I substituted each of the domain scores (Neuroticism, Extraversion, Openness, Agreeableness, and Conscientiousness) for Trait BPD (for original model, see Table 7). Results of these models are presented in Tables 24-28. The null models have been transposed from Table 3 for ease of model comparison. Consistent with a FFM personality model of BPD in which extremely high Neuroticism is a key underlying aspect of BPD, Neuroticism exerted main effects and interacted significantly with estradiol variables to predict BPD symptom expression. In each case except the Self-Harm subscale of the PAI-BOR, higher levels of Neuroticism were associated with greater BPD symptoms. Additionally, there were significant interactions between Neuroticism and *average estradiol* in three models. Among women *lower* in Neuroticism, higher average levels of estradiol were associated with lower levels of Affective Instability ($\gamma_{\text{LOWNEUROTICISM*AVGESTRADIOL}} = -.31, SE = .15, t(37) = -1.99, p = .04$), Identity Disturbance ($\gamma_{\text{LOWNEUROTICISM*AVGESTRADIOL}} = -.16, SE = .07, t(37) = -2.28, p = .02$), and the BSL-23 ($\gamma_{\text{LOWNEUROTICISM*AVGESTRADIOL}} = -.27, SE = .10, t(37) = -2.70, p = .01$). Among women *higher* in Neuroticism, higher average levels of estradiol were not significantly associated with Affective Instability ($\gamma_{\text{HIGHNEUROTICISM*AVGESTRADIOL}} = .08, SE = .19, t(37) = .46, p = .64$) or Identity Disturbance ($\gamma_{\text{HIGHNEUROTICISM*AVGESTRADIOL}} = .12, SE = .14, t(37) = .85, p = .40$), but were associated with higher scores on the BSL-23 ($\gamma_{\text{HIGHNEUROTICISM*AVGESTRADIOL}} = .29,$

Table 24

Multilevel Poisson Regression Models Predicting Weekly BPD Symptoms from the Interaction of Neuroticism with Average Estradiol and Weekly Deviations in Estradiol

Parameter	Model 1 (null)	Model 4 (with moderator)
<u>Dependent Variable: PAI-BOR Total Score</u>		
Fixed Effects		
Intercept	-.50* (.10)	-.51* (.09)
Avg Estradiol		-.02* (.09)
Weekly Deviation in Estradiol		.01 (.05)
Neuroticism		.31* (.09)
Neuroticism*Avg Estradiol		.06 (.08)
<i>Neuroticism*Estradiol Deviation</i>		-.03 (.04)
Random Parameters		
Intercept	.39* (.09)	.29* (.08)
Weekly Deviation in Estradiol		
Residual	.08* (.01)	.08* (.01)
-2 Restricted Log Pseudo-likelihood	240.55	239.20
<u>Dependent Variable: PAI-BOR Affective Instability Subscale</u>		
Fixed Effects		
Intercept	-.60* (.15)	-.64* (.12)
Avg Estradiol		-.11 (.13)
Weekly Deviation in Estradiol		-.02 (.07)
Neuroticism		.50* (.13)
Neuroticism*Avg Estradiol		.20* (.08)
<i>Neuroticism*Estradiol Deviation</i>		-.05 (.07)
Random Parameters		
Intercept	.83* (.23)	.83* (.22)
Weekly Deviation in Estradiol		
Residual	.16* (.02)	.16* (.02)
-2 Restricted Log Pseudo-likelihood	370.17	362.00†
<u>Dependent Variable: PAI-BOR Identity Disturbance Subscale</u>		
Fixed Effects		
Intercept	-.30* (.11)	-.32* (.09)
Avg Estradiol		-.02 (.10)
Weekly Deviation in Estradiol		.04 (.05)
Neuroticism		.32* (.10)
Neuroticism*Avg Estradiol		.14* (.04)
<i>Neuroticism*Estradiol Deviation</i>		-.10 (.09)
Random Parameters		
Intercept	.45* (.11)	.32* (.08)
Weekly Deviation in Estradiol		
Residual	.12* (.01)	.12* (.01)
-2 Restricted Log Pseudo-likelihood	274.93	264.30†

Table Continued on Next Page

<u>Dependent Variable: PAI-BOR Negative Relationships Subscale</u>		
	Fixed Effects	
Intercept	-.41* (.12)	-.41* (.11)
Avg Estradiol		-.04 (.11)
Weekly Deviation in Estradiol		.006 (.06)
Neuroticism		.38* (.11)
Neuroticism*Avg Estradiol		-.04 (.09)
<i>Neuroticism*Estradiol Deviation</i>		.08 (.06)
	Random Parameters	
Intercept	.53* (.14)	.42* (.12)
Weekly Deviation in Estradiol		
Residual	.16* (.02)	.16* (.02)
-2 Restricted Log Pseudo-likelihood	332.16	324.17†
<u>Dependent Variable: PAI-BOR Self-Harm Subscale</u>		
	Fixed Effects	
Intercept	-1.03* (.11)	-1.04* (.11)
Avg Estradiol		.01 (.12)
Weekly Deviation in Estradiol		.03 (.09)
Neuroticism		.05 (.11)
Neuroticism*Avg Estradiol		.008 (.10)
<i>Neuroticism*Estradiol Deviation</i>		-.05 (.09)
	Random Parameters	
Intercept	.35* (.12)	.39* (.14)
Weekly Deviation in Estradiol		
Residual	.18* (.02)	.18* (.03)
-2 Restricted Log Pseudo-likelihood	407.23	406.69
<u>Dependent Variable: Borderline Symptom Checklist—23 (BSL-23)</u>		
	Fixed Effects	
Intercept	2.05* (.16)	2.00* (.13)
Avg Estradiol		.001 (.14)
Weekly Deviation in Estradiol		-.12 (.07)
Neuroticism		.60* (.14)
Neuroticism*Avg Estradiol		.27* (.12)
<i>Neuroticism*Estradiol Deviation</i>		.006 (.92)
	Random Parameters	
Intercept	1.06* (.29)	.65* (.19)
Weekly Deviation in Estradiol		
Residual	2.34* (.31)	2.24* (.30)
-2 Restricted Log Pseudo-likelihood	379.72	369.59†

Table Continued on Next Page

Dependent Variable: McLean Screening Instrument for BPD (MSI-BPD)

	Fixed Effects	
Intercept	.17 (.22)	.15 (.21)
Avg Estradiol		-.07 (.22)
Weekly Deviation in Estradiol		-.11 (.09)
Neuroticism		.61* (.22)
Neuroticism*Avg Estradiol		.05 (.18)
<i>Neuroticism*Estradiol Deviation</i>		.06 (.08)
	Random Parameters	
Intercept	1.80* (.54)	1.58* (.50)
Weekly Deviation in Estradiol		
Residual	.68* (.09)	.69* (.09)
-2 Restricted Log Pseudo-likelihood	481.04	480.08

Note. Standard errors are in parentheses.

* $p < .05$.

†Change in -2 Restricted Log Pseudo-likelihood over the previous model is significant at $p < .05$.

Table 25

Multilevel Poisson Regression Models Predicting Weekly BPD Symptoms from the Interaction of Extraversion with Average Estradiol and Weekly Deviations in Estradiol

Parameter	Model 1 (null)	Model 4 (with moderator)
<u>Dependent Variable: PAI-BOR Total Score</u>		
Fixed Effects		
Intercept	-.50* (.10)	-.53* (.11)
Avg Estradiol		-.07 (.11)
Weekly Deviation in Estradiol		.02 (.05)
Extraversion		-.06 (.11)
Extraversion*Avg Estradiol		-.01 (.10)
<i>Extraversion*Estradiol Deviation</i>		.05 (.04)
Random Parameters		
Intercept	.39* (.09)	.40* (.10)
Weekly Deviation in Estradiol		
Residual	.08* (.01)	.08 (.01)
-2 Restricted Log Pseudo-likelihood	240.55	238.77
<u>Dependent Variable: PAI-BOR Affective Instability Subscale</u>		
Fixed Effects		
Intercept	-.60* (.15)	-.70* (.16)
Avg Estradiol		-.17 (.17)
Weekly Deviation in Estradiol		-.01 (.07)
Extraversion		-.19 (.16)
Extraversion*Avg Estradiol		-.15 (.16)
<i>Extraversion*Estradiol Deviation</i>		.07 (.06)
Random Parameters		
Intercept	.83* (.23)	.84* (.24)
Weekly Deviation in Estradiol		
Residual	.16* (.02)	.16* (.02)
-2 Restricted Log Pseudo-likelihood	370.17	368.72
<u>Dependent Variable: PAI-BOR Identity Disturbance Subscale</u>		
Fixed Effects		
Intercept	-.30* (.11)	-.34* (.11)
Avg Estradiol		-.07 (.12)
Weekly Deviation in Estradiol		.01 (.05)
Extraversion		.003 (.12)
Extraversion*Avg Estradiol		-.01 (.11)
<i>Extraversion*Estradiol Deviation</i>		.04 (.05)
Random Parameters		
Intercept	.45* (.11)	.46* (.12)
Weekly Deviation in Estradiol		
Residual	.12* (.01)	.12* (.01)
-2 Restricted Log Pseudo-likelihood	274.93	273.75

Table Continued on Next Page

<u>Dependent Variable: PAI-BOR Negative Relationships Subscale</u>		
		Fixed Effects
Intercept	-.41* (.12)	-.45* (.13)
Avg Estradiol		-.07 (.13)
Weekly Deviation in Estradiol		.05 (.06)
Extraversion		-.06 (.13)
Extraversion*Avg Estradiol		-.01 (.13)
<i>Extraversion*Estradiol Deviation</i>		.04 (.06)
		Random Parameters
Intercept	.53* (.14)	.55* (.15)
Weekly Deviation in Estradiol		
Residual	.16* (.02)	.16* (.02)
-2 Restricted Log Pseudo-likelihood	332.16	331.99
<u>Dependent Variable: PAI-BOR Self-Harm Subscale</u>		
		Fixed Effects
Intercept	-1.03* (.11)	-1.00* (.11)
Avg Estradiol		-.04 (.12)
Weekly Deviation in Estradiol		.05 (.10)
Extraversion		-.11 (.11)
Extraversion*Avg Estradiol		.10 (.11)
<i>Extraversion*Estradiol Deviation</i>		.07 (.08)
		Random Parameters
Intercept	.35* (.12)	.37* (.13)
Weekly Deviation in Estradiol		
Residual	.18* (.02)	.18* (.02)
-2 Restricted Log Pseudo-likelihood	407.23	331.11
<u>Dependent Variable: Borderline Symptom Checklist—23 (BSL-23)</u>		
		Fixed Effects
Intercept	2.05* (.16)	1.96* (.18)
Avg Estradiol		-.04 (.19)
Weekly Deviation in Estradiol		-.10 (.06)
Extraversion		-.10 (.19)
Extraversion*Avg Estradiol		-.10 (.18)
<i>Extraversion*Estradiol Deviation</i>		.05 (.06)
		Random Parameters
Intercept	1.06* (.29)	1.15* (.32)
Weekly Deviation in Estradiol		
Residual	2.34* (.31)	2.26* (.30)
-2 Restricted Log Pseudo-likelihood	379.72	377.15

Table Continued on Next Page

Dependent Variable: McLean Screening Instrument for BPD (MSI-BPD)		
	Fixed Effects	
Intercept	.17 (.22)	.11 (.25)
Avg Estradiol		-.08 (.27)
Weekly Deviation in Estradiol		-.11 (.08)
Extraversion		.03 (.25)
Extraversion*Avg Estradiol		-.01 (.24)
<i>Extraversion*Estradiol Deviation</i>		-.07 (.07)
	Random Parameters	
Intercept	1.80* (.54)	2.02* (.63)
Weekly Deviation in Estradiol		
Residual	.68* (.09)	.69* (.09)
-2 Restricted Log Pseudo-likelihood	481.04	480.26

Note. Standard errors are in parentheses.

* $p < .05$.

†Change in -2 Restricted Log Pseudo-likelihood over the previous model is significant at $p < .05$.

Table 26

Multilevel Poisson Regression Models Predicting Weekly BPD Symptoms from the Interaction of Openness to Experience with Average Estradiol and Weekly Deviations in Estradiol

Parameter	Model 1 (null)	Model 4 (with moderator)
<u>Dependent Variable: PAI-BOR Total Score</u>		
Fixed Effects		
Intercept	-.50* (.10)	-.52* (.09)
Avg Estradiol		-.01 (.09)
Weekly Deviation in Estradiol		-.001 (.04)
Openness		.21* (.09)
Openness*Avg Estradiol		-.23* (.10)
<i>Openness*Estradiol Deviation</i>		.04 (.04)
Random Parameters		
Intercept	.39* (.09)	.32* (.08)
Weekly Deviation in Estradiol		
Residual	.08* (.01)	.08* (.01)
-2 Restricted Log Pseudo-likelihood	240.55	236.98†
<u>Dependent Variable: PAI-BOR Affective Instability Subscale</u>		
Fixed Effects		
Intercept	-.60* (.15)	-.64* (.14)
Avg Estradiol		-.07 (.14)
Weekly Deviation in Estradiol		-.05 (.06)
Openness		.28 (.16)
Openness*Avg Estradiol		-.42* (.16)
<i>Openness*Estradiol Deviation</i>		.03 (.06)
Random Parameters		
Intercept	.83* (.23)	.70* (.20)
Weekly Deviation in Estradiol		
Residual	.16* (.02)	.16* (.02)
-2 Restricted Log Pseudo-likelihood	370.17	362.07†
<u>Dependent Variable: PAI-BOR Identity Disturbance Subscale</u>		
Fixed Effects		
Intercept	-.30* (.11)	-.33* (.10)
Avg Estradiol		-.03 (.11)
Weekly Deviation in Estradiol		-.01 (.05)
Openness		.17 (.11)
Openness*Avg Estradiol		-.21* (.11)
<i>Openness*Estradiol Deviation</i>		.02 (.04)
Random Parameters		
Intercept	.45* (.11)	.40* (.10)
Weekly Deviation in Estradiol		
Residual	.12* (.01)	.12* (.01)
-2 Restricted Log Pseudo-likelihood	274.93	272.63†

Table Continued on Next Page

Dependent Variable: PAI-BOR Negative Relationships Subscale

	Fixed Effects	
Intercept	-.41* (.12)	-.44* (.11)
Avg Estradiol		-.01 (.12)
Weekly Deviation in Estradiol		.03 (.06)
Openness		.17 (.12)
Openness*Avg Estradiol		-.20 (.13)
<i>Openness*Estradiol Deviation</i>		.03 (.04)
Random Parameters		
Intercept	.53* (.14)	.49* (.14)
Weekly Deviation in Estradiol		
Residual	.16* (.02)	.17* (.02)
-2 Restricted Log Pseudo-likelihood	332.16	330.48

Dependent Variable: PAI-BOR Self-Harm Subscale

	Fixed Effects	
Intercept	-1.03* (.11)	-1.04* (.11)
Avg Estradiol		.06 (.12)
Weekly Deviation in Estradiol		.01 (.09)
Openness		.18 (.12)
Openness*Avg Estradiol		-.19 (.12)
<i>Openness*Estradiol Deviation</i>		.13 (.08)
Random Parameters		
Intercept	.35* (.12)	.38* (.13)
Weekly Deviation in Estradiol		
Residual	.18* (.02)	.18* (.02)
-2 Restricted Log Pseudo-likelihood	407.23	405.97

Dependent Variable: Borderline Symptom Checklist—23 (BSL-23)

	Fixed Effects	
Intercept	2.05* (.16)	2.00* (.16)
Avg Estradiol		.06 (.16)
Weekly Deviation in Estradiol		-.12 (.06)
Openness		.29 (.16)
Openness*Avg Estradiol		-.53* (.18)
<i>Openness*Estradiol Deviation</i>		.03 (.05)
Random Parameters		
Intercept	1.06* (.29)	.90* (.25)
Weekly Deviation in Estradiol		
Residual	2.34* (.31)	2.27* (.30)
-2 Restricted Log Pseudo-likelihood	379.72	375.44†

Table Continued on Next Page

Dependent Variable: McLean Screening Instrument for BPD (MSI-BPD)

	Fixed Effects	
Intercept	.17 (.22)	.12 (.21)
Avg Estradiol		.03 (.22)
Weekly Deviation in Estradiol		-.09 (.07)
Openness		.60* (.22)
Openness*Avg Estradiol		-.53* (.23)
<i>Openness*Estradiol Deviation</i>		.06 (.06)
	Random Parameters	
Intercept	1.80* (.54)	1.49* (.47)
Weekly Deviation in Estradiol		
Residual	.68* (.09)	.70* (.09)
-2 Restricted Log Pseudo-likelihood	481.04	473.55†

Note. Standard errors are in parentheses.

* $p < .05$.

†Change in -2 Restricted Log Pseudo-likelihood over the previous model is significant at $p < .05$.

Table 27

Multilevel Poisson Regression Models Predicting Weekly BPD Symptoms from the Interaction of Agreeableness with Average Estradiol and Weekly Deviations in Estradiol

Parameter	Model 1 (null)	Model 4 (with moderator)
<u>Dependent Variable: PAI-BOR Total Score</u>		
Fixed Effects		
Intercept	-.50* (.10)	-.54* (.09)
Avg Estradiol		-.05 (.09)
Weekly Deviation in Estradiol		.02 (.05)
Agreeableness		-.16 (.10)
Agreeableness*Avg Estradiol		-.15 (.10)
<i>Agreeableness*Estradiol Deviation</i>		.04 (.04)
Random Parameters		
Intercept	.39* (.09)	.33* (.08)
Weekly Deviation in Estradiol		
Residual	.08* (.01)	.08* (.01)
-2 Restricted Log Pseudo-likelihood	240.55	238.22
<u>Dependent Variable: PAI-BOR Affective Instability Subscale</u>		
Fixed Effects		
Intercept	-.60* (.15)	-.66* (.14)
Avg Estradiol		-.16 (.14)
Weekly Deviation in Estradiol		-.02 (.07)
Agreeableness		-.27 (.14)
Agreeableness*Avg Estradiol		-.22 (.15)
<i>Agreeableness*Estradiol Deviation</i>		.06 (.06)
Random Parameters		
Intercept	.83* (.23)	.72* (.21)
Weekly Deviation in Estradiol		
Residual	.16* (.02)	.16* (.02)
-2 Restricted Log Pseudo-likelihood	370.17	368.41
<u>Dependent Variable: PAI-BOR Identity Disturbance Subscale</u>		
Fixed Effects		
Intercept	-.30* (.11)	-.34* (.10)
Avg Estradiol		-.08 (.10)
Weekly Deviation in Estradiol		.01 (.05)
Agreeableness		-.16 (.11)
Agreeableness*Avg Estradiol		-.12 (.11)
<i>Agreeableness*Estradiol Deviation</i>		.02 (.04)
Random Parameters		
Intercept	.45* (.11)	.41* (.10)
Weekly Deviation in Estradiol		
Residual	.12* (.01)	.12* (.01)
-2 Restricted Log Pseudo-likelihood	274.93	273.36

Table Continued on Next Page

<u>Dependent Variable: PAI-BOR Negative Relationships Subscale</u>		
	Fixed Effects	
Intercept	-0.41* (.12)	-0.45* (.10)
Avg Estradiol		-0.06 (.10)
Weekly Deviation in Estradiol		.03 (.07)
Agreeableness		-0.30* (.11)
Agreeableness*Avg Estradiol		-0.16 (.11)
<i>Agreeableness*Estradiol Deviation</i>		<i>-0.001 (.05)</i>
	Random Parameters	
Intercept	.53* (.14)	.39* (.11)
Weekly Deviation in Estradiol		
Residual	.16* (.02)	.17* (.02)
-2 Restricted Log Pseudo-likelihood	332.16	332.05
<u>Dependent Variable: PAI-BOR Self-Harm Subscale</u>		
	Fixed Effects	
Intercept	-1.03* (.11)	-1.05* (.11)
Avg Estradiol		.04 (.12)
Weekly Deviation in Estradiol		.05 (.09)
Agreeableness		.12 (.12)
Agreeableness*Avg Estradiol		-0.13 (.12)
<i>Agreeableness*Estradiol Deviation</i>		<i>.13 (.08)</i>
	Random Parameters	
Intercept	.35* (.12)	.41* (.14)
Weekly Deviation in Estradiol		
Residual	.18* (.02)	.18* (.02)
-2 Restricted Log Pseudo-likelihood	407.23	406.07
<u>Dependent Variable: Borderline Symptom Checklist—23 (BSL-23)</u>		
	Fixed Effects	
Intercept	2.05* (.16)	1.98* (.16)
Avg Estradiol		-0.04 (.16)
Weekly Deviation in Estradiol		-0.12 (.06)
Agreeableness		-0.27 (.16)
Agreeableness*Avg Estradiol		-0.35 (.18)
<i>Agreeableness*Estradiol Deviation</i>		<i>-0.01 (.05)</i>
	Random Parameters	
Intercept	1.06* (.29)	.92* (.26)
Weekly Deviation in Estradiol		
Residual	2.34* (.31)	2.26* (.30)
-2 Restricted Log Pseudo-likelihood	379.72	378.34

Table Continued on Next Page

Dependent Variable: McLean Screening Instrument for BPD (MSI-BPD)		
	Fixed Effects	
Intercept	.17 (.22)	.11 (.22)
Avg Estradiol		-.10 (.22)
Weekly Deviation in Estradiol		-.09 (.08)
Agreeableness		-.29 (.22)
Agreeableness*Avg Estradiol		-.33 (.23)
<i>Agreeableness*Estradiol Deviation</i>		-.01 (.07)
	Random Parameters	
Intercept	1.80* (.54)	1.70* (.54)
Weekly Deviation in Estradiol		
Residual	.68* (.09)	.70* (.09)
-2 Restricted Log Pseudo-likelihood	481.04	480.81

Note. Standard errors are in parentheses.

* $p < .05$.

†Change in -2 Restricted Log Pseudo-likelihood over the previous model is significant at $p < .05$.

Table 28

Multilevel Poisson Regression Models Predicting Weekly BPD Symptoms from the Interaction of Conscientiousness with Average Estradiol and Weekly Deviations in Estradiol

Parameter	Model 1 (null)	Model 4 (with moderator)
<u>Dependent Variable: PAI-BOR Total Score</u>		
Fixed Effects		
Intercept	-.50* (.10)	-.53* (.10)
Avg Estradiol		-.05 (.10)
Weekly Deviation in Estradiol		.01 (.10)
Conscientiousness		-.13 (.10)
Conscientiousness*Avg Estradiol		.02 (.10)
<i>Conscientiousness*Estradiol Deviation</i>		-.02 (.04)
Random Parameters		
Intercept	.39* (.09)	.38* (.10)
Weekly Deviation in Estradiol		
Residual	.08* (.01)	.08* (.01)
-2 Restricted Log Pseudo-likelihood	240.55	238.71
<u>Dependent Variable: PAI-BOR Affective Instability Subscale</u>		
Fixed Effects		
Intercept	-.60* (.15)	-.65* (.15)
Avg Estradiol		-.14 (.15)
Weekly Deviation in Estradiol		-.05 (.06)
Conscientiousness		-.18 (.15)
Conscientiousness*Avg Estradiol		.05 (.16)
<i>Conscientiousness*Estradiol Deviation</i>		-.01 (.05)
Random Parameters		
Intercept	.83* (.23)	.83* (.24)
Weekly Deviation in Estradiol		
Residual	.16* (.02)	.16* (.02)
-2 Restricted Log Pseudo-likelihood	370.17	368.84
<u>Dependent Variable: PAI-BOR Identity Disturbance Subscale</u>		
Fixed Effects		
Intercept	-.30* (.11)	-.33* (.11)
Avg Estradiol		-.07 (.11)
Weekly Deviation in Estradiol		-.01 (.05)
Conscientiousness		-.11 (.11)
Conscientiousness*Avg Estradiol		.01 (.11)
<i>Conscientiousness*Estradiol Deviation</i>		.01 (.04)
Random Parameters		
Intercept	.45* (.11)	.44* (.11)
Weekly Deviation in Estradiol		
Residual	.12* (.01)	.12* (.01)
-2 Restricted Log Pseudo-likelihood	274.93	272.18

Table Continued on Next Page

<u>Dependent Variable: PAI-BOR Negative Relationships Subscale</u>		
	Fixed Effects	
Intercept	-.41* (.12)	-.45* (.12)
Avg Estradiol		-.04 (.12)
Weekly Deviation in Estradiol		.03 (.06)
Conscientiousness		-.13 (.12)
Conscientiousness*Avg Estradiol		-.01 (.12)
<i>Conscientiousness*Estradiol Deviation</i>		-.05 (.05)
	Random Parameters	
Intercept	.53* (.14)	.53* (.15)
Weekly Deviation in Estradiol		
Residual	.16* (.02)	.16* (.02)
-2 Restricted Log Pseudo-likelihood	332.16	330.85
<u>Dependent Variable: PAI-BOR Self-Harm Subscale</u>		
	Fixed Effects	
Intercept	-1.03* (.11)	-1.04* (.11)
Avg Estradiol		.02 (.11)
Weekly Deviation in Estradiol		.01 (.09)
Conscientiousness		-.13 (.11)
Conscientiousness*Avg Estradiol		.08 (.11)
<i>Conscientiousness*Estradiol Deviation</i>		-.10 (.08)
	Random Parameters	
Intercept	.35* (.12)	.37* (.13)
Weekly Deviation in Estradiol		
Residual	.18* (.02)	.18* (.02)
-2 Restricted Log Pseudo-likelihood	407.23	405.01
<u>Dependent Variable: Borderline Symptom Checklist—23 (BSL-23)</u>		
	Fixed Effects	
Intercept	2.05* (.16)	1.98* (.33)
Avg Estradiol		2.27* (.30)
Weekly Deviation in Estradiol		-.11 (.07)
Conscientiousness		-.09 (.18)
Conscientiousness*Avg Estradiol		.08 (.18)
<i>Conscientiousness*Estradiol Deviation</i>		.02 (.04)
	Random Parameters	
Intercept	1.06* (.29)	1.15* (.33)
Weekly Deviation in Estradiol		
Residual	2.34* (.31)	2.27* (.30)
-2 Restricted Log Pseudo-likelihood	379.72	378.77

Table Continued on Next Page

Dependent Variable: McLean Screening Instrument for BPD (MSI-BPD)		
	Fixed Effects	
Intercept	.17 (.22)	.08 (.24)
Avg Estradiol		-.12 (.24)
Weekly Deviation in Estradiol		-.08 (.07)
Conscientiousness		-.07 (.24)
Conscientiousness*Avg Estradiol		.18 (.24)
<i>Conscientiousness*Estradiol Deviation</i>		-.03 (.05)
	Random Parameters	
Intercept	1.80* (.54)	1.98* (.62)
Weekly Deviation in Estradiol		
Residual	.68* (.09)	.69* (.09)
-2 Restricted Log Pseudo-likelihood	481.04	480.64

Note. Standard errors are in parentheses.

* $p < .05$.

†Change in -2 Restricted Log Pseudo-likelihood over the previous model is significant at $p < .05$.

$SE = .10, t(37) = 2.90, p = .006$). See Figures 38-40 for depictions of these interactions.

There were no significant interactions of Neuroticism with deviations in estradiol.

Also consistent with dimensional models of BPD in which high Openness to Experience (specifically, high scores on the facets of Openness to Actions and Openness to Feelings) are associated with higher levels of BPD, there were significant interactions of Openness to Experience with *average* levels of estradiol to predicting weekly PAI-BOR total score, PAI-BOR Affective Instability, PAI-BOR Identity Disturbance, the BSL-23, and the MSI-BPD. In the model predicting the PAI-BOR Total score, among women *lower* in Openness, higher average levels of estradiol were associated with higher scores ($\gamma_{\text{LOWOPENNESS} * \text{AVGESTRADIOL}} = .22, SE = .10, t(37) = 2.20, p = .03$); among women *higher* in Openness, higher average levels of estradiol were associated with lower scores ($\gamma_{\text{HIGHOPENNESS} * \text{AVGESTRADIOL}} = -.25, SE = .12, t(37) = -1.98, p = .05$). See Figure 41 for a graph of this interaction. In model predicting the Affective Instability subscale, among women *lower* in Openness, higher average levels of estradiol were associated with higher scores ($\gamma_{\text{LOWOPENNESS} * \text{AVGESTRADIOL}} = .35, SE = .14, t(37) = 2.50, p = .01$); among women *higher* in Openness, higher average levels of estradiol were associated with lower scores ($\gamma_{\text{HIGHOPENNESS} * \text{AVGESTRADIOL}} = -.50, SE = .20, t(37) = -2.50, p = .01$). See Figure 42 for a graph of the interaction. In the model predicting Identity Disturbance, among women *lower* in Openness, higher average levels of estradiol were not significantly associated with higher scores ($\gamma_{\text{LOWOPENNESS} * \text{AVGESTRADIOL}} = .17, SE = .18, t(37) = .33, p = .33$); among women *higher* in Openness, higher average levels of estradiol were associated with lower scores ($\gamma_{\text{HIGHOPENNESS} * \text{AVGESTRADIOL}} = -.25, SE = .11, t(37) = -2.10, p = .04$). This interaction is depicted in Figure 43. In the model predicting the BSL-23, among

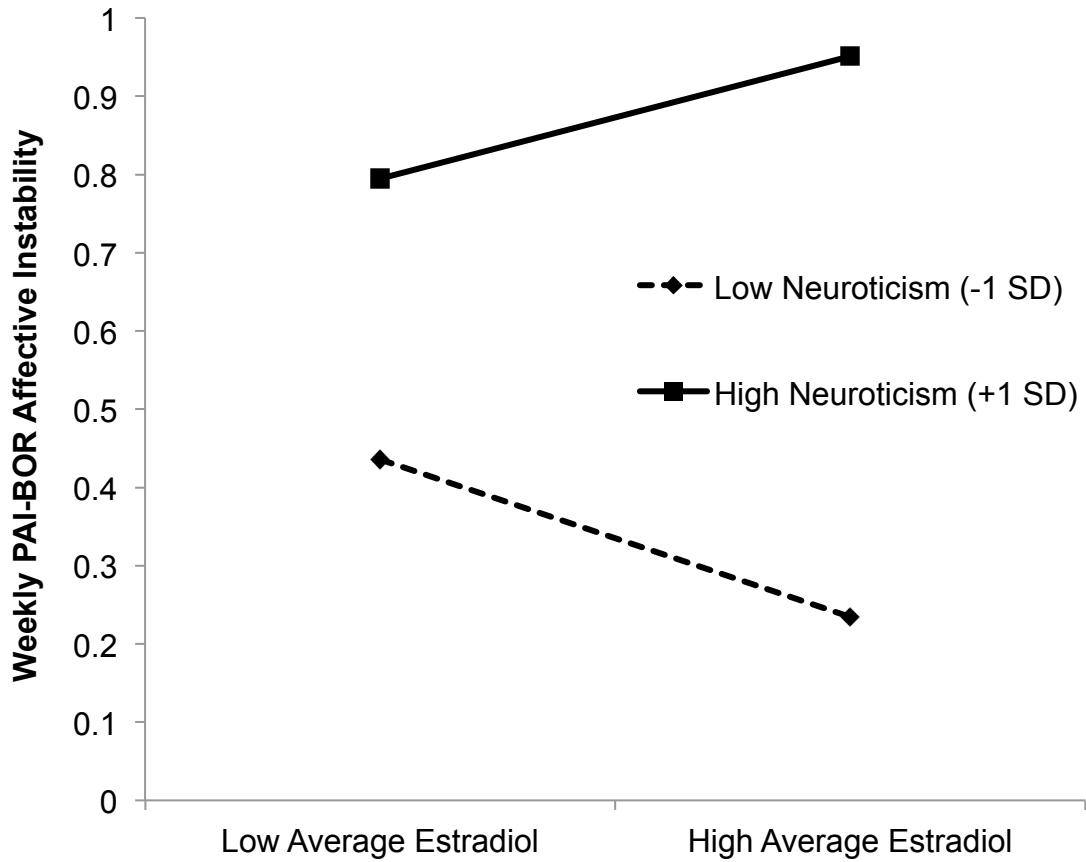


Figure 38. A graph of the interaction between Neuroticism and *average* estradiol predicting weekly PAI-BOR Affective Instability subscale score.

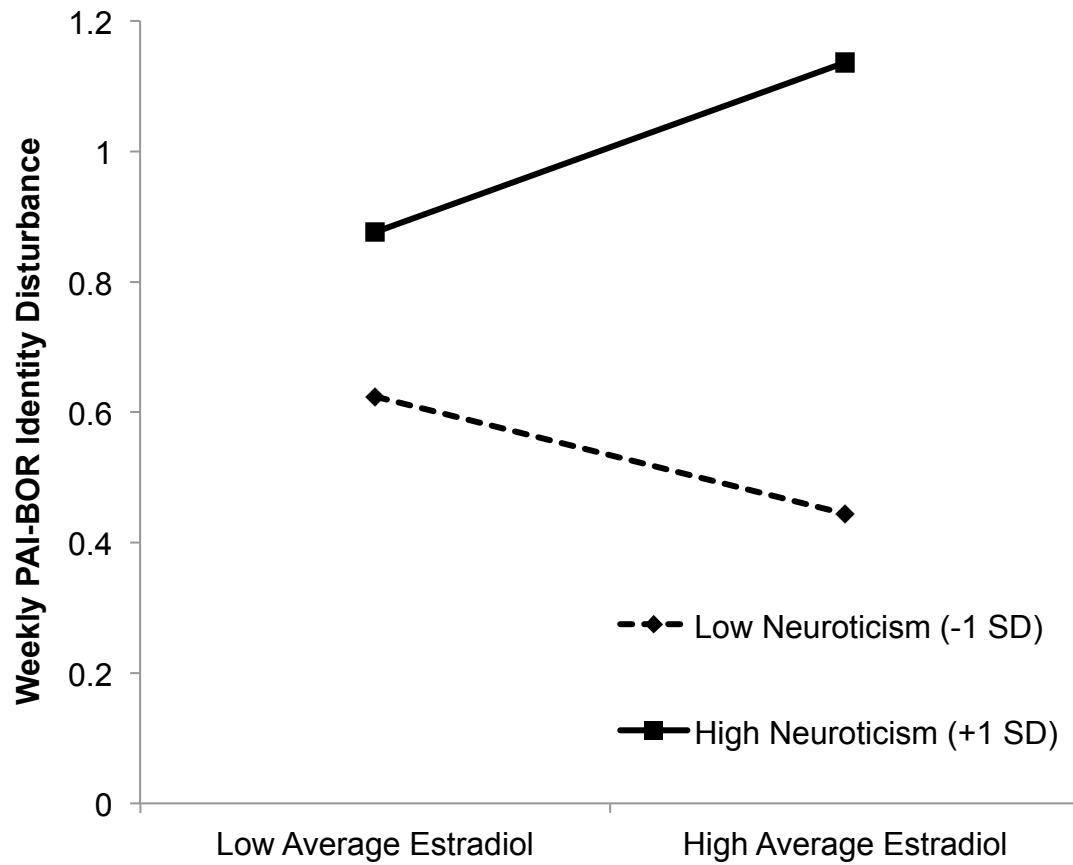


Figure 39. A graph of the interaction between Neuroticism and *average* estradiol predicting weekly PAI-BOR Identity Disturbance subscale score.

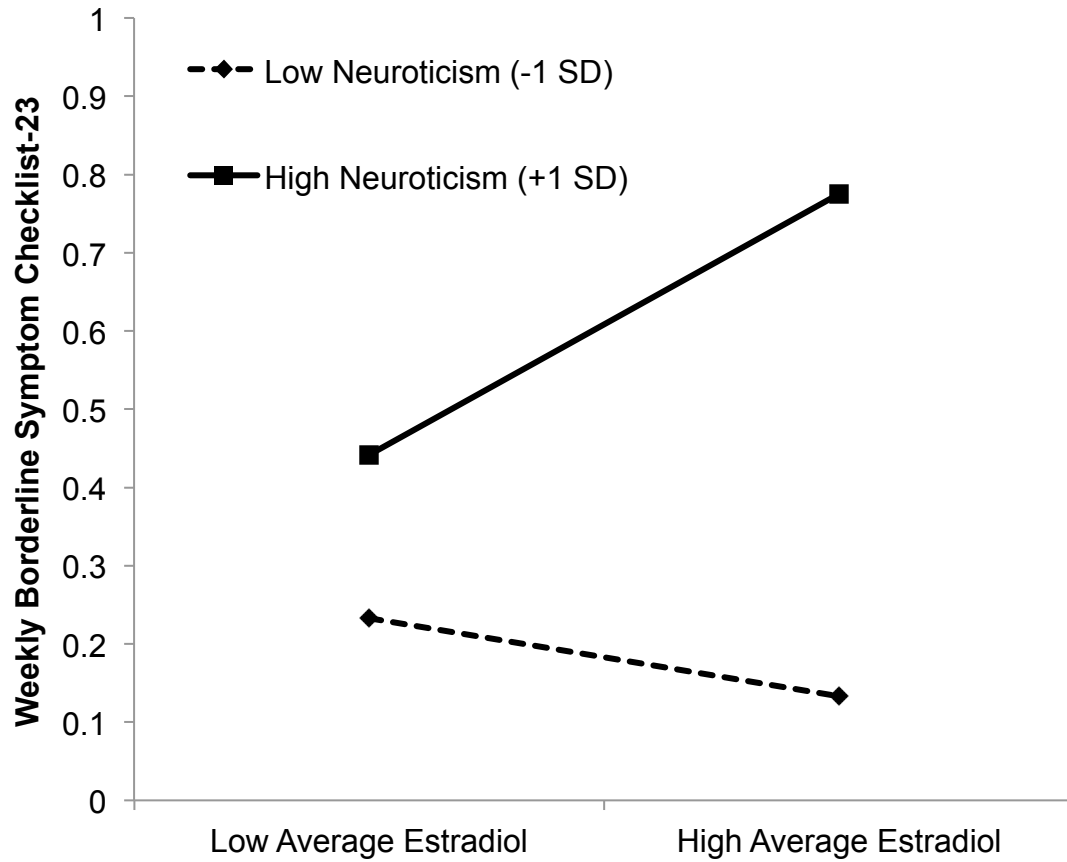


Figure 40. A graph of the interaction between Neuroticism and *average* estradiol predicting weekly BSL-23 score.

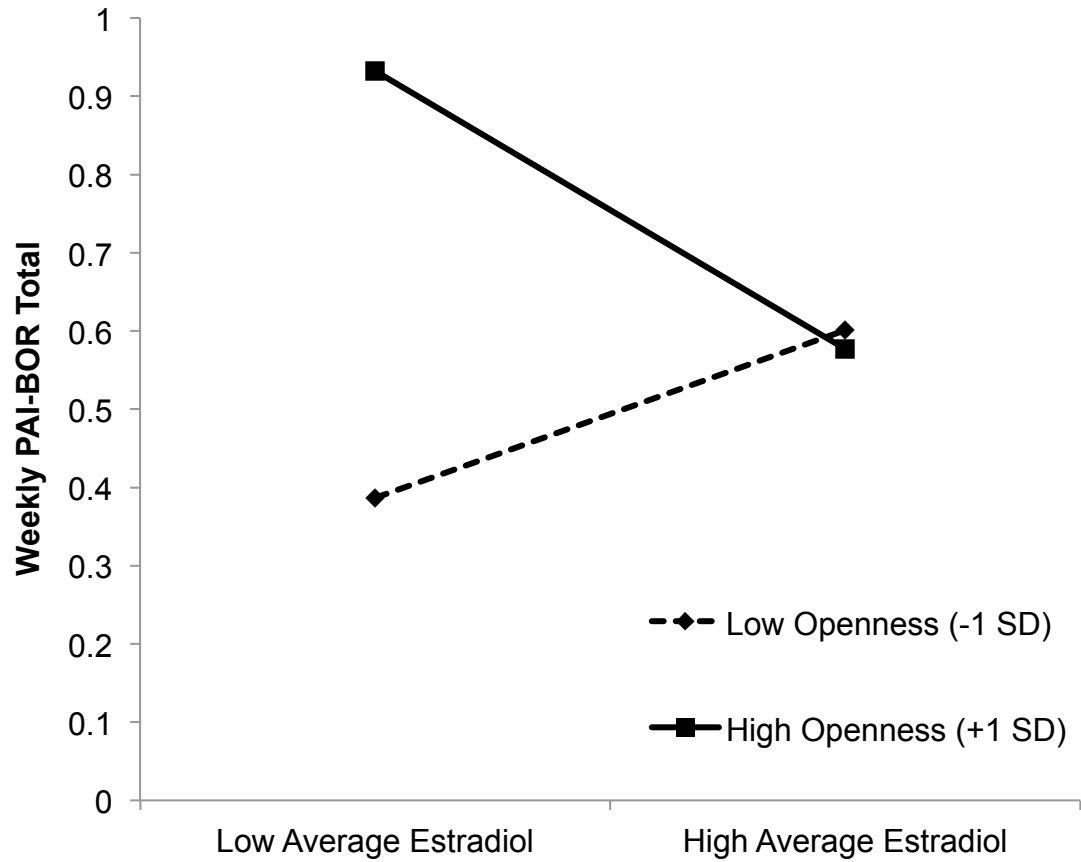


Figure 41. A graph of the interaction between Openness to Experience and average estradiol predicting weekly PAI-BOR total score.

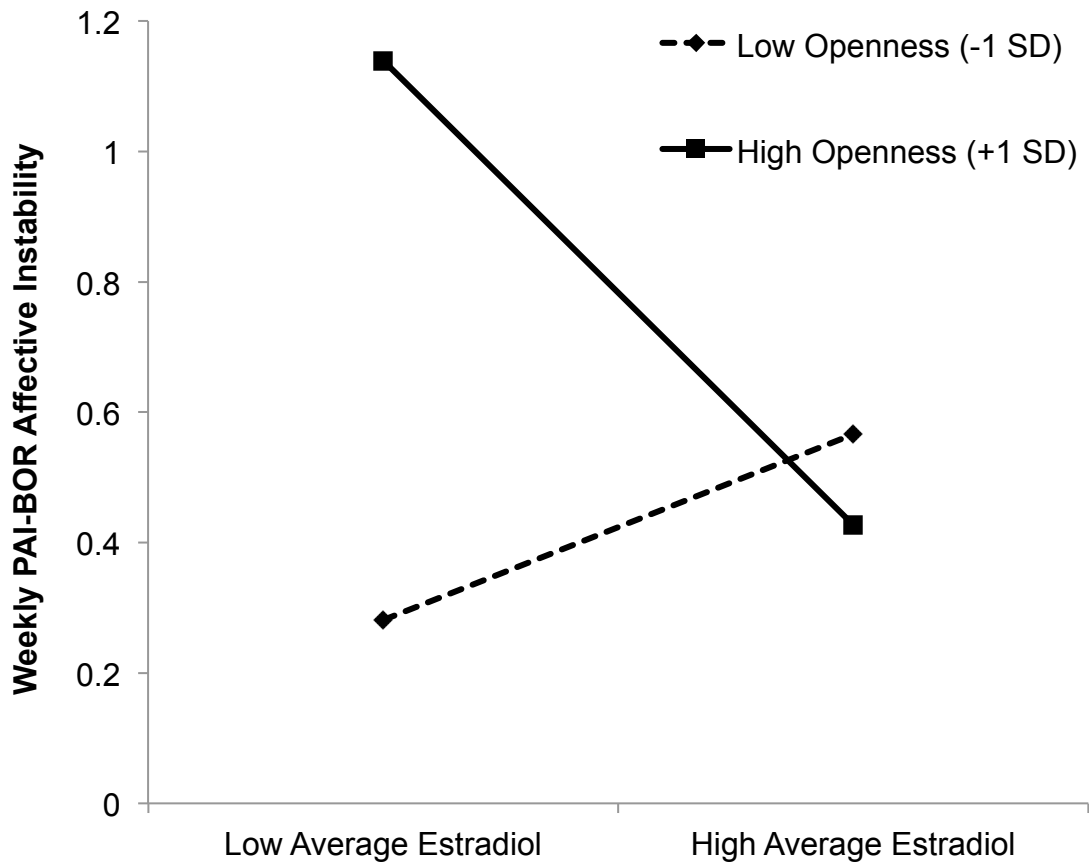


Figure 42. A graph of the interaction between Openness to Experience and average estradiol predicting weekly PAI-BOR Affective Instability subscale score.

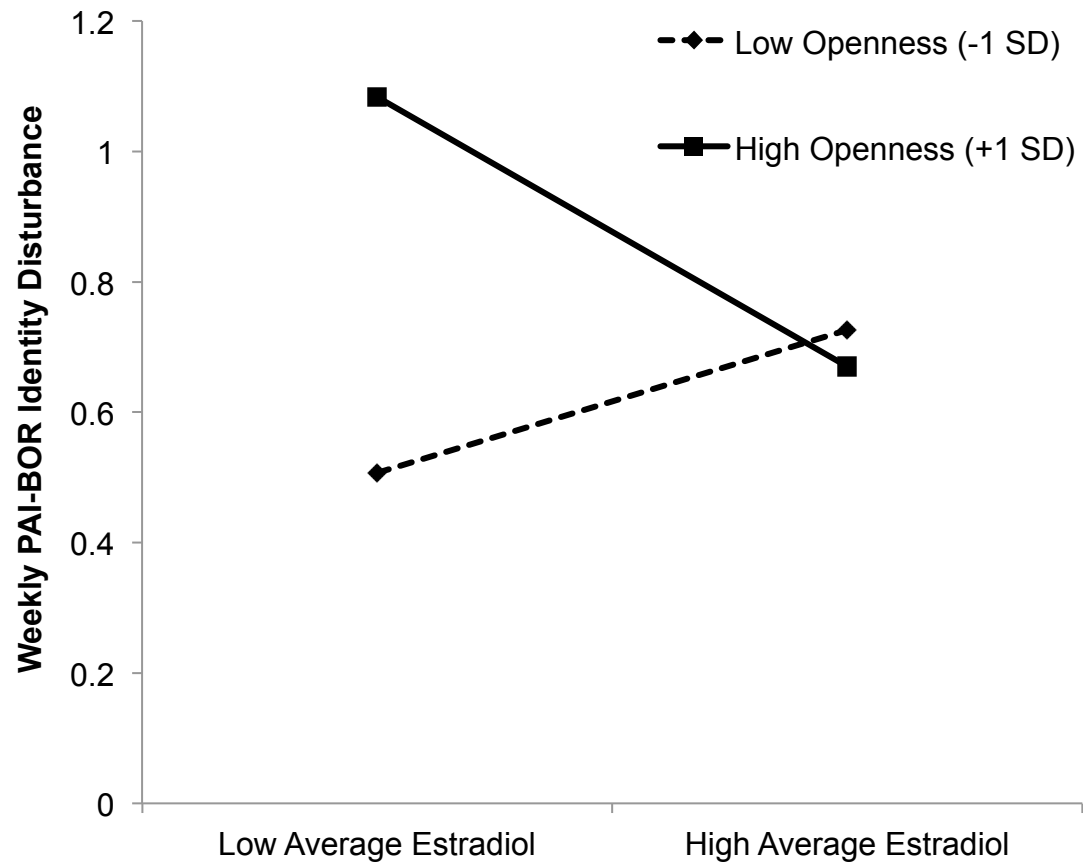


Figure 43. A graph of the interaction between Openness to Experience and average estradiol predicting weekly PAI-BOR Identity Disturbance subscale score.

women *lower* in Openness, higher average levels of estradiol were associated with higher scores ($\gamma_{\text{LOWOPENNESS} * \text{AVGESTRADIOL}} = .59, SE = .27, t(37) = 2.21, p = .03$); among women *higher* in Openness, higher average levels of estradiol were associated with lower scores ($\gamma_{\text{HIGHOPENNESS} * \text{AVGESTRADIOL}} = -.47, SE = .22, t(37) = -2.14, p = .03$). See Figure 44 for a graph of this interaction. Finally, in the model predicting number of symptoms endorsed on the MSI-BPD, among women *lower* in Openness, higher average levels of estradiol were associated with a higher number of symptoms endorsed on the MSI-BPD ($\gamma_{\text{LOWOPENNESS} * \text{AVGESTRADIOL}} = .51, SE = .24, t(37) = 2.12, p = .04$); among women *higher* in Openness, higher average levels of estradiol were associated with fewer symptoms endorsed on the MSI-BPD ($\gamma_{\text{HIGHOPENNESS} * \text{AVGESTRADIOL}} = -.50, SE = .21, t(37) = -2.42, p = .02$). See Figure 45 for a graph of this interaction. There were no interactions of Openness to Experience with weekly deviations in estradiol predicting BPD symptom expression.

In general, there were not significant main effects of or interactions of estradiol variables with Agreeableness or Conscientiousness, the other two FFM variables hypothesized to be abnormal in BPD. There was one exception: higher levels of Agreeableness were associated with lower scores on the PAI-BOR Negative Relationships subscale.

Types of Childhood Maltreatment as Alternative Moderators. Next, I substituted each scale of the CTQ for the Trait BPD variable (again, for original model, see Table 7). Results of models testing interactions of estradiol variables with the subscales of the CTQ are presented in Tables 29-33. Consistent with the biosocial theory, which posits that invalidation of a child's desires and emotions is associated with risk for BPD, both the

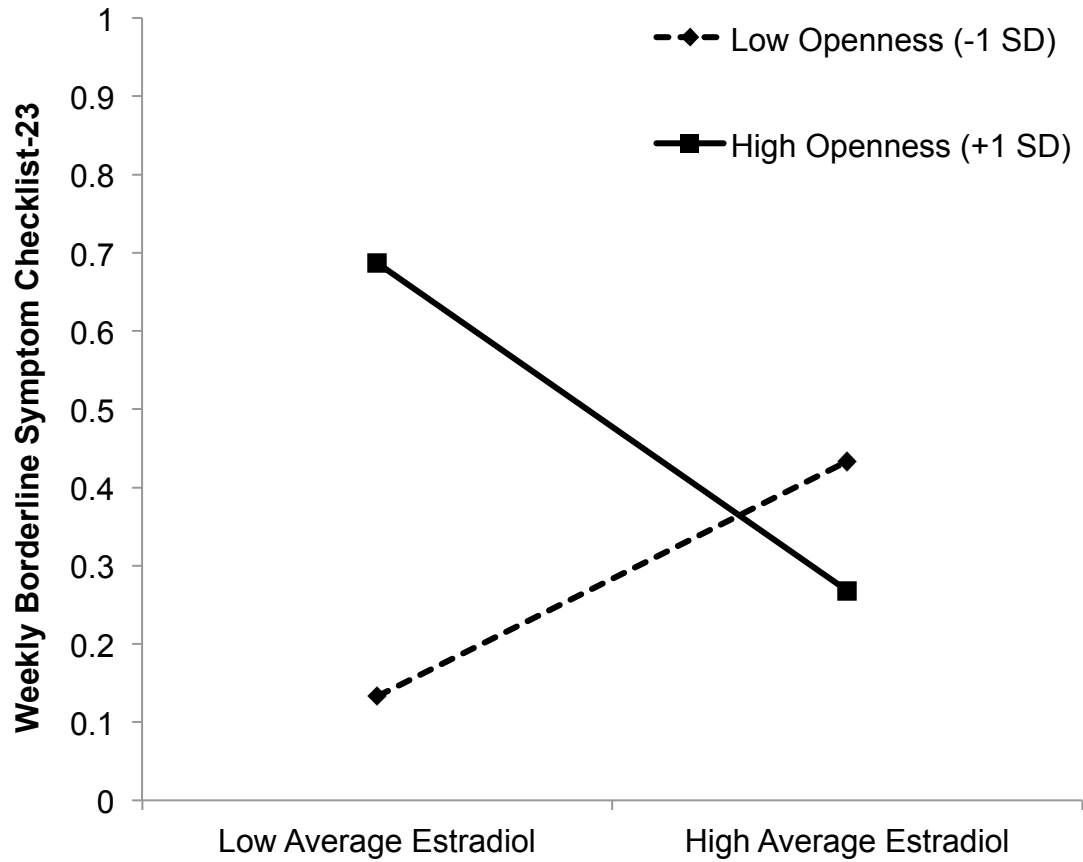


Figure 44. A graph of the interaction between Openness to Experience and average estradiol predicting weekly PAI-BOR Affective Instability subscale score.

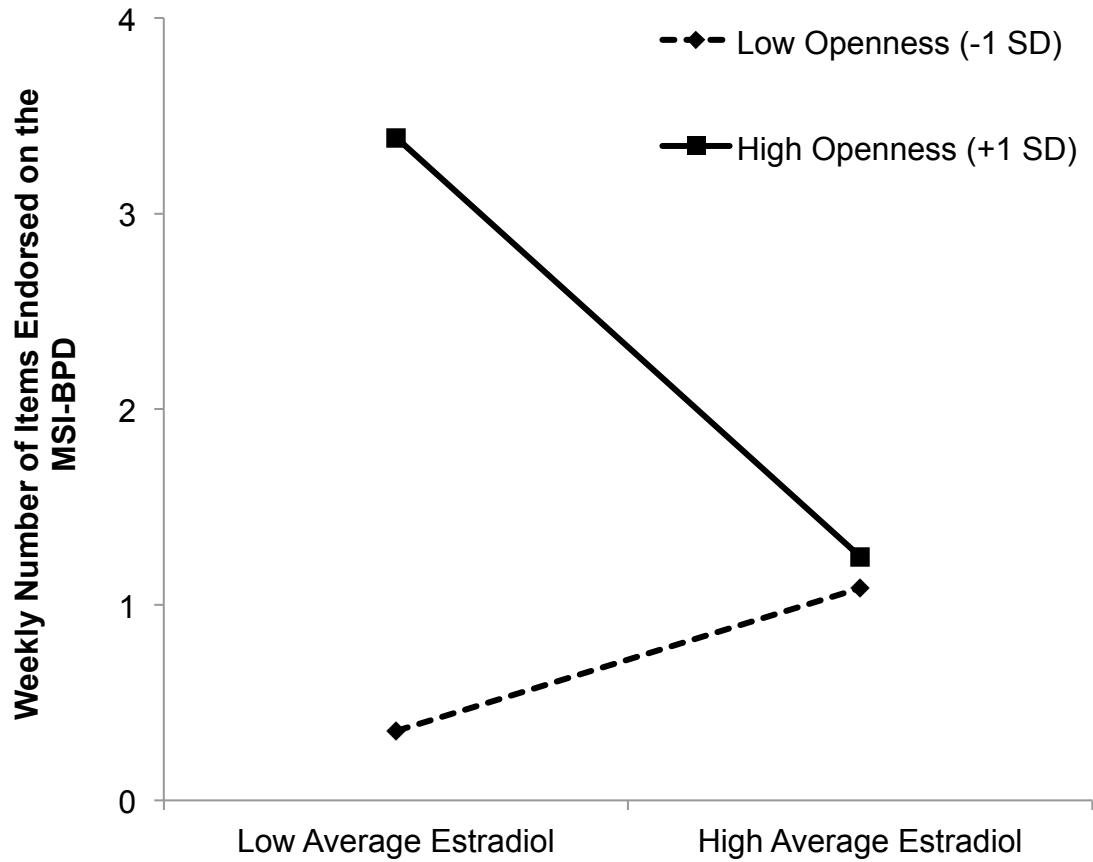


Figure 45. A graph of the interaction between Openness to Experience and average estradiol predicting weekly number of items endorsed on the MSI-BPD.

Table 29

Multilevel Poisson Regression Models Predicting Weekly BPD Symptoms from the Interaction of the Emotional Abuse scale of the CTQ with Average Estradiol and Weekly Deviations in Estradiol

Parameter	Model 1 (null)	Model 4 (with moderator)
<u>Dependent Variable: PAI-BOR Total Score</u>		
Fixed Effects		
Intercept	-.50* (.10)	-.53* (.10)
Avg Estradiol		-.14 (.10)
Weekly Deviation in Estradiol		.01 (.04)
Emotional Abuse		.30* (.13)
Emotional Abuse*Avg Estradiol		-.05 (.12)
<i>Emotional Abuse*Estradiol Deviation</i>		-.01 (.05)
Random Parameters		
Intercept	.39* (.09)	.33* (.08)
Weekly Deviation in Estradiol		
Residual	.08* (.01)	.08* (.01)
-2 Restricted Log Pseudo-likelihood	240.55	237.91
<u>Dependent Variable: PAI-BOR Affective Instability Subscale</u>		
Fixed Effects		
Intercept	-.60* (.15)	-.66* (.15)
Avg Estradiol		-.28 (.15)
Weekly Deviation in Estradiol		.04 (.06)
Emotional Abuse		.44* (.19)
Emotional Abuse*Avg Estradiol		-.07 (.18)
<i>Emotional Abuse*Estradiol Deviation</i>		.01 (.07)
Random Parameters		
Intercept	.83* (.23)	.69* (.20)
Weekly Deviation in Estradiol		
Residual	.16* (.02)	.17* (.02)
-2 Restricted Log Pseudo-likelihood	370.17	368.08
<u>Dependent Variable: PAI-BOR Identity Disturbance Subscale</u>		
Fixed Effects		
Intercept	-.30* (.11)	-.33* (.11)
Avg Estradiol		-.15 (.11)
Weekly Deviation in Estradiol		.01 (.05)
Emotional Abuse		.28* (.13)
Emotional Abuse*Avg Estradiol		.01 (.13)
<i>Emotional Abuse*Estradiol Deviation</i>		-.06 (.05)
Random Parameters		
Intercept	.45* (.11)	.39* (.10)
Weekly Deviation in Estradiol		
Residual	.12* (.01)	-.15 (.11)
-2 Restricted Log Pseudo-likelihood	274.93	272.34

Table Continued on Next Page

<u>Dependent Variable: PAI-BOR Negative Relationships Subscale</u>		
	Fixed Effects	
Intercept	-0.41* (.12)	-0.46* (.12)
Avg Estradiol		-.15 (.12)
Weekly Deviation in Estradiol		.03 (.06)
Emotional Abuse		.30* (.14)
Emotional Abuse*Avg Estradiol		-.01 (.15)
<i>Emotional Abuse*Estradiol Deviation</i>		-.01 (.06)
	Random Parameters	
Intercept	.53* (.14)	.47* (.13)
Weekly Deviation in Estradiol		
Residual	.16* (.02)	.17* (.02)
-2 Restricted Log Pseudo-likelihood	332.16	331.30
<u>Dependent Variable: PAI-BOR Self-Harm Subscale</u>		
	Fixed Effects	
Intercept	-1.03* (.11)	-1.01* (.12)
Avg Estradiol		.01 (.12)
Weekly Deviation in Estradiol		.01 (.09)
Emotional Abuse		.14 (.16)
Emotional Abuse*Avg Estradiol		-.10 (.15)
<i>Emotional Abuse*Estradiol Deviation</i>		.03 (.11)
	Random Parameters	
Intercept	.35* (.12)	.41* (.14)
Weekly Deviation in Estradiol		
Residual	.18* (.02)	.18* (.02)
-2 Restricted Log Pseudo-likelihood	407.23	406.69
<u>Dependent Variable: Borderline Symptom Checklist—23 (BSL-23)</u>		
	Fixed Effects	
Intercept	2.05* (.16)	1.99* (.17)
Avg Estradiol		-.18 (.17)
Weekly Deviation in Estradiol		-.12 (.06)
Emotional Abuse		.54* (.21)
Emotional Abuse*Avg Estradiol		-.07 (.20)
<i>Emotional Abuse*Estradiol Deviation</i>		-.01 (.05)
	Random Parameters	
Intercept	1.06* (.29)	.91* (.27)
Weekly Deviation in Estradiol		
Residual	2.34* (.31)	2.27* (.30)
-2 Restricted Log Pseudo-likelihood	379.72	369.74†

Table Continued on Next Page

Dependent Variable: McLean Screening Instrument for BPD (MSI-BPD)

	Fixed Effects	
Intercept	.17 (.22)	.08 (.24)
Avg Estradiol		-.26 (.24)
Weekly Deviation in Estradiol		-.06 (.07)
Emotional Abuse		.51 (.30)
Emotional Abuse*Avg Estradiol		-.02 (.29)
<i>Emotional Abuse*Estradiol Deviation</i>		-.07 (.08)
	Random Parameters	
Intercept	1.80* (.54)	1.81* (.57)
Weekly Deviation in Estradiol		
Residual	.68* (.09)	.70* (.09)
-2 Restricted Log Pseudo-likelihood	481.04	478.96

Note. Standard errors are in parentheses.

* $p < .05$.

†Change in -2 Restricted Log Pseudo-likelihood over the previous model is significant at $p < .05$.

Table 30

Multilevel Poisson Regression Models Predicting Weekly BPD Symptoms from the Interaction of the Emotional Neglect scale of the CTQ with Average Estradiol and Weekly Deviations in Estradiol

Parameter	Model 1 (null)	Model 4 (with moderator)
<u>Dependent Variable: PAI-BOR Total Score</u>		
Fixed Effects		
Intercept	-.50* (.10)	-.54* (.09)
Avg Estradiol		-.07 (.09)
Weekly Deviation in Estradiol		.01 (.04)
Emotional Neglect		.25* (.09)
Emotional Neglect*Avg Estradiol		-.02 (.09)
<i>Emotional Neglect*Estradiol Deviation</i>		-.02 (.03)
Random Parameters		
Intercept	.39* (.09)	.33* (.09)
Weekly Deviation in Estradiol		
Residual	.08* (.01)	.08* (.01)
-2 Restricted Log Pseudo-likelihood	240.55	237.74
<u>Dependent Variable: PAI-BOR Affective Instability Subscale</u>		
Fixed Effects		
Intercept	-.60* (.15)	-.67* (.14)
Avg Estradiol		-.19 (.14)
Weekly Deviation in Estradiol		-.03 (.07)
Emotional Neglect		.38* (.13)
Emotional Neglect*Avg Estradiol		-.01 (.13)
<i>Emotional Neglect*Estradiol Deviation</i>		-.01 (.05)
Random Parameters		
Intercept	.83* (.23)	.68* (.20)
Weekly Deviation in Estradiol		
Residual	.16* (.02)	.17* (.02)
-2 Restricted Log Pseudo-likelihood	370.17	369.20
<u>Dependent Variable: PAI-BOR Identity Disturbance Subscale</u>		
Fixed Effects		
Intercept	-.30* (.11)	-.33* (.10)
Avg Estradiol		-.08 (.10)
Weekly Deviation in Estradiol		.01 (.05)
Emotional Neglect		.26* (.10)
Emotional Neglect*Avg Estradiol		-.001 (.09)
<i>Emotional Neglect*Estradiol Deviation</i>		-.05 (.04)
Random Parameters		
Intercept	.45* (.11)	.40* (.10)
Weekly Deviation in Estradiol		
Residual	.12* (.01)	.12* (.01)
-2 Restricted Log Pseudo-likelihood	274.93	274.03

Table Continued on Next Page

<u>Dependent Variable: PAI-BOR Negative Relationships Subscale</u>		
	Fixed Effects	
Intercept	-0.41* (.12)	-0.47* (.12)
Avg Estradiol		-0.08 (.12)
Weekly Deviation in Estradiol		.02 (.06)
Emotional Neglect		.24* (.11)
Emotional Neglect*Avg Estradiol		.01 (.11)
<i>Emotional Neglect*Estradiol Deviation</i>		.001 (.05)
	Random Parameters	
Intercept	.53* (.14)	.49* (.14)
Weekly Deviation in Estradiol		
Residual	.16* (.02)	.17* (.02)
-2 Restricted Log Pseudo-likelihood	332.16	330.01
<u>Dependent Variable: PAI-BOR Self-Harm Subscale</u>		
	Fixed Effects	
Intercept	-1.03* (.11)	-1.03* (.11)
Avg Estradiol		.03 (.12)
Weekly Deviation in Estradiol		.01 (.09)
Emotional Neglect		.12 (.11)
Emotional Neglect*Avg Estradiol		-.11 (.10)
<i>Emotional Neglect*Estradiol Deviation</i>		-.003 (.08)
	Random Parameters	
Intercept	.35* (.12)	.40* (.14)
Weekly Deviation in Estradiol		
Residual	.18* (.02)	.18* (.02)
-2 Restricted Log Pseudo-likelihood	407.23	407.13
<u>Dependent Variable: Borderline Symptom Checklist—23 (BSL-23)</u>		
	Fixed Effects	
Intercept	2.05* (.16)	1.98* (.26)
Avg Estradiol		-0.08 (.16)
Weekly Deviation in Estradiol		-.13* (.06)
Emotional Neglect		.49* (.15)
Emotional Neglect*Avg Estradiol		.03 (.14)
<i>Emotional Neglect*Estradiol Deviation</i>		.01 (.04)
	Random Parameters	
Intercept	1.06* (.29)	.87* (.26)
Weekly Deviation in Estradiol		
Residual	2.34* (.31)	2.27* (.31)
-2 Restricted Log Pseudo-likelihood	379.72	369.90†

Table Continued on Next Page

Dependent Variable: McLean Screening Instrument for BPD (MSI-BPD)

	Fixed Effects	
Intercept	.17 (.22)	.09 (.22)
Avg Estradiol		-.13 (.23)
Weekly Deviation in Estradiol		-.08 (.08)
Emotional Neglect		.52* (.21)
Emotional Neglect*Avg Estradiol		-.05 (.20)
<i>Emotional Neglect*Estradiol Deviation</i>		-.003 (.05)
	Random Parameters	
Intercept	1.80* (.54)	1.71* (.55)
Weekly Deviation in Estradiol		
Residual	.68* (.09)	.71* (.09)
-2 Restricted Log Pseudo-likelihood	481.04	478.93

Note. Standard errors are in parentheses.

* $p < .05$.

†Change in -2 Restricted Log Pseudo-likelihood over the previous model is significant at $p < .05$.

Table 31

Multilevel Poisson Regression Models Predicting Weekly BPD Symptoms from the Interaction of the Physical Abuse scale of the CTQ with Average Estradiol and Weekly Deviations in Estradiol

Parameter	Model 1 (null)	Model 4 (with moderator)
<u>Dependent Variable: PAI-BOR Total Score</u>		
Fixed Effects		
Intercept	-.50* (.10)	-.52* (.11)
Avg Estradiol		-.14 (.12)
Weekly Deviation in Estradiol		-.001 (.04)
Physical Abuse		.26 (.24)
Physical Abuse*Avg Estradiol		-.08 (.21)
<i>Physical Abuse*Estradiol Deviation</i>		.01 (.05)
Random Parameters		
Intercept	.39* (.09)	.38* (.10)
Weekly Deviation in Estradiol		
Residual	.08* (.01)	.08* (.01)
-2 Restricted Log Pseudo-likelihood	240.55	238.75
<u>Dependent Variable: PAI-BOR Affective Instability Subscale</u>		
Fixed Effects		
Intercept	-.60* (.15)	-.62* (.16)
Avg Estradiol		-.32 (.17)
Weekly Deviation in Estradiol		-.04 (.06)
Physical Abuse		.51 (.34)
Physical Abuse*Avg Estradiol		-.23 (.31)
<i>Physical Abuse*Estradiol Deviation</i>		.01 (.08)
Random Parameters		
Intercept	.83* (.23)	.77* (.23)
Weekly Deviation in Estradiol		
Residual	.16* (.02)	.17* (.02)
-2 Restricted Log Pseudo-likelihood	370.17	370.04
<u>Dependent Variable: PAI-BOR Identity Disturbance Subscale</u>		
Fixed Effects		
Intercept	-.30* (.11)	-.36* (.12)
Avg Estradiol		-.10 (.12)
Weekly Deviation in Estradiol		.01 (.05)
Physical Abuse		.08 (.26)
Physical Abuse*Avg Estradiol		.11 (.23)
<i>Physical Abuse*Estradiol Deviation</i>		-.01 (.06)
Random Parameters		
Intercept	.45* (.11)	.43 (.11)
Weekly Deviation in Estradiol		
Residual	.12* (.01)	.12* (.01)
-2 Restricted Log Pseudo-likelihood	274.93	273.48

Table Continued on Next Page

<u>Dependent Variable: PAI-BOR Negative Relationships Subscale</u>		
	Fixed Effects	
Intercept	-0.41* (.12)	-0.45* (.14)
Avg Estradiol		-.13 (.14)
Weekly Deviation in Estradiol		.02 (.06)
Physical Abuse		.22 (.29)
Physical Abuse*Avg Estradiol		-.05 (.26)
<i>Physical Abuse*Estradiol Deviation</i>		.04 (.07)
	Random Parameters	
Intercept	.53* (.14)	.53* (.15)
Weekly Deviation in Estradiol		
Residual	.16* (.02)	.17* (.02)
-2 Restricted Log Pseudo-likelihood	332.16	331.24
<u>Dependent Variable: PAI-BOR Self-Harm Subscale</u>		
	Fixed Effects	
Intercept	-1.03* (.11)	-.97* (.13)
Avg Estradiol		-.04 (.13)
Weekly Deviation in Estradiol		.01 (.09)
Physical Abuse		.30 (.27)
Physical Abuse*Avg Estradiol		-.27 (.24)
<i>Physical Abuse*Estradiol Deviation</i>		-.01 (.13)
	Random Parameters	
Intercept	.35* (.12)	.40* (.14)
Weekly Deviation in Estradiol		
Residual	.18* (.02)	.18* (.02)
-2 Restricted Log Pseudo-likelihood	407.23	405.73
<u>Dependent Variable: Borderline Symptom Checklist—23 (BSL-23)</u>		
	Fixed Effects	
Intercept	2.05* (.16)	1.97* (.19)
Avg Estradiol		-.18 (.19)
Weekly Deviation in Estradiol		-.14 (.06)
Physical Abuse		.41 (.40)
Physical Abuse*Avg Estradiol		-.01 (.35)
<i>Physical Abuse*Estradiol Deviation</i>		.04 (.06)
	Random Parameters	
Intercept	1.06* (.29)	1.03* (.30)
Weekly Deviation in Estradiol		
Residual	2.34* (.31)	2.26* (.30)
-2 Restricted Log Pseudo-likelihood	379.72	376.69

Table Continued on Next Page

Dependent Variable: McLean Screening Instrument for BPD (MSI-BPD)

	Fixed Effects	
Intercept	.17 (.22)	.17 (.26)
Avg Estradiol		-.32 (.27)
Weekly Deviation in Estradiol		-.07 (.07)
Physical Abuse		.77 (.54)
Physical Abuse*Avg Estradiol		-.39 (.48)
<i>Physical Abuse*Estradiol Deviation</i>		-.02 (.09)
	Random Parameters	
Intercept	1.80* (.54)	1.89* (.60)
Weekly Deviation in Estradiol		
Residual	.68* (.09)	.71* (.09)
-2 Restricted Log Pseudo-likelihood	481.04	479.47

Note. Standard errors are in parentheses.

* $p < .05$.

†Change in -2 Restricted Log Pseudo-likelihood over the previous model is significant at $p < .05$.

Table 32

Multilevel Poisson Regression Models Predicting Weekly BPD Symptoms from the Interaction of the Physical Neglect scale of the CTQ with Average Estradiol and Weekly Deviations in Estradiol

Parameter	Model 1 (null)	Model 4 (with moderator)
<u>Dependent Variable: PAI-BOR Total Score</u>		
Fixed Effects		
Intercept	-.50* (.10)	-.54* (.10)
Avg Estradiol		-.10 (.10)
Weekly Deviation in Estradiol		.01 (.04)
Physical Neglect		.19 (.13)
Physical Neglect*Avg Estradiol		.001 (.13)
<i>Physical Neglect*Estradiol Deviation</i>		-.04 (.04)
Random Parameters		
Intercept	.39* (.09)	.37* (.09)
Weekly Deviation in Estradiol		
Residual	.08* (.01)	.08* (.01)
-2 Restricted Log Pseudo-likelihood	240.55	237.25
<u>Dependent Variable: PAI-BOR Affective Instability Subscale</u>		
Fixed Effects		
Intercept	-.60* (.15)	-.67* (.15)
Avg Estradiol		-.24 (.15)
Weekly Deviation in Estradiol		-.02 (.07)
Physical Neglect		.34 (.19)
Physical Neglect*Avg Estradiol		-.05 (.19)
<i>Physical Neglect*Estradiol Deviation</i>		-.06 (.06)
Random Parameters		
Intercept	.83* (.23)	.75* (.22)
Weekly Deviation in Estradiol		
Residual	.16* (.02)	.16* (.02)
-2 Restricted Log Pseudo-likelihood	370.17	370.10
<u>Dependent Variable: PAI-BOR Identity Disturbance Subscale</u>		
Fixed Effects		
Intercept	-.30* (.11)	-.34* (.11)
Avg Estradiol		-.10 (.11)
Weekly Deviation in Estradiol		.01 (.05)
Physical Neglect		.17 (.14)
Physical Neglect*Avg Estradiol		.07 (.14)
<i>Physical Neglect*Estradiol Deviation</i>		-.09 (.05)
Random Parameters		
Intercept	.45* (.11)	.42* (.11)
Weekly Deviation in Estradiol		
Residual	.12* (.01)	.12* (.01)
-2 Restricted Log Pseudo-likelihood	274.93	272.54

Table Continued on Next Page

<u>Dependent Variable: PAI-BOR Negative Relationships Subscale</u>		
	Fixed Effects	
Intercept	-0.41* (.12)	-0.47* (.12)
Avg Estradiol		-.10 (.12)
Weekly Deviation in Estradiol		.02 (.06)
Physical Neglect		.20 (.16)
Physical Neglect*Avg Estradiol		-.01 (.16)
<i>Physical Neglect*Estradiol Deviation</i>		.03 (.05)
	Random Parameters	
Intercept	.53* (.14)	.52* (.15)
Weekly Deviation in Estradiol		
Residual	.16* (.02)	.17* (.02)
-2 Restricted Log Pseudo-likelihood	332.16	331.18
<u>Dependent Variable: PAI-BOR Self-Harm Subscale</u>		
	Fixed Effects	
Intercept	-1.03* (.11)	-1.04* (.12)
Avg Estradiol		.01 (.12)
Weekly Deviation in Estradiol		.01 (.09)
Physical Neglect		.04 (.16)
Physical Neglect*Avg Estradiol		-.01 (.16)
<i>Physical Neglect*Estradiol Deviation</i>		-.001 (.10)
	Random Parameters	
Intercept	.35* (.12)	.42* (.15)
Weekly Deviation in Estradiol		
Residual	.18* (.02)	.18* (.02)
-2 Restricted Log Pseudo-likelihood	407.23	406.48
<u>Dependent Variable: Borderline Symptom Checklist—23 (BSL-23)</u>		
	Fixed Effects	
Intercept	2.05* (.16)	1.97* (.17)
Avg Estradiol		-.12 (.17)
Weekly Deviation in Estradiol		-.12 (.07)
Physical Neglect		.37 (.22)
Physical Neglect*Avg Estradiol		.03 (.22)
<i>Physical Neglect*Estradiol Deviation</i>		-.01 (.04)
	Random Parameters	
Intercept	1.06* (.29)	1.01* (.17)
Weekly Deviation in Estradiol		
Residual	2.34* (.31)	2.26* (.30)
-2 Restricted Log Pseudo-likelihood	379.72	377.11

Table Continued on Next Page

Dependent Variable: McLean Screening Instrument for BPD (MSI-BPD)

	Fixed Effects	
Intercept	.17 (.22)	.09 (.23)
Avg Estradiol		-.19 (.24)
Weekly Deviation in Estradiol		-.08 (.08)
Physical Neglect		.45 (.31)
Physical Neglect*Avg Estradiol		-.10 (.31)
<i>Physical Neglect*Estradiol Deviation</i>		.002 (.06)
	Random Parameters	
Intercept	1.80* (.54)	1.87* (.60)
Weekly Deviation in Estradiol		
Residual	.68* (.09)	.71* (.09)
-2 Restricted Log Pseudo-likelihood	481.04	480.60

Note. Standard errors are in parentheses.

* $p < .05$.

†Change in -2 Restricted Log Pseudo-likelihood over the previous model is significant at $p < .05$.

Table 33

Multilevel Poisson Regression Models Predicting Weekly BPD Symptoms from the Interaction of the Sexual Abuse scale of the CTQ with Average Estradiol and Weekly Deviations in Estradiol

Parameter	Model 1 (null)	Model 4 (with moderator)
<u>Dependent Variable: PAI-BOR Total Score</u>		
Fixed Effects		
Intercept	-.50* (.10)	-.55* (.10)
Avg Estradiol		-.09(.10)
Weekly Deviation in Estradiol		-.01 (.04)
Sexual Abuse		.09 (.10)
Sexual Abuse*Avg Estradiol		.12 (.08)
<i>Sexual Abuse*Estradiol Deviation</i>		-.15* (.06)
Random Parameters		
Intercept	.39* (.09)	.37* (.10)
Weekly Deviation in Estradiol		
Residual	.08* (.01)	.08* (.01)
-2 Restricted Log Pseudo-likelihood	240.55	234.20†
<u>Dependent Variable: PAI-BOR Affective Instability Subscale</u>		
Fixed Effects		
Intercept	-.60* (.15)	-.69* (.15)
Avg Estradiol		-.21 (.15)
Weekly Deviation in Estradiol		-.07 (.06)
Sexual Abuse		.16 (.15)
Sexual Abuse*Avg Estradiol		.12 (.12)
<i>Sexual Abuse*Estradiol Deviation</i>		-.21* (.09)
Random Parameters		
Intercept	.83* (.23)	.83* (.23)
Weekly Deviation in Estradiol		
Residual	.16* (.02)	.16* (.02)
-2 Restricted Log Pseudo-likelihood	370.17	360.97†
<u>Dependent Variable: PAI-BOR Identity Disturbance Subscale</u>		
Fixed Effects		
Intercept	-.30* (.11)	-.34* (.10)
Avg Estradiol		-.10 (.11)
Weekly Deviation in Estradiol		-.02 (.05)
Sexual Abuse		.11 (.10)
Sexual Abuse*Avg Estradiol		.16 (.09)
<i>Sexual Abuse*Estradiol Deviation</i>		-.24* (.07)
Random Parameters		
Intercept	.45* (.11)	.41* (.11)
Weekly Deviation in Estradiol		
Residual	.12* (.01)	.11* (.01)
-2 Restricted Log Pseudo-likelihood	274.93	267.30†

Table Continued on Next Page

<u>Dependent Variable: PAI-BOR Negative Relationships Subscale</u>		
		Fixed Effects
Intercept	-.41* (.12)	-.47* (.12)
Avg Estradiol		-.09 (.12)
Weekly Deviation in Estradiol		.02 (.06)
Sexual Abuse		.13 (.12)
Sexual Abuse*Avg Estradiol		.10 (.10)
<i>Sexual Abuse*Estradiol Deviation</i>		-.01 (.09)
		Random Parameters
Intercept	.53* (.14)	.52* (.15)
Weekly Deviation in Estradiol		
Residual	.16* (.02)	.17* (.02)
-2 Restricted Log Pseudo-likelihood	332.16	327.22†

<u>Dependent Variable: PAI-BOR Self-Harm Subscale</u>		
		Fixed Effects
Intercept	-1.03* (.11)	-1.06* (.11)
Avg Estradiol		.04 (.11)
Weekly Deviation in Estradiol		-.004 (.10)
Sexual Abuse		-.21 (.15)
Sexual Abuse*Avg Estradiol		.25* (.10)
<i>Sexual Abuse*Estradiol Deviation</i>		-.09 (.17)
		Random Parameters
Intercept	.35* (.12)	.35* (.12)
Weekly Deviation in Estradiol		
Residual	.18* (.02)	.18* (.02)
-2 Restricted Log Pseudo-likelihood	407.23	404.35†

<u>Dependent Variable: Borderline Symptom Checklist—23 (BSL-23)</u>		
		Fixed Effects
Intercept	2.05* (.16)	1.96* (.17)
Avg Estradiol		-.10 (.17)
Weekly Deviation in Estradiol		-.14 (.06)
Sexual Abuse		.14 (.17)
Sexual Abuse*Avg Estradiol		.29* (.14)
<i>Sexual Abuse*Estradiol Deviation</i>		-.15* (.07)
		Random Parameters
Intercept	1.06* (.29)	.65* (.19)
Weekly Deviation in Estradiol		
Residual	2.34* (.31)	2.24* (.30)
-2 Restricted Log Pseudo-likelihood	379.72	369.64†

Table Continued on Next Page

Dependent Variable: McLean Screening Instrument for BPD (MSI-BPD)

	Fixed Effects	
Intercept	.17 (.22)	.07 (.23)
Avg Estradiol		-.15 (.23)
Weekly Deviation in Estradiol		-.10 (.07)
Sexual Abuse		.03 (.23)
Sexual Abuse*Avg Estradiol		.33 (.20)
<i>Sexual Abuse*Estradiol Deviation</i>		-.13 (.12)
	Random Parameters	
Intercept	1.80* (.54)	1.81* (.59)
Weekly Deviation in Estradiol		
Residual	.68* (.09)	.71* (.09)
-2 Restricted Log Pseudo-likelihood	481.04	478.51†

Note. Standard errors are in parentheses.

* $p < .05$.

†Change in -2 Restricted Log Pseudo-likelihood over the previous model is significant at $p < .05$.

emotional abuse and emotional neglect subscales of the CTQ predicted higher BPD scores in all cases except in the prediction of the Self-Harm subscale. However, these subscales did not interact significantly with estradiol variables to predict BPD symptoms. The Sexual Abuse subscale, however, significantly interacted with estradiol variables to predict BPD symptom expression.

There were significant interactions between Sexual Abuse and higher average levels of estradiol predicting both the PAI-BOR Self-Harm subscale and the BSL-23, following a similar pattern as the interactions of Neuroticism and average estradiol in the previous section. In the model predicting the PAI-BOR Self-Harm subscale, among women reporting *higher* levels of Sexual Abuse, higher average levels of estradiol were associated with higher Self-Harm ($\gamma_{\text{HIGHSEXABUSE*AVGESTRADIOL}} = .30, SE = .09, t(37) = 3.33, p = .001$), whereas among women reporting *lower* levels of Sexual Abuse, higher average levels of estradiol were associated with lower Self-Harm ($\gamma_{\text{LOWSEXABUSE*AVGESTRADIOL}} = -.21, SE = .04, t(37) = -5.25, p < .0001$). See Figure 46 for a graph of this interaction. In the model predicting the BSL-23, among women reporting *higher* levels of Sexual Abuse, higher levels of average estradiol were associated with higher scores ($\gamma_{\text{HIGHSEXABUSE*AVGESTRADIOL}} = .21, SE = .08, t(37) = 2.62, p = .01$), whereas among women reporting *lower* levels of Sexual Abuse, higher levels of estradiol were associated with lower scores ($\gamma_{\text{LOWSEXABUSE*AVGESTRADIOL}} = -.40, SE = .14, t(37) = -2.85, p = .007$). See Figure 47 for a graph of this interaction.

There were also significant interactions between levels of Sexual Abuse and weekly deviations in estradiol predicting the PAI-BOR total scale, the PAI-BOR Affective Instability subscale, the PAI-BOR Identity Disturbance subscale, and the BSL-

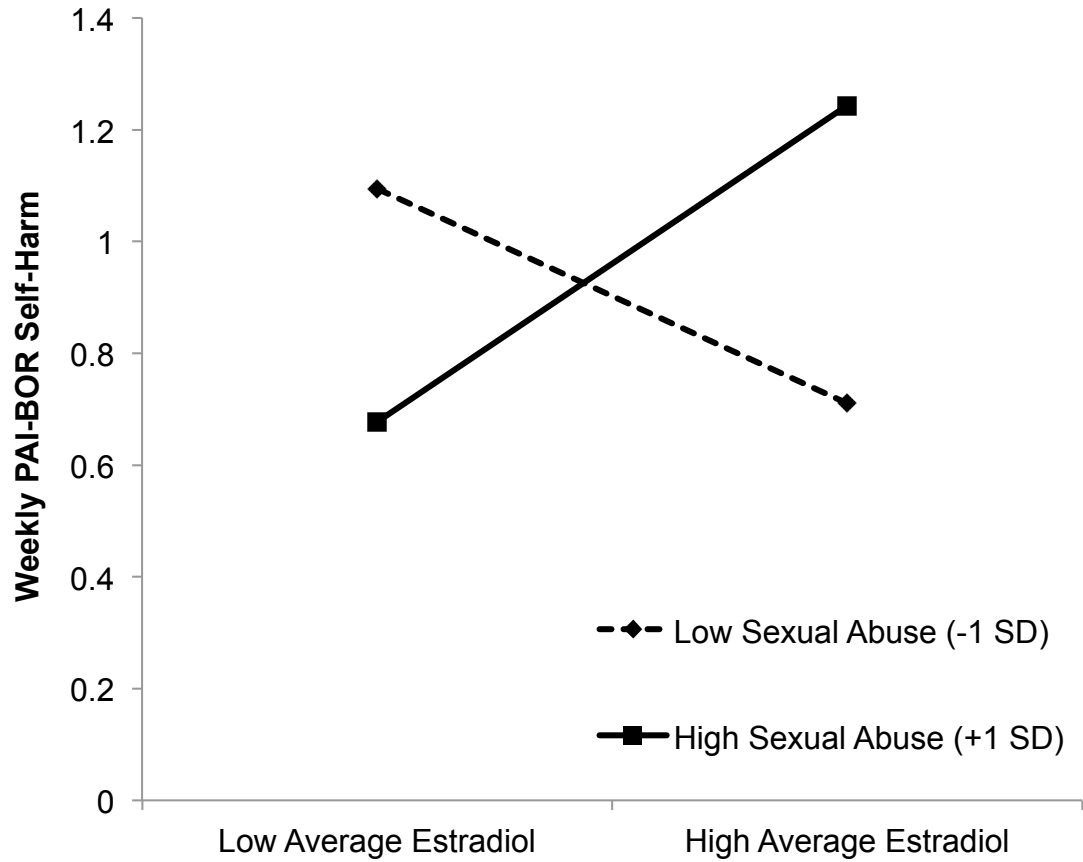


Figure 46. A graph of the interaction between self-reported levels of childhood sexual abuse and *average* estradiol predicting weekly PAI-BOR Self-Harm subscale score.

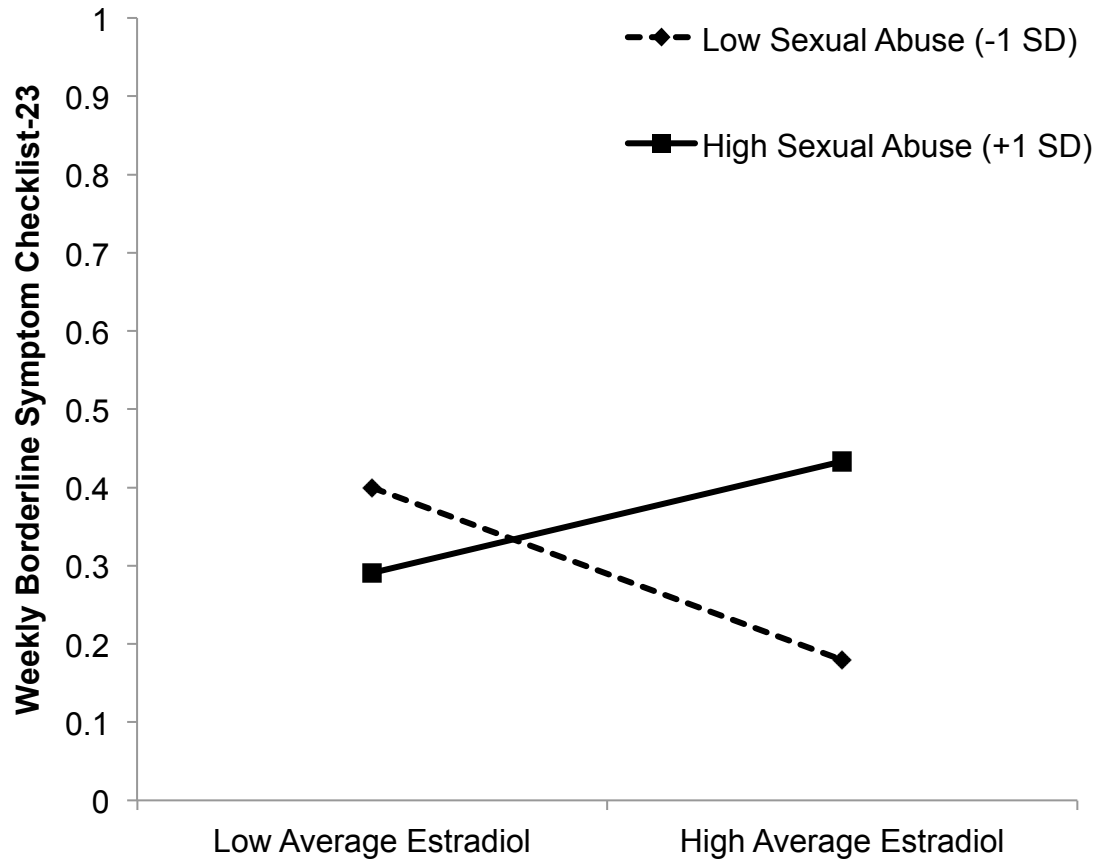


Figure 47. A graph of the interaction between self-reported levels of childhood sexual abuse and *average* estradiol predicting weekly BSL-23 score.

23, though they indicated that the impact of higher-than-usual estradiol was opposite from high average levels estradiol. In the model predicting the PAI-BOR total scale, among women reporting *higher* levels of Sexual Abuse, higher-than-usual levels of estradiol were associated with lower BPD symptom expression

($\gamma_{\text{HIGHSEXABUSE*ESTRADIOLDEVIATION}} = -.17, SE = .09, t(37) = -1.95, p = .05$) whereas among women reporting *lower* levels of Sexual Abuse, higher-than-usual levels of estradiol were associated with higher BPD symptom expression ($\gamma_{\text{LOWSEXABUSE*ESTRADIOLDEVIATION}} = .13, SE = .02, t(37) = 6.50, p < .0001$). A graph of this interaction can be found in Figure 48.

In the model predicting the PAI-BOR Affective Instability subscale, among women reporting *higher* levels of Sexual Abuse, higher-than-usual levels of estradiol were associated with lower levels of Affective Instability ($\gamma_{\text{HIGHSEXABUSE*ESTRADIOLDEVIATION}} = -.28, SE = .12, t(37) = -2.21, p = .02$), whereas among women reporting *lower* levels of Sexual Abuse, higher-than-usual levels of estradiol were not associated with lower Affective Instability ($\gamma_{\text{LOWSEXABUSE*ESTRADIOLDEVIATION}} = .14, SE = .10, t(37) = 1.32, p = .19$). See Figure 49 for a graph of this interaction. In the model predicting the PAI-

BOR Identity Disturbance scale, among women reporting *higher* levels of Sexual Abuse, higher-than-usual levels of estradiol were associated with lower Identity Disturbance ($\gamma_{\text{HIGHSEXABUSE*ESTRADIOLDEVIATION}} = -.26, SE = .09, t(37) = -2.82, p = .005$), whereas among women reporting *lower* levels of Sexual Abuse, higher-than-usual levels of estradiol were associated with higher Identity Disturbance

($\gamma_{\text{LOWSEXABUSE*ESTRADIOLDEVIATION}} = .21, SE = .08, t(37) = 2.66, p = .008$). See Figure 50 for a graph of this interaction. In the model predicting the BSL-23, among women reporting *higher* levels of Sexual Abuse, higher-than-usual levels of estradiol were

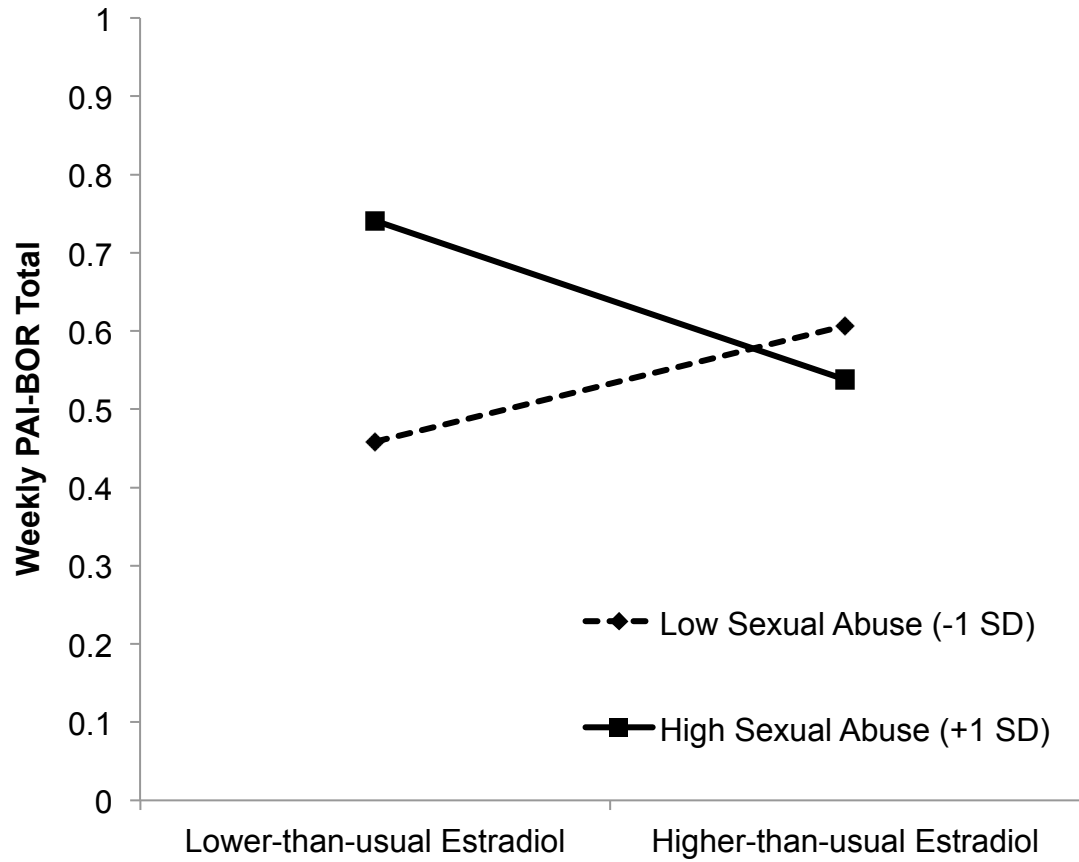


Figure 48. A graph of the interaction between self-reported levels of childhood sexual abuse and *deviation* in estradiol predicting weekly PAI-BOR total score.

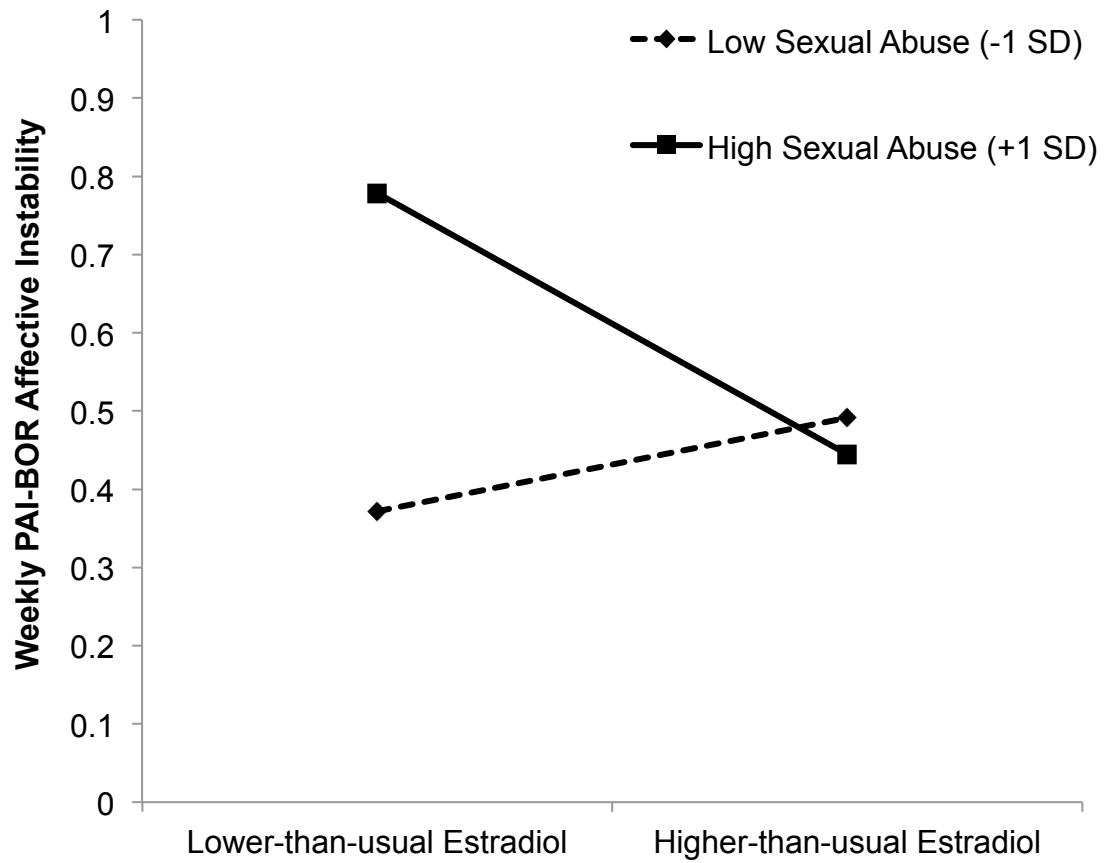


Figure 49. A graph of the interaction between self-reported levels of childhood sexual abuse and *deviation* in estradiol predicting weekly PAI-BOR Affective Instability subscale score.

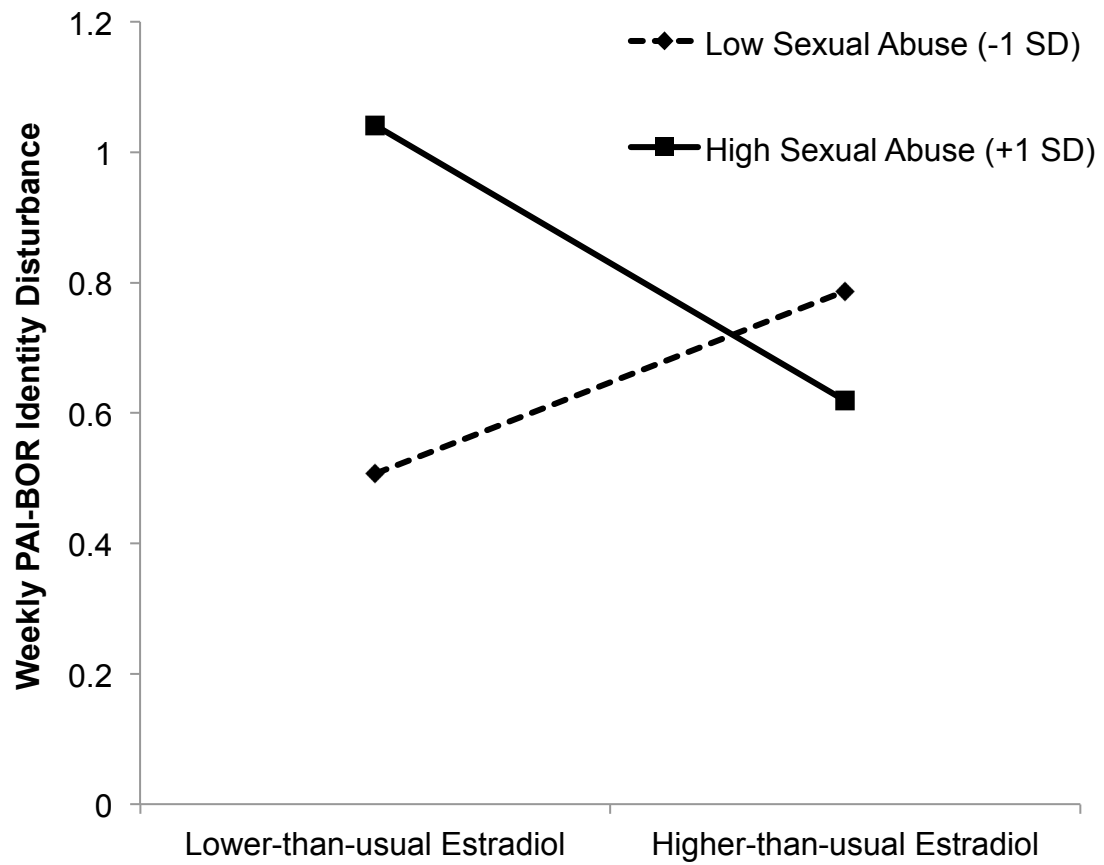


Figure 50. A graph of the interaction between self-reported levels of childhood sexual abuse and *deviation* in estradiol predicting weekly PAI-BOR Identity Disturbance subscale score.

associated with lower levels of BPD symptoms ($\gamma_{\text{HIGHSEXABUSE*ESTRADIOLDEVIATION}} = -.30$, $SE = .11$, $t(37) = -2.84$, $p = .005$), whereas among women reporting *lower* levels of Sexual Abuse, higher-than-usual levels of estradiol were not associated with BPD symptom expression ($\gamma_{\text{LOWSEXABUSE*ESTRADIOLDEVIATION}} = .01$, $SE = .09$, $t(37) = .16$, $p = .87$). A graph of this interaction can be found in Figure 51.

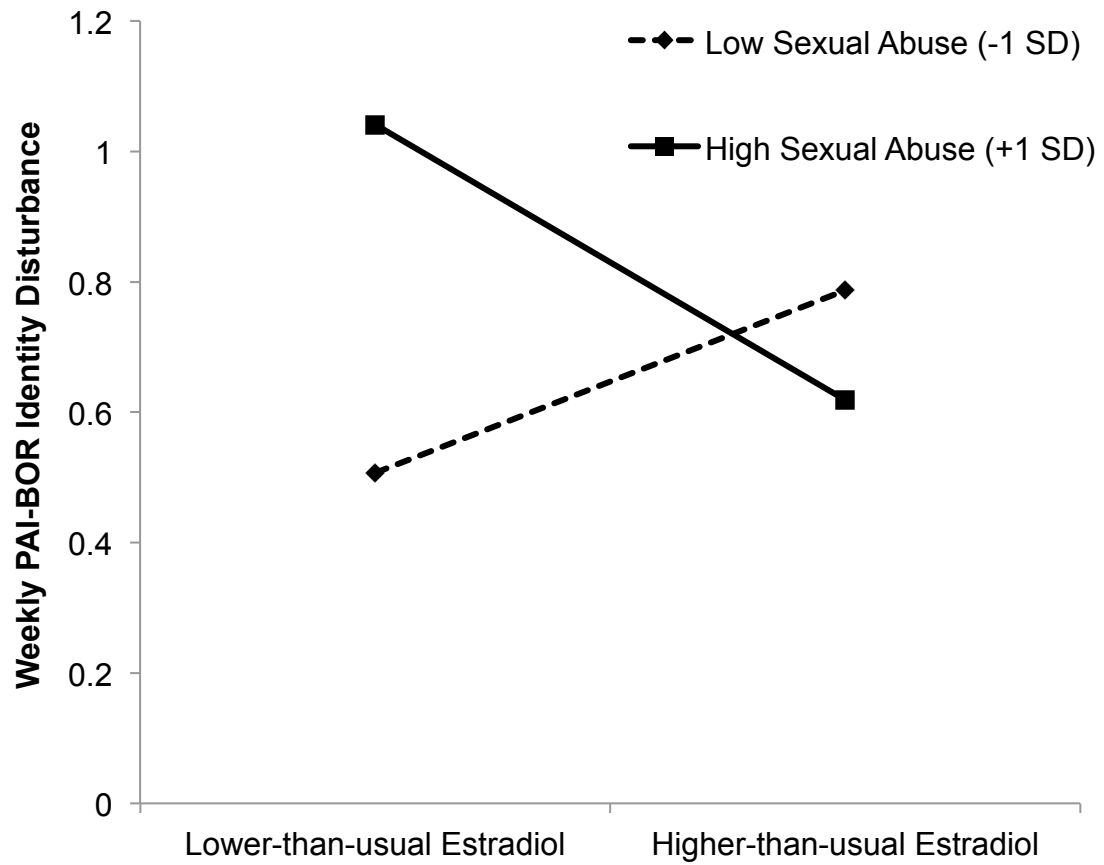


Figure 51. A graph of the interaction between self-reported levels of childhood sexual abuse and *deviation* in estradiol predicting weekly BSL-23 score.

Chapter Four: Discussion

Despite recent advancements in the understanding and treatment of individuals with Borderline Personality Disorder, very little work has addressed the physiological underpinnings of day-to-day and week-to-week variability in BPD symptom expression. Given the higher rates of diagnosis and impairment in women diagnosed with BPD (Grant et al., 2008), it has been suggested that the monthly reproductive cycle—and particularly changes in estradiol, a hormone that peaks naturally at ovulation—may play a role in modulating symptoms. The present study builds upon previous evidence that ovulation-related increases in estradiol are associated with greater emotional and behavioral symptoms of BPD, especially among women high in trait levels of BPD symptoms. The purpose of this project was to test a new cyclical vulnerability theory of BPD, which posits that women at risk for BPD experience exaggerated reductions in felt social acceptance at ovulation and consequent increases in BPD symptom expression at that time.

The cyclical vulnerability theory and its associated hypotheses were generally unsupported by the data. In fact, results of hypothesis tests actually support the opposite the pattern of ovulatory effects on BPD symptoms, in which individuals with high trait levels of affective and behavioral dysregulation report slightly lower levels of BPD symptoms when fertility and estradiol are higher, whereas women with low trait levels of these problems report slight increases in BPD symptoms when fertility and estradiol are higher. However, this pattern was not entirely consistent, and requires further exploration in subsequent studies. The results of specific hypothesis tests carried out for the present project are reviewed and discussed below, followed by discussions of potential

alternative theoretical explanations for these effects, how these effects compare to previously-established findings, and the ways in which the present study can be used to improve future work in this area.

Do Ovulation or Increases in Estradiol Predict Greater BPD Symptoms?

Hypothesis 1 predicted that all women would experience increases in symptoms of BPD at ovulation and when estradiol was relatively higher for them. Contrary to this prediction, there were no significant effects of conception probability on BPD symptoms at the daily level. At the weekly level, there was only one significant effect of within-person changes in estradiol, and it was in the opposite of the predicted direction; when women had higher-than-usual estradiol—likely at visits that were relatively closer to ovulation—they had *lower* scores on the Borderline Symptom Checklist (BSL-23), a measure of BPD symptoms based on the DSM-IV criteria for BPD. Zero-order correlations and significance tests suggested that the BSL-23 was more significantly related to a measure of actual diagnosis of BPD than the PAI-BOR or the MSI-BPD. In the present study, then, it appears that more extreme, clinically-relevant symptoms were more likely to lessen under conditions of higher-than-usual levels of estradiol.

Hypothesis 2 predicted that the effects of daily fertility and weekly fluctuations in estradiol would be moderated by trait levels of BPD such that women with higher levels of trait BPD would show exaggerated increases in BPD symptoms at higher levels of conception probability and higher-than-usual estradiol. Contrary to this prediction, there was no evidence for exaggerated symptom expression at higher fertility or higher-than-usual estradiol among women higher in trait BPD. Rather, there was some evidence for the opposite pattern. Women with *low* trait levels of BPD reported higher BPD symptoms

at higher levels of fertility and at higher-than-usual levels of estradiol, including higher scores on the daily and weekly total scales of the PAI-BOR. Women with *high* trait levels of BPD, on the other hand, reported symptom reduction at higher levels of fertility and higher-than-usual estradiol: lower scores on the weekly PAI-BOR total scale, the Affective Instability subscale, the Identity Disturbance subscale, and the BSL-23.

It would appear that, rather than becoming vulnerable at ovulation and during periods of increased estradiol, women higher in trait BPD actually experience periods of reduced symptoms and enhanced well-being. This also indicates, however, that women high in trait BPD may experience higher symptom expression at lower levels of fertility and estradiol—a different sort of cyclical vulnerability. Though the computation of effect sizes in multilevel Poisson regression is not straightforward, it is possible to discuss the practical implications of the estimates presented here in a couple of more practical ways. First, estimates in Poisson regression may be exponentiated to determine the percent change in the dependent variable that would be achieved with a one-unit increase in the predictor. In the present study, the average unstandardized estimate for the effect of deviations in estradiol on weekly BPD symptoms among women with high levels of trait BPD symptoms was $-.15$, which means that a one unit (i.e., 100%) increase above an individual's mean level of estradiol was associated with a 13.9% reduction in BPD symptoms among women high in trait BPD symptoms. Second, results can also be more clearly understood by discussing them in terms of change on the response scale. For example, an estimate of $-.15$ corresponds to a change in symptoms that is only about one-tenth the distance between “Sometimes True” and “False, Not True at All” on the

response scale for the PAI-BOR. So, although there does appear to be a cyclical effect on symptoms, the effects found here are relatively small.

Although clearly post-hoc, an evolutionary psychological interpretation of the present findings should be considered. Evolutionary psychologists have established that ovulation can have a profound effect on numerous psychological and behavioral processes of relevance to BPD symptoms. The effects found here appear to lend further support to the notion that affective and behavioral symptoms do change, however modestly, at the higher levels of fertility and higher-than-usual levels of estradiol that generally accompany ovulation. As mentioned earlier, higher fertility has been associated with increased implicit motivation to seek out social contact, and may be associated with an increased tendency toward agreeableness at that time so as to improve the chances of finding a mate (Schultheiss, Dargel, & Rhode, 2003). However, effects here seem to point toward a normalizing effect of ovulation on emotions and behavior, with women with high trait BPD and women with low trait BPD reporting slightly more “normal” (more average) levels of symptoms at ovulation and higher-than-usual estradiol. It is possible that these opposite effects indicate that higher fertility represents a cue for women to appear more emotionally and behaviorally stable (in the case of high-BPD women) or to appear more emotionally and behaviorally provocative and risk-taking (in the case of low-BPD women). Women who are better able to up- or down-regulate abnormally high or low levels of BPD-related traits at ovulation may appear healthier, more balanced, and hence more attractive to potential mates.

Do Ovulation or Increases in Estradiol Predict Lower Felt Acceptance?

Hypothesis 3a predicted that changes in felt acceptance would mediate the effect of fertility and higher-than-usual estradiol on BPD symptoms. Contrary to prediction, rather than feeling exaggerated decreases in felt acceptance at higher levels of fertility and higher-than-usual estradiol, women with higher levels of trait BPD showed *increases* in felt acceptance under these conditions. On the other hand, women with lower trait BPD symptoms reported feeling *less* social acceptance at ovulation, a finding that is consistent with previous work in this area demonstrating that normal women show decreases in self-esteem (Hill & Durante, 2009)—a construct intimately connected to feelings of social acceptance (Leary et al., 1998)—at ovulation. Overall, these findings suggest that, unlike normal women, women high in trait BPD may actually experience a boost in feelings of social acceptance at higher-than-usual levels of estradiol. Further, there was evidence that these differing effects of estradiol deviations on felt acceptance mediated the interactive effects of trait BPD and estradiol deviations on BPD features. That is, there is some evidence that women with higher levels of trait BPD evidenced fewer BPD symptoms at higher levels of estradiol because that higher estradiol was associated with feeling more socially accepted, which, in turn, was associated with lower BPD symptoms. It should also be noted that the effects of daily and weekly deviations in felt acceptance were, in every case, significant above and beyond mean levels of felt acceptance, highlighting the importance of within-person changes in feelings of belongingness and social wellbeing—especially for individuals with BPD.

It is possible that this effect can also be explained from an evolutionary perspective. These divergent effects of changes in estradiol on felt acceptance may be a

further example of a “normalizing” ovulatory effect that confers a reproductive benefit on both low-BPD and high-BPD women. In theory, it should be most reproductively beneficial to experience whatever social emotions at ovulation encourage socializing and mating. Individuals with BPD experience the world as an unsafe place in which people, including potential mates, cannot be trusted and in which social rejection is inevitable (Baer, Peters, Eisenlohr-Moul, Geiger, & Sauer, 2012). In order to increase social motivation at ovulation among *these* women, adjustments to the usual ovulatory pattern (of decreased felt acceptance and increased social motivation) may be necessary. Although normal women may, in some sense, require feelings of *lower* social acceptance at ovulation in order to be motivated to seek out greater social contact and potential mates, women with high trait BPD may require feelings of *higher* social acceptance at ovulation in order to feel safe enough to engage in the same social, mate-seeking behaviors.

Alternative, Proximal Mediators: Are Changes in Self-Control or Impulsivity Responsible for Cycle Effects?

Hypotheses 3b and 3c predicted that, among women higher in trait BPD, higher-than-usual levels of estradiol would be associated with decreased self-control and increased impulsivity, and that these changes would mediate the impact of changes in estradiol on BPD symptom expression. This hypothesis was also unsupported, and once again, significant effects were observed in the opposite of the predicted direction. Among women higher in trait BPD, higher-than-usual estradiol was associated with better self-control, lower Positive and Negative Urgency, lower Lack of Perseverance, and lower Lack of Premeditation. Among women low in trait BPD symptoms, none of these associations were significant. Insofar as poor self-control and elevated levels of these

types of impulsivity are consistent with BPD (de Ridder, Lensvelt-Mulders, Finkenauer, Stok, & Baumeister, 2012; Tragesser & Robinson, 2009), these findings are entirely consistent with the results presented above.

Further, both average levels of and relative changes in self-control and impulsivity significantly predicted BPD symptoms in expected directions in the full sample. This finding provides further validation of the presence and importance of self-control and impulsivity effects on BPD symptom expression, especially at the within-person level. Although average levels of self-control and impulsivity were associated with BPD symptoms, day-to-day fluctuations in these processes often had an equal or greater impact on daily symptoms. Further, mediational hypotheses confirmed that each of these variables at least partially mediated the interactive effect of Trait BPD and deviations in estradiol on BPD symptom expression.

Partially consistent with the existing evidence suggesting that Negative Urgency, Positive Urgency, and Lack of Premeditation are associated with BPD features (Tragesser & Robinson, 2009), the strongest and most consistent indirect effects were found for Negative Urgency, Lack of Premeditation, and Lack of Perseverance. Therefore, in the present study, ovulatory reductions in the tendency to respond to negative emotion with impulsive action, the tendency to act without thinking through the consequences of one's actions, and the tendency to have difficulty persisting in the face of difficulty were primarily responsible for estradiol-related changes in BPD symptom expression. It is possible that shifts in estradiol underlie changes in aspects of impulsivity or the capacity to regulate them, and such changes may account for the impact of the cycle on BPD symptoms.

Investigating Alternative Moderators of Cyclical and Hormonal Effects: Roles of FFM Personality and Childhood Maltreatment

Because BPD is a heterogeneous disorder, it may be questionable to rely on trait levels of BPD as a moderator of ovulatory or hormonal effects. It would be more preferable to identify moderators that capture central underlying traits or risk factors for BPD. Therefore, using two theoretical models of BPD (Five Factor Models of BPD and Linehan's biosocial theory) as my guide, I tested additional models in which five factor model domain scores and types of childhood maltreatment were substituted for Trait BPD in moderation models. These models provided some new insights into the reasons for differential effects of changes in estradiol on BPD symptom expression among women high and low in trait BPD symptoms.

Neuroticism, Estradiol, and BPD Symptoms. From the FFM perspective on personality disorders, the central characteristic of BPD is a high level of all aspects of Neuroticism (Widiger & Mullins-Sweatt, 2009). Therefore, I expected that Neuroticism would function as a moderator in similar ways to trait BPD. However, there were no significant interactions between Neuroticism and deviations in estradiol, suggesting that the moderating effect of trait BPD on deviations in estradiol is not due to extreme levels of Neuroticism, as might be expected. It is possible that some other, more specific characteristic of all or some of those with higher trait BPD—such as reactivity to sensations (Rosenthal, Ahn, & Geiger, 2011), rejection sensitivity (Ayduk et al., 2008), or a chronically elevated physiological stress response (e.g., Jogems-Kosterman, de Knijff, Kusters, & van Hoof, 2007)—is responsible.

Rather than modulating the effect of estradiol fluctuations, Neuroticism significantly altered the association between *trait* (or *average*) levels of estradiol and

BPD symptoms. Among women low in Neuroticism, higher average levels of estradiol buffered against BPD symptoms, predicting lower levels of affective instability, identity disturbance, and more extreme symptoms of BPD as measured on the BSL-23. Among women high in Neuroticism, however, higher average levels of estradiol were associated with higher levels of more extreme symptoms on the BSL-23. Therefore, it appears that Neuroticism may serve as a risk factor for negative reactions to chronically elevated estradiol, and low Neuroticism may allow women to benefit—at least in psychological terms—from chronically elevated estradiol. Notably, these between-person effects were generally about twice as large as the within-person effects found in the interactions between trait BPD and *deviations* in estradiol discussed earlier.

Openness to Experience, Estradiol, and BPD Symptoms. The FFM personality perspective also provides evidence that certain aspects of Openness to Experience—specifically, a tendency to be open to emotions and to actions—are high among individuals diagnosed with BPD. Like Neuroticism, Openness to Experience did not interact with deviations in estradiol to predict BPD symptoms, but did moderate the impact of *trait* levels of estradiol on BPD symptoms. Among women with higher Openness to Experience, higher trait levels of estradiol were associated with lower scores on the PAI-BOR total, the Affective Instability subscale, the Identity Disturbance subscale, the BSL-23, and the MSI-BPD. Among women low in Openness to Experience, there were smaller but significant effects of higher trait levels of estradiol predicting higher scores on each of these scales. When one inspects the graphs in Figures 20-24, it appears that high Openness to Experience actually serves as a risk factor for BPD

symptoms at low trait levels of estradiol—note that this in contrast to Neuroticism, which served as a risk factor at high levels of average estradiol.

Therefore, chronically low estradiol was associated with more symptoms among women with higher levels of Openness to Experience, though further work will be needed to elucidate the mechanisms of this effect. In one small cross-sectional study that tested all participants during menses, lower levels of estradiol were associated with higher self-reported risk taking (Balada, Torrubia, & Arqué, 1993). If this association is replicable, it is possible that higher Openness to Experience—and especially openness to actions—serves to disinhibit this tendency toward risk taking among women low in average estradiol. In any case, higher Openness to Experience has been established apart from Neuroticism as a fundamental FFM personality abnormality in BPD, and may therefore confer risk for BPD through much different pathways than Neuroticism.

Other FFM Personality Variables, Estradiol, and BPD Symptoms. Other aspects of FFM personality thought to be abnormal in BPD—including Agreeableness and Conscientiousness—did not interact with estradiol variables to predict symptoms in the present study. However, consistent with the FFM conceptualization, both Agreeableness and Conscientiousness showed negative zero-order correlations with average levels of BPD symptoms.

Sexual Abuse, Estradiol, and BPD Symptoms. Only one subscale of the Childhood Trauma Questionnaire, Sexual Abuse, moderated any effects of estradiol on BPD symptoms. The pattern in which Sexual Abuse moderated *average* levels of estradiol was similar to Neuroticism. Among women who reported greater experiences of having been sexually violated as a child, higher average levels of estradiol were

associated with higher scores on the PAI-BOR Self-Harm subscale as well as higher scores on the BSL-23. Each of these measures represents relatively extreme BPD symptoms, indicating that Sexual Abuse may prime women in some way to experience greater negative behavioral (i.e., externalizing) effects of chronically-elevated estradiol. It has been suggested (e.g., Figueroa & Silk, 1997) and demonstrated (Jogems-Kosterman, de Knijff, Kusters, & van Hoof, 2007) that sexual abuse and other childhood maltreatment is responsible for a chronically-activated physiological stress response (e.g., chronically elevated cortisol) in BPD. Such physiological changes secondary to childhood maltreatment may play a role in modulating the effects of average estradiol on BPD symptoms, and future studies should include measures of a chronic physiological stress activation. Crucially, these results are consistent with Linehan's discussion of the biosocial model of BPD; Linehan (1993) asserts that childhood Sexual Abuse, insofar as it blatantly ignores the wishes of the child, can be conceptualized as the most extreme form of the childhood invalidating environment theorized to play a central role in the development of BPD.

Several smaller interactions were found between greater reports of childhood sexual abuse and deviations in estradiol, though these within-person effects followed the opposite pattern. Among women reporting higher levels of childhood sexual abuse, higher-than-usual estradiol was associated with reductions in Affective Instability, Identity Disturbance, and the BSL-23, whereas women reporting low or no childhood sexual abuse had increases in symptoms at higher levels of estradiol. These effects are consistent with the interactive effects of trait BPD and deviations in estradiol, and may at least partially explain the moderating impact of trait BPD.

Having experienced sexual abuse in childhood may cause women to be more vigilant and use greater caution at times when high fertility primes sexuality. Such greater caution may, in some cases, translate into less BPD-related interpersonal and behavioral symptoms. However, these effects were smaller than the effects of average estradiol, indicating that the interaction of childhood sexual abuse and average levels of estradiol may have greater consequence for BPD symptom expression. These findings once again highlight that between-person effects (effects of average estradiol) and within-person effects (effects of estradiol deviations) may be quite different.

Contextualizing the Present Findings in the Extant Literature on the Cycle and BPD

In previous studies, ovulation-related variability and changes in estradiol were associated with both higher levels of and increases in BPD symptom expression (DeSoto et al., 2003). In the present study, I found evidence for such effects only inconsistently among those low in trait levels of BPD. There may be several reasons for these inconsistent findings.

First, unlike the DeSoto et al. (2003) study, the present was not designed to track women across specific points in the ovulatory cycle. Rather, it was designed to investigate the unique impact of naturally-occurring changes in estradiol on the expression of BPD symptoms across one ovulatory cycle. Only one of the DeSoto et al. (2003) studies examined the association of naturally-occurring variability in endogenous estradiol with BPD symptom expression, but it focused on between-person associations of such overall variability with trait levels of symptoms rather than within-person effects of week-to-week changes in estradiol. However, post-hoc correlations in the present study between an individual's overall variability in estradiol and measures of BPD

revealed no significant associations (all r 's less than .05, all p 's > .45). Therefore, not only were there no within-person effects consistent with those reported by DeSoto et al. (2003); the data from the present study also failed to replicate their primary reported effect.

Second, the present study focused on endogenous (naturally-occurring) rather than exogenous (synthetic) estrogen, whereas two of the three studies reported in DeSoto et al. (2003) reported a negative impact of hormonal contraceptives containing synthetic estrogen. This may be due to the fact that hormonal birth control and other exogenous forms of estrogen do differ in some ways from endogenous estradiol in their biochemical activity. Chronically elevated levels of estrogen due to taking hormonal contraceptives may have negative effects in some women similar to those found above, at least among women sensitive to higher levels of estrogen (see Kiesner, 2011).

There are a variety of physiological pathways through which chronically-elevated synthetic estrogen could exert negative effects in select women; however, one particularly plausible pathway is via elevated levels of C-reactive protein (CRP), a marker of inflammation, among women taking sustained doses of synthetic estrogens in the form of hormonal birth control (van Rooijen et al., 2006) and hormone replacement therapy (Eilertsen, Sandvik, Steinsvik, & Sandset, 2008). Several studies have provided evidence of a causal link between elevated levels of C-Reactive Protein to later depression and other symptoms of affective dysregulation (Gimeno et al., 2009; Matthews et al., 2010; Howren, Lamkin, & Suls, 2009). Given the centrality of poor affect regulation in BPD, it is possible that even mild increases in levels of inflammation mediate the negative affective consequences of chronically elevated synthetic estrogen

found in some women. While normal women may not experience negative effects of artificially elevated synthetic hormone, women with higher trait levels of BPD may experience negative effects such as those found by DeSoto et al. (2003) due to a chronically-activated stress response system that is unable to effectively downregulate inflammation via vagal inhibition (Thayer, 2009) or some other physiological pathway that is compromised during chronic stress.

Using Biosocial and Evolutionary Theories to Understand the Effects of the Cycle on BPD Symptoms

Though the original “estradiol peak” cyclical vulnerability model was not supported, an inverse “estradiol trough” cyclical vulnerability model was supported. Women higher in trait BPD reported greater symptoms when estradiol was lower-than-usual for them; these effects were mediated by a reduction in felt acceptance, and, more proximally, by changes in self-control and impulsivity. In an attempt to better understand the role of trait BPD in this model, alternative moderators were tested. Only Sexual Abuse mirrored the moderating role of trait BPD. Sexual Abuse, a social risk factor, interacted with cyclical changes to predict BPD symptom expression: among those reporting high levels of Sexual Abuse, lower-than-usual levels of estradiol (such as those found at menses or the premenstrual week) were associated with higher BPD symptom expression. This finding provides support for the idea that Linehan’s (1993) biosocial model may operate not only at a trait level, but at a state level, as well.

There are several potential physiological mechanisms through which *state* physiological predisposition to emotion dysregulation may occur at lower-than-usual levels of estradiol. First, recent evidence indicates that natural fluctuations (higher-than-

usual levels) of estradiol are associated with improvements in working memory (Segal, 2012), which may play a role in the ability to modulate negative emotion, feelings of rejection, or related urges (Schmeichel, Volokhov, & Demaree, 2008), premeditate the consequences of behavior (Bechara & Martin, 2004), or persist on goal-relevant tasks (McVay & Kane, 2009). This interpretation may be particularly relevant to the mediation of cyclical vulnerability effects by self-control and impulsivity. Second, despite evidence that long-term administration of exogenous estrogen elevates CRP (a marker of inflammation; see above), there is evidence that fluctuations in endogenous estrogen follow the opposite pattern, with higher-than-usual levels of estradiol predicting reductions in CRP (Blum et al., 2005). As noted previously, such inflammatory markers prospectively predict depressive symptoms, which may explain greater symptoms at lower-than-usual estradiol (Gimeno et al., 2009; Matthews et al., 2010; Howren, Lamkin, & Suls, 2009). However, it has yet to be established whether these quicker day-to-day and week-to-week changes in inflammation map onto changes in psychological symptoms.

Finally, within-person effects of estradiol may actually be due to the parallel variation of estradiol and oxytocin across the cycle. Oxytocin, a hormone involved in social cognition, affiliative behavior, and attachment, is estradiol-dependent throughout the cycle, and peaks naturally the day after ovulation (i.e., the day after luteinizing hormone surge; Shukovski, Healy, & Findlay, 1989). Furthermore, there is some evidence that trait oxytocin levels are abnormally low among women with BPD and women with a history of childhood maltreatment or trauma (Bertsch, Schmidinger, Neumann, & Herpertz, 2013), and that administration of oxytocin attenuates emotional

reactivity in BPD (Simeon et al., 2011; although cf. Bartz et al., 2011). It is possible, then, that the cyclical effect of estradiol on BPD symptoms may exist only due to oxytocin's potentially ameliorative effects on BPD symptoms.

From an evolutionary perspective, it would be reasonable to expect that such physiological changes that confer cyclical vulnerability to BPD symptoms among at-risk women would occur at points in the cycle when signaling reproductive fitness to potential mates and selecting a reproductively fit mate was less important. During the periods of low estradiol associated with the premenstrual and menstrual weeks, fertility is also low, so it may be less important for women at risk for BPD symptoms to (1) present themselves as normal and psychologically healthy and (2) have optimized cognitive capacity so as to inhibit impulses to mate with less-than-ideal mates and to seek out ideal mates. During periods of high fertility, however, increased estradiol may modulate the ability of at-risk women to accurately perceive social acceptance, to respond in helpful ways to emotion, think through behavior, and to persist in goal-related action. In addition to improving the experience of at-risk women, these positive ovulatory changes may serve to signal reproductive fitness to potential mates *and* to increase a woman's ability to carefully select a mate that is higher in reproductive fitness.

Using FFM and Biosocial Theories of BPD to Understand the Effects of Average Levels of Estradiol on BPD Symptoms

Unlike the effects of estradiol fluctuations, the effects of a woman's average levels of estradiol were moderated by all factors associated with risk for higher BPD symptoms, and the effects followed a pattern that was different from the within-person effects. Between women, higher average levels of estradiol was associated with greater

risk among those who reported risk factors for BPD such as Neuroticism or childhood sexual abuse, and low average estradiol was associated with greater risk among those high in Openness to Experience.

In the modified biosocial model presented and supported above, *trait* risk variables such as trait BPD or Sexual Abuse interact with *state* physiological variables to predict fluctuations in symptoms. However, in Linehan's (1993) classic biosocial model of BPD, childhood maltreatment such as Sexual Abuse interacts with trait physiological variables (those conferring risk for emotional dysregulation) to predict trait BPD symptoms. The findings that average levels of estradiol interacts with such risk factors as trait BPD, Sexual Abuse, and even FFM Neuroticism and Openness are probably best understood using Linehan's classic, trait-level biosocial framework in which psychosocial and personality factors interact with physiological dysregulation to produce trait BPD symptoms. In the case of three out of four of these risk-related moderators, higher scores on a measure of risk (trait BPD, Sexual Abuse, or Neuroticism) interacted synergistically with higher average estradiol to predict higher trait BPD symptoms.

In order to provide further illustration of the association between average levels of estradiol and BPD symptoms at any given assessment, two additional figures are provided. Figure 52 is a descriptive scatterplot of the (nonsignificant) association between average levels of estradiol and weekly BPD scores in the full sample. Figure 53 is a descriptive scatterplot of the association between average levels of estradiol and weekly BPD scores graphed according to the individual's self-reported trait BPD symptoms (as measured by the original trait version of the PAI-BOR) at recruitment. Upon inspection of the these figures, it seems clear that women in this sample with high

levels of trait BPD symptoms show a stronger, positive association between average levels of estradiol and BPD symptoms on any given week than women with low levels of trait BPD symptoms. Indeed, among women with moderate-to-low trait BPD symptoms, it would appear that the association between average levels of estradiol and weekly BPD symptoms is either nonsignificant or negative. While descriptive only, this apparent pattern should motivate additional research on the role of average levels of estradiol in BPD.

In what way might high levels of average estradiol represent a trait physiological risk factor for emotion dysregulation consistent with the biosocial model? There are several possibilities. First, in the present study, higher trait levels of estradiol were strongly associated with lower FFM Extraversion—particularly low warmth and low positive emotions (r 's ranging from $-.30$ to $-.36$). Although low Extraversion is not hypothesized to be central to BPD in the FFM dimensional model of BPD, low positive emotion and low warmth are nevertheless consistent with the dysregulated emotion and low Agreeableness found in BPD. Assuming these associations are replicable, high *average* levels of estradiol may serve as a physiological risk factor for low warmth and positive emotion that interacts with other BPD risk factors to predict trait BPD symptoms.

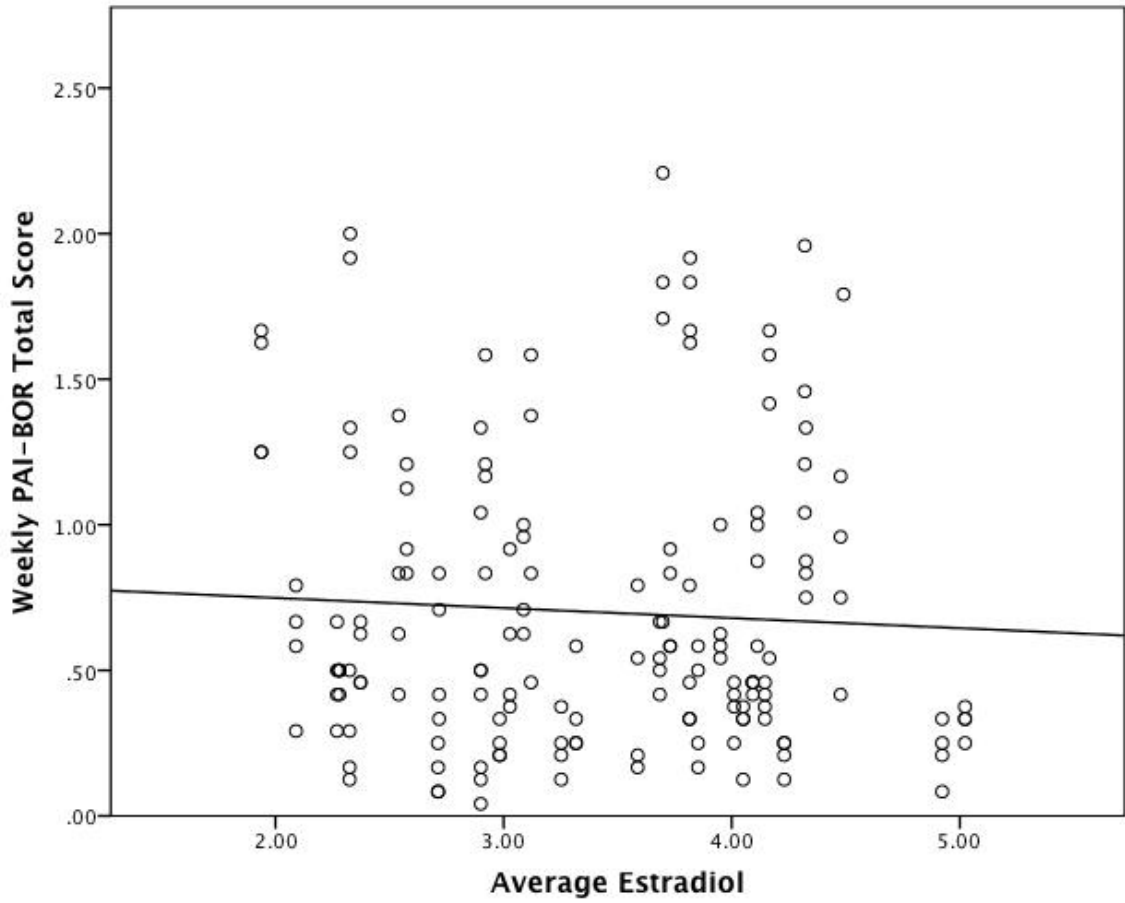


Figure 52. Descriptive scatterplot of the (nonsignificant) association between an individual’s average levels of estradiol across four weeks and their weekly PAI-BOR scores in the full sample.

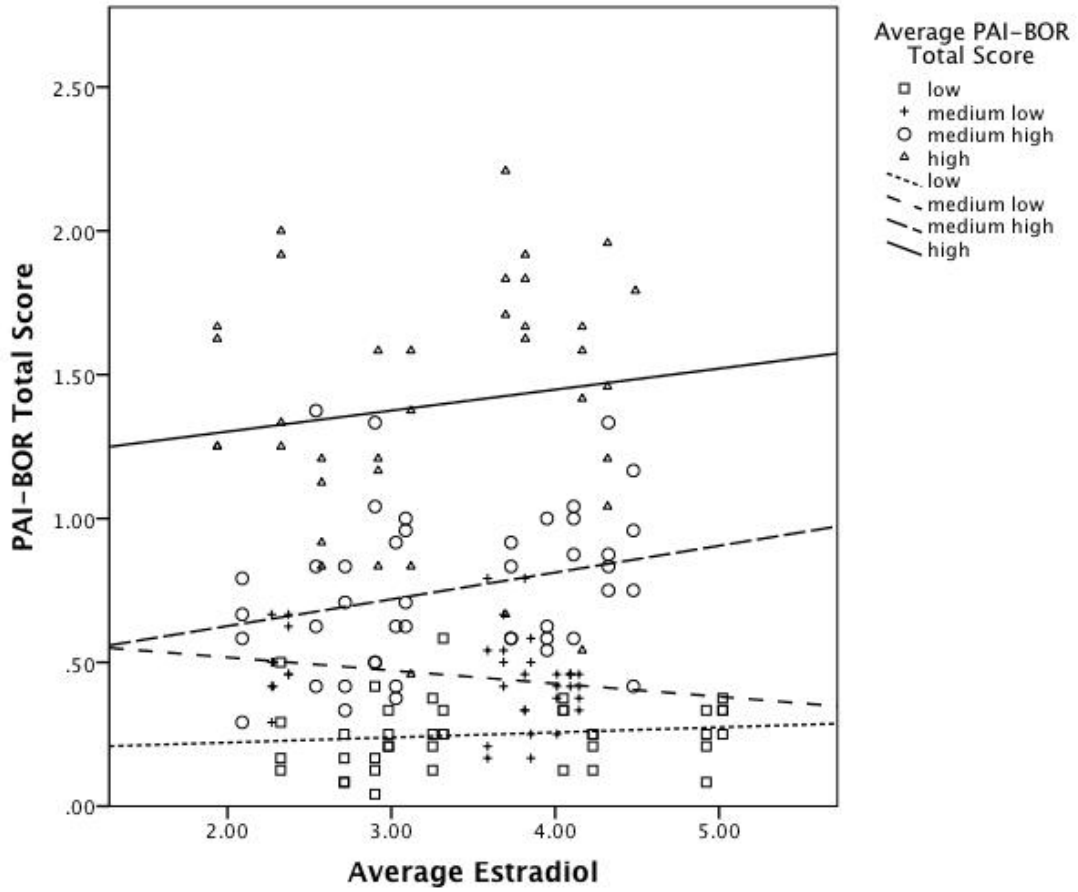


Figure 53. Descriptive scatterplot of the association between an individual’s average levels of estradiol across four weeks and weekly PAI-BOR scores graphed by quartiles of average PAI-BOR total score.

Other studies have found higher trait levels of estradiol among women high in alexithymia (a condition related to emotional awareness and expression) vs. controls (Ushiroyama, Ueki, Orino, & Ikeda, 1994), and higher trait levels of estradiol were associated with greater self-reported loneliness among a group of adolescent girls (Fujisawa, Nishitani, Obara, & Shinohara, 2012). These findings provide further evidence that *average* estradiol may be associated with other psychosocial risk factors for BPD-related symptoms (i.e., low Extraversion, high loneliness or alexithymia) that, in turn, interact with trait BPD, Neuroticism, or Sexual Abuse to predict higher trait symptoms. Notably, these effects are consistent with the evidence that chronic administration of exogenous estrogen in the context of hormonal contraceptives confer risk for BPD among women high in trait BPD (De Soto et al., 2003). The physiological mechanisms of these effects have not been studied; however, they may be similar to the physiological mechanisms through which chronic exogenous estrogen administration increases BPD symptom risk.

In the case of high Openness to Experience (which, from the FFM perspective, serves as a risk factor for BPD), it was *low* levels of average estradiol that were associated with higher BPD symptoms. Due to the divergent effects from other risk-related moderators, one can only assume that high levels of Openness to Experience exert moderating effects on BPD symptoms through different physiological pathways than trait BPD, Neuroticism, or Sexual Abuse. As mentioned previously, there is one study suggesting that risk taking is higher among women with low trait levels of estradiol (Balada, Torrubia, & Arqu e, 1993). It is possible that high Openness to Experience—and especially Openness to Actions, which is known to be high in BPD—serve to unmask

higher levels of risk-taking behavior among women low in estradiol. However, this is but one of many potential explanations for this finding, and a clear understanding of these results awaits further research.

Limitations and General Recommendations for Future Work in this Area

The project presented here is not without flaws, and the insights gained here may be used to refine the methodology of future projects on this topic. Perhaps most importantly, it is likely that the non-clinical sample used here created a floor effect in which the range of daily and weekly BPD symptoms was restricted (i.e., to the lower range). Although the sample was selected to create a flat distribution of BPD symptoms, the distributions for trait BPD were still relatively positively skewed, and only four of the individuals in the study met SCID-II criteria for BPD. Future studies will include a larger number of women overall, with a greater number of women meeting diagnostic criteria for BPD.

Another limitation concerns the definition and measurement of the ovulatory cycle. In the present study, the focus was relatively limited to ovulatory and estradiol effects. In the future, it would be appropriate to examine other hormones (e.g., progesterone) that can provide more information about hormonal changes in symptoms occurring at other points in the cycle (e.g., the luteal phase). Although the reliability of luteal-phase effects on mood-related issues (i.e., Premenstrual Syndrome or PMS) has recently been seriously questioned (Romans et al., 2012), it is possible that such effects are more robust among individuals with BPD, and that non-ovulatory cycle effects are important for understanding cycle-related variability in BPD symptom expression. A related point, mentioned previously, is that higher-than-usual levels of estradiol in the

current study may covary with some third hormonal variable (e.g., luteinizing hormone or oxytocin) that exerts more powerful ovulatory effects on BPD symptoms. In future studies, alternative ways of defining and measuring the cycle should be explored.

It is also possible that low statistical power played a role in the failure to identify significant small interactive effects at the weekly level. Therefore, the failure of some small effects presented in the current study to reach significance should not necessarily be interpreted as evidence for nonsignificance; larger sample sizes may be necessary to detect small interactive effects of trait variables with deviations in estradiol on BPD symptoms. On the other hand, several small interactive effects were detected, suggesting that these effects are reasonably robust and that low statistical power may not be responsible for the failure of some small interactive effects to reach significance.

Finally, the present study could have benefitted from the inclusion of more concrete measures of basic underlying traits and processes. The study failed to include measures of some core trait constructs that may have served as clearer, more powerful moderators of the effects of estradiol. Chronic physiological stress activation such as that found in post-traumatic stress, sensitivity to rejection or sensations, or some other risk variable that is associated with trait BPD may be truly responsible for the interactive effects seen here. Future studies should seek to pinpoint and measure such key constructs. The study also relied on daily and weekly self-report measures of BPD symptoms using heterogeneous symptom inventories; in the future, studies may be strengthened by the inclusion of more concrete measures of behavior, such as substance use, aggression, or observer reports of interpersonal functioning.

Clinical Implications and Directions for Future Clinical Research

The small effect sizes found here may indicate that cyclical vulnerability does not exert clinically meaningful effects on BPD. However, future studies should explore the possibility that the synergistic effects of the cycle with other daily and weekly variables may be more significant in the lives of women who suffer from diagnosable levels of BPD. Small cycle-related changes symptoms may become more significant in the face of the more serious stressors that often plague the lives of women with BPD. In the context of ongoing BPD-related stressors such as unemployment, physical disability, substance use or abuse, or even simply ongoing interpersonal stress, small cyclical changes in emotional or behavioral vulnerability (i.e., at low levels of estradiol and fertility) may have larger, more serious implications. Further, trait levels of estradiol may also interact with either acute or chronic stressors in the lives of women with BPD to predict symptoms. As mentioned previously, the present study used a sample of generally healthy undergraduate women, and though they were sampled to achieve higher sample levels of BPD than would be found in the general population, only four of the participants met SCID-II criteria for BPD. Therefore, it is crucial that future studies examine whether the effects found here generalize to clinical populations.

The most appropriate next step in this line of research would be another, larger prospective study aimed at replicating and extending the present findings to women with a diagnosis of BPD. Ideally, such a study would recruit groups of women clinically diagnosed with BPD, clinically diagnosed with an Axis I disorder such as Major Depressive Disorder, and women with no diagnosis. Inclusion of the Axis I group would allow for a determination of whether increases in fertility and estradiol benefit women

with risk for psychopathology in general or only women with a pattern of symptoms consistent with BPD. This study should also include progesterone, oxytocin, and other ways of measuring the cycle, and should aim to determine whether it is truly low fertility and estradiol that are the key cycle variables responsible for the effects observed here. A study that included more information about the individual's trait and weekly hormonal levels as well as a sample that provides a less restricted range of symptoms may prove elucidating.

If the effects observed here were found to be clinically significant in a larger study, the eventual goal of this program of research would be to conduct a randomized controlled trial of an adjunctive intervention (i.e., an addition to a broader empirically-supported treatment such as Dialectical Behavior Therapy) for women with BPD. Such an intervention would be carried out in the context of individual therapy. Daily symptom tracking sheets, which are already a standard part of Dialectical Behavior Therapy, would be modified to include information about cycle day and menses. After the individual had tracked emotional and behavioral symptoms across two cycles, the therapist would aggregate the data and determine whether a cycle or phase-related pattern had emerged. If so, the therapist would explain the results of this assessment to the client, and would encourage the client to focus the use of skills such as those learned in DBT on the problematic times of the month. Several different types of skills may be relevant to compensating for symptom vulnerability at certain times of the month, including the ability to exercise nonjudgmental present-centered awareness of physical symptoms or emotional lability (i.e., DBT mindfulness skills), the ability to label, understand, and respond effectively to emotions as they arise (i.e., DBT emotion regulation skills), the

ability to tolerate distress without acting on impulses (i.e., DBT distress tolerance skills), and the ability to interact with others in useful ways even in the presence of strong emotion (i.e., DBT interpersonal effectiveness skills). Such a study would seek to determine whether focusing the use of skills on times of the month that are generally problematic for the client might boost the effectiveness of the intervention.

Conclusions

The present study provides some interesting preliminary evidence about the role of fertility and estradiol in predicting BPD symptom expression. A great deal of additional work is needed to clarify the role of the cycle and related hormones in BPD symptom expression, to tease apart between-person and within-person effects of hormones, to clarify the key BPD-related traits responsible for moderated cycle effects, and to better understand the physiological and psychological mechanisms through which the cycle exerts its influence. If clear mechanisms of naturalistic change across the cycle can be established, medical, psychiatric, and psychotherapeutic attempts to improve functioning in BPD can become more timely, focused, and effective, reducing the considerable burden of this debilitating disorder.

References

- American Psychiatric Association (1994). *Diagnostic and statistical manual of mental disorders*, 4th ed. American Psychiatric Association, Washington, DC.
- Ayduk, Ö., Zayas, V., Downey, G., Cole, A. B., Shoda, Y., & Mischel, W. (2008). Rejection sensitivity and executive control: Joint predictors of borderline personality features. *Journal of Research in Personality*, *42*(1), 151-168.
- Baer, R., Peters, J., Eisenlohr-Moul, T. Geiger, P., & Sauer, S. (2012). Emotion-related cognitive processes in borderline personality disorder: A review of the empirical literature. *Clinical Psychology Review*, *32*(5), 359-369.
- Bardenstein, K.A., & McGlashen, T.H. (1988). The natural course of a residentially treated borderline sample: gender differences. *Journal of Personality Disorders*, *2*(1), 69–83.
- Bartz, J., Simeon, D., Hamilton, H., Kim, S., Crystal, S., Braun, A., & Hollander, E. (2011). Oxytocin can hinder trust and cooperation in borderline personality disorder. *Social Cognitive and Affective Neuroscience*, *6*(5), 556-563.
- Bechara, A., & Martin, E. (2004). Impaired decision making related to working memory deficits in substance addicts. *Neuropsychology*, *18*, 152-162.
- Bernstein, D. P., Stein, J. A., Newcomb, M. D., Walker, E., Pogge, D., Ahluvalia, T., & Zule, W. (2003). Development and validation of a brief screening version of the Childhood Trauma Questionnaire. *Child Abuse & Neglect*, *27*(2), 169-190.
- Bertsch, K., Schmidinger, I., Neumann, I. D., & Herpertz, S. C. (in press). Reduced plasma oxytocin levels in female patients with borderline personality disorder. *Hormones and Behavior*, doi: 10.1016/j.yhbeh.2012.11.013

- Blum, C. A., Müller, B., Huber, P., Kraenzlin, M., Schindler, C., De Geyter, C., & Puder, J. J. (2005). Low-grade inflammation and estimates of insulin resistance during the menstrual cycle in lean and overweight women. *Journal of Clinical Endocrinology & Metabolism*, *90*(6), 3230-3235.
- Bohus, M., Limberger, M. F., Frank, U., Chapman, A. L., Kuhler, T., & Stieglitz, R. (2007). *Psychopathology*, *40*, 126-132.
- Carver, C. S., Johnson, S. L., & Joormann, J. (2008). Serotonergic function, two-mode models of self-regulation, and vulnerability to depression: what depression has in common with impulsive aggression. *Psychological bulletin*, *134*(6), 912.
- Clarkin, J. F., Levy, K. N., Lenzenweger, M. F., & Kernberg, O. F. (2007). Evaluating three treatments for borderline personality disorder: A multiwave study. *American Journal of Psychiatry*, *164*, 922-928.
- Costa, P. T., & McCrae, R. R. (1992). Normal personality assessment in clinical practice: The NEO Personality Inventory. *Psychological Assessment*, *4*(1), 5.
- Cyders, M. A., & Smith, G. T. (2008). Emotion-based dispositions to rash action: positive and negative urgency. *Psychological Bulletin*, *134*(6), 807.
- Derefinko, K., DeWall, C. N., Metze, A. V., Walsh, E. C., & Lynam, D. R. (2011). Do different facets of impulsivity predict different types of aggression? *Aggressive Behavior*, *37*(3), 223-233.
- de Ridder, D. T., Lensvelt-Mulders, G., Finkenauer, C., Stok, F. M., & Baumeister, R. F. (2012). Taking stock of self-control: A meta-analysis of how trait self-control relates to a wide range of behaviors. *Personality and Social Psychology Review*, *16*(1), 76-99.

- DeSoto, M.C. Geary, D.C., Hoard, M.K., Sheldon, M. & Cooper, M. L. (2003). Estrogen variation, oral contraceptives, and borderline personality. *Psychoneuroendocrinology*, 28, 751-766.
- Duckworth, A. L., & Kern, M. L. (2011). A meta-analysis of the convergent validity of self-control measures. *Journal of Research in Personality*, 45(3), 259-268.
- Eilertsen, A. L., Sandvik, L., Steinsvik, B., & Sandset, P. M. (2008). Differential impact of conventional-dose and low-dose postmenopausal hormone therapy, tibolone and raloxifene on C-reactive protein and other inflammatory markers. *Journal of Thrombosis and Haemostasis*, 6(6), 928-934.
- Eisenlohr-Moul, T., Pond, R., DeWall, C. N, Lambert, N., Finkel, E., & Fincham, F. (under review). Ovulation, felt acceptance, and aggression.
- Ellis, A., Abrams, M., & Abrams, L. (2008). *Personality theories: Critical perspectives*. New York: SAGE Publications, Incorporated.
- Evardone, M., Alexander, G. M., & Morey, L. C. (2008). Hormones and borderline personality features. *Personality and Individual Differences*, 44, 278-287.
- Figuroa, E., & Silk, K. R. (1997). Biological implications of childhood sexual abuse in borderline personality disorder. *Journal of Personality Disorders*, 11(1), 71-92.
- First, M. B., & Gibbon, M. (1997). *User's guide for the structured clinical interview for DSM-IV axis II personality disorders: SCID-II*. Arlington, VA: American Psychiatric Publications.
- Fischer, S., Anderson, K. G., & Smith, G. T. (2004). Coping with distress by eating or drinking: role of trait urgency and expectancies. *Psychology of Addictive Behaviors*, 18(3), 269.

- Fujisawa, T. X., Nishitani, S., Obara, T., & Shinohara, K. (2011). Loneliness depends on salivary estradiol levels in adolescent females. *Neuroendocrinology Letters*, *33*(5), 525-529.
- Gardner, K., & Qualter, P. (2009). Reliability and validity of three screening measures of borderline personality disorder in a nonclinical population. *Personality and Individual Differences*, *46*(5), 636-641.
- Gimeno, D., Kivimaki, M., Brunner E. J., Elovainio, M., De Vogli, R., Steptoe, A., Kumari, M., Lowe, G. D. O., Rumley, A., Marmot, M., & Ferrie, J. E. Associations of C-reactive protein and interleukin-6 with cognitive symptoms of depression: 12-year follow-up of the Whitehall II study. *Psychological Medicine*, *39*, 413–23.
- Grant, B. F., Chou, S. P., Goldstein, R. B., Huang, B., Stinson, F. S., Saha, T. D., Smith, S. M., Dawson, D. A., Pulay, A. J., Pickering, R. P., & Ruan, J. R. (2008). *Journal of Clinical Psychiatry*, *69*(4), 533-545.
- Gruenewald, T. L., Kemeny, M. E., Aziz, N., & Fahey, J. L. (2004). Acute threat to the social self: Shame, social self-esteem, and cortisol activity. *Psychosomatic Medicine*, *66*(6), 915-924.
- Heatherton, T. F. & Polivy, J. (1991). Development and validation of a scale for measuring state self-esteem. *Journal of Personality and Social Psychology*, *60*, 895-910.
- Hill, S. E., & Durante, K. M. (2009). Do women feel worse to look their best? Testing the relationship between self-esteem and fertility status across the menstrual cycle. *Personality and Social Psychology Bulletin*, *35*(12), 1592-1601.

- Hofmann, W., Friese, M., & Strack, F. (2009). Impulse and self-control from a dual-systems perspective. *Perspectives on Psychological Science, 4*(2), 162-176.
- Howren, M. B., Lamkin, D. M., & Suls, J. (2009). Associations of depression with C-reactive protein, IL-1, and IL-6: a meta-analysis. *Psychosomatic Medicine, 71*(2), 171-186.
- Jogems-Kosterman, B. J., de Knijff, D. W., Kusters, R., & van Hoof, J. J. (2007). Basal cortisol and DHEA levels in women with borderline personality disorder. *Journal of Psychiatric Research, 41*(12), 1019-1026.
- Kiesner, J. (2011). One woman's low is another woman's high: Paradoxical effects of the menstrual cycle. *Psychoneuroendocrinology, 36*, 68-76.
- Leary, M.R. (2004). *The curse of the self: Self-awareness, egoism, and the quality of human life*. New York: Oxford University Press.
- Leary, M. R., Haupt, A. L., Strausser, K. S., & Chokel, J. T. (1998). Calibrating the sociometer: The relationship between interpersonal appraisals and the state self-esteem. *Journal of Personality and Social Psychology, 74*(5), 1290-1299.
- Lee, A. H., Wang, K., Scott, J. A., Yau, K. K., & McLachlan, G. J. (2006). Multi-level zero-inflated Poisson regression modelling of correlated count data with excess zeros. *Statistical Methods in Medical Research, 15*(1), 47-61.
- Lenzenweger, M. F., Lane, M. C., Loranger, A. W., & Kessler, R. C. (2007). DSM-IV personality disorders in the National Comorbidity Survey Replication. *Biological Psychiatry, 62*(6), 553-564.
- Linehan, M. (1993). *Cognitive-behavioral treatment of borderline personality disorder*. New York: The Guilford Press.

- Marteau, T. M., & Bekker, H. (2011). The development of a six-item short-form of the state scale of the Spielberger State—Trait Anxiety Inventory (STAI). *British Journal of Clinical Psychology, 31*(3), 301-306.
- Matthews, K. A., Schott, L. L., Bromberger, J. T., Cyranowski, J. M., Everson-Rose, S. A., & Sowers, M. F. (2010) Are there bi-directional associations between depressive symptoms and C-reactive protein in mid-life women? *Brain, Behavior, and Immunity, 24*, 96 –101.
- Mckinley, P. S., King, A. R., Shapiro, P. A., Slavov, I., Fang, Y., Chen, I. S., & Sloan, R. P. (2009). The impact of menstrual cycle phase on cardiac autonomic regulation. *Psychophysiology, 46*(4), 904-911.
- McVay, J. C., & Kane, M. J. (2009). Conducting the train of thought: Working memory capacity, goal neglect, and mind wandering in an executive-control task. *Journal of Experimental Psychology: Learning, Memory, and Cognition, 35*(1), 196.
- Morey, L.C., 1991. The personality assessment inventory: professional manual. Psychological Assessment Resources, Odessa, FL.
- Mullins-Sweatt, S. N., Jamerson, J. E., Samuel, D. B., Olson, D. R., & Widiger, T. A. (2006). Psychometric properties of an abbreviated instrument of the five-factor model, *Assessment, 13*(2), 119-137.
- Radloff, L.S. (1977). The CES-D scale: A self-report depression scale for research in the general population. *Applied Psychological Measurement, 1*, 385-401.

- Romans, S., Clarkson, R., Einstein, G., Petrovic, M., & Stewart, D. (2012). Mood and the Menstrual Cycle: A Review of Prospective Data Studies. *Gender Medicine, 9*(5), 361-384.
- Rosenthal, M. Z., Ahn, R., & Geiger, P. J. (2011). Reactivity to Sensations in Borderline Personality Disorder: A Preliminary Study. *Journal of Personality Disorders, 25*(5), 715-721.
- Russell, J., Moskowitz, D. S., Zuroff, D. C., Sookman, D., & Paris, J. (2007). Stability and variability of affective experience and interpersonal behavior in Borderline Personality Disorder. *Journal of Abnormal Psychology, 116*, 578-588.
- Sampson, R. J., & Laub, J. H. (1990). Crime and deviance over the life course: The salience of adult social bonds. *American Sociological Review, 60*–627.
- Schmeichel, B. J., Volokhov, R. N., & Demaree, H. A. (2008). Working memory capacity and the self-regulation of emotional expression and experience. *Journal of Personality and Social Psychology, 95*(6), 1526-1540.
- Schultheiss, O.C., Dargel, A., & Rhode, W. (2003). Implicit motives and gonadal steroid hormones: Effects of menstrual cycle phase, oral contraceptive use, and relationship status. *Hormones and Behavior, 43*, 293-301.
- Shukovski, L., Healy, D. L., & Findlay, J. K. (1989). Circulating immunoreactive oxytocin during the human menstrual cycle comes from the pituitary and is estradiol dependent. *Journal of Clinical Endocrinology & Metabolism, 68*(2), 455-460.

- Simeon, D., Bartz, J., Hamilton, H., Crystal, S., Braun, A., Ketay, S., & Hollander, E. (2011). Oxytocin administration attenuates stress reactivity in borderline personality disorder: a pilot study. *Psychoneuroendocrinology*, *36*(9), 1418-1421.
- Skodol, A. E., Gunderson, J. G., McGlashan, T. H., Dyck, I. R., Stout, R. L., Bender, D. S., Grilo, C. M., Shea, M. T., Zanarini, M. C., Morey, L. C., Sanislow, C. A., & Oldham, J. M. (2002). Functional impairments in patients with schizotypal, borderline, avoidant, or obsessive-compulsive personality disorder. *The American Journal of Psychiatry*, *159*(2), 276-283.
- Snijders, T., & Bosker, R. (1999). *Multilevel Analysis: An Introduction to Basic and Applied Multilevel Analysis*. Thousand Oaks, CA: Sage.
- Spielberger, C. D., Gorsuch, R. L., & Lushene, R. E. (1970). *Manual for the State-Trait Anxiety Inventory*. Palo Alto, CA: Consulting Psychologists Press.
- Stein, M. B., Pinsker-Aspen, J. H., & Hilsenroth, M. J. (2007). Borderline pathology and the personality assessment inventory (PAI): An evaluation of criterion and concurrent validity. *Journal of Personality Assessment*, *88*(1), 81-89.
- Stone, M. H., 1992. *Borderline personality disorder: course of illness*. In: Clarkin, J.F., Marziali, E., Munroe-Blum, H. (Eds.), *Borderline Personality Disorder: clinical and empirical perspectives*. The Guilford Press, New York, pp. 67–86.
- Swartz, M., Blazer, D., George, L., & Winfield, I. (1990). Estimating the prevalence of borderline personality disorder in the community. *Journal of Personality Disorders*, *4*(3), 257-272.

- Tangney, J. P., Baumeister, R. F., & Boone, A. L. (2004). High self-control predicts good adjustment, less pathology, better grades, and interpersonal success. *Journal of Personality, 72*, 271-322.
- Thayer, J. F. (2009). Vagal tone and the inflammatory reflex. *Cleveland Clinic Journal of Medicine, 76*(2), S23-S26.
- Thompson, E. R. (2007). Development and validation of an internationally reliable short-form of the positive and negative affect schedule (PANAS). *Journal of Cross-Cultural Psychology, 38*(2): 227-242.
- Tofghi, D., & MacKinnon, D. P. (2011). RMediation: An R package for mediation analysis confidence intervals. *Behavior Research Methods, 43*, 692-700.
- Tolpin, L. H., Gunthert, K. C., Cohen, L. H., & O'Neill, S. C. (2004). Borderline personality features and instability of daily negative affect and self-esteem. *Journal of Personality, 72*(1), 111-137.
- Tragesser, S. L., & Robinson, R. J. (2009). The role of affective instability and UPPS impulsivity in borderline personality disorder features. *Journal of Personality Disorders, 23*(4), 370-383.
- Twenge, J. M., Catanese, K. R., & Baumeister, R. F. (2002). Social exclusion causes self-defeating behavior. *Journal of Personality and Social Psychology, 83*, 606-615.
- Twenge, J. M., Catanese, K. R., & Baumeister, R. F. (2003). Social exclusion and the deconstructed state: Time perception, meaninglessness, lethargy, lack of emotion, and self-awareness. *Journal of Personality and Social Psychology, 85*, 409-423.
- Upton, B., Eisenlohr-Moul, T., Peters, J., & Baer, R. (under review). The role of rumination in borderline personality disorder.

- Ushiroyama, T., Ueki, M., Orino, I., & Ikeda, A. (1994). Alexithymia and undefined complaints in climacteric women: Prevalence, correlates and personality traits. *Research Communications in Psychology, Psychiatry & Behavior, 19*, 49-58.
- van Rooijen, M., Hansson, L. O., Frostegård, J., Silveira, A., Hamsten, A., & Bremme, K. (2006). Treatment with combined oral contraceptives induces a rise in serum C-reactive protein in the absence of a general inflammatory response. *Journal of Thrombosis and Haemostasis, 4*(1), 77-82.
- Verdejo-García, A., Bechara, A., Recknor, E. C., & Pérez-García, M. (2007). Negative emotion-driven impulsivity predicts substance dependence problems. *Drug and Alcohol Dependence, 91*(2), 213-219.
- Whiteside, S. P., & Lynam, D. R. (2001). The Five Factor Model and impulsivity: Using a structural model of personality to understand impulsivity. *Personality and Individual Differences, 30*, 669-689.
- Widiger, T. A., & Mullins-Sweatt, S. N. (2009). Five-factor model of personality disorder: a proposal for DSM-V. *Annual Review of Clinical Psychology, 5*, 197-220.
- Widiger, T.A., & Weissman, M.M. (1991). Epidemiology of borderline personality disorder. *Hospital and Community Psychiatry, 42*, 1015-1021.
- Wilcox, A.J., Dunson, D.B., Weinberg, C.R., Trussell, J., & Baird, D.D. (2001). Likelihood of conception with a single act of intercourse: Providing benchmark rates for assessment of post-coital contraceptives. *Contraception, 63*, 211-215.
- Zanarini, M. C., Vujanovic, A. A., Parachini, E. A., Boulanger, J. L., Frankenburg, F. R.,

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CURRICULUM VITAE

EDUCATION

University of Kentucky; Lexington, Kentucky

M.S. in Clinical Psychology

Date of Completion: August 2011

Thesis: *Self-regulation and liver function: Expanding an ecological model.*

Hillsdale College; Hillsdale, Michigan

B.A. in Psychology, Magna cum Laude

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PUBLICATIONS

Seegerstrom, S. C., Evans, D. R., & **Eisenlohr-Moul, T.** (2010). Optimism and pessimism dimensions in the Life Orientation Test-Revised: Method and meaning. *Journal of Research in Personality, 45*(1), 126-129.

Seegerstrom, S. C., Smith, T. W., & **Eisenlohr-Moul, T.** (2011). Positive psychophysiology: The body and self-regulation. In K. M. Sheldon, T. B. Kashdan, & M. F. Steger (Eds.), *Designing the Future of Positive Psychology: Taking Stock and Moving Forward* (pp. 25-40). New York: Oxford University Press.

Baer, R., Peters, J., **Eisenlohr-Moul, T.**, Geiger, P., & Sauer, S. (2012). Emotion-related cognitive processes in borderline personality disorder: A review of the empirical literature. *Clinical Psychology Review, 32*(5), 359-369.

Eisenlohr-Moul, T., Walsh, E., Charnigo, R., Baer, R., & Lynam, D. (2012). The “what” and the “how” of mindfulness: using interactions among the subscales of the Five-Facet Mindfulness Questionnaire to understand its relation to substance use. *Assessment, 19*(3), 276-286.

Eisenlohr-Moul, T., Burris, J., & Evans, D. (2012). Pain acceptance, self-regulatory fatigue, and psychological functioning in temporomandibular disorder. *Health Psychology.*

Eisenlohr-Moul, T., Seegerstrom, S. C., & Fillmore, M. (2012). “Pause and Plan” includes the liver: Self-regulatory effort slows alcohol metabolism in men with low trait self-control. *Biological Psychology, 91*(2), 229-231.

Eisenlohr-Moul, T., & Seegerstrom, S. C. (2013). Autonomy, relationships, and IL-6: Evidence for gender-specific effects. *British Journal of Health Psychology, 18*(2), 420-438.

de Leeuw, R., **Eisenlohr-Moul, T.**, Burris, J., & Bertrand, P. (2013). The association of smoking status with sleep disturbance, psychological functioning, and pain severity in patients with temporomandibular disorders. *Journal of Orofacial Pain, 27*(1), 32-41.

Evans, D.R., **Eisenlohr-Moul, T.**, Button, D., Baer, R., & Segerstrom, S.C. (in press). Self-regulatory deficits associated with unpracticed mindfulness strategies for coping with acute pain. *Journal of Applied Social Psychology*.

Sauer-Zavala, S., Walsh, E., **Eisenlohr-Moul, T.**, & Lykins, E. (in press). Dismantling mindfulness-based interventions: Differential effects of body scan, sitting meditation, and yoga. *Mindfulness*.

Eisenlohr-Moul, T., Peters, J., & Baer, R. (chapter in press). How do mindfulness-based interventions work? Strategies for studying the mechanisms of change.

Duke, A., Giancola, P., Begue, L., Bell, R., & **Eisenlohr-Moul, T.** (in press). Revisiting the Serotonin Deficiency Hypothesis of Human Aggression: A Meta-analysis. *Psychological Bulletin*.

Eisenlohr-Moul, T., Peters, J., & Baer, R. (chapter in press). Using mindfulness effectively in clinical practice: Two clinical case studies.

Peters, J., **Eisenlohr-Moul, T.**, Upton, B., & Baer, R. (in press) Nonjudgment as a moderator of the effect of present-centered awareness on psychological difficulties: Highlighting the importance of interactions in mindfulness assessment. *Personality and Individual Differences*.

POSITIONS HELD

2011-2013	University of Kentucky, Department of Psychology <i>Research Assistant, Center for Drug Abuse Research Translation</i> Principal Investigators: Richard Milich, Ph.D. and Don Lynam, Ph.D.
2010-2012	University of Kentucky, Division Orofacial Pain (Dentistry) <i>Psychological Research Associate</i> Principal Investigators: Charles Carlson, Ph.D., Reny de Leeuw, D.M.D.
2009-2011	University of Kentucky, Department of Psychology <i>Project Manager/Interviewer, "Thoughts, Stress, and Immunity in Older Adults"</i> Principal Investigator: Suzanne Segerstrom, Ph.D.

GRANT APPLICATIONS AND AWARDS

2012	University of Kentucky Center for Drug and Alcohol Research, Funded: "Ovulatory Hormone Shifts, Borderline Personality Disorder Symptoms, & Alcohol Use"
2011	Submitted NRSA Pre-Doctoral Training Grant: "Ovulation, Estradiol, and Symptom Expression in Borderline Personality Disorder"

CLINICAL EXPERIENCE

2009-2013	<i>CBT/DBT Psychotherapist to Individual Clients</i> Jesse G. Harris Psychological Services Center, Lexington, KY Supervisors: MaryBeth McGavran, Ph.D.; Ruth Baer, Ph.D.
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- 2009-2013 *Group Psychotherapist: Dialectical Behavior Therapy, Child Social Skills, Mindfulness-Based Stress Reduction*
Jesse G. Harris Psychological Services Center, Lexington, KY
Supervisors: Richard Milich, Ph.D. (SS); Ruth Baer, Ph.D. (DBT/MBSR)
- 2012-2013 *Groups Coordinator*
Jesse G. Harris Psychological Services Center, Lexington, KY
Supervisors: David Susman, Ph.D.
- 2010-2012 *Behavioral Medicine Resident*
University of Kentucky Orofacial Pain Center, Lexington, KY
Supervisors: Emily Brechting, Ph.D.; Charles Carlson, Ph.D., Jeffrey Okeson, D.M.D.

TEACHING EXPERIENCE

University of Kentucky

- 2008-2009 *Teaching Assistant, Introduction to Psychology*
- 2009-2012 *Frequent Guest Instructor, Dialectical Behavior Therapy Clinical Skills Course*
- 2011-2012 *Guest Speaker on the topic of Dialectical Behavior Therapy, Psychological Internship Course*

PROFESSIONAL ACTIVITIES

- 2011-Present *Founding Member, Diversity Task Force, University of Kentucky Clinical Psychology Program.*
- 2012-Present *Founding Member, Quantitative Psychology Interest Group, University of Kentucky Psychology.*
- 2012 & 2013 *Research Fellow, Mind and Life Summer Research Institute*

HONORS AND AWARDS

University of Kentucky

- 2013 *Winner, Nietzel Award (Awarded to the outstanding Ph.D. recipient from all Graduate Programs in Psychology at the University of Kentucky)*
- 2012-2013 *Winner/Fellow, Dissertation Year Fellowship (University of Kentucky Graduate School - Full Stipend Award)*
- 2012 *Recipient, Excellence in Clinical Practice Award*
- 2008-2013 *Recipient, Graduate Travel Award*
- 2008-2013 *Recipient, RCTF Travel and Research Awards*

Hillsdale College

- 2007 & 2008 *Winner, Outstanding Research in Psychology Scholarship*
- 2004-2008 *Recipient, Presidential Scholar Award (Half Tuition Scholarship)*