Exploring the Roles of Trauma, Emotion Dysregulation, and Negative Affect in Borderline Personality Disorder Symptoms among Young Women

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Abstract

By analyzing repeated measures data through advanced statistical methods, this study investigates the critical roles of trauma, emotion dysregulation and negative affect (NA) in terms of influencing borderline personality disorder (BPD) features among a group of urban city young women. Our results evidence significant connections between distinct trauma types and NA aspects, as well as a decline of NA and BPD features during emerging adulthood. Implications from our findings extended prior explorations by showing that several distinct NA-related forms of ED, such as proneness to anger expression, shame, and guilt, may serve as critical channels driving the significant link between emotional abuse and BPD symptoms. More importantly, our findings would potentially lead to novel trauma-informed treatments effectively targeting multiple ED forms among people with BPD. Finally, we contribute critical new information to the nascent topic area on emerging adulthood.

Table of Contents

1.0 Introduction 1

1.1 Statement of the Problem and the Importance of the Study 2

1.2 Definitions (Key Terms and Variables) 3

1.2.1 Conceptualizations of BPD and the Etiological Factors 3

1.2.2 Trauma in Relation to BPD Development 7

1.2.3 Emotion Dysregulation & Negative Affect 9

1.3 Theoretical Foundations of the Study 11

1.3.1 Biosocial Theory 11

1.3.2 Attachment Theory 15

1.3.3 Trauma Theory 18

1.3.4 Synthesis 23

1.4 Statement of the Purpose 24

1.4.1 Study Aims 25

2.0 Literature Review 29

2.1 Overview of the Problem 29

2.1.1 Basic Demographics 29

2.1.2 Variation in BPD Symptomatology 29

2.1.3 Previously Studied Etiological Factors 30

2.2 Prior Research in Relation to BPD Etiology 38

2.2.1 Prior Research in Relation to BPD Etiology 38

2.2.2 Foci and Topics 38

2.2.3 Limitations and Resolutions....39

2.3 Past Interventions.... 42

2.3.1 Psychotherapy....42

2.3.2 Pharmacotherapy....48

2.3.3 Underserved Populations....49

2.3.4 Synthesis....49

2.4 Relevance to Social Work.... 53

3.0 Methodology.... 55

3.1 Overview of the Study.... 55

3.1.1 Research Questions....57

3.2 Study Design.... 58

3.2.1 Data....58

3.2.2 Participants....59

3.3 Variables and Measures.... 60

3.3.1 Independent Variables....60

3.3.2 Dependent Variables....63

3.3.3 Mediator....64

3.3.4 Covariates....64

3.4 Data Analysis.... 66

3.4.1 Aim 1....67

3.4.2 Aim 2....68

3.4.3 Aim 3....68

3.4.4 Missing Data....69

3.4.5 Sample Size Justification 72

4.0 Results 72

4.1 Participant Characteristics 73

4.2 Preliminary Analyses 76

4.2.1 Internal Consistency if Measures 76

4.2.2 Control Variables 82

4.3 Aim 1 82

4.3.1 Regression 82

4.3.2 Cross-lagged Models 85

4.4 Aim 2 96

4.5 Aim 3 105

5.0 Discussion 108

5.1 Findings 109

5.1.1 Distinct Trauma Types and NA Types 109

5.1.2 A Decline of NA and BPD Features 112

5.2 Limitations 115

5.3 Implications 116

5.3.1 Implications for Practice 116

5.3.2 Implications for Policy 118

5.3.3 Implications for Research 119

5.4 Conclusion 120

Bibliography 121

List of Tables

Table 1 A Brief History of Trauma Studies Evolution……20

Table 2 Comparison of Five DBT Treatment Programs…….51

Table 3 A Detailed list of All Measures…….66

Table 4.1 Demographic Characteristics of Study Participants……...74

Table 4.2 Demographic Differences in BPD Scores……..75

Table 5.1 Reliability Statistics of All Measures--CTQ…….77

Table 5.2 Reliability Statistics of All Measures--Anger……...78

Table 5.3 Reliability Statistics of All Measures--GASP……...79

Table 5.4 Reliability Statistics of All Measures--PAI-BOR…….80

Table 5.5 Reliability Statistics of All Measures--DERS……...81

Table 6 Regression Models Predicting BPD Features …….84

Table 7 Path Parameters and Model Fit of Cross-lagged Models……..86,87

Table 8 Path Parameters of Latent Growth Curve Analyses……101

Table 9 Standardized Parameter Estimates for the Multi-level Mediation Model…….106

List of Figures

Figure 1 A Brief Timeline of Evolving BPD Conceptualizations….....6

Figure 2 Proposed Cross-lagged Model….....26

Figure 3 Proposed Latent Growth Curve Models….....26,27

Figure 4 Proposed Mediation Model ….....…….28

Figure 5 Cross-lagged Models of Trait Anger and BPD….....……..88

Figure 6 Cross-lagged Models of Anger Control and BPD….....……..89

Figure 7 Cross-lagged Models of Anger Out and BPD….....……..90

Figure 8 Cross-lagged Models of Anger In and BPD….....……..91

Figure 9 Cross-lagged Models of SW and BPD….....……..92

Figure 10 Cross-lagged Models of NSE and BPD….....……93

Figure 11 Cross-lagged Models of NBE and BPD….....……94

Figure 12 Cross-lagged Models of GR and BPD ….....95

Figure 13 Combined Boxplots of Individual Differences….....……97

Figure 14 Visualization of Correlations among NA and BPD ….....98

Figure 15 Example First-order Latent Growth Curve Model……...100

Figure 16 Curves of Factor Latent Growth Model Predicting Linear Trend in BPD from Linear Trend in Negative Affects…….104

Figure 17 A Path Diagram of Trauma Predicting BPD Partially Mediated via Emotion Dysregulation Dimensions…….107

Figure 18 Pooled Effect Size of Association between Emotional Abuse and BPD Features……...110

1.0 Introduction

Borderline Personality Disorder (BPD) is a challenging clinical disorder characterized by pervasive instability in a range of areas including interpersonal relationships, self-image, and affect, and by marked impulsivity that is manifested in various contexts such as overspending, promiscuous sex, substance use, and/or binge eating. DSM-5 indicates that the median population prevalence is from 1.6% to as high as 5.9%. In particular, BPD affects up to 2% of the general population, up to 10% of psychiatric outpatients, and 20% of psychiatric inpatients (APA, 2013).

Past studies concluded that BPD was more prevalent among females; however, more recent studies found no such gender difference, and also indicated the clinical presentations of specific symptoms (such as self-harm) revealed similar patterns across genders (Sansone et al., 2011). In terms of ethnic difference, studies have indicated inconsistent findings. A review of 17 studies (Akhtar, Byrne, & Doghramji, 1986) implied that less African Americans suffered from BPD. Chavira and colleagues compared three ethnic groups and found disproportionately higher rates of BPD in Hispanic than in Caucasian and African American participants (Chavira et al., 2003). However, Castaneda and colleagues (1985) reported no racial differences in a retrospective study among a clinical sample (Castaneda & Franco, 1985).

Both theories and extant research studies point to the critical roles of childhood trauma and emotion dysregulation in the developmental path for BPD. Despite the theoretical foundations and empirical evidence, our knowledge of the underlying mechanisms in the development and maintenance of BPD remains limited.

1.1 Statement of the Problem and the Importance of the Study

BPD leads to many adverse psychosocial impacts, including excessive, and yet potentially avoidable, utilization of medical services (Lieb et al., 2004). In terms of direct financial expenses related to BPD, a European study conducted a comprehensive analysis of costs in various areas including all healthcare costs, medication, informal care, productivity losses, and out-of-pocket expenses (van Asselt, Dirksen, Arntz, & Severens, 2007). A study of an adult population in the Netherlands (N=11,990,942) calculated a 1.1% prevalence rate for BPD (N=131,900). For the derived total BPD population, they concluded the total bootstrapped annual cost of BPD to be €2,222,763,789 (€1,372,412,403–€3,260,248,300), and for each individual the cost would be €16,852 (22% of this cost was healthcare related).

Apart from the aforementioned tangible costs, BPD creates functional impairment and instabilities at all levels, from individuals to families and societies. For example, studies indicate that chaotic interpersonal relationships as a consequence of BPD symptomatology often result in marital distress and violence. In addition, labile emotions and splitting behaviors characteristic of BPD are reported to negatively impact employment outcomes, and to be detrimental to the work environment for co-workers (Lieb et al., 2004).

The etiology of the disorder has been continuously debated since the 1930s when the term "borderline" was initially formalized by Stern (1938). Understanding the development of this disorder is essential in formulating early, effective intervention approaches, and in addressing the wide array of adverse impacts as mentioned before. Knowledge of key etiological factors such as emotion dysregulation and trauma exposure will allow for early identification of BPD traits, and therefore facilitate effective prevention and intervention.

1.2 Definitions – (Key Terms and Variables)

1.2.1 Conceptualizations of BPD and Etiological Factors

Early attempts in naming borderline personality disorder (BPD) originated from the psychoanalytic literature in the early 20th century. From 1930s through 1950s, while a group of researchers regarded BPD as a mild form of schizophrenia, others deemed it as a separate, distinct category of disorder (Goldstein, 1983). For the former work, several terminologies were coined to describe patients who appeared to have mild schizophrenic symptoms, such as "ambulatory schizophrenia" (Zilboorg, 1941), "pseudoneurotic schizophrenia" (Hoch & Polatin, 1949), and "latent psychosis" (Goldstein, 1983). For the latter group that considered this disorder to be distinct, Stern (1938) was the first to use "borderline" formally, Deutsch (1945) used "as if personality", and Frosch (1964) created a term "psychotic character".

BPD first became recognized as an official psychiatric diagnosis in the 1980s with the publication of the third edition of the American Psychiatric Association's Diagnostic and Statistical Manual of Mental Disorders (DSM-III; Spitzer & Williams, 1980). Different from the previous psychoanalytic approach, DSM-III's definition of BPD was based on a research-oriented approach, in which empirical methods and statistical models were utilized to identify a checklist of symptoms (Goldstein, 1983). Using a behavioral checklist, DSM-III laid out eight specific characteristics, including impulsivity, self-harm, unstable interpersonal relationships, intense inappropriate anger, identity disturbance, affective instability, intolerance of aloneness, and chronic feelings of emptiness or boredom. An individual who met five or more of those eight criteria would be diagnosed with BPD.

Carrying on the research-oriented approach, DSM-IV required five out of nine criteria to be met to diagnose BPD (Frances, 1994), including fear and avoidance of abandonment, unstable interpersonal relationships (i.e., fluctuating between idealization and devaluation), unstable sense of self, impulsivity in areas such as spending and substance use, suicidality and self-injurious behaviors, emotion dysregulation, chronic emptiness, uncontrolled or inappropriate anger, and transient stress-related paranoid ideation or dissociative symptoms.

Without significant change in the language from the fourth edition, DSM-5 defines BPD as a clinical disorder characterized by pervasive instability in a range of areas including interpersonal relationships, self-image, and affect, and by marked impulsivity that is manifested in various contexts (e.g., spending, sex, substance use, binge eating; American Psychiatric Association., 2013). One central characteristic of BPD individuals is their marked reactivity of mood, including intense episodic dysphoria, irritability, or anxiety lasting from a few hours to days, which usually results from a hypersensitivity to interpersonal stresses. Another key BPD feature concerns unstable and intense interpersonal relationships, which often result from sudden and dramatic shifts in their view of others (either idealizing or devaluating other people). Additionally, a persistently unstable sense of self is also characteristic of BPD, which could manifest in quick and extreme shifts in goals, values, sexual identity, friend types, and/or vocational aspiration (American Psychiatric Association., 2013). DSM-5 further indicates that the median population prevalence is from 1.6% to as high as 5.9%. In particular, BPD affects up to 2% of the general population, up to10% of psychiatric outpatients, and 20% psychiatric inpatients.

The term “borderline” was first used by Stern (1938) to refer to a clinical condition seemingly sharing both neurosis and psychosis; since then, “borderline” still remained as an inconsistent clinical construct until 1970s (Gunderson, 2009). Zanarini et al. (1997) described the

evolution of borderline as in six varying conceptualizations. The first conceptualization was provided by Kernberg (1967). From a theoretical perspective of object relations and ego psychology, his work thoroughly overviewed and integrated previous psychodynamic writings on BPD. In short, Kernberg (1975) conceptualized BPD as a stable pathological personality organization, with a pattern of ego strengths (such as relative intactness of reality testing and thought processes) and ego weaknesses (such as impulsivity, primitive ego defenses and emotional instability). The second one by Gunderson and Singer (1975), using clinical interviews and statistical analyses, described BPD as a specific form of personality disorder carrying certain characteristic features, such as intense affect and a history of impulsivity. The third one, most popular during the 1960s and 1970s, considered BPD as a schizophrenia spectrum disorder, and Wendy, Kety, Rosental and their colleagues (1967) were among the most prominent scholars representing this group (Goldstein, 1983). A fourth conceptualization, reflecting the work of Donald Klein (1977), highlighted dysphoria and affective lability, and therefore considered BPD as an affective spectrum disorder. The fifth one by Zanarini (1993) conceptualized BPD as an impulse spectrum disorder, bearing similar characteristics of impulse control disorders such as substance use disorders and eating disorder. The sixth one reflected a theoretical perspective of trauma, proposed by J Herman and Van der Kolk (1987), suggesting BPD to be a trauma spectrum disorder. After the sixth one, Linehan (1993) conceptualized BPD as a disorder of emotion dysregulation.

More recently, Gunderson (2009) gave a chronological framework of past conceptualizations. Integrating the overview from Zanarini and Frankenburg and that from Gunderson, we present the evolving variations in conceptualizing BPD from 1930s to present chronologically along with particular emphases on theories and approaches (See a visual display

in Figure 1). Before the 1970s, descriptions of the borderline construct were mostly psychoanalytically oriented, with varying etiological interpretations including abandonment and parenting issues. From the 1970s to 1980s, the general emphasis on empirical approach lead to the development of valid, measurable criteria of BPD. The decade of 1980-1990 became more research oriented and biological psychiatry focused. During this time period, various factors were examined, including BPD patients' responses to medications, BPD's overlap with posttraumatic stress disorder, and high correlations with trauma (especially physical and sexual abuse). In the 1990s, two psychobiological proclivities were proposed by Siever and Davis (1991): affective dysregulation and behavioral dyscontrol; and differential diagnostic questions in relation to bipolar disorder were also raised in this period of time. From 2000 to 2009, the advancement of neurobiological and genetic knowledge and technologies contributed to more evidence supporting the heritability of BPD, and some began to consider it as similar to a "brain disease" (Gunderson,

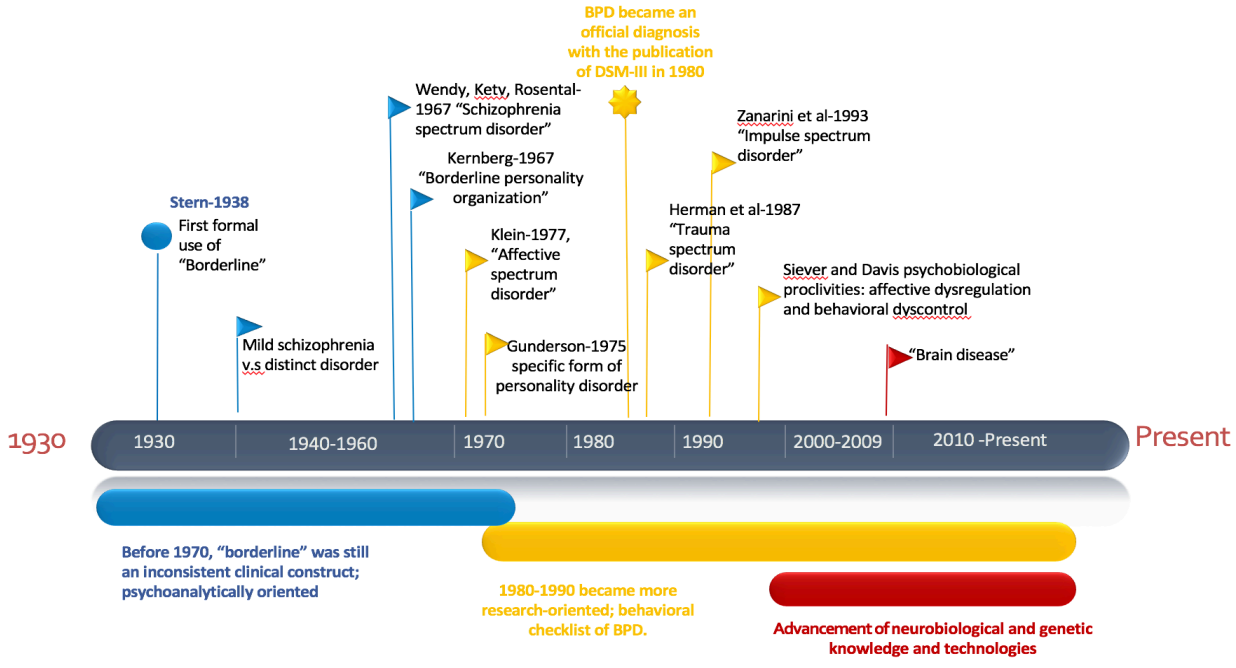


Figure 1 A Brief Timeline of Evolving BPD Conceptualizations.

2009).

1.2.2 Trauma in Relation to BPD Development.

Definitions of Trauma. In the late 19th century, a condition called hysteria was commonly observed among women, many of whom (while receiving treatment for hysteria) indicated psychologically traumatic experiences such as sexual abuse (Ringel & Brandell, 2011). It was the hysteria study that first motivated early academic explanations on trauma. The German word "trauma" originally denotes a wound inflicted on the physical body. Caruth (2016) provided the definition of trauma as a split in the mental processing of time, self, and the world. Freud's definition emphasized two major aspects: the unconscious act (infliction of a wound on the mind) and the repetitions of the act (Freud, 1920). Based on previous attachment theories and empirical work with trauma survivors, contemporary trauma theories in the 1990s further enriched the conceptualizations of trauma. For example, Herman's theory added the concept of the "dialectic of trauma" (such as oscillations between overwhelming emotions and emotional numbing), and she also stressed the multiple origins of complex trauma experience (Herman, 1992). Another branch of trauma theory, Freyd's betrayal trauma, is defined as the development of amnesia in order to maintain the previous trust after the violation of a safe attachment (Freyd, 1994, 1996).

Trauma and Mental Health. As described above, early trauma work originated from studies on hysteria in the 19th century (more history will be discussed in the theory section as follows). Martin Charcot, a French physician, was the first to posit that hysteria was a psychological problem as opposed to a physiological one (a disease originating in the uterus). While working with a group of women in the Salpetriere hospital, Charcot investigated the associations between trauma and mental illness (Ringel & Brandell, 2011). Freud and Janet, however, both influenced by Charcot, had disparate explanations on trauma. Whereas Freud

favored an intrapsychic conflict over external traumatic exposure, Janet focused on dissociation and emotion (Ringel & Brandell, 2011). In addition, Janet's work laid foundations for contemporary trauma theorists, who posited various etiological factors from attachment to dissociation/amnesia and emotion (more will be elaborated in the theory section) (Herman, 1992; Van der Kolk, 2003b).

Trauma and Personality Pathology. Extant studies have consistently identified significant simple correlations between childhood trauma and BPD. Accounting for the above associations, past literature has proposed potential mechanisms such as (1) attachment disruptions (Herman, Perry, & van der Kolk, 1989; Machizawa-Summers, 2007; Venta, Kenkel-Mikelonis, & Sharp, 2012; Weaver & Clum, 1993; Westphal et al., 2013), (2) dissociation (Freyd, 1994; Kaehler & Freyd, 2012), and (3) emotion dysregulation (which will be further explained in the following paragraph) (Gratz, Tull, Baruch, Bornovalova, & Lejuez, 2008; Tyrka, Wyche, Kelly, Price, & Carpenter, 2009). In addition, most literature focused on investigating multiple trauma types as opposed to one specific abuse form, and the commonly tested traumas included emotional abuse, verbal abuse, physical abuse, sexual abuse, and neglect, among which sexual abuse has been most frequently noted to be a significant predictor. Additional trauma indicators were comprised of items such as caregivers' failure to protect, denial of feelings, emotional withdrawal, and non-interpersonal trauma (Afifi et al., 2011; Battle et al., 2004; Goldman, D'Angelo, DeMaso, & Mezzacappa, 1992; Tyrka et al., 2009; Westphal et al., 2013).

ED as a Potential Agent. As aforementioned, a large group of BPD researchers place an emphasis on affect/emotion dysregulation (especially negative affect) associated with early attachment failures. For example, Tyrka et al. (2009)'s work associated the roots of BPD with a sensitivity to negative affect. Moreover, Gratz et al. (2008) directly tested the role of emotion

dysregulation and negative affect in the relationship between childhood trauma and BPD diagnosis status (and symptom count). Comparing individuals with substance use problems with and without BPD, the authors found that both childhood trauma (overall trauma level) and negative affect were significantly associated with BPD status and symptom counts. In addition, emotion dysregulation partially mediated the association between childhood trauma and BPD status, and emotion dysregulation fully mediated the relationship between childhood trauma and BPD symptom count as well as between negative affect and symptom count. In terms of the effect on specific trauma types, emotional abuse was the only significant predictor of BPD status, controlling other forms of abuse and negative affect.

1.2.3 Emotion Dysregulation & Negative Affect

Emotion and Affect. To date, there have been many debates over the terms of emotion and affect. Many scholars use affect and emotion interchangeably (Sheese, 2007), some consider affect as a narrower term in relation to emotion (e.g. only comprising some components of emotions, such as the behavioral experience), and others might consider affect as a hypernym (i.e. a broader construct that is subordinate to emotion). For the sake of consistency and simplicity, here we use the term of affect synonymously with emotion.

ED and NA. By definition, emotion regulation (ER) involves modifying any or all of the emotional processes of self or another(Gross, 2013; Gross & Jazaieri, 2014). Typical examples of those processes include the circumstances of experiencing certain emotions (e.g. when to feel angry), actual emotional states, evaluation of experienced emotions, and selecting how to respond (Sansone et al., 2011). Gross laid out three common features of ER: identification and activation of regulatory aims, skills/capabilities to achieve the aims, and the impacts/results. To fulfill the

tasks falling under these categories, Gross' (2013) ER Modal Model posits that an individual can activate a continuum of regulations over a series of processes in relation to emotions (from initiation to intensity to duration and more).

Nevertheless, ER is not necessarily an effortful or effective one. Effortful regulation is the attentional application of regulatory strategies in order to affect emotional outcomes (for example, one might work to inhibit his/her anger so that aggressive behaviors can be avoided); whereas effective regulation means that one is successful to alter the emotional processes either by increasing or reducing the responses (Rothbart & Bates, 2006), and it involves several common factors such as awareness, goals and strategies (Gross & Jazaieri, 2014). Therefore, emotion dysregulation, a namely unsuccessful regulation, is indeed considered a form of ER. Unsuccessful regulation usually fails to target one or several of ER areas (goals, awareness or strategies). For instance, an individual might lack awareness of his/her own emotions, be unable to activate a goal, and/or lack adaptive strategies to alter the emotional processes (Gross, 2013). Although generic ED can indeed incorporate dysfunctions regulating positive affects (e.g. problematic expression of joy), we consider dysregulation of negative affects more compatible with BPD etiology. In line with biosocial theoretical perspective of BPD, ED can be manifested as an excessive sensitivity to negative stimuli, a heightened intensity of emotional states, and a slow return to baseline (M. Linehan, 1993a, 1993b), all of which are characteristic of BPD individuals.

Shame, Guilt & Anger. Various lines of literature lend support to an elevated sensitivity to negative affects (e.g., shame and anger) and maladaptive strategies in managing negative affects among BPD individuals (Carpenter & Trull, 2013; Cheavens et al., 2005; Gratz et al., 2008). Shame is consistently associated with early experience of sexual abuse and results in a wide array of negative outcomes relevant for BPD pathology, including low self-esteem, negative self-

appraisals, intolerance of negative disapproval and problematic interpersonal relationships (Badour et al., 2017). BPD literature highlights the role of shame in terms of exacerbating and maintaining BPD features (Peters & Geiger, 2016). Likewise, persistent emotional states of anger, shame and guilt were also commonly identified among individuals who suffered from early childhood trauma (Badour et al., 2017). Persistent anger has been noted among trauma survivors, especially for those who later carry a diagnosis of PTSD. In fact, DSM-5 added two anger-related criteria (one is irritation and anger outbursts with little to no provocation; and the other is persistent negative emotional state, including shame, guilt or anger) to particularly address and to accentuate symptoms related to negative emotions (especially anger). Finally, maladaptive regulation of negative emotions contribute to exacerbation and maintenance of several psychopathologies, including BPD (Glück et al., 2017).

1.3 Theoretical Foundations of the Study

The discussions will be focused on (1) how the theories explain BPD etiology, (2) critical analysis of the theory, (3) synthesis of all chosen theories as an explanation of BPD etiology as well as a guidance and foundation for this study.

1.3.1 Biosocial Theory

Millon (Biology and Environment). Different biosocial theorists commonly accentuate the interrelations among various etiological factors, including inherent vulnerabilities, familial influences, early childhood trauma, and broader cultural and social impacts. Being one of the influential personality theorists, Millon (1969) posited a dynamic interplay between biology and environment while explaining multiple personality pathologies (including the borderline type).

According to Millon, each child is born with biologically based attributes that interact with the environment in many different ways, which in turn contribute to several personality variants.

A fundamental component of Millon's theory is based on a three-polarity model of personality: pleasure-pain, active-passive, and self-other (Millon, 1969). This model assumes that personality types are formed as learned strategies to maximize positive reinforcement and minimize punishment. The pleasure-pain polarity represents the positive or negative response from others, such as seeking emotional security or avoiding discomfort. The active-passive polarity means the behaviors utilized by individuals, either passive adaptations or active modifications of environments to suit own needs. The self-other polarity indicates the paradox between maximizing self-production and caring for others. Actually, borderline personality type is characterized by conflicts within all three polarities, as well as frequent fluctuations between opposites extremes. For example, the chaotic relationship pattern and frantic efforts to avoid abandonment can be understood as an interpersonal paradox. The emotional lability can be interpreted as the failure in modifying or adapting own moods to external reality.

Furthermore, Millon (1969) recognizes borderline pathology as an evolutionary continuum. In particular, he highlights three critical substrates in the developmental course of BPD: biological factors, disruptive experiences at early stage of life, and accumulation of adverse life experiences at later stages. These components intersect with each other in a dynamic manner. Biological mechanisms can include heredity or genetic variations. In regard to later environmental influences, both quality and quantity of those experiences play an essential role in the evolutionary course of BPD pathology.

Notably, the environmental aspect in Millon's formulation extends beyond the nuclear family environment and touches upon the pathogenic sources in contemporary societies. He posits

that development of psychopathology is highly correlated with disintegration of socio-cultural institutions (such as regulatory standards, beliefs, and goals) (Millon, 1969). For example, the contradictory standards in contemporary life may potentially lead to inconsistent marital/parental relationships and unstable rearing environment. If experiencing such inconsistency and confusion at an early age and cumulatively, a child born with genetic vulnerability to emotion dysregulation and impulsivities will be more likely to develop borderline personality features.

Linehan (Emotion Dysregulation). Operating from a similar biosocial perspective, Linehan conceptualizes the etiology of BPD as the genetic predisposition coupled with invalidating environmental factors. Based on Linehan's model, BPD is characterized primarily by dysfunction in emotion regulation across all aspects, but particularly with regard to negative affect, which subsequently leads to maladaptive response patterns during emotionally challenging events (M. Linehan, 1993a, 1993b). In addition, certain people are born with such traits as heightened emotional sensitivity, but they do not always develop BPD unless they are met with an invalidating developmental context.

As mentioned above, Linehan (1993) assumes that certain personality traits, such as emotional vulnerabilities, are inherited and thus biologically determined, although they may not be manifested pathologically. Among various personality predispositions, emotion regulation abilities include emotion sensitivity, emotion modulating, and ability to return to emotional baseline. Several studies have reported evidence of genetic influences in the developmental pathways to BPD. For example, in a longitudinal discordant twin study, Bornovalova and colleagues (2013) found that trauma, BPD traits and externalizing and internalizing disorders are all correlated. Further, their findings suggested that the relationship between trauma and BPD traits is better accounted for by genetic risk factors that overlap with externalizing and internalizing

disorders. This offers empirical evidence that genetic influences could serve a vital role in mediating the link between trauma and later development of BPD.

Consistent with what attachment theorists purported, Linehan (1993a) posits that an invalidating developmental context is featured by irresponsiveness or negative reactions toward a child's emotional displays. For example, families can contribute to an invalidating environment simply by ignoring, discounting, minimizing, denying, or judging a child's feelings. Consequently, the child does not learn how to conceptualize, regulate, or tolerate emotional responses under this environment with their emotions being constantly unsupported and invalidated; instead, they could develop maladaptive mechanisms in regulating emotions which fluctuate between emotional inhibition and emotional lability (Crowell et al., 2009). In particular, people who grow up in an invalidating environment learn to believe that their actions, thoughts, and feelings do not matter, or that they cannot trust their perceived emotions, which in turn hinders their ability to recognize and label their emotions. It can also lead to substance abuse or self-harm as a strategy to better cope with and control their emotions.

Biosocial Developmental Model (Impulsivity). Extending Linehan's theory, Crowell and colleagues (2009) have elaborated a biosocial developmental model of BPD. In particular, they argue that emotional behaviors might be influenced by another predisposition (impulsivity) in addition to emotion dysregulation, which could lead to the development of a severe subtype of BPD—impulsive BPD. A unique proposition underlying their extended model is that impulsivity and emotion dysregulation may manifest themselves independent of each other, and lead to different functioning problems. As shown in their extended model, they hypothesize that a developmental pathway for BPD that first starts with predisposing impulsivity (an independent group of heritable vulnerabilities), which later gets intensified by emotion regulation difficulties

coupled with environmental adversities, all dynamically leading to the final manifestations of BPD features (Crowell et al., 2009).

1.3.2 Attachment Theory

Bowlby. Initially developed by John Bowlby (1969) to conceptualize the relationship between a child and their primary caregivers, attachment theory was later extended to adult relationships. According to Bowlby (1969, 1989), the attachment system activates when individuals sense any adversity, which leads to subsequent behaviors eliciting support from attachment figures. Unavailable or irresponsive caregivers could potentially lead to the child experiencing anxiety or insecurity. On the other hand, caregivers would contribute to shape the child's "internal working models" of the self and significant others. This aforementioned attachment system evolves over time, shaped by experiences with and internal representations of these attachment figures. Over time, individuals develop attachment styles (such as secure attachment or insecure attachment), which dictate how they seek and maintain closeness to a person who can facilitate their inner capacity to cope with threats and dangers (Bowlby, 1969).

Ainsworth. Ainsworth and her colleagues (1978, 1979) expanded on Bowlby's work by conducting naturalistic, longitudinal research on attachment styles in infants and their mothers. Their studies showed three distinct attachment styles that infants employ to seek and maintain proximity to an attachment figure: secure, insecure-ambivalent, and insecure-avoidant. According to Ainsworth's work, behavior of caregivers to some extent determines the attachment style of infants. Specifically, responsive and available mothers promote the development of a secure attachment style, with which infants are easily comforted, quickly return to baseline, and are emotionally stable and explorative of the outside world; whereas inconsistent mothers lead to an ambivalent style and unavailable mothers contribute to an avoidant style (Ainsworth, 1978, 1979).

Adult Attachment. Ainsworth's early observations of mother–infant attachment patterns served as a foundation for other theorists to conceptualize how adult attachments are formed and whether they are related to attachment patterns that are observed in young children. Recent attachment theorists and scholars have continuously explored adults' representations of childhood attachment styles. George, Kaplan and Main (1985, 1996) were among the first to conduct adult attachment research and developed the Adult Attachment Interview (AAI) in order to assess adult attachment prototypes. From an adult attachment perspective, Shaver and Hazan (1993) conceptualized romantic love as an attachment process. Utilizing self-report, retrospective measures, they examined and classified adults into three categories corresponding to the three attachment styles of childhood.

Attachment and Trauma. Early attachment theories have laid foundations for contemporary trauma theories which gained particular public attention in 1990s (Radstone, 2007). For example, Freyd's work on betrayal trauma was conceptualized based on the attachment framework. His definition of "adaptive blindness" for childhood trauma actually means a re-integration of memory to avoid attachment disruptions (e.g. loss of trust in attachment figures) (Freyd, 1994; Kaehler & Freyd, 2012). Trauma in relation to BPD development has also been shown by empirical studies to be related to disruption in attachment bonds with primary caregivers (Scaer, 2001). For example, the protest and despair responses displayed in response to parental separation, as observed by Bowlby, parallel the hyper-arousal and numbing states found in some psychiatric disorders including personality disorders (J. L. Herman et al., 1989).

Attachment and BPD. The concepts of attachment system, internal working models and attachment styles all together are useful to comprehend the development of BPD especially in areas of emotion regulation and interpersonal relationships. BPD is characterized by significant

impairment, especially associated with inability to sustain stable relationships as a result of personal and emotional instability, which from the attachment perspective usually result from early disruption of child-caregiver relationships. This particular disruption would contribute to the unstable sense of self and others (Fonagy, 2000).

In particular, Bowlby's work posits that early experiences with caregivers would help to form mental representations of self, others and the world, called internal working models of social relationships, which have great impacts on emotion, memory and cognition development (Bowlby, 1969). Additionally, the internal working models would guide the social interactions of individuals, and later on help to form meaningful friendships, marriage, and other interpersonal relations. For example, children who perceive themselves as worthy of love and care are more likely to assume their attachment figures to be responsive to their needs, and later on form a stable, secure social relationship. Likewise, Ainsworth's work sheds light on the association between early attachment styles and the child's later development of personality, emotion regulation abilities and relations to the attachment figures.

In terms of empirical support, past literature reported empirical evidence supporting the theoretical connection between attachment styles and personality pathology. To date, several studies highlighted the relationship between attachment variables and development of PD (Levy et al., 2005). In particular, both retrospective and longitudinal studies have found connections between disorganization of the attachment system (i.e. insecure attachment styles) and BPD psychopathology. For example, two longitudinal studies reported significant associations between insecure attachment in early childhood and BPD symptoms on follow-up (Lyons-Ruth et al., 2005; Rogosch & Cicchetti, 2005). Levy (2005) also compiled results from a host of studies with regards to the association between attachment anxiety and BPD. Specifically, while some studies reported

inconsistent association between the avoidant attachment and BPD, other research demonstrated correlations between this construct and BPD when anxiety was high.

1.3.3 Trauma Theory

Co-fertilized by a number of fields including psychology, neuroscience and humanities, trauma theory had its inceptive origins from theories of deconstruction, post-structuralism and psychoanalysis (Caruth, 2016). As early as the late 19th century, assumptions on hysteria initially were based on a physiological etiology that the disease originated in the uterus; back then different work on hysteria from a group of theorists (such as Charcot, Janet and Freud) raised questions on the dynamic interplays among dissociation, intense emotions and traumatic memories (Ringel & Brandell, 2011). Questioning the physiological explanation of hysteria, Charcot's work, for the first time, shifted the focus to psychology while addressing the traumatic memories and dissociative symptoms among the hysterical women. Influenced somewhat by Charcot, Freud placed focus on unconscious conflict, repression and fantasy. Rejecting Freudian psychoanalytical approach, Janet then posited that dissociation was the main factor driving the dysfunctional integration of the memories that were due to unbearable traumatic events. In addition to the theoretical work, medical practice with war survivors (WWI&II and Vietnam War) further advanced understanding of experiences that were traumatic, which also helped to demarginalize the trauma-inflicted population and motivate more systematic study of trauma. Actually, the Vietnam war brought posttraumatic stress disorder into the public domain, and for the first time legitimized trauma experiences (from hysterical women to low moral soldiers to a psychological condition). In addition, the 1970s' feminists movement called for attention on domestic violence, which further improved conceptualizations of different trauma types. Influenced by Janet's dissociation theory, contemporary trauma theories boomed in the 1990s, as a joint effort from

fields of psychology, neuroscience and humanities (Ringel & Brandell, 2011). Please see Table 1 for a visual display of trauma work evolution.

Table 1.

A Brief History of Trauma Studies Evolution

Time	Study Focus	Population	Theoretical Explanations
Late 19th century	Hysteria	Woman	Physiological: Hysteria originated in uterus
Charcot	Hysteria	Woman	Psychological origin and mental health focus (dissociation)
Freud	Hysteria	Woman	Fantasy/Intrapsychic Conflict
Janet	Studied the influence of trauma on development of personality and behavior; Amnesia and integration of memory	Woman	Rejected fantasy/seduction theory; dissociation, emotions and memory
World War I&II	Amnesia and somatic symptoms	Soldiers	Systematic studies started; expanded the studies to men
Post-war	War neurosis	Soldiers	Explosion of shells caused the war neurosis (Shell Shock)
Post-war	War neurosis	Soldiers	Weak mental/moral character
Vietnam War	PTSD	All	DSM 1980s
1970s feminists	Domestic violence	Women and children	Raised attention to domestic violence
1990s (Herman and van der Kolk)	Built on previous theoretical and clinical work	All	Enriched the trauma work and improved clinical treatments

Physiology and Psychology. In the l9th century, hysteria was a common disease among women characterized by a great variety of psychological and somatic symptoms (including but are not limited to hallucinations, uncontrolled emotions and nervousness) (Smith-Rosenberg, 1972). It was held that hysteria originated in a wandering uterus (and the Victorian culture labeled those women as fallen and unfeminine). While conducting case studies with a group of women with hysteria, Freud discovered that many women with hysteria revealed traumatic memories, especially sexual trauma. However, later favoring the intrapsychic conflict over external traumatic exposure, Freud attributed the roots of intrusive memories mostly to "unacceptable nature of sexual and aggressive wishes" (Freud, 2015).

Emotion and Dissociation. While treating women diagnosed with hysteria, a group of theorists commonly noted emotional problems and dissociation in response to traumatic memories. Rejecting the physiological argument that hysteria originated in uterus, Charcot was the first to hypothesize a psychological mechanism underlying the disease, and to account for the traumatic experiences among hysterical patients from a mental health perspective. Although influenced by Charcot, Freud's psychoanalysis later on favored an intrapsychic conflict over the external trauma as aforementioned.

A student of Charcot, Pierre Janet's dissociation theory rejected Freud's theory of fantasy and seduction. His work advanced the understanding of dissociation, emotions and trauma. In particular, he believed that dissociation served the function of modulating unbearable memories associated with trauma. Janet's work placed a special emphasis on the role of intense emotions due to the traumatic experience. He believed that such emotions have the strongest effect on "the recently formed and least stable memories", and in turn on personality. Through investigating the impacts of trauma on people's personality, Janet discovered that emotions of high intensity were

responsive to patients' own perceptions of the traumatic experiences (Janet, 1901). Such emotional difficulties will in return have significant effects on forming social relationships and on the development of self (Radstone, 2007).

Trauma and Personality Pathology. Herman, one of the contemporary trauma theorists, posit that trauma events undermine people's adaptive skills and threaten the basic structures of self, and its connections to others and the world (Herman, 1992). A key theoretical underpinning of Herman's trauma perspective concerns the "dialectic of trauma", which means the oscillations between the intense feelings and no feelings, intrusive memories and amnesia, and impulsive action and inhibition. Herman points out that the alternating episodes between the intrusive and numbing symptoms actually represent patients' attempts on balancing the extremes, and yet the dysfunctional "balancing" efforts in turn lead to a self-perpetuating instability. Moreover, Herman highlighted that trauma experiences usually had complex, multiple origins, which could impact various life functioning and personality development areas. Finally, another critical aspect of Herman's theory describes the devastation that trauma brings to the basic structures of self in relation to others and the world due to the impaired regulation and adaptation abilities, hence having great implications for BPD development in relation to trauma (Herman, 1992).

Likewise, recognizing the complex origins and impacts of trauma, van der Kolk's developmental perspective particularly addressed the influence of early childhood trauma on children's later development in many aspects (Van der Kolk, 2003). In a discussion on the neurobiology of childhood trauma, he argued that early exposure to trauma would impair the neurobiological development of children, which might result in difficulties with cognitive processing, regulating emotions and establishing functional relationships. Furthermore, he noted emotion regulation inability as the most striking characteristic among children suffering from

chronic trauma, which subsequently leads to a variety of problems such as lacking a sense of self, impulsivity, affect dysregulation and relationship issues, which are indeed key aspects characteristic of BPD. Supporting the above theoretical arguments, through his empirical work, van der Kolk reported a significantly higher likelihood for traumatized children to develop a group of psychiatric disorders, including BPD (Van der Kolk, 2017)

Built on previous work of dissociation and emotion, Freyd's (2009) conceptualization of betrayal trauma was originally developed from the attachment framework. This branch of trauma theory addresses the pathological development of amnesia— "adaptive blindness"—for childhood trauma (Freyd, 1994, 1996). According to Freyd, a betrayal trauma is a type of trauma concerning a violation of trust for survival, which from attachment theoretical perspective is the disruption in the caregiver-child bond. In order to maintain the trust, the child who suffered interpersonal trauma (such as sexual abuse from a primary caregiver) would subsequently develop mechanisms in order to remain unconscious of the violation, which is often accomplished by dissociation. In terms of associations with BPD etiology, Kaehler and Freyd (2012) examined the betrayal trauma in regards to BPD development by recruiting a community sample. Results showed that high-betrayal trauma (interpersonal trauma: such as sexual abuse from a primary caregiver) was more significantly associated with BPD traits.

1.3.4 Synthesis

In general, attachment theory attributes the development of core BPD features to the roots of disrupted attachments, which is especially helpful to decipher how early attachment styles and internal working models may influence the later formulation of sense of self and others, as well as affective regulation abilities. Built upon and expanded from attachment theory, several psychotherapies have been developed to treat BPD symptoms, one of which is mentalization-based

treatment (more details will be elaborated in the following section). Having early origins from psychoanalysis and dissociation theory, different branches of trauma theories all together touched upon several aspects of emotion dysregulation in relation to personality pathology. While attachment theory and trauma theory mostly focus on the individual level of dysfunction, biosocial theory complements them by touching upon the macro level (such as social networks), as well as its transactions with the micro and mezzo levels (e.g. inherited vulnerabilities). Therefore, biosocial perspective can provide a dynamic and holistic lens while addressing biopsychosocial factors at each level as well as their interactions.

Indeed, each of the theories discussed above lends itself to one or more key aspects in the intertwined etiological web. In comparison, biosocial theory has some superiority over attachment theory in that it stresses the dynamic interplay among various etiologies in addition to individual factors. Likewise, contemporary trauma theory has its advantage over early attachment work as it does not reduce the etiology to a linear, single-factor explanation. Despite the limitations, attachment theories laid foundations for later work on dissociation, emotion, and memory, which are all key aspects to consider in BPD development. In conclusion, a research study informed by multiple theoretical frameworks would have great impact in terms of adequately addressing critical key variables including dimensions of ED and trauma and its subtypes, which all have valid theoretical foundations in the development of BPD.

1.4 Statement of the Purpose

As discussed above, attachment and trauma theories both have critical implications for potential etiological factors that are worth examination at micro and macro-level. Complementing those two theories, biosocial theory emphasizes the importance of dynamic interplay among

factors. Integrating those previously discussed theories, this study aims to decipher the role of two important etiological factors (trauma and ED) in the context of a variety of environmental influences, with a long-term goal to enhance knowledge on early, effective interventions as well as to improve macro-level policies in response to the problem.

1.4.1 Study Aims

In general, this research aims to advance the extant knowledge on BPD features in relation to early childhood trauma exposure and emotion dysregulation, via examining the paths from trauma to emotion dysregulation, and to BPD features. In particular, this proposed research aims to:

Aim 1: Examine the association patterns among trauma, ED, and BPD symptoms by (1) testing the relationships among all variables at baseline as well as (2) investigating the directional relationships between ED and BPD (controlling for trauma history) across time points.

Hypothesis 1a: Both trauma and ED will significantly predict higher BPD features at the baseline. Hypothesis 1b: Repeated measures of participants' negative affect (shame/guilt and anger) will predict participants' BPD symptoms across time points (Figure 2[1]).

[1] Two separate models will be conducted to test two sets of NA: (1) Shame/guilt and (2) anger. NA_{t1}= Shame/Guilt and Anger Scores at Baseline, NA_{t2}= Shame/Guilt and Anger Scores at Wave 2, NA_{t3}= Shame/Guilt and Anger Scores at Wave 3, BPD_{t1}= BPD Scores at Baseline, BPD_{t2}= BPD Scores at Wave2, BPD_{t3}=BPD Scores at Wave 3.

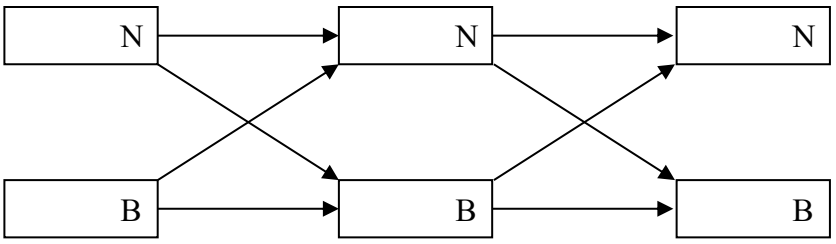
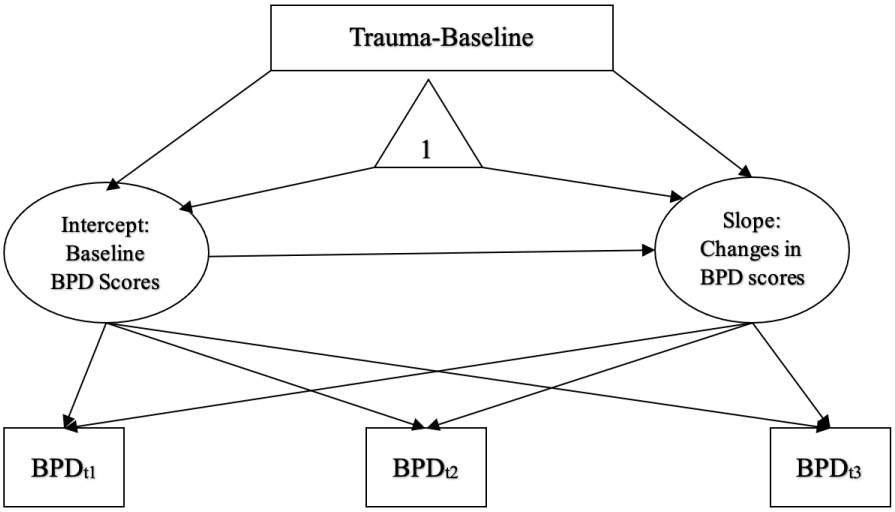


Figure 2 Proposed Cross-lagged Model

Aim 2: Examining the trajectory of BPD features and ED problems in the context of trauma history by testing the changes between waves.

Hypothesis 2: Trauma will predict both higher mean BPD scores at baseline, and steeper changes in BPD symptoms over time; similarly, trauma will predict both higher mean negative affect scores at baseline, and steeper changes in negative affect over time (Figure 3[2]).



[2] *Note*. For the NA outcome, two separate models will be conducted to test: (1) Shame/guilt and (2) Anger. NA_{t1}= Shame/Guilt and Anger Scores at Baseline, NA_{t2}= Shame/Guilt and Anger Scores at Wave 2, NA_{t3}= Shame/Guilt and Anger Scores at Wave 3, BPD_{t1}= BPD Scores at Baseline, BPD_{t2}= BPD Scores at Wave2, BPD_{t3}=BPD Scores at Wave 3.

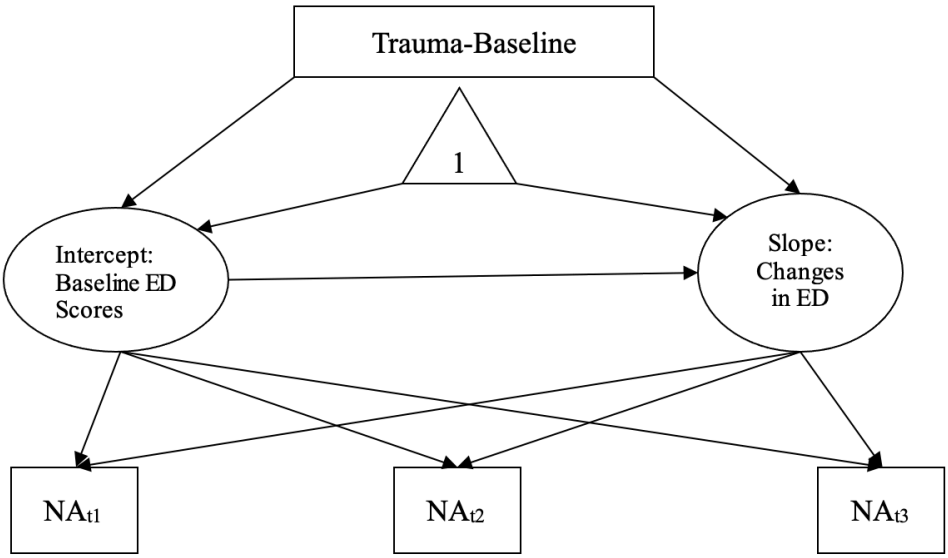


Figure 3 Proposed Latent Growth Curve Models

Aim 3: Investigate the mechanisms through which trauma plays a role in predicting BPD features (Figure 4[3]).

Hypothesis 3a: ED will partially mediate the path from trauma to BPD features at baseline.

Hypothesis 3b: Controlling for ED, trauma still predicts higher BPD features at baseline.

[3] Note. T1= Sexual Abuse, T2= Physical Abuse, T3=Emotional Abuse, T4=Physical Neglect, T5=Emotional Neglect.

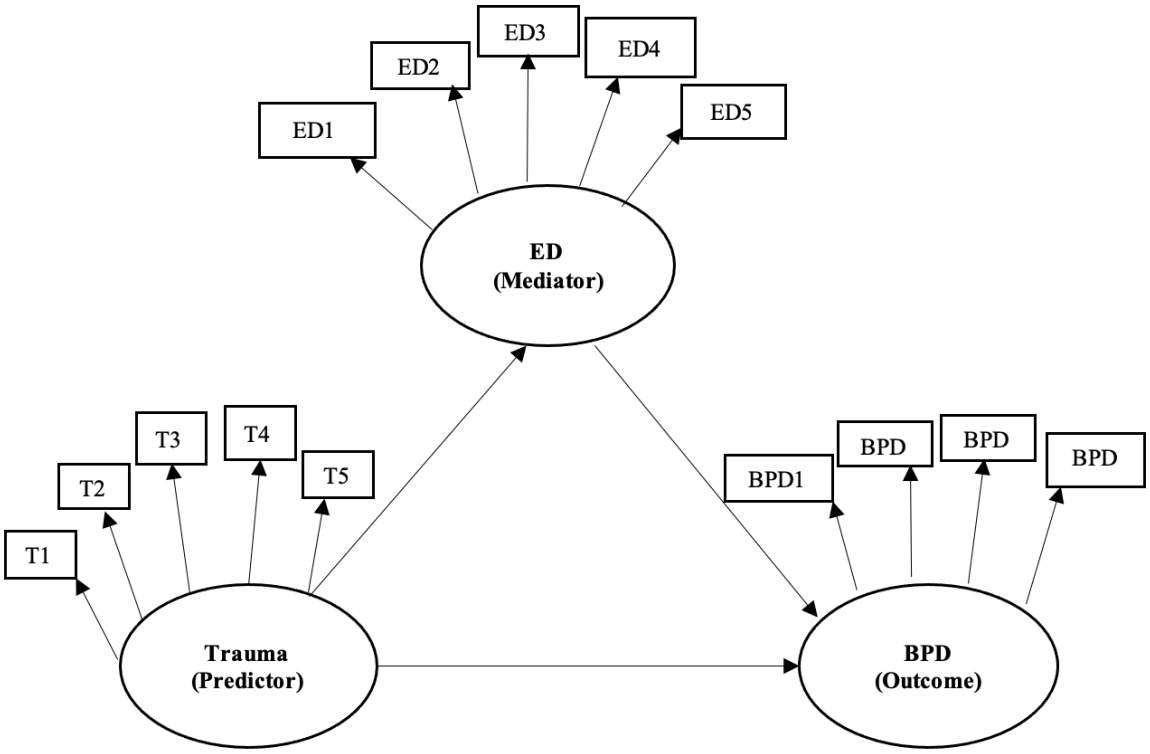


Figure 4 Proposed Mediation Model

2.0 Literature Review

The following sections depicts a comprehensive review on how the problem has been studied, including the trends and variations, previously investigated etiologies, methods of past research and their limitations, extant efforts to resolve the limitations, and current treatments/interventions. Finally, the importance of our study to social work will also be discussed.

2.1 Overview of the Problem Studied

2.1.1 Demographic Profile

As previously mentioned, BPD affects up to 2% of the general population, up to 10% of psychiatric outpatients, and 20% of psychiatric inpatients (American Psychiatric Association., 2013). Statistics from a national community sample (National Epidemiologic Survey on Alcohol and Related Conditions) indicated approximately a prevalence rate of 2.7% of BPD among adults in America. Specifically, BPD disproportionately affects the general population, with a slightly higher rate among females, low-income individuals, people under 30, and people that are not in a relationship (either separated or divorced). There is also a significantly higher BPD prevalence among Native Americans and Blacks, and a significantly lower rate among Asians (Tomko, Trull, Wood, & Sher, 2014). Extra variations for diverse populations are discussed as follows.

2.1.2 Variations in BPD Symptomatology among Different Racial/Ethnic/Gender groups

As mentioned above, past studies concluded that BPD was a more prevalent disorder among females. However, although the DSM-5 (APA, 2013) maintains that BPD is diagnosed predominantly in females, more recent studies found no such gender difference, indicating the clinical presentations of specific symptoms, such as self-harm, revealed similar patterns across different genders (Sansone et al., 2011).

In terms of racial and ethnic differences, studies have reported inconsistent findings. A review of 17 studies implied that fewer African Americans suffered from BPD (Akhtar, 1986). Castaneda & Franco (1985) reported no racial difference in a retrospective study among a clinical sample comprised of whites, blacks and Hispanics. A more recent study by Chavira and colleagues indicated disproportionately higher rates of BPD in Hispanic than in Caucasian and African American participants (Chavira et al., 2003). Additionally, there is some evidence that BPD symptomatology may differ across races. For example, Newhill et al. (2009) examined the affective and behavioral symptoms of BPD in a sample of 17 African Americans and 27 White Americans, with results indicating that African Americans with BPD experience greater affective intensity and emotional dysregulation, fewer self-harming behaviors, and more thoughts of interpersonal aggression than Whites.

2.1.3 Previously Studied Etiological Factors

Environmental factors. Past BPD studies have concluded three primary environmental factors for developing BPD, including early childhood separations, disturbed parental relations and early childhood abuse (Zanarini et al., 1997). A large body of literature, primarily behavioral studies, have examined various factors that might predict BPD development while trauma exposure remains at the forefront of the etiological research. These environmental adversities are generally considered potential risk factors for a wide array of mental disorders, including BPD; nevertheless, these risk factors do not necessarily serve as determinant or causal influences leading to such psychopathology. Finally, the likelihood of the risk can be greatly enhanced if these factors intersect with biological factors (such as inherited temperamental vulnerabilities) (See more discussion on the dynamic biopsychosocial interaction below).

Trauma. Past studies have consistently identified significant simple correlations between childhood traumas and BPD. In addition, most literature focused on investigating multiple trauma types as opposed to one specific abuse form, and the commonly tested traumas included emotional abuse, verbal abuse, physical abuse, sexual abuse, and neglect, among which sexual abuse has been most frequently noted to be a significant predictor. Additional trauma indicators were comprised of items such as caregivers' failure to protect, denial of feelings, emotional withdrawal, and non-interpersonal trauma (Afifi et al., 2011; Battle et al., 2004; Goldman et al., 1992; Tyrka et al., 2009; Westphal et al., 2013).

Aside from the trauma types, other dimensions of trauma have also been examined in the past studies, including the nature of trauma events, onset, and the role of perpetrator. For instance, Westphal and colleagues (2013) examined differential effects of interpersonal trauma (physical/sexual assault, unwanted sexual experiences, and assault with a weapon) and non-interpersonal trauma (natural disasters, accidents, illnesses, death of a close person), in which they identified that interpersonal trauma types were strongly associated with BPD whereas non-interpersonal ones were associated with BPD only if the traumatic experience had occurred during childhood. Battle and colleagues (2004) measured caretaker perpetrated sexual abuse and non-caretaker sexual abuse, and found that effect sizes of sexual abuse from caretakers were consistently larger than those of sexual abuse from non-caretakers, indicating the essential role of the perpetrator's relationship with the victim in accounting for the association.

Family environment. As often reported, adverse or invalidating rearing environments (that are not necessarily of a traumatic nature) can also contribute to BPD pathology. For example, a Japanese study reported that parental overprotection and trauma exposure were both significantly related to the BPD diagnosis (Machizawa-Summers, 2007). Similarly, Weaver & Clum (1993)

reported that high familial control in conjunction with frequent sexual abuse strongly predicted the severity of BPD symptomatology. Two more recent studies focusing on adolescent populations indicated that harsh parenting might be associated with subsequent BPD symptoms whereas positive parenting could have a reciprocal relationship with BPD features (Belsky et al., 2012; Stepp et al., 2014).

Macro-level environment. Another line of studies suggested that social and cultural factors, such as a lack of social and community support system, could add to the individual vulnerabilities for developing a BPD. For example, differential prevalence of BPD observed in different geographical areas was reported to be associated with the disparities in social capital (Paris & Lis, 2013). While discussing BPD etiologies, Millon (1969) suggests several pathogenic sources in contemporary societies. According to him, the increasing prevalence of BPD is highly correlated with disintegration of socio-cultural institutions (such as regulatory standards, beliefs, and goals). For example, the contradictory standards in contemporary life (caused by rapid industrializations, urbanization and high mobility) may potentially lead to inconsistent marital/parental relationships and unstable rearing environment. If experiencing such inconsistency and confusion at an early age and accumulatively, a child born with genetic vulnerability to emotion dysregulation and impulsivities will be more likely to develop borderline personality features. However, extant research that directly investigates such macro-level factors remain scarce, possibly due to the difficulties of operationalization and instrumentation.

Biological factors. In recent years, attempts on providing a genetic explanation of BPD development continue to grow. On average, twin studies report that the heritability of BPD to be approximately 40% (Amad et al., 2014). Nevertheless, understanding of the biological correlates is still limited due to the difficulties in integrating all the biopsychosocial influences as well as

making logical connections. Current biological research in the context of BPD mainly examines the roles of neurotransmitter genes, brain structure and function, and neurochemistry (Crowell et al., 2009).

While twin studies attempt to disentangle the impacts of genetic factors from shared/non-shared environmental factors, genetic studies aim to further identify the effects of specific genes. Neurotransmitter systems genes have been most prevalently examined, including serotonin (5-HT), dopamine, vasopressin, acetylcholine, and noradrenaline systems genes (Amad et al., 2014; Crowell et al., 2009). As far as the brain factors in relation to BPD pathology, neuroimaging studies have produced varied results which sometimes can be hard to interpret. In the following paragraphs we will review and discuss the currently discovered genetic and brain etiological factors of BPD.

Serotonin(5-HT) system. A growing body of literature has consistently reported significant associations between deficits in the central serotonin system and several BPD features, including impulsivity, suicidality, aggression and emotional lability (Amad et al., 2014; Maurex et al., 2009; Perez-Rodriguez et al., 2010; Zaboli et al., 2006). Specifically, researchers have investigated the following serotonin system genes: tryptophan hydroxylase (TPH-1 and -2, a rate-limiting enzyme) genes, serotonin receptor genes, serotonin transporter genes, and monoamine oxidase A (*MAOA*) genes. TPH-1 and -2 both control the synthesis process of serotonin, and *MAOA* gene is involved in the breakdown process of serotonin and norepinephrine.

Several studies have associated polymorphisms in TPH-1 genes with a list of BPD features such as suicidality, aggression and emotional lability (Amad et al., 2014). In addition, one study using the haplotype approach identified a significant connection between polymorphism in TPH-2 gene and symptoms of anxiety, depression and suicidal behavior among BPD individuals (Perez-Rodriguez et al., 2010). Focusing on candidate genes, another group of studies suggested that

5HTT (a serotonin transporter gene) might contribute to BPD related features of suicidal and impulsive behaviors, emotional lability, and addiction (Amad et al., 2014). As far as *MAOA*, past studies consistently reported its link to impulsivity and aggression. Some studies implied that polymorphisms in *MAOA* genes might interact with environmental risk factors, hence increasing the likelihood to develop impulsivity and aggression (Crowell et al., 2009).

Dopaminergic system. There exists a limited amount of work directly testing dopamine dysfunction among BPD population. Some studies reported the correlation between dopamine dysfunction and key dimensions of BPD such as emotional processing, impulse control and cognition. One study testing Catechol-O-methyltransferase gene (COMT, an enzyme that breaks down dopamine) indicated the potential interaction effect between dopamine and serotonin systems in the development of BPD (Amad et al., 2014). Limitations in this area reside within not only the amount of conducted studies, but also the unclear interpretations of identified connections. One example is the debate between hyperfunction and hypofunction of dopamine system. While some researchers suggest that dopamine abnormalities arise from hyperdopaminergic functioning that in turn leads to psychotic features in BPD (Joyce et al., 2014), others postulate that those abnormalities are indeed associated with hypodopaminergic functioning that accounts for impulsivity (Sagvolden et al., 2005). Crowell et al. (2009) have reviewed recent findings from a group of studies and they conclude that BPD features of impulsivity and negative affectivity are more consistently related to hypodopaminergic functioning state.

Other neurotransmitter systems. Additional neurotransmitter systems have also been examined, including vasopressin system, cholinergic system, noradrenergic system and hypothalamic–pituitary–adrenal (HPA) system, among which HPA is more often investigated. Past research has associated HPA axis hyperactivity (due to cortisol nonsuppression) with suicidal

behavior (Crowell et al., 2009). A more recent study supported the role of the HPA axis in the developmental course of BPD by showing that HPA genes (*FKBP5* and *CRHR* genes) were associated with BPD diagnosis, and the same study also found an interaction effect between HPA genes and environmental factors (childhood trauma) (Martin-Blanco et al., 2016).

Brain structure. In terms of brain structure, relatively more consistent results come from volumetric studies examining the regions of hippocampus and anterior cingulate cortex (ACC), as compared to the ones investigating other regions such as amygdala. Reportedly, BPD individuals often show volume reductions in hippocampus and ACC, and a negative correlation has also been observed by some studies between hippocampal volume and a history of childhood trauma (Amad et al., 2014). However, the observed results from other brain regions seemed to be varied and difficult to interpret. For example, in the amygdala region, some researchers found volumetric reductions whereas others found no significant difference or increased grey matter among BPD individuals relative to healthy controls. Further, different results can potentially point to distinct mechanisms for BPD symptomatology. While the reduced amygdala volumes could account for emotional dysregulation, the larger grey matter concentration might explain heightened responses to emotional stimuli (Minzenberg et al., 2008). It is meaningful to note that DSM uses a polythetic criteria set for BPD which might potentially lead to more than 200 different presentations of symptoms; and thus, heterogeneity of symptomatology within and across BPD samples can indeed account for inconsistent results across studies.

Brain function. fMRI and PET studies are overall consistent with each other in terms of finding functional abnormalities among BPD individuals. The most commonly reported finding concerns a greater amygdala activity of BPD participants relative to healthy controls during exposures to negative emotional stimuli (such as viewing emotion-inducing slides or performing

aggression provoking tasks). Additionally, amygdala activity shows a positive association with affective lability (assessed via self-report measures). Current research has also identified a negative connection between activities in the prefrontal cortex and the level of impulsivity among BPD population (O'Neill & Frodl, 2012).

In conclusion, multiple lines of evidence support the genetic and brain etiological factors of BPD, including polymorphisms in the neurotransmitter system genes as well as several brain structural and functional abnormalities. However, studies in this area sometimes produce mixed results and still require further replications. In addition, interpretations of the mechanisms remain somewhat unclear. One example regards the unresolved debate in dopamine dysfunction as aforementioned. Another example is the interpretation of observed amygdala activity as either direction could point to distinct pathogenesis of BPD. Finally, while considering candidate genes for BPD, the genetic mechanisms might be better explained by moderation or mediation through multiple groups of genes given the heterogeneity and high comorbidity (Amad et al., 2014).

Transactions between biological and environmental factors. The moderating effect in terms of the interactions between biology and environment has long been posited and tested by many BPD researchers. Stone (1977) accounted for the transactions during the development of BPD with a diathesis-stress model, in which he posited a dynamic relationship between biological factors and environmental factors. In his view, more environmental stressors will be needed for an individual with less temperamental vulnerability to develop a full-blown BPD (Stone, 1977). Similar to Stone's approach, Linehan's (1993a) biosocial model conceptualizes BPD etiology as inherited biological vulnerabilities coupled with environmental invalidation factors.

Behavioral studies have for long shown evidence supporting diathesis stress model and biosocial model in interpreting the BPD development. For example, one case-control study

examined the effects of personality traits on negative affect during interpersonal interactions in BPD individuals. The results showed that increases in perceived inferiority and emotional insecurity (environmental triggers) were related to more negative affect, and this relationship was moderated by personality traits, thus supporting the application of a diathesis stress model in predicting negative affect among a BPD sample (Kopala-Sibley et al., 2012) .

A small number of genetic studies have also tested the gene-environment interactions, providing some nascent biological explanations in this area. One study particularly analyzed the moderating effects of a serotonin transporter gene on the association between trauma and impulsivity of BPD individuals. Their results showed that traumatic events (such as experience of war or childhood abuse) had an inverse relationship with impulsivity in SS and SL genotypes (SS genotype has two copies of short-form deletion "s" alleles; SL genotype has one copy of "s" allele and one copy of long-form insertion allele)(Wagner et al., 2009). Another study reported that a history of abuse could be associated with a higher probability of BPD diagnosis among individuals with TPH1 polymorphism (risk-allele), thus accounting for the genetic mechanism by which trauma might impact the development of BPD (Wilson et al., 2012).

Taken together, many conceptualizations of BPD etiological factors exist. Earlier BPD researchers have more often investigated psychosocial factors primarily through cross-sectional survey designs. In recent years, the advancement of neurobiology contributes to a rapid growth in genetic and brain imaging studies. Despite limitations in methodologies (such as sample biases, measurement limitations, lack of causal inference methods, and unclear interpretation of mechanisms), together these studies shed light on a dynamic web of interrelated factors contributing to the development of BPD. Future research needs to build on extant findings in order to make further improvements, which will be elaborated and discussed in the later sections.

2.2 Prior Research in Relation to BPD Etiology

2.2.1 Methodologies of Past Research

Extant BPD etiology studies are predominantly cross-sectional survey studies, and a limited number of longitudinal studies exist. Causal inference methods were also very sparse when investigating certain etiological factors in accounting for the development of BPD. Chi square tests were most frequently used to compare frequency of multiple etiological factors among BPD and non-BPD groups. Finally, studies predominantly used simple correlations and multiple regression while claiming a significant effect of a certain factor (such as family environment or traumatic experience) on the development of BPD.

2.2.2 Focuses and Topics

Various etiological factors have been postulated and tested among the extant etiology literature. Recent studies have explored multiple etiologies of BPD, and the frequently reported factors include familial psychopathology, genetic factors, internalizing and externalizing psychopathology, and other environmental influences (such as trauma, familial control and parental overprotection). Examples of such studies are briefly discussed below:

Familial psychopathology. A group of researchers identified significant associations between both familial psychiatric disorders and sexual abuse with BPD development (Bandelow et al., 2005). Similarly, another group of researchers found a small magnitude of correlations between trauma and BPD criterion counts (Berenz et al., 2013). Given the small effect size, they postulated that BPD features might be better accounted for by familial factors other than trauma.

Environmental factors. Liotti and colleagues suggested that trauma exposure and losses suffered by the primary caretakers together increased the probability of developing BPD (Liotti,

Pasquini, & Cirrincione, 2000). Another study recruiting Japanese participants found that emotional abuse and neglect as well as parental overprotection were all significantly related to the BPD diagnosis (Machizawa-Summers, 2007). The study further suggested other factors, such as environmental and biological ones, might as well contribute to the development of BPD. Weaver and Clum (1993)'s work further stressed the effect of certain dimensions of trauma, such as duration and severity of sexual abuse. In particular, high familial control in conjunction with frequent sexual abuse was shown to be strongly associated with severity of BPD symptomatology (as measured by a dimensional BPD scale).

Comorbid Axis-I psychopathology and genetic factors. Afifi et al. (2011) utilized a national representative sample and their results suggested that Axis-I comorbidity might explain the identified significant associations between multiple childhood trauma events and most cluster A and B personality disorders. Another group of researchers found that trauma, BPD traits and externalizing (such as attention deficit hyperactivity disorder, conduct disorder and oppositional defiant disorder), and internalizing disorders (such as major depressive disorder, separation anxiety disorder and internalizing distress) were all correlated, but no causal relationship could be established (Bornovalova et al., 2013). In addition, their findings suggested that the relationship between trauma and BPD traits was better accounted for by genetic risk factors overlapping with externalizing and internalizing disorders. Likewise, Krabbendam and colleagues (2015) capitalized on a longitudinal design, and they reported that internalizing problems (for example, depression, dissociation, and other emotional problems) and externalizing problems (for example, bullying, fighting and vandalism) showed significant association with the development of BPD and comorbid BPD-ASPD (Krabbendam et al., 2015).

2.2.3 Limitations and Extant Resolutions

External validity. A large number of extant BPD etiological studies recruited predominately female participants, compromising generalizability to men with BPD. In addition to gender, certain age groups (such as adolescents and older adults) and ethnic minority groups were highly underrepresented across most BPD studies. The reasons for these disparities may be partially due to the different systems in which these underrepresented individuals may be found. For example, males with BPD are more likely to have comorbid substance abuse problems and may more frequently end up in the criminal justice system where their mental health needs are not identified or met (Zanarini et al., 1998)

Internal validity. Key threats to internal validity include selection bias, measurement issues, insufficient consideration of mediating/moderating factors, and lack of causal inference design. First, most studies recruited participants in outpatient or inpatient settings. Individuals who actively seek clinical treatment can very likely self-select into research studies due to similar reasons. Such selection bias might potentially lead to a larger effect size. Another threat to internal validity lies within the predominant reliance on retrospective self-report measures in assessing some etiological factors, as recall bias could potentially undermine the accuracy and completeness of information being retrieved. Furthermore, over-reporting of trauma among clinical populations can upwardly bias estimates and limit the variance in the sample, thereby influencing the power in identifying a causal relationship. In addition, some studies only examined the single pathway between one factor (such as sexual abuse) and BPD development, and overlooked the complex web of interrelated factors that may mediate the relationship (e.g. the genetic influence and its interrelationship with trauma).

As far as the research design, most current etiology studies do not utilize causal inference methods. One prominent problem concerns the biased regression analysis. Specifically, a majority

of the etiology studies are indeed correlational studies, and many relied on simple linear regression and logit regression to disentangle the effects of etiological factors (e.g., trauma) on BPD. Many studies did not sufficiently account for the influences of certain unobserved variables, e.g., genetic influences and comorbidity with major Axis-I disorders. In addition, the multicollinearity among concurrent psychiatric diagnoses were not sufficiently addressed. Taken together, causal claims about etiology cannot be established if merely reliant on the correlational evidence or biased regression estimation.

Attempts to resolve the above issues. A number of studies have attempted to enhance the research rigor in claiming causality by utilizing longitudinal or causal designs (such as co-twin and biometric modeling). As discussed in the previous sections, several twin studies have been conducted to isolate the causal effect of genetics aside from environmental influences. For example, one study capitalized on a discordant twin design and biometric modeling to identify how much causal effect trauma might have on BPD (Bornovalova et al., 2013). In particular, they examined the variation of monozygotic (MZ) and dizygotic (DZ) twin pairs in their BPD features by including a list of covariates: Shared environmental factors (effects that are theorized to be the same for both MZ and DZ twins), non-shared environmental variables, and genetic influences (shared 100% by MZ twins and 50% by DZ twins). They then disentangled the causal effect by comparing the BPD features between MZ and DZ twin pairs who differ on trauma experience (within-pair effect).

The authors also used biometric modeling in their design, a special type of structural equation modeling which incorporates two types of variables: Four manifest variables (trauma, BPD, and the internalizing and externalizing psychopathology, INX and ETX), and three latent variables (shared environmental influences, non-shared environments, and genetic factors)

(Bornovalova et al., 2013). Consistent with discordant twin analyses, the results from biometric modeling identified moderate to large genetic effects, small effects for non-shared environment factors (but negative effects for sexual abuse), and very small interaction effects.

Efforts of including the understudied populations. There have been a few attempts to include past understudied subpopulations. For instance, one study examined trauma exposure and BPD among a group of low-income, minority primary care patients (Westphal et al., 2013), and another study examined the patterns of trauma and its correlation with BPD among a Japanese sample (Machizawa‐Summers, 2007).

Overall, general statistical analyses and research designs as discussed above can be applied to understudied and underserved subgroups. However, additional covariates should be considered in building specific statistical models for each subpopulation. For example, socio-economic-status variables may show different roles or effects for low-income groups versus other populations. Another example is the inclusion of particular cultural norms and values (such as parental over-protection for Asian communities) as a primary factor when studying underserved ethnic minorities.

2.3 Past Interventions

2.3.1 Psychotherapy Treatment

A number of psychotherapies have been developed to treat BPD with proven efficacy in decreasing BPD symptoms (such as suicidality, self-harm, and use of hospitals, emergency rooms, substances and medications). Nevertheless, a recent meta-analysis showed only modest benefits of BPD psychotherapies over controlled treatment condition in terms of reducing BPD or BPD-relevant outcomes (Cristea et al., 2017). Examples of these therapies include schema-focused

cognitive therapy (SFT) (Cousineau & Young, 1997), dialectical behavioral therapy (DBT) (Linehan, 1993a, 1993b), transference focused psychotherapy (Clarkin et al., 2007), structural analysis of social behavior (Benjamin, 1996), mentalization based therapy (MBT) (Bateman & Fonagy, 2009), good psychiatric management (Gunderson et al., 2018), and Systems Training for Emotional Predictability and Problem Solving (STEPPS) (Blum et al., 2008). In the following paragraphs, we present basic concepts and techniques of six primary treatment programs (DBT, SFT, MBT, TFP, GPM and STEPPS), their reported empirical evidence, and a critical analysis in reference to the social science theories.

DBT. Marsha Linehan developed DBT initially to treat chronically suicidal individuals, and later she modified it into a cognitive behavioral treatment for BPD (Linehan, 1993a). DBT was developed partially on the biosocial theoretical assumption that BPD individuals lack the ability to regulate emotions in various contexts. Furthermore, invalidating personal and environmental factors contribute to the lack of functional coping strategies, and therefore reinforce dysfunctional behaviors (Panos et al., 2014).

In general, DBT targets the following areas: (1) interpersonal chaos, (2) labile emotions, (3) impulsiveness, and (4) confusion about self/cognitive dysregulation. It also aims to decrease maladaptive behaviors including life-threatening acts, therapy-interfering behaviors, and qualify-of-life-interfering behaviors (Linehan, 1993a). From a recent meta-analysis assessing the efficacy of DBT, Panos and colleagues identified five randomized controlled trials in a systematic search, and found that DBT demonstrated efficacy in stabilizing and controlling self-destructive behavior and improving patient compliance; and yet, DBT was found only marginally better than treatment as usual in reducing attrition during treatment (Panos et al., 2014). Despite the findings, the data was insufficient to indicate that DBT is successful in long-term treatment. Additionally, a recent

meta-analysis indicated a small effect size of BPD psychotherapies, including DBT, relative to controlled treatment condition (treatment as usual) in terms of reducing BPD-relevant outcomes (Cristea et al., 2017).

SFT. Fundamentally, SFT assumes that unmet childhood needs (such as love, attention, acceptance, safety, and more), usually because of inadequate parenting, lead to maladaptive modes, hence accounting for BPD pathology. The concept of modes represents the patient as child, parent, and healthy adult. Different modes reflect underlying coping styles of BPD individuals, which can include abandoned/abused child, angry/impulsive child, detached protector, punitive parent and healthy adult. Incorporating both cognitive and experiential work (such as gestalt techniques, e.g., "empty chair"), SFT proposes four mechanisms of change in the therapy: (1) limited reparenting, (2) experiential imagery and dialogue work, (3) cognitive restructuring and education, and (4) behavioral pattern breaking, which are to be carried out in three phases (1) bonding and emotional regulation, (2) schema mode change, and (3) development of autonomy (Kellogg & Young, 2006).

In terms of treatment efficacy, a group of researchers conducted a randomized controlled trial to evaluate the effectiveness of adding an eight-month SFT group to treatment-as-usual (TAU) individual psychotherapy for BPD patients (Cristea et al., 2017). In comparison to TAU, SFT group patients showed significant improvements in terms of reduced BPD-related symptoms, reduced psychiatric symptoms severity, and improved global functioning; further, SFT participants also had significantly lower drop-out rates relative to TAU patients.

MBT. As a form of social cognition, mentalization refers to the mental capability to understand others' behaviors in terms of various mental states such as feelings, needs and desires. As purported by MBT, this mental capability was greatly influenced by early attachments and later

mirroring relationships. Therefore, disturbed attachments or trauma may contribute to a mentalization failure or loss that is linked with an incoherent sense of self, hence accounting for core features of BPD (Fonagy & Luyten, 2009). A primary goal of MBT is to make sense of the interconnectedness among the core features of BPD at various levels, and it suggests therapeutic interventions to help patients mentalize in the context of attachment relationships (Fonagy & Luyten, 2009). According to a mentalization-based developmental model of BPD, childhood adversities (disruptions in attachment) in the caring environment lead to early emotional difficulties, which in turn develop into more chronic impairments when coupled with other traumatic experiences.

In terms of intervention techniques, MBT takes the form of both group and individual therapy, with a primary goal of addressing mentalizing problems, and it also includes therapeutic steps such as demonstration of empathy, exploration, clarification, identification of affect status, and mentalization of the relationship (Bateman & Fonagy, 2013). Fonagy and colleagues (2008, 2009) conducted two randomized trials and reported significant treatment effect of MBT as compared with treatment as usual and structured clinical management. Specifically, in the first study, patients showed significant improvement in areas of suicidality, diagnosis status, service use, medication use, global functioning and vocational status as compared with TAU at five-year follow-up. When comparing with structured clinical management in the second study, both treatment groups demonstrated significant improvements in outcomes such as self-report symptoms, suicidality, hospitalizations and more; however, MBT group showed quicker and larger decrease in self-report symptoms and crisis situations (Bateman & Fonagy, 2008, 2009)

TFP. Standing for transference-focused psychotherapy, TFP is a form of structured psychodynamic therapy that is developed from the theoretical perspective of object relations. A

fundamental assumption underpinning TFP is that people with BPD suffer from severe identity diffusion, and hence lack an integrated sense of self and others which is primarily due to negative emotions (Kernberg et al., 2008). Consistent with traditional object relationship therapies, the main strategies utilized in TFP include (re)activation of BPD individuals' split-off object relations through observation and interpretations in the transference process (the therapeutic redirection of expressed feelings and emotions). Specifically, to address the split representations of self and others, TFP utilizes several key techniques including treatment contracting, reflection, clarification, confrontation, and interpretation (Kernberg et al., 2008).

Evaluating the efficacy of TFP, Clarkin and colleagues (2001) compared the clinical conditions of patients prior to and after one year of treatment. They reported a significant reduction in suicidality, and half of the patients no longer met criteria for the BPD diagnosis (Clarkin et al., 2001). In another randomized controlled trial that compared three treatments (DBT, TFT and Supportive Treatment-ST), the results showed that TFP predicted significant improvements in 10 out of the 12 measured outcomes (such as suicidality, impulsivity, anger, etc.), whereas DBT only resulted in improvements in 5 of 12 outcomes, and ST resulted in improvements in 6 of 12 outcomes. Additionally, both TFP and DBT showed efficacy in reducing suicidality, but only TFP predicted significant improvement in impulsivity (Clarkin et al., 2007).

GPM. Good Psychiatric Management (GPM) is a type of generalist treatment that combines case management and psychotherapy (utilizing both psychodynamic and cognitive-behavioral strategies) (Gunderson et al., 2018). GPM considers BPD as a disorder of interpersonal hypersensitivity, which proposes that BPD symptoms are exhibited in interpersonal contexts (Gunderson et al., 2018). Despite genetic deficits, BPD patients are believed to have capabilities to overcome the interpersonal stress and have long-term goals for satisfactory lives. Based on the

patients' emotional reactions to the interpersonal stressors, clinicians support the patients as collaborative partners and encourage them to better cope with the stressors. GPM is different from other therapies in that it not only improves the BPD patients' views of themselves but also improves social adaptations skills, which in turn promotes collaborative therapeutic relationships and interpersonal relationships. In terms of its efficacy, one randomized controlled study compared GPM to DBT after one-year treatment as well as at two-year follow-up. Their results evidenced similar efficacy of GPM and DBT in a plethora of areas, such as suicidal behavior, self-harm, interpersonal relationships, general psychopathology, and more (McMain et al., 2009; McMain et al., 2012).

STEPPS. Systems Training for Emotional Predictability and Problem Solving (STEPPS) is a twenty-week, manual-based psychoeducational group treatment, usually prescribed as a supplement to regular treatment for BPD (Blum et al., 2008). STEPPS combines several cognitive behavioral components and skill training sessions, but it does not provide individual therapies. Additionally, utilizing a systems-based model, STEPPS engages a variety of support networks (such as families and health care providers) in the treatment for the patients through psychoeducation. Specifically, STEPPS (Blum et al., 2008) first educates patients about the disorder, with the goal of replacing misconceptions with an awareness of emotions, thoughts and behaviors in relation to BPD. Second, trainers teach emotion management skills, including "distancing, communicating, challenging, distracting and problem management" (p.469). Third, trainers teach several skills to manage behaviors, such as "goal setting, healthy eating behaviors, sleep hygiene, regular exercise" and more (p.469). Sessions take the form of a seminar, each lasting for two hours. Meanwhile, a two-hour evening psychoeducation session is also held for family members and significant others.

As far as the reported efficacy, one randomized controlled trial was conducted to investigate the effectiveness of adding 20-week STEPPS to TAU among a group of BPD outpatients (Blum et al., 2008). STEPPS demonstrated significant beneficial effects in terms of improving impulsivity, negative affectivity, mood, and global functioning; in addition, STEPPS participants showed significant improvements in multiple items measured by Zanarini Rating Scale for BPD; however, there was no significant difference in areas of self-harm, suicide attempts and hospitalization. Finally, most reported gains were also maintained during the one-year follow-up.

2.3.2 Pharmacotherapy

As psychotherapy is generally considered as main treatment for BPD, appropriate medication may be beneficial in terms of stabilizing certain symptoms especially under crisis situations. Past studies investigating the efficacy of a combination of pharmacotherapy and psychotherapy have shown some results supporting utilizing medication in conjunction with traditional psychotherapy such as DBT (Nelson et al., 2014). For example, Linehan and colleagues (2008) investigated the efficacy of using olanzapine and DBT in treating BPD individuals with high irritability and anger by comparing individuals treated with DBT only to those treated with DBT and medication. Their results indicated more rapid decreases in symptoms of irritability and aggression for people treated with both DBT and olanzapine. A recent systematic review compiled results from 27 randomized controlled trials, reporting significant effects with mood stabilizers and second-generation antipsychotics in treating core BPD symptoms, yet no evidence was shown in decreasing overall BPD severity (Lieb et al., 2010). From extant findings, it appears that medication treatment shows efficacy while targeting specific symptoms and under crisis, and it should be used as a supplement to primary psychosocial treatments when needed.

2.3.3 Interventions Targeting Underserved Populations

As aforementioned, there are gender and racial/ethnic biases in BPD etiological studies. The reasons for these disparities may be partially due to the different systems in which these underrepresented individuals may be found, for example, males with BPD are more likely to have comorbid substance abuse problems and may more frequently end up in the criminal justice system where their mental health needs are not identified or met (Zanarini et al., 1998). Other sources of disparities may come from implicit biases related to gender stereotypes and racial/ethnic stereotypes. Borderline personality disorder is often assumed to primarily affect White females and, thus, little attention has been paid as to how the disorder manifests and what differences may exist with persons of color with BPD or men with BPD.

Indeed, these aforementioned disparities might potentially lead to inaccurate diagnoses and delayed or inappropriate treatment for understudied BPD subgroups. Further, it is highly beneficial to supplement regular therapy with a systems-based, culturally competent psychoeducation component. In that sense, STEPPS seems to be an effective and even necessary supplement to regular treatment. One advantage of STEPPS is that the program provides education and trainings to a reinforcement team that includes patients' families, friends and other professionals, all of whom serve as the support system. Another strength of this program is the flexible format (both face-to-face and teleconference) that requires relatively less intensive training of mental health professionals. In practice, Blum and Black have adapted the STEPPS program for correctional facilities and have also conducted trainings via teleconferences.

2.3.4 Synthesis

In comparison, the above psychotherapies can be differentiated from each other based on their underlying social science theories, fundamental assumptions of BPD etiology, and how each

treatment was proven to predict improvement in BPD related symptoms (See Table 2). In terms of BPD pathology, DBT, MBT, GPM and SFT assume a deficit-oriented origin (such as inherited vulnerabilities, emotion dysregulation, interpersonal hypersensitivity and mentalization failure), whereas TFT takes a neutral stance and instead focuses on internal conflict (Kernberg et al., 2008). Despite its similar deficit-oriented theoretical core (assumption of inherited hypersensitivity), GPM is considered a generalist treatment which combines case management and multiple psychotherapy strategies, which to a certain extent is strengths focused. Finally, despite only being an adjunct treatment, the systems-based approach of STEPPS is considered holistic and strengths-based.

Table 2

Comparison of Five DBT Treatment Programs

	Interventions				
	Dialectical Behavioral Therapy	**Mentalization-based Treatment**	**Transference-focused Therapy**	**Schema-focused Therapy**	**STEPPS**
Reflected Theories	Biosocial Eastern philosophy	Attachment Theory Expanded	Psychodynamic Object Relations	Psychodynamic Object Relations	Systems Social capital
Assumptions	BPD is mainly due to emotion dysregulation coupled with invalidating environmental factors.	BPD is a disorder due to mentalization failure /loss because of early attachment disruptions.	BPD is mainly due to an internal conflict (internalized split-off object relations).	BPD is mainly due to unmet childhood needs that are because of inadequate parenting.	N/A
Strengths	Efficacy in stabilizing and controlling self-destructive behavior and improving patient compliance.	Efficacy in outcomes such as self-report symptoms, suicidality, hospitalizations, etc. Quicker and larger decrease in self-report symptoms and crisis.	Proven efficacy in reducing suicidality and impulsivity; quicker and larger decreases in crises and self-reported symptoms.	Improvements in BPD related symptoms, psychiatric symptoms severity, and global functioning; lower drop-out rates.	It engages a variety of support networks (families and health care providers); adaptable to various BPD subpopulations; flexible formats (teleconference and face-to-face).
Weakness	Insufficient evidence supporting its long-term effectiveness. Need extended training for therapists and commitment from patients.	Difficult to apply during emotional arousal state. Limited RCT data. Need extended training for therapists and commitment from patients.	Limited RCT data. Need extended training for therapists and commitment from patients.	Limited RCT data. Need extended training for therapists and commitment from patients.	Supplementary treatment only and does not offer individual therapy sessions. Insufficient evidence of efficacy in decreasing suicide, self-harm and hospitalization.

As mentioned above, randomized controlled studies have indicated the efficacy of all six programs in reducing BPD relevant symptoms especially during crises, such as suicidality, hospitalization, self-harm and so on. These programs seem to share several features that can account for their proven effectiveness, which happens to be consistent with that proposed by Bateman and Fonagy (2000): highly structured, long-term treatment, clearly focused, theoretically coherent, and integrated with other services.

Aside from the noted strengths, the current interventions are still to large extent symptom-reducing oriented. The reported effectiveness was mostly related to short-term crisis stabilization (such as reducing suicidality and hospitalization), and yet little was known about the long-term effect in improving social functioning and temperamental symptoms (such as chronic anger and negative emotions that tend to remit slowly). Nevertheless, the lack of evidence of long-term treatment effectiveness is possibly in part due to inadequate provision of long-term programs per se. Actually, one meta-analysis evaluating the effectiveness of long-term psychodynamic psychotherapy (LTPP) did indicate significantly larger effects of LTPP than short-term treatments in overall effectiveness, target symptomatology, and personality functioning (Leichsenring & Rabung, 2008), providing some evidence of long-term treatment although not specifically in the context of BPD.

In a more recent appraisal of available treatments for personality disorders, Bateman et al. (2015) indicated that despite the success of current interventions, people with severe personality disorders still pose a danger of harm to themselves and others, and they continue to feel miserable about life and have low social functioning. Overall, long-term psychotherapies are still lacking in the context of chronic mental illness and personality disorders. The above limitation could largely be caused by low insurance coverage in long-term treatment of personality disorders as opposed

to more financial incentives in addressing crisis situations. Another source could be a lack of valid measurement tools in terms of assessing and evaluating temperament and functioning, which should be adequately addressed in the future research.

2.4 Importance/Relevance to Social Work

Social work is a profession that intersects with multiple disciplines, including the provision of systemic health care and related social services to BPD individuals. Indeed, as the primary providers for people with all forms of severe mental illness, social workers encounter people with BPD and their families at almost all levels, from psychiatric emergency units to community and family services.

Current practice in the field of medicine utilizes a symptom-reducing oriented and gender/race biased approach. In comparison, carrying on a tradition of holistic practice, social workers can offer more effective and comprehensive assistance to BPD individuals, from individual system to family context, from local environment to society level, from biological correlates to social factors. This is especially meaningful in formulating non-biased, accurate, and culturally competent diagnostic tools for various BPD populations. As mentioned above, the way BPD symptomatology manifests itself differs across age, gender and racial groups due to variations embedded in the intertwined web of biology, psychology, culture and social environment. The way people seek treatment also differs across gender and racial groups. One study shows distinct gender difference in treatment utilizations: Men are more likely to utilize substance use rehabilitation programs and are less likely to use pharmacotherapy and psychotherapy services than women with BPD (Fishman, 2014). All of the above discrepancies require a multifaceted diagnostic tool that challenges the current symptom-reducing oriented and gender/race biased approach.

In addition, social work profession's core value of competence mandates all social workers to continuously advance their own expertise and skills in relation to their practical fields. Given the complexity and diversity of the problem, it is especially important to equip social workers with appropriate knowledge and skills of effective treatment approaches while working with BPD individuals. For example, toward the aim of developing more effective diagnosis tools and interventions for a variety of BPD subpopulations, a solid knowledge base of different manifestations of BPD as well as underlying mechanisms needs be built. Further, a comprehensive and dynamic assessment requires all practitioners to go beyond clinical symptoms and obtain full trajectories of patients' psychosocial functioning over time. While meeting this core value, this study will contribute to our current knowledge on BPD etiology, as well as generate meaningful implications for prevention and intervention strategies.

As the agent of social change, social workers tend to address various social problems and challenge social injustice by drawing on their expertise, values and skills (Assembly, 2008). Consistent with this value, social workers are ethically responsible to address the difficulty of treating and managing BPD populations. Given its adverse psychosocial impacts (such as functional instabilities to families and societies), the treatment and management of BPD patients have always been challenges for the health care sectors as well as other social services agencies. Managing BPD remains difficult even with the availability of a wide array of evidence-based psychosocial programs (from psychodynamic therapies, to cognitive behavioral therapies, to psychoeducation programs), and the advancement of medication treatment, as evidenced by the high rate of 20% inpatient admission and outpatient referrals (Bateman et al., 2015). In addition, psychosocial treatments generally require intensive training of mental health professionals and

support staff persons, which further adds to the challenges to current health care and social service systems in both tangible and intangible forms.

Finally, long-term, integrated treatments are currently sparse, which requires social workers' advocacy for macro-level policy changes. A few examples of changes are highly needed, including but are not limited to: (1) to incorporate trauma assessment/screening/treatment into traditional BPD psychotherapies (such as DBT and MBT), given its high correlation with BPD symtopms; (2) to integrate BPD assessment/screening with primary care; (3) to offer specialized, community-based services to BPD individuals, including case management, counseling, and crisis hotline; and (4) to allocate NIMH research grants in particular to fund research focusing on previously underrepresented populations such as ethnic and racial minority groups, adolescents, and males. Overall, efforts from social workers at macro-levels will be highly critical in order to achieve accurate early assessment, to reduce the utilization rate of intensive treatment services, to achieve economically efficient use of health care system among this population, to equitably distribute services among minority groups and male patients who are often under diagnosed and served, and to increase equal insurance coverage for long-term BPD treatment (especially temperamental symptoms) from a broader goal of eliminating stigma associated with personality disorder.

3.0 Methodology

The following sections depict key components of methods employed, including type of the study, sample and data, measures (and key information on the reliability and validity), and detailed descriptions of data analyses and justifications.

3.1 Overview of the Study

As synthesized in our review of past literature, significant correlations between trauma and BPD have been consistently noted across a myriad of BPD studies; further, multiple mechanisms were proposed and yet have not been thoroughly understood or rigorously examined, such as attachment issues, dissociation and emotion dysregulation (Afifi et al., 2011; Battle et al., 2004; Goldman, D'Angelo, DeMaso, & Mezzacappa, 1992; Tyrka et al., 2009; Westphal et al., 2013).

In addition, ED has remained on the research forefront targeting the intersection of trauma and BPD, in which an elevated sensitivity to several negative affects was specifically noted, such as shame, guilt and anger (Carpenter & Trull, 2013; Cheavens et al., 2005; Gratz et al., 2008). As also aforementioned, trauma, attachment and biosocial theories all have shed light on several aspects in the intertwined etiological web, as well as a dynamic interplay among various factors. Last but not least, we take into consideration a unique factor—developmental stage—in our study design. Specifically, our participants aged approximately between 18 to 24 at Wave 1, which is a special age group called emerging adulthood (from late adolescence to early adulthood according to Arnett's conceptualization,(Arnett, 2001)). This stage is typically characterized of unpredictable instabilities and heterogeneities in varied biopsychosocial areas. Taken all together, early childhood trauma might affect a certain group of individuals among whom higher sensitivity to negative affects is manifested in later years. This hypersensitivity intersects with the unpredictable shifts during emerging adulthood, in which further swift and unstable changes are theoretically suggested.

Informed by past theoretical frameworks and empirical studies, this study analyzes repeated measures data from a study of BPD symptomology in an at-risk community sample of young women, hypothesizing that ED (especially negative affect such as guilt, shame and anger) will be a mediating factor in the link between trauma and BPD symptoms.

3.1.1 Research Questions and Aims

My research questions concern three main aspects: 1) Are there significant associations among early childhood trauma, BPD features, and ED/NA? 2) What role does ED/NA play in the association between trauma and BPD? 3) Is trauma associated with changes in BPD features and/or ED (NA) features over time?

Answering the above questions, this study will be focused on examining the potential association between trauma and risks for developing BPD, specifically to the following aims: Examine the association patterns among trauma, ED, and BPD symptoms by (1) testing the relationships among all variables at baseline as well as investigating the directional relationships between ED and BPD (controlling for trauma history) across time points (Figure 2), (2) examining the trajectory of BPD features and ED problems in the context of trauma history (Figure 3), and (3) investigating the mechanisms through which trauma plays a role in predicting BPD features (Figure 4).

Aim 1: Examine the associations among trauma, ED, and BPD symptoms by (1) testing the relationships among all variables at baseline as well as (2) investigating the directional relationships between ED and BPD (controlling for trauma history) across time points.

Hypothesis 1a: Both trauma and ED will significantly predict higher BPD features at the baseline.

Hypothesis 1b: Repeated measures of participants' negative affect (shame/guilt and anger) will predict participants' BPD symptoms across time points (Figure 2).

Aim 2: Examining the trajectory of BPD features and ED problems in the context of trauma history by testing the changes between waves.

Hypothesis 2: Trauma will predict both higher mean BPD scores at baseline, and steeper changes in BPD symptoms over time; similarly, trauma will predict both higher mean negative affect scores at baseline, and steeper changes in negative affect over time (Figure 3).

Aim 3: Investigate the mechanisms through which trauma plays a role in predicting BPD features (Figure 4).

Hypothesis 3a: ED will partially mediate the path from trauma to BPD features at baseline.

Hypothesis 3b: Controlling for ED, trauma still predicts higher BPD features at baseline.

3.2 Study Design

This study analyzes data collected from a substudy of young women who were recruited from a larger community-based longitudinal study (Pittsburgh Girls Study [PGS]; R01 MH56630; PI: Rolf Loeber). The following sections describe in detail the original population from which the subsample was drawn, as well as the procedures in which the substudy data were collected in terms of recruitment and assessments.

3.2.1 Data and Procedures

As aforementioned, participants were recruited from the ongoing PGS for a substudy on young women's personality features, impulsive aggression, and self-harm (Women's Emotions and Relationships Study [WERS]; K01 MH101289 and American Foundation for Suicide Prevention [AFSP] YIG-0-131-14; PI: Lori Scott). The larger PGS involves an urban community sample of 2,450 women who were initially recruited in 1999 and 2000 when they were ages 5 to 8 years old. PGS participants were identified by oversampling from neighborhoods in which at least 25% of families were living at or below poverty level (see Keenan et al., 2010 for further details on PGS recruitment and study design) .

Participants from the larger PGS over the age of 18 were identified for the WERS substudy based on self-reports of recent aggressive behavior, suicidality, or self-injury. Screening procedures are further described below (see Scott et al. (2017, 2019) for additional details). A total of 166 women were recruited and consented to participate in the study.

During initial WERS assessments (Wave 1), participants completed a battery of clinical interviews and self-report measures (See measures section for detailed information on all the measures for the current study). Follow-up assessments (Waves 2 and 3) occurred 6- and 12-months, respectively, after the initial assessment. At each follow-up, participants completed a variety of clinical interviews and self-report measures.

Participants ranged in age from 18 to 25 ($M = 21.51$, $SD = 1.50$). Consistent with the diverse demographics of the PGS, 72.2% of participants self-identified as racial or ethnic minority (70.1% African American; 2.1% multi-racial) and about 36% did not work either due to disability, being housemaker or other unspecified reasons.

3.2.2 Participants for this Study

This proposed research study will analyze the WERS sample as described above. Given our particular focus on trauma, only those WERS participants who completed baseline the Childhood Trauma Questionnaire (CTQ; (Bernstein et al., 2003)) will be included for the primary analyses (N=144, *Mean* (CTQ)=46.25, *SD* (CTQ)=16.32). It is important to note that trauma measure was only administered during Wave 1 based on the rationale that childhood trauma experiences happened in the past and were not expected to change during the follow-up year. Like most longitudinal studies, this sample suffered from missingness as some participants partially completed the follow-up assessments, which will be addressed and further discussed in the data analyses section as follows.

3.3 Variables and Measures

The following sections describe variables of interests as well as relevant covariates (a list of all variables and measures are summarized in Table 4). In addition, the literature support for the measures will also be discussed in the following sections. Figures are attached following the texts to give a visual demonstration of all models (see Figure 2-4).

3.3.1 Independent Variables and Measures

Trauma and ED. The primary independent variables in the multivariate linear models (baseline models) will be trauma and ED, which are measured by Childhood Trauma Questionnaire Short Form (D. P. Bernstein et al., 2003), and Difficulties in Emotion Regulation Scale, respectively (only used at baseline models; repeated measures such as Anger/Guilt/Shame are described as follows) (Gratz & Roemer, 2004).

Trauma. The Childhood Trauma Questionnaire Short Version (CTQ-SF; (D. P. Bernstein et al., 2003)) items ask about experiences from early childhood to adolescence, which are rated on a 5-point scale with response options ranging from Never True to Very Often True. The CTQ-SF has five clinical scales—physical, sexual, and emotional abuse, and physical and emotional neglect, which are defined as follows: (1) Sexual abuse is defined as "sexual contact or conduct between a child younger than 18 years of age and an adult or older person." (2) Physical abuse is defined as, "bodily assaults on a child by an adult or older person that posed a risk of or resulted in injury." (3) Emotional abuse is defined as, "verbal assaults on a child's sense of worth or well-being or any humiliating or demeaning behavior directed toward a child by an adult or older person." (4) Physical neglect is defined as, "the failure of caretakers to provide for a child's basic physical needs, including food, shelter, clothing, safety, and health care." (5) Emotional neglect is

defined as, “the failure of caretakers to meet children’s basic emotional and psychological needs, including love, belonging, nurturance, and support.” Research findings have supported the validity and reliability of CTQ-SF across clinical and community populations (D. Bernstein & Fink, 1998; D. P. Bernstein et al., 2003). In a study conducted among a combination of clinical and community samples (*N*=1978), Bernstein and colleagues reported that the CTQ-SF’s items held essentially the same meaning across all four samples. Moreover, the scale demonstrated good criterion validity in a subsample of adolescents when compared to the independent ratings of four types of childhood trauma obtained from the Child Maltreatment Ascertainment Interview.

ED. Difficulties in Emotion Regulation Scale (DERS) DERS is a 36-item self-report measure, developed to assess emotion dysregulation comprehensively, including items that reflect difficulties in six emotional dimensions: Non-acceptance, Goals, Impulse, Strategies and Clarity (Gratz & Roemer, 2004). More specifically, Non-acceptance means non-accepting reactions to negative emotions or stress; the Goals dimension contains items reflecting difficulties in engaging in goal-directed behaviors (such as concentrating or accomplishing tasks); the Impulse dimension consists of items that describe difficulties with controlling behaviors under negative emotions; the Awareness (reverse-coded) means the abilities to attend to and recognized emotions; the Strategies dimension includes items that evaluate limited access to regulation strategies; and Clarity measures the level of one lacks clarity of own emotions. Each item of DERS is rated on a 5-point scale according to the frequency of which participants believe each item applies to their situation (1 denotes “almost never” to 5 indicates “almost always”). Reportedly, the authors indicated good internal consistency for both the DERS total scale (α=0.94), and its subscales (α ranges from .80 to .91).

Negative Affect (NA). Repeated measures of shame/guilt and anger will be treated as the main predictors in the cross-lagged structural equation model (Figure 2). Negative affects "guilt and shame" are measured by the Guilt and Shame Proneness scale (GASP; Cohen, Wolf, Panter, Insko, & psychology, 2011), whereas "Anger" is measured by State Trait Anger Expression Inventory 2 (STAXI-2; Spielberger, 1999).

Shame/Guilt. The GASP is a 16-item self-report scale that assesses individuals' tendencies to experience shame and guilt (not just presence of those emotions) following embarrassing or offensive events across different settings (Cohen et al., 2011). GASP consists of two shame subscales (negative behavior-evaluations and repair action tendencies) and two guilt subscales (negative self-evaluations and withdrawal action tendencies). For the two guilt subscales, negative behavior-evaluations items address bad feelings about one's action, whereas repair items describe behavioral intentions such as correcting one's mistakes (e.g., "you would try to act more considerately toward your friends"). As far as the shame subscales, negative self-evaluations consist of items about feeling bad about oneself, whereas withdrawal items address tendencies to hide from the public (e.g., "you would avoid the guests until they leave"). Each item of the GASP is rated on a 7-point scale, with "1" indicating "very unlikely" and "7" indicating "very likely", based on the proneness of having shame or guilt emotions (which participants believe they will have) in response to scenarios such as making mistakes at the workplace or committing small crimes. Cohen et al. (2011) reported good construct validity across all the subscales when comparing with other measures, as well as moderate to high reliability of the subscales (α ranges from .61 to .71).

Anger. The original STAXI-2 is 57-item self-report measure which comprises of six subscales: State Anger, Trait Anger, Anger Expression-In, Anger Expression-Out, Anger Control-

In, and Anger Control-Out (Spielberger, 1999). We utilized an abbreviated anger scale that included only Trait Anger, Anger Expression-In, Anger Expression-Out, and Anger Control (we used mean scores of both Control-in and out scores, which were also reverse coded). In terms of each subscale, Trait Anger measures the disposition to experience anger with or without provocation; Anger Expression-In assesses the frequency of controlling one's angry feelings; Anger Expression-Out measures how often one takes actions upon his/her anger; and Anger Control measures one's ability to control one's anger by utilizing positive outlets (Control-out) or calming oneself down (Control-in). Past studies reported overall high reliability (α above .70 for nonclinical population and above .80 for clinical populations), and good validity of original STAXI-2 among clinical and non-clinical populations (Lievaart, Franken, & Hovens, 2016). The internal consistency of our anger scale among this current sample will be elaborated in results section.

3.3.2 Dependent Variables and Measures

BPD Symptomatology. The primary dependent variable in all models is BPD features. In cross-lagged models and latent growth curve models, dimensional scores from the full-length Semi-structured Interview for DSM-IV Personality (SIDP-IV; (Pfohl, Blum, & Zimmerman, 1997)) were used. In the mediation analysis with latent construct, scores from the self-report scale of Personality Assessment Inventory-Borderline Features (PAI-BOR; (Morey, 1991))were used to account for four symptomatic subconstructs.

BPD Diagnosis. The Semi-structured Interview for DSM-IV Personality (SIDP-IV; (Pfohl, Blum, & Zimmerman, 1997)) was used to generate BPD scores for the cross-legged models and latent growth curve models (Figure 2 and 3). As a semi-structured interview, SIDP-IV was designed to assess the diagnostic criteria for the 10 personality disorders (PD) listed in DSM-IV.

Questions are organized based on themes such as work style, interpersonal relationships, interest, activities and more, as opposed to being differentiated by disorders. Each item is rated on a scale from 0 to 3. Dimensional scores (a sum of all BPD items scores) were used as an index of BPD symptomatology severity .

Self-Report BPD Features. The Personality Assessment Inventory-Borderline Features Scale (PAI-BOR, (Morey, 1991)) dimensionally measures four categories of symptoms underlying BPD: affective instability, identity problems, negative emotions, and self-harm . A later study that replicated Morey's method and administered the PAI-BOR among a chronic-pain population reported good internal consistency reliability of this scale (Mean Cronbach alpha=.79). The authors further validated the factor structure of the PAI-BOR using the same sample (Karlin et al., 2005). The PAI-BOR is used to measure subconstructs of BPD in our mediation analysis model (Figure 4).

Negative Affect (NA). In the latent growth curve model (Figure 3), repeated measures of Shame/guilt and Anger were used as the outcome variables, which again were measured by GASP (Cohen et al., 2011) and the STAXI (Spielberger et al., 1999), respectively.

3.3.3 Mediator and Measures

ED. As described in the previous sections, the latent construct of ED of baseline mediation models was measured via the Difficulties in Emotion Regulation Scale (Gratz & Roemer, 2004). Again, the DERS measures six emotional subconstructs (manifest variables) of ED (the latent factor), including non-acceptance, goals, impulse, awareness, strategies, and clarity.

3.3.4 Covariates and Measures

Demographic differences in our outcome variables were fully examined in order to select potential control variables. In particular, demographic differences in CTQ scores were assessed

using Pearson's Chi-Square and one-way ANOVA tests (or *t*-tests) for categorical and continuous variables respectively in R (R Core Team, 2017). In the cross-legged models, trauma history was treated as a control variable.

Table 3.

A Detailed list of All Measures

Variables	Measures	Description	Model
ED/NA	DERS	36-item self-report measure that assesses emotion dysregulation in six emotional dimensions: Non-acceptance, Goals, Impulse, Strategies and Clarity	Mediation analysis model
	STAXI-2	Trait Anger, Anger Expression-In, Anger Expression-Out, Anger Control.	Cross-lagged model and latent growth curve model
	GASP	16-item self-report measure with two shame subscales and two guilt subscales, which assesses individuals' tendencies to shame and guilt (not just presence of those emotions)	Cross-lagged model and latent growth curve model
BPD	SIDP-IV	Semi-structured interview that assesses the diagnostic criteria for the 10 PDs listed in DSM-IV. For each PD, criterion scores were summed, and these summed scores were used as an index of PDs.	Cross-lagged model and latent growth curve model
	PAI-BOR	Self-report scale that measures four symptomatic categories: affective instability, identity problems, negative emotions and self-harm	Mediation analysis model
Trauma	CTQ	Self-report scale that measures five types of trauma—physical, sexual, and emotional abuse, and physical and emotional neglect.	In all models

3.4 Data Analysis

3.4.1 Aim 1

To examine the general associations among trauma, BPD features and ED levels, multivariate linear models were conducted, controlling for a variety of demographic covariates. Mean scores of each subscale from CTQ-SF and DERS were entered as the primary predictors both simultaneously and stepwise. Means of each subscale (e.g., sexual abuse and physical abuse) as well as the mean of the full-length scales (e.g., the entire CTQ) were tested separately. Finally, performance of different models (model fit indices) were evaluated and interpreted.

To further verify the directional relationships between negative affect and BPD over time, a cross-lagged model was specified using structural equation modeling. Cross-lagged models can be used when (1) two variables are measured at the same point in time and across two or more time points, and (2) the interest is to learn the nature of their influences on each other. This type of model assumes causality while true experimental design is not available, which will be further explained using our model as follows (Anderson & Kida, 1982).

Two sets of negative affect were tested separately: (1) Shame and guilt, and (2) Anger. Figure 2 demonstrates that in each model, there are eleven correlations in total: Four cross-lagged correlations (NA_{t1} & BPD_{t1}, NA_{t2} & BPD_{t3}, BPD_{t1} & NA_{t2}, BPD_{t2} & NA_{t3}), four auto-correlations (NA_{t1} & NA_{t2}, NA_{t2} & NA_{t3}, BPD_{t1} & BPD_{t2}, BPD_{t2} & BPD_{t3}), and three synchronous correlations (NA_{t1} & BPD_{t1}, NA_{t2} & BPD_{t2}, NA_{t3} & BPD_{t3}). The correlations of interest in causal analysis are the cross-lagged ones. The basic rationale here is that if the correlation NA_{t1} & BPD_{t1} is stronger than BPD_{t1} & NA_{t2} and if NA_{t2} & BPD_{t3} is stronger than BPD_{t2} & NA_{t3}, then we could infer that NA is a stronger cause of BPD; however, if these cross-lagged correlations are equal, we then conclude that they do not cause each other (there might be a third variable causing NA and BPD).

Two assumptions have to be met: (1) NA and BPD have to be measured at the same point over time, and (2) the structural equation of our model stays unchanged between measurement time points (in other words, no additional independent variables would be added to the model as time goes by) (Anderson & Kida, 1982).

3.4.2 Aim 2

Latent growth curve structural equation models were specified to examine the prediction of NA and BPD from trauma using repeated measures of NA and BPD (Figure 2). Structural equation modeling is a form of multivariate analysis used to evaluate "arguably" causal models by examining the relationships between a dependent variable and two or more independent variables (McDonald & Ho, 2002).

3.4.3 Aim 3

This research also investigates the potential mediating channels from a history of trauma to higher BPD features through ED (measured via DERS) at baseline. In the mediation analysis (with latent constructs), the structural model comprises the latent predictor **Trauma**, the latent outcome variable **BPD Symptomatology,** and the mediator **ED**. The measurement model is specified as follows: the latent construct **Trauma** is measured by five trauma subtypes (physical abuse, sexual abuse, emotional abuse, physical neglect and emotional neglect), **BPD** by four symptomatic categories (affective instability, identity problems, negative emotions and self-harm), and **ED** by six emotional subconstructs (non-acceptance, goals, impulse, awareness, strategies, and clarity) (see Figure 3 inserted before). The above mediation analysis with latent factors will be performed in R via structural equation modeling.

As aforementioned, structural equation modeling is a form of multivariate analysis used to evaluate "arguably" causal models by examining the relationships between a dependent variable

and two or more independent variables (McDonald & Ho, 2002). The effects of each independent variable on the dependent variable are shown both directly and indirectly through other independent variables. Sobel's (1982) test for mediation was used to examine whether the indirect effects between trauma and mediator, and mediator and BPD are significant. Since Sobel's (1982) test requires a sampling distribution of indirect effects be normal, the results could be potentially biased given the likely violation of normality. To address this possibility, a bootstrapping method was applied, which does not make any assumptions about the sampling distribution (Shrout & Bolger, 2002).

3.4.4 Missing Data Analysis

In longitudinal studies, each unit is usually measured at baseline and then repeatedly over time. Missing data is a common problem under such scenarios because many subjects are not available to be measured at all time points. For example, in this proposed research study some participants may have missed the second assessment wave or the third assessment wave, or missed both waves, although all of them have taken the baseline assessments. Such incomplete data presents a considerable modeling and statistical challenges.

According to Rubin (1976), there are three important mechanisms of missingness: (i) *missing completely at random* (MCAR) refers to the case when missingness is completely unrelated to the data; (ii) *missing at random* (MAR) refers to the case in which the missingness depends on the observed data but, given the observed data, it does not depend on the unobserved outcome data; (iii) *missing not at random* (MNAR) is the most general mechanism where missingness may depend on the unobserved data in addition to the observed data.

In general, the MCAR and MAR mechanisms are often referred to as being *ignorable*, because statistical inferences can proceed by analyzing the observed data only, either via

maximum likelihood estimation or multiple imputation approaches (Robins & Rotnitzky, 1995; Scharfstein, Rotnitzky, & Robins, 1999). This is made possible because there is no need to explicitly addressing a parametric model to account for the missing data when incorporating all available information. Consequently, the resulting parameter estimators usually are unbiased. For the ignorable cases, those methods are included in a variety of statistical software such as R (lme and nlme).

Although the ignorable mechanisms (MCAR and MAR) are often reasonable and can be handled in a relatively straightforward manner, there are many important situations where the MAR assumption (missingness does not depend on the unobserved outcome data given the observed data) is unlikely to hold. For example, in our proposed study, it is reasonable to believe that women with more severe BPD Symptomatology are more likely to miss one of the following assessment waves due to more difficulties with balancing daily activities and managing mental health. If so, the outcome variables (BPD Symptomatology score) will be closely related to their own missingness. Due to this complicated missingness mechanism, MNAR is also referred as being *nonignorable.* Unfortunately, for the nonignorable case, those ignorable-based methods are subject to bias and often lead to false conclusions.

Handling nonignorable missing data requires a statistical model that includes all parameters of the joint distribution of outcome variables and the corresponding missing-data indicator (the missingness). More specifically, this means that the statistical analysis must incorporate a sub-model that characterizes the missing-data indicator (e.g., a logistic regression that predicts if the outcome is observed or not). This specification can be further classified into two major types of models: *selection model* and *pattern-mixture model* (Little & Rubin, 2014).

Based on the above methods proposed by Little and Rubin (2014), the missingness of outcome variable Y can be coined as an indicator random variable R denoting if Y is observed or not, i.e., $R = 1$ if Y is observed and 0 otherwise. Then the outcome Y and the missing-data indicator R have a joint distribution given covariates X, $P(Y, R \mid X)$. In the *selection modeling approach*, the joint distribution has the following factorization: $\mathrm{P}(Y, R \mid X) = P(Y \mid X) \times P(R \mid Y, X)$. This factorization implies a two-stage model in which the marginal distribution $P(Y \mid X)$ corresponds to the main analysis model as if all data is complete (e.g., a growth model) and the conditional distribution $P(R \mid Y, X)$ typically refers to a regression model that uses the outcome Y to predict the probability of missing data (Little & Rubin, 2014).

Usually, the selection model relies on some strict distributional assumptions such as multivariate normality due to the difficulty of modeling $P(R \mid Y, X)$ since Y is missing whenever $R = 1$. In contrast, the *pattern-mixture modeling approach* factorizes the joint distribution as $\mathrm{P}(Y, R \mid X) = P(Y \mid R, X) \times P(R \mid X)$ (Hogan & Laird, 1997). The preceding factorization also implies a two-stage model. However, it first provides a marginal distribution of R, $P(R \mid X)$ describing the incidence of different missing data patterns, and then provides a conditional distribution $P(Y \mid R, X)$ representing the main analysis model (Little & Rubin, 2014). Such a factorization conveys the strategy of stratification: we first stratify the sample into subgroups according to their missing data pattern, and then estimate the parameters in the main model (e.g., a growth model) separately within each pattern. For our proposed research study, there are four missing data patterns: (1, 1), (0, 0), (1, 0) and (0, 1), where the first indicator and second indicator denote the missingness of the 2nd and 3rd assessment waves, noting that all women attended the first baseline assessment.

For example, our proposed Aim2 applies a growth curve model to this missing data. We consider the most general MNAR mechanism as we discussed above and consider both *selection modeling approach* and *pattern-mixture modeling approach.* Both models describe the same joint distributions of the outcome variables and their missing-data indicators. However, two frameworks require different distributional assumptions and likely will produce different estimates of the growth curve model (Diggle & Kenward, 1994; Hedeker & Gibbons, 1997; Wu & Carroll, 1988). Unfortunately, those assumptions are usually nontestable and thus it is not easy to judge which framework is more accurate. In the end, as suggested by Enders (2011), we will apply a sensitivity analysis on two frameworks using the same data.

3.4.5 Sample Size Justification

In terms of the acceptable sample size for structural equation modeling, a review of SEM studies reported that the median sample size for testing an indirect effect was about 142.5, and about 40% of all the studies included less than 150 participants (Fritz & MacKinnon, 2007). Although typically a sample size over 200 is preferred to test SEM models, it appears that a size of 145 is above the median and within the acceptable range.

4.0 Results

This section depicts the analytical plans designed to investigate the proposed research questions: 1) Are there significant associations among early childhood trauma, BPD features, and ED/NA at baseline? 2) What role does ED/NA play in the association between trauma and BPD? 3) Is trauma associated with changes in BPD features and/or ED (NA) features overtime? First, this section presents the demographic characteristics of young women who participated in this study. Second, preliminary analyses were performed including internal consistency of all measures

and evaluation and selection of potential control variables; finally, results on each aim were presented respectively with supporting tables and figures.

4.1 Participants Characteristics

As aforementioned, 72.2% of participants identified as racial or ethnic minority. 74.3% of the participants self-identified as heterosexual, whereas 25.7% as non-heterosexual (16% bisexual; 8.3% gay/lesbian/homosexual; 1.4% unsure about sex orientation). The majority of the participants had never married (93.1%), and only 6.9% had married or lived with someone. Regarding education level, 9.7% did not graduate high school, 41. 7% had graduated high school or HS equivalent diploma, and 46.5% were in college or graduated 2-year college or 4-year college. Only 2.1% were in or completed graduate/professional school. As far as employment, 63.2% worked (38.2% had part time jobs; 25% had full time jobs; 1.4% were homemaker; 1.4% did not work due to disability; the remaining 34% did not work due to other reasons) (Table 4.1 &4.2).

Table 4.1

Demographic Characteristics of Study Participants (N=144)

Variables	*n* (%)
Age: Mean at Wave 1 (Range)	21.51 (18.83-24.91)
Race/Ethnicity	
African American	101 (70.1)
White	40 (27.8)
Multiracial	3 (2.1)
Sexual orientation	
Heterosexual orientation	107 (74.3)
Bisexual orientation	23 (16)
Gay/lesbian/homosexual orientation	12 (8.3)
Not sure	2 (1.4)
Marital status	
Never married	134 (93.1)
Married/living with someone	10 (6.9)
Education level	
Grade 7 to 12 did not graduate high school	14 (9.7)
High school/HS equivalent	60 (41.7)
College (graduated 2-year or 4-year college/part college)	67 (46.5)
Graduate/professional school (completed/part graduate or professional school)	3 (2.1)
Employment status	
Homemaker	2 (1.4)
Did not work due to disability	2 (1.4)
Did not work	49 (34)
Worked full time	36 (25)
Worked part time	55 (38.2)

Table 4.2
Demographic Differences in BPD Scores

Multi-factor ANOVA Results

	Df	Sum Sq	Mean Sq	F
Race	2	142.70	71.35	3.47
Hispanic	1	0.40	0.42	0.02
Marriage	1	27.00	26.95	1.31
Education	6	122.60	20.44	0.99
Employment	4	186.30	46.57	2.27
Sexual Orientation	3	184.20	61.41	2.99
Residuals	126	2590.60	20.56	

Tukey Post Hoc (Race and Sexual Orientation) 95% CI

	Difference	Lower	Upper	p
Multi v.s. Black	5.33	-0.97	11.63	0.11
White v.s. Black	-1.29	-3.30	0.72	0.29
White v.s.Multi	-6.62	-13.05	-0.18	0.04
Gay/lesbian/homosexual v.s. Bisexual	-2.37	-6.57	1.83	0.46
Heterosexual v.s. Bisexual	-2.49	-5.20	0.23	0.09
Not sure v.s. Bisexual	2.77	-5.93	11.47	0.84
Heterosexual v.s. Gay/lesbian/homosexual	-0.12	-3.71	3.48	1.00
Not sure v.s. Gay/lesbian/homosexual	5.14	-3.88	14.16	0.45
Not sure v.s. Heterosexual	5.25	-3.17	13.68	0.37

4.2 Preliminary Analyses

4.2.1 Internal Consistency of Measures

We conducted preliminary analyses to examine the psychometric properties of all measures and results showed good internal consistency for all among our sample (α ranged from .80-.91, see Table 5.1-5.5).

Table 5.1-5.5
Reliability Statistics of All Measures

Table 5.1

	CTQ (alpha=.86, n=144)				
	std.r	r.cor	r.drop	mean	sd
CTQ1	0.41	0.39	0.33	1.50	0.92
CTQ2rev	0.50	0.47	0.42	1.80	1.18
CTQ3	0.55	0.53	0.50	2.10	1.24
CTQ4	0.49	0.47	0.42	1.40	0.91
CTQ5rev	0.40	0.37	0.33	1.70	1.01
CTQ6	0.50	0.48	0.42	1.30	0.79
CTQ7rev	0.66	0.65	0.61	1.90	1.10
CTQ8	0.53	0.52	0.48	1.80	1.24
CTQ9	0.45	0.42	0.38	1.30	0.82
CTQ10	-0.31	-0.37	-0.38	2.70	1.34
CTQ11	0.70	0.70	0.65	1.60	1.01
CTQ12	0.54	0.51	0.49	2.60	1.50
CTQ13rev	0.46	0.45	0.38	2.60	1.27
CTQ14	0.53	0.51	0.47	2.50	1.25
CTQ15	0.71	0.71	0.65	1.50	0.98
CTQ16	-0.39	-0.44	-0.46	2.30	1.28
CTQ17	0.57	0.56	0.51	1.20	0.72
CTQ18	0.60	0.60	0.56	2.40	1.42
CTQ19rev	0.52	0.51	0.46	3.00	1.29
CTQ20	0.64	0.65	0.60	1.60	1.23
CTQ21	0.64	0.63	0.59	1.30	0.86
CTQ22	-0.42	-0.46	-0.49	2.80	1.31
CTQ23	0.63	0.64	0.59	1.50	1.10
CTQ24	0.58	0.59	0.54	1.60	1.25
CTQ25	0.65	0.64	0.61	2.20	1.49
CTQ26rev	0.49	0.46	0.42	1.90	1.19
CTQ27	0.66	0.67	0.61	1.50	1.27
CTQ28rev	0.61	0.60	0.55	2.50	1.33

Table 5.2

	Anger (alpha=.85, n=144)				
	std.r	r.cor	r.drop	mean	sd
STAXI11	0.61	0.62	0.58	2.60	1.08
STAXI12	0.67	0.68	0.65	2.60	1.09
STAXI13	0.62	0.63	0.60	2.20	1.10
STAXI14	0.60	0.59	0.57	2.50	1.02
STAXI15	0.50	0.49	0.46	2.10	0.97
STAXI16	0.59	0.58	0.55	1.80	0.93
STAXI17	0.62	0.63	0.60	2.30	1.03
STAXI18	0.56	0.55	0.52	2.20	1.09
STAXI19	0.67	0.67	0.64	2.00	1.05
STAXI20	0.53	0.53	0.49	2.40	1.08
STAXI21	-0.20	-0.23	-0.29	2.40	0.88
STAXI22	0.38	0.36	0.33	2.60	0.93
STAXI23	0.25	0.22	0.17	2.70	1.05
STAXI24	-0.02	-0.05	-0.11	2.40	0.99
STAXI25	0.40	0.37	0.35	2.10	0.90
STAXI26	0.50	0.48	0.45	2.90	1.01
STAXI27	0.51	0.50	0.47	2.60	1.11
STAXI28	0.12	0.10	0.03	2.30	0.85
STAXI29	0.52	0.50	0.47	2.00	1.06
STAXI30	0.32	0.30	0.23	2.50	1.00
STAXI31	-0.05	-0.08	-0.15	2.40	0.88
STAXI32	0.51	0.50	0.47	2.40	0.97
STAXI33	0.53	0.51	0.48	2.20	1.15
STAXI34	0.67	0.67	0.64	1.90	0.88
STAXI35	0.07	0.03	-0.03	2.40	0.94
STAXI36	0.45	0.43	0.39	1.90	0.94
STAXI37	0.66	0.66	0.63	2.50	1.04
STAXI38	-0.04	-0.08	-0.14	2.00	0.90
STAXI39	0.61	0.61	0.58	2.20	1.02
STAXI40	0.19	0.16	0.10	2.90	0.94
STAXI41	0.64	0.63	0.59	2.70	1.00
STAXI42	0.64	0.65	0.62	2.50	1.02
STAXI43	0.35	0.32	0.29	2.60	1.02
STAXI44	-0.01	-0.04	-0.11	2.40	0.85

Table 5.3

GASP (alpha=.80, n=144)					
	std.r	r.cor	r.drop	mean	sd
GASP1	0.45	0.39	0.35	4.00	2.10
GASP2	0.42	0.35	0.31	5.40	1.80
GASP3	0.69	0.68	0.62	4.60	2.10
GASP4	0.42	0.36	0.31	3.10	1.80
GASP5	0.36	0.30	0.24	5.10	1.80
GASP6	0.54	0.50	0.44	5.00	1.90
GASP7	0.25	0.16	0.13	3.50	1.60
GASP8	0.19	0.10	0.05	3.20	2.00
GASP9	0.59	0.56	0.50	5.00	2.00
GASP10	0.65	0.64	0.58	4.90	1.90
GASP11	0.61	0.58	0.52	5.50	1.50
GASP12	0.37	0.30	0.26	2.80	1.70
GASP13	0.62	0.59	0.53	3.80	2.00
GASP14	0.63	0.61	0.54	4.90	2.00
GASP15	0.54	0.51	0.44	5.20	1.60
GASP16	0.64	0.63	0.56	5.00	1.80

Table 5.4

	PAI-BOR (alpha=.86, n=144)				
	std.r	r.cor	r.drop	mean	sd
paibor1	0.67	0.67	0.62	3.00	0.89
paibor2	0.62	0.62	0.57	2.40	0.96
paibor3	0.62	0.61	0.58	2.30	1.05
paibor4	0.69	0.69	0.65	2.80	0.99
paibor5	0.59	0.58	0.54	2.30	1.09
paibor6	0.55	0.53	0.50	2.60	1.08
paibor7rev	0.52	0.49	0.45	3.20	0.82
paibor8	0.48	0.45	0.41	2.20	1.03
paibor9	0.48	0.45	0.42	3.10	0.91
paibor10	0.50	0.46	0.43	2.10	0.94
paibor11	0.40	0.37	0.33	2.70	1.05
paibor12rev	0.42	0.40	0.35	2.90	0.98
paibor13	0.53	0.51	0.46	1.60	0.82
paibor14rev	0.32	0.27	0.23	2.40	0.94
paibor15	0.61	0.60	0.56	2.50	1.01
paibor16	0.49	0.46	0.42	2.40	1.13
paibor17	0.47	0.44	0.40	1.20	0.53
paibor18	0.57	0.55	0.52	2.50	1.08
paibor19rev	0.24	0.19	0.16	3.20	0.88
paibor20rev	0.30	0.26	0.21	2.60	0.86
paibor21	0.56	0.54	0.48	1.80	0.87
paibor22	0.40	0.38	0.31	3.00	1.06
paibor23	0.48	0.45	0.39	1.60	0.88
paibor24rev	0.33	0.29	0.25	2.90	0.84

Table 5.5

DERS (alpha=.91, n=144)					
	std.r	r.cor	r.drop	mean	sd
DERS1rev	0.38	0.36	0.32	2.80	1.08
DERS2rev	0.30	0.29	0.23	2.30	1.14
DERS3	0.56	0.55	0.52	2.80	1.24
DERS4	0.56	0.55	0.51	2.20	0.98
DERS5	0.62	0.61	0.58	2.40	1.10
DERS6rev	0.23	0.21	0.17	2.90	1.16
DERS7rev	0.32	0.31	0.26	3.00	1.16
DERS8rev	0.16	0.15	0.10	2.40	1.15
DERS9	0.53	0.51	0.48	2.40	1.10
DERS10rev	0.17	0.16	0.10	2.60	1.26
DERS11	0.51	0.50	0.47	2.50	1.22
DERS12	0.43	0.42	0.39	2.00	1.09
DERS13	0.54	0.53	0.50	2.60	1.31
DERS14	0.56	0.55	0.52	2.10	1.27
DERS15	0.64	0.64	0.61	2.20	1.28
DERS16	0.63	0.63	0.60	2.50	1.33
DERS17rev	0.13	0.10	0.06	2.80	1.28
DERS18	0.55	0.54	0.51	2.90	1.16
DERS19	0.65	0.65	0.62	2.40	1.29
DERS20rev	0.35	0.33	0.29	3.00	1.16
DERS21	0.56	0.56	0.52	2.20	1.17
DERS22rev	0.29	0.27	0.23	2.70	1.09
DERS23	0.51	0.50	0.48	2.30	1.30
DERS24rev	0.50	0.49	0.44	3.20	1.20
DERS25	0.46	0.45	0.42	2.10	1.16
DERS26	0.52	0.50	0.48	2.80	1.20
DERS27	0.62	0.61	0.58	2.40	1.23
DERS28	0.66	0.66	0.63	2.20	1.11
DERS29	0.69	0.69	0.66	2.40	1.23
DERS30	0.67	0.67	0.65	2.30	1.27
DERS31	0.64	0.63	0.61	2.00	1.18
DERS32	0.53	0.52	0.50	2.60	1.17
DERS33	0.52	0.50	0.48	2.20	1.13
DERS34rev	0.19	0.16	0.12	3.40	1.16

DERS35	0.51	0.50	0.48	2.60	1.12
DERS36	0.65	0.65	0.62	3.00	1.27

4.2.2 Selection of Potential Control Variables

To select potential control variables, demographic differences in BPD scores were assessed using Multi-factor Analysis of Variance (ANOVA) in R. Results from Multi-factor ANOVA evidenced no significant between-group differences in BPD scores except for Race [$F(2) = 3.47$, $p = .03$](Again see Table 4.2 inserted before).Tukey Post Hoc results revealed that individuals who self-identified as multi-racial had a significant higher BPD scores in relative to their white counterparts . Given the significant difference, race was selected as the control variable in all our multivariate regression models.

4.3 Aim 1 (Baseline & Cross-lag)

4.3.1 Baseline Multiple Regression

Table 6 presents parameters and model fit indices of all our multiple regression models. The initial model comprised five single trauma types as main predictors. Results from Model 1 indicated that only emotional abuse (b=.68, t=3.12, p<.001) was significantly associated with higher BPD features. The overall model R^2 was significant, accounting for approximately 25% of the variance. Model 2 included DERS as an additional predictor. This model showed that EA (b=.41, t=2.32, p=.02), PA (b=.56, t=2.30, p<.001) and DERS (b=.29, t=8.66, p<.001) were all significantly correlated with higher BPD scores. There is a significant increase in model R^2, indicating an improvement in model performance. In Model 3, we introduced four additional predictors: Trait Anger, Anger Expression-out, Anger Expression-in and Anger Control (this variable was reversed coded). Model 3 further improved from model (ΔR^2= 8%,p<.001), and it

revealed that EA (b=.39, t=2.43, p=.02), DERS (b=.17, t=4.53, p<.001), as well as Anger Expression Out (b=2.89, t=1.33, p=.04) were all significantly associated with BPD scores.

In the final model, four subconstructs of shame/guilt were added, and results demonstrated that EA (b=.35, t=2.10, p=.04), DERS (b=.19, t=4.98, p<.001), Guilt (repair; b=2.41, t=3.04, p<.001), and Shame (negative self-evaluation; b=-1.27, t=-2.66, p=.03) were all significantly associated with BPD scores. It is important to note that all significant associations were positive except for the one between Guilt-repair and BPD scores. The final model was significantly improved from model 3, accounting for about 62% of the variance. Interestingly, both people with no job due to being disabled and those with a full-time employment showed significantly heightened BPD features in relative to homemakers.

Table 6.
Regression Models Predicting BPD Features (N=144)

Variables	Model 1					Model 2					Model 3					Model 4				
	b	se	t	p		b	se	t	p		b	se	t	p		b	se	t	p	
(Intercept)	23.36	7.33	3.19	0.00	**	0.81	6.43	0.13	0.90		-11.93	7.49	-1.59	0.11		-19.87	8.37	-2.37	0.02	*
CTQ_EA	0.68	0.22	3.12	0.00	**	0.41	0.18	2.32	0.02	*	0.39	0.16	2.43	0.02	*	0.35	0.17	2.10	0.04	*
CTQ_PA	0.45	0.30	1.50	0.14		0.56	0.24	2.30	0.02	*	0.32	0.23	1.42	0.16		0.33	0.22	1.48	0.14	
CTQ_SA	0.05	0.19	0.29	0.77		0.22	0.15	1.46	0.15		0.19	0.14	1.41	0.16		0.10	0.14	0.76	0.45	
CTQ_EN	0.14	0.23	0.61	0.54		-0.01	0.18	-0.07	0.94		0.02	0.17	0.10	0.92		0.00	0.17	-0.03	0.98	
CTQ_PN	-0.25	0.31	-0.82	0.41		-0.35	0.25	-1.40	0.17		-0.29	0.23	-1.26	0.21		-0.29	0.23	-1.28	0.20	
DERS_Total						0.29	0.03	8.66	0.00	***	0.17	0.04	4.53	0.00	***	0.19	0.04	4.98	0.00	***
T_Anger											2.24	1.40	1.59	0.11		2.69	1.42	1.90	0.06	
Ang_Con											0.95	1.24	0.76	0.45		0.92	1.24	0.74	0.46	
Ang_Out											2.89	1.42	2.04	0.04	*	1.85	1.47	1.26	0.21	
Ang_In											1.78	1.34	1.33	0.19		1.74	1.30	1.33	0.19	
NBE																-0.51	0.63	-0.80	0.42	
GR																2.41	0.79	3.04	0.00	**
NSE																-1.27	0.58	-2.19	0.03	*
SW																0.55	0.59	0.94	0.35	
No (Disabled)	16.00	10.17	1.57	0.12		9.52	8.20	1.16	0.25		20.89	7.83	2.67	0.01	**	26.97	7.85	3.44	0.00	***
No (Other)	3.43	7.12	0.48	0.63		1.44	5.72	0.25	0.80		6.04	5.34	1.13	0.26		8.61	5.26	1.64	0.10	
Yes (Full-time)	1.96	7.17	0.27	0.79		2.20	5.75	0.38	0.70		7.75	5.37	1.44	0.15		11.16	5.34	2.09	0.04	*
Yes (Part-time)	-3.32	7.06	-0.47	0.64		-3.46	5.66	-0.61	0.54		2.61	5.32	0.49	0.62		5.43	5.25	1.03	0.30	
R2adjusted	0.25					0.52					0.60					0.62				
F	6.36					16.37					16.26					13.98				
df	9,134					10, 135					14, 129					18, 125				
p (ΔR2)						<.001					<.001					0.03				

Note. * p<.05, ** p<.01, *** p<.001.

4.3.2 Cross-lagged Models

We conducted eight sets of cross-lagged models using SEM methods in R. For example, in the model of Anger Control predicting BPD, BPD at wave 1 is predicted by Anger Control at wave 1; BPD at wave 2 is predicted by BPD and Anger Control scores preceding it; and BPD at wave 3 is predicted by BPD and Anger Control preceding it. In addition, concurrent paths (e.g BPD 1 and Anger 1) were modeled as correlations (Figures 5-12). Overall, the model fit indices were not very satisfactory. However, this was expected as we maintained all the nonsignificant paths in order to verify that the reverse patterns (e.g. the reverse path from BPD to Anger) are indeed not significant (See Table 7 for specifics of each model).

In Table 7 and Figures 5-12, we present the path parameters of all cross-lagged models. In general, we failed to detect a consistent directional pattern from Anger/Shame/Guilt to BPD, in that NA measures were not associated with BPD in either direction. However, we discovered a reverse significant direction in the model of NBE and BPD. In particular, higher scores of NBE at wave 2 and wave 3 were significantly predicted by BPD scores at previous waves (i.e. from BPD2 to NBE3; from BPD1 to NBE2).

Table 7
Path Parameters and Model Fit of Cross-lagged Models

T_Anger (Significant X^2, CFI- .93, TLI=.74, RMSEA=.19, SRMR=.057)						
		se	z	beta	p	r^2
BOR2 ~						0.26
	T_Anger1	0.41	1.86	0.16	0.06	
	BOR1	0.09	3.17	0.41	0.00	
T_Anger2 ~						0.29
	T_Anger1	0.07	5.17	0.47	0.00	
	BOR1	0.01	1.47	0.14	0.14	
BOR3 ~						0.36
	T_Anger2	0.59	-0.35	-0.04	0.73	
	BOR2	0.13	4.05	0.62	0.00	
T_Anger3 ~						0.37
	T_Anger2	0.09	5.58	0.57	0.00	
	BOR2	0.01	0.57	0.05	0.57	

Anger Control (Significant X^2, CFI= .95, TLI=.81, RMSEA=.13, SRMR=.05)						
		se	z	beta	p	r^2
BOR2 ~						0.24
	Ang_Con1	0.36	1.21	0.08	0.23	
	BOR1	0.08	4.16	0.47	0.00	
Ang_Con2 ~						0.21
	Ang_Con1	0.07	4.96	0.37	0.00	
	BOR1	0.01	3.27	0.23	0.00	
BOR3 ~						0.37
	Ang_Con2	0.31	2.27	0.13	0.02	
	BOR2	0.09	5.40	0.56	0.00	
Ang_Con3 ~						0.36
	Ang_Con2	0.08	7.02	0.60	0.00	
	BOR2	0.01	0.28	0.02	0.78	

Anger Out (Significant X^2, CFI=.96, TLI=.85, RMSEA=.13, SRMR=.05)						
BOR2 ~						0.24
	Ang_Out1	0.40	0.96	0.08	0.34	
	BOR1	0.09	3.65	0.45	0.00	
Ang_Out2 ~						0.19
	Ang_Out1	0.07	4.64	0.40	0.00	
	BOR1	0.01	1.08	0.09	0.28	
BOR3 ~						0.36
	Ang_Out2	0.45	0.64	0.06	0.52	
	BOR2	0.10	4.67	0.57	0.00	
Ang_Out3 ~						0.36
	Ang_Out2	0.09	5.59	0.55	0.00	
	BOR2	0.01	1.42	0.11	0.15	

Anger In (Significant X^2, CFI=.94, TLI=.76, RMSEA=.16, SRMR=.06)						
BOR2 ~						0.24
	Ang_In1	0.40	-1.23	-0.08	0.22	
	BOR1	0.09	4.19	0.52	0.00	
Ang_In2 ~						0.17
	Ang_In1	0.07	4.49	0.41	0.00	
	BOR1	0.01	0.09	0.01	0.93	
BOR3 ~						0.37
	Ang_In2	0.49	-1.12	-0.09	0.26	
	BOR2	0.10	5.17	0.63	0.00	
Ang_In3 ~						0.25
	Ang_In2	0.07	4.80	0.41	0.00	
	BOR2	0.01	1.96	0.17	0.05	

GR (Significant X^2, CFI=.94, TLI=.78, RMSEA=.13, SRMR=.05)

BOR2 ~						0.24
	GR1	0.22	-0.19	-0.01	0.85	
	BOR1	0.08	4.24	0.48	0.00	
GR2 ~						0.23
	GR1	0.06	5.57	0.43	0.00	
	BOR1	0.02	-1.80	-0.18	0.07	
BOR3 ~						0.36
	GR2	0.27	-0.31	-0.03	0.76	
	BOR2	0.09	5.58	0.60	0.00	
GR3 ~						0.17
	GR2	0.13	3.05	0.41	0.00	
	BOR2	0.02	0.64	0.05	0.52	

NBE (Significant X^2, CFI=.90, TLI=.61, RMSEA=.19, SRMR=.06)

BOR2 ~						0.24
	NBE1	0.20	-0.25	-0.02	0.80	
	BOR1	0.08	4.25	0.48	0.00	
NBE2 ~						0.27
	NBE1	0.06	5.60	0.45	0.00	
	BOR1	0.02	-2.54	-0.18	0.01	
BOR3 ~						0.36
	NBE2	0.21	-0.12	-0.01	0.90	
	BOR2	0.09	5.48	0.60	0.00	
NBE3 ~						0.25
	NBE2	0.08	5.66	0.50	0.00	
	BOR3	0.02	0.54	0.04	0.01	

NSE (Significant X^2, CFI=.92, TLI=.70, RMSEA=.18, SRMR=.06)

BOR2 ~						0.24
	NSE1	0.19	-0.87	-0.07	0.39	
	BOR1	0.08	4.08	0.47	0.00	
NSE2 ~						0.33
	NSE1	0.06	6.72	0.53	0.00	
	BOR1	0.02	-1.85	-0.13	0.07	
BOR3 ~						0.36
	NSE2	0.19	1.02	0.08	0.31	
	BOR2	0.09	5.57	0.61	0.00	
NSE3 ~						0.34
	NSE2	0.09	5.91	0.55	0.00	
	BOR2	0.03	-1.58	-0.12	0.12	

SW (Significant X^2, CFI=.93, TLI=.73, RMSEA=.13, SRMR=.056)

BOR2 ~						0.24
	SW1	0.20	-0.57	-0.04	0.57	
	BOR1	0.08	4.26	0.49	0.00	
SW2 ~						0.13
	SW1	0.06	4.77	0.36	0.00	
	BOR1	0.02	-0.32	-0.03	0.75	
BOR3 ~						0.36
	SW2	0.20	0.44	0.03	0.66	
	BOR2	0.09	5.62	0.60	0.00	
SW3 ~						0.12
	SW2	0.10	2.76	0.30	0.01	
	BOR2	0.02	1.59	0.14	0.11	

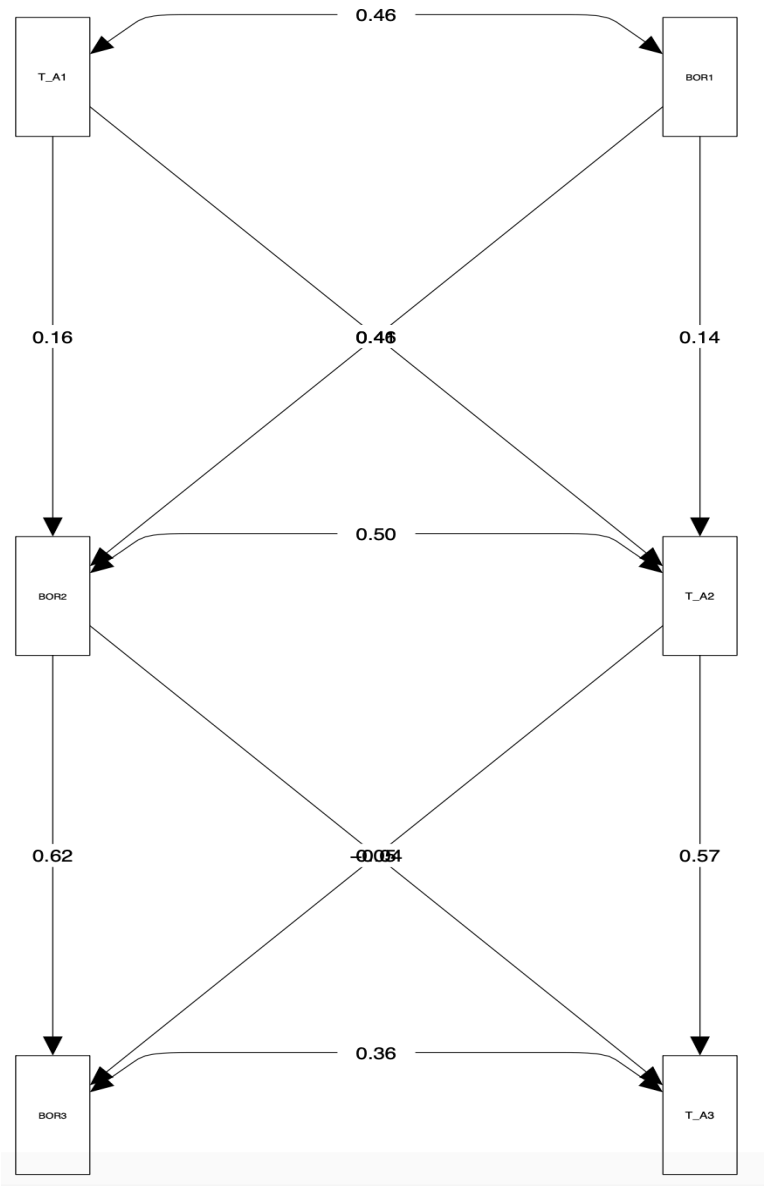


Figure 5 Cross-lagged Models of Trait Anger and BPD

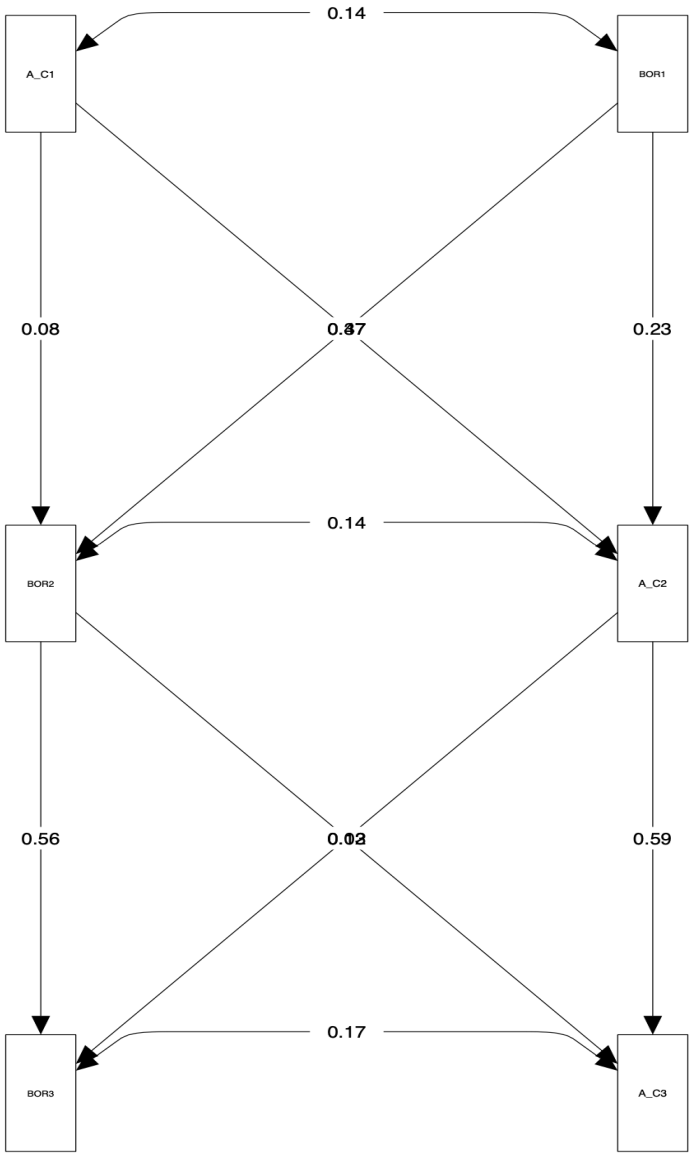


Figure 6 Cross-lagged Models of Anger Control and BPD

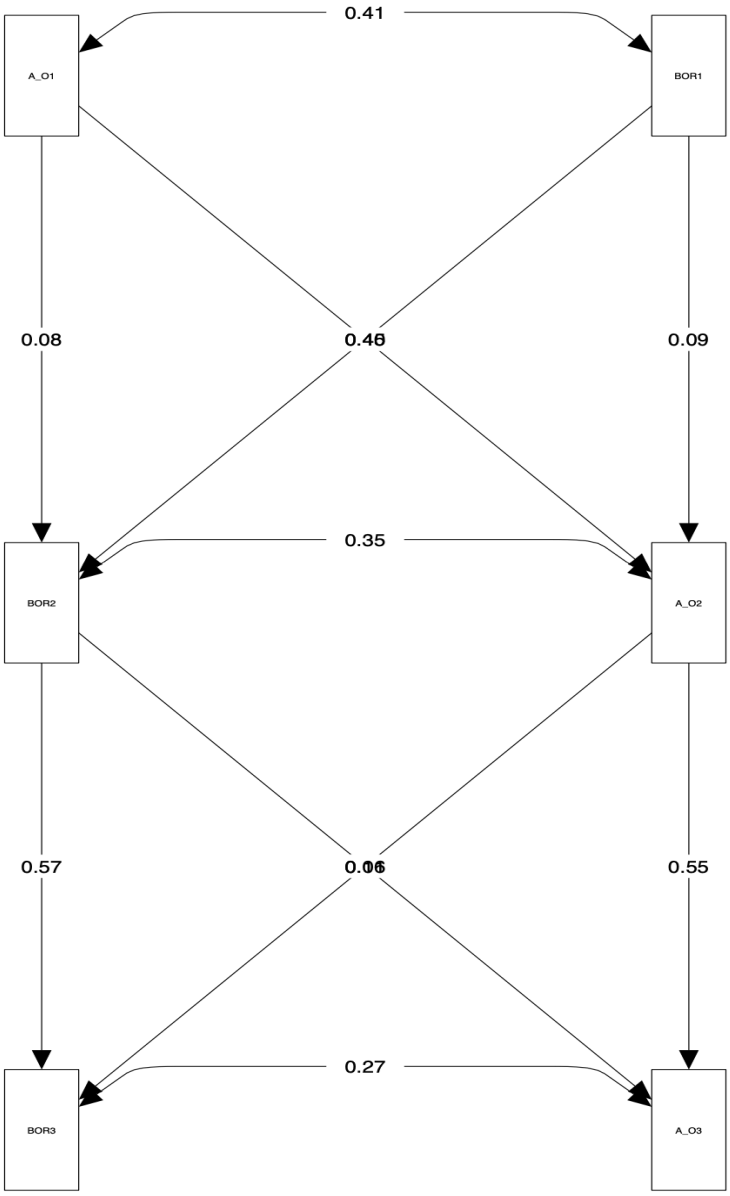


Figure 7 . Cross-lagged Models of Anger Out and BPD

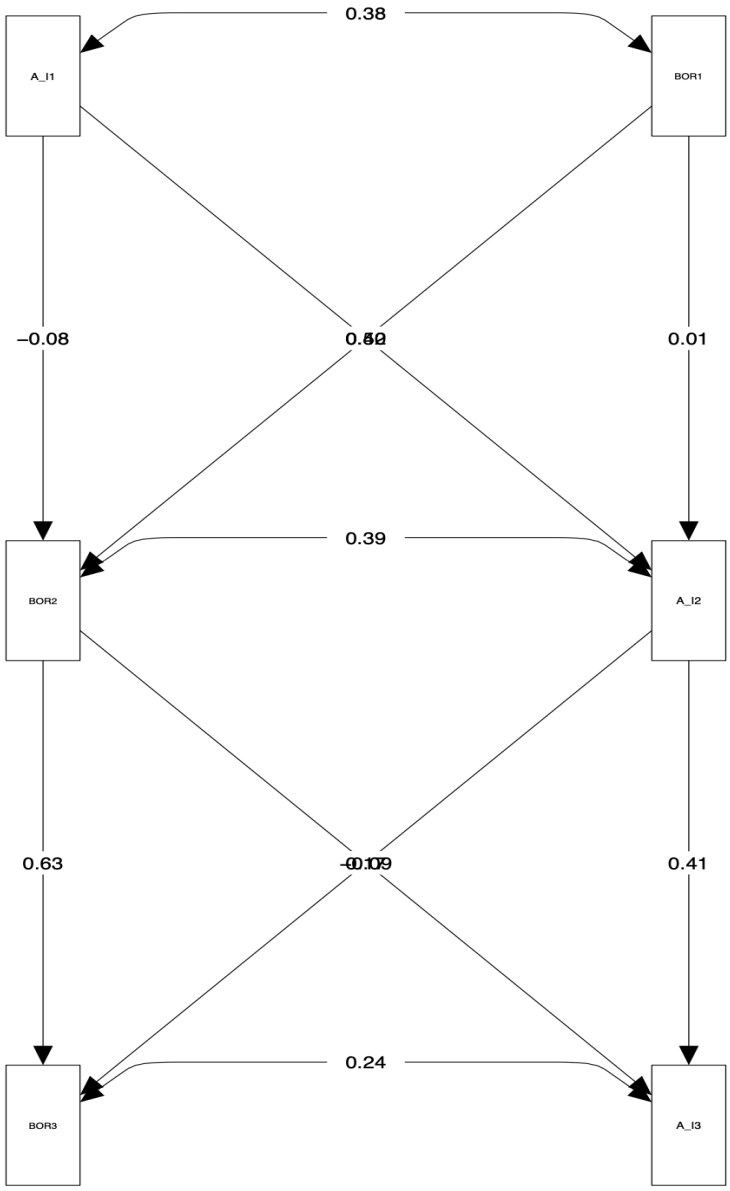


Figure 8 Cross-lagged Models of Anger In and BPD

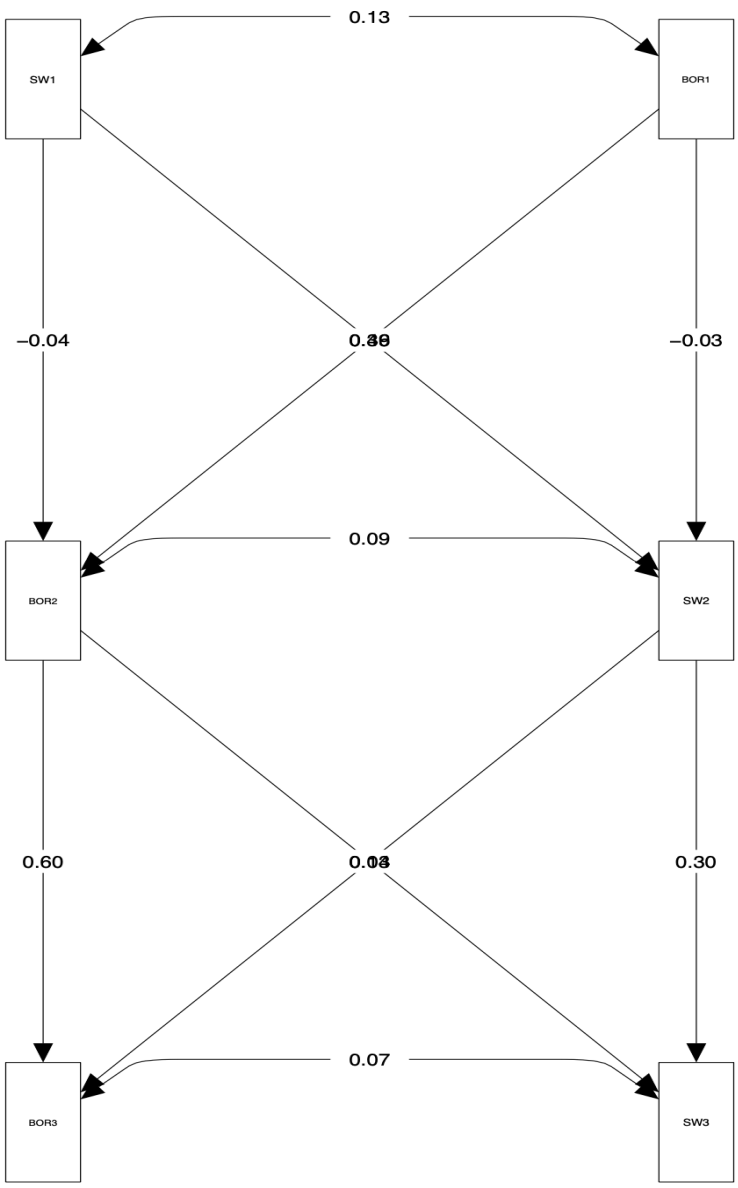


Figure 9 Cross-lagged Models of SW and BPD

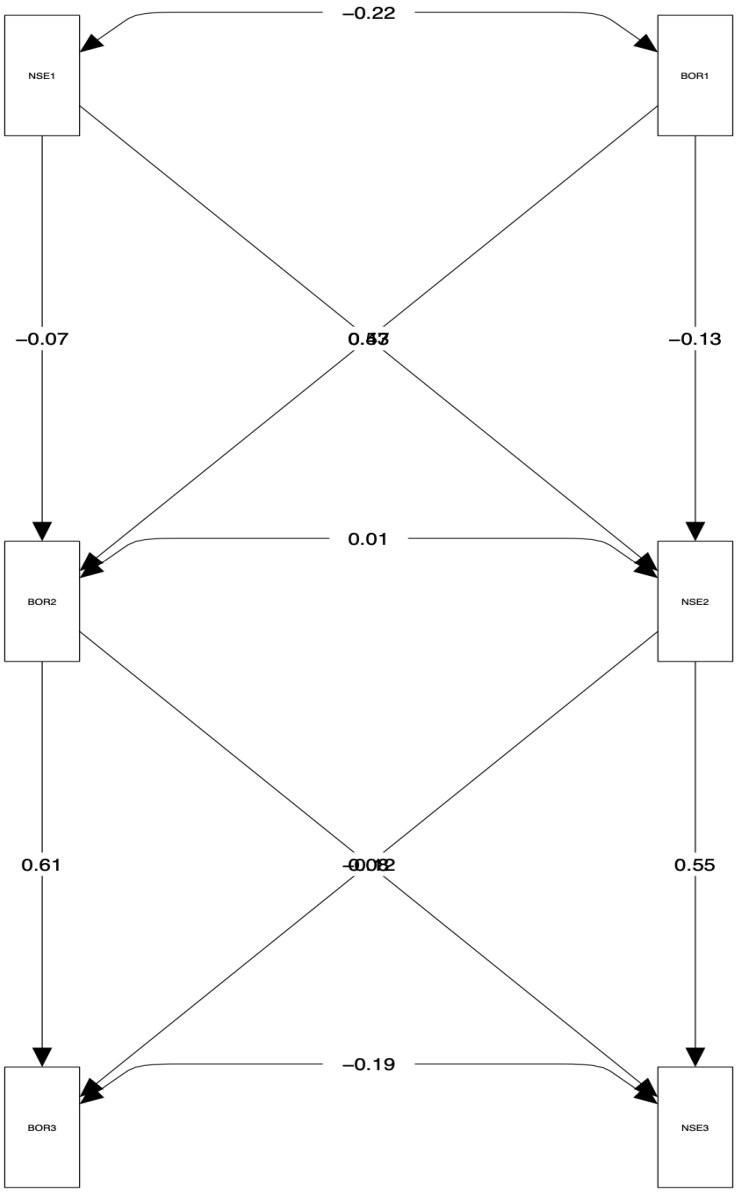


Figure 10 Cross-lagged Models of NSE and BPD

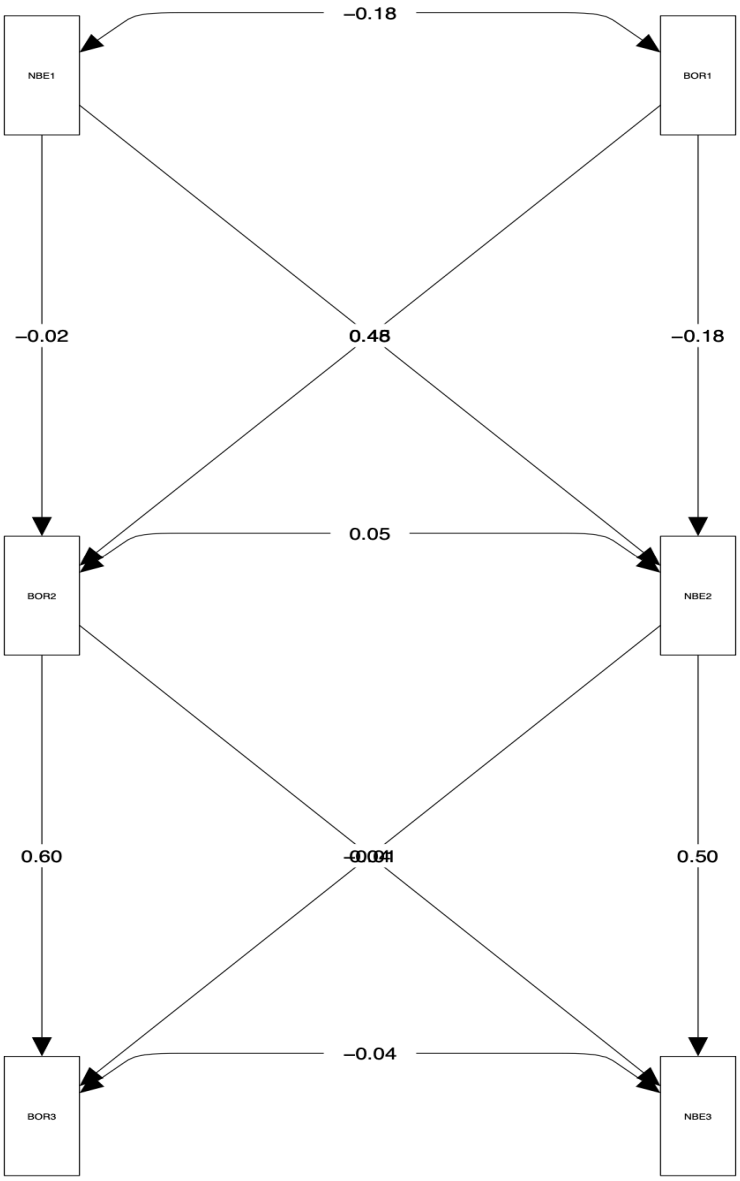


Figure 11 Cross-lagged Models of NBE and BPD

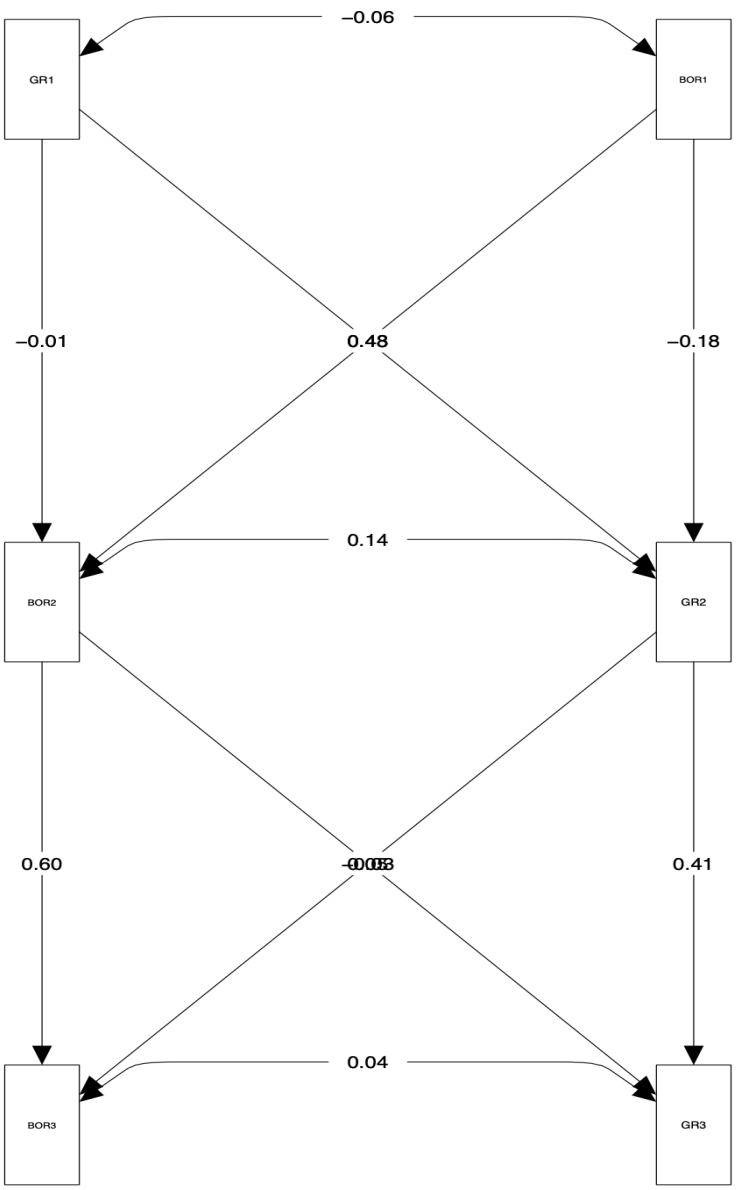


Figure 12 . Cross-lagged Models of GR and BPD

4.4 Aim 2 (Latent Growth Curve)

Descriptive statistics (e.g. *mean, sd and multivariate analysis of variance*), and visual inspection of linear relationships among NA (Anger Control, Anger Expression, Shame and Guilt), trauma, and BPD all together revealed that (1) there might be significant changes in all measures over time, and (2) there was a consistent decreasing trend in NA and BPD (this is indeed inconsistent with our original hypotheses) (See Figures 13-14).

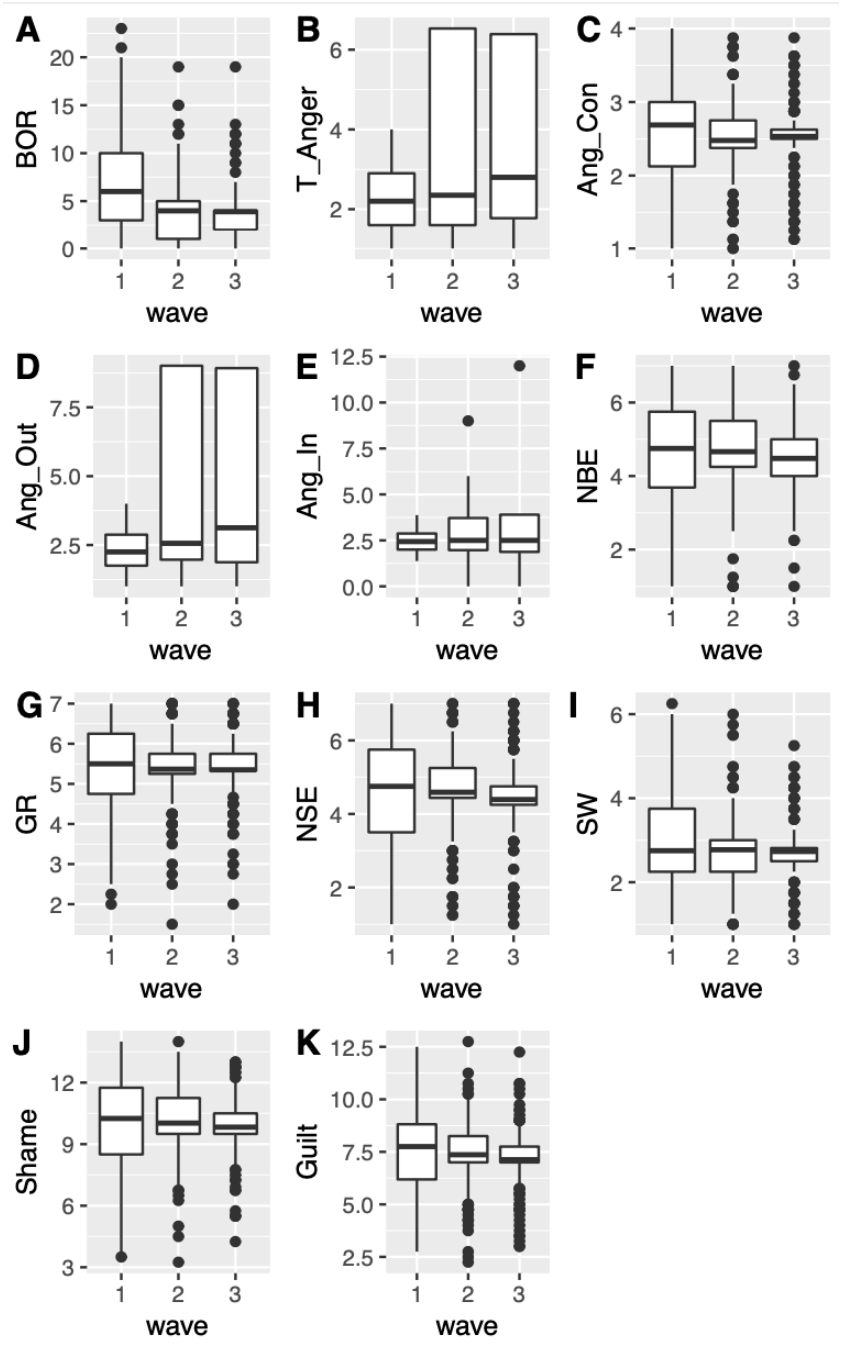


Figure 13 Combined Boxplots of Individual Differences in Key Variables Across Three Time Points

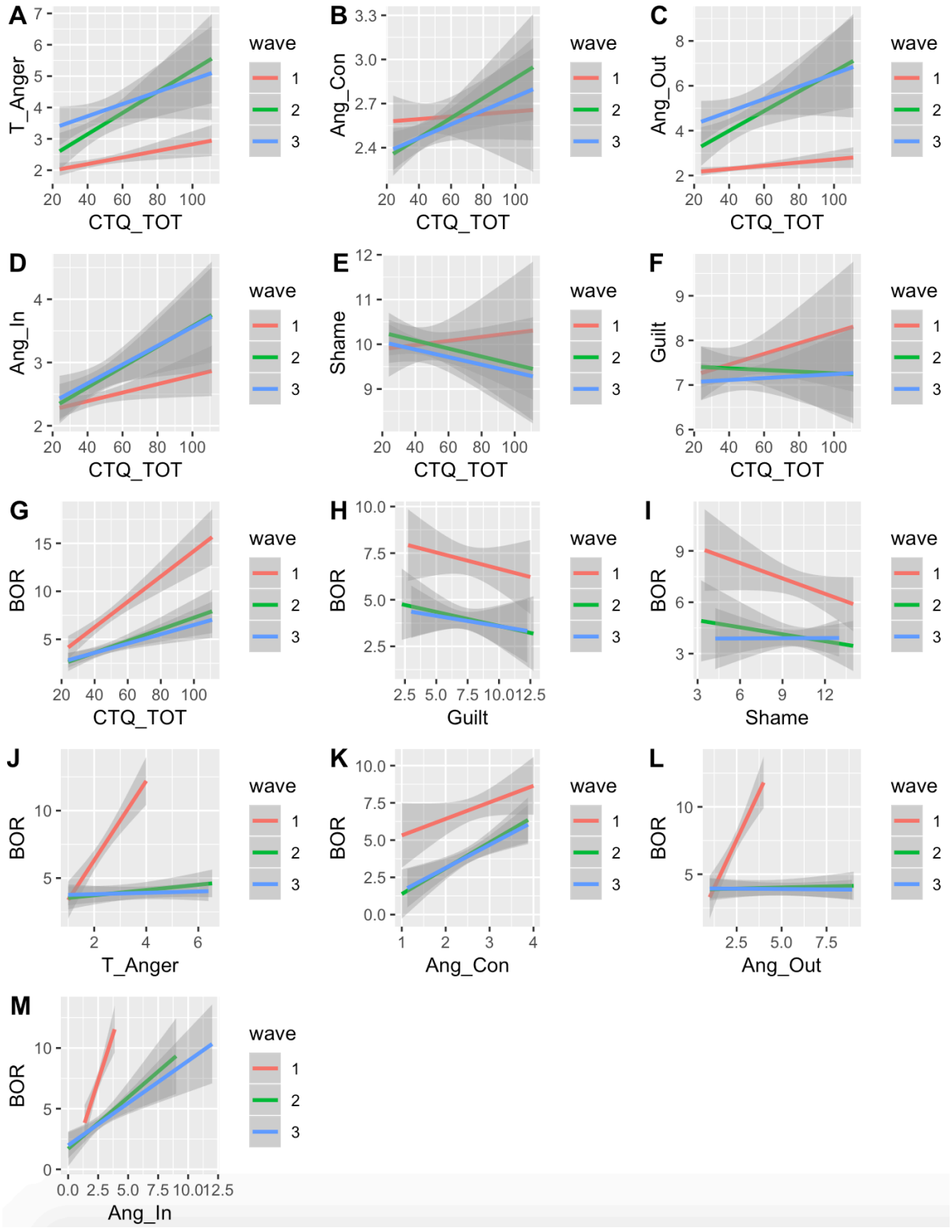


Figure 14 Visualization of Correlations among NA Measures and BPD Scores Grouped by Waves

Five sets of first-order latent growth curve models were conducted for four individual NA measures and BPD measure (both clinical interviews and self-report measures; See sample path diagram in Figure 15). As can be seen in Table 8, there was good model fit for Anger Control [$X^2(8,144)=.087$, $p=.77$; $CFI=1.00$; $RMSEA=.00$, 90% CI (.11, .15); $SRMR=.01$], Anger Expression [$X^2(8,144)=3.37$, $p=.07$; $CFI=.98$; $RMSEA=.13$, 90% CI (.00, .29); $SRMR=.03$], Shame [$X^2(8,144)=.73$, $p=.39$; $CFI=.98$; $RMSEA=.00$, 90% CI (.00, .14); $SRMR=.02$] and Guilt [$X^2(8,144)=.07$, $p=.79$; $CFI=.99$; $RMSEA=.00$, 90% CI (.00, .14); $SRMR=.01$], but not BPD [significant X^2; $CFI=.70$; $RMSEA=.47$; 90% CI (.34, .62); $SRMR=.12$]. When prediction from trauma to NA and BPD was added to all five models, it significantly undermined the model fits for all, and we merely detected significant predictions from trauma to mean intercept of Anger expression and mean intercept of Shame over time.

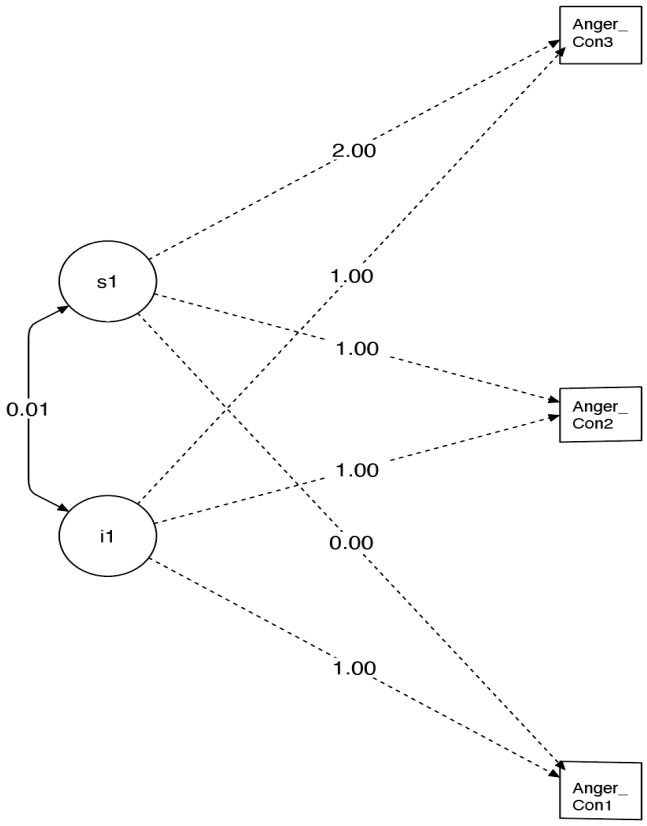


Figure 15 . Example First-order Latent Growth Curve Model

As expected, the growth parameters indicated that there was significant variability in baseline Anger Control (z=51.96, b=6.73, p<.001), Anger Expression (z=50.55, b=5.92, p<.001), Shame (z=58.19, b=6.46, p<.001) and Guilt (z=46.64, b=5.11, p<.001); as well as the rates of change in Anger Control (z=2.67, b=.64, p<.05), Anger Expression (z=-4.68, b=-1.61, p<.001), and Guilt (z=-2.64, b=-.33, p<.05) across three waves. Given the poor model fit of BPD, a reliable interpretation of the parameters cannot be made; nevertheless, the previous inspections on descriptive statistics indeed indicated a relatively stable slope over time, and non-significant change in the means of BPD from wave 2 to wave 1.

Table 8
Path Parameters of Latent Growth Curve Analyses

		B	*SE*	*Z*	*P*	*beta*	*r^2*
a) Anger Control X^2 (8,144)=.087, p=.77; CFI=1.00; RMSE=.00; 90% CI (.11, .15); SRMR=.01							
Variance	Anger Control 1	0.25	0.05	4.78	0.00	0.66	0.335
	Anger Control 2	0.14	0.02	5.83	0.00	0.49	0.515
	Anger Control 3	0.07	0.04	1.76	0.08	0.27	0.728
Mean i & s	i	2.41	0.05	51.96	0.00	6.73	
	s	0.06	0.02	2.67	0.01	0.64	
b) Anger Expression X^2 (8,144)=3.37, p=.07; CFI=.98; RMSEA= .13, 90% CI(.00, .29); SRMR=.03							
Variance	Anger Express 1	1.66	0.40	4.10	0.00	0.55	0.452
	Anger Express 2	0.84	0.16	5.26	0.00	0.42	0.585
	Anger Express 3	0.58	0.30	1.95	0.05	0.36	0.645
Mean i & s	i	6.93	0.14	50.55	0.00	5.92	
	s	-0.29	0.06	-4.68	0.00	-1.61	
c) Shame X^2 (8,144)=.73, p=.39; CFI=.98; RMSEA= .00 90% CI(.00, .21); SRMR=.02							
Variance	Shame 1	2.68	0.61	4.40	0.00	0.53	0.475
	Shame 2	1.24	0.24	5.24	0.00	0.43	0.573
	Shame 3	1.40	0.41	3.46	0.00	0.58	0.416
Mean i & s	i	10.08	0.17	58.19	0.00	6.46	
	s	-0.11	0.09	-1.25	0.21	-0.42	

d) Guilt X^2 (8,144)=.07, p=.79; CFI=.99; RMSEA= .00 90% CI(.00, .14); SRMR=.005							
Variance	Guilt 1	2.83	0.60	4.69	0.00	0.61	0.386
	Guilt 2	1.28	0.24	5.43	0.00	0.49	0.512
	Guilt 3	1.72	0.41	4.19	0.00	0.78	0.222
Mean i & s	i	7.55	0.16	46.64	0.00	5.11	
	s	-0.21	0.08	-2.64	0.01	-0.33	
e) BPD Model 1 significant X^2; CFI=.70; RMSEA= .47 90% CI(.34, .62); SRMR=.12							
Variance	BPD1	15.47	3.21	4.82	0.00	0.65	0.348
	BPD2	6.45	1.20	5.36	0.00	0.50	0.500
	BPD3	2.50	1.79	1.40	0.16	0.30	0.698
Mean i & s	i	6.03	0.40	15.07	0.00	2.10	
	s	-1.15	0.19	-6.06	0.00	-1.52	

In addition to first order LGM, we performed a curves-of-factor latent growth curve model (two-level model). As compared with the first order model (only assessing the change in a single item), the two-level model focused on the overall trajectory in the construct of NA using multiple measures across three time points, as well as to investigate its longitudinal relationship with BPD scores. As can be seen in Figure 16, a latent variable of NA was created at each time point with individual NA indicators (e.g. Anger Expression). Other parameters specified in the model included: A common slope and intercept of NA across three waves, slope and intercept of BPD over time, as well as the prediction from longitudinal growth in NA to longitudinal growth in BPD. Considering that the model performance is very poor, the interpretation can be problematic. However, informed by first-order models, the longitudinal changes in some of the measures (e.g. guilt and BPD) were not significant, and thus a failure to detect consistent longitudinal association pattern between NA and BPD is understandable.

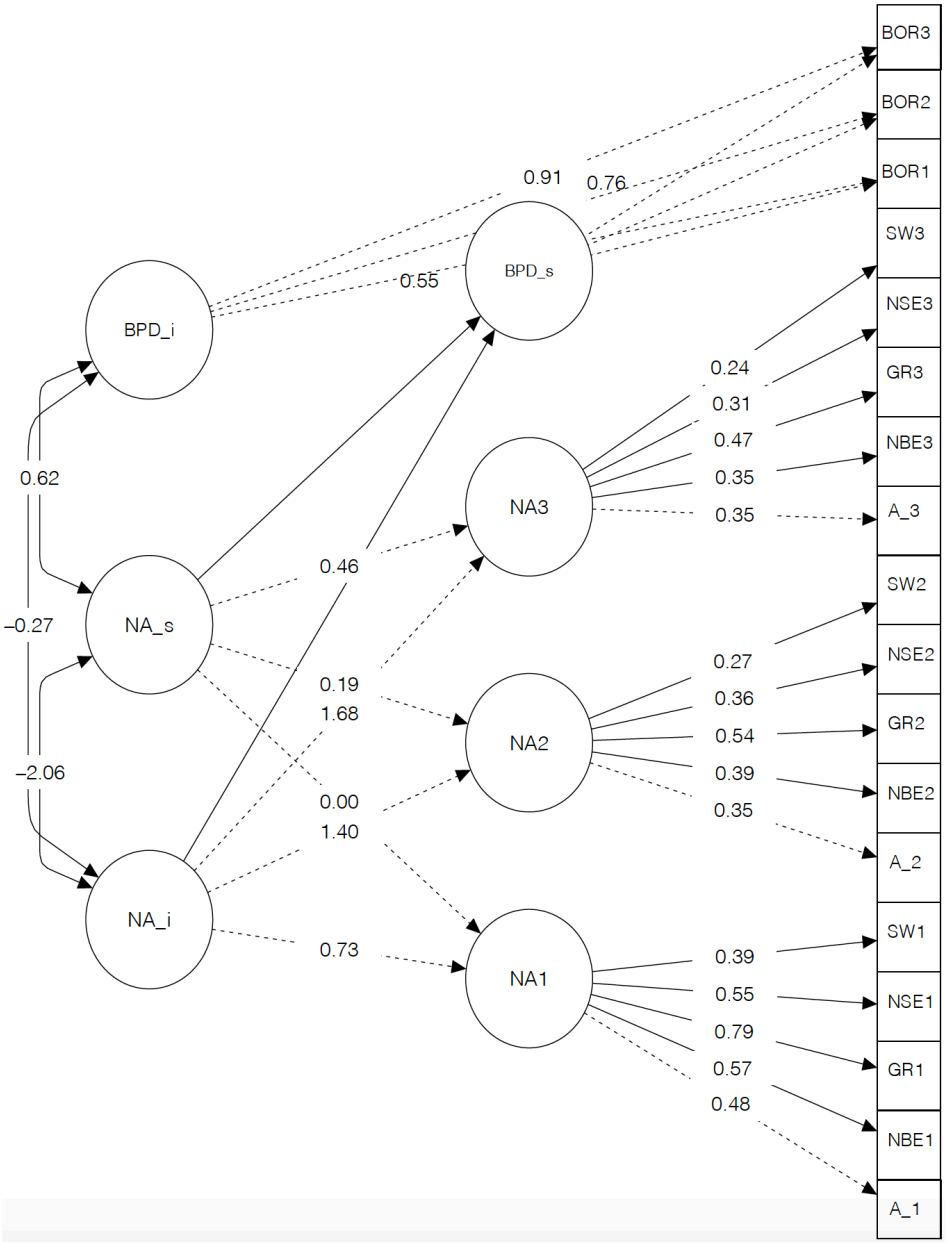


Figure 16 . Curves of Factor Latent Growth Model Predicting Linear Trend in BPD from Linear Trend in Negative Affects[4]

[4] *Note.* NA=Negative Affects, A_1=Anger expression, NBE=Guilt-negative-behavior-evaluation, GR=Guilt-repair, NSE=Shame-negative-self -valuation, W=Shame-withdraw.

4.5 Aim 3 (Mediation SEM)

4.5.1 Mediation with Latent Factors

The initial SEM model had unsatisfactory performance (*CFI* =.67, *SRMR*=.12, and *RMSEA* =.13 (90%*CI*: .11~ .14)). Model performance and subsequent modifications can be evaluated using two main types of statistics: (1) *Wald* statistics (estimated increase in X^2 given a prior estimated path parameter fixed to a known value) and (2) *LaGrange Multiplier* method (predicted decrease in X^2 given a prior fixed path parameter were to be estimated) (Mueller & Hancock, 2018).

The stepwise multivariate *Wald* test in *Lavaan* (Rosseel, 2012) indicated that four non-significant paths can be eliminated from the initial model (the predictions of ED by emotional awareness, both guilt subconstructs, and one shame subscale of negative self-evaluation). The *LaGrange Multiplier* method was subsequently applied for further diagnosis and modification. From the results, five covariances (See Figure 17 and Table 9) were added iteratively to improve the model performance. In this procedure, only covariances underlying the same factor were selected iteratively (e.g. ED manifest variables were allowed to covary); whereas cross-loadings (variables measuring across factors: e.g. ED subconstruct with Trauma subconstruct) were not allowed given that it will be theoretically misleading.

Table 9.
Standardized Parameter Estimates for the Multi-level Mediation Model

Measurement Model							
		B	***SE***	***z***	***p***	***β***	***R^2***
Trauma	→Childhood emotional abuse	1				.78	.61
	→Childhood physical abuse	.73	.09	8.13	.00	.77	.59
	→Childhood sexual abuse	.68	.12	5.66	.00	.52	.27
	→Childhood emotional neglect	.74	.11	6.64	.00	.61	.37
	→Childhood physical neglect	.46	.08	5.79	.00	.54	.29
ED	→Non-acceptance	1				.53	.28
	→Goal-directed behavior	.90	.15	6.13	.00	.59	.35
	→Impulse control	1.68	.26	6.36	.00	.84	.71
	→Emotional regulation strategies	1.68	.23	7.43	.00	.71	.50
	→Emotional clarity	.62	.14	4.35	.00	.45	.20
	→Anger control	-.10	.03	-3.99	.00	-.46	.21
	→Ange expression	.49	.08	6.19	.00	.79	.62
	→Shame-withdraw	.15	.04	3.65	.00	.36	.13
BPD	→Affective instability	1				.87	.75
	→Identity problems	.81	.09	8.55	.00	.69	.47
	→Negative emotions	.75	.09	8.15	.00	.65	.43
	→Self-harm	.72	.10	7.44	.00	.68	.47
Structural Model							
		B	***SE***	***z***	***p***	***β***	***R^2***
BPD	→Trauma (a2)	.29	.06	5.05	.00	.36	.82
	→ED (a1)	.83	.14	5.88	.00	.74	
ED	→Trauma (a3)	.21	.08	2.68	.01	.29	.08
Indirect Effect	a1*a3	.17	.06	2.86	.00	.21	
Total Effect	a2+(a1*a3)	.46	.08	5.70	.00	.57	

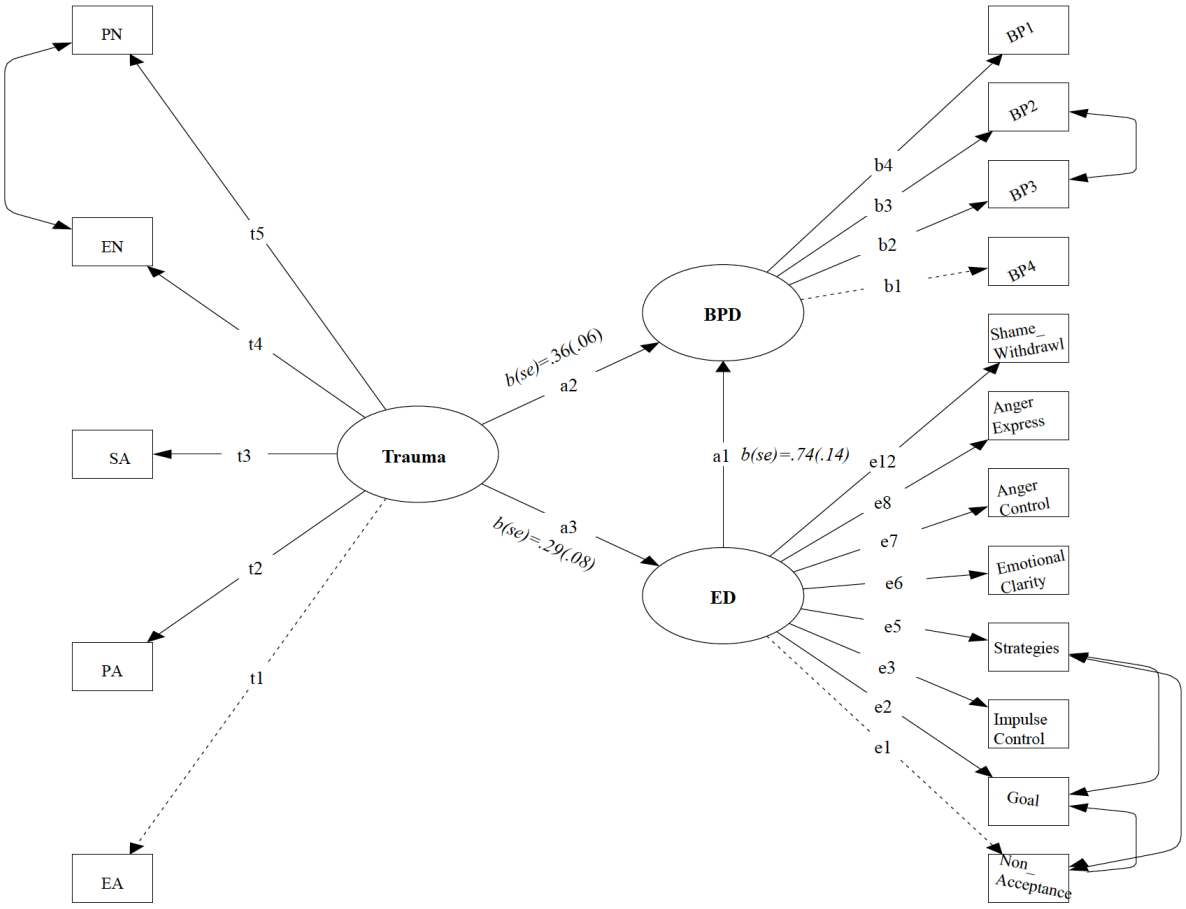


Figure 17 A Path Diagram of Trauma Predicting BPD Partially Mediated via Emotion Dysregulation Dimensions[5]

[5] PN=Physical Neglect, EN=Emotional Neglect, SA=Sexual Abuse, PA=Physical Abuse, EA=Emotional Abuse, BP 1=Affective instability, BP 2= Identity problems, BP 3= Negative Relations, BP 4=Self-harm. Measurement model parameters which were omitted here for a more clear and concise display. All parameters were significant except for three ED subconstructs. Parameters of the paths displayed via dotted lines were fixed. Double arrow lines stand for the covariances among subconstructs.

The modified model was significantly improved from the initial model despite no significant difference from the observed model (ΔX^2=367.57). However, the following indices showed an overall good fit of the final model [*CFI* =.93, *SRMR*=.067, and *RMSEA* =.06 (90%*CI*: .04~ .07)]. As can be seen in Table 8, the final model revealed that three factors were generally well identified with good construct validity.

From information presented in Figure 11 and Table 8, there were significant direct effects of trauma on ED (b=.36, z =5.05, p <.001) and ED on BPD (b=.74, z =5.88, p <.001). After accounting for the indirect effect of trauma on BPD via ED (Δb=.21, z =2.86, p <.01), the total effect of trauma on BPD remained significant (b=.57, z =5.70, p <.001). In other words, higher childhood trauma significantly predicted heightened BPD symptomatology, partially mediated through ED. In addition, trauma showed a significant and unique effect after controlling for the indirect effect via ED.

5.0 Discussion

Our study contributes to an improved understanding of associations among trauma, ED, and BPD features in a diverse group of low-income young women. Trauma experience, especially emotional abuse, is critical in accounting for significantly more BPD features among young women, partially through underlying emotion regulation difficulties. Importantly, the direct effect from early childhood trauma remained significant after controlling for the indirect path from ED, indicating a unique role of trauma in affecting BPD. Interestingly, we discovered a declining trajectory in shame, guilt, anger and BPD features among this group of young women, adding new knowledge to the literature about the unique psychopathology during emerging adulthood.

5.1 Findings

5.1.1 Distinct Trauma Types & NA Aspects

Multiple regression results evidenced a consistent, significant effect of emotional abuse (EA) as opposed to the fact that other types of trauma did not appear significant in most of our models. Actually, emotional abuse significantly predicted BPD features across all our models, confirming the previous findings on the salient relationship between childhood emotional abuse and BPD symptoms (Carvalho et al., 2014; Kuo et al., 2015, Laporte et al., 2011;2012; Wota et al., 2014; Zhang et al., 2013). A recent meta-analysis of the effects of childhood trauma and BPD symptoms reported (1) a highest frequency of EA (as compared with other trauma types) among BPD individuals with a trauma history, as well as (2) a moderate, pooled effect size of EA (See Figure 18 , Yuan, Lee, Eack & Newhill, 2020). Commonly posited etiological explanations by past studies accounting for such effects of EA included emotion dysregulation, attachment disturbance and a dynamic biosocial interaction (Carvalho Fernando et al., 2014; Fossati, Gratz, Somma, Maffei, & Borroni, 2016; Kuo, Khoury, Metcalfe, Fitzpatrick, & Goodwill, 2015; Laporte, Paris, Guttman, Russell, & Correa, 2012).Indeed, this finding confirms Linehan's line of biosocial theory, in which BPD etiology is conceptualized as a dynamic interplay between inherited vulnerabilities and invalidating environments (M. Linehan, 1993a). According to CTQ scales, examples of emotional abuse comprised "People in my family called me things like stupid, lazy, or ugly" and "I thought my parents wished I had never been born"(Bernstein et al., 2003) . Those verbal assaults are typical of invalidating environments, where belittling of feelings, and suppression of negative emotions frequently happen. Therefore, emotional abuse is key to invalidating environments which elevates risk of BPD development and the severity of symptoms.

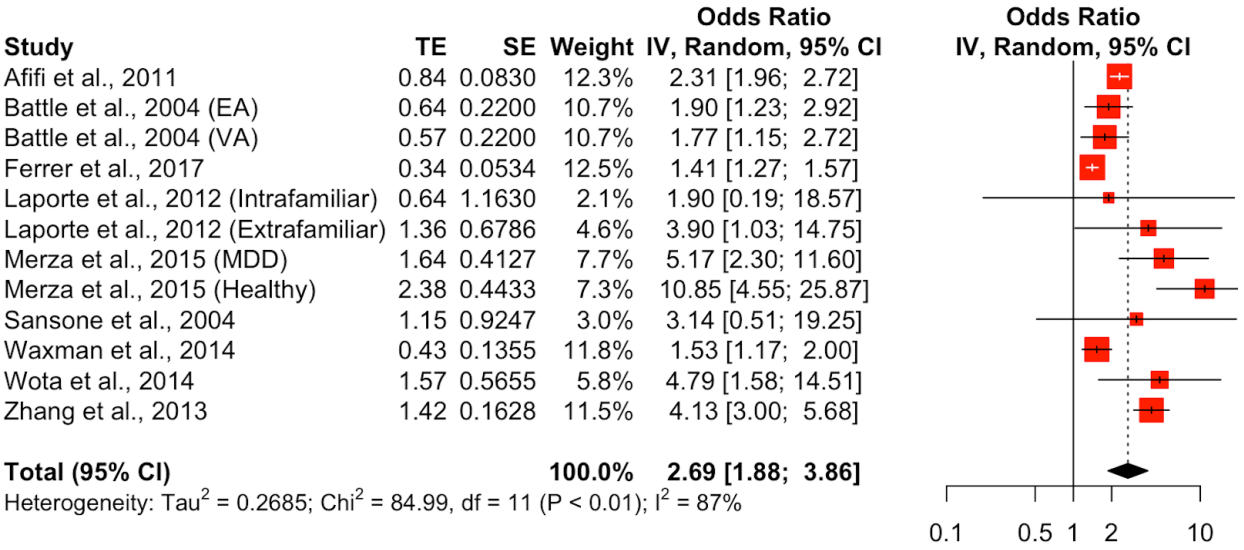


Figure 18[6] Pooled Effect Size of Association between Emotional Abuse and BPD Features.

More meaningfully, our results extended prior explorations by showing that several distinct NA-related forms of ED, such as proneness to anger expression, shame, and guilt, may play critical roles in relation to BPD symptoms.

In particular, two kinds of guilt and shame were shown to significantly associate with BPD features at baseline. One is a repair type of guilt that defines a tendency to act overly nice towards others due to feeling guilt, while the other is a negative self-evaluation type of shame which denotes an unfavorable appraisal of self as a result of feeling shame. These findings are in line with attachment theoretical explanations on a trauma-BPD link. Griffin and Bartholomew (1994)

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[6] See more in Yuan, Y., Hyunji, L., Eack, S., & Newhill, C. (2020, Manuscript Ready for Submission). A Systematic Review and Meta-analysis of the Association between Early Childhood Trauma and Borderline Personality Disorder with Critical Etiological Implications.

conceptualize adult attachment styles as follows: positive self and other (secure pattern), positive self and negative other (dismissing pattern), negative self and positive other (preoccupied pattern), and negative self and other (fearful pattern). Operating from that, guilt-repair is comparable to a positive-other dimension while shame-negative-self is analogous to negative-self dimension. Taken together, the function of these two ED forms resembles that of a preoccupied anxiety attachment, which has been consistently marked among people with early traumatic exposure and a later development of BPD (Baryshnikov et al., 2017; Battle et al., 2004; Fossati et al., 2016).In addition to shame and guilt, a proneness to anger expression was also detected in terms of correlation with high BPD symptoms. In fact, these three emotions (shame, guilt and anger) are all part of social emotions, which primarily arise in interpersonal context, such as by interacting with a close friend or observing a stranger. These social emotions will in turn motivate and influence how people's actions with others (Hareli & Parkinson, 2008). Socially maladaptive regulation of such emotions can undermine one's abilities to manage interpersonal relationships and vice versa. Such intertwined link between interpersonal context and social emotions, hence, is highly compatible with BPD symptomatology.

Inconsistent with a large body of BPD literature, our findings did not support a significant effect of sexual abuse on BPD. Consistent with other research (insert citations), our participants endorsed the lowest mean score of sexual abuse (*Mean*=7.56, *SD*=5.14) relative to other trauma types. Nevertheless, some child welfare research as well as studies utilizing national samples show that emotional abuse and physical neglect are indeed more common as opposed to sexual abuse (Afifi et al., 2011; Hengartner et al., 2013; Waxman et al., 2014).

Finally, our results supported a partial indirect effect of ED in terms of associating history of trauma with heightened BPD features using baseline data. This finding is consistent with

multiple lines of BPD literature. For example, Carvalho Fernando et al. (2014) investigated how unique aspects of emotion dysregulation might be differentially associated with distinct trauma types in accounting for higher BPD features. They found that emotional neglect was related to less adaptive emotion regulation abilities, whereas emotional abuse was associated with higher dysfunctional or maladaptive emotion regulation strategies. These associations did not emerge for other trauma types. It is notable that the effect of trauma remained significant even after accounting for ED, indicating a unique role of trauma in exacerbating BPD symptoms that is worthy of further investigation. Finally, we did not find a significant effect of trauma in predicting the trajectory of NA and BPD scores, due to an unexpected declining trend over time, which will be elaborated in the following section.

5.1.2 A Decline of NA and BPD Features & Emerging Adulthood

From the results of latent growth curve analyses, we discovered a decreasing trend in shame, guilt, anger and BPD scores over time. In comparison with NA measures, BPD features were highly stable among this group of young women, in which the rate of changes remained almost the same across three time points (there was a negligible decrease over time) and there was no significant difference in mean scores from wave 2 to 3. Although this was somewhat inconsistent with our original hypotheses, it actually added to the new knowledge on a nascent intersectional topic area that is focused on the biopsychosocial changes among a unique and yet previously understudied age group called emerging adulthood.

According to Arnett's conceptualization (2001), emerging adulthood is a critical developmental period (roughly from late adolescence to early twenties or approximately 18-25), during which young people experience considerable changes and explorations in a diversity of areas. In this period, there are a plethora of unpredictable instabilities and heterogeneities facing

this group, spanning categories of education, employment, sexual orientation, relationship status, living environments and more. In addition to instabilities, ambiguity is another key feature of this stage, leading to substantial explorations in one's roles in various facets of life (such as relationships with others and worldviews, etc.). Aside from external social changes, internal transitions also occur, such as neurobiological, emotional and cognitive transformations.

From the theoretical perspective of emerging adulthood (e.g., high ambiguity and heterogeneity), these aforementioned internal biopsychological changes do not necessarily follow a smooth, linear pattern; instead, a combination of continuity and instability might exist (Arnett, 2001). For instance, an emerging adult will continue to use the same ER strategy (as was used during childhood) to regulate anger such as rumination, and yet he/she can also discover different purposes and functions of that regulatory strategy (Schulenberg & Zarrett, 2006).

BPD pathology, like most forms of psychopathology, mirrors unsuccessful adaptions to the exchange between a person and his/her changing environments (Schulenberg & Zarrett, 2006). Compatible with BPD features, heightened impulsivity (such as risk-taking behaviors) and emotional lability are very typical during this developmental stage due to the demographic and social changes at multiple levels compounded by physiobiological factors. Therefore, in emerging adulthood theory, BPD development during this period should reflect an unstable fluctuation as well as more elevations in features such as impulsivity and emotion lability. Nevertheless, this line of theoretical proposition indeed is not quite consistent with our results nor past BPD literature that suggested a decline of BPD symptomatology in early adulthood in comparison with adolescence (Bornovalova et al., 2009) (Our results further discussed below).

As mentioned above, in contrary to theoretical propositions from emerging adulthood literature, our findings consistently point to a linear declining trend in all NA measures as well as

in BPD scores. Despite the deviation from emerging adulthood theory, our results were supported by some empirical BPD studies investigating adolescence and young adulthood. For example, some researchers reported a linear decline in BPD traits among young adults (especially towards the late twenties) in relation to adolescence (Bornovalova et al., 2009; Chanen et al., 2008; Lenzenweger et al., 2004). Conversely, other researchers discovered high dysregulation in regard to certain negative affects from late adolescence to EA, which include failure to regulate anger, over-suppression of fearful emotions, and significant passive regulation (e.g. avoidance) of sadness (Zimmermann & Iwanski, 2014). Some BPD researchers found worsening and unstable BPD features among adolescents and young adults (Wright et al., 2016). In the context of trauma, scholars indicated that early childhood adversities might add to the risks for developing mental health disorders during this critical developmental period, including personality disorders (Briggs-Gowan et al., 2010; Dvir et al., 2014; Lejonclou et al., 2014).

While extant findings on BPD features among emerging adults are rare and somewhat mixed, reports are relatively more consistent on the developmental changes in emotion regulation, and also more in line with our findings. Reportedly, in comparison with childhood and early adolescence, emerging young adults are more likely to (1) show an increase in awareness and insights on one's own emotions, as well as improved adaptive strategies of regulation; (2) display a growth in self-regulation in comparison to a decrease in external regulation (i.e., reliance on others); (3) utilize more varied regulatory strategies which are dependent on the specific nature of experienced emotions per se (such as anger, shame and guilt); and (4) a general improvement in adaptive regulatory strategies (Carstensen et al., 2003; Zimmermann & Iwanski, 2014). A systematic review on developmental trends in emotion regulation identified a focus on emotional self-efficacy (in both expressing positive affects and managing negative affects) during emerging

adulthood (Rawana et al., 2014). Further, there is some evidence that supports the gender difference during the period of emerging adulthood. For instance, one study indicates that males tend to use more passive regulation strategies such as avoidance whereas females use more strategies such as social support and rumination, which some argue might be due to different gender roles or ways of socialization (Saarni et al., 2007).

All in all, our findings in the respect of NA were highly compatible with past emotion regulation literature. Regarding BPD features, there existed different discussions among past BPD literature, some of which echoed with our results. Future studies can recruit different age groups in order to make a more rigorous comparison in various areas of psychopathology.

5.2 Limitations

One limitation of our study concerns the predominant use of self-report measures (with the exception of our interview measure of BPD features), which can lead to recall biases as noted in most studies utilizing self-report measures. In terms of participants, we had only females (though the sample is diverse with regard to race and socioeconomic status); hence, generalizability to other genders is limited. Further, our age range is restricted to emerging adulthood, hence generalizability to other developmental stages can be limited.

Finally, there might be undetected confounding effects that we did not sufficiently consider, which might be unique to this age group. As indicated in the previous sections, this age group is featured by high uncertainty and ambiguity. Current empirical studies are somewhat inconsistent in terms of reporting patterns and features pertaining to personality development during this developmental period. More longitudinal studies will be required to obtain more reliable knowledge. Despite the limitation, we comprehensively investigate emotion regulation

and several distinct forms of negative affects in affecting BPD symptoms during emerging young adulthood

5.3 Implications

5.3.1 Implications for Practice

Our findings generate implications for future practice with those with BPD suffering from early childhood trauma. First, early screening of trauma-related symptoms and trauma-informed perspective should be integrated into traditional BPD treatments. Second, emotional regulation difficulties should be targeted when treating people with trauma experience. Third, it can be especially useful to address trauma-related negative emotions, such as shame, guilt and anger.

For future social work practice, ongoing individual and group psychotherapy are still the keys to successful treatment for clients with BPD, and there are many different therapies with proven efficacy for this population (see a previous chapter on the interventions comparisons). All of these treatments work, in terms of decreasing suicidality, self-harm, and use of hospitals, emergency rooms, substances and medications; however, certain approaches work more or less well with different individuals, and all can be enhanced by adding a trauma-informed perspective and aspects of trauma-informed care. A trauma-informed perspective stems from the increasing recognition of the significant incidence and prevalence of trauma affecting a wide range of individuals, families and communities (J. Herman, 1992). The development of trauma-informed interventions has been guided by a recognition of the complex web of neurological, biological, psychological, and social factors shaping the effects of trauma and understanding the various paths for recovery.

Regarding trauma care relevant to BPD populations, as inspired by our study, a trauma-informed emotion regulation skills training can potentially include topics such as (1) mindfulness of trauma-related emotions, (2) validation of negative emotions, and (3) reappraisal of negative experiences. Furthermore, for BPD individuals without a diagnosis of trauma and stressor-related disorders, facilitating a supportive empathic dialogue at minimum would promote early and effective screening for trauma symptomatology. As noted earlier, emotional abuse and emotion-related invalidation were highly prevalent among BPD populations (with or without a co-occurring trauma diagnosis); therefore, emotion regulation skill training targeting emotional invalidation can potentially lead to effective results.

For those with active co-occurring diagnoses, trauma informed treatments work by integrating traditional BPD psychotherapies (such as DBT) with trauma-processing narratives, trauma-informed psychoeducation sessions, and exposure-based techniques (such as imaginal exposure, in vivo exposure and prolonged exposure). These approaches differ from traditional BPD treatments in that they extend beyond a here-and-now focus (as in some psychodynamic and behavioral therapies), and further address the past traumatic experiences and specifically target trauma-related negative emotions through narratives and exposure protocols (Aase & Sagvolden, 2005; Harned et al., 2012, 2014; Pabst et al., 2014; Steuwe et al., 2016). Last but not least, taking control of therapy-interfering or other high-risk behaviors can be critical before implementing any type of trauma care or related treatments. Crises such as high levels of life-threatening (e.g. suicidal attempts) and/or therapy-interfering behaviors (e.g. frequent rescheduling or being late for sessions) before processing traumatic memories and emotions, given that the presence of aforementioned crises might prevent the individuals from effectively discussing and managing emotion about the trauma, or they may not have the skills yet to regulate the emotions. To this aim,

it will be necessary to conduct an early evaluation of the risks, establish a trusting therapeutic relationship as well as develop action plans to ensure safety (M. M. Linehan et al., 2012)

5.3.2 Implications for Policy

A trauma-informed perspective in the treatment of BPD should be integrated into how services are designed and the policies that drive them. Despite increasing public attention, translation of a trauma-informed perspective into legislations and policies is still of high uncertainty, and little is known regarding the status and progress of trauma-informed policies and public systems (Bowen & Murshid, 2016; Purtle & Lewis, 2017). Purtle and Lewis (2017) reported that policies and laws targeting trauma increased greatly since the first bill mentioning trauma-informed care was introduced in 2015. In addition, from their results, youth were the most commonly targeted population; and sectors conducting the services comprised mainly of juvenile courts, child protective services and mental health facilities.

In other words, those who received significant attention from the criminal justice system or clinical facilities would benefit most from the current trauma-informed care, indeed indicating a lack of proactive approach in current policies and services. In order to address that, policies should effectively target a wider range of sectors by taking more proactive action among younger populations. Informed by our results, some young adults might not necessarily display severe mental health symptoms, and yet may still suffer from the aftermath of early childhood adversities. In other words, these individuals with low clinical symptoms may not qualify for or do not show up at traditional treatment programs; however, their needs cannot be neglected. Therefore, having outreach programs and community services might be more effective to target such population. Further, policies should effectively reflect trauma-informed care principles such as safety, empowerment, trustworthiness, transparency, choice, and more (Bowen & Murshid, 2016).

Last but not least, support by the client's family for his or her treatment and recovery is very important for treatment adherence and success, and service providers must recognize that caring for someone with BPD can produce trauma in family members. Self-help advocacy groups, such as the National Alliance on Mental Illness (NAMI) and the National Education Alliance for Borderline Personality Disorder (NEABPD) can provide critical support and psychoeducation for families.

5.3.3 Implications for Research

Future research can compare the differential effects between momentary emotional reactions and stable traits in exacerbating BPD symptoms after traumatic exposure in order to gain more knowledge about the specifics of ED. Moreover, different age groups can be recruited (such as adolescents and adults in the late twenties) in order to further advance the current knowledge on different developmental ages. In addition, research studies can utilize prospective measures and causal inference techniques to improve the research design. For example, although repeated measures of actual traumatic experiences may not be feasible, assessments of trauma-related symptoms pre and post interventions can be easily administered; therefore a causal inference can be subsequently conducted using a regression-discontinuity design (by assigning a cut-off threshold above or below where an intervention happened, and differencing the two adjacent points next to the cut-off point) (Murnane & Willett, 2010). Finally, to correct the recall biases of self-report surveys, additional data sources might be obtained from substantiated child abuse cases in order to cross reference with the self-reported trauma history.

5.4 Conclusion

Our results contribute to new knowledge on the underlying factors driving the connection between early childhood trauma and BPD symptomatology, from the perspective of emotion dysregulation. Implications from our findings extended prior explorations by showing that several distinct NA-related forms of ED, such as proneness to anger expression, shame, and guilt, may serve as critical channels driving the significant link between emotional abuse and BPD symptoms. More importantly, our findings would potentially lead to novel trauma-informed treatments effectively targeting multiple ED forms among people with BPD. Finally, we contribute critical new information to the nascent topic area on emerging adulthood.

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