The Role of Maternal Invalidation in the Development of BPD symptoms in Adolescent Girls

by

Diana Jean Whalen

BS, University of Pittsburgh, 2005

MS, University of Pittsburgh, 2009

Submitted to the Graduate Faculty of the Kenneth P. Dietrich School of Arts and Sciences

in partial fulfillment

of the requirements for the degree of

Doctor of Philosophy

University of Pittsburgh

2014

UNIVERSITY OF PITTSBURGH

DIETRICH SCHOOL OF ARTS AND SCIENCES

This dissertation was presented

by

Diana Jean Whalen

It was defended on

April 8, 2013

and approved by

Michael Sayette, Professor, Psychology, University of Pittsburgh

Daniel S. Shaw, Professor and Department Chair, Psychology, University of Pittsburgh

Stephanie D. Stepp, Assistant Professor, Psychiatry and Psychology, University of Pittsburgh

Elizabeth Votruba-Drzal, Assistant Professor, Psychology, University of Pittsburgh

Dissertation Advisor: Jennifer S. Silk, Associate Professor, Psychiatry and Psychology, University of Pittsburgh

The Role of Maternal Invalidation in the Development of BPD symptoms in Adolescent Girls

Diana Jean Whalen, PhD

University of Pittsburgh, 2014

The Role of Maternal Invalidation in the Development of BPD symptoms in Adolescent Girls

Diana J. Whalen, PhD

University of Pittsburgh, 2014

Specific parenting mechanisms contributing to feelings of emotional invalidation in children and adolescents have not been adequately explored in the development of borderline personality disorder (BPD). The work in this area has been limited by cross-sectional studies using retrospective reports of adult samples and a limited range of parenting measures, which may be subject to bias. This study extends previous work by exploring associations between maternal invalidation and adolescent symptoms of BPD, with a short-term longitudinal design involving three assessments across one year, at baseline (Time 1), 6 months (Time 2), and 12 months (Time 3). Observational data were collected on 74 mother-adolescent dyads during a structured conflict discussion task at Time 2. It was hypothesized that invalidating maternal responses during the structured conflict discussion, at Time 2, would be associated with BPD symptoms at Time 3, even after controlling for BPD symptoms at Time 1. Results indicated that critical maternal behaviors during the structured conflict discussion task were predictive of BPD symptoms 6 months later, even after controlling for initial BPD symptoms, indicating that invalidating parenting may play a role in the maintenance and development of BPD among at-risk adolescents. Lower positive dyadic behavior also predicted BPD symptoms 6 months later at trend levels, suggesting that the mother-adolescent relationship may also contribute to the development of BPD. Future directions and clinical implications are discussed.

TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

1.0 INTRODUCTION

Borderline personality disorder (BPD) is a severe psychological disorder impacting millions of lives each year (Grant et al., 2008). Individuals with BPD experience broad dysfunction across emotions, behaviors, relationships, and sense of self. BPD is associated with considerable functional impairment, including social stigma, reduced treatment response, elevated risk for suicide, as well as poor social, occupational, and academic outcomes (Bagge et al., 2004; Bagge, Stepp, & Trull, 2005; Bender et al., 2001; Skodol, Pagano, et al., 2005; Soloff, Lynch, & Kelly, 2002; Trull, Useda, Conforti, & Doan, 1997; Zweig-Frank & Paris, 2002). With suicide rates almost 50 times higher than the general population (Holm & Severinsson, 2011), BPD is a major public health problem of enormous scale and concern. Given this, it is somewhat surprising that little empirical work has focused on the development and maintenance of BPD.

Twenty years ago, Linehan (1993) articulated a biosocial model for the development of BPD, stating that complex transactions between a child's pre-existing emotional vulnerability and family environments experienced as invalidating by the child lead to BPD. Despite prominence in the etiological literature, components of this theory, such as familial invalidation have gone largely untested. This project tested the familial invalidation component of Linehan's model in a sample of adolescent girls and their mothers. This project was unique in several ways. First, the project measured BPD symptoms across three time points, spanning one year. Next, it included observed, rather than retrospectively reported measures of invalidation. The project

aimed to operationalize invalidation in a more specific and concrete way than prior work. By capturing invalidation in the moment, this study was able to identify specific aspects of parenting and the mother-adolescent relationship that contribute to a child's sense of invalidation. In this project, it was hypothesized that the following invalidating parenting behaviors will be associated with BPD: increased critical parenting, increased dismissive parenting, dyadic negative escalation, as well as lower positive relationship qualities in the dyad.

1.1 DEFINING BORDERLINE PERSONALITY DISORDER

Diagnostic symptoms of BPD include: (1) frantic efforts to avoid abandonment, (2) a pattern of unstable and intense interpersonal relationships characterized by alternating between extremes of idealization and devaluation, (3) markedly and persistently unstable self-image or sense of self, (4) chronic feelings of emptiness, (5) transient, stress-related paranoid ideation or severe dissociative symptoms, (6) recurrent suicidal behavior, gestures, threats, or self-mutilating behavior, (7) impulsivity in at least two areas that are potentially self-damaging, (8) affective instability due to a marked reactivity of mood, and (9) inappropriate, intense anger or difficulty controlling anger (American Psychiatric Association, 2000). Even after symptoms of BPD remit, those with prior diagnoses still experience clinically significant emotion dysregulation and functional impairment in their social relationships (Choi-Kain, Zanarini, Frankenburg, Fitzmaurice, & Reich, 2010).

Three symptom clusters have been identified from these nine criteria, namely affect dysregulation, impulsivity or behavioral dysregulation, and interpersonal dysfunction (Sanislow et al., 2002). Most empirical work focuses on affect dysregulation and/or impulsivity in those

with BPD (Crowell, Beauchaine, & Linehan, 2009). By contrast, few studies have investigated experiences of familial invalidation, despite clinical suggestions that patterns of dysfunctional relationships may uniquely characterize BPD (Gunderson, 2007). Familial invalidation can lead an individual to doubt their social perceptions, creating difficulties in forming and maintaining interpersonal relationships (Stanley & Siever, 2009).

Impairment from BPD symptoms often begins in adolescence, suggesting that symptoms of the disorder are likely to be detectable at an earlier age (DSM-IV-TR; APA, 2000). Specific features of BPD, such as self-harm, impulsivity and affective instability, often present during childhood and/or adolescence and are predictive of receiving a BPD diagnosis as an adult (Siever, Livesley, Gunderson, Pfohl, & Widiger, 2002; Zanarini et al., 2006). In adolescent samples, dimensional measures of psychopathology have been shown to be more reliable and valid than categorical measures (Markon, Chmielewski, & Miller, 2011). In addition, the field appears to be moving toward a continuous as opposed to categorical classification system for personality disorders (Miller, Morse, Nolf, Stepp, & Pilkonis, 2012; Trull, Distel, & Carpenter, 2011; Widiger, 2006). It has been suggested that dimensional models of personality disorders better account for the vast heterogeneity among patients with BPD and diminish the unclear diagnostic boundaries between BPD and other disorders (Blashfield & Intoccia, 2000; Clark, 2007; Trull & Durrett, 2005).

Developmental models of BPD outline several precursors to the disorder, including affective dysfunction, impulsivity, and relationship disturbances in at-risk children (Crick, Murray-Close, & Woods, 2005; Crowell et al., 2009; Putnam & Silk, 2005). One component of these models is that children at-risk for the development of BPD may possess genetic predispositions for emotion vulnerability and dysregulation, as well as other BPD traits.

Research has shown genetic linkages to both BPD and BPD symptoms (Lis, Greenfield, Henry, Guilé, & Dougherty, 2007; Skodol, Siever, et al., 2002). Heritability is high for both BPD and traits associated with BPD, such as neuroticism and negative emotionality (Nigg & Hill, 1994; White, Gunderson, Zanarini, & Hudson, 2003). In a large scale, multi-national twin study, BPD features were found to be moderately genetically influenced (42%), similar across the three countries, and lacked gender differences (Distel et al., 2008). Environmental factors unique to the individual accounted for the remaining 58% of the variance in BPD features, suggesting that environmental factors also play an important role in the development of BPD (Distel et al., 2008).

In addition to the potential genetic linkages of BPD, research has also shown that strong gender disparities exist in the diagnosis. BPD is especially damaging to women; women with BPD are disproportionally represented in clinical populations and are major consumers of health care resources (Eaton et al., 2008; Grant et al., 2008; Lieb, Zanarini, Schmahl, Linehan, & Bohus, 2004; Skodol, Gunderson, et al., 2005; Skodol & Bender, 2003; Soeteman, Hakkaart-van Roijen, Verheul, & Busschbach, 2008; Zanarini, Frankenburg, Khera, & Bleichmar, 2001). Furthermore, results from a nationally representative sample suggest that women with BPD suffer from more mental and physical disability than men with BPD (Grant et al., 2008). Thus, it is particularly important to study the development of BPD in adolescent girls, who appear to be at a greater risk for the disorder and functional impairment resulting from the disorder than adolescent boys.

1.2 DEFINING INVALIDATION

Transactional processes between an emotionally vulnerable child and a home environment perceived as invalidating by the child, where both the child and environment influence each other over time, are theorized to contribute to the development of BPD (Linehan, 1993, p. 39). The constructs of emotional vulnerability and familial invalidation have yet to be empirically validated in the development of BPD. According to Linehan, an invalidating environment is one, "in which the communication of private experiences is met by erratic, inappropriate or extreme responses" (Linehan, 1993, p. 49). Linehan's model asserts that invalidating parenting styles trivialize, punish, and/or intermittently reinforce the child's personal experiences and emotional expression. Over time, the child learns to distrust his or her internal emotional compass and begins to look toward the environment for guidance on how to respond. Although this definition is often cited, it is important to define invalidation in a way that can be more easily operationalized in research. One reason that research on Linehan's concept of invalidation has lagged behind work on the other critical tenants of her theory is the difficulty in defining and methodologically capturing invalidation. In this project, we used observations of parent-adolescent interactions to assess specific parenting behaviors and dyadic relationship factors that are believed to reflect familial invalidation.

Invalidating environments can be characterized by high levels of critical and dismissive responses to a child's emotional experiences and expressions (Fruzzetti, Shenk, & Hoffman, 2005; Linehan, 1993). For example, a child may experience invalidation when parents criticize (e.g., "it's no big deal") or punish (e.g., stop crying, or I'll give you a reason to cry") their

emotional expression. In addition, invalidation can occur when the child's emotional expression is ignored or dismissed, and parents fail to notice and acknowledge the child's emotions. In contrast to parental responses that are more supportive, invalidation fails to reflect to the child that his or her response is acceptable and understandable. These types of invalidating statements can be particularly detrimental for children who possess pre-existing emotionally vulnerable (Linehan, 1993).

Generally, research on invalidating parenting has been defined under two broad categories: critical and dismissive (Crowell et al., 2009; Eisenberg, Spinrad, & Eggum, 2010). In this project, critical aspects of parenting included: high levels of negative affect, dominance, and conflict. Dismissive aspects of parenting included: higher levels of denial, task avoidance, and withdrawal. It was hypothesized that both high levels of critical parenting and high levels of dismissive parenting will contribute to the development of BPD in adolescence. These invalidating parenting behaviors include responses that dismiss or ignore a child's emotions, punish emotional disclosure, or blame or criticize the child for his/her emotional experience. Consequently, labeling internal experiences becomes difficult for the child and instead, their emotional reactions are grounded in cues from their social environment (McMain, Korman, & Dimeff, 2001).

In addition, it is critical to recognize that invalidating parenting behaviors are both a contributing factor in the development of BPD and a response to BPD symptoms in youth. Children with BPD features may behave in ways that make validation difficult or impossible. At times, critical and dismissive parenting responses may be both appealing and effective in order to help the child cope with overwhelming emotions. Because of this, it is important to study these transactional processes at a dyadic level, rather than at the individual level of the parent or

adolescent. For example, a transactional escalation of negative affect may characterize the emotional communication between adolescents at-risk for BPD and their mothers. At other times, a dismissive response may serve to de-escalate the child's emotions. Both the mother and adolescent can exacerbate each other's negative affect and behavior, creating a snowball effect. In addition, the opposite may also be true, that positive emotions stagnate and neither partner is able to build positivity or support for the other. Low relationship quality, mutuality, and satisfaction are additional key transactional processes that may contribute to and/or result from an individual's sense of invalidation and have yet to be studied in the development of BPD. Using this definition, it is expected that negative escalation, low relationship quality, and mutuality will also be related to the development of BPD symptoms.

1.3 RETROSPECTIVE STUDIES OF FAMILIAL INVALIDATION

Several studies have tested aspects of Linehan's theory, however, invalidation has not been consistently operationalized or measured in a methodologically rigorous fashion (Cheavens et al., 2005; Reeves, James, Pizzarello, & Taylor, 2010; Rosenthal, Cheavens, Lejuez, & Lynch, 2005; Sauer & Baer, 2009). Overall, invalidation was typically defined as a sense of not being heard or understood by one's parents as a child and usually assessed retrospectively in a self-report questionnaire completed by adults with BPD symptoms. For example, Cheavens and colleagues (2005) focused on one type of invalidation, perceived parental criticism and measured this through a retrospective report using the parental criticism subscale of the Multi-dimensional Perfectionism Scale (Frost, Marten, Lahart, & Rosenblate, 1990). The authors found that in a nonclinical sample of 202 college students, perceived parental criticism was a predictor of BPD

symptoms. The relationship between parental criticism and BPD was strengthened when the young adults reported using thought suppression as an emotion regulation strategy. These results suggest that parental criticism may be important to study in the development of BPD.

Another study from the same research group (Rosenthal et al., 2005) used childhood sexual abuse as a proxy for invalidation and considered this to be a distal risk factor in the development of BPD. The authors found that both childhood sexual abuse and negative affectivity predicted BPD symptoms in a community sample of adults, but that negative affectivity was a stronger predictor. The two studies reviewed above were cross-sectional in design, and thus it is unclear whether invalidating environments would have predicted BPD across time. Prospective data are needed to directly test this relationship.

Reeves and colleagues (2010) used a large, female undergraduate sample (*n*= 1044) to test Linehan's biosocial theory. They found support for both emotion dysregulation and emotional vulnerability being associated with BPD symptoms. However, invalidation was not a significant predictor. The authors defined invalidation according to a questionnaire measure: the Socialization of Emotion Scale (SES) developed by Krause, Mendelson, & Lynch (2003). This scale assesses a respondent's memories of his or her caregivers' responses to the respondent's emotional displays as a child. The questionnaire presents 12 scenarios (e.g., a child crying after an accident) and six potential parental responses to each scenario. Respondents rate how likely their maternal and/or paternal caregiver was to give each type of response. Krause et al. (2003) identified the Distress Reactions, Punitive Reactions, and Minimization Reactions scales as corresponding to Linehan's concept of invalidation. However, these scales, individually or as a total score, were not able to predict increases in BPD symptoms in the sample.

Using a sample of undergraduates (*n*= 104), Sauer and Baer (2009) found that self-reported childhood emotional vulnerability and self-reported levels of parental invalidation in childhood were related to current BPD symptoms. Similarly to Reeves and colleagues (2010), they defined invalidation according to the Socialization of Emotion Scale (SES; Krause, Mendelson, & Lynch, 2003). Their findings also replicated the work of Cheavens and colleagues (2005) in that thought suppression and fear of emotions completely mediated the relationship between invalidating environments and BPD symptoms. One limitation of this work, however, is that all assessments occurred at a single point in time and the assessment of invalidation was retrospective.

Extending their previous work, Sauer and Baer (2010) used additional self-report measures that assess both emotional vulnerability and invalidation using 519 undergraduate students. In this study, parents were also asked to retrospectively report on their child's emotional vulnerability and their parenting style using the Coping with Children's Negative Emotions Scales (CCNES; Fabes et al., 2002). They found that both measures were correlated with current BPD symptoms. However, invalidation was not a significant predictor of BPD symptoms when childhood emotional vulnerability was added to the equation.

Although the studies reviewed above represent an important first step in developing empirical support for the invalidation construct of Linehan's theory, conclusions that can be drawn from these studies are limited by the use of retrospective reports, a single assessment, and questionnaires as the only measure of the construct of invalidation. Furthermore, all of the studies used college student or young adult samples. It may be necessary to investigate parental invalidation earlier in development. There have been only a few studies that have examined

familial invalidation using prospective and independent methods. This literature is reviewed below.

1.4 PROSPECTIVE STUDIES OF FAMILIAL INVALIDATION

Prospective studies of invalidating family environments and BPD have focused on factors such as neglect, abuse, and parental criticism and dismissiveness (Bandelow et al., 2005; Bradley, Zittel Conklin, & Westen, 2005; Carlson, Egeland, & Sroufe, 2009; Johnson et al., 2001) all of which imply to the child that their emotional experiences, expressions and perceptions are not acceptable. Parenting practices that focus on the child's emotional displays, such as criticism and dismissiveness may be especially relevant to the development of BPD given the nature of both affective and interpersonal dysfunction often found in the disorder. Often, families do not discuss abuse and neglect and the child's feelings about such events can be ignored. While abuse and neglect may represent more severe and damaging forms of invalidation, the message sent to the child by other less extreme forms, such as parental dismissiveness, is the same: you are not important, worthy, or accurate; the feelings you have, you shouldn't have; the behaviors you do, you shouldn't do.

An invalidating environment is likely shaped by specific characteristics of both the caregiver and the child and their interactions over time (Linehan, 1993; Crowell, et al., 2009). In one of the only prospective longitudinal studies of BPD, Carlson et al (2009) found that BPD symptoms could be predicted from early emotion and environmental variables measured from infancy through adolescence. These included abuse, neglect, family stress as well as child-centered problems with attachment, behavior, emotion-regulation, and self-regulation. These

findings underscore the importance of incorporating both child and dyadic effects into models of BPD development. However, the dyadic or transactional nature of invalidation has yet to be studied in BPD. Studies that have assessed relations between BPD, abuse, neglect, and parenting practices are reviewed below.

1.4.1 BPD, Child Abuse, and Neglect

Relations between BPD and childhood abuse/neglect are well researched. Abusive and neglectful environments are hypothesized to be invalidating not only due to the nature of the act, but also because the child's interpretation of and emotions related to the abuse or neglect is frequently ignored or met with disbelief. Zanarini and colleagues (1997) found that 92% of patients with BPD retrospectively reported the experience of parental neglect and emotional invalidation before the age of 18, with emotional invalidation being a significant, unique predictor of a BPD diagnosis. Both neglect and emotional under-involvement (e.g., not caring about what the child is doing, feeling, or thinking) by caretakers, an extreme form of emotional invalidation, appear to contribute to the development of BPD (Soloff et al., 2002). In an older sample of college students, neglect, emotional, and sexual abuse predicted borderline personality traits (Igarashi et al., 2010). Neglect sends the message that the child is not important or worthy of attention. Over time, the child may internalize this message and begin to feel that their thoughts or emotions are not worthy of expression, understanding, or regulation, potentially leading to emotional inhibition.

In a prospective study, Widom and colleagues (Widom, Czaja, & Paris, 2009) used 500 documented cases of child physical and sexual abuse as well as neglect to determine whether these children were at an increased risk for BPD in adulthood. Results confirmed their

hypotheses in that significantly more abused and/or neglected children met criteria for BPD as adults, compared to demographically matched controls. Interestingly, several parental factors mediated the relationship between childhood abuse/neglect and BPD including, having a parent dependent on drugs and/or alcohol, unemployed, with less than a high school education, and/or with a diagnosis of drug abuse, major depressive disorder, and posttraumatic stress disorder. More recent evidence replicated these findings and suggests that this type of childhood adversity, such as witnessing domestic violence, parental incarceration, and parental suicide attempts are strongly and consistently associated with several Cluster B personality disorders, including BPD (Afifi et al., 2011).

Childhood sexual abuse is arguably one of the most invalidating events a person can experience. In BPD populations, sexual abuse has been reported to be as high as 75% in both inpatient and outpatient samples (Battle et al., 2004; Silk, Lee, Hill, & Lohr, 1995) and there is evidence that the prevalence of childhood sexual abuse is higher in BPD than in other disorders. For example, a history of childhood sexual abuse has been shown to discriminate between BPD populations and depressed, non-BPD populations in both adolescents and adults (Horesh, Orbach, Gothelf, Efrati, & Apter, 2003). In a Norwegian longitudinal study, childhood abuse and neglect, combined with fewer protective factors, such as creativity or academic talent, differentiated between adults with BPD and a healthy control group of adults (Helgeland & Torgersen, 2004).

Extending the research using questionnaire assessments of abuse, a recent meta-analysis of fMRI studies (Nunes et al., 2009) found that BPD patients had smaller hippocampal volumes than healthy controls, particularly in those with a history of childhood abuse. Moreover, in BPD patients but not in controls, right hippocampal volumes were negatively associated with

aggressiveness. This suggests that patients with BPD who had experienced abuse in childhood were also likely to be more aggressive in adulthood.

Despite the amount of work linking BPD symptoms to abuse and neglect, these relations do not appear to be specific. Many abused and neglected children do not develop psychopathology, or they may develop other disorders apart from BPD. For example, 90% of childhood sexual abuse victims do not go on to develop BPD. While many individuals with BPD report abuse and neglect in their childhoods, these experiences are neither necessary nor sufficient for the development of BPD. Complex interactions are likely to exist among genetic vulnerabilities, personality traits, and other factors, such as parental invalidation, abuse, and neglect.

1.4.2 BPD and Parenting Practices

The biosocial model describes the importance of parental responses to emotional displays throughout development. Specific parenting practices or parenting behaviors defined by both content and socialization goals (Morris, Silk, Steinberg, Myers, & Robinson, 2007) can also contribute to invalidation, such as ignoring and/or negating emotions, intermittently reinforcing extreme emotional displays, and oversimplifying problems (Linehan, 1993). Research has shown that parents play a key social role in the acquisition and refinement of skills and strategies for experiencing and regulating affect (Eisenberg, Cumberland, & Spinrad, 1998; Gottman, Katz, & Hooven, 1997; Morris et al., 2007). In normative samples, parental criticism or invalidation of children's emotions has been associated with social and emotional difficulties in early childhood (Eisenberg et al., 2010; Fruzzetti et al., 2005; Silk et al., 2009) and psychological distress in adulthood (Krause et al., 2003). Furthermore, family environments characterized by conflict and

aggression combined with relationships that are unsupportive and/or neglectful represent vulnerabilities that are hypothesized to disrupt emotional processing and psychological functioning (Repetti, Taylor, & Seeman, 2002; Silk et al., 2007). These family environments and parenting styles could be characterized as invalidating toward the child or adolescent. While widely researched in Axis I disorders, less attention has been given to Axis II disorders, such as BPD.

Parental responses to emotion deliver explicit and implicit messages to children about appropriate ways to experience and regulate emotions (Eisenberg, Fabes, & Murphy, 1996), and these responses may be related to the development of BPD. Parental responses to children's emotions have been broadly characterized as supportive, critical, and dismissive. Supportive responses to children's affect encourage and acknowledge the child's emotional expressions, as well as model adaptive emotion regulation strategies. For example, a parent might use positive listening and speaking skills that demonstrate an understanding of the child's perspective or in adolescence, promote a reasoned discussion of a disagreement.

Parents who are uncomfortable with emotional expression or who feel unable to manage their own affect may minimize or criticize their children's emotional expression (Gottman, Katz, & Hooven, 1996). Critical parental responses are visibly negative responses to children's emotions and include mocking, threatening, or punishing the child for displaying affect. Dismissive responses include minimizing, ignoring or trivializing the child's emotion expression or encouraging the child to ignore or suppress her feelings. Over time, dismissive and critical responses interfere with the adaptive development of emotion expression and regulation strategies by encouraging the child to suppress affect and use avoidant and/or aggressive regulation strategies (Eisenberg et al., 1996). These responses can become intermittently

reinforcing to the child, since a parent may begin by ignoring the child's emotion and later, as the child has become more dysregulated, responds in a highly critical or dominant manner. Thus, BPD symptoms, particularly those associated with emotional avoidance or aggressive behaviors, may develop when parents respond to children's affective experiences with dismissive or critical responses.

Despite the fact that the role of peers increases during adolescence, the family continues to play a critical socializing role (Morris et al., 2007). Parents represent key socializing agents throughout development and continue to shape an adolescent's emotional experiences both direct and indirectly. In adolescent depression, there is evidence suggesting that family relations more strongly predict adolescent outcomes than peer relations (Stocker, Richmond, Rhoades, & Kiang, 2007). Furthermore, family relationships seem to be an even stronger predictor of psychopathology and related outcomes for girls than boys (Adrian et al., 2009; Repetti et al., 2002).

Parental invalidation has been linked to adolescent distress, behavior problems, general psychopathology, as well as depression (Fruzzetti et al., 2005). Maternal overprotection, lack of emotional availability, and rejection has been correlated with BPD symptoms in adulthood, leading to an impaired ability to reflect on the emotional states of others (Ghiassi, Dimaggio, & Brune, 2010). In a clinical intervention study, teaching mothers of adolescents with BPD to be less invalidating toward their adolescent was associated with improvements in the adolescents' depressive symptoms, self-esteem, and relationship satisfaction (Fruzzetti, Shenk, & Hoffman, 2005). Using data from one of the two longitudinal studies of personality disorders, Reich and Zanarini (2001) found that adult patients with BPD remembered more mood reactivity, poorer

frustration tolerance, and more difficulties with separation from caretakers between ages 6 and 17.

The Children in the Community Study (CIC; Cohen, Crawford, Johnson, & Kasen, 2005) has provided a longitudinal prospective account of parenting, parental psychopathology, and child outcomes, and was one of the only studies to look at child rearing in parents with Axis II disorders, but not specifically BPD. In ten recent papers using self-report data from late childhood and early adolescence, they have shown that: (1) parental personality disorder is associated with problematic parenting behaviors; (2) maladaptive parenting behaviors are predictive of later personality disorder symptoms in offspring; and (3) parental personality disorder is associated with symptoms and disorders in offspring (Bezirganian, Cohen, & Brook, 1993; Cohen, Chen, et al., 2005; Cohen, Crawford, et al., 2005; Johnson et al., 2001; Johnson, Cohen, Brown, Smailes, & Bernstein, 1999; Johnson, Smailes, Cohen, Brown, & Bernstein, 2000; Johnson et al., 2002). For example, Johnson and colleagues (2002) found that invalidating parental behaviors such as ignoring, neglecting, or dismissing the child's emotions impairs the parents' ability to socialize emotions effectively, which in turn is associated with increased child suicidality, a common symptom of BPD. In addition, children who experienced verbal abuse were more than three times likely to suffer from a PD in adolescence and adulthood, including BPD (Johnson et al., 2001). These results remained significant even after controlling for other child and parent variables such as emotionality, psychopathology, and other forms of abuse/neglect. More recent work from this project has shown that both harsh punishment and low levels of parental affection were associated with the development of BPD and BPD symptoms in adolescence and adulthood (Johnson et al., 2006).

Aspects of parenting have also been investigated among self-harming adolescents, a portion of whom are likely to develop BPD during later adolescence or adulthood. During a conflict discussion task, families of self-harming adolescents demonstrated less positive affect, more negative affect, and lower mutuality compared with a control group of adolescents who did not self-harm (Crowell et al., 2008). Family relationships characterized by a lack of support for managing emotions combined with higher levels of conflict have also been associated with adolescent emotion dysregulation and self-harm (Adrian, Zeman, Erdley, Lisa, & Sim, 2011). Yap and colleagues (Yap et al., 2011; Yap, Allen, & Ladouceur, 2008) have found that adolescents reported using more maladaptive emotion regulation strategies if their mothers reported using invalidating emotion socialization practices, such as criticism or neglect response to their adolescents' positive emotion expressions. More maladaptive emotion regulation strategies reported by adolescents were also associated with mothers who displayed aversive behavior, such as dominance and withdrawal during a structured mother–adolescent interaction. This type of work involving conflict discussions and emotion-related parenting practices have yet to be extended to Axis II conditions in adolescent samples.

From a theoretical view, parents can invalidate the emotional expressions of their children, especially when the parent responds with criticism or neglect. This may in turn lead to children who inhibit or question their emotions and emotional responses. A chronic invalidation of emotional experiences may disrupt the adaptive development of emotion processing systems. As a result, the child may experience increases in BPD symptoms, including affect instability, high sensitivity to emotional stimuli, increased intensity of emotional states, and a slow return to emotional baseline. Despite its place as a major construct in Linehan's theory, maternal invalidation and specific dyadic processes associated with invalidation remain largely untested.

Invalidating family environments that involve abuse, neglect, and several emotion-related parenting practices have shown to be associated with BPD. However, relatively little is known about what invalidation may look like as it is occurring, during interpersonal interactions and how it is experienced by the adolescent at that moment. In fact, there have been no direct face-to face assessments of invalidation in those with or at-risk for BPD. The proposed study will assess maternal and dyadic aspects of invalidation, as they occur, during a structured discussion task.

1.5 ADOLESCENCE IS A CRITICAL TIME TO STUDY BPD

Research has largely ignored the development of personality disorder symptoms. It is clear that on the morning of their 18th birthday, individuals do not wake with a personality disorder that was not present the day before. BPD has been theorized to begin around puberty; however most clinicians and researchers are reluctant to make the diagnosis until adulthood (Zanarini et al., 2001). Longitudinal work suggests that BPD symptoms peak between adolescence and young adulthood and tend to burn out by middle age (Paris, 2003). When compared to adults with BPD, BPD symptoms in adolescents are equally reliable, stable, and valid (Chanen, Jovev, McCutcheon, Jackson, & McGorry, 2008; Chanen & Kaess, 2011; Westen, Shedler, Durrett, Glass, & Martens, 2003) and can be diagnosed with equal frequency (Becker, Grilo, Edell, & McGlashan, 2002). However, the criteria for diagnosing BPD differ at the two ages: affective instability and anger are the best diagnostic predictors for adolescents, whereas impulsivity may be the best diagnostic predictor for adults (Becker et al., 2002). Thus, there is a critical need for research on symptoms of personality disorders before the full diagnostic criteria are met.

Adolescence represents an ideal and critical time to study the development of personality disorders, and BPD specifically. During adolescence, there are widespread changes in brain maturation, socialization, and identity development. Several symptoms of BPD are likely to cause functional impairments during adolescence, creating adverse consequences that are difficult for the adolescent to overcome, given the differences in specific brain circuitry at this age. For example, adolescents who experience intense levels of negative emotions that take longer than average to dissipate may turn to self-damaging behavioral responses to control these emotions (Silk, Steinberg, & Morris, 2003). Deliberate self-harm may even be used to quell these intense emotional experiences.

The vast numbers of physical, cognitive, and emotional changes that occur during this transitional period make adolescence a critical time to study the development of BPD. Furthermore, many problematic trajectories, including relationship difficulties, may solidify during adolescence (Dahl, 2004). Even in relatively healthy adolescents, the shifts across many facets of day-to-day experience create emotionally intense situations, which strain the adolescent's developing emotion regulation skills. During adolescence, individuals are able to more flexibly engage in cognitive processing, depending on the social and motivational context (Crone & Dahl, 2012). These findings have been used to help explain why adolescents find a variety of risky behaviors, such as reckless driving and binge drinking more appealing. In healthy adolescents, however, these situations can present as opportunities for personal growth and emotional development.

On the other hand, for those adolescents who will go on to development of BPD, these normative challenges may lead to emotions with even greater levels of intensity, and when coupled with invalidating environments, may alter the adolescent’s flexibility in engaging the

neural systems underlying emotion processing and sensitivity to social and affective influences (Crone & Dahl, 2012). It would be hypothesized that adolescents who later develop BPD would experience affect instability around several negative emotions, such as anger, sadness, and shame, above and beyond that of healthy adolescents. For example, adolescents who will later go on to develop BPD may be less accurate or inefficient at processing these emotions, and may have memory biases toward social situations involving these emotions than adolescents who do not go on to develop BPD (Cole, Luby, & Sullivan, 2008). If an adolescent feels intense anger, he or she may be likely to act out in aggression toward the self, perhaps through deliberate self-harm, and aggression toward others through both physical and relational acts. Following this behavioral outburst, the same adolescent may also feel intense shame over the consequences of the actions. Feeling intense shame after anger maintains a negative focus on the self and may impede social relationships (Cole et al., 2008). Over time, this pattern of affect instability combined with differences in brain functioning and environmental invalidation is likely to impair social and emotional development.

Childhood BPD symptoms, such as affective instability and relationship disturbances are expected to show variable pathways into adulthood ranging from resilience to pathology. However, for some children, these symptoms may be indicative of risk for later BPD and warrant increased research attention. Furthermore, the presence of symptoms combined with environmental invalidation may be especially problematic for children and adolescent at-risk for BPD (Stepp, Olino, Klein, Seeley, & Lewinsohn, 2013). There has been little work on children specifically at-risk for the development of BPD. Gratz and colleagues (Gratz et al., 2009) found support for the independent and interactive roles of affective dysfunction and sensation seeking in childhood BPD symptoms. Children aged 9-13 and their primary caretakers completed

questionnaires that assessed personality traits, emotion regulation, ego control, risk taking, sensation seeking, and psychopathology. Caregiver reported deficits in self and emotion regulation partially mediated the relation between affective dysfunction and child BPD symptoms. This study was the first to examine these relationships in children at-risk for BPD. Crick and colleagues (Crick et al., 2005) also found that emotional sensitivity uniquely predicted borderline personality features in a community sample of children over time. Results also indicated that relational aggression predicted BPD features in children over time, as changes in relational aggression were uniquely associated with changes in BPD features. Both studies suggest that the same emotional deficits theorized to be central to BPD in adulthood are also associated with childhood borderline personality symptoms. As stated earlier, general deficits in self and emotion regulation strategies have also been shown to prospectively contribute to the development of BPD (Carlson et al., 2009).

Female adolescents in particular may represent an ideal population in which to study the development of BPD. A BPD diagnosis is three times more common in females than males (Bjorklund, 2006), and this discrepancy is theorized to persist throughout development. In clinical settings, 75% of those with BPD are female (Skodol & Bender, 2003). Although there have been few empirical studies of adolescent females who develop BPD, it can be hypothesized that they represent a group of greater risk. Girls are especially vulnerable to emotional disorders during adolescence. Research suggests that adolescent girls are at higher risk for emotional distress during adolescence than adolescent boys (Hilt, Cha, & Nolen-Hoeksema, 2008). Before puberty, girls and boys exhibit similar rates of depression (Angold, Costello, & Worthman, 1998). However, during puberty and throughout adolescence, girls become twice as likely to experience a major depressive episode (Conley & Rudolph, 2009). Other risk factors for BPD,

such as sexual abuse have been shown to be more common among females than males (McClellan, Adams, Douglas, McCurry, & Storck, 1995). In terms of BPD criteria, adolescent girls are more likely to report affect instability whereas adolescent boys are more likely to endorse impulsivity (Aggen, Neale, Røysamb, Reichborn-Kjennerud, & Kendler, 2009). Furthermore, research has shown that adolescent girls present with BPD symptoms that are more consistent with the adult BPD diagnosis than adolescent boys, who are more likely to present with aggressive and antisocial symptoms (Bradley et al., 2005). Given the increases in affective dysregulation and other risk factors during adolescence, female adolescents represent an ideal population for investigating the development of BPD.

1.6 SIGNIFICANCE

This study was the first observational assessment investigating the roles of maternal and dyadic facets of invalidation in predicting future BPD symptoms, measuring the construct before the onset of a full BPD diagnosis. The study tested Linehan's (1993) assertion that invalidating aspects of the adolescent's environment would predict increases in BPD symptoms over time. Here, invalidating maternal responses were defined as critical and dismissive parenting behaviors that could be directly observed. It is likely that not all critical and dismissive parenting is interpreted as invalidating by the adolescent; therefore one aim of this study was to determine the more precise markers of invalidation during conflict: Which specific maternal behaviors, during a structured conflict interaction, would be associated with BPD symptoms in the future? As such, invalidating maternal responses, particularly more critical and dismissive responses to adolescent's emotions and behavior during a structured interaction task were investigated. In

addition, the transactional or dyadic aspects of invalidation, such as negative escalation and low relationship quality were explored. There have been no studies examining the face-to-face interactions of adolescents with BPD features. Furthermore, this is the first study of which I am aware that empirically tested this component of Linehan's biosocial model (1993) in adolescents prior to the onset of the disorder.

1.7 HYPOTHESES

Invalidating maternal responses during a conflict discussion were hypothesized to be associated with BPD symptoms in adolescent girls. In this project, invalidating responses included both critical (intense negative affect) and dismissive (withdrawing from the conflict) parenting behaviors. Furthermore, dyadic aspects of invalidation, including negative escalation and low dyadic positive relationship qualities were explored. Maternal invalidation was hypothesized to predict BPD symptoms in adolescent girls across one year. Furthermore, it was hypothesized that these findings would hold after controlling for the girls' initial BPD symptoms, measured at Time 1.

H1: Invalidating maternal responses at Time 2 (6 months) were expected to be associated with BPD symptoms at Time 3 (1 year).

H1a: Critical maternal responses, coded as negative affect, dominance, and conflict were expected to be associated with BPD symptoms at Time 3.

H1b: Dismissive maternal responses, coded as denial, task avoidance, and withdrawal were expected to be associated with BPD symptoms at Time 3.

H1c: Dyadic codes of negative escalation, low relationship quality, and low mutuality were expected to be associated with BPD symptoms at Time 3.

H2: Invalidating maternal responses at Time 2 were expected to be associated with BPD symptoms at Time 3, while controlling for baseline levels (Time 1) of BPD symptoms.

H2a: Critical maternal responses, coded as negative affect, dominance, and conflict were expected to be associated with BPD symptoms at Time 3, while controlling for baseline levels of BPD symptoms.

H2b: Dismissive maternal responses, coded as denial, task avoidance, and withdrawal were expected to be associated with BPD symptoms at Time 3, while controlling for baseline levels of BPD symptoms.

H2c: Dyadic codes of negative escalation, low relationship quality, and low mutuality were expected to be associated with BPD symptoms at Time 3, while controlling for baseline levels of BPD symptoms.

2.0 METHOD

2.1 PARTICIPANTS

74 adolescent girls and their mothers participated in the project (Total n= 148). The adolescents (all age 16) and their mothers were recruited from the Girls Personality Study (Dr. Stepp's K Award, n =110). The first 74 girls enrolled in the Girls Personality Study were asked to enroll in the proposed study. In the K01 study, 110 16-year old girls and their biological mothers were assessed 4 times across 18 months using psychiatric interviews and questionnaires.

Participants were recruited from the Girls Personality Study (PI: Stephanie Stepp) through the ongoing Pittsburgh Girls Study (PGS; R01 MH56630, PI: Rolf Loeber). This multiple cohort, prospective study investigates the developmental precursors and risk factors for conduct problems and co-morbid conditions in a population sample of inner-city girls (Hipwell et al., 2002; Keenan et al., 2010). This high-risk sample was comprised of 2,451 girls who were between 5 and 8 years old at the start of data collection in 2000/01. Participants in the current project consisted of those girls who turned 16 years of age in 2010-2012 and their biological mothers.

The demographic characteristics of the sample for this study are presented in Tables 8-10. Overall, the sample was diverse in terms of race and socio-economic status. Twenty-six participants were Caucasian (35.13%) and 48 were African American (64.86%). The majority of mothers completed 12 or more years of education (*N*= 68; 92%). A substantial minority of the families in this study reported receiving public assistance (43.2%, *N*= 32).

2.1.1 PGS Recruitment Procedure

The participants of the main PGS were recruited from a sample of 103,238 households in the City of Pittsburgh. Of the 2,875 potential participants, 2,451 agreed to participate in the first phase of the longitudinal study, yielding a recruitment rate of 85.3%.

2.1.2 PGS Sample Characteristics

At the time of the first interview, 588 of the girls were five, 630 were six, 611 were seven, and 622 were eight years old. Fifty-two percent of the sample is African-American, 42% is Caucasian, and the remaining girls are described as multi-racial or another race. Seventy percent of the sample reported 12 or more years of education and 32% reported receiving some type of public assistance.

After families completed the PGS annual interview for the adolescent's 16 year-old assessment, all girls completed the affect instability section of the PAI-BOR. Girls' scores were calculated into groups (high/low) for the purpose of recruitment from the larger study only. Girls scoring higher than 12 were part of the high AI group and girls scoring 11 or lower were part of the low AI group. Dr. Stepp's project aimed to recruit an equal number of high and low AI girls. This project includes the first 74 girls who participated in the Girls Personality Study, regardless of AI scores.

2.2 PROCEDURES

The University of Pittsburgh Institutional Review Board approved all procedures (IRB # PRO09100184). Mothers and adolescents enrolled in Dr. Stepp's study participated in an additional 15-minute assessment during their Time 2 interview, approximately six months following study enrollment.

	Completed By:		**Time 1 Baseline**	**Time 2 6 months**	**Time 3 12 months**	**Time 4 18 months**	**Domain Assessed**
Name of Measure	**Mother**	**Girl**					
Interview with Evaluator							
Structured Clinical Interview for DSM-IV Personality Disorders	X	X	X	X	X	X	Personality disorder symptoms
Parent-Adolescent Interaction							
Hot Topics	X	X		X			Invalidation

Table 1. Dr. Stepp's Assessment Schedule with Interaction Task

Adolescents were informed of study procedures and provided informed assent; mothers provided consent for adolescent participation. This project incorporates data from their initial Time 1 assessment, as well as assessments at Time 2 and 3. During Time 2, mothers and daughters were interviewed for Axis II psychopathology and participated in a structured conflict discussion task. Mothers and their daughters also completed two later assessments as part of Dr. Stepp's project (12 and 18 months) following the initial visit.

2.3 MEASURES

2.3.1 Axis II Psychopathology

The Structured Interview for DSM-IV Personality Disorders (SIDP-IV) was used to assess Axis II disorders at Times 1, 2 and 3 in the adolescents (Pfohl, Blum, & Zimmerman, 1997). Each adolescent and her mother were interviewed to determine the adolescent's current Axis II symptoms and diagnoses. Parents and adolescents were interviewed separately, with clinical interviewers integrating data from both informants and using clinical judgment to arrive at a final diagnosis (Pfohl, Blum, & Zimmerman, 1997). Typically, if parents and adolescents provided differing information for a criterion, the higher of the two scores was used. The questions are grouped into 10 areas of functioning (e.g., close relationships, work style, perception of others) rather than by diagnoses. Following the interview, each criterion is rated on a 4-point scale (0 = not present; 1 = subthreshold features; 2 = clearly present, clinically significant; 3 = prominent symptom). Scores of 2 or higher are necessary to consider the criteria present. Dimensional scores were calculated for each diagnosis by summing the component

criterion scores (0 to 4). The dimensional scores for each personality disorder were used as an index of personality disorder. In prior work, dimensional scores of personality disorders have been shown to have greater test-retest reliability and stability, particularly in adolescent samples (Chanen et al., 2004; Clark, 2009; Crick et al., 2005).

At each 6-month follow-up assessment, participants were re-interviewed using the SIDP-IV. Again, mothers and adolescents were interviewed separately with clinicians integrating data from both informants. The interview period covered the time from the last assessment up until the time of the interview. An expert administrator trained all interviewers on the assessment of personality disorders using the SIDP-IV. Reliability meetings were held with the interviewers where all interviewers watched video and independently rated the interviews. Any disagreements were discussed and a clinical consensus was reached. Inter-rater agreement was calculated on each of the 9 criteria for BPD using Intra-class correlations (ICC). ICC for each BPD criteria ranged from .54 to 1 (M = .71; SD= .14).

2.3.2 Familial Invalidation: Mother-Daughter Behavioral Interaction

Mothers and daughters were videotaped while completing a structured discussion task designed to elicit typical displays of negative affect. Mother-daughter dyads interacted during a "hot topics" task designed to elicit conflict and negative emotion (O'Connor, Hetherington, Reiss, & Plomin, 1995). First, mothers and daughters completed a brief questionnaire about common areas of conflict among adolescents and their mothers. Dyads were asked to discuss the conflict rated most highly in terms of frequency and severity by both members of the dyad during an 8-minute videotaped discussion (Furman & Shomaker, 2008; McMakin et al., 2011).

2.4 CODING

The Interactional Dimensions Coding System-Revised IDCS-R (Furman & Shomaker, 2008) system includes both individual and dyadic codes and was used to code the mother-daughter interactions. The IDCS-R is an observational coding system that was originally designed to assess couples' interactions during problem solving and was modified for use with adolescents (Furman & Shomaker, 2008). A team of trained coders, blind to each participant's interview data and study hypotheses, rated observable behavior, facial expressions, and the verbal content of each interaction. Tapes were randomly assigned to each coder. Participants were rated on a five-point Likert scale with half-point intervals (1= *extremely uncharacteristic* to 5= *extremely characteristic*) for individual and dyadic codes.

Code	Rated	Definition
1. Positive Affect	1-5 rating	Positivity of facial expressions, body positioning, and emotional tone
2. Negative Affect	1-5 rating	Negativity of facial expressions, body positioning, and emotional tone
3. Problem Solving	1-5 rating	Define a problem and work toward a satisfactory solution
4. Denial	1-5 rating	Rejection of a problem's existence or personal responsibility
5. Dominance	1-5 rating	Achievement of control or influence over partner
6. Task Avoidance	1-5 rating	Avoidance of the assigned discussion topic or task
7. Support/ Validation	1-5 rating	Positive listening/speaking skills that demonstrate understanding
8. Conflict	1-5 rating	Expressed struggle between two people with incompatible goals
9. Withdrawal	1-5 rating	Affect and behavior designed to avoid interacting with the partner

10. Communication skills	1-5 rating	Individual's ability to convey thoughts and feelings in a constructive manner
11. Promoting Autonomy	1-5 rating	Behaviors aimed at promoting a reasoned discussion of a disagreement
12. Support Seeking	1-5 rating	Behaviors aimed at increasing care from partner
13. Positive Escalation	1-5 rating	A sequential pattern where positive behavior from one partner is followed by positive behavior from the other partner, creating a snowball effect
14. Negative Escalation	1-5 rating	A sequential pattern where negative behavior from one partner is followed by negative behavior from the other partner, creating a snowball effect
15. Mutuality	1-5 rating	The dyad's sense of 'we-ness' and reciprocity
16. Relationship Quality	1-5 rating	General health of the relationship
17. Satisfaction	1-5 rating	Degree to which the pair would consider themselves happy in the relationship

Table 2. Coding System

There are 12 scales on which the adolescent and mother were coded: overall positive affect, overall negative affect, problem solving, denial, dominance, task avoidance, support-validation, conflict, withdrawal, communication skills, promoting autonomy, and support seeking. Each adolescent and mother received separate scores. It was hypothesized that mothers who exhibit higher levels of invalidation would show greater negative affect, dominance, and conflict during the interactions. These mothers were also expected to display higher levels of denial, task avoidance, and withdrawal.

In addition, the five dyadic codes include: positive escalation, negative escalation, mutuality, relationship quality, and satisfaction. The mother and daughter in each dyad receive the same score on each of these codes. It was hypothesized that negative escalation, low mutuality, and low relationship quality during the interactions would predict BPD symptoms.

A trained coding team comprised of a master, reliability coder and several research assistants completed all conflict discussion task coding. Twenty-one percent of the tapes were coded by all members of the team and were used to calculate inter-rater agreement. Each of the coders was compared to the "reliability coder." Intra-class correlations coefficients for the codes of interest ranged from .69 to .93 (M = .79; SD= .07), consistent with other research using the IDCS-R coding system (Furman & Shomaker, 2008; McMakin et al., 2011).

3.0 DATA SCREENING AND PREPARATION

3.1 INCOMPLETE/MISSING DATA

All 74 participants completed the SIDP-IV at Time 1. 74 also completed the SIDP-IV at Time 2 and 60 completed the SIDP-IV at Time 3. All 74 participants completed the hot topics questionnaires and mother-adolescent discussion at Time 2.

3.2 NORMALITY ANALYSIS

Skew, kurtosis, and the Kolmogorov-Smirnov test of normality were used to explore univariate normality for all study variables. For BPD symptoms, an examination of the histograms and normality plots indicated that both W1 and W3 BPD symptoms were positively skewed. This finding was anticipated given the low base rate of BPD in the general population. Log transformations were performed on these variables.

3.3 POWER

The Gpower program was used to calculate statistical power (Faul, Erdfelder, Lang, & Buchner, 2007). With an N of 74 subjects (total of 148 mothers and daughters) and three to four predictor variables, this sample will have power above .8 to detect medium to large effects (f^2=

.15 to 35) using multiple regression. Tabachnick & Fidell (2007) also provide a formula for calculating sample size within a multiple regression framework that takes into account the number of independent variables used: N > 50 + 8m (where m= the number of independent variables). Using this formula, this study has enough power to incorporate three predictor variables. With an n of 60 subjects, who completed the W3 assessment, this study has power above .8 to detect only large effects.

3.4 PRINCIPAL COMPONENTS ANALYSIS OF INTERACTION CODES

In order to reduce the number of variables, an exploratory principal components analysis was conducted on the maternal and dyadic interaction codes hypothesized to be associated with the development of BPD. For maternal behaviors, the 6 codes hypothesized to be indicative of invalidation (negative affect, denial, dominance, task avoidance, conflict, and withdrawal) were subjected to principal components analysis (PCA). A separate PCA was performed on the five dyadic codes (negative escalation, mutuality, relationship quality, satisfaction, and positive escalation). Prior to performing PCA, data was determined to be suitable for analysis, as an inspection of the correlation matrix revealed the presence of several coefficients .3 and above.

For maternal codes, the principal components extraction method revealed the presence of two components with eigenvalues exceeding 1, explaining 36.57% and 34.52% of the variance respectively. An inspection of the screeplot revealed a clear break after the second component. The two-component solution explained a total of 71.09% of the variance. To aid in the interpretation of these two components, varimax rotation was performed. The rotated solution revealed the presence of simple structure, with both components showing several strong loadings

and all variables loading substantially on only one component. The interpretation of the two components was consistent with the hypothesized maternal behaviors: critical and dismissive. Negative affect, conflict and dominance loaded strongly on the 'critical' factor, whereas withdrawal, task avoidance, and denial loaded strongly on the 'dismissive' factor.

Item	Component		Rotated Component		Communalities
	Component 1	Component 2	Component 1	Component 2	
Negative Affect	.87	-	.87	.14	.77
Conflict	.83	-.2	.86	-	.74
Dominance	.58	-.44	.67	-29	.54
Withdrawal	.13	.86	-	.87	.76
Task Avoidance	-	.87	-.18	.85	.76
Denial	.61	.57	.45	.71	.76

Table 3. Pattern and Structure Matrix with Varimax Rotation of Two-Factor Solution of Maternal Codes

For dyadic codes, PCA revealed the presence of two components with eigenvalues exceeding 1, explaining 67.17% and 21.06% of the variance respectively. An inspection of the screeplot revealed a clear break after the second component. The two-component solution explained a total of 88.24% of the variance. To aid in the interpretation of these two components, varimax rotation was performed. The rotated solution revealed the presence of simple structure,

with both components showing a number of strong loadings and all variables loading substantially on only one component. The interpretation of the two components revealed negative and positive relationship components. Mutuality, positive escalation, relationship quality, and satisfaction loaded strongly on the 'positive' factor, whereas negative escalation loaded strongly on the 'dismissive' factor.

Item	Component		Rotated Component		Communalities
	Component 1	Component 2	Component 1	Component 2	
Negative Escalation	-.23	.96	-.06	.99	.98
Mutuality	.88	.3	.92	.15	.87
Positive Escalation	.89	.09	.89	-.06	.79
Relationship Quality	.93	.03	.93	-.13	.87
Satisfaction	.93	-.16	.89	-.31	.89

Table 4. Pattern and Structure Matrix with Varimax Rotation of Two-Factor Solution of Dyadic Codes

3.5 PRELIMINARY ANALYSES

Descriptive statistics and intercorrelations for BPD symptoms and maternal codes are presented below. To identify potential covariates, preliminary analyses were also conducted to

examine the influence of the following demographic factors: race, maternal education, and public assistance on the variables of interest (e.g. maternal codes, dyadic codes and BPD symptoms). All analyses were performed with IBM SPSS Statistics, version 20.

3.5.1 Descriptive Statistics

Descriptive statistics for BPD symptoms and ratings for conflict discussion codes are presented below in Figures 1-2 and Tables 5-6.

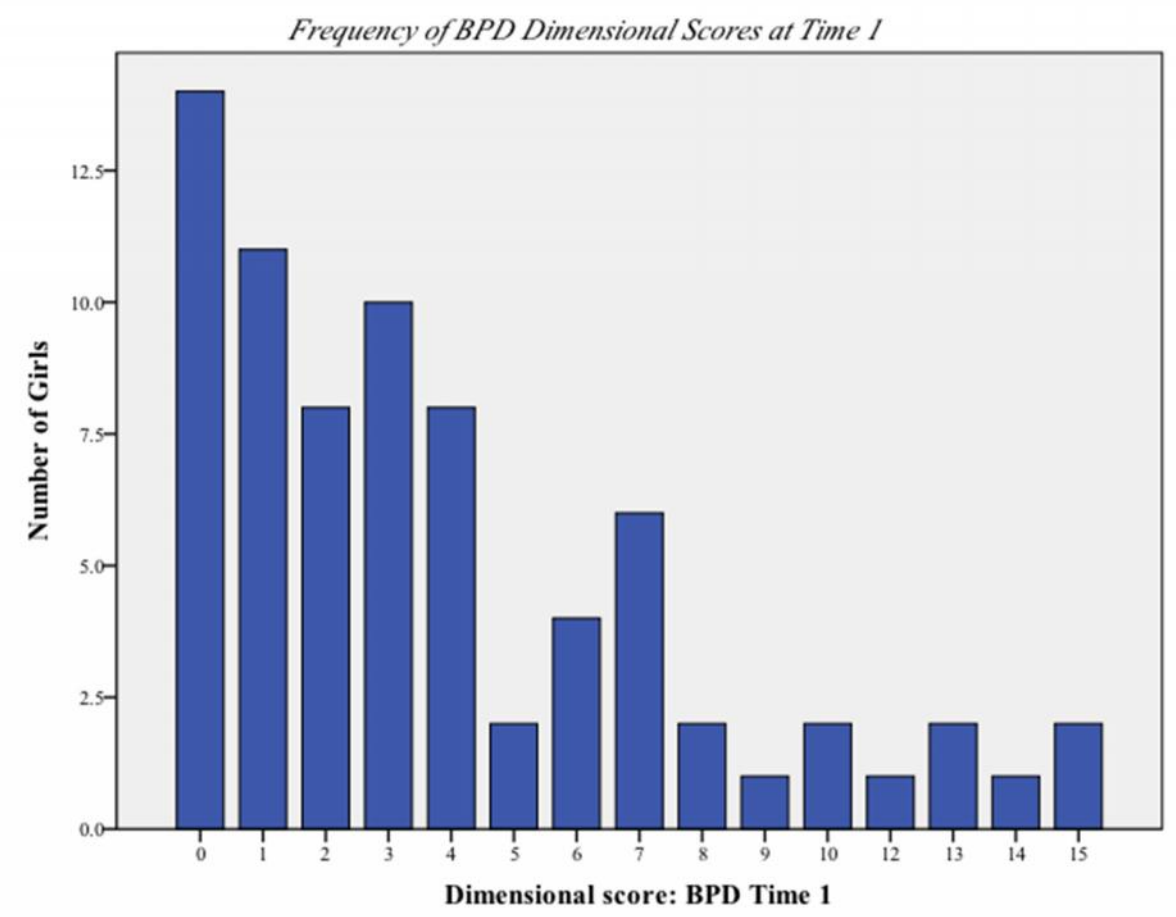

Figure 1. Frequency of BPD Dimensional Scores (untransformed) at Time 1

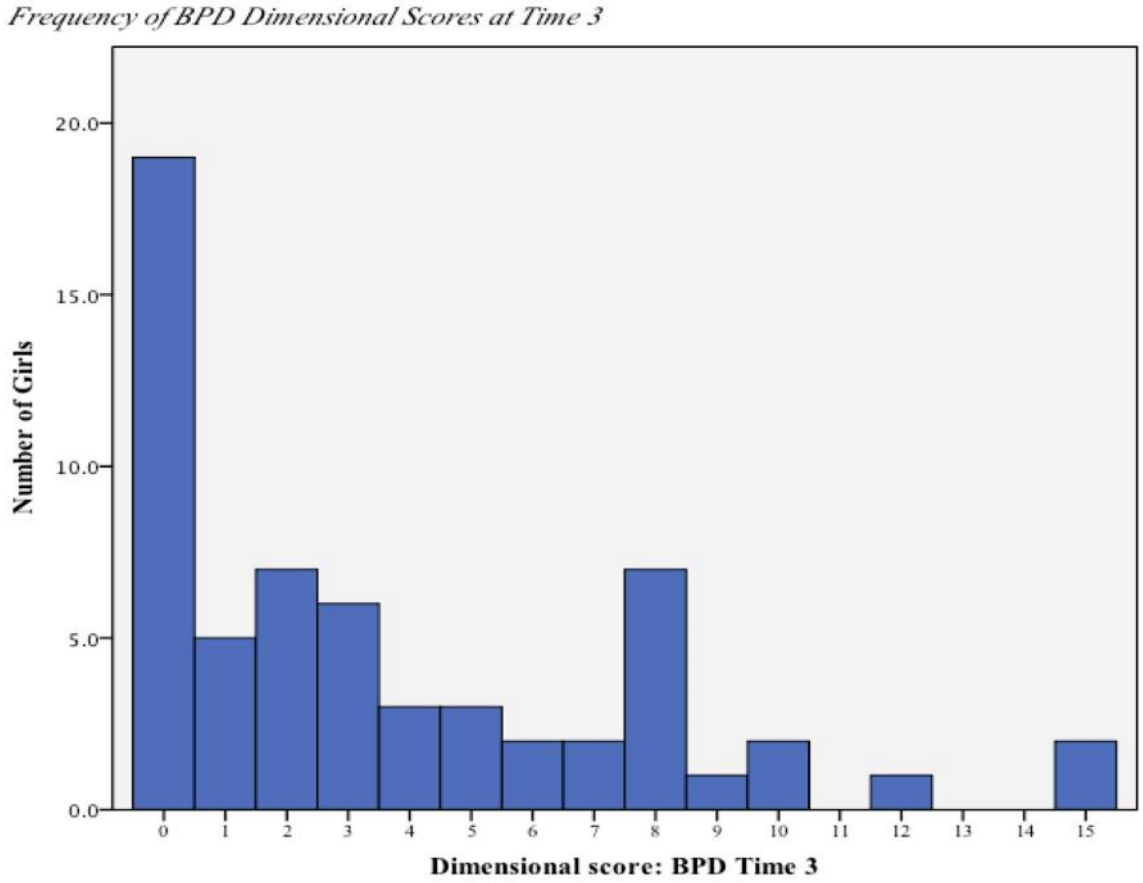

Figure 2. Frequency of BPD Dimensional Scores (untransformed) at Time 3

Variable	Mean	*SD*	Range
Dimensional Score: BPD T1	3.95	3.9	0-15
Dimensional Score: BPD T3	3.62	3.95	0-15

Table 5. Descriptive Statistics for BPD Symptoms

	Variable	Mean	*SD*	Range
Individual Codes	Negative Affect	2.78	.69	1.5-5
	Denial	1.78	.85	1-5
	Task Avoidance	1.75	.89	1-5
	Dominance	2.38	.82	1-4.5
	Conflict	2.5	.76	1-5
	Withdrawal	1.55	.70	1-4
	Relationship Quality	2.62	.95	1-5
	Satisfaction	2.76	.89	1-5
	Positive Escalation	2.02	.86	1-4
	Mutuality	2.67	1.03	1-5
Factors	Critical Factor	7.65	1.84	4-14.5
	Dismissive Factor	5.08	1.99	3-12
	Dyad: Negative Escalation	2.32	.85	1-5
	Dyad: Positive Factor	10.07	3.44	4-18.5

Table 6. Descriptive Statistics on Coded Variables

3.5.2 Intercorrelations

Intercorrelations between BPD symptoms and interaction codes are presented in Table 7.

	Dimensional score: BPD T1	Dimensional score: BPD T3	Critical factor	Dismissive factor	Dyad: Negative escalation	Dyad: Positive Factor

Dimensional score: BPD T1	--					
Dimensional score: BPD T3	.67**	--				
Critical factor	.26*	.37**	--			
Dismissive factor	.04	.03	.02	--		
Dyad: Negative escalation	.29*	.35**	.67**	-.13	--	
Dyad: Positive Factor	-.24*	-.33**	-.36**	-.35**	-.16	--

*p<.05; **p<.01

Table 7. Intercorrelations Between BPD symptoms and Interaction Codes

3.5.3 Covariate Analysis

To identify potential covariates, preliminary analyses were conducted to examine the influence of the following demographic factors: race, maternal education, and public assistance on the variables of interest (e.g. maternal and dyadic codes and BPD symptoms).

Tables 8-10 present the results of independent-samples t-tests conducted to compare the BPD dimensional scores and conflict discussion codes by race, maternal education, and a family's receipt of public assistance. Receipt of public assistance was assessed annually as part of the larger Pittsburgh Girls Study. Families were asked whether or not they had received any form of public assistance, such as TANEF or WIC in the past year. Responses were dichotomously coded as 'yes' or 'no.' There was no significant difference in BPD scores or conflict discussion codes for white and minority participants (Table 8). There were also no

significant differences in BPD scores or codes when mothers had less than 12 years of education compared to mothers with 12 or more years of education (Table 9). BPD dimensional scores at time 1 were higher among those girls whose families received public assistance (*M*= 5.13 *SD*= 4.39) compared to those whose families did not receive public assistance, *M*= 3.05 *SD*= 3.33; $t_{(72)}$ = -2.31, p<.05 (Table 10). Therefore, public assistance was included as a covariate in analyses for hypothesis 2.

		White (*n*=26)		Minority (*n*=48)			
	Variable	*M*	*SD*	*M*	*SD*	*t*	*p*
Coding Variables	Critical Factor	7.77	1.55	7.59	1.99	.39	.70
	Dismissive Factor	4.71	1.59	5.28	2.16	-1.18	.24
	Dyad: Negative Escalation	2.33	.75	2.32	.91	.02	.98
	Dyad: Positive Factor	10.35	3.68	9.93	3.28	.50	.62
Symptom Variables	BPD symptoms Time 1	3.38	3.74	4.25	4.05	-.9	.37
	BPD symptoms Time 3	3.90	4.23	3.48	3.9	.4	.70

Table 8. Descriptive Statistics and t-Tests comparing White to Minority Mothers on Hypothesized Variables of Interest

	Variable	<12 years Education (*n*=6)		>=12 years Education (*n*=68)		*t*	*p*
		M	*SD*	*M*	*SD*		
Coding Variables	Critical Factor	7.42	1.28	7.67	1.89	.33	.74
	Dismissive Factor	6.5	3.2	4.96	1.83	-1.85	.07
	Dyad: Negative Escalation	2.25	.93	2.33	.85	.22	.82
	Dyad: Positive Factor	9.33	2.66	10.14	3.47	.55	.58
Symptom Variables	BPD symptoms Time 1	6.33	5.43	3.74	3.76	-1.56	.12
	BPD symptoms Time 3	6.20	4.38	3.38	3.86	-1.55	.13

Table 9. Descriptive Statistics and t-Tests comparing Mothers with low education to Mothers with higher levels of education on Hypothesized Variables of Interest

		No Assistance (n=42)		Received Assistance (n=32)			
	Variable	*M*	*SD*	*M*	*SD*	*t*	*p*
Coding Variables	Critical Factor	7.57	1.73	7.76	2.01	-.45	.66
	Dismissive Factor	4.81	1.83	5.44	2.16	-1.35	.18
	Dyad: Negative Escalation	2.29	.82	2.36	.90	-.31	.76
	Dyad: Positive Factor	10.68	3.46	9.28	3.21	1.77	.08
Symptom Variables	BPD symptoms Time 1	3.05	3.33	5.13	4.39	-2.31	.02*
	BPD symptoms Time 3	2.79	3.44	4.63	4.34	-1.79	.08

***p<.05**

Table 10. Descriptive Statistics and t-Tests comparing Families who received Public Assistance to Families not receiving Public Assistance on Hypothesized Variables of Interest

3.6 ANALYSIS PLAN: HYPOTHESIS 1

To investigate whether invalidating maternal responses at Time 2 were related to BPD symptoms at Time 3, simple linear regressions were conducted in SPSS. This allowed for the investigation of the independent effects of specific maternal invalidation factors and dyadic codes on BPD symptoms. Separate regressions were run for the hypothesized codes of interest: critical factor, dismissive factor, negative escalation, and dyadic positive factor. β weights and R^2 are reported.

3.7 ANALYSIS PLAN: HYPOTHESIS 2

Adolescent BPD symptoms at Time 1 were controlled when investigating hypothesis 2, whether invalidating maternal responses are related to BPD symptoms at Time 3. In these models, public assistance was entered as a covariate. Again, β weights and R^2 are reported.

3.8 ANALYSIS PLAN: HIERARCHICAL REGRESSIONS

To investigate whether invalidating maternal responses or dyadic codes were uniquely related to BPD symptoms at Time 3, hierarchical linear regressions were conducted in SPSS. This allowed for the investigation of the combined effects of specific maternal invalidation and dyadic codes on BPD symptoms.

First, the maternal critical factor was entered into the regression equation. Following this, any dyadic factors found to be significantly related to BPD symptoms in the prior regression analyses were entered. β weights and changes in R^2 are reported. Adolescent BPD symptoms at Time 1 and receipt of public assistance were controlled when investigating hypothesis 2, whether invalidating maternal responses are related to the development of BPD symptoms across one year. Again, β weights and changes in R^2 are reported.

4.0 RESULTS

4.1 CLINICAL CHARACTERISTICS

The BPD dimensional scores of the sample are summarized above in Figures 1-2 and Table 5. At Times 1 and 3, BPD dimensional scores ranged from 0-15, indicating a low to moderate level of symptoms. At Time 1, 5 girls (6.7%) were at or above the diagnostic threshold for BPD (5 or 6 out of 9 symptoms) and an additional 5 girls (6.7%) had 4 of the 9 required symptoms. At Time 3, 5 girls (8.3% of sample) were above the diagnostic threshold for BPD (5 out of 9 symptoms) and an additional 8 girls (13.3% of sample) had 4 of the 9 required symptoms. Time 1 BPD symptoms were strongly correlated with Time 3 BPD symptoms (r= .67, p< .001). 17 girls (out of 60) experienced an increase in BPD symptoms from Time 1 to Time 3. Group comparisons (e.g. high and low BPD) were not made, as BPD is theorized to exist on a continuum.

All adolescents and their mothers completed a brief questionnaire that assessed the frequency and severity of common conflicts that occur between adolescents and their parents. The most common argument selected was the “attitude and manners” of the adolescent. Most often, the argument chosen was a topic that the mother and dyad argued about daily (31.1% of the dyads). The majority of the sample (60.9%, n= 55) reported that the level of intensity/severity of the arguments surrounding the topic chosen was somewhat, very, or extremely bad. Therefore, the topics selected for the conflict task were both frequent and intense arguments.

4.2 RESULTS FOR HYPOTHESIS 1

Analyses for hypothesis 1 investigated whether invalidating maternal responses at Time 2 predicted BPD symptoms at Time 3 using linear regression. The dismissive factor was not predictive of the dependent variable and was removed from the analyses. Multicollinearity, tolerance, and VIF were examined. No variables had tolerance values less than .10 (range = .48 - 1) or VIF values above 10 (range= 1-2.08).

The Normal Probability Plot (P-P) of the Regression Standardised Residual and scatterplots were examined. The P-P plot shows a reasonably straight diagonal line, suggesting no major deviations from normality. There does not appear to be a clear or systematic pattern in the residuals from the scatterplot, again suggesting no major deviations from normality. An inspection of Mahalanobis distances revealed that no outliers were present in the data (all values were beneath 16.27). The maximum value for Cook's Distance was .63, again suggesting no major problems with outliers influencing the results of the model. In sum, preliminary analyses ensured no violation of the assumptions of normality, linearity, multicollinearity, and homoscedasticity.

Linear regressions were conducted with the following predictors of interest: maternal critical factor, dyadic negative escalation, and dyadic positive factor (Tables 11-13). BPD symptoms at Time 3 were the dependent variable. The regressions were run using both the transformed and untransformed dependent variable. The results were the same, therefore, for ease of interpretation the untransformed results are presented below. All three variables separately predicted BPD symptoms at Time 3. The maternal critical factor explained 14% of the

variance in BPD symptoms at Time 3, $F_{(1,58)}$ = 9.20, p< .01 (β = .37, p<.01). Dyadic negative escalation explained 12% of the variance in BPD symptoms at Time 3, $F_{(1,58)}$ = 7.92, p< .01 (β = .35, p<.01). The dyadic positive factor explained approximately 11% of the variance in BPD symptoms at Time 3, $F_{(1,58)}$ = 7.19, p< .01 (β = -.33, p<.01).

Variable		*B*	*SE*	β	R^2
	Constant	-2.44	2.05		0.14**
	Critical factor	0.79	0.26	0.37**	

***p<.05; **p<.01**

Table 11. Individual Regression Table: Maternal Critical Factor Predicting BPD symptoms

Variable		*B*	*SE*	β	R^2
	Constant	-0.12	1.41		0.12**
	Negative Escalation	1.61	0.57	0.35**	

***p<.05; **p<.01**

Table 12. Individual Regression Table: Negative Escalation Predicting BPD symptoms

Variable		*B*	*SE*	β	R^2
	Constant	7.50	1.53		0.11**
	Dyad: Positive	-0.39	0.14	-0.33**	

***p<.05; **p<.01**

Table 13. Individual Regression Table: Dyadic Positive Behaviors Predicting BPD symptoms

4.3 HYPOTHESIS 2

Analyses for Hypothesis 2 investigated whether invalidating maternal responses at Time 2 predicted BPD symptoms at Time 3, after controlling for BPD symptoms at Time 1 using hierarchical linear regression (Tables 14-16). Separate regressions were conducted for each predictor variables: maternal critical factor, dyadic negative escalation, and dyadic positive factor while controlling for BPD symptoms at Time 1 and receipt of public assistance. The covariate, receipt of public assistance did not alter the regression findings, therefore it was removed from the analyses.

The maternal critical factor significantly predicted BPD symptoms at Time 3, while controlling for BPD symptoms at Time 1. While controlling for BPD symptoms at Time 1, the dyadic positive factor predicted BPD symptoms at Time 3 at trend level. The critical maternal factor uniquely explained 4% of the variance in BPD symptoms at Time 3, $F_{(2,57)} = 27.72$, $p<$ .000 ($\beta = .21$, $p<.05$) over and above Time 1 BPD symptoms. The dyadic positive factor uniquely explained approximately 3% of the variance in BPD symptoms at Time 3, $F_{(2,57)} = 26.64$, $p<$ .000 ($\beta = -.18$, $p=.07$). Dyadic negative escalation uniquely explained 2% of the variance in BPD symptoms at Time 3, $F_{(2,57)} = 25.93$, $p<$ .000 ($\beta = .16$, $p=.1$).

Model	Variable	*B*	*SE*	β	ΔR^2
Step 1	Constant	0.96	0.54		.45***
	Time 1 BPD symptoms	0.67	0.10	.67***	
Step 2	Constant	-2.26	1.59		.04*
	Time 1 BPD symptoms	0.62	0.10	.62**	
	Critical factor	0.45	0.21	.21*	

***p*<.05; ***p*<.01; ***p<.000**

Table 14. Maternal Critical Factor Predict BPD symptoms at Time 3, Controlling for BPD Symptoms at Time

Model	Variable	*B*	*SE*	β	ΔR^2
Step 1	Constant	0.96	0.54		.45***
	Time 1 BPD symptoms	0.67	0.10	.67***	
Step 2	Constant	3.26	1.35		.03$^{\pm}$
	Time 1 BPD symptoms	0.63	0.10	.63**	
	Dyadic Positive	-0.21	0.11	-.18$^{\pm}$	

±*p*=.07; **p*<.05; ***p*<.01; ***p<.000

Table 15. Dyadic Positive Behaviors Predict BPD symptoms at Time 3, Controlling for BPD Symptoms at Time 1

Model	Variable	*B*	*SE*	β	ΔR^2
Step 1	Constant	0.96	0.54		.45***
	Time 1 BPD symptoms	0.67	0.10	.67***	
Step 2	Constant	-0.61	1.10		.02
	Time 1 BPD symptoms	0.63	0.10	.62**	
	Negative Escalation	0.76	0.46	.16	

***p*<.05; ****p*<.01; *****p*<.000

Table 16. Negative Escalation Predict BPD symptoms at Time 3, Controlling for BPD Symptoms at Time 1

4.4 HIERARCHICAL REGRESSIONS

Hierarchical regressions were conducted to determine the combined effects of the predictor variables (Tables 17-18). More specifically, these analyses allowed for the comparison of maternal versus dyadic codes in the prediction of BPD symptoms at Time 3. The maternal critical factor was entered at Step 1, explaining 13.7% of the variance in BPD symptoms at Time 3, $F_{(1,58)} = 9.20$, $p < .01$ ($\beta = .37$, $p<.01$). After entry of the negative escalation and the dyadic positive factor at Step 2, the total variance explained by the model as a whole was 20.8%, $F_{(3,56)} = 4.90$, $p < .01$. Negative escalation and dyadic positive behavior explained an additional 7% of the variance in BPD symptoms, after controlling for critical maternal responses, $R^2\Delta = .07$, $F\Delta_{(2, 56)} = 2.51$, $p = .09$. In the final model, there were no significant predictors of BPD symptoms at

Time 3. At trend level, the dyadic positive factor continued to predict BPD symptoms (β = -.25, *p*= .06).

Variable		*B*	*SE*	β	ΔR^2
Step 1	Constant	-2.44	2.05		0.14**
	Critical factor	0.79	0.26	0.37**	
Step 2	Constant	2.01	3.02		0.07
	Critical factor	0.29	0.37	0.13	
	Negative escalation	1.01	.0.75	0.22	
	Positive Factor	-0.29	0.15	-0.25±	

±*p*=.06; **p*<.05; ***p*<.01

Table 17. Hierarchical Regression Table: Predicting BPD symptoms from Critical Maternal Responses, Negative Escalation, and Dyadic Positive Factor

Next, hierarchical regressions were conduced while controlling for BPD symptoms at Time 1 (Table 18). More specifically, these analyses allowed for the comparison of maternal versus dyadic codes in the prediction of BPD symptoms, while controlling for earlier levels of BPD symptoms. BPD symptoms at Time 1 were entered in the first step of the regression equation. This explained 45% of the variance in BPD symptoms at Time 3, $F_{(1,58)}$ = 47.84, *p*< .000 (β = .67, *p*<.000). The maternal critical factor was entered at Step 2 and the combined effect of both Time 1 BPD symptoms and critical maternal responses explained 49% of the variance in BPD symptoms at Time 3, $F_{(2,57)}$ = 27.72, *p*< .000. In this model, both BPD symptoms at Time 1

and the maternal critical factor were statistically significant, with BPD symptoms having a higher beta value (β = .62, p<.000) than critical maternal responses (β = .21, p<.05).

After entry of the negative escalation and the dyadic positive factor codes at Step 3, the total variance explained by the model as a whole was 51%, $F_{(4,55)}$ = 14.30, p<.000. Negative escalation and the dyadic positive factor only explained an additional 1.7% of the variance in BPD symptoms, after controlling for BPD symptoms at Time 1 and the maternal critical factor, $R^2\Delta$ = .02, $F\Delta_{(2, 55)}$ = 0.40, *ns*. In the final model, only BPD symptoms at Time 1 were statistically significant (β = .59, p<.000).

Model	Variable	*B*	*SE*	β	ΔR^2
Step 1	Constant	0.96	.0.54		0.45***
	Time 1 BPD symptoms	0.67	0.10	0.67***	
Step 2	Constant	-2.26	1.59		0.04*
	Time 1 BPD symptoms	0.62	0.10	0.62**	
	Critical factor	0.45	0.21	0.21*	
Step 3	Constant	0.16	2.42		0.02
	Time 1 BPD symptoms	0.59	0.10	0.59**	
	Critical factor	0.26	0.29	0.12	
	Negative escalation	0.34	0.61	0.07	
	Dyadic: Positive Factor	-0.16	0.12	-0.14	

***p*<.05; ****p*<.01; *****p*<.000**

Table 18. Hierarchical Regression Table: Predicting BPD symptoms from Time 1 BPD symptoms, Critical Maternal Responses, Negative Escalation, and Relationship Quality/Mutuality

5.0 DISCUSSION

5.1 SUMMARY

This study assessed maternal and dyadic facets of invalidation, including critical and dismissive parenting behaviors, dyadic negative escalation, and low positive dyadic qualities on the development of BPD and BPD symptoms in adolescent girls. The results of this study indicate that a critical maternal response factor, including as negative affect, dominance, and conflict, predicted BPD symptoms across one year, even after controlling for BPD symptoms at baseline. At trend level, aspects of the overall mother-daughter relationship, namely a positive factor including low satisfaction, mutuality, and relationship quality also predicted BPD symptoms across one year, even after controlling for baseline BPD symptoms. However, when hierarchical regressions were conducted, including all of these variables in a single model, none of independent predictors contributed unique variance to BPD symptoms. These findings are discussed in more detail below.

5.2 RELATION BETWEEN MATERNAL INVALIDATION AND BPD

Maternal invalidation, specifically critical responses, predicted BPD symptoms in adolescent girls across one year. These results provide support for Linehan's original theory (1993) that invalidating aspects of the home environment are related to the development of BPD.

Critical responses in this study included high levels of negative affect, dominance, and conflict. Although the conflict discussion task was designed to elicit these types of responses, maternal critical behaviors still predicted BPD symptoms. There may be several reasons for this distinction. First, mothers of girls with more BPD symptoms may be reacting or responding to the negative behaviors of their adolescent. High levels of negative affect, dominance, and conflict are common among individuals with BPD and are often reported in their interpersonal relationships. Girls with more BPD symptoms may be more likely to elicit this type of behavior from their mothers. However, these results held even when controlling for prior levels of BPD symptoms, suggesting that higher levels of maternal criticism may promote the maintenance of BPD symptoms over time.

These findings fit with self-report studies showing that individuals with BPD report that their relationships with parents were highly conflictual (Cheavens et al., 2005; Gunderson & Lyoo, 1997). For example, Cheavens and colleagues (2005) found that in a nonclinical sample of college students, perceived parental criticism was a predictor of BPD symptoms. The relationship between criticism and BPD was strengthened when the young adults reported using thought suppression as an emotion regulation strategy. The findings from this study extend previous work by showing that observed critical behaviors, rather than perceived parental criticism is also associated with BPD symptoms across time. Furthermore, this study has replicated these findings in a younger sample of adolescents, suggesting that processes of invalidation may be present and ongoing earlier in the development of BPD.

Interpersonal sensitivity has been theorized to be a critical component for discriminating BPD from other disorders (Stanley & Siever, 2009). In fact, many of the prominent symptoms of BPD such as aggression, suicidal behavior, and emotional reactivity often occur in interpersonal

contexts. Perhaps some of the interpersonal sensitivity seen in individuals with BPD is shaped from early and continued interactions with highly critical caregivers. Receiving maternal criticism in response to one's emotions and behaviors may create difficulties in the adolescent's interpersonal relationships. The highly criticized adolescent may misperceive others' intentions and affect, attribute malevolent intentions to social partners when none exist, and have a decreased ability to understand the mental state of others. All of these social deficits have been found in adults with BPD (Fonagy, Luyten, & Strathearn, 2011; Roepke, Vater, Preissler, Heekeren, & Dziobek, 2013; Stepp, Smith, Morse, Hallquist, & Pilkonis, 2011), especially among those who experienced invalidation, such as sexual abuse. The results from this study indicate that maternal criticism may be another pathway to the development of the interpersonal sensitivity commonly seen among BPD adults.

The findings from this study held even while controlling for initial BPD symptoms. Thus, critical maternal responses contribute unique variance to the development of BPD. Experiencing high levels of negative affect, dominance, and conflict from a mother may signal to the daughter that she is undesirable and/or incapable as a social partner, and perhaps even as an individual. This may be particularly detrimental for adolescents at-risk for developing BPD given the suggestion that forming a cohesive sense of identity may be linked to feelings of positive interpersonal contact and connectedness (Stanley & Siever, 2009). Without this positive sense of connection and acceptance from their mothers, adolescents at-risk for developing BPD may continue to view themselves inherently unacceptable.

The hypothesis that dismissive maternal responses would also be predictive of BPD was not supported. There may be several reasons for this. First, the dismissive maternal behaviors observed and coded during the task might not be representative of the kinds of dismissive

parenting behaviors that may lead to BPD. In this study, dismissive parenting was assessed through denial, task avoidance, and withdrawal. While these maternal behaviors may not be particularly validating during an argument, they may not be interpreted as invalidating by girls either. Although the behaviors in this construct were labeled as dismissive, they could more accurately represent a lack of task engagement rather than invalidation. Dismissive parenting and the hypothesized relations to BPD might be more emotional than what was captured by these codes. Emotional withdrawal and emotion-based denial might be more associated with BPD and not fully captured in this coding system, which focused more on the cognitive and physical aspects of these codes. For example, the mother may have engaged in task avoidance or withdrawal during the task because she was embarrassed to be on camera or unsure of how to complete the task. Mothers may have been denying their role in the conflict, rather than denying the girl's feelings.

Alternatively, dismissive maternal responses may actually be an effective strategy that can be used to help emotionally vulnerable adolescent's regulate intense negative emotions. Perhaps, mothers who used more dismissive strategies assisted their adolescent in down-regulating intense negative emotions, and lessened the likelihood that BPD symptoms would increase over time. Dismissive strategies might also be used in combination with highly critical responses, without the mother realizing that she may be intermittently reinforcing the adolescent's extreme emotional displays. For example, if the first response to an adolescent's negative affect is dismissive, the adolescent may feel that she needs to increase her emotional expression in order to be understood and fully convey her suffering. By increasing emotional intensity, the adolescent might elicit a second, more critical response from the mother. Through these types of interactions, the adolescent learns that only intense and dramatic expressions of

negative affect get a response from the mother. Future research should continue to explore the role of dismissive parenting strategies in order to determine whether there are conditions under which these strategies might be beneficial for the adolescent or serve to intermittently reinforce intense displays of negative affect.

5.3 RELATION BETWEEN DYADIC CODES AND BPD

Aspects of the overall mother-daughter relationship, namely lower levels of positive features including relationship quality, satisfaction, and mutuality may also to be related to BPD symptoms across one year, even when controlling for earlier BPD symptoms. Although only at trend level, these findings suggest that the dyadic and transactional nature of invalidation may be important to take into account as another unique predictor of BPD symptoms over time. In particular, they lend support to the transactional relationship between emotional vulnerability and invalidation, a key aspect of Linehan's theory (1993). If lower positive aspects of the mother-daughter relationship predict BPD symptoms over time that suggests that both the mother and daughter play a role in the development of adolescent BPD.

This is the first study to assess observed aspects of relationship satisfaction, quality, and mutuality in adolescents at-risk for BPD and their mothers. The relationship can be viewed as a transactional system, were there are ongoing daily interactions between the adolescent and her mother. These interactions provide feedback to both the mother and daughter about the nature of the relationship. It seems likely that adolescents with more BPD features are challenging social partners. As a mother, it may be difficult to form a high quality partnership, experience mutuality and feel satisfied with the relationship. The findings from this study also support Thomas and

Chess' (1977) goodness of fit model, in that the adolescent's challenging characteristics and behaviors may be a poor match for a mother with equally challenging characteristics and behaviors, or a mother who is uncomfortable with strong negative emotions. Mothers of these adolescents may require additional support to adaptively respond to their adolescent's behaviors and emotions.

Dyadic negative escalation also predicted BPD symptoms across 6 months, but this finding was no longer significant when controlling for prior levels of BPD symptoms. These findings indicate that it is important to emphasize the bidirectional nature of the relationship between invalidation and the development of BPD. Individuals with BPD have been theorized to be highly sensitive appraisers of emotional cues in their environments and social partners (Domes, Schulze, & Herpertz, 2009; Fonagy et al., 2011; Linehan, 1993). Research evidence has also supported these theories (Fertuck et al., 2009; Fertuck, Grinband, & Stanley, 2013; Lynch et al., 2006; Wagner & Linehan, 1999). Therefore, adolescent girls with BPD may become aware of their mother's negative emotions more quickly and anticipate conflict or rejection. The girls may react in an extreme way to avoid or override the expected conflict or rejection by their social partner. When adolescents are reacting in more extreme ways, it is likely that parents will need to respond in more extreme ways, creating a negative escalation cycle. These data reveal that it is not simply high negative, critical affect that characterizes adolescent girls at-risk for BPD and their mothers. Instead, there seems to be an inability to form a mutually satisfying relationship and failures by both partners to regulate negative affect in a way that is interpersonally effective and responsive to the task demands and opportunities.

5.4 HIERARCHICAL REGRESSION FINDINGS

The hierarchical regression analyses did not confirm that the maternal critical factor, over and above dyadic behaviors, was uniquely associated with BPD. This may due to the small sample size (n=60), as this study was underpowered to detect small-to-medium effects. In addition, two of the predictors: maternal critical behaviors and dyadic negative escalation were correlated. When combined with the small sample size, this may have limited the ability to examine the independent effects of those variables on BPD symptoms. While the overall models were significant, the independent predictors were not, suggesting that as a group maternal criticism, dyadic negative escalation, and low positive qualities of the dyad do explain variance in BPD symptoms over time. However, one set of predictors is not more important than the other.

This lack of specificity may have implications for future observational research on BPD. Given that maternal and dyadic variables were strongly correlated, it is unclear what predictive value one provides over and above the other. Perhaps, the dyadic processes that occur during interactions are the most clinically relevant for the development of BPD, a disorder that is characterized by interpersonal sensitivity and instability. Assessing dyadic processes takes into account the behavior of both the adolescent and the mother in response to one another. Linehan's original definition of invalidation also focuses on the response of the parent to the child's emotions and continued transactions between the two. Thus, it is important to not just focus on the maternal critical behaviors, but to investigate *when* the mothers are engaging in critical behaviors: what has the adolescent said or done before the maternal behaviors occurred; what is the adolescent's affect? Does the mother's criticism precede the adolescent's negative affect? More specific contextual factors may be critical to address in future studies on the development

of BPD. Furthermore, the use of second-by-second coding schemes may be necessary to tease apart these complex relations (Crowell et al., *in press*).

5.5 STRENGTHS

A strength of this study was the operationalization and measurement of invalidation. Invalidation was observationally measured through specific critical and dismissive aspects of maternal behavior as well as dyadic relationship quality and negative escalation. It is hoped that future work can build on this foundation and continue to investigate specific maternal and dyadic behaviors that may lead to the development of BPD. Furthermore, this is the first study that has observed invalidation during a structured conflict discussion between mothers and daughters varying on BPD symptoms. Prior research has relied primarily on retrospective reports of parental invalidation completed by adult patients with BPD. This study measured BPD across several time points spanning one year and was able to prospectively predict BPD symptoms.

Another strength of this study was the ability to examine dyadic aspects of the interaction and how these may contribute to the development of BPD symptoms. Although there was limited support for the role of negative escalation in the prediction of BPD symptoms over and above prior BPD symptoms, this is the first study to assess this inter-individual process of invalidation. Positive and negative aspects of the dyadic relationship are likely impacted by both partners. Perhaps during adolescence, the nature and quality of the parent-adolescent relationship is more strongly influenced by the parent than the adolescent. Future research should continue to

investigate this possibility and incorporate additional measures of maternal psychopathology, temperament, and attachment.

This study recruited a community sample of adolescents and therefore, these results are likely to be more generalizable than the results of a purely clinical sample. The adolescents who participated in this project had low to moderate features of BPD and it is likely that the invalidation findings may have been more severe in a sample with more clinically significant and impairing symptoms. In addition, this study was strengthened by the recruitment of a sample diverse in terms of racial and socio-economic background.

5.6 LIMITATIONS

Despite the merits of this study, there are several limitations worthy of consideration. This study has strength in its short-term longitudinal design. However, it is likely that BPD symptoms and parenting behaviors have been transacting for many years prior to enrollment in this project. BPD symptoms may have been present and developing in these girls before enrollment and these earlier symptoms may have altered parenting earlier in childhood. A longer follow-up period and/or additional assessments at an earlier age may help to create more clear pathways to the development of BPD. Furthermore, few girls met full diagnostic criteria for BPD, thus this study may have assessed symptoms of psychopathology that are not specific or unique to BPD.

This study was unable to assess the uniqueness of each predictor variable to the development of BPD, as opposed to other psychopathology. It is likely that maternal criticism, low quality mother-adolescent relationships, and negative escalation contribute to the

development of other disorders as well. Perhaps, factors such as maternal criticism are not unique to the prediction of specific diagnoses, but may be uniquely related to the development of personality or temperament traits, such as neuroticism that may underlie and predict several disorders. Future work using samples of adolescents with varying Axis I and II disorders might help elucidate the specificity of these predictors to the development of psychopathology or underlying traits that may predispose individuals toward specific types of psychopathology.

Another limitation is the assessment of BPD symptoms in the adolescents only. Given the work indicating strong genetic linkages in BPD and BPD symptoms (Lis et al., 2007), it is likely that many of the mothers also had symptoms and/or diagnoses of BPD. It would be anticipated that the prediction of adolescent BPD symptoms might be stronger when any co-occuring maternal psychopathology is considered. Alternatively, maternal BPD may interact with aspects of parenting and dyadic relationship components, serving as an exacerbating factor in the development of BPD.

Another limitation of this study was the lack of fathers and adolescent boys in the sample. It is possible that invalidating parenting behaviors could be affected by gender of the parent and/or adolescent. Fathers may respond to the emotions of his adolescent daughter very differently, and could influence the development of BPD in an alternative way. Adolescent girls may also experience invalidation differently from mothers as opposed to fathers. Furthermore, the predictive strength of the dyadic codes between father and daughter as opposed to mother and daughter may shift. However, it is especially important to study the development of BPD in adolescent girls, given that they appear to be at much greater risk for the future development of BPD than adolescent boys.

The coding system used to analyze the interaction data was designed to provide an overall snapshot of each mother and dyad's affect and behavior. Because of this, some of the subtler, moment-to-moment shifts in maternal affective responding may not have been captured. Perhaps, a more fine-grained, sentence-level coding system might capture invalidation in a more comprehensive way. Finally, the relatively small sample size limited the power to detect smaller effects. Hopefully, future work can continue to observationally assess mother-daughter interactions in larger samples and perhaps combine these interactions with other methods such as psychophysiological responses and ecological momentary assessment.

5.7 FUTURE DIRECTIONS

Future research should continue to build upon the findings of this work and refine the definition and measurement of invalidation. Although this study represents an important step in the measurement of invalidation, it will be crucial for future work to continue to assess invalidation longitudinally and in different situations (e.g. a positive context, with other family members and friends). In addition, it will be interesting to investigate positive maternal and adolescent behaviors at the level of the individual and dyad and whether low levels of positive behaviors are equally predictive in the development of BPD. Perhaps specific combinations of low positive maternal behaviors and high negative maternal behaviors lead to BPD over time.

It would be interesting to observe and code the mothers and daughters during a positive task, rather than a conflict discussion that pulls for negative affect. There may be differences in the validation of positive emotions as well as negative ones. A lack of support for positive emotions in social situations may be especially detrimental to those adolescents who are at-risk

for the development of BPD because of the theorized interpersonal sensitivity in this group. In addition, future work could employ a positive mood induction or positive discussion task, prior to the conflict discussion. It may be interesting to assess the balance of invalidating parenting behaviors and transition between contexts under these conditions.

It would also be exciting to link observational data to psychophysiological data in a sample of adolescents at high-risk for BPD (Crowell et al., 2008). Over time, maternal invalidation may change the psychophysiology of offspring, who become less reactive or blunted to emotional information. Adolescents may have learned that their reactions are not accepted or treated with respect and are instead ignored or met with criticism. This type of work would extend the correlates and consequences of invalidation into physically observable signs.

The heterogeneity of BPD is well documented and patients often present with varying clinical profiles, symptoms, and Axis I and II comorbidity (Skodol, Gunderson, et al., 2002). This heterogeneity might also imply different developmental pathways to the disorder (Nigg, Silk, Stavro, & Miller, 2005) and as a result, differential effects of parental invalidation. Perhaps, specific sub-groups of adolescents with BPD symptoms may be more sensitive to the effects of maternal invalidation and poor relationships. For example, adolescents endorsing the interpersonal sensitivity criterion of BPD might be more negatively impacted by maternal criticism and poor relationship quality over time. Using large-scale longitudinal methods, future work should aim to address these conceptual questions.

Clinically, these findings underscore the importance of assessing and beginning to treat BPD symptoms earlier in development. In this community sample of adolescents, BPD symptoms were stable across one year, and for a subset of girls, indicative of clinically significant functional impairment. The findings may also be helpful in the development of a

therapy targeting invalidation in mothers of adolescents with BPD symptoms. This therapy could enhance parenting practices by decreasing the use of dismissive or critical strategies, or focus more on improving aspects of the relationship between mother and daughters. These types of clinical interventions provide unique opportunities to reduce parental invalidation through both psychoeducation and change strategies since we are able to modify parenting behaviors more easily than underlying neurobiology and genetics. Participating in a parenting intervention earlier in development may help decrease the likelihood that BPD will develop in adolescents, as their mothers may become more validating. A related consequence of increased maternal validation may be increased dyadic relationship quality, mutuality, and satisfaction.

Adolescents with BPD symptoms were more likely to experience invalidation, in the form of criticism, from their mothers during a conflict discussion. Furthermore, they were more likely to maintain their BPD symptoms over time, when their mothers displayed more invalidating behaviors during the interaction. Lower positive aspects of the dyadic relationship also predicted BPD symptoms across time. These findings suggest that parenting, and more specifically critical aspects of parenting may contribute to the development of BPD in adolescents. In addition, aspects of the parent-adolescent relationship contribute to the development of BPD, including lower mutuality, satisfaction, and relationship quality. Future work should continue to use ecologically valid paradigms to determine the nature and the extent of parental invalidation in adolescents at-risk for developing BPD.

6.0 BIBLIOGRAPHY

Adrian, M., Zeman, J., Erdley, C., Lisa, L., Homan, K., & Sim, L. (2009). Social contextual links to emotion regulation in an adolescent psychiatric inpatient population: do gender and symptomatology matter? *Journal of Child Psychology and Psychiatry, and Allied Disciplines*, *50*(11), 1428–1436. doi:10.1111/j.1469-7610.2009.02162.x

Adrian, M., Zeman, J., Erdley, C., Lisa, L., & Sim, L. (2011). Emotional dysregulation and interpersonal difficulties as risk factors for nonsuicidal self-injury in adolescent girls. *Journal of Abnormal Child Psychology*, *39*(3), 389–400. doi:10.1007/s10802-010-9465-3

Afifi, T. O., Mather, A., Boman, J., Fleisher, W., Enns, M. W., Macmillan, H., & Sareen, J. (2011). Childhood adversity and personality disorders: Results from a nationally representative population-based study. *Journal of Psychiatric Research*, *45*(6), 814–822. doi:10.1016/j.jpsychires.2010.11.008

Aggen, S. H., Neale, M. C., Røysamb, E., Reichborn-Kjennerud, T., & Kendler, K. S. (2009). A psychometric evaluation of the DSM-IV borderline personality disorder criteria: age and sex moderation of criterion functioning. *Psychological Medicine*, *39*(12), 1967–1978. doi:10.1017/S0033291709005807

American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders: DSM-IV-TR*. American Psychiatric Pub.

Angold, A., Costello, E. J., & Worthman, C. M. (1998). Puberty and Depression: The Roles of Age, Pubertal Status and Pubertal Timing. *Psychological Medicine*, *28*(01), 51–61. doi:null

Bagge, C. L., Nickell, A., Stepp, S. D., Durrett, C., Jackson, K., & Trull, T. J. (2004). Borderline personality disorder features predict negative outcomes 2 years later. *Journal of Abnormal Psychology*, *113*(2), 279–288. doi:10.1037/0021-843X.113.2.279

Bagge, C. L., Stepp, S. D., & Trull, T. J. (2005). Borderline personality disorder features and utilization of treatment over two years. *Journal of Personality Disorders*, *19*(4), 420–439. doi:10.1521/pedi.2005.19.4.420

Bandelow, B., Krause, J., Wedekind, D., Broocks, A., Hajak, G., & Rüther, E. (2005). Early traumatic life events, parental attitudes, family history, and birth risk factors in patients with borderline personality disorder and healthy controls. *Psychiatry Research*, *134*(2), 169–179. doi:10.1016/j.psychres.2003.07.008

Battle, C. L., Shea, M. T., Johnson, D. M., Yen, S., Zlotnick, C., Zanarini, M. C., Sanislow, C. A., et al. (2004). Childhood Maltreatment Associated With Adult Personality Disorders: Findings From the Collaborative Longitudinal Personality Disorders Study. *Journal of Personality Disorders*, *18*(2), 193–211. doi:10.1521/pedi.18.2.193.32777

Becker, D. F., Grilo, C. M., Edell, W. S., & McGlashan, T. H. (2002). Diagnostic Efficiency of Borderline Personality Disorder Criteria in Hospitalized Adolescents: Comparison With Hospitalized Adults. *American Journal of Psychiatry*, *159*(12), 2042–2047.

Bender, D. S., Dolan, R. T., Skodol, A. E., Sanislow, C. A., Dyck, I. R., McGlashan, T. H., Shea, M. T., et al. (2001). Treatment utilization by patients with personality disorders. *The American Journal of Psychiatry*, *158*(2), 295–302.

Bezirganian, S., Cohen, P., & Brook, J. S. (1993). The impact of mother-child interaction on the development of borderline personality disorder. *The American Journal of Psychiatry*, *150*(12), 1836–1842.

Bjorklund, P. (2006). No man's land: gender bias and social constructivism in the diagnosis of borderline personality disorder. *Issues in Mental Health Nursing*, *27*(1), 3–23. doi:10.1080/01612840500312753

Blashfield, R. K., & Intoccia, V. (2000). Growth of the Literature on the Topic of Personality Disorders. *American Journal of Psychiatry*, *157*(3), 472–473. doi:10.1176/appi.ajp.157.3.472

Bradley, R., Zittel Conklin, C., & Westen, D. (2005). The borderline personality diagnosis in adolescents: gender differences and subtypes. *Journal of Child Psychology and Psychiatry, and Allied Disciplines*, *46*(9), 1006–1019. doi:10.1111/j.1469-7610.2004.00401.x

Carlson, E. A., Egeland, B., & Sroufe, L. A. (2009). A prospective investigation of the development of borderline personality symptoms. *Development and Psychopathology*, *21*(4), 1311–1334. doi:10.1017/S0954579409990174

Chanen, A. M., Jackson, H. J., McGorry, P. D., Allot, K. A., Clarkson, V., & Yuen, H. P. (2004). Two-Year Stability of Personality Disorder in Older Adolescent Outpatients. *Journal of Personality Disorders*, *18*(6), 526–541. doi:10.1521/pedi.18.6.526.54798

Chanen, A. M., Jovev, M., McCutcheon, L. K., Jackson, H. J., & McGorry, P. D. (2008). Borderline Personality Disorder in Young People and the Prospects for Prevention and Early Intervention. *Current Psychiatry Reviews*, *4*(1), 48–57. doi:10.2174/157340008783743820

Chanen, A. M., & Kaess, M. (2011). Developmental Pathways to Borderline Personality Disorder. *Current Psychiatry Reports*. doi:10.1007/s11920-011-0242-y

Cheavens, J. S., Zachary Rosenthal, M., Daughters, S. B., Nowak, J., Kosson, D., Lynch, T. R., & Lejuez, C. W. (2005). An analogue investigation of the relationships among perceived parental criticism, negative affect, and borderline personality disorder features: the role of thought suppression. *Behaviour Research and Therapy*, *43*(2), 257–268. doi:10.1016/j.brat.2004.01.006

Choi-Kain, L. W., Zanarini, M. C., Frankenburg, F. R., Fitzmaurice, G. M., & Reich, D. B. (2010). A longitudinal study of the 10-year course of interpersonal features in borderline personality disorder. *Journal of Personality Disorders*, *24*(3), 365–376. doi:10.1521/pedi.2010.24.3.365

Clark, L. A. (2007). Assessment and Diagnosis of Personality Disorder: Perennial Issues and an Emerging Reconceptualization. *Annual Review of Psychology*, *58*(1), 227–257. doi:10.1146/annurev.psych.57.102904.190200

Clark, L. A. (2009). Stability and Change in Personality Disorder. *Current Directions in Psychological Science*, *18*(1), 27–31. doi:10.1111/j.1467-8721.2009.01600.x

Cohen, P., Chen, H., Kasen, S., Johnson, J. G., Crawford, T., & Gordon, K. (2005). Adolescent Cluster A personality disorder symptoms, role assumption in the transition to adulthood, and resolution or persistence of symptoms. *Development and Psychopathology*, *17*(2), 549–568.

Cohen, P., Crawford, T. N., Johnson, J. G., & Kasen, S. (2005). The children in the community study of developmental course of personality disorder. *Journal of Personality Disorders*, *19*(5), 466–486. doi:10.1521/pedi.2005.19.5.466

Cole, P. M., Luby, J., & Sullivan, M. W. (2008). Emotions and the Development of Childhood Depression: Bridging the Gap. *Child development perspectives*, *2*(3), 141–148. doi:10.1111/j.1750-8606.2008.00056.x

Conley, C. S., & Rudolph, K. D. (2009). The Emerging Sex Difference in Adolescent Depression: Interacting Contributions of Puberty and Peer Stress. *Development and Psychopathology*, *21*(02), 593–620. doi:10.1017/S0954579409000327

Crick, N. R., Murray-Close, D., & Woods, K. (2005). Borderline personality features in childhood: a short-term longitudinal study. *Development and Psychopathology*, *17*(4), 1051–1070.

Crone, E. A., & Dahl, R. E. (2012). Understanding adolescence as a period of social–affective engagement and goal flexibility. *Nature Reviews Neuroscience*, *13*(9), 636–650. doi:10.1038/nrn3313

Crowell, S. E., Baucom, B. R., McCauley, E., Potapova, N. V., Fitelson, M., Barth, H., & Smith, C. J. (*in press*). Mechanisms of contextual risk for adolescent self-injury: Invalidation and conflict escalation in mother-child interactions. *Journal of Clinical Child and Adolescent Psychology.*

Crowell, S. E., Beauchaine, T. P., & Linehan, M. (2009). A biosocial developmental model of borderline personality: Elaborating and extending Linehan's theory. *Psychological Bulletin*, *135*(3), 495–510. doi:10.1037/a0015616

Crowell, S. E., Beauchaine, T. P., McCauley, E., Smith, C. J., Vasilev, C. A., & Stevens, A. L. (2008). Parent-child interactions, peripheral serotonin, and self-inflicted injury in adolescents. *Journal of Consulting and Clinical Psychology*, *76*(1), 15–21. doi:10.1037/0022-006X.76.1.15

Dahl, R. E. (2004). Adolescent brain development: a period of vulnerabilities and opportunities. Keynote address. *Annals of the New York Academy of Sciences*, *1021*, 1–22. doi:10.1196/annals.1308.001

Distel, M. A., Trull, T. J., Derom, C. A., Thiery, E. W., Grimmer, M. A., Martin, N. G., Willemsen, G., et al. (2008). Heritability of borderline personality disorder features is similar across three countries. *Psychological Medicine*, *38*(9), 1219–1229. doi:10.1017/S0033291707002024

Domes, G., Schulze, L., & Herpertz, S. C. (2009). Emotion recognition in borderline personality disorder-a review of the literature. *Journal of Personality Disorders*, *23*(1), 6–19. doi:10.1521/pedi.2009.23.1.6

Eaton, W. W., Martins, S. S., Nestadt, G., Bienvenu, O. J., Clarke, D., & Alexandre, P. (2008). The Burden of Mental Disorders. *Epidemiologic reviews*, *30*, 1–14. doi:10.1093/epirev/mxn011

Eisenberg, N, Fabes, R. A., & Murphy, B. C. (1996). Parents' reactions to children's negative emotions: relations to children's social competence and comforting behavior. *Child Development*, *67*(5), 2227–2247.

Eisenberg, N., Cumberland, A., & Spinrad, T. L. (1998). Parental Socialization of Emotion. *Psychological Inquiry*, *9*(4), 241–273. doi:10.1207/s15327965pli0904_1

Eisenberg, N., Spinrad, T. L., & Eggum, N. D. (2010). Emotion-related self-regulation and its relation to children's maladjustment. *Annual Review of Clinical Psychology*, *6*, 495–525. doi:10.1146/annurev.clinpsy.121208.131208

Fabes, R.A., Poulin, R.E., Eisenberg, N., & Madden-Derdich, D.A. (2002). The Coping with Children's Negative Emotions Scale (CCNES): Psychometric properties and relations with children's emotional competence. *Marriage & Family Review, 34*, 285–310.

Faul, F., Erdfelder, E., Lang, -G., & Buchner, A. (2007). G*Power 3: A flexible statistical power analysis program for the social, behavioral, and biomedical sciences. *Behavior Research Methods*, *39*, 175–191.

Fertuck, E. A, Jekal, A., Song, I., Wyman, B., Morris, M. C., Wilson, S. T., Brodsky, B. S., et al. (2009). Enhanced "Reading the Mind in the Eyes" in borderline personality disorder compared to healthy controls. *Psychological Medicine*, *39*(12), 1979–1988. doi:10.1017/S003329170900600X

Fertuck, E. A., Grinband, J., & Stanley, B. (2013). Facial trust appraisal negatively biased in borderline personality disorder. *Psychiatry Research*. doi:10.1016/j.psychres.2013.01.004

Fonagy, P., Luyten, P., & Strathearn, L. (2011). Borderline personality disorder, mentalization, and the neurobiology of attachment. *Infant Mental Health Journal*, *32*(1), 47–69. doi:10.1002/imhj.20283

Frost, R. O., Marten, P., Lahart, C., & Rosenblate, R. (1990). The dimensions of perfectionism. *Cognitive Therapy and Research*, *14*(5), 449–468. doi:10.1007/BF01172967

Fruzzetti, A. E., Shenk, C., & Hoffman, P. D. (2005). Family interaction and the development of borderline personality disorder: a transactional model. *Development and Psychopathology*, *17*(4), 1007–1030.

Furman, W., & Shomaker, L. B. (2008). Patterns of interaction in adolescent romantic relationships: Distinct features and links to other close relationships. *Journal of Adolescence*, *31*(6), 771–788. doi:10.1016/j.adolescence.2007.10.007

Ghiassi, V., Dimaggio, G., & Brune, M. (2010). Dysfunctions in understanding other minds in borderline personality disorder: a study using cartoon picture stories. *Psychotherapy Research: Journal of the Society for Psychotherapy Research*, *20*(6), 657–667. doi:10.1080/10503307.2010.501040

Gottman, J. M., Katz, L. F., & Hooven, C. (1996). Parental meta-emotion philosophy and the emotional life of families: Theoretical models and preliminary data. *Journal of Family Psychology*, *10*(3), 243–268. doi:http://dx.doi.org.pitt.idm.oclc.org/10.1037/0893-3200.10.3.243

Gottman, J. M., Katz, L. F., & Hooven, C. (1997). *Meta-Emotion: How Families Communicate Emotionally*. Psychology Press.

Grant, B. F., Chou, S. P., Goldstein, R. B., Huang, B., Stinson, F. S., Saha, T. D., Smith, S. M., et al. (2008). Prevalence, correlates, disability, and comorbidity of DSM-IV borderline personality disorder: results from the Wave 2 National Epidemiologic Survey on Alcohol and Related Conditions. *The Journal of Clinical Psychiatry*, *69*(4), 533–545.

Gratz, K. L., Tull, M. T., Reynolds, E. K., Bagge, C. L., Latzman, R. D., Daughters, S. B., & Lejuez, C. W. (2009). Extending extant models of the pathogenesis of borderline personality disorder to childhood borderline personality symptoms: the roles of affective dysfunction, disinhibition, and self- and emotion-regulation deficits. *Development and Psychopathology*, *21*(4), 1263–1291. doi:10.1017/S0954579409990150

Gunderson, J. G, & Lyoo, I. K. (1997). Family problems and relationships for adults with borderline personality disorder. *Harvard Review of Psychiatry*, *4*(5), 272–278.

Gunderson, J. G. (2007). Disturbed relationships as a phenotype for borderline personality disorder. *The American Journal of Psychiatry*, *164*(11), 1637–1640. doi:10.1176/appi.ajp.2007.07071125

Helgeland, M. I., & Torgersen, S. (2004). Developmental antecedents of borderline personality disorder. *Comprehensive Psychiatry*, *45*(2), 138–147. doi:10.1016/j.comppsych.2003.09.001

Hilt, L. M., Cha, C. B., & Nolen-Hoeksema, S. (2008). Nonsuicidal self-injury in young adolescent girls: moderators of the distress-function relationship. *Journal of Consulting and Clinical Psychology*, *76*(1), 63–71. doi:10.1037/0022-006X.76.1.63

Hipwell, A. E., Loeber, R., Stouthamer-Loeber, M., Keenan, K., White, H. R., & Kroneman, L. (2002). Characteristics of girls with early onset disruptive and antisocial behaviour. *Criminal Behaviour and Mental Health: CBMH*, *12*(1), 99–118.

Holm, A. L., & Severinsson, E. (2011). Struggling to recover by changing suicidal behaviour: Narratives from women with borderline personality disorder. *International Journal of Mental Health Nursing*, *20*(3), 165–173. doi:10.1111/j.1447-0349.2010.00713.x

Horesh, N., Orbach, I., Gothelf, D., Efrati, M., & Apter, A. (2003). Comparison of the suicidal behavior of adolescent inpatients with borderline personality disorder and major depression. *The Journal of Nervous and Mental Disease*, *191*(9), 582–588. doi:10.1097/01.nmd.0000087184.56009.61

Igarashi, H., Hasui, C., Uji, M., Shono, M., Nagata, T., & Kitamura, T. (2010). Effects of child abuse history on borderline personality traits, negative life events, and depression: a study among a university student population in Japan. *Psychiatry Research*, *180*(2-3), 120–125. doi:10.1016/j.psychres.2010.04.029

Johnson, J. G, Cohen, P., Brown, J., Smailes, E. M., & Bernstein, D. P. (1999). Childhood Maltreatment Increases Risk for Personality Disorders During Early Adulthood. *Arch Gen Psychiatry*, *56*(7), 600–606. doi:10.1001/archpsyc.56.7.600

Johnson, J. G, Cohen, P., Smailes, E. M., Skodol, A. E., Brown, J., & Oldham, J. M. (2001). Childhood verbal abuse and risk for personality disorders during adolescence and early adulthood. *Comprehensive Psychiatry*, *42*(1), 16–23. doi:10.1053/comp.2001.19755

Johnson, J. G, Smailes, E. M., Cohen, P., Brown, J., & Bernstein, D. P. (2000). Associations between four types of childhood neglect and personality disorder symptoms during adolescence and early adulthood: findings of a community-based longitudinal study. *Journal of Personality Disorders*, *14*(2), 171–187.

Johnson, J. G., Cohen, P., Gould, M. S., Kasen, S., Brown, J., & Brook, J. S. (2002). Childhood Adversities, Interpersonal Difficulties, and Risk for Suicide Attempts During Late Adolescence and Early Adulthood. *Arch Gen Psychiatry*, *59*(8), 741–749. doi:10.1001/archpsyc.59.8.741

Keenan, K., Hipwell, A., Chung, T., Stepp, S., Stouthamer-Loeber, M., Loeber, R., & McTigue, K. (2010). The Pittsburgh Girls Study: Overview and Initial Findings. *Journal of Clinical Child & Adolescent Psychology*, *39*(4), 506–521. doi:10.1080/15374416.2010.486320

Krause, E. D., Mendelson, T., & Lynch, T. R. (2003). Childhood emotional invalidation and adult psychological distress: the mediating role of emotional inhibition. *Child Abuse & Neglect*, *27*(2), 199–213. doi:16/S0145-2134(02)00536-7

Lieb, K., Zanarini, M. C., Schmahl, C., Linehan, M. M., & Bohus, M. (2004). Borderline personality disorder. *Lancet*, *364*(9432), 453–461. doi:10.1016/S0140-6736(04)16770-6

Linehan, M. M. (1993). *Cognitive Behavioral Treatment of Borderline Personality Disorder*. New York: Guilford Press.

Lis, E., Greenfield, B., Henry, M., Guilé, J. M., & Dougherty, G. (2007). Neuroimaging and genetics of borderline personality disorder: a review. *Journal of Psychiatry & Neuroscience: JPN*, *32*(3), 162–173.

Lynch, T. R., Rosenthal, M. Z., Kosson, D. S., Cheavens, J. S., Lejuez, C. W., & Blair, R. J. R. (2006). Heightened sensitivity to facial expressions of emotion in borderline personality disorder. *Emotion (Washington, D.C.)*, *6*(4), 647–655. doi:10.1037/1528-3542.6.4.647

Markon, K. E., Chmielewski, M., & Miller, C. J. (2011). The reliability and validity of discrete and continuous measures of psychopathology: A quantitative review. *Psychological Bulletin*, *137*(5), 856–879. doi:10.1037/a0023678

McClellan, J., Adams, J., Douglas, D., McCurry, C., & Storck, M. (1995). Clinical characteristics related to severity of sexual abuse: a study of seriously mentally ill youth. *Child Abuse & Neglect*, *19*(10), 1245–1254.

McMain, S., Korman, L. M., & Dimeff, L. (2001). Dialectical behavior therapy and the treatment of emotion dysregulation. *Journal of Clinical Psychology*, *57*(2), 183–196.

McMakin, D. L., Burkhouse, K. L., Olino, T. M., Siegle, G. J., Dahl, R. E., & Silk, J. S. (2011). Affective Functioning Among Early Adolescents at High and Low Familial Risk for Depression and Their Mothers: A Focus on Individual and Transactional Processes across Contexts. *Journal of Abnormal Child Psychology*, *39*(8), 1213–1225. doi:10.1007/s10802-011-9540-4

Miller, J. D., Morse, J. Q., Nolf, K., Stepp, S. D., & Pilkonis, P. A. (2012). Can DSM-IV borderline personality disorder be diagnosed via dimensional personality traits?

Implications for the DSM-5 personality disorder proposal. *Journal of Abnormal Psychology*, *121*(4), 944–950. doi:10.1037/a0027410

Morris, A. S., Silk, J. S., Steinberg, L., Myers, S. S., & Robinson, L. R. (2007). The Role of the Family Context in the Development of Emotion Regulation. *Social Development (Oxford, England)*, *16*(2), 361–388. doi:10.1111/j.1467-9507.2007.00389.x

Nigg, J. T., & Hill, H. (1994). Genetics of personality disorders: Perspectives from personality and psychopathology research. *Psychological Bulletin*, *115*(3), 346–380. doi:10.1037/0033-2909.115.3.346

Nigg, J. T., Silk, K. R., Stavro, G., & Miller, T. (2005). Disinhibition and borderline personality disorder. *Development and Psychopathology*, *17*(4), 1129–1149.

Nunes, P. M., Wenzel, A., Borges, K. T., Porto, C. R., Caminha, R. M., & de Oliveira, I. R. (2009). Volumes of the hippocampus and amygdala in patients with borderline personality disorder: a meta-analysis. *Journal of Personality Disorders*, *23*(4), 333–345. doi:10.1521/pedi.2009.23.4.333

O'Connor, T. G., Hetherington, E. M., Reiss, D., & Plomin, R. (1995). A twin-sibling study of observed parent-adolescent interactions. *Child Development*, *66*(3), 812–829.

Paris, J. (2003). Personality disorders over time: precursors, course and outcome. *Journal of Personality Disorders*, *17*(6), 479–488.

Pfohl, B., Blum, N., & Zimmerman, M. (1997). *Structured Interview for Dsm-IV Personality: Sidp-IV*. American Psychiatric Pub.

Putnam, K. M., & Silk, K. R. (2005). Emotion dysregulation and the development of borderline personality disorder. *Development and Psychopathology*, *17*(4), 899–925.

Reeves, M., James, L. M., Pizzarello, S. M., & Taylor, J. E. (2010). Support for Linehan's biosocial theory from a nonclinical sample. *Journal of Personality Disorders*, *24*(3), 312–326. doi:10.1521/pedi.2010.24.3.312

Reich, D. B., & Zanarini, M. C. (2001). Developmental aspects of borderline personality disorder. *Harvard Review of Psychiatry*, *9*(6), 294–301.

Repetti, R. L., Taylor, S. E., & Seeman, T. E. (2002). Risky families: Family social environments and the mental and physical health of offspring. *Psychological Bulletin*, *128*(2), 330–366. doi:10.1037/0033-2909.128.2.330

Roepke, S., Vater, A., Preissler, S., Heekeren, H. R., & Dziobek, I. (2013). Social cognition in borderline personality disorder. *Frontiers in Neuroscience*, *6*. doi:10.3389/fnins.2012.00195

Rosenthal, M. Z., Cheavens, J. S., Lejuez, C. W., & Lynch, T. R. (2005). Thought suppression mediates the relationship between negative affect and borderline personality disorder symptoms. *Behaviour Research and Therapy*, *43*(9), 1173–1185. doi:10.1016/j.brat.2004.08.006

Sanislow, C. A., Morey, L. C., Grilo, C. M., Gunderson, J. G., Shea, M. T., Skodol, A. E., Stout, R. L., et al. (2002). Confirmatory factor analysis of DSM-IV borderline, schizotypal, avoidant and obsessive-compulsive personality disorders: findings from the Collaborative Longitudinal Personality Disorders Study. *Acta Psychiatrica Scandinavica*, *105*(1), 28–36.

Sauer, S. E., & Baer, R. A. (2009). Relationships between thought suppression and symptoms of borderline personality disorder. *Journal of Personality Disorders*, *23*(1), 48–61. doi:10.1521/pedi.2009.23.1.48

Sauer, S. E., & Baer, R. A. (2010). Validation of Measures of Biosocial Precursors to Borderline Personality Disorder: Childhood Emotional Vulnerability and Environmental Invalidation. *Assessment*, *17*(4), 454 –466. doi:10.1177/1073191110373226

Siever, L. J., Livesley, W. J., Gunderson, J. G., Pfohl, B., & Widiger, T. A. (2002). The borderline diagnosis II: biology, genetics, and clinical course. *Biological Psychiatry*, *51*(12), 951–963.

Silk, J. S., Steinberg, L., & Morris, A. S. (2003). Adolescents' Emotion Regulation in Daily Life: Links to Depressive Symptoms and Problem Behavior. *Child Development*, *74*(6), 1869–1880.

Silk, J. S., Vanderbilt-Adriance, E., Shaw, D. S., Forbes, E. E., Whalen, D. J., Ryan, N. D., & Dahl, R. E. (2007). Resilience among children and adolescents at risk for depression: Mediation and moderation across social and neurobiological contexts. *Development and Psychopathology*, *19*(3), 841–865. doi:10.1017/S0954579407000417

Silk, J. S., Ziegler, M. L., Whalen, D. J., Dahl, R. E., Ryan, N. D., Dietz, L. J., Birmaher, B., et al. (2009). Expressed emotion in mothers of currently depressed, remitted, high-risk, and low-risk youth: links to child depression status and longitudinal course. *Journal of Clinical Child and Adolescent Psychology: The Official Journal for the Society of Clinical Child and Adolescent Psychology, American Psychological Association, Division 53*, *38*(1), 36–47. doi:10.1080/15374410802575339

Silk, K., Lee, S., Hill, E., & Lohr, N. (1995). Borderline personality disorder symptoms and severity of sexual abuse. *Am J Psychiatry*, *152*(7), 1059–1064. doi:

Skodol, A. E., & Bender, D. S. (2003). Why are women diagnosed borderline more than men? *The Psychiatric Quarterly*, *74*(4), 349–360.

Skodol, A. E., Gunderson, J. G., Pfohl, B., Widiger, T. A., Livesley, W. J., & Siever, L. J. (2002). The borderline diagnosis I: psychopathology, comorbidity, and personality structure. *Biological Psychiatry*, *51*(12), 936–950.

Skodol, A. E., Gunderson, J. G., Shea, M. T., McGlashan, T. H., Morey, L. C., Sanislow, C. A., Bender, D. S., et al. (2005). The Collaborative Longitudinal Personality Disorders Study (CLPS): overview and implications. *Journal of Personality Disorders*, *19*(5), 487–504. doi:10.1521/pedi.2005.19.5.487

Skodol, A. E., Pagano, M. E., Bender, D. S., Shea, M. T., Gunderson, J. G., Yen, S., Stout, R. L., et al. (2005). Stability of functional impairment in patients with schizotypal, borderline, avoidant, or obsessive-compulsive personality disorder over two years. *Psychological Medicine*, *35*(3), 443–451.

Skodol, A. E., Siever, L. J., Livesley, W. J., Gunderson, J. G., Pfohl, B., & Widiger, T. A. (2002). The borderline diagnosis II: biology, genetics, and clinical course. *Biological Psychiatry*, *51*(12), 951–963.

Soeteman, D. I., Hakkaart-van Roijen, L., Verheul, R., & Busschbach, J. J. V. (2008). The economic burden of personality disorders in mental health care. *The Journal of Clinical Psychiatry*, *69*(2), 259–265.

Soloff, P. H., Lynch, K. G., & Kelly, T. M. (2002). Childhood abuse as a risk factor for suicidal behavior in borderline personality disorder. *Journal of Personality Disorders*, *16*(3), 201–214.

Stanley, B., & Siever, L. J. (2009). The Interpersonal Dimension of Borderline Personality Disorder: Toward a Neuropeptide Model. *American Journal of Psychiatry*, *167*(1), 24–39. doi:10.1176/appi.ajp.2009.09050744

Stepp, S. D., Olino, T. M., Klein, D. N., Seeley, J. R., & Lewinsohn, P. M. (2013). Unique Influences of Adolescent Antecedents on Adult Borderline Personality Disorder Features. *Personality Disorders: Theory, Research, and Treatment*, No Pagination Specified. doi:10.1037/per0000015

Stepp, S. D., Smith, T. D., Morse, J. Q., Hallquist, M. N., & Pilkonis, P. A. (2011). Prospective Associations Among Borderline Personality Disorder Symptoms, Interpersonal Problems, and Aggressive Behaviors. *Journal of Interpersonal Violence*. doi:10.1177/0886260511416468

Stocker, C. M., Richmond, M. K., Rhoades, G. K., & Kiang, L. (2007). Family Emotional Processes and Adolescents' Adjustment. *Social Development, 16*(2), 310–325. doi:10.1111/j.1467-9507.2007.00386.x

Tabachnick, B. G., & Fidell, L. S. (2007). Using Multivariate Statistics: Pearson Education Inc. *Boston, MA*.

Thomas, A., & Chess, S. (1977). *Temperament and development* (Vol. xv). Oxford, England: Brunner/Mazel.

Trull, T. J., Distel, M. A., & Carpenter, R. W. (2011). DSM-5 Borderline Personality Disorder: At the Border Between a Dimensional and a Categorical View. *Current Psychiatry Reports*, *13*(1), 43–49. doi:10.1007/s11920-010-0170-2

Trull, T. J., & Durrett, C. A. (2005). Categorical and Dimensional Models of Personality Disorder. *Annual Review of Clinical Psychology*, *1*, 355–380. doi:10.1146/annurev.clinpsy.1.102803.144009

Trull, T. J., Useda, J. D., Conforti, K., & Doan, B. T. (1997). Borderline personality disorder features in nonclinical young adults: 2. Two-year outcome. *Journal of Abnormal Psychology*, *106*(2), 307–314.

Wagner, A. W., & Linehan, M. M. (1999). Facial expression recognition ability among women with borderline personality disorder: implications for emotion regulation? *Journal of Personality Disorders*, *13*(4), 329–344.

Westen, D., Shedler, J., Durrett, C., Glass, S., & Martens, A. (2003). Personality diagnoses in adolescence: DSM-IV axis II diagnoses and an empirically derived alternative. *The American Journal of Psychiatry*, *160*(5), 952–966.

White, C. N., Gunderson, J. G., Zanarini, M. C., & Hudson, J. I. (2003). Family Studies of Borderline Personality Disorder: A Review. *Harvard Review of Psychiatry*, *11*(1), 8–19. doi:10.1080/10673220303937

Widiger, T. A. (2006). *Dimensional Models of Personality Disorders: Refining the Research Agenda for Dsm-v*. American Psychiatric Pub.

Widom, C. S., Czaja, S. J., & Paris, J. (2009). A prospective investigation of borderline personality disorder in abused and neglected children followed up into adulthood. *Journal of Personality Disorders*, *23*(5), 433–446. doi:10.1521/pedi.2009.23.5.433

Yap, M. B. H., Allen, N. B., & Ladouceur, C. D. (2008). Maternal Socialization of Positive Affect: The Impact of Invalidation on Adolescent Emotion Regulation and Depressive Symptomatology. *Child Development*, *79*(5), 1415–1431. doi:10.1111/j.1467-8624.2008.01196.x

Yap, M. B. H., Allen, N. B., O'Shea, M., di Parsia, P., Simmons, J. G., & Sheeber, L. (2011). Early adolescents' temperament, emotion regulation during mother-child interactions, and

depressive symptomatology. *Development and Psychopathology*, *23*(1), 267–282. doi:10.1017/S0954579410000787

Zanarini, M. C., Frankenburg, F. R., Khera, G. S., & Bleichmar, J. (2001). Treatment histories of borderline inpatients. *Comprehensive Psychiatry*, *42*(2), 144–150. doi:10.1053/comp.2001.19749

Zanarini, M., Williams, A., Lewis, R., Reich, R., Vera, S., Marino, M., Levin, A., et al. (1997). Reported pathological childhood experiences associated with the development of borderline personality disorder. *Am J Psychiatry*, *154*(8), 1101–1106. doi:

Zanarini, M. C., Frankenburg, F. R., Ridolfi, M. E., Jager-Hyman, S., Hennen, J., & Gunderson, J. G. (2006). Reported Childhood Onset of Self-Mutilation Among Borderline Patients. *Journal of Personality Disorders*, *20*, 9–15. doi:10.1521/pedi.2006.20.1.9

Zweig-Frank, H., & Paris, J. (2002). Predictors of outcome in a 27-year follow-up of patients with borderline personality disorder. *Comprehensive Psychiatry*, *43*(2), 103–107.