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Testing Emotion Regulation and Parasympathetic Nervous System Deficits as a Mechanism for the Transmission of Borderline Personality Disorder

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TESTING EMOTION REGULATION AND PARASYMPATHETIC NERVOUS

SYSTEM DEFICITS AS A MECHANISM FOR THE TRANSMISSION OF

BORDERLINE PERSONALITY DISORDER

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TESTING EMOTION REGULATION AND PARASYMPATHETIC NERVOUS SYSTEM DEFICITS AS A MECHANISM FOR THE TRANSMISSION OF BORDERLINE PERSONALITY DISORDER

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ABSTRACT

The present study explored the role of parental physiological state and parental emotion regulation (ER) deficits on the relationship between parent borderline personality disorder (BPD) symptoms and child BPD symptoms. Participants were 110 adolescents aged 11-13 years and their legal guardians who completed measures of BPD symptom severity and emotion dysregulation before engaging in an interpersonal conflict discussion task while being monitored for peripheral psychophysiological signals (i.e., respiratory sinus arrhythmia; RSA). Multiple mediation analyses were conducted to examine the model proposed in this study. The results revealed that parent BPD symptoms predicted lower parent baseline RSA at trend level, but was not predictive of RSA reactivity. Parent RSA did not predict parent ER deficits or child BPD symptoms. However, parental BPD symptoms predict increased parent ER deficits which, in turn, predict greater child BPD symptoms. These findings suggest that the transmission of BPD from parent to child may be in part due to the ER strategies at use during parenting and not merely the existence of the disorder itself.

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CHAPTER I

INTRODUCTION

Borderline Personality Disorder (BPD) is a disorder marked by unstable and intense interpersonal relationships, impulsivity, instability of affect, and difficulty controlling anger (American Psychiatric Association, 2013). Borderline PD is found in 1-2% of the general population (Lenzenweger, Lane, Loranger, & Kessler, 2007), but the prevalence increases in clinical settings. About 10% of mental health outpatients and 15-20% of inpatients are diagnosed with BPD (Widiger & Weissman, 1991), making BPD the most commonly diagnosed personality disorder among mental health inpatients. The high prevalence of BPD is matched only by its devastating effects. Over 60% of those with BPD attempt suicide (Kullgren, Renberg, & Jacobsson, 1986) and 10% successfully commit suicide, which is fifty times higher than the death by suicide rate of the general population (Skodol et al., 2002). Additionally, individuals with BPD are more likely to experience physical health problems, poor treatment response, social stigma, and comorbid psychiatric disorders (Stepp, 2012). The high prevalence, mortality rates, and

potential concurrent problems among those with BPD make it an important disorder to understand, prevent, and properly treat.

1.1 Borderline Personality Disorder in Adolescence

Borderline PD emerges early in development, as the average reported age of onset is 17.3 years old (Zanarini et al., 2006). While different viewpoints exist regarding whether BPD can develop during adolescence, even subclinical levels of BPD may have severe consequences during this period which affect the critical development of autonomy, self-identity, and emotion regulation (ER). A failure to develop these interpersonal skills may have long lasting effects into adulthood. For example, young girls with BPD symptoms experience worse performance academically and socially as well as increased mental health problems (Bagge et al., 2004; Wright, Zalewski, Hallquist, Hipwell, & Stepp, 2016). Wright and colleagues also found that BPD symptoms were associated with inferior social skills and self-perception among girls between the ages of 14 and 17 years. As BPD symptoms increase, risk for unsuccessfully developing good social skills and self-identity also increases, which could lead to future difficulties with interpersonal functioning and self-esteem (Bender, Morey, & Skodol, 2011). Such deficits carry through adulthood as evidenced by research that suggests BPD symptoms in adolescence predict low social support, life satisfaction, and functioning in adulthood (Winograd, Cohen, & Chen, 2008).

While not commonly diagnosed in adolescents, core BPD features appear to emerge early in development. In particular, anger and impulsivity may emerge during childhood in the form of Oppositional Defiant Disorder (ODD) and Attention Deficit

Hyperactivity Disorder (ADHD) (Stepp, 2012), two disorders often linked to BPD development later in adulthood (Stepp, Burke, Hipwell, & Loeber, 2012). Further, the distress experienced by many individuals with BPD may first be experienced in mood and affective disorders. In particular, adolescent Major Depressive Disorder (MDD) and Generalized Anxiety Disorder (GAD) are often comorbid with adolescent BPD symptoms (Sharp & Fonagy, 2015) and early precursors to the development of BPD in adulthood (Stepp, Olino, Klein, Seeley, & Lewinsohn, 2013). However, early onset of ODD, GAD, ADHD, and MDD does not necessarily indicate the development of BPD. Borderline PD shares core features from both externalizing and internalizing disorders, so identifying risk factors that are unique to BPD onset are critical for early detection and preventive measures.

1.2 Risk Factors

Familial BPD diagnosis is one significant risk factor for the development of the disorder. It is estimated that between .8-25% of those with family members diagnosed with BPD (White, Gunderson, Zanarini, & Hudson, 2003) and 11.5% of those with first-degree relatives diagnosed with BPD (Nigg & Goldsmith, 1994) will go on to develop it themselves. Specifically, maternal BPD diagnosis is related to BPD symptoms in their offspring in childhood (Weiss et al., 1996), adolescence, (Barnow et al., 2013), and adulthood (Stepp, Olino, Klein, Seeley, & Lewinsohn, 2013). This familial pattern may suggest that biological and behavioral processes play a role in the transmission of the disorder.

Linehan's biosocial model provides one framework for understanding how parental BPD symptoms play a role in child BPD development (1993). This model reflects the diathesis between genetic and physiological vulnerabilities that predispose a child to experience emotion dysregulation and environmental stress in the form of invalidating environments that disregard a child's emotional experience. She argues that some individuals are genetically predisposed to develop BPD symptoms, but that whether they actually go on to develop the disorder is dependent on the environment in which they were raised. A child with biological vulnerabilities raised in a validating environment may not develop BPD just as a child with low biological vulnerability raised in an invalidating environment may be protected from developing BPD in adulthood.

Crowell, Beauchaine, and Linehan developed a developmental framework in which the biosocial model operates (2009). They point out that because traits like impulsivity and emotional intensity emerge early in childhood and are highly heritable (Beauchaine & Neuhaus, 2008), while physiological markers of emotion dysregulation don't emerge until sometime between preschool and middle school (Beauchaine, Gatzke-Kopp, & Mead, 2007), that the biosocial model depends on various developmental stages. Some of the symptoms of BPD are biological, while some are shaped and maintained by the environment, specifically emotion dysregulation.

1.3 Environmental and Social Contributors

Linehan's model suggests that these biological factors operate within the context in which the child is raised. Children raised in invalidating environments where they are not emotionally supported or feel ignored and have a predisposition to experience

emotions intensely are more likely to develop BPD. There are several different types of invalidating environments that are strongly linked to BPD. Crawford and colleagues found that children growing up in a family with low socioeconomic status are significantly more likely to develop BPD in adulthood (2009). Chronic stressors in school or home life in adolescence (Cohen et al., 2008) and childhood abuse, whether sexual, physical, or emotional (Bornovalova et al., 2013) also significantly predict the development of BPD. Perhaps the most significant environmental risk factor associated with BPD is parental BPD symptoms or emotion dysregulation. Parental behaviors can foster insecure interpersonal relationships and inability for children regulate their own emotions.

1.4 Emotion Regulation

Rothbart and Rueda identify several parental behaviors that may encourage maladaptive mechanisms of ER to emerge in their children. They are: the invalidation of emotions and the failure to demonstrate appropriate emotional expression, interactions that reinforce emotional arousal, and poor fit between child temperament and parenting style (2005). Mothers with BPD may demonstrate some of these behaviors, as motherhood may present a specific challenge to women with BPD. These women may struggle with fluctuations between over-control and passivity and have an inability to handle their children's emotions, which in turn creates problems in their children's ability to regulate their own emotions (Stepp, Whalen, Pilkonis, Hipwell, & Levine, 2012). Mothers with BPD tend to be less sensitive, more overprotective, and more likely to engage in maladaptive behaviors like role reversal (Eyden, Winsper, Wolke, Broome, & MacCallum, 2016). Evidence suggests that mothers with BPD also display problems

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promoting relatedness and are more likely to inhibit autonomy in their adolescent children (Frankel-Weldheter, Macfie, Strimpfel, & Watkins, 2015). The same study found that the children of mothers with BPD receive less validation and emotional engagement from their mothers which, according to Linehan's theory, would increase their risk for developing BPD themselves.

While not all children of mothers with BPD go on to develop BPD themselves, they are still at higher risk for developing a wide variety of psychiatric disorders, utilizing maladaptive ER strategies, and other adverse effects throughout all stages of development. Infants of mothers with BPD are less attentive and less interested in maternal interaction than those of healthy mothers (Newman, Stevenson, Bergman, & Boyce, 2007). Twelve month old babies present with inconsistent attachment to their mothers and even more disorganized behavior and negative mood towards strangers (Hobson et al., 2009). Between ages four and seven years, children of mothers with BPD experience poorer ER, greater fears of abandonment, and incongruent self-representation (Macfie & Swan, 2009). Young adult children of mothers with BPD experience more problems than their peers who were raised by a mother without a psychiatric disorder. Adolescents aged eleven to eighteen years experience significantly more problems with attention, delinquency, and aggressive behavior if they were raised by a mother with BPD (Barnow, Spitzer, Grabe, Kessler, & Freyberger, 2006). These same children report increased anxiety, depression, impulsivity, self-esteem problems, and suicidal ideation or behaviors compared to their peers.

1.5 Parasympathetic Nervous System

One biological vulnerability known to be adversely affected by parental behaviors is the parasympathetic nervous system (PNS). The PNS serves the function of a brake on heart rate through the vagus nerve. Respiratory sinus arrhythmia (RSA) is one way of measuring the influence of the vagal brake on the heart (Porges, 2007). Behaviorally, the vagal brake enables one to engage or disengage from their environment and facilitate ER. In social situations, the vagal brake allows for calm states, but if risk is detected in the environment, the vagal brake can be withdrawn and facilitate defensive reactions in order to modulate attention to a new stimuli or stressor in the environment. Once these external stimuli are no longer present, individuals should show vagal recovery, or return to their resting RSA level (Gentzler, Santucci, Kovacs, & Fox, 2009). RSA levels are highly heritable both at rest and during stress (Snieder, Boomsma, Van Doornen, & De Geus, 1997), which could one explanation for the transmission of physiological vulnerabilities from parent to child.

Low resting RSA is related to use of less adaptive ER strategies (Gentzler, Santucci, Kovacs, & Fox, 2009) and more maladaptive ER strategies (Santucci et al., 2008). ER deficits are at the core of BPR, as many of the behaviors displayed by individuals with BPD are either due to or as a form of ER. These individuals often utilize thought suppression as a way of attempting to regulate their negative emotions (Cheavens et al., 2005), although this form of ER is considered to be maladaptive because it heightens levels of negative emotions as well as interpersonal dysfunction (Gross & John, 2003). In fact, Gross and John found that thought suppression in individuals with BPD leads to increased physiological arousal and more intense emotions (1993). High resting

RSA is related to higher regulatory control and less negative emotional responses to stress (Fabes & Eisenberg, 1997). Children and adolescents with high resting RSA are at lower risk for delinquency (Pine et al., 1998) and may be protected from adverse effects of parental conflict (Katz & Gottman, 1995). Adults with lower resting RSA tend to experience more social anxiety (Movius & Allen, 2005), are at higher risk for depression (Yaroslavsky, Bylsma, Rottenberg, & Kovacs, 2013), and are more likely to self-injure (Wielgus, Aldrich, Mezulis, & Crowell, 2016). Interpersonally, high resting RSA is associated with social competence and assertiveness (Beauchaine, 2001), while higher levels of RSA withdraw are associated with better social skills (Graziano, Keane, & Culkins, 2007).

Given the links between ER, interpersonal functioning, and PNS activity, it is not surprising that those with BPD have some impairment in this system. The literature is varied, as some find differences in PNS activity within this group while some do not. It is well documented that individuals with BPD experience negative reactivity and intense and unstable interpersonal feelings. Physiologically, those with BPD likely have lower resting RSA, heightened responses to emotional stimuli, and a slower return to baseline (Kuo & Linehan, 2009). However, research by Austin, Riniolo, and Porgues found that a group with BPD did not have significant differences in baseline RSA than a control group but when presented with affective stimuli, the BPD group displayed decreased RSA over the course of the experiment, while the control group had a gradual increase in RSA activity (2007). These mixed findings may reflect use of a negative mood induction that does not capture the interpersonal component in BPD.

1.6 Purpose for Study

Although the body of research surrounding the causes, factors, and outcomes of BPD is rapidly expanding, there are still many areas left unexplored or unclarified. Surprisingly, mothers with BPD are not often researched, and the mechanisms by which BPD is transmitted from parent to child is researched even less. Knowledge about how this disorder passes from parent to child is crucial to understanding the most important factors involved in the development and maintenance of maladaptive ER strategies, negative social interactions, and other distressing symptoms of BPD. Not only does this information help develop more successful treatment plans and early intervention programs, but it could also provide useful insight into how to stop the cycling from parent BPD to child BPD. Additionally, more knowledge about the onset of BPD can help adolescents with BPD features develop more typical interpersonal behaviors and provide them with information about adaptive ER strategies to prevent even more functional impairment in the future. Exploring the presentation of BPD in younger individuals may lead to more information that could aid in the development of successful treatments and screening tools for this population. While much research has been done on the role of the PNS in ER, there has been a significant lack of research about how this functions within the context of BPD.

The current study aims to examine the mechanistic role of maternal ER deficits between parental and offspring BPD symptoms.

Hypothesis I. Parental BPD symptoms will predict lower parental RSA levels, which will in turn predict higher parental ER deficits and atypical RSA reactivity in response to a stressful interpersonal task with their offspring.

Hypothesis II. Parental RSA levels will predict BPD symptoms in their offspring.

Hypothesis III. Parental ER deficits mediate the relationship between parental RSA levels and offspring BPD symptoms.

Hypothesis IV. Parental BPD symptoms will predict offspring BPD symptoms, as mediated by parental RSA levels and parental ER deficits.

CHAPTER II

METHODS

2.1 Participants

Adolescents (N=110) aged 11-13 and legal guardians will be recruited from child psychiatric clinics at Western Psychiatric Institute and Clinic by posting flyers and brochures around the clinic. Parents will call a member of research staff or complete a permission to contact form through a member of clinic staff. The youth will be screened with the emotional instability screener and the BPD screener after parental consent has been given.

2.2 Measures

2.2.1 Childhood Borderline Personality Disorder Symptoms.

The Childhood Interview for DSM-IV Borderline Personality Disorder (CI-BPD) is a semi-structured clinical interview designed to be used with children and adolescents to assess BPD symptoms. There are 9 criteria reflecting the 9 symptoms of BPD.

Interviewers rate each item as either "0" for absent of symptoms, "1" if the symptom is probably present, or "2" if the symptom is definitely present.

2.2.2 Adult Borderline Personality Disorder Symptoms.

The Structured Interview for DSM-IV Personality (SIDP-IV) is a semi-structured interview designed to assess BPD symptoms in adults. There are 9 criteria reflecting each of the 9 symptoms of BPD. Interviewers rate each item as either "0" for not present, "1" for almost present, "2" for present, and "3" for strongly present.

2.2.3 Emotion Regulation.

The Difficulties in Emotion Regulation Scale (DERS) is a 36 item measure assessing individual differences in aspects of emotion regulation. Participants respond on a 5 point Likert scale to statements like "When I'm upset, I lose control over my behaviors".

2.2.4 Respiratory Sinus Arrhythmia.

Electrocardiogram (ECG) electrodes will measure resting RSA levels using the MP150 Data Acquisition System and software from Mindware Technologies. The electrodes will be placed on the left and right rib cage at the heart level in a modified Lead-II configuration. Signals will be acquired at a frequency of 2000 Hz and submitted through a 0.01 high-pass filter. Interbeat intervals of the ECG will be interpolated and the ECG series will be transformed using Fast Fourier analysis. The high-frequency (HF) power band of heart rate variability (0.12-0.40 Hz) will be used to calculate RSA activity during a three-minute resting baseline.

2.3 Procedure

Participants will participate in a phone screening, though which the child will be administered the Emotional Instability screener and the BPD screener. Then, participants will come into the lab for a baseline assessment that will take between 4-5 hours. During this visit, the participants are informed of the lab procedures and sign informed assent and consent forms. The parent and child will complete several measures of BPD symptoms, childhood internalizing and externalizing behaviors, parenting practices, and other family variables. Then, parent and child will be interviewed separately using the Structured Interview for DSM-IV – Borderline Section (SIDP-IV-BOR) for the parent and the Schedule for Affective Disorders and Schizophrenia in School-Age Children – Present and Lifetime version (K-SADS-PL) for the child. Parent and child will both complete the Positive and Negative Affect Schedule (PANAS-X47) and then researchers will attach transducers to both individuals to record their physiological signals.

Then, the dyad will engage in a five-minute period where they sit silently while holding a hand grip with minimal exertion for three minutes and then 20% strength for two minutes. This will be followed with two "vanilla baseline" periods during which each individual takes two minutes to read an article out loud. Then, the two will engage in another two minutes of silence. Parent and child will then rate the PANAS-X again. The two will then engage in the Personal Stressor Disclosure Task. Adolescents will review a list of common stressors prior to the interaction and identify topics that often create issues between the child and parent. The parent and their child will then discuss this topic for eight minutes. After this discussion, both will complete the PANAS-X again. The dyad will then engage in a debriefing discussion during which the two will plan a family

vacation together to end the interaction positively. This process will be repeated again in 9 and 18 months.

2.4 Data Analyses

All analyses will be completed using SPSS version 23 (SPSS IBM, 2014). Descriptive statistics including range, mean, standard deviation, and correlations will be performed.

Hypothesis I will be tested by running a mediation model of parental RSA levels on parental BPD symptoms and parental ER deficits in Mplus.

Hypothesis II will be tested by running a regression of parental resting RSA on offspring BPD symptoms in Mplus.

Hypothesis III will be tested by running a mediation model of parental ER deficits on parental RSA levels and offspring BPD symptoms in Mplus.

Hypothesis IV will be tested by running a mediation model of parental RSA levels and parental ER deficits on parental BPD symptoms and offspring BPD symptoms in Mplus.

CHAPTER III

RESULTS

3.1 Descriptive Analyses

Means, standard deviations, and bivariate correlations are presented in Table 1. Pearson correlations were conducted to examine bivariate correlations between all variables. Higher scores on the DERS was significantly related to increased parent BPD symptom severity, $r = .44$ and increased child BPD symptom severity, $r = .30$ ($p < .01$). Higher parent age was significantly related to decreased BPD symptom severity, $r = -.28$, $p < .01$. Child BPD symptom severity was significantly related to child gender, with females reporting increased BPD symptom severity, $r = .28$, $p < .01$. Surprisingly, baseline RSA was unrelated to any study variable other than change in RSA, $r = -.51$ ($p < .01$). Age and gender of both parent and child were examined as potential covariates.

3.2 Hypothesis Testing

The first aim of this study was to determine whether parental BPD symptom severity predicted parental DERS score via parental RSA level (Figure 1). Two mediation

models were conducted to determine the predictive effects of parental BPD symptoms on parental ER deficits via parental baseline RSA (Figure 1, Paths A & B) and via parental RSA reactivity (Figure 1, Paths C & D). Parent age and gender were examined as covariates. As hypothesized, parental BPD symptoms predicted parental RSA levels at baseline (Figure 1, Path A), but only at trend level, , $\beta = -.28$, $R^2 = .14$, $p = .07$. However, parental baseline RSA did not significantly predict parental DERS score (Figure 1, Path B), $\beta = .18$, $R^2 = .23$, $p = .16$. Contrary to expectation, parental BPD symptoms did not predict parental RSA reactivity (Figure 1, Path C), $\beta = .05$, $R^2 = .04$, $p = .76$. Parental RSA reactivity did not significantly predict parental DERS score (Figure 1, Path D), $\beta = -.11$, $R^2 = .23$, $p = .30$.

The second aim of this study was to determine whether parental RSA levels predict offspring BPD symptoms. To determine this relationship, two regression models were conducted with parental resting RSA and parental RSA reactivity as predictors and child BPD symptom severity as the outcome (Figure 1, Paths F & G). Age and gender of both parent and child were examined as covariates. Contrary to expectation, parental RSA levels at baseline had no significant effect on child BPD symptoms (Figure 1, Path F), $\beta = .03$, $p = .81$. Change in parental RSA reactivity did not have a significant effect on child BPD symptoms (Figure 1, Path G), $\beta = -.02$, $p = .88$.

The third aim of this study was to determine how parental RSA levels predict child BPD symptoms through parental ER deficits. In order to examine this relationship, two mediation models were run with parental baseline RSA and parental RSA reactivity as predictors, parental DERS score as a mediator (Figure 1, Paths B & D), and child BPD

symptom severity as the outcome (Figure 1, Path E). Age and gender of both parent and child were examined as covariates. As noted above, neither parent RSA levels at baseline nor parent RSA reactivity were not significantly predictive of parental DERS score (Figure 1, Paths B & D). As hypothesized, parental DERS score was significantly predictive of child BPD symptom severity (Figure 1, Path E), $\beta = .26$, $R^2 = .14$, $p = .01$.

The final aim of this study was to determine how parental BPD symptoms predict child BPD symptoms through both parental RSA levels and parental ER difficulties. In order to examine this relationship, two mediation models were run, first with parental BPD as the predictor, parental RSA level at baseline as the first mediator (Figure 1, Path A), parental DERS score as the second mediator (Figure 1, Path B), and child BPD symptoms as the outcome (Figure 1, Path E). The second model was run with parental BPD as the predictor, parental RSA reactivity as the first mediator (Figure 1, Path C), parental DERS score as the second mediator (Figure 1, Path D), and child BPD symptoms as the outcome (Figure 1, Path E). Age and gender of both parent and child were examined as covariates. In this full model, parental BPD symptoms were significantly predictive of parental RSA at baseline, $\beta = -.31$, $R^2 = .21$, $p = .05$, which, in turn, were predictive of parental DERS score at trend level, $\beta = .22$, $R^2 = .28$, $p = .07$. Parent DERS score was significantly predictive of child BPD symptoms, $\beta = .27$, $R^2 = .16$, $p = .01$. In the second model, Parent BPD symptoms were not significantly predictive of parent RSA reactivity, $\beta = .01$, $R^2 = .06$, $p = .65$. Parent RSA reactivity was not significantly predictive of parental DERS score, $\beta = -.11$, $R^2 = .28$, $p = .27$. The direct effect of parent BPD symptoms on child BPD symptoms was not significant, $\beta = .04$, $p = .73$.

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CHAPTER IV

DISCUSSION

The present study examined the relationship between parent and child BPD symptoms as transmitted through parental resting RSA, parental RSA reactivity, and parental ER deficits. This study was informed in part by Linehan's biosocial model (1993), which suggests that transmission of BPD from parent to child is caused by a combination of physiological and environmental factors. According to Linehan's model, children with physiological vulnerabilities towards emotion dysregulation are more likely to develop BPD if they are raised in an invalidating environment. This study incorporates both of these elements by examining the role of RSA as a physiological marker of emotion dysregulation and parental ER deficits as contributing to the invalidating environment of the child. Specifically, this study tested parental RSA and parental ER deficits as mediators between parent and child BPD symptoms.

The first hypothesis of this study, that the relationship between parental BPD symptoms and parental ER deficits is mediated by parental RSA at baseline and RSA

reactivity, was partially supported. The present study found that greater BPD symptom severity predicted decreased baseline RSA at trend level when controlling for adult age and gender but were significant when controlling for both child and adult demographics. This finding is partially supported by previous research that suggests that individuals with BPD display lower RSA levels than controls (Kuo, Fitzpatrick, Metcalfe, & McMain, 2015; Weinberg, Klonsky, & Hajcak, 2009) as well as studies illustrating lower RSA levels in psychiatric disorders with marked emotion dysregulation (Gross & John, 2003; Movius & Allen, 2005; Wielgus, Aldrich, Mezulis, & Crowell, 2016; Yaroslavsky, Bylsma, Rottenberg, & Kovacs, 2013). However, a study by Austin and colleagues did not find significant differences in RSA levels between individuals with BPD and controls, but these findings may be due to small sample size (2007). The present study had a relatively small sample of participants with BPD, as only 14 parents and 32 children met criteria for the disorder. A larger sample of clinically impaired participants may have shown a significant relationship between parental BPD symptoms and RSA at baseline.

The present study also found that parental BPD symptoms did not predict RSA reactivity, which is consistent with previous research. Weinberg and colleagues found that there was not a significant difference in RSA reactivity between BPD participants and controls in a five-minute math stressor task (2009). However, Austin and colleagues found that significant differences in RSA reactivity between controls and BPD participants did not occur until the third of three ten-minute film clips (2007). It could be that the interpersonal conflict task in the present study was not long enough to elicit a detectable physiological change. This study measured baseline RSA levels as participants

were sitting quietly and their stressor was a series of films, two of which depicted conflicts. In the present study, participants were talking during both baseline and the conflict discussion task, which may have interfered with the ability to detect a difference between RSA at baseline and RSA during the interpersonal stressor task. These findings suggest that RSA reactivity in BPD may only be detectible after longer exposure to external stressors.

Contrary to prior research, the present study found that neither parental RSA at baseline nor parental RSA reactivity significantly predicted parental ER deficits. Previous research suggests that low levels of RSA are related to use of less adaptive ER strategies (Fabes & Eisenberg, 1997; Gentzler, Santucci, Kovacs, & Fox, 2009). Gentzler and colleagues found that RSA reactivity in children was predictive of adaptive, but not maladaptive ER responses (2009), which contracts the findings in this study in which there was no effect of RSA reactivity on ER difficulties. As noted above, these findings may have been affected by the presence of speech during baseline and conflict discussion. Additionally, many studies that have found significant relationships between RSA reactivity and ER have utilized affective film clips to elicit emotion reactivity. It could be that our conflict discussion task was not emotionally evocative enough to detect a change in physiological responses.

The second hypothesis of this study, that parental RSA levels would predict child BPD symptoms, was not supported. Neither parental RSA levels at baseline nor RSA reactivity predicted BPD symptoms in the child. Previous research notes that PNS activity is related to social processes and ER strategies (Beauchaine, 2001; Gentzler, Santucci, Kovacs, & Fox, 2009; Movius & Allen, 2005; Santucci et al., 2008). It was

hypothesized that lower resting RSA in the parent would lead to increased symptoms of BPD in their children because of their parent's biological vulnerability to emotion dysregulation and use of less prosocial behaviors, but this hypothesis was not supported by this study.

The third hypothesis of this study, that parental RSA levels would predict child BPD symptoms through parental ER deficits, was partially supported. As noted above, parental RSA reactivity was not predictive of parental emotion dysregulation, contrary to previous research. However, consistent with expectation, increased parental ER deficits predicted increased child BPD symptoms. Previous research suggests that the way a parent processes and displays emotions has significant implications for the way their children will display and process their own emotions (Lunkenheimer, Shields, & Cortina, 2007) which may lead them to developing problems with emotion dysregulation themselves (Eyden, Winsper, Wolke, Broome, & MacCallum, 2016; Frankel-Weldheter, Macfie, Strimpfel, & Watkins, 2015). Stepp and colleagues suggest that mothers with difficulty engaging in adaptive ER strategies may be unable to handle their children's emotions, which in turn creates problems in their children's ability to regulate their own emotions (2012). These findings suggest that parents with ER deficits, not just parents with BPD, are likely to have children with symptoms of BPD, indicating that parent behaviors and emotional responses are important mediators in the relationship and transmission of BPD and ER deficits from parent to child.

The final hypothesis, that parental BPD symptoms predict child BPD symptoms through parental RSA levels and parental ER deficits, was partially supported. It was found that parental BPD symptoms were not directly related to child BPD symptoms,

suggesting that paternal ER deficits mediated this relationship. This finding is consistent with previous research. Macfie and Swan note that not all children of mothers with BPD will go on to develop BPD themselves, but it may be exposure to certain aspects of BPD that determines who is more likely (2009). They point out failure at tasks of attachment, self-development, and emotion regulation as potential mediators in the relationship between mother and child BPD development. Parents with BPD tend to offer their children less validation and emotional engagement (Frankel-Weldheter, Macfie, Strimpfel, & Watkins, 2015), are more likely to engage in maladaptive behaviors like role reversal (Eyden, Winsper, Wolke, Broome, & MacCallum, 2016), and are more likely to invalidate their children's emotions and fail to demonstrate appropriate emotion expression themselves (Rothbart & Rueda, 2005). The present study suggests that these parental behaviors and maladaptive ER responses may at least be partially responsible for the transmission of the disorder from parent to child.

4.1 Limitations

The findings of this study should be considered with a number of limitations. First, this study only contained 14 parents and 32 children who meet criteria for BPD. A larger BPD sample size would likely increase the statistical power of these findings. Second, BPD is most commonly diagnosed in women. The majority of children in this sample were male (n = 70). If only women and their daughters were included in this sample, there may have been increased severity or prevalence of BPD symptoms. Additionally, parent gender was correlated with parent BPD symptoms at trend level and child gender was significantly correlated with child BPD symptoms, which may have reduced the relationships among the variables in this study. Third, as noted above, the

measures of RSA in this study may have been confounded by speech. Participants were speaking in both the baseline and conflict discussion phases of the study and this could change the way respiration is measured and interfere with true levels of RSA. Finally, the present study did not control for disorders that are comorbid disorders or other diagnoses that are commonly associated with ER deficits (e.g., MDD and GAD).

4.2 Future Research

The design of this study reveals several limitations that should be addressed in future research. First, utilizing a sample with more individuals diagnosed with BPD could improve statistical findings. Second, controlling for comorbid disorders could account for confounding effects on emotion dysregulation. Third, looking at another measure of PNS activity could help better understand the role of physiological responses to emotion dysregulation without the potential confound of variable respiration and speech in RSA. Finally, assessing only females within this study may increase the statistical significance of these findings.

4.3 Strengths and Clinical Implications

This study is one of the first to investigate the role of parental PNS activity in the relationship between parental BPD and child BPD symptoms. Prior studies have examined the role of the PNS within BPD, but no known research has examined how parental physiology and emotion dysregulation influence the transmission of BPD from parent to child.

This study is clinically significant because it illustrates that parental BPD predicts child BPD only when ER deficits are used as a mediator. This indicates that transmission

from parent to child may not be due to the presence of the disorder itself, but to the ER strategies the parent engages in to feel better. While this study did not find significant results for transmission through RSA, the trend level significance suggests that there may be a relationship between the transmission of BPD and RSA in a different sample. This study will help researchers further understand the physiological and ER influences on parent to child BPD transmission, which may improve intervention efforts and treatment practices. Finally, this study is clinically and scientifically important because it combines behavioral and biological observations as advocated by the National Institute of Mental Health (NIMH, 2013).

CHAPTER VI

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APPENDIX

Table 1. Descriptive statistics and bivariate correlations of study variables.

Variables	M (SD)	1.	2.	3.	4.	5.	6.	7.	8.
1. P_BPD	5.71 (5.60)	--							
2. DERS	74.14 (22.91)	.44**	--						
3. RSA baseline	5.67 (1.55)	-.16	.12	--					
4. RSA reactivity	.20 (.78)	.02	-.16	-.51**	--				
5. C_BPD	8.59 (4.62)	.15	.30**	.02	-.04	--			
6. P_Age	40.95 (7.62)	-.28**	-.04	-.15	.04	.06	--		
7. P_Gender	1.90 (.30)	.19+	-.01	-.10	-.09	.15	.03	--	
8. C_Age	12.57 (.96)	-.09	-.04	-.00	.01	.02	.22*	-.02	--
9. C_Gender	1.38 (.49)	-.00	.21+	-.16	-.02	.28**	-.05	.01	.18+

Note. P_BPD = parent BPD symptom severity, DERS = Difficulties in Emotion Regulation Scale total, RSA baseline = vanilla baseline RSA, RSA reactivity = change in RSA, C_BPD = child BPD symptom severity, P_Age = parent age at interview, P_Gender, parent gender (higher score indicates female), C_Age = child age at interview, C_Gender = child gender (higher score indicates female)
** = significant at .01 level, * = significant at .05 level, + = trend level significance
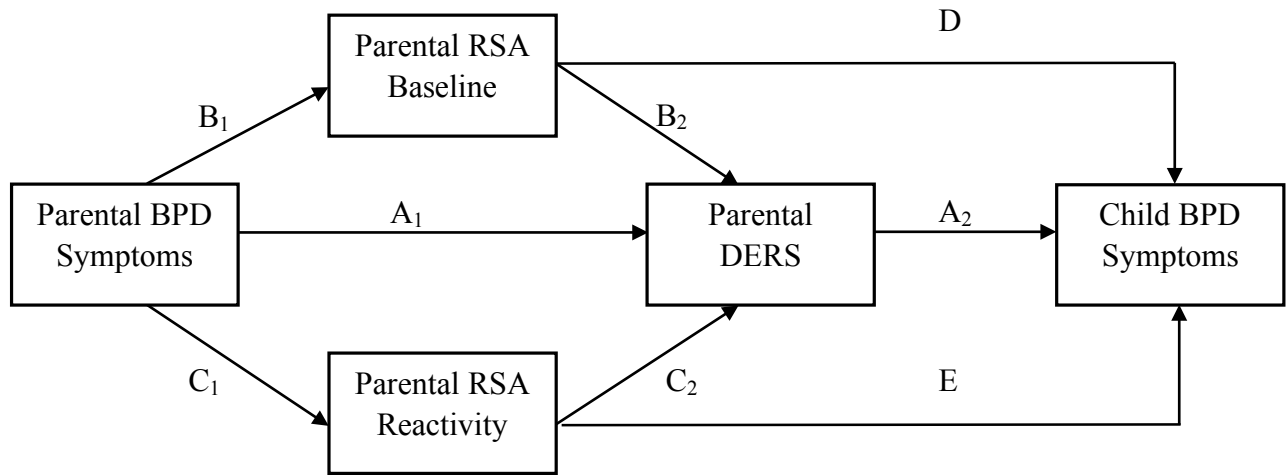
FIGURES

Figure 1. Conceptual model of transmission of BPD symptoms from parent to child via parental PNS activity and parental ER deficits. This figure illustrates the hypothesized mediation effects of parental BPD symptoms and parental RSA (B & C) on parental DERS score, as well as direct predictions of parent RSA (D & E) and parental DERS score (A_2) on child BPD symptoms.
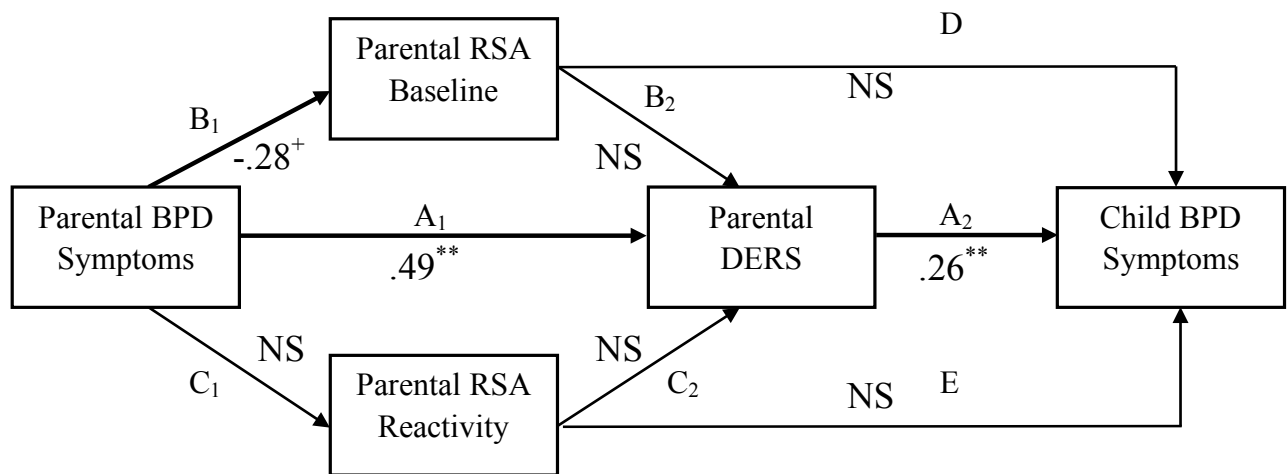

Figure 2. Standardized effects of parental BPD and its relationship to RSA baseline, RSA reactivity, DERS, and child BPD symptoms. A1 = direct effect of parental BPD on parental DERS; B_1 = direct effect of parental BPD on parental RSA baseline; B_2 = direct effect of parental RSA baseline on parental DERS score; C_1 = direct effect of parental BPD symptoms on parental RSA reactivity; C_2 = direct effect of parental RSA reactivity on parental DERS; A_2 = direct effect of parental DERS on child BPD; D = direct effect of parental RSA baseline on child BPD; E = direct effect of parental RSA reactivity on child BPD.
**p < .01. +p < .10. NS = Not Significant.