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The Effect of Emotional Vulnerability and Invalidation on Emotion Dysregulation in Early Adolescence: An Empirical Investigation of Linehan's Biosocial Theory of Borderline Personality Disorder

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Table of Contents

List of Tables

List of Figures

Sarah Crystal
324 words

Abstract

The current study examined the relationship between emotional vulnerability, invalidation, and emotion dysregulation as they predicted borderline features in a community sample of young adolescents. Emotional vulnerability, as measured by trait negative affect (trait NA), as well as the psychophysiological component of basal vagal tone, as measured by respiratory sinus arrhythmia (RSA), were proposed as risk factors for borderline features. Emotion dysregulation as indexed both by psychophysiological indices (vagal tone in response to stress i.e., RSA reactivity) and self-report measures was hypothesized to function as a mediator between trait NA and borderline features. A moderated mediation model was then proposed with parental invalidation moderating the relationship between trait NA and emotion dysregulation. A total of 101 youth, 53% female, with a mean age of 12.82 (*SD*=0.83) completed a laboratory task to measure their RSA at rest and while completing a stressor task. Trait NA, parental invalidation, emotion dysregulation, and borderline features were assessed through self-report questionnaires. Support was found when models were assessed cross-sectionally, using self-report measures only. The direct effect of trait NA on borderline features was significantly mediated by emotion dysregulation. Furthermore parental invalidation did function as a moderator between trait NA and emotion dysregulation. The full moderated mediation model was also significant. When measured using psychophysiological indices, no relationship was found between any study variables. Results indicate that child temperament, specifically trait NA, and invalidating parenting interact to produce emotional dysregulation, which is related to increased borderline pathology among

adolescents. However, the study did not implicate the involvement of physiological vulnerabilities and patterns of responding in the development of borderline features. This study suggests that understanding the risk for the development of borderline features in adolescence needs more rigorous and continued research, particularly in understanding the biological risk and role of psychophysiological responding to stress in the development of the disorder. Further exploration of how these variables are related will be important in understanding the etiology of borderline features across development.

CHAPTER I

Introduction and Literature Review

Purpose

Borderline personality disorder (BPD) is a complex and extremely debilitating psychiatric disorder that is characterized by persistent and pervasive emotional dysregulation that impairs multiple domains of an individual's functioning. Epidemiological studies estimate that BPD affects approximately 1-6% of the general population (Grant et al., 2008). BPD accounts for a disproportionate amount of individuals in mental health treatment facilities, accounting for about 10% of individuals seeking outpatient services and up to 20% of inpatient psychiatric patients (Bender et al., 2001; Grant et al., 2008; Zanarini, Frankenburg, Khera, & Bleichmar, 2001). Of these, approximately 70% of individuals with BPD self-injure and up to 10% eventually commit suicide, a rate 50 times that observed in the general population (Black, Blum, Pfohl, & Hale, 2004). Additionally, BPD is the most prevalent and debilitating of the personality disorders observed in psychiatric hospitals and thus among the more costly diagnoses facing the health care system (Bender et al. 2001; Comtois et al. 2003; Trull et al. 2003). Despite its prevalence and clinical complexity, only recently has attention been paid to the disorder within childhood or adolescence.

Currently, there is reluctance to diagnose children and adolescents with BPD, especially in clinical practice (Chanen & McCutcheon, 2008; Griffiths, 2011). This reluctance has historically resulted from the belief that personality traits are not stable until adulthood, and, up until the Diagnostic and Statistical Manual of Mental Disorders 5th Edition, diagnostic criteria did not allow diagnosis of personality disorders prior to

age 18. Although delays in diagnosis and treatment are common, there is evidence that BPD does emerge between adolescence and emerging adulthood, suggesting that it is imperative to diagnose earlier and therefore be able to provide earlier interventions (Chanen & McCutcheon, 2013). Over the past decade there has been a major increase in empirical studies examining BPD among adolescent populations (Sharp & Tackett, 2014, Miller, Muehlenkamp, & Jacobson, 2008), that has generated better understanding of the etiology, course, and prognosis of the disorder, as well as allowed for tailored early treatment approaches.

Oftentimes when the diagnosis is applied to children and adolescents, common adaptations include, "borderline pathology," "borderline features," or "borderline traits" to reflect the belief that personality is in flux during development (Crick, Murray-Close, & Woods, 2005), as well as to account for the dimensional approach in classifying symptoms. Nevertheless, research suggests that particular sets of symptoms and maladaptive coping strategies are manifest during adolescence that indicate heightened risk for later development of the disorder (Bradley, Conklin & Westen, 2005; Miller et. al., 2008; Westen & Chang 2000). Much recent research indicates that borderline features can be reliably observed during childhood and adolescence, that borderline features during adolescence have been shown to predict poor functioning over time, in academic, occupational and interpersonal/social domains, and that borderline features in youth strongly predict BPD in adulthood (Bradley et al. 2005; Sharp & Fonagy, 2015; Westen & Chang 2000; Winograd, Cohen & Chen, 2008). Thus, understanding the mechanisms that confer risk for the development and maintenance of the disorder in adolescence may help inform prevention and intervention efforts.

Theories on the development of BPD focus on individual and environmental factors that contribute to the disorder. The most widely known and empirically supported theoretical model is Linehan's biosocial model (1993). The biosocial model focuses on the individual differences in the capacity to regulate affect, behavior and physiology (Crowell, Beauchaine, Linehan, 2009; Linehan, 1993) and how these processes are impacted by environmental factors. Specifically, the theory states that BPD emerges from continual transactions between individual vulnerabilities and environmental influences, specifically, when an emotionally vulnerable, reactive child is raised in an invalidating environment. Over time, these emotionally vulnerable children do not learn to effectively regulate emotions or behaviors, display dysfunctional physiological responses, and are characterized by pervasive emotion dysregulation. According to the model, emotion dysregulation, defined as the inability to effectively modulate emotions, behavior and physiology, lies at the heart of the disorder.

In an effort to expand upon previous findings and examine the development of borderline features in a developmental population, the current study examines the relationship between emotional vulnerability, emotion dysregulation, parental invalidation, and borderline pathology in a young-adolescent sample. It is hypothesized that emotional vulnerability will predict borderline features in my sample. Moreover, it is hypothesized that this relationship will be mediated by emotional dysregulation. Furthermore, the mediation relationship will then be moderated by parental invalidation such that the effect of high emotional vulnerability on borderline features will be strongest for youth with invalidating parents.

Defining and Measuring Borderline Personality in Early Adolescence

As a diagnostic category, BPD is a personality disorder characterized by pervasive patterns of instability in interpersonal relationships, impulse control, self-regulation, and self-image. Diagnostically, DSM-5 marked a change in the classification of personality pathology because personality disorder criteria specifies that all but one personality disorder can be diagnosed in individuals prior to age 18. According to DSM-5, BPD is a personality disorder diagnosed in individuals who meet at least five out of nine diagnostic criteria including: frantic efforts to avoid real or imagined abandonment; pattern of unstable and intense interpersonal relationships; identity disturbance; impulsivity in potentially self-damaging areas; recurrent suicidal behavior, gestures, threats or self-injurious behavior; affective instability; chronic feelings of emptiness; inappropriate, intense anger; and transient, stress-related paranoid ideation or severe dissociative symptoms. Regarding diagnosis in youth, DSM-5 states,

> "Personality disorder categories may be applied with children or adolescents in those relatively unusual instances in which the individual's particular maladaptive personality traits appear to be pervasive, persistent, and unlikely to be limited to a particular developmental state or another mental disorder. It should be recognized that the traits of a personality disorder that appear in childhood will often not persist unchanged into adult life. For a personality disorder too be diagnosed in an individual younger than 18 years, the features must have been present for at least 1 year" (American Psychiatric Association, 2013, p. 647).

This diagnostic shift is important because it legitimizes recent research indicating that BPD constitutes a valid and reliable diagnosis in adolescence (Chanen & McCutcheon,

2008; Miller et al., 2008; Sharp & Romero, 2007). Nevertheless, many clinicians do not diagnose youth with BPD (Laurenssen, Hutsebaut, Feenstra, Van Busschbach, & Luyten, 2013) due to concerns about stigma, and questions regarding diagnostic reliability and stability among youth (Kernberg, Weiner, & Bandenstein, 2000; Laurenssen, Hustebaut, Feenstra, Van Busschbachl, & Luyten, 2013; Miller et. al., 2008).

Prior to the past few decades, BPD had been mostly studied in adulthood and was largely ignored by developmental researchers. Therefore, until recently much of what was known about precursors to and the development of the disorder was from retrospective adult reports. However, recent studies have demonstrated that children with borderline pathology exhibit similar risk factors as adults with BPD (Zelkowitz, Paris, Guzder & Feldman, 2001), that recognizable features of the disorder are apparent during adolescence (Bradley et al. 2005; Westen & Chang, 2000), and that BPD in youth is related to poorer psychosocial functioning in adolescence (Wright, Zalewski, Hallquist, Hipwell, & Stepp, 2016).

Some recent epidemiological studies suggest a moderate prevalence of BPD pathology in adolescents. Chabrol and colleagues (2001) surveyed more than 1300 French high school students using the Screening Test for Comorbid Personality Disorders (STCPD; Dowson, 1992) and Revised Diagnostic Interview for Borderlines (DIB-R; Zanarini, Gunderson, Frankenburg, & Chauncey, 1989). They estimated an overall frequency of 14% for individuals with BPD, with peaks in diagnosis appearing in early and late adolescence. Chabrol et al. (2004) examined BPD rates in 616 high school adolescents and found that 6% of their sample met the clinical cut-off for receiving a BPD diagnosis based on the Borderline Personality Inventory (BPI; Leichsenring, 1999).

In a larger scale epidemiological study of 10,000 11 year-olds assessed in Great Britain, Zanarini (2003) found that 3.3% of children met full diagnostic criteria for BPD. The overall prevalence rate for BPD found in adults in the general population is estimated at 2% (APA, 2000), suggesting that not all children and adolescents continue to display clinically significant BPD symptoms throughout their lives.

Clearly the assessment and measurement of BPD in youth, as well as adults, varies across studies and therefore likely impacts prevalence rates. Crick and colleagues (2005) were the first researchers to develop a psychometrically sound measure to reliably assess BPD in youth (BPFS-C; Crick, Murray-Close & Woods, 2005). Their goal was to prospectively assess dimensional borderline features among nonclinical samples. These researchers modified the Borderline Scale of the PAI (Morey, 1991), a reliable and valid instrument used to assess borderline personality features among adults. The scales included in the BPFS-C include four domains: affective instability, identity problems, negative relationships, and self-harm. Based on extensive literature reviews, the authors identified five childhood indicators of BPD theorized to represent unique aspects of the disorder (Geiger and Crick, 2001). These indicators include a hostile, paranoid world-view, intense unstable, inappropriate emotion, overly close relationships, and impulsivity. The authors used linear mixed modeling in a prospective study of 4th to 6th graders to evaluate the degree to which the five indicators and children's BPFS-C scores tracked together over three time points. Results indicated that the theoretical indicators of borderline pathology in childhood tracked together with children's borderline personality features as assessed by the BPFS-C over the course of a year.

Regarding stability of BPD symptoms over time, Crick et al (2005) found that individual differences in borderline personality features were moderately stable over the course of a year, even across transitions from one school year to another. Stepp et al. (2010) also examined the underlying features of BPD including impulsivity, negative affectivity, and interpersonal aggression, in 6-12-year-old girls. This study examined teacher and parent report of child behaviors and found stability in features across informants, across a seven-year period.

Alternatively, Wright and colleagues (2016) found significant variability in adolescent BPD symptoms across time. Girls in their study demonstrated trajectories of increasing, decreasing, and stable symptoms. This finding is important because it is congruent with the adult BPD findings (Morey & Hopwood, 2013) that personality pathology is not necessarily an "enduring pattern that is stable and of long duration" (American Psychiatric Association, 2013, pp. 646–647). Instead, it supports the idea that symptoms of personality disorders shift and change over time depending on context and development of the individual over time (Vachon et al., 2013; Wright, Pincus, & Lenzenweger, 2011). Chanen et al. (2004) also assessed the stability of BPD among 101 adolescent outpatients over a 2-year period using the SCID-II. Eleven of the 101 participants (10%) met criteria for BPD at baseline, and 12 of 96 participants (12.5%) met criteria for BPD at follow-up, suggesting moderate support for the stability of BPD in adolescents.

Given that there is variability in BPD symptoms over time, research has also examined which diagnostic criteria are most likely to remit and which are more resistant to change. It appears that more enduring features of the disorder include impulsivity,

affective instability, and anger, whereas self-injury, fears of abandonment, and identity disturbance may be more likely to reduce with age (McGlashan et al., 2005; Zanarini, Frankenburg, Hennen, Reich, & Silk, 2005). Stepp and Pilkonis (2008) examined age-related changes across a 30-year span, in 8 DSM-III-R criteria for BPD. The criteria included were unstable relationships, impulsivity, affective instability, intense anger, suicidal behavior, identity disturbance, feelings of emptiness, and frantic efforts to avoid abandonment. Findings indicated that impulsive and suicidal behaviors decreased with age, whereas other symptoms did not. Their results also indicated that emotional distress, measured by levels of anxiety and depression was higher in individuals with BPD, and that emotional distress did not decrease with age or reduced BPD symptoms. These findings continue to lend support of the heterogeneity in symptom presentation of BPD across time, and suggest that although symptoms may be different across childhood/adolescence and adulthood, they remain impairing.

Recent research has also indicated that BPD in adolescence is associated with multiple problematic outcomes. The Children Community Study is an ongoing investigation of the course of personality disorders in a sample of approximately 800 youth. Findings from this sample indicate that early adolescent BPD predicted subsequent grade retention, school dropout, and social problems at two-year follow-up (Bernstein et al., 1993). Within this sample, elevated borderline symptoms were associated with more intimate relationship break-ups, chronic stress, and abuse over the next four years (Daley, Burge, & Hammen, 2000), as well as relationship dysfunction, academic underachievement, more semesters on probation, and expulsion over the next two years (Bagge et al., 2004; Trull, Useda, Conforti, & Doan, 1997).

Wright and colleagues examined the co-development of BPD symptoms with multiple domains of psychosocial functioning in a large sample of adolescent girls aged 14-17 (Wright, Zalewski, Hallquist, Hipwell, & Stepp, 2016). This study assessed BPD using a symptom scale (International Personality Disorders Examination [IPDE-BOR]; Loranger et al., 1994) where scores above a certain cut-off indicated clinical significance. This study found that increased BPD symptoms were related to psychosocial dysfunction, and decreased BPD symptoms were related psychosocial improvements, over time. More specifically, results indicated that increasing BPD symptoms were related to worsening social, academic, and mental health outcomes throughout adolescence, whereas improved symptoms over time were associated with improvements in these domains. Furthermore, their analyses controlled for internalizing and externalizing pathology suggesting that the specific developmental associations of BPD with social skills, self-perception, and sexual activity went above and beyond general associations with psychopathology.

Winograd and colleagues (2008) also investigated the relationship of adolescent borderline symptoms to subsequent psychosocial functioning based on data from the Children in the Community cohort. This study examined symptoms of BPD at age 14 as predictors of work/school/homemaker role function, social function (social support, relationship quality), and life satisfaction over the subsequent 20 years. Results indicated that adolescents with higher levels of early adolescent borderline symptoms scored consistently lower in role function, social function, and life satisfaction from mid-adolescence through mid-adulthood. Borderline symptoms in adolescence predicted lower academic and occupational attainment, less partner involvement, and fewer attained adult developmental milestones. Adolescent borderline symptoms were also

associated with adult borderline symptoms, borderline diagnosis, general impairment, and the need for social services at mean age 33. These effects were evident despite symptom decline with age and were independent of adolescent Axis I diagnoses.

Given the research reviewed, it appears that BPD symptoms can be reliably detected and diagnosed in youth. While the temporal stability of BPD in youth is less clear, research suggests that for a small, clinically significant portion of youth, BPD symptoms persist into adulthood. Additional prospective, longitudinal studies are needed to identify those at risk, and understand the factors that impact the developmental trajectory of BPD.

Theoretical Models of BPD

Early Psychodynamic Theories. Clinical exploration and research of BPD began in the early 19th century when clinicians were unsure how to categorize patients displaying a combination of neurotic and psychotic symptoms; the term "borderline" represented this distinction. Prior to the nineteen-sixties the focus was on clinical descriptions of the disorder as clinicians struggled to fit individuals into existing diagnostic categories. Clinicians such as Knight (1953) and Stern (1938) wrote foundational articles on BPD, and Stern coined the term "borderline personality" to describe low-functioning, difficult-to-treat psychiatric patients whose symptoms lay between neurosis and psychosis.

Otto Kernberg (1967) developed the first psychodynamic conceptualization of the disorder and proposed the idea of a Borderline Personality Organization (BPO), which represented a level of personality organization or dysfunction that was between neurotic and psychotic personality organizations. BPO was characterized by distortions in reality

perception (as opposed to the genuine loss of contact with reality characteristic of psychosis), immature and maladaptive defenses (problematic emotion regulation strategies), and an inability to form complex, integrated representations of others (contributing to interpersonal instability).

Masterson (1972) developed an object-relations formulation for BPD that emphasized internalized relational patterns between individuals with BPD and their primary caregivers. He stated that children who develop BPD develop internal representations of others who respond to their legitimate expression of needs and affect with withdrawal or hostility. He believed that individuals with BPD subsequently recreated these dynamics in their adult relationships. Masterson (1976, 1989) argued that the emotional instability of individuals with BPD stems from a basic fear of abandonment that develops in the young child as a result of the primary caregiver's emotional withdrawal, unavailability, or over-protection. Within this framework the fear of abandonment is posited to trigger recurrent episodes of dysregulated emotion.

After much psychoanalytic theorizing, Gunderson and Singer (1975) published the first literature review integrating theory, clinical descriptions and psychological test findings regarding BPD and shortly thereafter developed the first assessment for BPD, the Diagnostic Interview for Borderline Patients (Gunderson & Kolb, 1978; Gunderson, Kolb, & Austin, 1981). Subsequently, Spitzer, Endicott, and Gibbon (1979) detailed the background and development of the diagnostic criteria for BPD that became the basis for the BPD criterion introduced in the *Diagnostic and Statistical Manual of Mental Disorders-III (*DSM-III; American Psychiatric Association, 1980).

Biosocial Model. In contrast to the early psychodynamic theories of BPD, contemporary theory and current empirically supported treatment for BPD is based predominantly on Linehan's biosocial model (1993). The biosocial theory depicts a transactional, etiological model, that encompasses various factors in the development and maintenance of BPD, including biology, environment, and learning. In a transactional model, risk factors are thought to influence each other reciprocally, resulting in continual changes over time to an individual, dyad or environmental circumstance. The biosocial theory states that BPD develops from the transaction between biological child factors and environmental contexts that together create difficulties managing emotions. In particular the model emphasizes the following factors to explain the development and maintenance of BPD: (1) the role of early child emotionality, which is influenced by genetics and early biological development; (2) the role of parenting and other family or caregiver responses to the child; (3) the development of pervasive emotional dysregulation.

The three main emotional characteristics of Linehan's theorized emotionally vulnerable child are (1) having a low threshold for triggering an emotional reaction to internal or external stimuli, (2) typically experiencing very strong emotional reactions, and (3) typically requiring a long time to return to a calm emotional state after experiencing an emotion. Specifically, the theory states that BPD develops when emotionally vulnerable children encounter invalidating developmental environments. Invalidating experiences actively call into question a child's understanding of her emotions. In its most extreme form invalidation can be thought of as a type of emotionally abusive or neglectful environment. Invalidation of a child's emotional expression can involve repeatedly trivializing it (e.g., "it's not that big of a deal that you

hurt yourself on your bike") or punishing it (e.g., "stop crying or I'll give you something to cry about"), or repeatedly neglecting to validate a child's emotional expression by ignoring her emotional displays (e.g., a child expresses anger over a friend stealing his toy and the parent neglects to acknowledge the child's emotional state). In this type of environment, invalidating responses take the place of healthier responses to a child's emotional displays, which would involve validating the child's response by acknowledging it and indicating that the response is understandable.

Linehan (1993) argued that the main consequence of an emotionally vulnerable child experiencing repeated invalidation of their emotional responses is emotional dysregulation. She defined emotion dysregulation as poor control over one's emotional state, an inability to inhibit maladaptive behaviors under conditions of high emotional arousal, and an inability to engage in goal-directed behavior while in a highly emotional state (1993). According to the model, emotion dysregulation is considered the core feature and difficulty, rather than a symptom of BPD. Thus, emotion dysregulation is theorized to cause individuals with BPD to engage in their characteristically dysfunctional behaviors such as self-harm, suicidality, risky sexual activity, splitting, etc.

Factors Affecting the Development of Borderline Personality Disorder

Temperament. Linehan describes an 'emotional vulnerability' that refers to a biologically mediated predisposition involving heightened sensitivity and reactivity to emotional stimuli, and a slow return to baseline level of emotional arousal (1993). Within the developmental literature, this emotional vulnerability can be well characterized by the construct temperament. Temperament refers to early appearing components of personality that remain relatively constant through development (Roberts

& DelVecchio, 2000). Thomas and Chess introduced the notion that innate individual differences in reaction and motivation were 'temperament.' Not only did their theory delineate multiple temperamental dimensions, it also emphasized the influence of reciprocal interactions between the child and its environment on the adjustment of the child. Thomas and Chess developed the concept 'goodness-of-fit' suggesting that parenting should be tailored to a child's unique temperament to assure healthy psychological development (Thomas & Chess, 1977). They also introduced the construct 'difficult temperament,' which defined a cluster of emotional and behavioral styles that are particularly challenging for caregivers and render children particularly vulnerable to developing psychopathology, regardless of their environment. Buss and Plomin (1975) modified Thomas and Chess's model by framing temperament as a developmental precursor of adult personality. Most contemporary discussions of temperament adhere to the definition put forth by Rothbart and colleagues (Rothbart & Derryberry, 1981) that defines temperament as:

> …Constitutionally based individual differences in reactivity and self-regulation as observed in the domains of emotionality, motor activity, and attention. By reactivity, we mean characteristics of the individual's responsivity to changes in stimulation, as reflected in somatic, autonomic, and endocrine nervous systems. By self-regulation, we mean processes modulating this reactivity, including behavioral approach, avoidance, inhibition, and attentional self-regulation. In our view, individual differences in temperament constitute the earliest expression of personality and the substrate from which later personality develops" (Rothbart & Posner, 2006, p. 466).

Fundamental to their theory is that temperamental differences are largely determined by the responsiveness of underlying psychobiological processes. In this regard, reactivity refers to physiological excitability of neural systems, whereas self-regulation refers to the processes enabling the modulation of this automatic, involuntary reactivity.

Despite different models of temperament, research has consistently demonstrated that certain temperament dimensions are associated with poor outcomes, and thus confer risk for the development of psychopathology (Rettew & McKee, 2005). Negative Affectivity (trait NA) is one temperamental domain that has been well established as conferring risk for the development of many psychological problems including depression (Anthony, Lonigan, Hooe, & Phillips, 2002), anxiety (Klein, Durbin, & Shankman, 2009), substance use (Willem, Bijttebier, Claes, Sools, Vandenbussche & Nigg, 2011), and BPD (Joyce et al., 2003), and represents one of the temperamental domains that comprised Thomas and Chess' 'difficult temperament" construct.

Trait NA is characterized as the experience and display of intense and frequent negative emotions, such as sadness, fear, anger and frustration (Rothbart et al., 2001), and difficulty regulating these states. Trait NA is stable into adulthood with high trait NA adults characterized by the tendency to experience a wide variety of negative emotions, to have negatively charged relationships, and to be vulnerable to the adverse effects of stress (Tellegen, 1985; Watson & Clark, 1984). Children with high trait NA are more likely to become easily upset, remain aroused for long periods of time and have difficulty recovering from these emotional experiences. Therefore the link between trait NA and various psychological disorders associated with problems regulating mood, anxiety and behavior, is unsurprising.

Trait NA and Borderline Pathology. Consistent with research in other domains of psychopathology, an association between high trait NA and BPD has been found in multiple studies (Joyce et al., 2003; Paris, 2005; Reich & Zanarini, 2001; Saulsman & Page, 2004). Bornovalova and colleagues (2006) examined the relationship between trait NA and environmental factors associated with BPD in a sample of inner-city adults with comorbid substance use and found that BPD was associated with higher trait NA (instability, reactivity and sensitivity). Another study by Joyce and colleagues (2003) examined retrospective childhood temperament and environmental experiences as risk factors for BPD in a sample of adult outpatients with comorbid depression. Their findings demonstrated that trait NA uniquely predicted development of BPD, and that the risk for developing the disorder tripled when individuals also experienced childhood abuse and/or neglect. Clarkin and Posner (2005) also demonstrated that trait NA was associated with BPD such that individuals with BPD had higher levels of trait NA than controls. Findings from various other studies also demonstrate the link between trait NA and BPD (Cheavens et al., 2004; Gratz, Tull, Baruch, Bornovalova, & Lejuez, 2008; Stepp et al., 2016).

Dixon-Gordon at colleagues (2016) examined the relationship between trait NA, maternal problem solving and supportive/validating interpersonal strategies, and BPD among adolescents. In their study, adolescent girls and their mothers completed a conflict discussion task, and maternal problem solving, support/validation, and participants' negative affect were coded. Results demonstrated a 3-way interaction of participants' trait NA, maternal problem solving, and maternal support/validation, such that that participants' negative affect was only associated with BPD severity in the context of low

maternal support/validation and high maternal problem solving. This suggests that although high trait NA is a risk for BPD severity in adolescent girls, and in general, in this study maternal interpersonal emotion regulation strategies moderated this relationship. Whereas maternal problem solving in addition to low support/validation was associated with a stronger trait NA-BPD relationship, maternal problem solving paired with high support/validation was associated with an attenuated relationship. The authors suggested that among adolescents high in trait NA, maternal problem solving in the presence of low maternal support/validation might have been viewed by participants as insensitive to their distress and thus invalidating. These findings are consistent with much theoretical and empirical literature suggesting that the interaction between trait NA and parental invalidation is crucial in the development of BPD (Linehan, 1993; Sauer and Baer, 2010).

Parasympathetic Responsivity. Emotional vulnerability can also be represented as a biological/physiological vulnerability. According to the psychophysiology literature, the emotional vulnerability proposed by Linehan may be more directly conceptualized and measured via physiological indices. Much literature has linked measures of the autonomic nervous system to various psychological conditions (Beauchaine, 2001; Beauchaine, Katkin, Strassberg, & Snarr, 2001). Of particular importance to the development of emotion dysregulation and subsequent BPD may be the functioning of the parasympathetic nervous system (PNS). The PNS is responsible for regulating the expression of our reactive systems (i.e., the sympathetic nervous system) and returning us to homeostasis, and thus regulating our emotional states. One reliable and noninvasive index of PNS activity is respiratory sinus arrhythmia (RSA; also referred to as vagal

tone). Parasympathetic regulation of cardiac activity, indexed by RSA, is associated with individual differences in emotional vulnerability (Beauchaine, 2001; Butler, Wilhelm, & Gross, 2006; Porges, 1995a, 1995b). Likewise, basal RSA is often conceptualized as an index of biological vulnerability to emotion dysregulation that well characterizes the emotional vulnerability Linehan proposes in the development of BPD.

Porges' polyvagal theory provides a coherent framework for generating hypotheses about the emotional/physiological vulnerabilities implicated in the development of BPD. Polyvagal theory (Porges, 1995; 2001; 2003; 2007) is an evolutionary approach that relates autonomic nervous system (ANS) functioning to behavior and explores parasympathetic control over heart period via the vagus nerve, a primary component of the ANS. The theory specifies two functionally distinct branches of the vagus nerve, both of which originate in the medulla, but that serve different evolutionary purposes. More specifically, each branch is associated with a different adaptive behavioral strategy, both of which are inhibitory in nature via the PNS. The vagal system is in opposition to the sympathetic-adrenal system, which is involved in mobilization behaviors. Thus, while measures of sympathetic nervous system activation (including increased heart rate, blood pressure, faster, shallower breathing and sweat secretion) are associated with emotional responding, measures of autonomic nervous system activation via the vagus nerve (i.e., RSA) are associated with an individual's capacity for emotion regulation (Beauchaine, Klein, Crowell, Derbidge, & Gatzke-Kopp, 2009).

The more primitive vagal branch termed the 'vegetative vagus' originates in the dorsal motor nucleus and mediates reflexive cardiac activity and is responsible for

primitive threat responses like orienting, feigning death, and immobilization (Beauchaine, 2001; Porges, 1995). Due to phylogenetic development, humans have evolved a more sophisticated system to enrich behavioral and affective responses to an increasingly complex environment (Porges, 2001). The more evolved, ventral branch of the vagus, emanates from the nucleus ambiguous and is also known as the 'smart vagus' because it is associated with the regulation of fight-or-flight behaviors and is linked to social communication and self-soothing behaviors (Beauchaine, Gatzke-Kopp, & Mead, 2007). Specifically it allows for sustained attention and engagement with the environment via suppression of sympathetic influence on the heart. These functions follow a phylogenetic hierarchy, where the most primitive systems are activated only when the more evolved structures fail. These neural pathways regulate our autonomic states and the expression of emotional and social behavior. Thus, according to this theory, physiological state dictates the range of behavior and psychological experience.

The smart vagus exerts an important influence on cardiac functioning. According to the model, in non-threatening situations the more evolved vagus branch sends constant inhibitory signals to the heart, acting as a brake on heart rate and allowing individuals to engage in tasks that require minimal mobilization of the sympathetic nervous system. Increased vagal control, or any activity away from baseline, is referred to as vagal reactivity (RSA reactivity). In threatening or distressing situations however, vagal influence rapidly decreases, there is little inhibition to the heart and rapid mobilization (flight/flight) can be activated and individuals revert to phylogenically older responses like freezing, or the sympathetically activated flight-or-fight response. This decrease in vagal influence is referred to as vagal withdrawal.

The measurement of RSA in humans has become a novel index of emotional vulnerability and reactivity and is frequently examined in populations with affect regulation difficulties. RSA is a measurable, noninvasive way to examine how the vagus modulates heart rate activity in response to stress. RSA refers to rhythmic fluctuations in heart rate associated with respiration, which result from activity of the vagus. During inspiration, vagal activity is attenuated and heart rate accelerates; during expiration vagal activity is reinstated, causing heart rate to slow. Research has demonstrated that RSA is primarily determined by the activity of the 'smart vagus' (Berntson et al., 1997; Porges, 1997), which, as stated earlier, evolved to facilitate the complex emotion responses and social behavior seen in humans. Thus, measures of RSA are thought to provide a noninvasive window into the relationship between the evolutionarily recent vagal system and affective experience.

There are two ways in which RSA is measured and linked with emotional responding: basal RSA and RSA reactivity. Basal RSA refers to levels of RSA at baseline when an individual is at a resting state. RSA reactivity refers to changes in RSA from a baseline resting state to an emotionally arousing state. Basal RSA is hypothesized to represent an individual's general capacity for emotion regulation, while RSA reactivity to stress or emotionally evocative stimuli is hypothesized to represent in-vivo regulatory capacities (Beauchaine, 2001). Given this distinction, basal RSA well characterizes the emotional vulnerability proposed in the developmental theory of BPD. Moreover, individual differences in basal RSA are associated with emotion regulation capabilities, with low RSA conferring risk for psychopathology and high RSA buffering against risk (Beauchaine, 2001; Katz & Gottman, 1997; Shannon, Beauchaine, Brenner, Neuhaus, &

Gatzke-Kopp, 2007). Research consistently demonstrates that high basal RSA in children and adolescents is a marker for better emotion regulatory capacities, and it has been associated with positive affect, coping, and social competence and inversely related to negative affect (Eisenberg et al., 1996; Fabes, Eisenberg, & Eisenbud, 1993; Mezzacappa et al., 1996). By contrast, low basal RSA has been observed across numerous populations characterized by poor emotion regulation, including severe conduct problems (Beauchaine et al., 2007), nonsuicidal and suicidal self-injury (Crowell et al., 2005), trait hostility (Sloan, Shapiro, Bigger, Bagiella, Steinman & Gorman, 1994), and both depression and anxiety disorders (Lyonfelds et al., 1995; Rechlin et al., 1994; Rottenberg et al., 2003; Thayer et al., 1996; Yeragani et al., 1993).

Basal RSA and Borderline Pathology. Only a few studies to date have examined RSA as a vulnerability for BPD. Instead, most studies have looked at other indices of physiological responding related to sympathetic activation, such as heart rate (HR) or skin conductance responses (SCRs), with mixed findings. For example, Elices and colleagues (2012) found that individuals with BPD showed smaller increases in HR in response to films designed to elicit fear, anger and sadness, versus healthy controls. Additionally, Kuo and Linehan (2009) found that individuals with BPD exhibited more SCRs during a resting baseline period versus healthy controls, but not versus individuals with social anxiety disorder (SAD).

A few studies have, however, examined RSA in individuals with BPD. A study by Kuo et al. (2016) found that adult males diagnosed with BPD exhibited reduced basal RSA versus healthy controls. Similarly, Kuo and Linehan (2009) found reduced basal RSA in BPD participants versus individuals with SAD and healthy controls.

Emotional Vulnerability Should Predict Borderline Features in Adolescence

In general, studies among adults diagnosed with BPD lend support for the association between trait NA and BPD. Therefore, it is likely that trait NA should predict borderline features in a young adolescent sample. The literature on the relationship between basal RSA and BPD also lends support to the hypothesis that low basal RSA is a likely risk factor for the development of BPD. Therefore, basal RSA should also predict borderline features in my adolescent sample.

Emotion Dysregulation as a Mediator

According to the biosocial model, the mechanism by which early emotional vulnerability (trait NA and RSA) and BPD are linked is emotion dysregulation. Thus, children with high trait-NA and/or low basal RSA who display frequent and intense emotional reactions are more likely to become adolescents and subsequently adults who are unable to modulate their emotional experiences. The field of emotion regulation examines how individuals experience, express and manage their emotions (Gross, 1998) and emotion regulation has been defined by Gross (1998, p. 275) as "the processes by which individuals influence which emotions they have, when they have them, and how they experience and express these emotions." The ability to regulate emotions effectively is essential to well-being (Mayer, Salovey, & Caruso, 2004) and mental health (Kring & Werner, 2004), and has been linked to better physical health (Carre, Mittmann, Woodin, Tabares, & Yoshimoto, 2005; Kubzansky & Thurston, 2007), the experience of increased positive affect (Gross & John, 2003), as well as higher levels of relationship satisfaction (Levenson, Carstensen, & Gottman, 1994; Wachs & Cordova, 2007). In contrast, individuals who lack emotion regulation skills or who use ineffective emotion regulation

strategies are more likely to be more emotionally reactive (Gross & Levenson, 1997) and experience various forms of psychopathology including BPD (Aldao, Nolen-Hoeksema, & Schweizer, 2010; Gross, 1998; Southam-Gerow & Kendall, 2002).

Emotion dysregulation refers specifically to the inability to control and modulate one's affective arousal to such a degree that emotions become out of control. During states of intense negative or aversive emotional arousal individuals often lose track of their long term goals, have reduced capacity for problem solving or thinking and tend to engage in behaviors aimed at reducing their negative arousal, regardless of negative consequences (Linehan & Heard, 1992; Shedler & Westen, 2004).

According to the biosocial theory (Linehan, 1993) emotion dysregulation in BPD involves (1) high baseline sensitivity (i.e., emotions that are too intense or easily triggered), and (2) greater reactivity to stressors (i.e., deficient control or modulation of the emotional response once it has begun) and 3) a slow return to baseline functioning. A significant amount of research has demonstrated that individuals with BPD have difficulties with emotion regulation and that their emotional responses are dysregulated (Salsman & Linehan, 2012). According to the literature, individuals with BPD tend to use maladaptive emotion regulation strategies (e.g., suppression), and self-reported emotion dysregulation has been consistently linked to BPD (Glenn & Klonsky, 2009).

Self-Reported Dysregulation. Emotion dysregulation is often assessed via an individual's subjective report on their experience. For example, individuals with BPD are exposed to a stressful or emotionally evocative stimuli and then asked to rate their emotional experience in some way. Studies that assess dysregulation in this way have consistently found that individuals with BPD report more negative emotional intensity in

general (Ebner-Priemer et al., 2007; Henry et al., 2001; Koenigsberg et al., 2002; Levine, Marziali, & Hood, 1997), but findings are mixed on whether they report more distress or dysregulation in response to stress (Russell, Moskowitz, Zuroff, Sookman, & Paris, 2007). For example, Kuo and Linehan (2009) looked at self-reported emotion dysregulation before and after exposing participants with BPD, SAD and healthy controls, to emotional film clips. They found that individuals with BPD reported higher emotion dysregulation at baseline versus individuals with SAD or healthy controls. However, they did not find differences in self-reported emotion dysregulation during or after sad film induction. Gratz and colleagues (2010) examined emotional responding in adults diagnosed BPD versus healthy controls in response to a standard arithmetic stressor task (the PASAT-C) and receiving negative feedback about their performance on the PASAT-C. Between BPD and healthy controls, no differences were found in anxiety, hostility or irritability from baseline to stressor. However, within the BPD group, differences emerged specifically for the emotion of shame from the stressor period to receiving negative feedback. This study suggests that emotional responding in individuals with BPD may in fact be more nuanced and that individuals with BPD not have difficulty regulating all emotions. Instead, there may be context-dependent and/or emotion-specific dysregulation in BPD.

RSA Reactivity. Although much literature exists that examines emotion dysregulation via self-report methods, fewer studies examine the biological basis of emotion dysregulation. As outlined earlier, experience of emotions and the process by which they are regulated involves an essential physiological component. While basal measures of RSA can be understood as an individual's basic capacity for emotion

regulation, the primary role of the vagus is to respond flexibly to environmental demands and regulate SNS activity. Thus, RSA reactivity in response to stress or an emotionally charged event may represent a more meaningful measure of emotion regulation or dysregulation.

In terms of RSA reactivity, much research with infants, children, and adults has found decreases in RSA during negative emotional experiences (Beauchaine, 2001; Friedman & Thayer, 1998; Gottman et al., 1995; Thayer et al., 1996). More specifically, vagal withdrawal occurs in response to a variety of emotional stimuli including sad film clips (Rottenberg, Salomon, Gross, & Gotlib, 2005), anxiety-provoking public speaking tasks (Rottenberg, Clift, Bolden, & Salomon, 2007), and mental arithmetic tasks designed to cause frustration (Mezzacappa, Kelsey, Katkin, & Sloan, 2001). Decreased RSA in response to stress can signify the inability to appropriately engage or disengage defense systems (Porges, 2004). Atypical vagal influence has been related to multiple emotional disorders and emotional states, including depression (Carney, Saunders, Freedland, Stein, Rich, & Jaffe 1995; Rechlin et al., 1994; Rottenberg et al., 2003), anxiety (Lyonfields et al., 1995; Thayer et al., 1996), worry (Hofmann, Moscovitch, Litz, Kim, Davis, & Pizzagalli, 2005), self-injury (Crowell et al., 2005), stress (Allen & Crowell, 1989) and BPD (Austin et al., 2007; Weinberg et al., 2009). High vagal control, indexed by increased RSA in response to stress, on the other hand, has been associated with enhanced ability to cope with life stressors (Fabes & Eiseinberg, 1997).

RSA Reactivity and BPD. A study by Austin and colleagues (2007) provided the first documented evidence of autonomic nervous system differences between controls and individuals diagnosed with BPD. RSA was monitored during the presentation of film

clips of varying emotional content. Findings showed no differences between individuals with BPD and controls in terms of basal RSA, however during the experiment the groups exhibited contrasting trajectories. Individuals with BPD displayed decreasing RSA, whereas individuals in the control group displayed increasing RSA. By the end of the experiment, the groups differed significantly on RSA. From the perspective of Porges' polyvagal theory this suggests that the BPD group ended in a physiological state that supports the mobilization behaviors of fight and flight, while the control group ended in a physiological state that supports social engagement behaviors.

Weinberg and colleagues (2009) attempted to replicate Austin and colleagues study (2007) and examined ANS functioning before, during, and following a social stressor task. In this study, contrary to Austin and colleagues findings, individuals with BPD displayed reduced basal RSA compared to controls. Moreover, the two groups showed different RSA trajectories, with the BPD group characterized by reduced RSA. Group differences, however, were not statistically significant. The researchers stated that the trend in their study might have become significant if their emotional stressor had continued for longer so that it was the same length as the Austin et al., study. Moreover, BPD participants reported more negative affect in response to the stressor task versus controls.

Kuo and Linehan (2009) also examined RSA reactivity in response to emotional films in among young adults diagnosed with BPD compared to individuals diagnosed with SAD and healthy controls. Contrary to what was expected, they found that individuals with BPD exhibited a significant increase in RSA from basal to sad film induction period, whereas the SAD group exhibited decreasing RSA. This study also

examined sympathetic activation and found that individuals with BPD did not exhibit a change in SCRs from baseline to sad film period whereas the healthy controls and SAD individuals exhibited increased SCRs in response to the emotional film.

Gratz and colleagues (2013) examined dysregulation via high frequency heart rate variability (HF HRV) which is a similar index to RSA in that high HF HRV is related to increased parasympathetic control and low HF HRV is related to poor regulator control. They exposed individuals with BPD (with and without avoidant personality disorder (APD)) and healthy controls to the PASAT. At baseline, they found no differences in HF HRV between groups. However, when they examined the change in HF HRV from baseline to the most difficult level of the PASAT they found that BPD individuals (with co-morbid APD) displayed decreased HF HRV (indicative of emotion dysregulation or poor emotion regulation capacity) versus the healthy controls who exhibited a slight increase in HF HRV in response to the stressor. In this study researchers also assessed emotion dysregulation via self-report indices and found that individuals with BPD (with and without co-morbid APD) reported using more dysregulated emotion regulation strategies versus healthy controls.

Weinberg and colleagues (2009) also examined RSA in BPD individuals in response to a lab stressor. Similarly to other studies, BPD participants exhibited significantly lower RSA at baseline, as well as during the stressor. RSA however neither increased nor decreased from baseline to stressor, whereas the control group exhibited a non-significant increase in RSA from baseline to stressor.

Other studies have failed to find differences in physiological responding in individuals with BPD. Kuo and colleagues (2016) did not find different patterns of RSA

responsivity when adults with BPD versus healthy controls were exposed to negative stimuli. In this study however, sympathetic activation as indexed by Cardiac Sympathetic Index (CSI; Toichi et al., 1997) did show differences between groups. For BPD participants, CSI was higher during baseline, stressor and recovery versus controls. Additionally for individuals with BPD, CSI increased from the first half to the second half of the stressor, whereas CSI decreased from first to second half of the stressor in the control group, suggesting less arousal.

Emotion Dysregulation and BPD. According to the existing literature, the relationship between self-reported emotion dysregulation and BPD is well supported. The physiological literature examining the relationship between RSA reactivity and BPD, however, is more mixed. Some studies have found expected patterns of reactivity with individuals with BPD exhibiting RSA reactivity (reduced RSA) in response to stress. Other studies have not found this pattern. However, I predicted that emotion dysregulation in the form of both self-reported dysregulation and RSA reactivity would predict increased borderline features. I also hypothesize that emotion dysregulation (self-reported dysregulation and RSA reactivity) will mediate the relationship between emotional vulnerability and borderline features.

Parental Invalidation as a Moderator

Thus far I have discussed the predictive role of trait NA and basal RSA on borderline pathology and the mediating role of emotional dysregulation as evidenced both by self-reported dysregulated strategies, and by RSA reactivity on the relationship between emotional vulnerability and borderline features. However, according to the biosocial model, emotion dysregulation and resultant borderline pathology arises in the

presence of an invalidating context. The following section reviews the literature linking invalidation with BPD as well as literature examining the effects of invalidation on emotion regulatory capacities. This will elucidate the role of invalidation as a moderator of the relationship between emotional vulnerability and emotion dysregulation in predicting borderline pathology.

A large body of existing research focuses on emotion-related parenting practices that are believed to contribute to the development of mental health problems in offspring. This literature suggests that how parents discuss and respond to their children's emotions play a key role in the children's subsequent perception, expression, and regulation of emotions (Eisenberg, Cumberland & Spinrad, 1998, Eisenberg et al., 2001). Specifically, positive parental responses to child emotions have been linked with positive emotional and social outcomes for children, and include parental responsiveness to distress and encouragement of emotional expression (Field, 1994; Roberts, 1999; Roberts & Strayer, 1987). Alternatively, negative parental responses to children's emotional expressions encourage the suppression of emotion or the use of avoidant, aggressive and/or dysfunctional emotion regulation strategies (Buck, 1984; Eisenberg et al., 1996; Roberts & Strayer, 1987). These negative responses are associated with a variety of social and emotional problems in children (Gottman, Katz, & Hooven, 1996) as well as with avoidant-insecure attachments (Cassidy, 1994).

One specific type of negative parental responses to child emotional displays has been termed "invalidating responses" (Linehan, 1993). Invalidation occurs when individuals responsible for taking care of a child (typically parents) repeatedly criticize, trivialize, punish, reject or erratically reinforce their child's communication of internal

experiences, such as their thoughts and feelings. More specifically, invalidating responses involve high negative emotion (e.g., disgust, contempt, condescension, or other emotions associated with disrespect), high levels of negative judgment (e.g., the child's feelings, desires, actions, or thoughts are "wrong"), or illegitimatizing the child's valid experiences (Linehan, 1993). Invalidation also refers to responding to dysfunctional child behaviors with support or acceptance (i.e., to validating unacceptable behaviors or displays of emotion). According to Linehan, validating responses from the environment guide children in learning how to label, control, and understand their emotions and emotional reactions. However, pervasive invalidation promotes dysfunctional emotion regulation capacities, ultimately, undermining the development of a cohesive self-identity, emotion regulation, and behavioral control characteristic of BPD.

Invalidation and BPD. The etiological role of pervasive invalidation has been implicated in the development of adolescent emotional dysregulation, depression (Yap, Allen, & Ladouceur, 2008) dysfunction in romantic relationships in adults with BPD (Selby, Braitwaite, Joiner, & Fincham, 2008), and has also been associated with higher rates of impulsivity, interpersonal sensitivity, and aggression (Cheavens et al., 2004). Significant research supports the relationship between invalidating environments and the development of BPD (Dixon-Gordon, Whalen, Scott, Cummings, & Stepp, 2016; Dubo, Zanarini, Lewis, & Williams, 1997; Fruzzetti et al., 2005; Johnson, Cohen, Chen, Kasen, & Brook, 2006; Sauer & Baer, 2010; Selby, Braithwaite, Joiner, & Fincham, 2008; Stepp, Whalen, Pilkonis, Hipwell, & Levine, 2012; Zanarini & Frankenburg, 2007). Empirical evidence also demonstrates a link between invalidation and emotion regulation difficulties during childhood (Eisenberg et al., 1998). For BPD, the combination of being

emotionally vulnerable and chronically invalidated is most likely to result in escalating, dysregulated emotion and the kind of pervasive emotion dysregulation associated with BPD.

Hong, Ilardi and Lishner (2011) examined the impact of childhood invalidation and childhood sexual abuse (CSA) and borderline symptoms in college students. They found that experiences of childhood invalidation directly predicted borderline symptoms whereas CSA alone did not. They also found an interaction between CSA and invalidation creating a synergistic effect that culminated in the development of borderline pathology. In their study regardless of the occurrence of CSA, early experiences of invalidation were predictive of higher levels of psychopathology. Sturrock, Francis and Carr (2009) also examined the association between childhood invalidation, distress tolerance and borderline symptoms. Their findings were consistent with the prevailing theories; reports of maternal invalidation in childhood were associated with current levels of self-reported BPD symptomatology and poor distress tolerance (which can be understood as an index of emotion dysregulation) mediated the relationship between invalidation and borderline symptoms.

Stepp and colleagues (2014) assessed children ages 5-8 and their parents over a 9-year period and found moderate associations between child BPD symptoms and increases in harsh parenting behaviors. Specifically, BPD symptoms and harsh parenting practices were related such that fluctuations in BPD symptom severity were directly related to changes in harsh parenting behaviors across a period of nine years. Additionally, the authors demonstrated a reciprocal association between adolescent BPD symptoms and adolescent-reported harsh punishment and low caregiver warmth.

Invalidation and Emotion Dysregulation. There is also a small body of literature examining the relationship between invalidation and physiological indices of emotion dysregulation. Woodberry, Gallo and Nock (2008) examined physiological arousal in response to in-vivo invalidation among young women with high levels of BPD symptoms. Women with BPD symptoms and controls were assigned to hear either a validating ("Most people find this set of anagrams really frustrating" spoken in a warm tone) or invalidating comment ("There's no need to get really frustrated. They're just anagrams." spoken in a puzzled, mildly critical tone) over the intercom, during a frustrating unsolvable anagram task. In this study, physiological arousal was measured via SNS reactivity as evidenced by skin conductance level (SCL). Interestingly, the researchers did not find significantly greater increases in arousal in response to invalidation in individuals with BPD symptoms as compared to controls. Subjectively however, they found that individuals with BPD symptoms reported a relatively more negative initial response to invalidation when compared to validation and to controls; the BPD features group reported feeling significantly less positive and less comfortable with their emotions across comments in spite of no significant differences in physiological arousal. These null findings are consistent with other research that has failed to demonstrate direct physiological evidence of hyperarousal measured via SNS activity in individuals with BPD (e.g., elevated skin conductance response (Herpertz, Kunert, Schwenger, Sass, 1999) typically associated with heightened affective intensity (Lang, Bradley & Cuthbert, 1998). These results however should also be interpreted with caution due to the small sample size (N=41).

A more recent study by Shenk and Fruzzetti (2011) assessed the psychophysiological impact of validating versus invalidating responses on patterns of emotional reactivity in a sample of 60 college students. The researchers hypothesized that under stressful conditions individuals who received invalidating responses would demonstrate higher degrees of emotional reactivity as evidenced by greater emotional (self-reported negative affect) and physiological (heart rate and SCLs) responses when compared to individuals who received validating responses. Results indicated that condition had an effect of self-reported negative affect such that participants in the invalidating condition reported significantly higher negative affect throughout the study when compared to those participants in the validating condition. Condition also had an effect on heart rate (participants in the invalidating condition had significantly increased heart rate in comparison to participants in the validating condition) and SCL (participants in the invalidating condition displayed significantly higher SCL in comparison to those individuals in the validating condition). Their findings demonstrated that validating and invalidating responses produced distinct trajectories of emotional reactivity across subjective and physiological (SNS) measures, which is consistent with current models of emotion regulation and dysregulation (Gross, 1998; Linehan, 1993).

Taken together, the literature linking invalidation and BPD provides abundant support for the biosocial model and suggests the crucial impact of invalidation on the development of borderline pathology. Additionally, there is some support for the impact of invalidation on autonomic functioning as evidenced by SNS reactivity. Thus, invalidation appears empirically to impact emotional reactivity. Currently no studies have investigated the relationship between invalidation and PNS functioning. However, given

preliminary support for the relationship between SNS reactivity and invalidation, invalidation is likely linked to the emotion regulatory functions of the vagus. Thus, invalidation may represent a potential moderator in the prediction of borderline pathology via its influence on the development of an individual's capacity for emotion regulation.

The Current Study

My dissertation sought to empirically test Linehan's biosocial model examining the process of emotion dysregulation in the development of borderline pathology. In particular, the relationships between emotional vulnerability, invalidation, and emotion dysregulation were examined as they predicted borderline features in a community sample of young adolescents. While extant research has established relationships among some of these variables, none of them have been examined in a developmental sample nor used the regulatory index of RSA. My conceptual model is shown in Figure 1 and examined the following hypotheses:

1. Emotional vulnerability (trait NA and basal RSA) would predict adolescent borderline features (concurrently and prospectively) such that adolescents high in trait NA or low in basal RSA would display increased borderline features.
2. The relationship between emotional vulnerability (trait NA and basal RSA) and adolescent borderline features (concurrently and prospectively) would be mediated by emotion dysregulation (self-reported emotion dysregulation and RSA reactivity).
3. Parental invalidation would moderate the effects of emotional vulnerability on emotion dysregulation.

a. Adolescents with high trait NA who experience high levels of invalidation would evidence the most borderline features (concurrently and prospectively).
b. Adolescents with low basal RSA who experience high levels of invalidation would evidence the most borderline features (concurrently and prospectively).

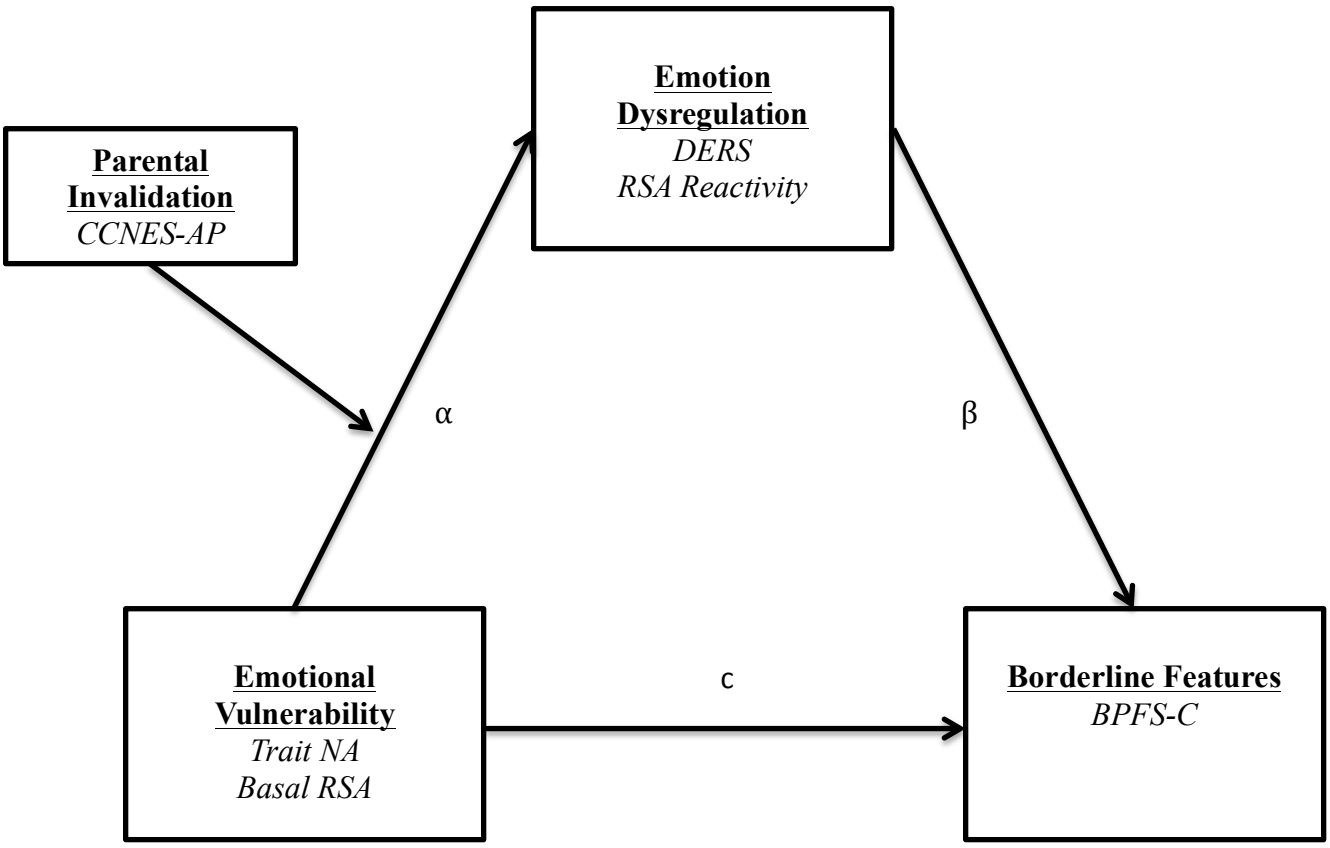

Figure 1. Conceptual model of emotion dysregulation mediating the relationship between emotional vulnerability and borderline features in adolescence, and the moderating role of invalidation on the relationship between emotional vulnerability and emotion dysregulation.

CHAPTER II

Method

Sample and Participant Selection

Eligibility. My study was part of a larger federally funded research study[1]. For the federally funded research study a community sample of never depressed youth was recruited. For this larger study, youth were recruited from public and private middle school classrooms in a metropolitan area in the Pacific Northwest. Youth between ages 10 and 14 years were recruited through brief in-class presentations from graduate students and through emails and newsletters directed at these student's parents. Youth completed an initial screening to assess current depression symptoms before being invited for full participation and the laboratory visit. Current depressive symptoms were assessed from child report on the Children's Depression Inventory 2nd Edition (CDI; Kovacs, 2011). Youth who were above the clinical cutoff for depression on the CDI, a score of fourteen or higher, received reimbursement for their completion of the initial screener but did not participate in further data collection. If the youth had a CDI score of thirteen or lower, their parents were contacted by phone to be interviewed about the youth's current medications. Some forms of psychiatric medications (specifically, stimulant medications) can interfere with physiological measurement and excluded the youth from participating. If these criteria were all met, participants were invited to complete the laboratory visit portion of the study. Therefore, for inclusion in the current study, all youth had a CDI score below 14.

National Institute of Mental Health: *Cognitive and Physiological Pathways from Temperament to Adolescent Depression*, PI: Amy Mezulis.

Power Analysis. From my review I could find no standardized methods for *a priori* estimates of sample size for moderated mediation models. To insure that the analyses were not underpowered, the statistical technique of bootstrapping was utilized. Bootstrapping uses the available data to take thousands of random sample observations to create an approximate sampling distribution. This method is non-parametric, does not require normally distributed data, and increases power to detect moderate effect sizes with smaller samples (Fritz & MacKinnon, 2007; Mallinckrodt, Abraham, Wei, & Russell, 2006). Efron and Tibshirani (1993) have found bootstrapping to be useful for studies utilizing small or moderate sample sizes ranging between 20 and 80 participants. Rucker and colleagues (2011) recommend that when using bootstrapping, moderate sample sizes are preferable. Simulations have found that, with power set at 0.80, a sample size of 71 is sufficient to find significant mediation when the α and β paths are moderately related (Fritz & MacKinnon, 2007). Based on this information, the current study aimed to recruit a total of 100 participants.

Participants. A total of 322 adolescents in grades 5-8 completed the screening phase of the federally funded study. Of this group, 40 youth exceeded the clinical cut-off for depression on the CDI and were not eligible for the remainder of the federally funded study. Therefore, they were excluded from this current study as well. From the 219 youth that were eligible, 111 youth completed the baseline and follow-up measures relevant to this study. No psychophysiological data was collected on two participants due to child discomfort with the protocol. Eight adolescents had unusable RSA data due to electrode or other recording errors. This resulted in a final sample size of 101.

There was minimal missing data on all self-report measures. Across all measures, to be considered a valid scale score no more than three items could be omitted from a specific scale. There were no instances where this occurred on any measure and all youth had complete data. This resulted in a final sample size for all analyses of 101 youth. All measures were reviewed for potential outliers along with tests of Skewness and kurtosis. Table 1 contains the final summary of the tests of Skewness and kurtosis, along with the means and standard deviations of the study variables.

Table 1. *Means, Standard Deviations, Ranges, Cronbach's Alpha, Skew, and Kurtosis for Study Variables*

Variable	*N*	*M*	*SD*	Min.	Max.	α	Skew	Kurtosis
1. Sex	101	53% female	-	-	-	-	-	-
2. Age	101	12.82	0.83	11.35	14.55	-	0.35	-0.88
3. Trait-NA	101	2.70	0.51	1.36	3.88	0.79	-0.25	-0.10
4. Basal RSA	101	7.10	0.98	4.10	9.64	-	-0.12	0.33
5. P Invalidation	101	2.54	0.99	1.00	4.89	0.88	0.32	-0.67
6. DERS	101	68.75	17.62	36	119	0.90	0.37	-0.26
7. RSA Reactivity	101	-0.56	0.60	-2.42	0.83	-	-0.39	0.42
8. RSA Stress	101	6.49	0.98	2.80	9.20	-	-0.25	1.61
9. BPFS-C at baseline	101	52.70	9.99	32	79	0.86	0.21	-0.45
10. BPFS-C at 6-month follow-up	101	48.23	11.26	26	81	0.90	0.60	-0.02

Note. Temperamental Trait-NA (Trait-NA), Dysregulated Emotion Regulation Strategies (DERS), P Invalidation (Parental Invalidation), Borderline Personality Features Scale in Children (BPFS-C).

The final sample size of 101 youth was maintained across all analyses. Participants in the study were younger adolescents (M=12.82, SD=0.83). Females (53%) were somewhat overrepresented in comparison to males in the sample. In terms of race and ethnicity, 71.3% of participants identified as Caucasian/White, 0.9% identified as Black/African-American, 6.1% identified as Asian, 0.9% identified as Hispanic/Latino,

6.1% identified as biracial/more than one reported race, and 3.5% identified as unknown/not reported.

Procedure

Participants were recruited via in-class presentations and emails to parents of 5th through 8th grade children attending participating schools. Once the adolescent had assented and parents consented to screening, adolescents completed screening questionnaires including youth report of trait NA. Researchers working directly with the adolescent were blinded to the results of these initial screening measures. Once adolescents were found eligible for participation in the full study, they were scheduled for a laboratory visit. At this visit, adolescents reported on parental invalidation, emotion regulation difficulties and baseline borderline features. Participating adolescents were then hooked up to physiological measuring equipment by a same-sex experimenter and run through a paced computerized paradigm. Adolescents were told that the purpose of the experiment was to monitor their body's reactions while their problem-solving abilities were tested through a computerized word activity. Before physiological measurement began, adolescents were told to avoid unnecessary movement and talking during the experiment to reduce interference on physiological measurements. Adolescents also received some instruction on and practice of anagrams to insure that they understood the instructions for the computerized word activity, which functioned as the stressor task. The paradigm included a two-minute baseline measurement period, and a five-minute stressor task. The baseline measurement period involved the youth sitting quietly at the computer as neutral nature scenes were presented; the basal RSA measurement was collected during this time. For the stressor task twenty anagrams were presented with a

timer at the top of the screen counting down from five minutes. Of the twenty anagrams, only five of them were solvable and the other fifteen were designed to be unsolvable. Unsolvable anagrams are a common task used for stress induction in studies of physiological reactivity of vagal tone (Mezulis & Rudolph, 2012; Reynard, Gevirtz, Berlow, Brown, & Boutelle, 2011; Zuroff, Mongrain, & Santor, 2004). RSA data continued to be collected during the stressor task time period. After completing these tasks the participant were debriefed about the true purpose of the study, to observe their response to a stressful situation, and informed that not all of the anagrams were solvable. Participants were observed by experimenters throughout the study and were allowed to request to discontinue the study at any time. If a participant appeared overly distressed during the study, experimenters evaluated whether the youth should continue with the study. No participants discontinued the study for this reason.

Six months post the lab visit all participants were contacted via phone and invited to return to assess changes in borderline features. All 101 adolescents and their parent returned to complete the follow-up portion of the study and reported on borderline features at that time.

Measures

Trait NA. Emotional vulnerability was assessed with the NA superscale of the Early Adolescent Temperament Questionnaire-Revised (EATQ-R; Ellis & Rothbart, 2001). The NA superscale is composed of 21 self-report items, and contains the temperament traits of *frustration* (e.g., "It bothers me when people are slow about getting ready for things"), *shyness* (e.g., "I am very shy"), and *fear* (e.g., "I feel scared when I enter a darkened room at home"). Adolescents were asked to rate items on a 5-point

Likert scale ranging from 1 (*almost always untrue of me*) to 5 (*almost always true of me*), with higher scores indicating greater trait-NA. Total trait-NA scores were obtained by computing a weighted average across relevant items. Previous research has provided evidence of good internal reliability of the instrument, with Cronbach's alphas varying between 0.65 and 0.82 (Ellis & Rothbart, 2001). In the current study, the NA superscale demonstrated adequate reliability of $\alpha = .79$.

Basal RSA. Emotional vulnerability was also assessed physiologically, via adolescents' cardiac activity. Adolescents' cardiac activity was recorded throughout a 4-minute seated resting baseline. All recordings occurred in the same laboratory suite with standardized temperature and lighting. Participants were asked to refrain from use of caffeine and stimulant medication for 36 hours prior to the laboratory session, and oral confirmation of their adherence to this protocol was obtained from both parent and adolescent upon arrival. Only two adolescents were currently using other medications (1 SSRI and 1 antihistamine). Medication use did not impact findings and thus was not controlled for in subsequent analyses. Disposable pre-gelled Ag/AgCl electrodes were placed on their chests and abdomens using a Lead II placement. Electrocardiograph (ECG) data were acquired continuously using Biopac MP150 Data Acquisition Unit (Goleta, CA) and sampled at 1000 Hz. ECG data were processed offline using Mindware Technologies HRV 3.0.10 analysis program (Gahanna, OH). Data were visually inspected for movement artifacts or incorrect placement of markers by the automated scoring algorithm and corrected as needed by trained graduate research assistants. The resulting inter-beat interval time series was subjected to a fast Fourier transformation by the Mindware software, and power in the respiratory frequency band (.15-.40 Hz) was

derived from the spectral density function. Respiration rates were examined and all fell within the expected range, therefore I elected not to control for respiration. RSA values were extracted in 30-second epochs. The average RSA value across the four minutes of the resting baseline was used to create a single basal RSA score. Altogether, there was RSA data available for 101 children. Range and mean value for basal RSA were consistent with published literature for community developmental samples (see Table 1; Zisner & Beauchaine, in press).

Self-Report Emotion Dysregulation. Self-reported emotion dysregulation was assessed with the Difficulties in Emotion Regulation Scale (DERS; Gratz & Roemer, 2004). The DERS is a 36 item self-report questionnaire that assesses clinically relevant difficulties in emotion regulation. Items are scored on six scales: Lack of Emotional Awareness (e.g., I pay attention to how I feel), Lack of Emotional Clarity (e.g., I am clear about my feelings), Difficulties Controlling Impulsive Behaviors when Distressed (e.g., When I'm upset I become out of control), Difficulties Engaging in Goal-Directed Behavior When Distressed (e.g., When I'm upset I have difficulty getting work done), Nonacceptance of Negative Emotional Responses (e.g., When I'm upset I become angry with myself for feeling that way), and Limited Access to Effective Emotion Regulation Strategies (e.g., When I'm upset I believe there is nothing I can do to make myself feel better). Items are scored on a five-point scale ranging from 1 (*almost never*) to 5 (*almost always*). Previous research has provided support for the reliability of a total DERS score within both clinical (e.g., Fox et al., 2007; Gratz et al., 2008) and nonclinical populations (e.g., Gratz & Roemer, 2004; Johnson et al., 2008). Total scores were obtained by summing all items so that higher scores indicate greater difficulties in emotion regulation

(i.e., greater emotion dysregulation). In the current study the DERS demonstrated strong reliability of $\alpha = .90$.

RSA Reactivity. Emotion dysregulation was also assessed physiologically, via adolescents' cardiac activity. Using the same procedure as described above (see section on Basal RSA), adolescents' cardiac activity was assessed during a 5-minute stressor task (stressor task described in detail above under Procedure section). RSA values were extracted in 30-second epochs. The average RSA value across the seven minutes of the stressor was used to create a single RSA stress score. Adolescent's basal score was subtracted from their RSA stress score from to obtain a change score. This change score is termed RSA reactivity, and represents the change in RSA from basal to stress, with negative values indicating greater RSA withdrawal (dysregulation) during stress. Altogether, there was RSA Reactivity data available for 101 children. Range and mean value for RSA Reactivity were consistent with published literature for community developmental samples (see Table 1; Zisner & Beauchaine, in press).

RSA Stress. As described above in the RSA Reactivity section, the average RSA value across the seven minutes of the stressor was used to create a single RSA stress score. This RSA stress score was also used as a measure of emotion dysregulation to the laboratory stressor. Higher values of RSA stress indicated greater regulation, whereas lower scores indicated poorer regulation, vagal withdrawal, and more emotion dysregulation. Range and mean value for RSA stress were consistent with published literature for community developmental samples (see Table 1; Zisner & Beauchaine, in press).

Parental Invalidation. Parental responses to their adolescent's negative emotions were assessed with the adolescent self-report form of the Coping with Children's Negative Emotions Scale – Adolescent Perception Version (CCNES-AP; Fabes & Eisenberg, 1998). The CCNES-AP is a self-report measure in which adolescent's respond to 12 hypothetical situations in which they experience distress (e.g. "When I get down because I had a bad day, my parent usually…"). Adolescents indicated the likelihood that their parent would display each of the six possible responses to the situation ranging from 1 (*very likely*) to 7 (*very likely*). The measure yields 6 subscales: problem-focused reactions (e.g. "helps me think of things to do to get my problem solved"), emotion focused reactions (e.g. "tries to get me to think of good things that happened"), expressive-encouragement reactions (e.g., "listens to me talk about my feelings"), minimization reactions (e.g. "tells me that I really have nothing to be sad about"), punitive reactions (e.g. "tells me to straighten up and stop sulking around the house"), and distress reactions (e.g. "becomes obviously uncomfortable when s/he sees I am feeling down"). The CCNES-AP yields two composite scales supportive (problem-focused, emotion-focused, expressive encouragement) and nonsupportive/invalidating (minimization, punitive, dismissive). Only the invalidation scale was used in the current study and invalidating responses were calculated as averages of the subscales (DeBoard-Lucas et al., 2010; Fabes et al., 2001, 2002; Nelson et al., 2009). Total invalidation scores were obtained by computing a weighted average across relevant items with higher scores indicating greater levels of invalidation. Adolescents were allowed to choose the parent reported on; most chose to report on their mother (80%) but some chose to report

on their father (19.1%). In the current study the invalidating scale of the CCNES-AP demonstrated adequate reliability of α = .88.

Borderline Features. Borderline personality features were measured using the Borderline Personality Features Scale for Children (BPFS-C: Crick, Murray-Close and Woods, 2005). The BPFS-C is a modified version of the Borderline Features scale of the Personality Assessment Inventory (PAI; Morey, 1991), a reliable and valid instrument used to assess borderline personality features among adults. It was developed for use with children ages nine and older. It consists of age-appropriate items adapted from the original PAI to reflect four domains: affective instability, identity problems, negative relationships, and self- harm. It includes 24 items rated on a 5-point Likert-type scale with responses ranging from 1 (*not at all true)* to 5 (*always true*). Example items include: "My feelings are very strong. For instance, when I get mad, I get really, really mad. When I get happy, I get really, really happy" and "I feel that there is something important missing about me, but I don't know what it is." Scores are summed to yield a total borderline personality features score, with higher scores indicating greater levels of borderline features. The BPFS-C has demonstrated good internal consistency in community samples (α = .76; Crick, Murray-Close and Woods, 2005). In the current study BPFS-C demonstrated adequate reliability at baseline (α = .86) and the 6-month follow-up (α = .90).

CHAPTER III

Results

Data Screening and Analysis

Prior to analysis, I inspected data for missing values, outliers, data entry accuracy and consistency, multicollinearity, and normality of distribution for continuous variables. No variables were significantly skewed or kurtotic, indicating the data met the basic assumptions of normality needed for analyses. See Table 1 (above).

Descriptives. Means, standard deviations, and other descriptive data are presented in Table 1. Trait NA was positively correlated with self-reported DERS and borderline features at baseline and follow-up and negatively correlated with basal RSA. Basal RSA was negatively correlated with RSA reactivity. DERS was positively correlated with parental invalidation, and borderline features at baseline and follow-up. Parental invalidation was positively correlated with borderline features at follow-up only, as well as with sex; therefore the decision was made to control for sex in all analyses that included this variable. RSA stress was negatively correlated with Trait NA, and positively correlated with basal RSA, and RSA reactivity. Borderline features at baseline and follow-up were positively correlated.

Table 2. *Correlations for All Study Variables*

Variable	1	2	3	4	5	6	7	8	9
1. Sex	-	-	-	-	-	-	-	-	-
2. Age	-0.12	-	-	-	-	-	-	-	-
3. Trait-NA	0.16	-0.06	-	-	-	-	-	-	-
4. Basal RSA	0.05	0.09	-0.22*	-	-	-	-	-	-
5. Parental Invalidation	-0.21*	-0.02	0.10	-0.01	-	-	-	-	-
6. DERS	-0.10	-0.02	0.33**	-0.13	0.56**	-	-	-	-
7. RSA Reactivity	-0.08	-0.06	0.00	-0.30**	0.12	0.01	-	-	-
8. RSA Stress	0.00	0.06	-.22**	0.82***	0.06	-0.12	0.31***	-	-
9. BPFSC at baseline	-0.09	-0.15	0.54**	-0.02	0.19	.60**	-0.05	-0.05	-
10. BPFSC at 6-month follow up	-0.07	-0.05	0.30**	-0.03	0.32**	.60**	0.05	0.00	0.54**

Note: + $p < .10$, * $p < .05$, ** $p < .01$, *** $p < .001$. Temperamental Negative Affectivity (Trait NA); Dysregulated Emotion Regulation Strategies (DERS); P Invalidation (Parental Invalidation); Borderline Personality Features Scale for Children (BPFS-C)

Cross-Sectional Analyses

Data analytic plan. My study examined a moderated mediation model. The independent variables were emotional vulnerability, measured by trait NA and basal RSA, separately. The dependent variable was borderline features reported at baseline and 6-months. The proposed mediators were emotion dysregulation, measured by RSA reactivity, RSA stress, and DERS, separately. The proposed moderator was parental invalidation. Participant age and ethnicity were not correlated with predictor or outcome variables and so they were not controlled for in my analyses. Participant sex was entered as a covariate for all moderation analyses given its correlation with parental invalidation.

Analysis. Linear regression was used to estimate main effects and the PROCESS macro in SPSS (Hayes, 2012) was used to estimate and probe mediation, moderation and moderated mediation effects. The PROCESS macro for SPSS (Hayes, 2012: Model 4) was used to conduct mediation analyses. Six models were tested to examine whether

emotional vulnerability predicted borderline features at baseline directly and indirectly through emotion dysregulation. The six models represented the two measurements of emotional vulnerability (trait NA, basal RSA) and three measures of emotion dysregulation (DERS, RSA reactivity, RSA stress). PROCESS mean-centered all variables prior to analysis. Indirect effects are considered significant when the confidence intervals do not include "0" whereas confidence intervals including "0" are considered non-significant.

The PROCESS macro for SPSS (Hayes, 2012: Model 1) was used to conduct moderation analyses. Six models were tested to examine whether parental invalidation moderated the relationship between emotional vulnerability and emotion dysregulation. The six moderation models represented the moderation of emotional invalidation on the relationship between emotional vulnerability and emotion dysregulation using two measurements of emotional vulnerability (trait NA, basal RSA), and three measures of emotion dysregulation (DERS, RSA reactivity, RSA stress). PROCESS mean-centered all variables prior to analysis.

The PROCESS macro for SPSS (Hayes, 2012: Model 7) was used to conduct moderated mediation analyses. In these analyses I combined the mediation and moderation models to estimate the conditional indirect effect of emotional vulnerability on borderline features through emotion dysregulation as moderated by parental invalidation on the *a* path. I used 10,000 bootstrap samples to create bias-corrected confidence intervals (95%) to evaluate the statistical significance of indirect and direct effects. Six models were tested to examine whether emotional vulnerability predicted borderline features directly and indirectly through emotion dysregulation, and whether

this relationship was moderated on the *a* path by parental invalidation. The six models represented the two measurements of emotional vulnerability (trait NA, basal RSA) and three measures of emotion dysregulation (DERS, RSA reactivity, RSA stress). PROCESS mean-centered all variables prior to analysis.

Main Effects: Did emotional vulnerability predict borderline features at baseline?

Trait NA as a proposed independent variable. I hypothesized that child reported trait NA would predict borderline features at baseline. Because age, sex and ethnicity were not correlated with predictor or outcome variables, they were not controlled for in this analysis. Results indicated that trait NA was associated with borderline features at baseline *B(SE)*= 10.62(1.67), *t*=6.35, *p*=.000).

Basal RSA as a proposed independent variable. I hypothesized that basal RSA would predict borderline features at baseline. Because age, sex and ethnicity were not correlated with predictor or outcome variables, they were not controlled for in this analysis. Basal RSA was not associated with borderline features at baseline (*B(SE)*=-.17(1.03), *t*=-.17, *p*=.87).

Mediation: Did emotion dysregulation mediate the relationship between emotional vulnerability and borderline features at baseline?

1. Trait NA as a proposed IV and DERS as a proposed mediator. First I tested the effects of trait NA on borderline features at baseline, both directly and indirectly, through self-reported emotion dysregulation as measured by the DERS. Results indicated a positive and statistically significant indirect effect (α=3.11, 95% CI= 1.17 to 5.49) and supported a partial mediation. Because the *a* path was positive and significant (α=11.58, *p*=.000) participants with greater trait NA also reported more emotion dysregulation. The

b path was also positive and significant (α =.27, *p*=.000), suggesting that participants with greater emotion dysregulation also reported increased borderline features. The positive and statistically significant indirect effect links these together: participants who were higher in trait NA reported increased emotion dysregulation on average, and the more emotion dysregulation that was reported, the higher reported borderline features. The significant direct effect (c'=7.51, 95% CI=4.51 to 10.50) suggests that there was still an effect of trait-NA on borderline features when the mediating variable DERS and its effects are included in the model, but that this effect was slightly smaller when the mediating variable was included in the model.

Table 3. *DERS as a Mediator between Trait NA and Borderline Features at Baseline*

Path	Variable	*B*	*SE*	*t*	*p*
α	Trait NA to DERS	11.58	3.30	3.51	.000
β	DERS to Borderline Features	.27	.04	6.20	.000
c	Total Effect	10.62	1.67	5.31	.000
c'	Direct Effect	7.51	1.51	5.00	.000
		M	*SE*	*LL 95% CI*	*UL 95% CI*
α*β	Indirect Effect	3.11	1.08	1.17	5.49

Note. LL = lower limit; CI= confidence interval; UL = upper limit.

2. Trait NA as a proposed IV and RSA reactivity as a proposed mediator. Next I tested the effects of trait NA on borderline features at baseline, both directly and indirectly, through emotion dysregulation as measured by RSA reactivity. Results indicated a negative and non-significant indirect effect (α=-.00, CI=-.50 to .38) and did not support mediation. Neither the *a* (α=.00, *p*=.97) nor *b* (α=-.85, *p*=.55) paths in this model were significant. Trait NA significantly predicted borderline features at baseline

with and without the mediator included in the model suggesting that the presence of this mediating variable did not impact the strength of the relationship.

Table 4. *RSA Reactivity as a Mediator between Trait NA and Borderline Features at Baseline*

Path	Variable	*B*	*SE*	*t*	*p*
α	Trait NA to RSA Reactivity	.00	.12	.04	.979
β	RSA Reactivity to Borderline Features	-.85	1.42	-.59	.533
c	Total Effect	10.62	1.67	6.35	.000
c’	Direct Effect	10.62	1.68	6.34	.000
		M	*SE*	*LL 95% CI*	*UL 95% CI*
α*β	Indirect Effect	-.00	.21	-.50	.38

Note. LL = lower limit; CI= confidence interval; UL = upper limit.

3. Basal RSA as a proposed IV and DERS as a proposed mediator. Next I tested the effects of basal RSA on borderline features at baseline, both directly and indirectly, through self-reported emotion dysregulation as measured by the DERS. Results indicated a negative and non-significant indirect effect (α =-.78, CI=-1.86 to .31) and did not support mediation. The *a* path was non-significant (α=-2.262, *p*=.21), suggesting no meaningful relationship between basal RSA and DERS. The *b* path was significant (α =.34, *p*=.00) suggesting that higher emotion dysregulation is related to greater borderline features at baseline. However the indirect path from predictor to outcome was non-significant, with and without inclusion of the mediator suggesting these variables are not meaningfully related in this sample.

Table 5. *DERS as a Mediator between Basal RSA and Borderline Features at Baseline*

Path	Variable	*B*	*SE*	*t*	*p*
α	Basal RSA to DERS	-2.26	1.79	-1.26	.210
β	DERS to Borderline Features	.34	.05	7.50	.000
c	Total Effect	-.17	1.03	-.17	.869
c’	Direct Effect	.61	.83	.74	.463
		M	*SE*	*LL 95% CI*	*UL 95% CI*

α*β	Indirect Effect	-.78	.55	-1.86	.31

Note. LL = lower limit; CI= confidence interval; UL = upper limit.

4. Basal RSA as a proposed IV and RSA reactivity as a proposed mediator.

Lastly I tested the effects of basal RSA on borderline features at baseline, both directly and indirectly, through physiologically assessed emotion dysregulation as measured by RSA reactivity. Results indicated a negative and non-significant indirect effect (α=.18, CI=-.49 to .98) and did not support mediation. The *a* path was negative and significant (α =-.18, *p* = .00), suggesting that higher basal RSA is related to less RSA reactivity (less vagal withdrawal). The *b* path was non-significant (α =-.98, *p*=.58), suggesting no meaningful relationship between RSA reactivity and borderline features at baseline in this sample. The indirect path from predictor to outcome was non-significant, with and without inclusion of the mediator suggesting these variables are not meaningfully related in this sample.

Table 6. *RSA Reactivity as a Mediator between Basal RSA and Borderline Features at Baseline*

Path	Variable	*B*	*SE*	*t*	*p*
α	Basal RSA to RSA Reactivity	-.18	.06	-3.14	.002
β	RSA Reactivity to Borderline Features	-.98	1.77	-.56	.579
c	Total Effect	-.17	1.03	-.17	.869
c’	Direct Effect	-.35	1.08	-.32	.746
		M	*SE*	*LL 95% CI*	*UL 95% CI*
α*β	Indirect Effect	.18	.36	-.49	.98

Note. LL = lower limit; CI= confidence interval; UL = upper limit.

5. Trait NA as a proposed IV and RSA stress as a proposed mediator. Next I tested the effects of trait NA on borderline features at baseline, both directly and indirectly, through emotion dysregulation as measured by RSA stress. Results indicated a negative and non-significant indirect effect (α=-.34, CI=-1.64 to .24) and did not support

mediation. The *a* path was negative and significant (α=-.43, *p*=.03) suggesting that higher trait NA was related to lower RSA stress (more vagal withdrawal during stress). The *b* path was nonsignificant (α=.78, *p*=.38) in this model were significant suggesting no meaningful relationship between RSA stress and borderline features at baseline in this sample. Trait NA significantly predicted borderline features at baseline with and without the mediator included in the model suggesting that the presence of this mediating variable did not impact the strength of the relationship.

Table 7. *RSA Stress as a Mediator between Trait NA and Borderline Features at Baseline*

Path	Variable	*B*	*SE*	*t*	*p*
α	Trait NA to RSA Stress	-.43	.19	-2.26	.026
β	RSA Stress to Borderline Features	.78	.89	.88	.379
c	Total Effect	10.62	1.67	6.35	.000
c'	Direct Effect	10.95	1.72	6.38	.000
		M	*SE*	*LL 95% CI*	*UL 95% CI*
α*β	Indirect Effect	-.34	.44	-1.64	.24

Note. LL = lower limit; CI= confidence interval; UL = upper limit.

6. Basal RSA as a proposed IV and RSA stress as a proposed mediator. Lastly I tested the effects of basal RSA on borderline features at baseline, both directly and indirectly, through physiologically assessed emotion dysregulation as measured by RSA stress. Results indicated a negative and non-significant indirect effect (α=-.80, CI=-3.81 to 2.16) and did not support mediation. The *a* path was positive and significant (α = .82, *p* = .000), suggesting that lower basal RSA is related to lower RSA stress (more withdrawal during stress). The *b* path was non-significant (α =-.98, *p*=.58), suggesting no meaningful relationship between RSA stress and borderline features at baseline in this sample. The indirect path from predictor to outcome was non-significant, with and without inclusion of the mediator suggesting these variables are not meaningfully related in this sample.

Table 8. *RSA Stress as a Mediator between Basal RSA and Borderline Features at Baseline*

Path	Variable	*B*	*SE*	*t*	*p*
α	Basal RSA to RSA Stress	.82	.06	13.98	.000
β	RSA Stress to Borderline Features	-.47	1.02	-.46	.65
c	Total Effect	-.17	1.03	-.17	.87
c'	Direct Effect	.63	1.77	.36	.72
		M	*SE*	*LL 95% CI*	*UL 95% CI*
α*β	Indirect Effect	-.80	1.52	-3.81	2.16

Note. LL = lower limit; CI= confidence interval; UL = upper limit.

Moderation: Did parental invalidation moderate the effect of emotional vulnerability on emotion dysregulation?

1. Trait NA as a proposed IV and DERS as a proposed DV. I entered trait NA as predictor, DERS as outcome variable, and parental invalidation as moderator; child sex was entered as a covariate. Parental invalidation did moderate the effect of trait NA on self-reported emotion dysregulation (*B(SE)*=-8.78 (3.16), *t*=-2.78, *p*= .00). Trait NA predicted emotion dysregulation at low and moderate levels of parental invalidation but at high levels of parental invalidation it did not (Conditional effects. Low levels of invalidation: *B(SE)*=18.76(4.25), *t*=4.41, *p*=.00; medium levels of invalidation: *B(SE)*=10.08(3.03), *t*=3.33, *p*=.00; high levels of invalidation: *B(SE)*=1.40(4.45), *t*=.32, *p*=.75). This indicated that at low levels of trait NA, higher invalidation is associated with higher emotion dysregulation. However, at high levels of trait NA, adolescents experience high levels of emotion dysregulation, regardless of invalidation. The lowest emotion dysregulation is seen for adolescents with low trait NA and low invalidation, as would be expected. See Figure 1.

Table 9. *Parental Invalidation as a Moderator between Trait NA and DERS*

Path	Variable	*B*	*SE*	*t*	*p*

	Invalidation to DERS	29.07	8.51	3.41	.000
β	Trait NA to DERS	32.38	8.45	3.83	.000
	Interaction of Invalidation * DERS	8.78	3.16	-2.78	.006
Invalidation		*Effect*	*SE*	*t*	*p*
-1 SD	-.99	18.76	4.25	4.41	.000
Mean	.00	10.07	3.03	3.33	.001
+1 SD	.99	1.40	4.45	.31	.754

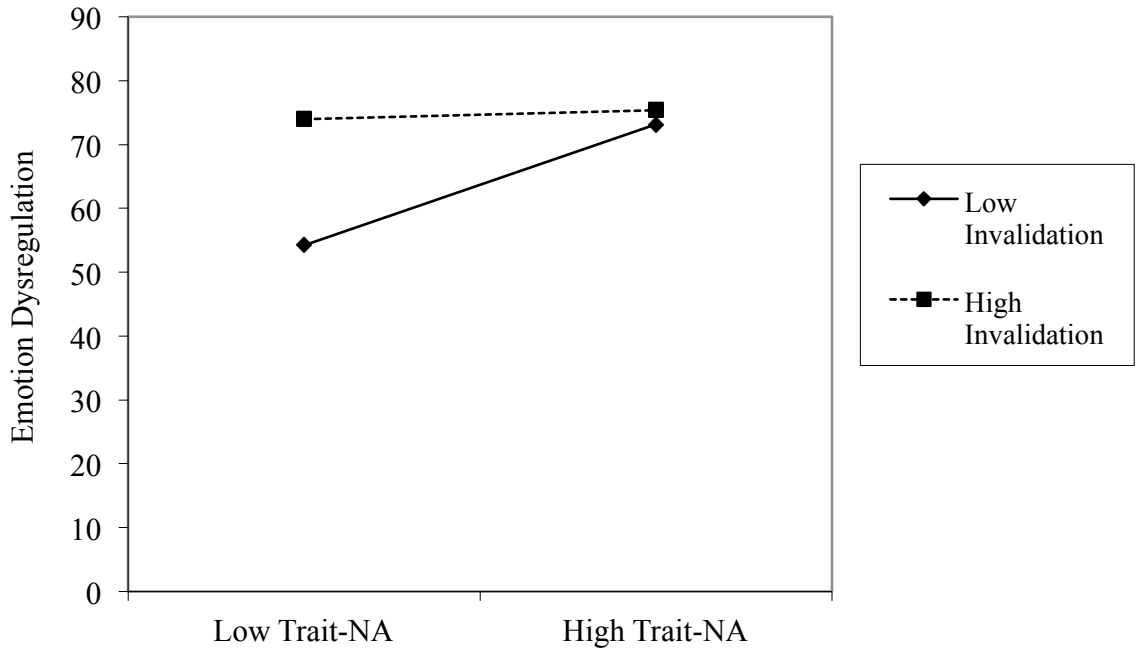

Figure 1. Interaction of Trait NA and Invalidating Parenting Predicting Emotion Dysregulation.

2. Trait NA as a proposed IV and RSA reactivity as a proposed DV. I entered trait NA as predictor, RSA reactivity as outcome variable and parental invalidation as moderator; child sex was entered as a covariate. Parental invalidation did not moderate the effect of trait NA on RSA reactivity (*B(SE)*=-.02(.13), *t*=-.17, *p*= .86).

Table 10. *Parental Invalidation as a Moderator between Trait NA and RSA Reactivity*

Path	Variable	*B*	*SE*	*t*	*p*
β	Invalidation to RSA Reactivity	.06	.06	.99	.323

	Trait NA to RSA Reactivity	.00	.12	-.17	.987
	Interaction of Invalidation * RSA Reactivity	-.02	.12	-.17	.862
Invalidation		*Effect*	*SE*	*t*	*p*
-1 SD	-.99	.02	.17	.14	.891
Mean	.00	.00	.12	.02	.988
+1 SD	.99	-.02	.18	-.11	.912

3. Basal RSA as a proposed IV and DERS as a proposed DV. I entered basal RSA as predictor, DERS as outcome variable and parental invalidation as moderator; child sex was entered as a covariate. Parental invalidation did not moderate the effect of basal RSA on self-reported emotion dysregulation (*B(SE)*=-.02(1.76), *t*=-.01, *p*=.99).

Table 11. *Parental Invalidation as a Moderator between Basal RSA and DERS*

Path	Variable	*B*	*SE*	*t*	*p*
	Invalidation to DERS	6.21	1.73	3.60	.000
β	Basal RSA to DERS	-2.15	1.71	-1.26	.211
	Interaction of Invalidation * DERS	-.02	1.76	-.01	.992
Invalidation		*Effect*	*SE*	*t*	*p*
-1 SD	-.99	-2.13	2.34	-.91	.364
Mean	.00	-2.15	1.71	-1.26	.211
+1 SD	.99	-2.17	2.54	-.85	.395

4. Basal RSA as a proposed IV and RSA reactivity as a proposed DV. I entered basal RSA as predictor, RSA reactivity as outcome variable and parental invalidation as moderator; child sex was entered as a covariate. Parental invalidation did not moderate the effect of basal RSA on RSA reactivity (*B(SE)*=-.02(.06), *t*=-.28, *p*=.78).

Table 12. *Parental Invalidation as a Moderator between Basal RSA and RSA Reactivity*

Path	Variable	*B*	*SE*	*t*	*p*
	Invalidation to RSA Reactivity	.06	.06	1.04	.302
β	Basal RSA to RSA Reactivity	-.18	.06	-3.09	.003
	Interaction of Invalidation * RSA Reactivity	-.02	.06	-.28	.784
Invalidation		*Effect*	*SE*	*t*	*p*
-1 SD	-.99	-.17	.08	-2.05	.043
Mean	.00	-.18	.06	-3.09	.003

+1 SD	.99	-.20	.09	-2.27	.026

5. Trait NA as a proposed IV and RSA Stress as a proposed DV. I entered trait NA as predictor, RSA stress as outcome variable and parental invalidation as moderator; child sex was entered as a covariate. Parental invalidation did not moderate the effect of trait NA on RSA stress (*B(SE)*=.32(.20), *t*=1.61, *p*= .11).

Table 13. *Parental Invalidation as a Moderator between Trait NA and RSA stress*

Path	Variable	*B*	*SE*	*t*	*p*
β	Invalidation to RSA Stress	.10	.10	1.05	.298
	Trait NA to RSA Stress	-.46	.19	-2.35	.021
	Interaction of Invalidation * RSA Stress	.32	.20	.61	.111
Invalidation		*Effect*	*SE*	*t*	*p*
-1 SD	-.99	-.77	.27	-2.85	.005
Mean	.00	-.50	.19	-2.35	.021
+1 SD	.99	-.14	.28	-.50	.621

6. Basal RSA as a proposed IV and RSA Stress as a proposed DV. I entered basal RSA as predictor, RSA stress as outcome variable and parental invalidation as moderator; child sex was entered as a covariate. Parental invalidation did not moderate the effect of basal RSA on RSA reactivity (*B(SE)*=-.02(.06), *t*=-.28, *p*=.78).

Table 14. *Parental Invalidation as a Moderator between Basal RSA and RSA Stress*

Path	Variable	*B*	*SE*	*t*	*p*
β	Invalidation to RSA Stress	.06	.06	1.04	.30
	Basal RSA to RSA Stress	.82	.06	13.83	.000
	Interaction of Invalidation * RSA Stress	-.02	.06	-.28	.783
Invalidation		*Effect*	*SE*	*t*	*p*
-1 SD	-.99	.83	.08	10.31	.000
Mean	.00	.82	.06	13.83	.000
+1 SD	.99	.80	.09	9.12	.000

Moderated Mediation: **Did emotion dysregulation mediate the relationship between emotional vulnerability and borderline features at baseline, and is this relationship moderated by parental invalidation?**

1. Trait NA as a proposed IV and DERS as a proposed mediator. I entered trait NA as predictor, borderline features at baseline as outcome, DERS as mediator and parental invalidation as the *a* path moderator; child sex was entered as a covariate. Piecemeal results are reported above for simple mediation and moderation. The index of moderated mediation was significant (*B(SE)*=-2.360(1.02), 95% CI=-4.558 to -.481). That is, DERS mediated the relationship between trait NA and borderline features at baseline, and the relationship between trait NA to DERS was moderated by parental invalidation. This indicates that participants who were higher in trait NA reported increased emotion dysregulation on average, and the more emotion dysregulation that was reported, the higher reported borderline features. However, parental invalidation impacted the relationship between trait NA and emotion dysregulation such that the relationship only held under low and moderate levels of parental invalidation.

Table 15. *Moderated Mediation Analysis*

Path	Variable	*B*	*SE*	*t*	*p*
α	Constant to DERS	74.38	5.05	14.71	.000
	Trait NA to DERS	10.69	3.08	3.47	.000
β	Constant to Borderline Features	38.34	4.10	9.35	.000
	DERS to Borderline Features	.26	.04	5.89	.000
c'	Direct Effect	7.51	1.51	4.97	.000
	Invalidation to DERS	29.07	8.51	3.41	.000
	Interaction of Trait NA*Invalidation	-8.78	3.26	-2.78	.006
	Sex (control)	-3.41	3.16	-1.08	.283
Index of Moderated Mediation		*Index*	*Boot SE*	*LL 95% CI*	*UL 95% CI*
		-2.30	.98	-4.40	-.53

Invalidation		*Indirect Effect*	*Boot SE*	*LL 95% CI*	*UL 95% CI*
-1 SD	-.99	5.01	1.22	3.00	8.00
Mean	.00	2.76	.94	1.17	4.90
+1 SD	.99	.50	1.50	-2.10	3.73

Note. LL = lower limit; CI= confidence interval; UL = upper limit.

2. Trait NA as a proposed IV and RSA reactivity as a proposed mediator. I entered trait NA as predictor, borderline features at baseline as outcome, RSA reactivity as mediator and parental invalidation as the *a* path moderator; child sex was entered as a covariate. Piecemeal results are reported above for simple mediation and moderation. The index of moderated mediation was non-significant (*B(SE)*=.02(.26), 95% CI=-.32 to .88).

Table 16. *Moderated Mediation Analysis*

Path	Variable	*B*	*SE*	*t*	*p*
α	Constant to RSA Reactivity	-.48	.20	-2.38	.019
	Trait NA to RSA Reactivity	.02	.12	.02	.988
β	Constant to Borderline Features	57.81	2.76	20.96	.000
	RSA Reactivity to Borderline Features	-1.09	1.40	-.78	.437
c'	Direct Effect	11.22	1.67	6.74	.000
	Invalidation to RSA Reactivity	.06	.06	.99	.323
	Interaction of Trait NA*Invalidation	-.02	.13	-.17	.862
	Sex (control)	-.07	.13	-.53	.595

Index of Moderated Mediation	*Index*	*Boot SE*	*LL 95% CI*	*UL 95% CI*
	.02	.26	-.32	.88

Invalidation		*Indirect Effect*	*Boot SE*	*LL 95% CI*	*UL 95% CI*
-1 SD	-.99	-.03	.35	-1.11	.50
Mean	.00	-.00	.22	-.48	.44
+1 SD	.99	.02	.33	-.46	.93

Note. LL = lower limit; CI= confidence interval; UL = upper limit.

3. Basal RSA as a proposed IV and DERS as a proposed mediator. I entered basal RSA as predictor, borderline features at baseline as outcome, DERS as mediator and parental invalidation as the *a* path moderator; child sex was entered as a covariate.

Piecemeal results are reported above for simple mediation and moderation. The index of moderated mediation was non-significant (*B(SE)*=-.01(.68), 95% CI=-1.33 to 1.32).

Table 17. *Moderated Mediation Analysis*

Path	Variable	*B*	*SE*	*t*	*p*
α	Constant to DERS	70.12	5.45	12.86	.000
	Basal RSA to DERS	-2.15	1.71	-1.26	.211
β	Constant to Borderline Features	30.14	4.30	7.01	.000
	DERS to Borderline Features	.34	.05	7.39	.000
c'	Direct Effect	.63	.83	.75	.454
	Invalidation to DERS	6.21	1.73	3.60	.005
	Interaction of Basal RSA*Invalidation	-.02	1.76	-.01	.992
	Sex (control)	-.89	3.41	-.26	.793

Index of Moderated Mediation	*Index*	*Boot SE*	*LL 95% CI*	*UL 95% CI*
	-.01	.68	-1.33	1.32

Invalidation		*Indirect Effect*	*Boot SE*	*LL 95% CI*	*UL 95% CI*
-1 SD	-.99	-.73	.74	-2.13	.82
Mean	.00	-.74	.61	-1.95	.45
+1 SD	.99	-.74	1.05	-2.69	1.43

Note. LL = lower limit; CI= confidence interval; UL = upper limit.

4. Basal RSA as a proposed IV and RSA reactivity as a proposed mediator. I entered basal RSA as predictor, borderline features at baseline as outcome, RSA reactivity as mediator and parental invalidation as the *a* path moderator; child sex was entered as a covariate. Piecemeal results are reported above for simple mediation and moderation. The index of moderated mediation was non-significant (*B(SE)*=.02(.17), 95% CI=-.26 to .50).

Table 18. *Moderated Mediation Analysis*

Path	Variable	*B*	*SE*	*t*	*p*
α	Constant to RSA Reactivity	-.50	.19	-2.68	.009
	Basal RSA to RSA Reactivity	-.18	.06	-3.09	.003
β	Constant to Borderline Features	54.99	3.33	16.51	.000
	RSA Reactivity to Borderline	-1.09	1.77	-.62	.539

	Features				
c'	Direct Effect	-.32	1.10	-.30	.771
	Invalidation to RSA Reactivity	.06	.06	1.04	.302
	Interaction of Basal RSA*Invalidation	-.02	.06	-.28	.784
	Sex (control)	-.05	.12	-.41	.679
Index of Moderated Mediation		*Index*	*Boot SE*	*LL 95% CI*	*UL 95% CI*
		.02	.17	-.26	.50
Invalidation		*Indirect Effect*	*Boot SE*	*LL 95% CI*	*UL 95% CI*
-1 SD	-.99	.18	.39	-.38	1.34
Mean	.00	.20	.36	-.47	1.00
+1 SD	.99	.22	.41	-.40	1.34

Note. LL = lower limit; CI= confidence interval; UL = upper limit.

5. Trait NA as a proposed IV and RSA Stress as a proposed mediator. I entered trait NA as predictor, borderline features at baseline as outcome, RSA stress as mediator and parental invalidation as the *a* path moderator; child sex was entered as a covariate. Piecemeal results are reported above for simple mediation and moderation. The index of moderated mediation was non-significant (*B(SE)*=.28(.37), 95% CI=-.14 to 1.55).

Table 19. *Moderated Mediation Analysis*

Path	Variable	*B*	*SE*	*t*	*p*
α	Constant to RSA Stress	6.27	.42	19.72	.000
	Trait NA to RSA Stress	-.46	.19	-2.35	.02
β	Constant to Borderline Features	52.76	6.14	8.60	.000
	RSA Stress to Borderline Features	.87	.87	1.00	.32
c'	Direct Effect	11.59	1.71	6.80	.000
	Invalidation to RSA Stress	.10	.10	1.04	.30
	Interaction of Trait NA*Invalidation	.32	.20	1.61	.11
	Sex (control)	.14	.20	.50	.50
Index of Moderated Mediation		*Index*	*Boot SE*	*LL 95% CI*	*UL 95% CI*
		.30	.37	-.14	1.55
Invalidation		*Indirect Effect*	*Boot SE*	*LL 95% CI*	*UL 95% CI*
-1 SD	-.99	-.67	.72	-2.70	.32

Mean	.00	-.40	.46	-1.75	.19
+1 SD	.99	-.12	.40	-1.45	.34

Note. LL = lower limit; CI= confidence interval; UL = upper limit.

6. Basal RSA as a proposed IV and RSA stress as a proposed mediator. I entered basal RSA as predictor, borderline features at baseline as outcome, RSA stress as mediator and parental invalidation as the *a* path moderator; child sex was entered as a covariate. Piecemeal results are reported above for simple mediation and moderation. The index of moderated mediation was non-significant (*B(SE)*=.02(.17), 95% CI=-.25 to .51).

Table 20. *Moderated Mediation Analysis*

Path	Variable	*B*	*SE*	*t*	*p*
α	Constant to RSA Stress	6.57	.19	34.67	.000
	Basal RSA to RSA Stress	.82	.06	13.83	.000
β	Constant to Borderline Features	62.71	12.15	5.16	.000
	RSA Stress to Borderline Features	-1.10	1.80	-.62	.54
c'	Direct Effect	.78	1.78	.44	.663
	Invalidation to RSA Stress	.06	.06	1.04	.302
	Interaction of Basal RSA *Invalidation	-.02	.06	-.28	.784
	Sex (control)	-.05	.12	-.41	.679
Index of Moderated Mediation		*Index*	*Boot SE*	*LL 95% CI*	*UL 95% CI*
		.02	.17	-.25	.51
Invalidation		*Indirect Effect*	*Boot SE*	*LL 95% CI*	*UL 95% CI*
-1 SD	-.99	-.91	1.57	-4.27	1.96
Mean	.00	-.89	1.52	-3.94	2.06
+1 SD	.99	-.87	1.49	-3.76	2.14

Note. LL = lower limit; CI= confidence interval; UL = upper limit.

Prospective Analyses

Data analytic plan. All prospective data analyses were also conducted with SPSS 20.0. Linear regression was used to estimate main effects and the PROCESS macro in SPSS (Hayes, 2012) were used to estimate and probe moderation and moderated

mediation effects. My prospective analyses were identical to my cross-sectional analyses with the exception of my dependent variable.

Main Effects: Did emotional vulnerability predict change in borderline features at 6-month follow-up?

Trait NA as a proposed independent variable. I hypothesized that child reported temperament would prospectively predict change in borderline features at 6-month follow-up. Because age, sex and ethnicity were not correlated with predictor or outcome variables, they were not controlled for in this analysis; borderline features at baseline were entered as a covariate. Trait NA did not predict change in borderline features from baseline to follow-up (*B(SE)*= .11(2.24), *t*=.05, *p*=.96).

Basal RSA as a proposed independent variable. I hypothesized that emotional vulnerability assessed physiologically via basal RSA would prospectively predict change in borderline features at 6-month follow-up. Because age, sex and ethnicity were not correlated with predictor or outcome variables, they were not controlled for in this analysis; borderline features at baseline were entered as a covariate. Basal RSA did not predict change in borderline features at follow-up (*B(SE)*=-.21(.98), *t*=-.21, *p*= .83).

Mediation: Did emotion dysregulation mediate the relationship between emotional vulnerability and change in borderline features at follow-up?

The PROCESS macro for SPSS (Hayes, 2012: Model 4) was used to conduct mediation analyses. Six models were tested to examine whether emotional vulnerability predicted change in borderline features at follow-up indirectly through emotion dysregulation. The six models represented the two measurements of emotional

vulnerability (trait NA, basal RSA) and three measures of emotion dysregulation (DERS, RSA reactivity, RSA Stress). PROCESS mean-centered all variables prior to analysis.

1. Trait NA as a proposed IV and DERS as a proposed mediator. First I tested the effects of trait NA on change in borderline features at follow-up, both directly and indirectly, through self-reported emotion dysregulation as measured by the DERS. Borderline features at baseline were entered as a covariate. Results indicated a positive and non-significant indirect effect (α=.12, 95% CI= -1.90 to 2.27) and did not support mediation. The *a* path was positive and non-significant (α=.45, *p*=.89). The *b* path was positive and significant (α =.27, *p*=.00), suggesting that higher emotion dysregulation is related to greater borderline features at follow-up. However the indirect path from predictor to outcome was non-significant, with and without inclusion of the mediator suggesting these variables are not meaningfully related in this sample.

Table 21. *DERS as a Mediator between Trait NA and Borderline Features at Follow-up*

Path	Variable	*B*	*SE*	*t*	*p*
α	Trait NA to DERS	.45	3.34	.14	.892
β	DERS to Borderline Features	.27	.06	-.01	.000
c	Total Effect	.11	2.24	.05	.961
c’	Direct Effect	-.01	2.06	-.00	.995
		M	*SE*	*LL 95% CI*	*UL 95% CI*
α*β	Indirect Effect	.12	1.05	-1.90	2.27

Note. LL = lower limit; CI= confidence interval; UL = upper limit.

2. Trait NA as a proposed IV and RSA reactivity as a proposed mediator. Next I tested the effects of trait NA on change in borderline features at follow-up, both directly and indirectly, through emotion dysregulation as measured by RSA reactivity. Borderline features at baseline were entered as a covariate. Results indicated a positive and non-significant indirect effect (α=.07, CI=-.30 to 1.17) and did not support mediation. Neither

the *a* (α=.05, *p*=.73) nor *b* (α=1.36, *p*=..40) paths in this model were significant. Trait NA did not significantly predicted borderline features at follow-up with or without the mediator included in the model suggesting that the presence of this mediating variable did not impact the strength of the relationship.

Table 22. *RSA Reactivity as a Mediator between Trait NA and Borderline Features at Follow-up*

Path	Variable	*B*	*SE*	*t*	*p*
α	Trait NA to RSA Reactivity	.05	.14	.35	.726
β	RSA Reactivity to Borderline Features	1.36	1.60	.85	.398
c	Total Effect	.11	2.24	.05	.961
c'	Direct Effect	.04	2.24	.02	.985
		M	*SE*	*LL 95% CI*	*UL 95% CI*
α*β	Indirect Effect	.07	.31	-.30	1.17

Note. LL = lower limit; CI= confidence interval; UL = upper limit.

3. Basal RSA as a proposed IV and DERS as a proposed mediator. Next I tested the effects of basal RSA on change in borderline features at follow-up, both directly and indirectly, through self-reported emotion dysregulation as measured by the DERS. Borderline features at baseline were entered as a covariate. Results indicated a negative and non-significant indirect effect (α =-.57, CI=-1.51 to .03) and did not support mediation. The *a* path was non-significant (α=-2.08, *p*=.15), suggesting no meaningful relationship between basal RSA and DERS. The *b* path was significant (α =.27, *p*=.00) suggesting that higher emotion dysregulation is related to greater borderline features at follow-up. However the indirect path from predictor to outcome was non-significant, with and without inclusion of the mediator suggesting these variables are not meaningfully related in this sample.

Table 23. *DERS as a Mediator between Basal RSA and Borderline Features at Follow-up*

Path	Variable	*B*	*SE*	*t*	*p*
α	Basal RSA to DERS	-2.08	1.44	-1.45	.151
β	DERS to Borderline Features	.28	.06	4.37	.000
c	Total Effect	-.21	.98	-.21	.834
c’	Direct Effect	.37	.90	.41	.685
		M	*SE*	*LL 95% CI*	*UL 95% CI*
α*β	Indirect Effect	-.57	.38	-1.51	.03

Note. LL = lower limit; CI= confidence interval; UL = upper limit.

4. Basal RSA as a proposed IV and RSA reactivity as a proposed mediator.

Lastly I tested the effects of basal RSA on change in borderline features at follow-up, both directly and indirectly, through physiologically assessed emotion dysregulation as measured by RSA reactivity. Borderline features at baseline were entered as a covariate. Results indicated a negative and non-significant indirect effect (α=-.26, CI=-1.34 to .28) and did not support mediation. The *a* path was negative and significant (α =-.18, *p* = .002), suggesting that higher basal RSA is related to less RSA reactivity (less vagal withdrawal). The *b* path was non-significant (α =1.39, *p*=1.68), suggesting no meaningful relationship between RSA reactivity and borderline features at follow-up in this sample. The indirect path from predictor to outcome was non-significant, with and without inclusion of the mediator suggesting these variables are not meaningfully related in this sample.

Table 24. *RSA Reactivity as a Mediator between Basal RSA and Borderline Features at Follow-up*

Path	Variable	*B*	*SE*	*t*	*p*
α	Basal RSA to RSA Reactivity	-.18	.06	-3.14	.002
β	RSA Reactivity to Borderline Features	1.39	1.68	.83	.411
c	Total Effect	-.21	.98	-.21	.834
c’	Direct Effect	.05	1.02	.05	.961

		M	*SE*	*LL 95% CI*	*UL 95% CI*
α*β	Indirect Effect	-.26	.38	-1.34	.28

Note. LL = lower limit; CI= confidence interval; UL = upper limit.

5. Trait NA as a proposed IV and RSA stress as a proposed mediator. Next I tested the effects of trait NA on change in borderline features at follow-up, both directly and indirectly, through emotion dysregulation as measured by RSA stress. Borderline features at baseline were entered as a covariate. Results indicated a negative and non-significant indirect effect (α=-.18, CI=-1.77 to .76) and did not support mediation. Neither the *a* (α=-.54, *p*=.02) nor *b* (α=.33, *p*=.74) paths in this model were significant. Trait NA did not significantly predict borderline features at follow-up with and without the mediator included in the model suggesting that the presence of this mediating variable did not impact the strength of the relationship.

Table 25. *RSA Stress as a Mediator between Trait NA and Borderline Features at Follow-up*

Path	Variable	*B*	*SE*	*t*	*p*
α	Trait NA to RSA Stress	-.54	.23	-2.38	.019
β	RSA Stress to Borderline Features	.33	1.01	.33	.745
c	Total Effect	.11	2.24	.05	.961
c’	Direct Effect	.29	2.31	.12	.902
		M	*SE*	*LL 95% CI*	*UL 95% CI*
α*β	Indirect Effect	-.18	.58	-1.77	.76

Note. LL = lower limit; CI= confidence interval; UL = upper limit.

6. Basal RSA as a proposed IV and RSA stress as a proposed mediator. Lastly I tested the effects of basal RSA on change in borderline features at follow-up, both directly and indirectly, through physiologically assessed emotion dysregulation as measured by RSA stress. Borderline features at baseline were entered as a covariate. Results indicated a positive and non-significant indirect effect (α=1.13, CI=-1.58 to 4.15)

and did not support mediation. The *a* path was positive and significant (α = .82, *p* = .000), suggesting that lower basal RSA is related to lower RSA stress (more withdrawal during stress). The *b* path was non-significant (α =1.39, *p*=.41), suggesting no meaningful relationship between RSA stress and borderline features at follow-up in this sample. The indirect path from predictor to outcome was non-significant, with and without inclusion of the mediator suggesting these variables are not meaningfully related in this sample.

Table 26. *RSA Stress as a Mediator between Basal RSA and Borderline Features at Follow-up*

Path	Variable	*B*	*SE*	*t*	*p*
α	Basal RSA to RSA Stress	.82	.06	13.92	.000
β	RSA Stress to Borderline Features	1.39	1.68	.83	.411
c	Total Effect	-.21	.98	-.21	.834
c'	Direct Effect	-1.34	1.68	-.79	.429
		M	*SE*	*LL 95% CI*	*UL 95% CI*
α*β	Indirect Effect	1.13	1.45	-1.58	4.15

Note. LL = lower limit; CI= confidence interval; UL = upper limit.

Moderated Mediation: **Did emotion dysregulation mediate the relationship between emotional vulnerability and change in borderline features at follow-up, and is this relationship moderated by parental invalidation?**

The PROCESS macro for SPSS (Hayes, 2012: Model 7) was used to conduct prospective moderated mediation analyses. In these analyses I combined the mediation and moderation models to estimate the conditional indirect effect of emotional vulnerability on change in borderline features at follow-up through emotion dysregulation as moderated by parental invalidation on the *a* path. I used 10,000 bootstrap samples to create bias-corrected confidence intervals (95%) to evaluate the statistical significance of indirect and direct effects. Six models were tested to examine whether emotional vulnerability predicted borderline features directly and indirectly through emotion

dysregulation, and whether this relationship was moderated on the *a* path by parental invalidation. The six models represented the two measurements of emotional vulnerability (trait NA, basal RSA) and three measures of emotion dysregulation (DERS, RSA reactivity, RSA stress). PROCESS mean-centered all variables prior to analysis.

1. Trait NA as a proposed IV and DERS as a proposed mediator. I entered trait NA as predictor, borderline features at follow-up as outcome, DERS as mediator and parental invalidation as the *a* path moderator; child sex and borderline features at baseline were entered as covariates. Piecemeal results are reported above for simple mediation and moderation. The index of moderated mediation was significant (*B(SE)*=-2.13(.92), 95% CI=-4.37 to -.65). This suggests that although main effects and simple mediation effects were non-significant, the overall model was significant, indicating that the effect of trait NA on the change in borderline features at follow-up, through DERS, depends on the level of parental invalidation. This effect is significant at low levels of invalidation only.

Table 27. *Moderated Mediation Analysis*

Path	Variable	*B*	*SE*	*t*	*p*
α	Constant to DERS	20.45	10.30	1.99	.050
	Trait NA to DERS	.50	3.19	.16	.875
β	Constant to Borderline Features at FU	12.39	6.96	1.78	.078
	DERS to Borderline Features at FU	.27	.06	4.32	.000
c'	Direct Effect	-.02	2.14	-.01	.994
	Invalidation to DERS	4.26	1.39	3.07	.003
	Interaction of Trait NA*Invalidation	-7.86	2.73	-2.87	.005
	Sex (control)	-.37	2.78	-.13	.895
	Borderline Features at baseline (control)				

Index of Moderated Mediation	*Index*	*Boot SE*	*LL 95% CI*	*UL 95% CI*
	-2.13	.92	-4.37	-.65

Invalidation		*Indirect Effect*	*Boot SE*	*LL 95% CI*	*UL 95% CI*
-1 SD	-.99	2.24	1.24	.38	5.43
Mean	.00	.14	.86	-1.43	1.98

+1 SD	.99	1.97	1.27	-4.59	.34

Note. LL = lower limit; CI= confidence interval; UL = upper limit.

2. Trait NA as a proposed IV and RSA reactivity as a proposed mediator. I entered trait NA as predictor, borderline features at follow-up as outcome, RSA reactivity as mediator and parental invalidation as the *a* path moderator; child sex and borderline features at baseline were entered as covariates. Piecemeal results are reported above for simple mediation and moderation. The index of moderated mediation was non-significant (*B(SE)*=-.04(.32), 95% CI=-1.05 to .41).

Table 28. *Moderated Mediation Analysis*

Path	Variable	*B*	*SE*	*t*	*p*
α	Constant to RSA Reactivity	-.09	.47	-.18	.855
	Trait NA to RSA Reactivity	.08	.15	.52	.607
β	Constant to Borderline Features at FU	17.33	7.49	2.32	.022
	RSA Reactivity to Borderline Features at FU	1.34	1.63	.82	.412
c'	Direct Effect	.15	2.33	.06	.949
	Invalidation to RSA Stress	.07	.06	1.09	.280
	Interaction of Basal RSA *Invalidation	-.03	.13	-.23	.817
	Sex (control)	-.09	.13	-.70	.489
	Borderline Features at baseline (control)	.61	.12	5.22	.000

Index of Moderated Mediation	*Index*	*Boot SE*	*LL 95% CI*	*UL 95% CI*
	-.04	.32	-1.05	.41

Invalidation		*Indirect Effect*	*Boot SE*	*LL 95% CI*	*UL 95% CI*
-1 SD	-.99	.14	.49	-.38	2.00
Mean	.00	.10	.32	-.24	1.27
+1 SD	.99	.06	.40	-.41	1.46

Note. LL = lower limit; CI= confidence interval; UL = upper limit.

3. Basal RSA as a proposed IV and DERS as a proposed mediator. I entered basal RSA as predictor, borderline features at follow-up as outcome, DERS as mediator and parental invalidation as the *a* path moderator; child sex and borderline features at baseline were entered as covariates. Piecemeal results are reported above for simple

mediation and moderation. The index of moderated mediation was non-significant (*B(SE)*=-.32(.41), 95% CI=-1.21 to .45).

Table 29. *Moderated Mediation Analysis*

Path	Variable	*B*	*SE*	*t*	*p*
α	Constant to DERS	16.59	8.74	1.90	.061
	Basal RSA to DERS	-2.13	1.39	-1.53	.128
β	Constant to Borderline Features at FU	12.40	5.76	2.15	.034
	DERS to Borderline Features at FU	.27	.06	4.34	.000
c'	Direct Effect	.37	.91	.40	.687
	Invalidation to DERS	4.40	1.43	3.09	.002
	Interaction of Basal RSA *Invalidation	-1.16	1.44	-.81	.422
	Sex (control)	.06	2.77	.02	.982
	Borderline Features at baseline (control)	.32	.11	2.91	.005

Index of Moderated Mediation	*Index*	*Boot SE*	*LL 95% CI*	*UL 95% CI*
	-.32	.41	-1.21	.45

Invalidation		*Indirect Effect*	*Boot SE*	*LL 95% CI*	*UL 95% CI*
-1 SD	-.99	-.27	.43	-1.29	.46
Mean	.00	-.59	.37	-1.46	.02
+1 SD	.99	-.90	.65	-2.42	.17

Note. LL = lower limit; CI= confidence interval; UL = upper limit.

4. Basal RSA as a proposed IV and RSA reactivity as a proposed mediator. I entered basal RSA as predictor, borderline features at follow-up as outcome, RSA reactivity as mediator and parental invalidation as the *a* path moderator; child sex and borderline features at baseline were entered as covariates. Piecemeal results are reported above for simple mediation and moderation. The index of moderated mediation was non-significant (*B(SE)*=-.02(.18), 95% CI=-.52 to .26)

Table 30. *Moderated Mediation Analysis*

Path	Variable	*B*	*SE*	*t*	*p*
α	Constant to RSA Reactivity	-.25	.37	-.68	.497
	Basal RSA to RSA Reactivity	-.18	.06	-3.08	.002
β	Constant to Borderline Features at FU	17.08	6.20	2.76	.007
	RSA Reactivity to Borderline Features at	1.37	1.69	.81	.421

	FU				
c'	Direct Effect	.06	1.02	.05	.957
	Invalidation to RSA Reactivity	.07	.06	1.16	.249
	Interaction of Basal RSA *Invalidation	-.01	.06	-.19	.852
	Sex (control)	-.053	.12	-.45	.652
	Borderline Features at baseline (control)	-.00	.01	-.78	.437
Index of Moderated Mediation		*Index*	*Boot SE*	*LL 95% CI*	*UL 95% CI*
		-.12	.18	-.52	.26
Invalidation		*Indirect Effect*	*Boot SE*	*LL 95% CI*	*UL 95% CI*
-1 SD	-.99	-.23	.42	-1.59	.25
Mean	.00	-.25	.37	-1.22	.29
+1 SD	.99	-.27	.40	-1.45	.24

Note. LL = lower limit; CI= confidence interval; UL = upper limit.

5. Trait NA as a proposed IV and RSA Stress as a proposed mediator. I entered trait NA as predictor, borderline features at follow-up as outcome, RSA stress as mediator and parental invalidation as the *a* path moderator; child sex and borderline features at baseline were entered as covariates. Piecemeal results are reported above for simple mediation and moderation. The index of moderated mediation was non-significant (*B(SE)*=.12(.41), 95% CI=-.39 to 1.49).

Table 31. *Moderated Mediation Analysis*

Path	Variable	*B*	*SE*	*t*	*p*
α	Constant to RSA Stress	5.59	.75	7.45	.000
	Trait NA to RSA Stress	-.58	.23	-2.51	.013
β	Constant to Borderline Features at FU	15.23	9.46	1.61	.111
	RSA Stress to Borderline Features at FU	.35	1.01	.34	.732
c'	Direct Effect	.46	2.40	.19	.849
	Invalidation to RSA Stress	.09	1.1	.93	.356
	Interaction of Trait NA*Invalidation	.33	.20	1.67	.099
	Sex (control)	.17	.20	.86	.390
	Borderline Features at baseline (control)	.01	.01	1.01	.316
Index of Moderated Mediation		*Index*	*Boot SE*	*LL 95% CI*	*UL 95% CI*
		.12	.41	-.39	1.49
Invalidation		*Indirect Effect*	*Boot SE*	*LL 95% CI*	*UL 95% CI*

-1 SD	-.99	-.32	.94	-2.76	1.24
Mean	.00	-.20	.62	-1.73	.84
+1 SD	.99	-.09	.44	-1.47	.48

Note. LL = lower limit; CI= confidence interval; UL = upper limit.

6. Basal RSA as a proposed IV and RSA stress as a proposed mediator. I entered basal RSA as predictor, borderline features at follow-up as outcome, RSA stress as mediator and parental invalidation as the *a* path moderator; child sex and borderline features at baseline were entered as covariates. Piecemeal results are reported above for simple mediation and moderation. The index of moderated mediation was non-significant (*B(SE)*=.02(.18), 95% CI=-.52 to .25).

Table 32. *Moderated Mediation Analysis*

Path	Variable	*B*	*SE*	*t*	*p*
α	Constant to RSA Stress	6.82	.37	18.30	.000
	Basal RSA to RSA Stress	.82	.06	13.80	.000
β	Constant to Borderline Features at FU	7.40	13.08	.57	.572
	RSA Stress to Borderline Features at FU	1.37	1.70	.81	.421
c'	Direct Effect	-1.31	1.70	-.77	.442
	Invalidation to RSA Stress	.07	.06	1.16	.249
	Interaction of Basal RSA *Invalidation	-.01	.06	-.19	.853
	Sex (control)	-.05	.12	-.45	.653
	Borderline Features at baseline (control)	-.00	.01	-.78	.437

Index of Moderated Mediation		*Index*	*Boot SE*	*LL 95% CI*	*UL 95% CI*
		-.02	.18	-.52	.25
Invalidation		***Indirect Effect***	***Boot SE***	***LL 95% CI***	***UL 95% CI***
-1 SD	-.99	1.13	1.45	-1.52	4.24
Mean	.00	1.12	1.42	-1.57	4.06
+1 SD	.99	1.10	1.43	-1.61	3.98

Note. LL = lower limit; CI= confidence interval; UL = upper limit.

Post Hoc Analyses

The first hypothesis of my study was that emotional vulnerability would predict borderline features concurrently and a change in borderline features prospectively. When

emotional vulnerability was measured via trait NA this hypothesis was supported concurrently. However emotional vulnerability did not predict a change in features over a 6-month period. To examine the change from baseline to follow-up, a paired samples t-test was conducted to compare mean levels of borderline features from baseline to follow up. Borderline features significantly decreased from baseline (M=52.69, SD=9.98) to follow up (M=48.22, SD=11.25) time points ($t(100) = 4.40$, $p = .000$).

CHAPTER IV

Discussion

The goal of my current study was to examine factors that contribute to the development of borderline features in early adolescence. I examined whether child emotional vulnerability, in the forms of temperament and physiological reactivity, interacted with parental invalidation to produce emotion dysregulation. I then examined whether or not emotion dysregulation was related to current and/or subsequent borderline features. In order to do so I recruited a total of 101 adolescents. Three primary hypotheses were tested: 1) emotional vulnerability (in the form of trait NA and basal RSA) would predict borderline features at baseline and follow-up; 2) the relationship between emotional vulnerability and borderline features at baseline and follow-up would be mediated by emotion dysregulation (measured via self-report emotional dysregulation and RSA reactivity); 3) parental invalidation would moderate the effects of emotional vulnerability on emotion dysregulation. Hypotheses were tested cross-sectionally and prospectively. Partial support was found for some cross-sectional analyses, and one prospective analysis. In this section I will discuss my results, their fit with the extant literature, implications and limitations of the study.

Emotional Vulnerability Predicted Borderline Features

Trait NA predicted borderline features at baseline. In my study, I found a direct relationship between emotional vulnerability measured via trait NA and borderline features measured at baseline. This finding is consistent with extant literature that has found an association between temperament and BPD (Bornovalova et al., 2006, Cheavens et al., 2004, Clarkin & Posner, 2005, Gratz, et al., 2008, Joyce et al., 2003;

Paris, 2005; Reich & Zanarini, 2001; Saulsman & Page, 2004). Given that trait NA is characterized as an early tendency to display heightened sensitivity and reactivity to emotional stimuli, and BPD is a disorder characterized by intense emotion dysregulation, it is not surprising that youth who are high in trait NA report elevated borderline features in early adolescence.

When examined prospectively, however, no relationship was found between trait NA and change in borderline features measured at follow-up. To further examine this finding, post-hoc analyses were conducted. A paired samples t-test demonstrated a significant decrease in borderline features at follow-up, which may account for the non-findings prospectively. This decrease in borderline features over a 6-month period suggests that emerging borderline features fluctuate throughout adolescence, in both directions. Given that BPD is considered a personality disorder that is viewed as an emerging pattern of biological, emotional, cognitive, and behavioral indexes, it makes sense that these features may wax and wane with development and maturation. Across development, youth may display borderline features at a given time, however as they acquire additional emotion regulation skills and or have specific protective factors in place in their lives, they may diverge from a trajectory of developing psychopathology. Whereas other youth who initially display borderline features may continue to display problematic behaviors and develop dysfunctional patterns over time, which may lead to an eventual diagnosis of BPD.

Overall, my finding lend partial support to the hypothesis that emotional vulnerability assessed via temperament is an early risk factor for the development of borderline features in early adolescence.

Basal RSA did not predict borderline features at baseline or follow-up.

Results indicated that basal RSA was not related to borderline features measured at baseline or follow-up. Therefore, the current results do not demonstrate that emotional vulnerability in the form of lower basal RSA is related to borderline features in my young adolescent community sample. Although lower basal RSA has been empirically linked to many problematic outcomes (Cohen et al., 2000, 1998; Friedman & Thayer, 1998; Lyonfields, Borkovec, & Thayer, 1995; Rechlin, Weis, Spitzer, & Kaschka, 1994; Thayer, Friedman, & Borkovec, 1996), and some studies have found reduced RSA in individuals with BPD (Kuo et al., 2016, Kuo & Linehan. 2009, Weinberg et al., 2009), there is also evidence that individuals with BPD do not display lower basal RSA in comparison with nonclinical controls (Austin et al., 2007). In this sample, there are several factors that may account for the lack of relationship between basal RSA and borderline features.

First, my study used a community versus a clinical sample. Most of the research that has examined physiological responding in relation to borderline features has done so with adult, clinical samples. In these studies, individuals with clinical diagnoses and impairments display altered patterns of physiological arousal. It is possible that youth in my sample had not yet developed the physiological risk factors and profiles of responding that would make them susceptible to developing borderline features. With only a 6-month follow up, perhaps we did not allow enough time for a change in the development of physiological responding.

Additionally, the level of borderline features reported by participants in my study appeared to be slightly lower than what has previously been reported in community

samples (M=53.27 at baseline and 48.23 at 6-month follow up versus M=59.73 in Crick et al., 2005). Therefore it is possible that the range of borderline features reported here is somewhat restricted, likely due to my recruitment strategy, and thus, we did not have adequate variability in the outcome measure to detect a relationship between RSA and borderline features.

One additional explanation for the non-findings may be that basal RSA only confers risk for borderline features under certain conditions. Specifically among developmental samples, it is important to examine the role of environmental context in understanding the relationship between basal RSA and borderline features. In my study we examined one theoretically driven moderator, parental invalidation, and did not find support for this moderator.

Emotion Dysregulation as a Mediator

Self-reported emotion dysregulation partially mediated the relationship between trait NA and borderline features at baseline

Results indicated that emotion dysregulation measured via self-report, partially mediated the relationship between trait NA and borderline features at baseline. However, all other mediations were non-significant. Emotion dysregulation measured via self-report did not mediate the relationship between trait NA and borderline features at follow-up. Emotion dysregulation measured via self-report did not mediate the relationship between basal RSA and borderline features at baseline or follow up. Additionally, emotion dysregulation measured via RSA reactivity did not mediate the relationship between trait NA and borderline features at baseline or follow-up, nor did it mediate the relationship between basal RSA and borderline features at baseline or follow

up. First I will discuss the one significant finding, then the multiple non-significant findings.

Emotion dysregulation measured via self-report, partially mediated the relationship between trait NA and borderline features at baseline. In this model, emotion dysregulation was assessed using a self-report measure looking at youth's difficulties with emotion regulation. Youth with higher scores on this measure have more difficulty regulating their emotions. My findings indicated that adolescents who reported higher trait NA, those who self-reported as more reactive and as experiencing more negative emotions, also reported increased difficulties with emotion regulation, and this was partially responsible for their development of borderline features. This finding is consistent with Linehan's biosocial theory suggesting that emotion dysregulation is a key feature of BPD. It also indicates that emotion dysregulation is one mechanism through which trait NA and borderline features are related. The fact that emotion dysregulation only partially mediated the relationship between trait NA and borderline features suggests that there are additional factors not assessed in this study by which trait NA is related to borderline pathology, and/or the measure used to assess emotion dysregulation did not fully capture the construct. This is one potential avenue for future research looking at the mechanisms which child temperament relates to borderline pathology.

Emotion dysregulation measured via self-report did not mediate the relationship between trait NA and borderline features at follow-up. Given the lack of association between trait NA and change in borderline features at follow up, there is no existing relationship between these two variables for self-reported emotion dysregulation to mediate.

RSA reactivity did not mediate the relationship between emotional vulnerability and borderline features

When emotion dysregulation was assessed physiologically using RSA reactivity, all mediation results were non-significant. RSA reactivity did not mediate the relationship between trait NA and borderline features at baseline or follow up. Additionally, RSA reactivity did not mediate the relationship between basal RSA and borderline features at baseline or follow up. In this study, RSA reactivity was not associated with trait NA or borderline features at baseline or follow-up. Given the lack of these associations the non-significant mediations are not surprising. Moreover, these null results are not entirely surprising as basal RSA and RSA reactivity have been inconsistently associated with these constructs throughout the literature. Although previous research has indicated that low basal RSA and more vagal withdrawal is seen in psychiatric disorders characterized by emotion dysregulation (Beauchaine, 2001; Porges, 1995, 2001, 2003, 2007) not all studies with individuals with BPD have found this to be true. Austin and colleagues (2007) did not find lower basal RSA in BPD versus control participants, although they did find contrasting trajectories of RSA for BPD versus control participants watching emotional film clips. It is important to note that their sample was solely a community female sample, which limits generalizability of their results. Conversely, Weinberg and colleagues (2009) found that BPD participants exhibited lower basal RSA and decreased RSA over the course of their 15-minute laboratory paradigm. However, during their 5-minute stressor task BPD and control groups did not show differences in RSA reactivity. Weinberg and colleagues hypothesized that perhaps differences in RSA reactivity would have been observed if their stressor had continued for longer than 5-minutes.

Applied to the current study, perhaps a longer stressor period would have resulted in greater RSA reactivity and subsequent associations between RSA reactivity and trait NA and borderline features. Additionally, differences in RSA reactivity and basal RSA examined in relation to BPD have typically occurred within clinical samples. This study did not look at a clinical versus control sample. Therefore we were unable to compare RSA reactivity between individuals with BPD and controls. Thus, it is possible that the expected differences between individuals who endorsed elevated borderline features and those who did not were not observed in this sample.

Parental Invalidation as a Moderator of the Relationship between Emotional Vulnerability and Emotion Dysregulation

Parental invalidation moderated the effect of trait NA on self-reported emotion dysregulation

My results indicated that parental invalidation moderated the relationship between trait NA and emotion dysregulation measured via self-report. All other examined moderations were non-significant. This finding indicated that the lowest level of emotion dysregulation was seen in youth with low trait NA who experienced low parental invalidation, which would be expected. However, adolescents who reported low trait NA and experienced high parental invalidation self-reported greater emotion dysregulation. Adolescents who reported high trait NA, self-reported high levels of emotional dysregulation, regardless of the level of parental invalidation they experienced. This finding suggests that in families where parents displayed low or moderate levels of invalidation, a protective effect was observed such that low trait NA predicted low dysregulation, and lower borderline features. This finding is consistent with the biosocial

theory in the sense that emotional vulnerability does make children more sensitive to environmental context, as well as with empirical findings suggesting that a transactional relationship between child and parent predicts child functioning (Stepp et al., 2014). However, instead of finding support for the idea that higher emotional vulnerability places children at the greatest risk, my study demonstrated that adolescents with low temperamental risk (low trait NA) were more vulnerable to the effects of invalidation. Adolescents with low trait NA thrived in low invalidating environments (they displayed the lowest emotion dysregulation), but struggled in highly invalidating environments (displaying high emotion dysregulation). In contrast, adolescents with high temperamental risk (high trait NA) displayed high emotion dysregulation regardless of their environment (level of parental invalidation). Applied to the development of BPD, this suggests that invalidating parenting has a strong impact on child functioning such that even a child with relatively low temperamental risk can develop emotion regulation difficulties when exposed to invalidation.

Parental invalidation did not moderate the effect of basal RSA on self-reported emotion dysregulation or RSA reactivity

As stated earlier, all other moderations examined were non-significant. Parental invalidation did not moderate the relationship between trait NA and emotional vulnerability measured physiologically via RSA reactivity. Additionally, when emotional vulnerability was measured physiologically using basal RSA, no moderations were found; parental invalidation did not moderate the relationship between basal RSA and emotion dysregulation measured via self-report or RSA reactivity. Given the lack of relationship found between trait NA and RSA reactivity, and basal RSA and self-reported

emotion dysregulation, these non-significant moderations are not necessarily surprising. However, given the negative association between basal RSA and RSA reactivity it was expected that this relationship might have been moderated by parental invalidation; although it was not.

While some studies have demonstrated an effect of invalidation on psychophysiological responding (Shenk & Fruzzetti, 2011), others have not (Woodberry et al., 2008). Critiques of studies that examine the impact of childhood invalidation on emotion regulation or subsequent psychopathology include the reliance on retrospective self-report or cross-sectional designs, limiting inferences that can be drawn. These studies have suggested that in order to determine if invalidation impacts emotion regulation in general and specifically in individuals with BPD, validation and invalidation must be directly manipulated while subjective and psychophysiological responses are measured. My study did not address this limitation and relied on self-report assessment of invalidation. We did not examine how in-vivo invalidation impacts psychophysiological functioning, which may have been more effective. This is also an important avenue for future research.

Moderate Support for Overall Moderated Mediation Model

My final hypothesis was a full moderated mediation model suggesting that emotion dysregulation would mediate the relationship between emotional vulnerability and borderline features, and that parental invalidation would moderate the relationship between emotional vulnerability and emotion dysregulation. This hypothesis was partially supported, cross-sectionally and prospectively, when emotional vulnerability was measured by trait NA, emotion dysregulation was measured via self-reported, and

borderline features were assessed at baseline. No other models were significant. Thus, youth who reported higher trait NA reported more subjective emotion dysregulation on average, and the more emotion dysregulation that was reported, the higher borderline features. However, parental invalidation impacted the relationship between trait NA and emotion dysregulation such that the relationship only held under low and moderate levels of parental invalidation. The implications of these findings and the limitations of the current study are discussed below.

Theoretical and Clinical Implications

The current study has some important theoretical implications. Some support is provided for the biosocial theory of borderline personality disorder that proposes that BPD is the result of a transactional relationship between vulnerability to emotion dysregulation and an invalidating environment. My study indicates that even individuals low in temperamental risk may be more reactive to invalidating environments and go on to develop problems regulating their emotions, and subsequent psychopathology. The current study does not provide evidence for biological vulnerability in BPD in the form of low basal RSA, nor does it support meaningful physiological changes in RSA that are related to temperament or invalidating parenting. This is not to say that these relationships do not exist, but that more research in this domain is necessary.

The current study also has some implications for the psychophysiological literature. One important finding of this study was that self-reported and physiological indices of emotional vulnerability and emotional dysregulation did not correlate in expected ways. Basal RSA was negatively correlated with trait NA indicating that these two constructs are related in a meaningful way. We would expect that higher trait NA

would be related to lower basal RSA since both high trait NA and low basal RSA confer risk for problems and are viewed as trait-level constructs. Basal RSA, however, was not significantly correlated with any other variables in the study. Likewise, RSA reactivity, while negatively correlated with basal RSA, was not significantly correlated with any other study variables. RSA reactivity and self-reported emotion dysregulation were not correlated as expected suggesting that they do not similarly represent emotion dysregulation. The question of how psychophysiological measures map on to subjective experiences is ongoing in the psychophysiological research and there is no definitive answer. This study suggests that there is not a perfect relationship between an individual's subjective report of their emotional responses and physiological responses to stress.

The finding that subjective and psychophysiological variables are uncorrelated is not uncommon (Kuo et al., 2013). In addition to the explanation provided above, it also suggests that perhaps self-report and physiological measures of emotional regulation may be differentially sensitive to various stimuli. Within the current study self-reported emotion dysregulation represented an adolescent's overall view of how they respond to stress/emotionally charged situations. Whereas adolescent's measured physiological emotional dysregulation represented their in-vivo responsivity to a stressor. Although both measurements were used to represent the construct of emotion dysregulation, they do not actually represent the same thing. Perhaps it would have been more theoretically sound to assess adolescent's subjective emotional experience before, during, and directly following the stressor task as a measure of emotion dysregulation that more precisely maps on to RSA reactivity. Additionally, although polyvagal theory makes hypotheses

about vagal tone and its relationship to emotion regulation capacities, RSA is an imperfect measure (Grossman, Karemaker, & Wieling, 1991; Grossman & Kollai, 1993) and there continue to be inconsistent findings with RSA (Morales et al., 2015).

The current study also has implications for how issues related to temperament (specifically trait NA), parental invalidation, and emotion dysregulation can be addressed in clinical practice. My study suggests that interventions for children with highly invalidating parents and children are important, because invalidation can negatively impact a child with low temperamental risk. It also suggests that intervention should focus on child emotions/behaviors and parenting techniques and strategies in response to children's emotions and behaviors. Trait NA and parental invalidation are easily identifiable risk factors and can be quickly and reliably assessed through clinical interview or objective ratings, and may provide important insights and directions for therapists in terms of therapeutic interventions. Based on my findings, addressing child and parent factors as well as the parent-child relationship is important in terms of preventing emotion dysregulation and subsequent borderline pathology.

Limitations

This study has a number of limitations. First, it was overly reliant on self-report questionnaire measures to assess temperament, emotional dysregulation, and borderline features. While questionnaire measures are easily administered and widely used in the social sciences, they contain weaknesses related to reporter bias and issues of validity. Additionally, the variability in borderline features and invalidation was quite limited, likely due to using a community sample. Moreover, our symptom follow-up was only 6-months which may not have been a long enough period of time to measure important

changes in the development of borderline features. Furthermore, this study only considered one emotional vulnerability/individual child risk factor for the development of borderline personality disorder. However, other risk factors have been suggested. Impulsivity is another trait characteristic that has been empirically linked to the development of borderline pathology in childhood/adolescence. Much research has focused on early life trait impulsivity and its relationship to emotion dysregulation and emerging borderline pathology (Belsky et al., 2012; Bornovalova, Hicks, Iacono, & McGue, 2013; Hinshaw et al., 2012; Stepp et al., 2012).

Another limitation of this study is the sample, including both age of participants and recruitment strategies used to obtain the final sample population. My sample consisted of young adolescent (mean age = 12.82, SD=0.83). While some research has exampled borderline features in younger adolescent samples (Crick et al., 2005, Stepp et al., 2010) the majority of studies tend to examine features in older adolescent populations, such as high schoolers (Chabrol et al., 2001; Chabrol et al., 2004; Winograd et al., 2008; Wright et al., 2016). As mentioned earlier, children borderline features tend to be are less stable in younger children, especially at the lower end of symptom severity. Thus fact that my young sample had lower symptom severity overall is likely related to their younger age, and the reduced severity likely impacted the relationship between borderline features and other study variables such as RSA. For example, at low levels, borderline personality disorder features are not necessarily problematic and are not associated with clinical problems. Therefore the expected relationships between borderline pathology and emotion dysregulation found in the clinical literature are unlikely to hold up within this population.

Additionally, due to this study being embedded in a larger study with specific exclusion criteria (discussed above), our sample did not include adolescents that would most likely have been at the high end of the borderline scale, displaying the most severe symptoms. By excluding these participants my study was not able to examine the relationship between young adolescents who are experiencing the most clinically severe borderline symptoms, emotional vulnerability, and psychophysiology. A wider range of borderline features and inclusion of these specific individuals may have led to findings more consistent with the clinical literature linking borderline features to lower basal RSA and increased RSA reactivity, specifically.

One methodological consideration to take into account when interpreting the results of this study has to do with the use of stress induction tasks. My study makes the assumption that youth's physiological response to an induced laboratory stressor provides insight into that youth's response to a real life stressor. Bush and colleagues (2011) examined changes in RSA across social, cognitive, sensory, and emotional challenge tasks. They found that individuals reacted significantly differently depending on the type of task, therefore suggesting that the type of stressor can alter findings across studies. Studies specifically examining BPD individuals have also examined the type of stimuli used to elicit negative emotions/stress. Currently there is no consensus on which methods are the most effective at eliciting emotional reactivity (Kuo et al., 2013). However some studies have noted differences in RSA depending on the type of stimulus used (Kuo et al., 2013).

It is also important to take into account the conceptual and measurement overlap in the constructs related to emotional vulnerability and dysregulation. For example,

negative affectivity can be conceptualized as a measure of reactivity related to emotion dysregulation (Rothbart & Posner, 2006) as well as a trait measure of general risk of psychopathology (trait NA). In addition, Basal RSA and RSA reactivity are commonly used interchangeably as measures of emotional capacity and regulatory abilities, both conceptually and statistically (Sulik, Eisenberg, Silva, Spinrad & Kupfer, 2012). The construct overlap and conflicting findings in the literature suggest that the model proposed in the current study may be imperfect. This does not diminish the findings in this study; instead it suggests that more research related to these constructs is indicated.

Future Research

As stated earlier, adolescents in this study were not asked to rate their emotional experiences or disclose regulation strategies they implemented during the stress induction. Future investigations of emotion regulation processes in the development of BPD may want to assess for regulation strategies or emotional experiences before, during, and after a stress/emotion induction. This may allow for a better understanding of self-report and physiological patterns and may lead to more convergence between the two forms of measurement.

Future studies may also want to assess invalidation in multiple ways. In addition to obtaining self-report measures on parental responses, collecting in-vivo data on how invalidating feedback impacts an individual's emotional and physiological functioning may be helpful in elucidating the relationship between these variables. Overall, additional prospective, longitudinal studies are important to more rigorously examine the development of borderline pathology

Conclusion

This study sought to test the biosocial model of BPD in a young adolescent sample to better understand the development of the disorder. Results indicated that child temperament, specifically trait NA, and invalidating parenting interact to produce emotional dysregulation, which is related to increased borderline pathology among adolescents. However, the study did not implicate the involvement of physiological vulnerabilities and patterns of responding in the development of borderline features. This study suggests that understanding the risk for the development of borderline features in adolescence needs more rigorous and continued research, particularly in understanding the biological risk and role of psychophysiological responding to stress in the development of the disorder. Further exploration of how these variables are related will be important in understanding the etiology of borderline features across development.

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