Modeling the Influence of Childhood Trauma on Rate of Symptom Change Among Psychiatric Inpatients

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Modeling the Influence of Childhood Trauma on Rate of Symptom Change Among Psychiatric Inpatients

A Dissertation Presented

by

ALESSANDRO T. PISELLI

Submitted to the Graduate School of the University of Massachusetts Amherst in partial fulfillment of the requirements for the degree of

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Modeling the Influence of Childhood Trauma on Rate of Symptom Change Among Psychiatric Inpatients

A Dissertation Presented

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I would like to begin by thanking my advisor. Richard Halgin has been an unwaivering source of guidance, inspiration, and support since I first stepped into his office as an undergraduate in the fall of 1998. His thoughtful as a teacher has served as the model toward which I strive. His guidance as a mentor has helped me to navigate the many barriers to training as a clinical scientist. His feedback as an advisor has introduced welcome clarity in both my writing and clinical work. Now, over fourteen years later, his friendship is helping me to create a career in which I can share those same gifts with others.

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ABSTRACT

MODELING THE INFLUENCE OF CHILDHOOD TRAUMA ON RATE OF SYMPTOM CHANGE AMONG PSYCHIATRIC INPATIENTS

SEPTEMBER 2013

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Clinical wisdom suggests that adults with histories of childhood trauma will have difficulty engaging in psychotherapy. I examined the relationship between early childhood trauma and treatment response in the form of rate of symptom decline among a group of 202 adults recruited into the longitudinal Austen Riggs Center Follow-Along Study. Participants were recruited at admission to the hospital and provided extensive demographic and clinical data at baseline, including retrospective recall of childhood traumatic experiences using the Traumatic Antecedents Interview (TAI) and narrative responses to the Relationship Anecdote Paradigm (RAP) used to generate ratings on the Social Cognition and Object Relations Scale (SCORS). Participant symptoms were assessed at baseline and again every six to eight months for up to four years using the Symptom Checklist-90-Revised (SCL-90-R). Hierarchical Linear Modeling (HLM) was used to perform growth curve modeling of the symptom change data, which demonstrated an overall linear rate of decline and significant unexplained variability.
The hypothesis that degree of childhood trauma would predict rates of symptom change failed to receive support, as did other related hypotheses. Only participant age predicted differences in rates of symptom decline, with younger participants’ symptoms declining more rapidly than those of older participants. The findings indicate the following: (1) symptom severity tends to decline linearly after hospital admission, (2) individual rates of symptom change vary considerably, and (3) rates of symptom decline may slow as people age. Implications for clinical research and practice are discussed.
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CHAPTER I

MODELING THE INFLUENCE OF CHILDHOOD TRAUMA ON RATE OF
SYMPTOM CHANGE AMONG PSYCHIATRIC INPATIENTS

A. Introduction

Timely recovery following a psychiatric crisis requires responsive treatment and
social support. The aid of mental health professionals, friends, and family can mitigate
the severity of psychiatric symptoms and hasten a return to normal functioning (Chou &
Chronister, 2012; Williams & Mfoafo-M’Carthy, 2006). Yet the very symptoms of
psychiatric illnesses often interfere with access to support and recovery. For example,
specific phobias involve intense fear that causes a person to avoid the very type of
exposure that would mitigate the fear response. Similarly, the consequences of early and
chronic psychological trauma create barriers to treatment and support. Although
therapists are attuned to this complexity (Knight, 2009), few studies have successfully
demonstrated the links between childhood trauma and response to treatment.

The present study explored the relationship between early childhood trauma and
symptom change among adults during and after inpatient psychiatric hospitalization.
Specifically, the study examined whether adults who reported early, chronic, varied, or
severe childhood trauma experienced slower reductions in the severity of their psychiatric
symptoms. In addition, the study assessed whether impairments in interpersonal
functioning could account for the proposed relationship between childhood trauma and
response to treatment in adulthood. The following sections provide selected reviews of
the literature on: (1) the nature and incidence of child maltreatment, (2) the consequences
of early life trauma on development and adult functioning, (3) the relationship between
childhood trauma and treatment response, and (4) methodological issues in the measurement and study of childhood trauma and treatment response.

B. Childhood Trauma

The present discussion focuses on traumatic events during childhood such as exposure to abuse, neglect, and domestic violence. The U. S. Department of Health and Human Services Fourth National Incidence Study of Child Abuse and Neglect (NIS-4; Seddak et al., 2010) differentiates three types of abuse: physical, sexual, and emotional; and three types of neglect: physical, emotional, and educational. Sadly, children who experience one form of maltreatment are much more likely to experience others because categories of abuse and neglect typically co-occur within families (Baker & Maiorino, 2010). As a result, trauma researchers have difficulty isolating the effects of each category of traumatic experience and tend to group together all forms of child maltreatment.

The prevalence of child maltreatment is alarming, especially given that reports likely underestimate the actual frequency with which children are abused and or neglected. According to the NIS-4 (Seddak et al., 2010), one in 58 children experienced harmful maltreatment and another one in 25 was at risk for maltreatment during a one year period during 2005 and 2006. Of children who experienced demonstrable harm in that year, 44% experienced abuse and 61% experienced neglect.

Childhood trauma has a destructive effect on the individual trauma survivor. The U. S. Department of Health and Human Services, Child Welfare Information Gateway (2008) reports that child maltreatment results in a wide range of negative physical, psychological, and behavioral outcomes including brain injury, psychiatric illness,
substance abuse, delinquency, difficulty in intimate relationships, and death. Scholars agree that individuals who experience psychological trauma experience various negative consequences that share key features. Knafo (2004) summarized the leading perspectives by outlining four common features of posttraumatic disorders: (1) unprocessed or dysregulated affect; (2) dissociated memories of the traumatic experience; (3) physiological reactivity that persists at a nonverbal level; and (4) intense fear associated with experiences of helplessness and threat. As a result, the traumatized individual may vacillate between states of numb separation from feeling and intense reexperience of the trauma.

Though researchers tend to group many forms of childhood trauma under the term maltreatment, some points of clarification are warranted. First, the term *trauma* itself can refer to either an experience or a consequence. Thus a person could be said to have experienced a traumatic event and then suffered a resulting psychological trauma. Second, trauma can take many forms. The NIS-4 identified six categories of child maltreatment (Sedlak et al., 2010), each of which could be applied to countless specific forms of abuse or neglect. Third, childhood trauma may consist of a single terrifying event or persistent abuse and neglect. The duration of traumatic events are typically classified as either *acute*, meaning limited in duration, or *chronic*, meaning lasting in duration. It is important to note that the same terms can also be used to describe the duration of the resulting syndrome (i.e., a chronic syndrome or an acute syndrome). Fourth and finally, the timing of traumatic experiences can vary. Childhood trauma can occur at any age and any stage of development.
Some suggest that a person’s age at the onset of traumatic experience has bearing on the scope and nature of the resulting syndrome. Freud (Freud & Strachey, 1964) and Erikson (1950) suggested that exposure to trauma during earlier stages of development would result in more fundamental, and therefore more severe, psychological impairments. Roth, Newman, Pelcovitz, van der Kolk, and Mandel (1997) found evidence that the risk of developing a severe psychiatric disorder is determined by a complex relationship between the type, duration, and onset of abuse. They argued that early onset is likely related to more chronic and pervasive patterns of abuse.

Some scholars emphasize the duration of exposure to trauma as the factor that determines the resulting psychopathology. In her pioneering work on childhood trauma, Terr (1991) distinguishes between the consequences of acute and chronic trauma, suggesting that children who experience extended abuse and neglect develop a distinct symptom pattern that includes dissociation and rage. Similarly, Streeck-Fischer and van der Kolk (2000) assert that chronic exposure to trauma may result in fundamental problems with self-regulation that contribute to impairment across many spheres of functioning.

Among psychological disorders, the *DSM-IV-TR* diagnosis of Post-Traumatic Stress Disorder (PTSD; American Psychiatric Association, 2000) provides a framework for identifying a post-traumatic syndrome. Criterion A requires that a person has experienced an event involving actual or perceived threat of death or serious injury and intense emotions such as fear, helplessness, or horror. The experience may be brief or prolonged, and the severity of the disorder is presumed to be greater when the traumatic event is longer in duration, more severe, and experienced more directly. The resulting
syndrome is defined by criteria organized into the following three symptom categories: (B) intrusion of traumatic memories, (C) psychic avoidance or numbness, and (D) hyperarousal to danger. Criteria E and F require at least one month of significant functional impairment and define the syndrome as acute if lasting less than three months or chronic if lasting more.

In May 2013 the American Psychiatric Association (APA) will publish the fifth edition of the DSM (DSM-5; APA, 2013). Among planned revisions, the diagnostic criteria for PTSD will be expanded to describe the syndrome as it appears in children (Friedman, Resick, Bryant, & Brewin, 2011). In the new criteria, children may demonstrate the re-experiencing of symptoms (Criterion B) in play or frightening dreams, though traumatic content may not be apparent in the latter. Some avoidance and numbing symptoms (Criterion C) will be moved to a new criterion designating negative alterations in cognition and mood. Children will need to have experienced at least two symptoms to meet the threshold for this new criterion, as well as for the hyperarousal criterion (Criterion D). (The threshold for these criteria will be three symptoms for adults.) Beyond these planned changes, Scheeringa, Zeanah, and Cohen (2011) recommend an alternate algorithm for PTSD in children which (1) eliminates the requirement for the traumatic event to have involved the experience of intense fear, helplessness or horror (Criterion A2); (2) reduces the threshold for the new cognition and mood criterion to one symptom; (3) allows for variability in the onset of symptoms given chronic exposure to trauma; and (4) delineates more developmentally sensitive and specific symptoms.
Reports of childhood trauma are more common in clinical samples. Baker and Maiorino (2010) compiled data on the adult recall of childhood maltreatment from studies utilizing the Childhood Trauma Questionnaire (CTQ). The CTQ is a widely used instrument that assesses the frequency with which adults report childhood experiences of physical abuse, sexual abuse, emotional abuse, physical neglect, and emotional neglect. The investigators found that, in the general population, 15% of adults reported experiencing severe abuse and 13% reported experiencing neglect. In contrast, they found higher rates in clinical samples where 32% reported severe abuse and 19% reported neglect.

Rates of childhood maltreatment are particularly high among individuals diagnosed with certain psychiatric diagnoses such as Borderline Personality Disorder (BPD). In a widely cited study, Herman, Perry, and van der Kolk (1989) found that reports of childhood abuse were most common and severe among subjects diagnosed with BPD, as compared to those diagnosed with other personality and mood disorders, and those with less severe borderline traits. Of those diagnosed with BPD in their sample, 71% reported having experienced physical abuse, 68% reported having experienced sexual abuse, and 62% reported having witnessed serious domestic violence.

In summary, child maltreatment can take many forms and is unfortunately common. Childhood trauma can result in severe impairment that affects many spheres of functioning and endures into adulthood. The diagnosis of PTSD outlines typical pathological responses to childhood trauma in adulthood, and an upcoming revision to the diagnostic system will expand the symptom criteria to more accurately represent the experiences of children with the disorder. Adults in treatment more often report histories
of childhood trauma, and such reports are especially common for adults with certain
diagnoses such as BPD. Theory suggests that the timing and duration of trauma in
childhood may play an important role in determining the severity of the subsequent
psychiatric problems.

C. Complex or Developmental Trauma

In recent decades, a number of scholars have argued that the consequences of
chronic childhood trauma are more complex and pernicious than the PTSD diagnosis
suggests (e.g., Cloitre et al., 2009; Roth et al., 1997; Streeck-Fischer & van der Kolk,
2000; van der Kolk, 1997; van der Kolk, 2002; van der Kolk & Fisler; 1994; van der
Kolk, Roth, Pelcovitz, Sunday, & Spinazzola, 2005). In their introduction to a special
issue of the Journal of Traumatic Stress, van der Kolk and Courtois (2005) argued that
chronic forms of trauma result in systemic impairments that are not reflected in the PTSD
symptom criteria. Some scholars have begun to use the term complex trauma to describe
the enduring syndrome that follows exposure to persistent psychological and
physiological crisis (Courtois, 2008; Gingrich, 2009).

In discussing upcoming revisions to the DSM-IV, van der Kolk and colleagues
have lobbied for the creation of a diagnosis that captures the impairments seen in
complex trauma (van der Kolk, 2005; van der Kolk & Courtois, 2005; van der Kolk et al.,
2009). A few years ago, van der Kolk and Courtois (2005) argued for the adoption of a
supplementary diagnostic category named Disorders of Extreme Stress, Not Otherwise
Specified (DESNOS), which would describe a syndrome consisting of a wide range of
impairments including disruptions in emotional states, sense of self, consciousness and
memory, relationships, and physical health. Even those who believed the change was not
necessary (e.g., Kilpatrick, 2005) conceded that chronic trauma is associated with greater
degrees of impairment. More recently, van der Kolk and colleagues (van der Kolk, 2005;
vander Kolk et al., 2009) proposed the creation of a new diagnostic category,
Developmental Trauma Disorder (DTD), intended to conceptualize childhood trauma
more accurately by placing it in a developmental framework. The DTD diagnosis would
differ from PTSD in a number of ways, including the following: (1) allowing for the
absence of caregiving (e.g., neglect) to serve as a trauma, (2) specifying problems with
affect modulation and awareness, (3) capturing a wider array of behavioral problems
(e.g., self-harm and inhibited exploration), (4) highlighting fundamental problems in
relationships (e.g., with trust and reciprocity), and (5) defining the syndrome as chronic
in that symptoms must last for at least six months (van der Kolk et al., 2009).

According to a review of the relevant literature (Margolin & Vickerman, 2007),
scholars believe that chronic trauma creates dysfunction in six domains: (1) affect
regulation, (2) cognition and consciousness, (3) self-concept, (4) behavioral control, (5)
interpersonal relationships, and (6) biological functioning. Victims of chronic forms of
childhood trauma, such as persistent maltreatment, often vacillate between affective
constriction and reactivity, therefore limiting their ability to process and manage
emotions in a variety of settings. They also become increasingly sensitized to real or
perceived signals of danger, and become preoccupied with intrusive memories or worries.
Chronically maltreated children manage these disruptive thoughts and feelings by
becoming either hypervigilant to, or disconnected from, their internal experiences and the
outside world. As a consequence, they may become impulsive or aggressive, or may
withdraw from social interactions. Finally, mounting evidence suggests that exposure to

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trauma causes high levels of neurological arousal which, when chronic, will disrupt a child’s physiological regulation and neurodevelopment.

Van der Kolk and Coutois (2005) theorize that the core feature of complex trauma is an underlying “breakdown in the capacity to regulate internal states” (p. 386). More specifically, these and other authors argue that chronic trauma undermines a developing child’s ability to regulate affect (Cloitre et al., 2009; Streeck-Fischer & van der Kolk, 2000; van Dijke, 2008). Streeck-Fischer and van der Kolk (2000) describe children who experience repeated and multiple forms of abuse as “literally [being] ‘out of touch’ with their feelings” (p. 905). They go on to explain that, as a result, these are children who “act instead of plan… take, rather than ask” (p. 905), and fear change because their familiar experience of terror seems safer.

McDermott (2004) proposed that a developmental perspective is needed in order to understand the complex causal influences at play in childhood trauma. He characterized trauma as a developmental insult that deflects a child’s normative growth trajectory. From this perspective, a child with sufficient baseline health, individual strengths, family support, and social resources has the best chance of returning to normal functioning following acute trauma. McDermott argued that chronic trauma is associated with even worse outcomes because exposure to repeated abuse and neglect often reflects larger failures in an otherwise protective social or family system.

Margolin and Vickerman (2007) suggested that the consequences of childhood trauma compound by interfering with a child’s ability to access protective external supports. For example, affect dysregulation can lead to interpersonal aggression and withdrawal that may result in punishment and rejection rather than care and support.
Without considerable external support from caregivers, a child who struggles to self-regulate will also have difficulty following rules, accepting limits, expressing needs, and receiving comfort.

The pathological consequences of chronic trauma can become particularly intractable when disrupting a child’s basic capacity to relate to others. Object relations theory provides a framework for understanding the development of relational capacities. According to early object relations theorists such as Klein (1946), infants translate early experiences with caretakers into internal objects, which are basic and lasting representations of the self and others as entities that are both separate and connected. Winnicott (1953), another early object relations theorist, suggested that experience with a good enough mother, one who recognizes and meets a child’s basic needs, enables the infant to develop an understanding of the real world without being overwhelmed by frustration and anxiety. Winnicott believed that severe disruption of the mother-child relationship interferes with the process of ego-development and cripples the child’s ability to connect with others, as well as with him or herself. Furthermore, he believed that early impairments in object relations could result in severe psychopathologies such as psychosis and personality disorder (Winnicott, 1965).

Attachment theory offers another framework for understanding relational development and dysfunction. Bowlby (1960) created the foundations for attachment theory, proposing that a child learns to feel safe and secure when his or her mother is responsive to basic needs. Bowlby’s theories were extended by Ainsworth (Ainsworth, Blehar, Waters, & Wall, 1978), who studied mother-child interactions and characterized three distinct attachment styles: (1) secure, (2) anxious/avoidant, and (3)
anxious/ambivalent. Later, Crittendon and Ainsworth (1989) described a fourth category of children whose attachment style had become disorganized. From an attachment perspective, parental neglect (i.e., inconsistent or unresponsive caretaking) predisposes a child to develop an ambivalent attachment style. In contrast, parental abuse (i.e., hostile or rejecting caretaking) contributes to the development of an avoidant attachment style. These theorists believed that the development of a disrupted attachment style sets the stage for later relational problems in adulthood.

Contemporary scholars have drawn a connection between disrupted attachment and the fundamental dysregulation seen in complex trauma (Bailey, Moran, & Pederson, 2007; Leahy, Pretty, & Tenenbaum, 2003; Liotti, 2004; Pearlman & Courtois 2005; van der Kolk & Fisler, 1994; Williams, 2006). For example, evidence is currently emerging that victims of childhood maltreatment demonstrate a heightened neurological stress response in adulthood (Carpenter et al., 2010). Streeck-Fischer and van der Kolk (2000) assert that “the mother plays the critical role of psychoneurobiological regulator of the child’s affective states” (p. 906). They explain that children who experience chronic maltreatment and disorganized attachment to parents or caregivers will suffer unmet basic needs, and will therefore learn to ignore internal regulatory cues. Streeck-Fischer and van der Kolk predict that infants who develop avoidant attachment styles will have difficulty obtaining care or relief through relationships, while those who develop ambivalent attachment styles will become overly reactive to their internal states. In either case, theory suggests that disrupted attachment limits one’s abilities to regulate affect and maintain healthy relationships.
Modern conceptualizations of Borderline Personality Disorder (BPD) provide a biopsychosocial model for the development of dysregulated affect and problems with attachment and relating. The *DSM-IV-TR* (APA, 2000) defines BPD as a pervasive disorder characterized by both interpersonal and intrapersonal instability, as indicated by the presence of at least five of the following nine diagnostic criteria: (1) frantic efforts to avoid abandonment, (2) a pattern of unstable and intense relationships, (3) identity disturbance, (4) self-damaging impulsivity, (5) recurrent suicidality, (6) affect instability, (7) chronic feelings of emptiness, (8) dysregulated anger, and (9) paranoid ideation or dissociative symptoms. In an influential work on the disorder, Linehan (1993) suggested that emotional dysregulation is the central feature of BPD, creating a parallel between the diagnosis of BPD and recent conceptualizations of complex trauma.

Citing widely acknowledged problems with the current diagnostic criteria for BPD, Fruzzetti, Shenk, and Hoffman (2005) argued the disorder arises from the interaction of vulnerabilities in the individual and invalidation from the environment. This transactional model highlights the developmental nature of problems with self-regulation, and explains how exposure to childhood trauma might contribute to the etiology of BPD (Herman et al., 1989; McDermott, 2004; Sansone & Sansone, 2007). Some argue that abusive or neglectful caregiving results in impoverished object representations and sets the stage for the inter- and intra-personal problems seen in the disorder (Bradley & Westen, 2005; Kernberg & Michels, 2009). While childhood trauma does not necessarily result in BPD, the disorder provides a developmental framework that connects early experiences with caregivers to lasting problems managing self-states and relationships.
In summary, scholars argue that chronic childhood trauma contributes to a wide variety of negative outcomes that interact with one another, disrupt development, and produce complex and enduring syndromes. A child who experiences maltreatment is therefore at risk for developing deficits in affect regulation, basic object relations, and interpersonal attachment. These consequences of these deficits compound by impeding the child’s access to external supports, thus establishing pathologies that resist change. The diagnosis of BPD demonstrates the developmental pathways that may connect early trauma, self-regulation, relational capacities, and lasting psychiatric disorder in adulthood.

D. **Complex Trauma and Response to Treatment**

State of the art treatment for PTSD includes various therapies and techniques. Foa, Keane, Friedman, and Cohen (2009) identify the first-line treatments for PTSD in adults and children based on the existing evidence base. They find support for treatments including: cognitive-behavioral therapy (CBT), psychopharmacotherapy, group therapy, psychodynamic therapy, psychosocial rehabilitation, and couples and family therapy. They also find support for specific techniques including eye movement desensitization and reprocessing (EMDR), the targeted use of hypnosis, and creative arts therapies. Foa and colleagues highlight the importance of providing treatment for comorbid disorders such as depression and substance abuse. They point out that less is known about best practices in the treatment of more complex forms of PTSD.

Courtois (2008) reviewed the clinical and empirical knowledge base to offer recommendations for the assessment and treatment of complex trauma. She cautions that the syndrome involves a multiplicity of problems which complicate treatment, requiring a
multimodal approach that addresses the biopsychosocial, spiritual, developmental, and relational features of the syndrome. Such problems include hypervigilance to perceived danger, poor self-regulation, difficulty tolerating traumatic memories, shame and anxiety regarding trauma, and difficulty forming stable interpersonal attachments. Drawing on the work of Herman (1997), Courtois outlines a three stage model for the assessment and treatment of complex trauma: (1) establish safety within the therapeutic relationship and ensure the client has a basic set of coping skills, (2) explore and process traumatic memories and emotions, and (3) consolidate gains and develop a life that is no longer ruled by trauma. The model prioritizes the development of the client’s sense of safety and ability to self-regulate as prerequisites for the difficult work of revisiting and overcoming a history of trauma. Courtois suggests that the therapeutic relationship serves a key role in this process by providing the client with “an opportunity to rework attachment difficulties from the past within the therapeutic context” (p. 93).

Complex trauma can result in a number of secondary impairments that, if unaddressed, may reduce the effectiveness of treatment. For example, van der Kolk, van der Hart, and Burbridge (2002) note that many people with traumatic histories are prone to recreating traumatic relationships in their adult lives. In another work, van der Kolk (2004) points out that survivors of complex trauma can experience “automatic physiological responses” (p. 278) that remain unchanged even after successful treatment. Van Dijke (2008) highlights the need to address co-occurring mood and substance abuse disorders that result from many years of problems with affect regulation. Lastly, Gingrich (2009) calls attention to the many forms of dissociative defense (e.g., amnesia, derealization, and identity confusion) that trauma survivors use to cope with their
experiences. All of these secondary problems maintain the traumatic syndrome and can inhibit progress in treatment.

Like Courtois (2008), a number of scholars give special importance to the quality of the client’s bond to the therapist (Leahy et al., 2003; Liotti, 2004; Pearlman & Courtois, 2005; Williams, 2006). The quality of the therapeutic relationship is especially important in work with chronically traumatized individuals for two reasons. First, problems with attachment may limit a client’s ability to engage in an adaptive working alliance (Dalenberg, 2004). Second, the difficult task of forming and maintaining a secure attachment may have a curative effect in and of itself (Pearlman & Courtois, 2005). Williams (2006) asserts that attachment to an abusive or neglectful caregiver can be devastating to a child’s sense of security and capacity to relate to others. She explains that, by offering a secure relationship and accurate mirroring, the therapist can help the traumatized client to integrate his or her sense of self, and to experience the hope that he or she can receive care and relief. Others (Leahy et al., 2003; Liotti, 2004) argue that problems with disorganized attachment can be addressed through focused work on the therapeutic relationship.

Given the complicating factors outlined above, people who have experienced chronic trauma are sometimes described as treatment resistant, and believed to require services adapted to their specific needs (Fromm, 2006). Following decades of research on the treatment of combat veterans that demonstrated limited efficacy, Shalev (1997) concluded that the treatment model needed revision in light of the apparent intractability of the veterans’ symptoms. Despite recent improvements in treatment, Spinazzola, Blaustein, and van der Kolk (2005) criticized the corpus of research on the treatment of
PTSD for omitting subjects with comorbid disorders who experience worse outcomes and are more representative of those diagnosed with the disorder. Fromm (2006) suggests that treatment resistance may be the result of inter-generational trauma that confuses a client’s ability to relate to people in positions of authority, thus interfering with many standard forms of treatment.

Shapiro and Plakun (2009) argue that treatment resistant patients are “doomed to chronic crisis” (p. 286) in standard short-stay hospitals and low-intensity outpatient settings. They suggest this patient population needs treatment settings that are flexible enough to allow patients to be in charge of their recovery and intensive enough to challenge long-standing patterns of maladaptive relating and behavior. Yet, Geller (2006) notes that long-term intensive treatment settings have become rare in the United States due to problems with cost-containment and outcome focus, and are now limited to only a few private psychiatric hospitals.

In summary, complex trauma impairs not only functioning, but also the capacity to engage in treatment. The presence of impairment across multiple domains interferes with treatment effectiveness and necessitates a more strategic approach to therapy. Among other difficulties, impaired relational and attachment capacities undermine a client’s ability to form a secure alliance with the therapist. Individuals who suffer complex trauma may require specialized treatment settings to address such barriers effectively. Yet, many hospitals that offer such specialized treatment have closed, and those that remain are facing the challenges of managing cost and focusing treatment more effectively.
E. **Methodological Issues in the Study of Childhood Trauma**

Two methodological issues warrant discussion before proceeding to the hypotheses of the present study. First, the assessment of childhood trauma among adults has proven difficult due to a number of reporting problems. Second, attempts to establish causality between childhood trauma and adult psychopathology may over-simplify complex phenomena that develop over many years. The following section briefly summarizes the challenges related to these methodological issues.

The retrospective measurement of childhood trauma poses a number of significant challenges. Survivor reports of traumatic experiences may be inaccurate, incomplete, or unavailable due to avoidance, pathological forgetting, emotional numbing, social withdrawal, or extreme sensitivity to the recall of traumatic memories (Flack, Litz, Weathers, & Beaudreau, 2002). As a result, adult reporters may fail to fully disclose their traumatic histories, or may avoid situations in which they could be asked to make such disclosures. Despite controversy regarding the validity of recovered traumatic memories (Laney & Loftus, 2008; Loftus, 1993; Ornstein, Ceci, & Loftus, 1998), research evidence suggests that trauma victims are able to recall their traumatic experiences with accuracy and stability (Drapeau & Perry, 2004; Maughan & Rutter, 1997; Paivio, 2001). Questions about repression aside, survivors are likely to want to avoid re-experiencing the thoughts and feelings associated with experiences of trauma (Christianson & Lindholm, 1998; McNally, 2003). As a result, a sample of adults asked to report on childhood trauma may under-report traumatic experiences and under-represent populations of interest.
Efforts to identify causal links between childhood trauma and adult psychopathology have been frustrated by complexity on both sides of the suggested connection. In a critique of the preceding corpus of psychological science, Richters (1997) argued that human development is an open system in which many different factors can result in a given outcome (equifinality), and that any single factor can contribute to the occurrence of a variety of outcomes (multifinality). Research on the consequences of childhood trauma offers evidence of such open system complexity. For example, studies examining the development of personality dysfunction demonstrate that factors such as gender, resilience, and ego functioning determine the pathway between childhood maltreatment and a range of possible personality disorders (Beachaine, Klein, Crowell, Derbidge, & Gatzke-Kopp, 2009; Kim, Cicchetti, Rogosch, & Manly, 2009). While diagnostic complexity is common in people diagnosed with PTSD, existing treatment research excludes those with co-occurring diagnoses, a constraint that threatens the utility and generalizability of the evidence base (Spinazzola, Blaustein, & van der Kolk, 2005). The search for causal links between childhood trauma and negative outcomes requires a developmental perspective that accounts for the influence of many contributing factors and measures a range of potential interrelated outcomes.

F. **The Present Study**

The present study examined the relationship between childhood trauma and response to treatment. Adults hospitalized for intensive inpatient psychiatric treatment provided detailed reports of childhood trauma and longitudinal symptom severity. Response to treatment was operationalized as the rate of decline in psychiatric symptoms over the months and years following hospital admission. The primary analyses
investigated the relationship between reports of childhood trauma and rates of decline in symptom severity. The following hypotheses guided evaluation of the proposed relationships:

**Hypothesis 1.** Change in symptom severity following psychiatric hospitalization would fit a model describing slowing quadratic decline.

**Hypothesis 2.** Reports of early, chronic, varied, and severe childhood trauma would be associated with slower rates of decline in symptom severity.

**Hypothesis 3.** Impairments in object relations would be associated with slower rates of decline in symptom severity.

A series of secondary analyses explored the relationships between the extent of childhood trauma and other study variables. These exploratory analyses were intended to examine the relationships between childhood trauma, psychiatric diagnosis, and impairment in object relations. The following hypotheses guided exploration of the expected relationships:

**Hypothesis 4.** Reports of more severe childhood trauma would be associated with greater impairment across all object relations dimensions.

**Hypothesis 5.** Reports of more severe childhood trauma would be associated with greater likelihood of being diagnosed with posttraumatic stress disorder (PTSD) and borderline personality disorder (BPD).
CHAPTER II

METHOD

A. Setting

Participants for the present study were admitted for treatment at the Austen Riggs Center (ARC), a psychiatric hospital and residential treatment facility in Western Massachusetts that serves adults with treatment-refractory psychiatric disorders. The ARC provides voluntary, long-term treatment across a continuum of services including inpatient hospitalization, residential treatment, and step-down programs (Austen Riggs Center, 2007). Treatment consists of intensive psychodynamic psychotherapy (4 sessions per week), psychopharmacologic intervention, insight-oriented group therapy, family therapy, and an activities program designed to support rehabilitation. The ARC is an internationally recognized institution of historical significance, which has been staffed by prominent scholars such as Erik Erikson, David Rapaport, and Margaret Brenman-Gibson. The ARC is now one of the few remaining treatment facilities that provide intensive and long-term psychotherapy to treatment-resistant patients.

B. Participants

The sample consisted of 226 patients taking part in the prospective, naturalistic, and longitudinal ARC Follow-Along Study (see Perry et al., 2009; Shapiro & Plakun, 2009). The Follow-Along Study (FAS) recruited patients admitted to the ARC between September 1992 and August 2001. Participation in the FAS was voluntary and did not affect patients’ treatment. Participants completed follow-up interviews every 6 to 8 months for up to four years after admission to the ARC.
Other researchers have published reports using the FAS data set, which is extensive and exceeds the scope of the present study. Perry and colleagues (2009) reported high rates of psychiatric comorbidity in the sample. FAS patients averaged 3.5 Axis I diagnoses at admission and 4.7 mood disorder diagnoses across their lifetimes. The sample had median Global Assessment of Functioning (GAF) scores of 42 at intake and 53 over the year prior to hospitalization. Many participants (78.5%) had been hospitalized at least once prior to coming to the ARC, and some (21.5%) had more than five prior hospitalizations. Shapiro and Plakun (2009) reported that 80% of the FAS sample had treatment-refractory mood disorders, and even more met criteria for at least one personality disorder.

Of the participants recruited into the FAS, 202 provided sufficient data to be included in the present study. The 24 excluded participants lacked measurements of symptom severity, the outcome variable in the primary analyses. All descriptive statistics that follow were based on the sample of 202 participants unless otherwise stated. Participants averaged 31.2 years of age (SD = 10.5) and were primarily Caucasian (95.0%), female (75.2%), and never married (66.8%). Participants were primarily members of the upper socioeconomic class (53.5%), with fewer in the middle (32.7%) and lower classes (13.9%).

The average length of treatment in the sample was 11.1 months (SD = 21.1). With regard to the diagnoses of interest, 40.6% of the sample had been diagnosed with BPD, 27.2% had been diagnosed with PTSD, and 18.3% had been diagnosed with both. According to participant reports, 50.5% had experienced interpersonal trauma by the age of 7 years; 41.1% had experienced interpersonal trauma across each period of early,
middle, and late childhood; and 43.1% had experienced three or more different types of trauma during childhood.

C. Measures

The present study made use of both baseline and follow-up measures from the FAS data set. For each follow-up measurement, time was recorded as the number of weeks that had passed since admission to the hospital. Duration of treatment was recorded as the numbers of days from admission to discharge at the ARC.

Basic demographic data included participant age, biological sex, marital status, race, and socioeconomic status. All demographic data were collected through self-report during the initial research interview. For the purposes of the present study, race was coded as either Caucasian or Other given that very few (5.3%) of the participants identified as non-White. Marital status was coded as Never Married or Married (i.e., married, divorced, separated, or widowed).

1. Hollingshead Two-Factor Index of Social Status (Hollingshead-II). The Hollingshead-II is an algorithm that calculates a score representing social status given information about an individual or family’s education, residence, and occupational status (Hollingshead, 1975; Hollingshead & Redlich, 1958). Scores range from 8 to 66, and are categorized in order to form five social strata: (1) upper middle class, (2) middle class and minor professionals, (3) white-collar workers, (4) blue-collar workers, and (5) unskilled laborers. The sample for this study was distributed as follows: 53.5% upper class, 4.5% middle class, 15.8% white-collar, 12.4% blue-collar, and 13.9% unskilled laborers.
2. **Symptom Checklist-90-Revised (SCL-90-R).** The SCL-90-R (Derogatis, 1994; Derogatis & Melisaratos, 1983) is a 90-item self-report inventory designed to assess a range of psychological symptoms in adult populations. The SCL-90-R prompts the respondent to rate his or her degree of distress on a 5-point Likert-type scale ranging from 0 (“not at all”) to 4 (“extremely”). Raw scores on the measure are computed to form the following nine domain-specific symptom scales: somatization, obsessive-compulsive, interpersonal sensitivity, depression, anxiety, hostility, phobic anxiety, paranoid ideation, and psychoticism. Overall psychological distress is represented on a global symptom scale called the General Severity Index (GSI), which is calculated as the sum of all 90 items divided by 90, and generates a score from 0 to 4. Scores in the 90th percentile (scores 1.33 standard deviations above the relevant normative mean) are thought to represent high levels of psychological distress. Evaluation of the SCL-90-R demonstrates that the scale is a reliable and valid measure of symptom severity, with high internal consistency and test-retest reliability (Derogatis, 1994).

3. **Traumatic Antecedents Interview (TAI).** The TAI is a semi-structured interview that probes participants regarding interpersonal forms of childhood trauma including physical abuse, sexual abuse, witnessing violence, physical neglect, emotional neglect, significant separations, losses, domestic chaos, verbal abuse, and parental discord. The version of the TAI used for the FAS is adapted from its original form (Herman et al., 1989), and includes information about two potentially protective factors: the presence of caretakers or confidants, and the display of mutual affection between caregivers. Trained raters review TAI interview transcripts and provide ratings for each source of trauma within three age-defined periods: early childhood (0-6 years), middle
childhood (7-12 years), and adolescence (13-18 years). (See Appendix A for a sample TAI scoring form.) The TAI rating scales differ for each form of trauma. Some forms of trauma are rated as simple counts of the number of perpetrators (i.e., physical abuse, sexual abuse, witnessing violence, significant separations, losses, and verbal abuse). Other forms of trauma are rated as either present or absent (i.e., physical neglect, emotional neglect, domestic chaos, parental discord, and parental mutual affection). The presence of caretakers or confidants was rated as a count of the number of such individuals, but with a maximum value of two. According to Roy and Perry (2004), the reliability of the adapted TAI variables ranged from acceptable to excellent (median Intraclass R = .73). The present study limited analyses to the following nine types of traumatic experiences: physical abuse, sexual abuse, witnessing violence, physical neglect, emotional neglect, significant separations, losses, domestic chaos, and verbal abuse.

4. Social Cognition and Object Relations Scale (SCORS). The Social Cognition and Object Relations Scale – Global Rating Method (SCORS-G; Hilsenroth, Stein, & Pinsker, 2007) provides a framework for clinician-generated ratings of a respondent’s representations of interpersonal events. Trained coders rate responses on a number of dimensions including: complexity of representations of people (COM), affective quality of representations (AFF), emotional investment in relationships (EIR), understanding of social causality (SC), and experience and management of aggressive impulses (AGG). These dimensions can be broadly categorized as reflecting more affective (AFF, EIR, and AGG) or more cognitive (COM and SC) aspects of object relations. Ratings are based on narrative responses and made on a 7-point scale ranging
from 1 (extremely pathological) to 7 (ideally healthy). (See Appendix B for a summary of the SCORS scales.) Researchers have generated SCORS ratings using narrative data based on early memories (Fowler, Hilsenroth, & Handler, 1995), projective test responses (Ackerman, Clemence, Weatherill, & Hilsenroth, 1999), and recall of psychotherapy sessions (Peters, Hilsenroth, Eudell-Simmons, Blagys, & Handler, 2006).

SCORS ratings for the present study were generated from narrative data obtained using the Relationship Anecdote Paradigm (RAP; Luborsky, 1998). The RAP is a semi-structured interview designed to obtain narrative accounts of a meaningful interpersonal interactions. Respondents are asked to identify and describe an interesting, important, or troublesome interaction with a parent, boss, or therapist (see Appendix C for the RAP Interview Form). Respondents are prompted to describe how the interaction began, what they and the other person said or did, why the interaction happened as it did, how the interaction ended, and what wishes or regrets they had about the interaction. The aim of the RAP is to generate a detailed account of an interaction that includes the respondent’s expression and understanding of the inter- and intra-personal features of the exchange.

Evaluation of the SCORS indicates that the rating system demonstrates good to excellent interrater reliability (Peters et al., 2006). SCORS ratings for the present study were generated by two trained raters; myself and an advanced graduate student who had recently completed a doctoral dissertation using the SCORS system. The two raters established reliability by rating practice narratives from the SCORS training manual and calculating intraclass correlation coefficients (ICC) using a two-way mixed-effects model. These ICCs ranged from good to excellent: 0.68 for COM, 0.85 for AFF, 0.73 for EIR, 0.64 for SC, and 0.72 for AGG. The two raters then established reliability with a
trained SCORS coder at the ARC by rating randomly selected RAP narratives from data not used in the present study. These ICCs also ranged from good to excellent: 0.63 for COM, 0.74 for AFF, 0.62 for EIR, 0.77 for SC, and 0.64 for AGG.

Once reliability had been established, the principal investigator generated SCORS ratings for each of the 204 participants in the FAS who had RAP data. The second rater double-coded one-third (69) of the RAP narratives selected at random from the sample. Ratings that differed by two or more points were reviewed and re-coded based on the original RAP narrative. ICCs for the resulting ratings were calculated using a one-way single measure reliability model and ranged from good to excellent: 0.68 for COM, 0.75 for AFF, 0.69 for EIR, 0.75 for SC, and 0.68 for AGG. The SCORS data used for analysis in the present study consisted of the single-coded ratings and the average of the double-coded ratings. The sample for the present study (N = 202) contained 193 participants with SCORS ratings and 9 for whom RAP data were not available.

D. Procedure

1. Recruitment of participants. Patients admitted at the ARC between September 1992 and August 2001 were invited to participate in the FAS. Their participation was entirely voluntary and did not affect their treatment at the ARC in any way. Within two days of admission to the hospital, a research coordinator interviewed potential participants and completed a Guided Clinical Interview (GCI). The GCI included all of the baseline measures (e.g., demographics, SCL-R-90, TAI, and SCORS narratives, and diagnostic interview) and took between 2.5 and 5 hours. Research staff followed up with patients approximately every 6 to 8 months in order to complete a set of measurements that included the SCL-90-R. Participants were paid $100 for providing
data at each time point. Follow-up data collection took place regardless of whether the patient remained in treatment at the ARC or had been discharged. Study data were collected, de-identified, and stored by the research staff at the ARC.

I gained access to the FAS data through a research collaboration with the ARC research staff. The principal investigator completed a Data Sharing and Confidentiality Agreement (see Appendix D) stating that a preliminary version of the proposal had been approved by the ARC Independent Review Board on August 6, 2010.

2. Measure development. The research hypotheses called for the creation of a set of trauma variables derived from the original TAI data. The creation of these trauma variables proceeded through the following three steps detailed below: (1) simplification of the TAI scales to facilitate comparability, (2) transformation of the data into variables representing specific features of childhood trauma, and (3) creation of a single variable representing the overall severity of childhood trauma.

The first step in measure development was to simplify the TAI data to enable comparison across types of trauma. The TAI data used in this study consisted of ratings for nine different types of traumatic experiences across three periods of childhood. Ratings for those experiences were made on different scales (i.e., dichotomous, rank-order, and continuous) and therefore could not be compared directly or aggregated easily (e.g., a rating of 1 did not have the same meaning for physical abuse and physical neglect). To enable comparison, the trauma variables were re-coded as dichotomous (i.e., either absent or present). The resulting set of variables indicated whether or not participants had endorsed each of the nine types of trauma as having occurred during each of three periods of childhood.
The second step in measure development was to transform the data to represent the following three features of childhood trauma: age of onset, chronicity, and variety. Age of onset was coded as a numeric variable reflecting the earliest period of childhood during which any traumatic experience was reported to have occurred; (0) no trauma reported, (1) adolescence, (2) middle childhood, or (3) early childhood. Chronicity was coded as the number of periods of childhood during which any traumatic experience was reported to have occurred (i.e., 0, 1, 2, or 3). Lastly, variety was coded as the number of different types of trauma reported across all three periods of childhood (i.e., an integer from 0 to 9).

The third step in measure development was to create a single variable reflecting the cumulative severity of childhood trauma. Drawing on the work of Doorenbos, Verbitsky, Given, and Given (2005), Item Response Theory (IRT) was used to create a unitary trauma variable within a Hierarchical Linear Modeling framework. Initially developed to address measurement issues in educational research, item response models distinguish between the apparent difficulty of test items and the latent ability of respondents. IRT analyses result in latent trait scales that are less skewed and more meaningful than simple transformations (e.g., the sum or proportion of correct responses). These latent trait scales combine test responses in an additive manner while accounting for measurement error and the relative weight of each test item. Applied here, IRT modeled the relative rarity of each type of trauma and the cumulative severity of each participant’s traumatic experiences.

The item response model used in this study was a one-parameter logistic (or Rasch) model. According to this model, the probability (log-odds) of a participant
reporting a particular category of trauma depends on the prevalence of that experience in the sample and the overall severity of that person’s traumatic experiences. The relationship between the overall trauma severity and the probability of endorsing any one type of trauma is fit to a logistic item characteristic curve (see Figure 1), which provides a continuous approximation of the dichotomous outcome (i.e., trauma absent or present). The model makes the following assumptions: trauma severity and category prevalence are additive, item responses are independent, each item discriminates equally between high and low levels of trauma severity, and the latent variable is distributed normally in the target population.

E. **Analytic Strategy**

The primary analyses used Hierarchical Linear Modeling (HLM) to model changes in symptom severity over time. The secondary analyses used hierarchical and logistic regression to predict psychiatric diagnosis and impairment in object relations based on reports of childhood trauma. Analyses addressed each of the five research hypotheses in turn: (1) fitting an unconditional HLM model describing symptom change over time, (2) fitting a conditional HLM model predicting symptom change from reports of childhood trauma, (3) adding object relations ratings as predictors in the conditional HLM model, (4) exploring the relationship between childhood trauma and impairments in object relations using hierarchical regression, and (5) exploring the relationship between childhood trauma and the likelihood of psychiatric diagnoses using logistic regression.

HLM offers advantages for the study of individual change over time (Raudenbush & Bryk, 2002). Specifically, other statistical techniques fail to model change explicitly or to accurately represent change data as repeated measurements nested within persons.
In contrast, HLM is able to model individual growth and is designed to represent multilevel data structures. In an HLM growth curve model, each person’s individual growth trajectory is represented at level one (level-1) and the trajectory defining parameters (i.e., the intercept and slope) become outcomes at a second level (level-2). Given this robust and flexible framework, HLM is able to account for the non-independence of repeated measurements, tolerate irregularly spaced measurements, adjust for missing measurements, improve estimations of individual growth, and generate more reliable models of change.

1. **Unconditional model for symptom change.** Hypothesis 1 stated that change in symptom severity would fit a model describing slowing quadratic decline over time. This hypothesis was tested through the construction and comparison of two unconditional models, one describing linear change in symptom severity and a second describing quadratic change. The first model was defined by the following: (Equation 1) the level-1 model predicting linear change in symptom severity over time, (Equation 2) the level-2 model predicting the level-1 intercept coefficient across participants, and (Equation 3) the level-2 model predicting the level-1 slope coefficient across participants.

\[
GSI_{ij} = \beta_{0j} + \beta_{ij}(\text{Month}_{ij}) + r_{ij} \tag{1}
\]

\[
\beta_{0j} = \gamma_{00} + u_{0j} \tag{2}
\]

\[
\beta_{ij} = \gamma_{10} + u_{ij} \tag{3}
\]

Equation 1 modeled within-subject (level-1) variation in symptom severity as the relationship between symptom severity (GSI) and time (Month) at each follow-up (i), for each participant (j). The intercept ($\beta_{0j}$) and slope ($\beta_{ij}$) of the equation were allowed to vary, thus modeling unique symptom trajectories for each participant. The remaining
within-subject variance in symptom severity (i.e., the variance not predicted by the model) was represented in a residual term ($r_{ij}$).

Equations 2 and 3 modeled differences in symptom severity between subjects (level-2) by making participant intercepts and slopes into outcomes. The random intercept ($\beta_{0ij}$) was described in terms of a fixed effect representing the overall average symptom severity across participants at time zero ($\gamma_{00}$) and the individual participant’s deviation from that average intercept ($u_{0ij}$). Similarly, the random slope ($\beta_{1ij}$) was described in terms of a fixed effect representing the overall average rate of change in symptom severity across participants ($\gamma_{10}$) and the individual participant’s deviation from that average slope ($u_{1ij}$). This first model was unconditional in that the random coefficients were not made dependent on other variables. Successful estimation of this model would assess linear change in symptom severity over time.

The time variable (Month) was centered at 1 year post-admission so that the model intercepts would be statistically and clinically meaningful. Leaving time uncentered, such that month equaled 0 at admission to the hospital, would have been inappropriate given that few participants completed GSI ratings on that day. GSI measurements took place at irregular intervals, with the average time elapsed since admission as follows: 1.6 months at baseline (SD = 1.2), 9.4 months at the first follow-up (SD = 1.9), 17.5 months at the second follow-up (SD = 3.8), 25.5 months at the third follow-up (SD = 4.2), and 33.9 months at the fourth follow-up (SD = 6.7). With time centered at 1 year post-admission, the model intercepts represented estimates of symptom severity that fit within the span of time covered by most participants’ GSI ratings. With regard to completeness of the GSI data, 32.2% of participants gave ratings for exactly one
time point (N = 65), 18.3% for two (N = 37), 13.9% for three (N = 28), 17.3% for four (N = 35), and 18.3% for five (N = 37).

In addition to assessing linear change in symptom severity, the first unconditional model indicated whether there was sufficient variance remaining at each level to merit continued modeling. With significant remaining variance, a second unconditional model was constructed to assess whether the trajectory of change in symptom severity over time followed a quadratic path in which the rate of change diminished over time. This second unconditional model expanded on the first by adding a quadratic term at level-1 (month squared) with a corresponding random coefficient ($\beta_{2j}$) and equation at level-2. This random quadratic term was equated with a fixed effect representing the overall average rate of quadratic change in symptom severity across participants ($\gamma_{20}$) and the individual participant’s deviation from that average change ($u_{2j}$). The results from these two models provided a test of Hypothesis 1. If the linear model indicated significant variance, and the quadratic model indicated significant slowing quadratic change, then Hypothesis 1 would be supported. The most parsimonious model would be used in subsequent analyses.

2. **Conditional model using reports of childhood trauma.** Hypothesis 2 stated that reports of childhood trauma would be associated with slower rates of decline in symptom severity. Testing this hypothesis required the creation and comparison of a series of conditional HLM models in which trauma and control variables were included as between-subject (level-2) predictors of individual symptom trajectories. Predictor variables were added to the model in the following sequence: (1) basic demographic variables, (2) duration of treatment, (3) psychiatric diagnoses, and (4) childhood trauma
variables. At each step, non-significant predictors were pruned and significant predictors were retained.

Sequential improvements in model fit could not be tested directly because imputation was used to complete the level-2 data. Nine of the 202 participants used in these analyses did not have SCORS ratings. A recent upgrade to the HLM statistical package (HLM 7.21s; Raudenbush, Bryk, & Congdon, 2012) was used to perform multiple imputation for these nine participants. This method generated five sets of estimated SCORS ratings and five associated HLM models. The results of these five models were then aggregated to provide final parameter estimates. While HLM provides a significance test for comparing nested models, this technique is not available when using aggregated parameter estimates. Instead, model comparison was limited to the retention of variables with significant fixed effects and the informal observation of reductions in parameter variance.

Hypothesis 2 predicted that higher reports of childhood trauma on one or more of the four trauma variables (i.e., onset, chronicity, variety, and severity) would predict slower decline in psychiatric symptoms. These trauma variables were included as level-2 predictors of the intercepts and slopes that defined participants’ symptom trajectories. A significant fixed effect on the intercept parameter would indicate that a variable was predictive of symptom severity one year after intake. A significant fixed effect on the slope parameter would indicate that a variable was predictive of the linear rate of decline in symptoms at that time point. A significant fixed effect on the quadratic parameter would indicate that a variable was predictive of the rate of change in symptom decline (i.e., slowing) at that time point. Significant effects of any of these trauma variables on
either the slope or quadratic parameters would support Hypothesis 2, and the variables would be said to moderate the relationship between symptom severity and time.

3. **Conditional model using object relations.** Hypothesis 3 predicted that impairments in object relations would be associated with slower rates of decline in symptom severity. To test this hypothesis, object relations (SCORS) variables were added to the final model from Hypothesis 2 as between-subject (level-2) predictors of individual symptom trajectories.

The hypothesis was that lower SCORS ratings would be associated with either slower or slowing symptom recovery. As was the case for Hypothesis 2, significant fixed effects would indicate that SCORS ratings were predictive of symptom severity, linear decline in symptoms, or rate of change in symptom decline one year after intake. As stated above, the use of imputation precluded direct tests of improvements in model fit. Instead, improvements in model fit were assessed informally by observing reductions in variance after the inclusion of SCORS variables as predictors of the growth parameters. A significant fixed effect and an apparent reduction in growth parameter variance would support Hypothesis 3. Such results would indicate that impairment in object relations moderates the relationship between symptom severity and time.

Once a final growth curve model was reached, its adequacy was assessed through an examination of the residual variance components. These HLM models assumed that level-1 and 2 residuals are normally distributed around zero, and that their degrees of variance are not significantly different across time or coefficient values. To assess whether the final model met these assumptions, level-1 and 2 residuals were graphed and inspected visually for signs of non-normality and heteroscedasticity (unequal variance
across time or coefficient value). Evidence of either assumption violation could call into question the p values and suggest that the model had failed to explain meaningful variance in the outcome.

4. Predicting impairments in object relations from childhood trauma.

Hypothesis 4 stated that more severe childhood trauma would be associated with greater impairments in object relations. Multiple hierarchical regression was used to test whether the addition of trauma severity variables improved significantly on models predicting SCORS ratings. These analyses were limited to one trauma variable and composite SCORS variables in order to reduce the number of significance tests and the risk of type I error. The trauma severity variable was chosen because of its superior statistical properties and its meaningfulness as a measure of the degree of childhood trauma. Three composites of the SCORS variables were used: (1) an overall average of the five dimensions, (2) an average of the three affective dimensions, and (3) an average of the two cognitive dimensions. Thus, Hypothesis 4 predicted that greater overall severity of trauma would predict lower overall, affective, or cognitive SCORS ratings.

Three multiple hierarchical regression models were created to test Hypothesis 4, one for each of the composite SCORS variables (i.e., overall, affective, and cognitive averages). Within each model, predictor variables were added in separate blocks in the following order: demographics variables, psychiatric diagnoses of BPD or PTSD, and trauma severity. A significance test for change in R squared ($R^2$) indicated whether or not the addition of each block of variables improved model fit. Predictors showing significant effects were retained in the final, most parsimonious, models. Significant effects of trauma severity in the predicted direction would support Hypothesis 4,
suggesting that greater overall trauma severity was predictive of greater impairment in object relations, controlling for demographics and diagnosis. Support for both Hypotheses 3 and 4 would offer some indication that impairment in object relations served as a mediator between childhood trauma and poorer treatment response. Supportive conclusions from these analyses were contingent on tolerable levels of collinearity among related predictors (e.g., trauma severity and diagnosis of PTSD).

5. **Predicting psychiatric diagnoses from childhood trauma.** Hypothesis 5 stated that more severe childhood trauma would be associated with greater likelihood that a participant had been diagnosed with PTSD and BPD. Given that these outcome variables were dichotomous, logistic regression was used to test whether greater trauma severity was associated with higher probability of each psychiatric diagnosis. Logistic regression simulates dichotomous outcomes by fitting a continuous logarithmic probability curve, thus allowing predictor variables to contribute to the odds that a participant would experience either outcome. As with the analyses for Hypothesis 4, trauma severity was used in order to reduce the number of significance tests and the risk of type I error.

Two logistic regression models were created to test Hypothesis 5, one for each psychiatric diagnosis (i.e., BPD and PTSD). Within each model, predictor variables were added in three blocks in the following order: demographics variables, object relations impairment, and trauma severity. A chi-square ($\chi^2$) test indicated whether or not the addition of each block improved model fit. Predictors showing significant effects were retained in the final, most parsimonious, models. The adequacy of the final models was assessed by examining their predictive accuracy with regard to sensitivity (percent of
correct positive predictions) and specificity (percent of correct negative predictions).

Significant effects of trauma severity in the predicted direction would support Hypothesis 5, suggesting that greater overall trauma severity was predictive of greater likelihood of these psychiatric diagnoses, controlling for demographics and impairment in object relations.
CHAPTER III

RESULTS

A. **Descriptive Statistics**

Table 1 provides descriptive statistics for the continuous and categorical variables prior to imputation of the SCORS data. Intercorrelations among predictor variables (see Table 2) were generally non-significant or small to moderate in size. The largest correlations were between age, marital status, and childhood socioeconomic status, but none were large enough to negatively impact the analyses. The mean symptom severity score at intake was 1.48 (SD = 0.68) on the GSI. Emotional neglect was the most common type of trauma reported by participants (61.1%), followed by significant separations (49.6%), sexual abuse (37.6%), verbal abuse (31.0%), physical abuse (30.1%), witnessing violence (18.1%), physical neglect (15.0%), losses (13.3%), and domestic chaos (10.6%).

B. **Unconditional Models (Hypothesis 1)**

The coefficients and variance components for each step in the sequence of models are presented in Table 3. Construction of the unconditional linear growth model revealed that on average, symptom severity levels one year after hospital admission (i.e., time zero) were significantly different from zero ($\gamma = 1.3588$, $t(201) = 30.77$, $p < .001$). Also, the linear rate of change in symptom severity levels was negative and significantly different from zero ($\gamma = -0.0111$, $t(201) = -5.21$, $p < .001$). On average, participants reported symptom severities that were non-zero one year after admission and declining over time. Estimates of the variance components indicated that significant variability remained in symptom severity one year after hospitalization ($\tau_{00} = 0.3001$), $\chi^2 (136) =$
732.54, \( p < .001 \), and in the rate of symptom decline (\( \tau_{10} = 0.0002 \)), \( \chi^2 \) (136) = 224.19, \( p < .001 \). The presence of sufficient unexplained variability in these parameters (demonstrated graphically in Figure 2) permitted subsequent efforts to model quadratic symptom change and predict differences among individual symptom trajectories.

Successful testing of the unconditional linear model was followed by construction of the unconditional quadratic model, which tested for curvilinear change in symptom severity over time. Time-squared was added at level-1 as a predictor of symptom severity. This quadratic term failed to reach significance and was dropped from further analyses. The resulting model described non-zero symptom severity one year after admission with linear decline in symptom severity over time.

C. **Conditional Models (Hypothesis 2)**

Completion of a base unconditional model was followed by the sequential construction and pruning of a series of conditional models. This series of models tested whether variables in the following sequence predicted the level or slope of participants’ symptom trajectories: (1) demographic variables, (2) duration of treatment, (3) psychiatric diagnoses, and (4) childhood trauma variables.

In the first set of conditional models, the following demographic variables were added as predictors of participants’ symptom trajectories; age, sex, race, marital status, current socioeconomic status, and childhood socioeconomic status. Of these six predictors, sex had a significant fixed effect on symptom level one year after admission (\( \gamma = 0.2815 \), \( t(195) = 2.91, p < .01 \), and age had a significant effect on symptom slope (\( \gamma = 0.0006 \), \( t(195) = 2.12, p < .01 \). Thus, women experienced significantly higher levels of symptom severity than men one year after admission, and older participants experienced
less negative (slower) rates of decline in symptom severity. These two variables were retained at level-2, sex as a predictor of the intercept and age as a predictor of both the intercept and the slope. (Predictors of the slope are included as predictors of the intercept even in the absence of a fixed effect on the latter.)

In the second set of conditional models, the duration of treatment was added as a predictor of symptom trajectory. The first of these models tested for a non-linear effect of participant age by including age squared as a predictor of both coefficients. Age squared was not significantly related to either coefficient, so it was dropped from the model. Duration of treatment was then added and had a significant fixed effect on the intercept ($\gamma = 0.0052$), $t(198) = 3.78, p < .001$. Thus, higher symptom severity one year after admission was associated with more months of treatment at the ARC. An additional model tested for an interaction effect between age and treatment duration by including their product as a predictor of both coefficients. This interaction term was not significantly related to either coefficient, so it was dropped from the model. Treatment duration was retained at level-2 as a predictor of the intercept.

In the third set of conditional models, psychiatric diagnoses of BPD and PTSD were added as separate dichotomous predictors of symptom trajectory. The effect of PTSD diagnosis was not significant, but BPD diagnosis had a significant fixed effect on the intercept ($\gamma = 0.4988$), $t(197) = 5.88, p < .001$. Thus, participants diagnosed with BPD had higher symptom severity one year after admission than participants not diagnosed with BPD. BPD was retained at level-2 as a predictor of the intercept.

In the fourth set of conditional models, reports of childhood trauma were added as predictors of symptom trajectory. First, each of the four trauma variables (i.e., onset,
chronicity, variety, and severity) were added to the model separately. When added individually, variety had a significant fixed effect on the intercept ($\gamma = 0.0431), t(196) = 2.02, p < .05, and the comparable fixed effect of severity neared significance ($\gamma = -0.0012), t(199) = -1.43, p = .15. In an effort to distinguish their unique effects, variety was tested in combination with each of the other trauma variables, which were highly correlated ($r = 0.68$ for onset, $0.75$ for chronicity, and $0.95$ for severity). Variety had a significant fixed effect on the intercept when included in a model along with chronicity ($\gamma = 0.0635), t(195) = 2.00, p < .05. None of the other tests resulted in significant effects. Given the results of these and the previous models, variety was retained as a predictor of the intercept. Thus, participants who reported having experienced more types of trauma during childhood provided higher symptom severity ratings one year after admission. The effect of sex on the intercept dropped below significance with variety of trauma added to the model ($\gamma = 0.1579), t(196) = 1.73, p = .09. Sex was retained because it had a significant effect in earlier models and may have served as an important control variable.

D. Final Conditional Model (Hypothesis 3)

Three models were created to test whether object relations predicted symptom trajectory. Two of the models used composite versions of the SCORS variables that averaged all of the SCORS dimensions, only the cognitive dimensions, or only the affective dimensions. None of the composite SCORS variables had significant effects on the model parameters, and they were not included in the final model. The final conditional model is presented in the following: (Equation 4)
hospital; (Equation 5) the level-2 model predicting the level-1 intercept coefficient across participants given their age, sex, whether or not they had been diagnosed with BPD, the duration of their stay at the ARC, and the variety of childhood traumatic experiences that they had reported at baseline; and (Equation 6) the level-2 model predicting the level-1 slope coefficient across participants given their age.

\[
GSI_{ij} = \beta_{0j} + \beta_{ij}(\text{Month}_{ij}) + r_{ij}
\]  
\[
\beta_{0j} = \gamma_{00} + \gamma_{01}(\text{Age}_{ij}) + \gamma_{02}(\text{Sex}_{ij}) + \gamma_{03}(\text{BPD}_{ij}) + \gamma_{04}(\text{Duration}_{ij}) + \gamma_{05}(\text{Variety}_{ij}) + u_{0j}
\]  
\[
\beta_{ij} = \gamma_{10} + \gamma_{11}(\text{Age}_{ij}) + u_{ij}
\]

In the final model (see Figure 3), the average GSI symptom severity level one year after admission was 1.0480, and the average rate of decline in GSI score was 0.0118 points per month. GSI levels were 0.4541 points higher among participants diagnosed with BPD as compared to those not diagnosed with the personality disorder. GSI levels one year after admission were higher among participants who remained in treatment at the ARC for longer periods of time (0.0063 GSI points per additional month of treatment). GSI levels were also higher among participants who reported experiencing more variety of childhood trauma (0.0445 GSI points per additional type of trauma reported). The rate of decline in GSI score was slower among older participants as compared to younger participants (0.0005 GSI points per month slower for every year older that participants were at admission). As stated earlier, the effects of sex and age on the intercept were retained in the model although they were not significant. The effects of the final model are presented graphically in Figures 4 and 5.

Residual analyses assessed the assumptions of the final conditional model and demonstrated it to be adequate and interpretable. Visual inspection of the level-1...
residuals supported the assumption that they were normally distributed around zero (see Figure 6). The level-1 residuals were observed to trend away from normality at higher values, but to a tolerable degree. Variance in the level-1 residuals appeared to be independent of the values fitted by the model and independent of time (see Figure 7). Non-constant variance (heteroscedasticity) was visible in the level-1 residuals across fitted values, an assumption violation that could result in misestimation of standard errors, but did not appear severe enough to merit concern.

The level-2 models provided residual estimates for both intercepts and slopes. Visual inspection of these residuals supported the assumption that they were normally distributed around zero (see Figures 8 and 9). A plot of the observed random effects indicated that they were bivariate normally distributed (see Figure 10). Variance in the level-2 residuals for the intercepts appeared to be independent of the predictor variables age and diagnosis (see Figure 11), as well as duration of treatment and variety of trauma (see Figure 12). Variance in the level-2 residuals for the slopes appeared to be independent of the predictor variable age (see Figure 13).

E. **Predicting SCORS from Childhood Trauma (Hypothesis 4)**

A series of multiple hierarchical regression analyses were performed to test whether greater childhood trauma predicted impairment in object relations. Overall trauma severity was chosen as the predictor variable because of its superior statistical properties. Within each model, predictor variables were added in the following three blocks: (1) age, sex, marital status, race, current SES, childhood SES; (2) diagnoses of BPD and PTSD; and (3) trauma severity. These analyses were conducted on the data set after the imputation of SCORS data for 9 participants. The variance inflation factors
(VIFs) indicated that levels of multicollinearity among these variables were low, with the highest being 1.97 for age, 1.71 for marital status, and 1.33 for diagnosis of BPD.

Three models were tested using three different composite SCORS variables as outcomes. Final coefficients for each model are presented in Table 4. The first model predicted the average of all SCORS dimensions, accounted for 2.4% of variance (adjusted $R^2 = .024$), and failed to reach overall significance. Within this first model, the effect of trauma severity approached significance ($\beta = -0.09$), $t = -1.73$, $p = .09$. The second model predicted the average of the affective SCORS dimensions, accounted for 2.4% of variance (adjusted $R^2 = .024$), and failed to reach overall significance. Within this second model, the effect of trauma severity approached significance, ($\beta = -0.10$), $t = -1.75$, $p = .08$, as did the effect of diagnosis of BPD, ($\beta = -0.26$), $t = -1.79$, $p = .08$. The third model predicted the average of the cognitive SCORS dimensions, accounted for 1.4% of variance (adjusted $R^2 = .014$), and failed to reach overall significance. Within this third model, the effect of sex reached significance, ($\beta = 0.44$), $t = 2.15$, $p < .05$, women obtained higher ratings on the cognitive SCORS dimensions than men.

F. Predicting Diagnosis from Childhood Trauma (Hypothesis 5)

Logistical regression analyses were completed to test whether the severity of childhood trauma predicted diagnoses of BPD and PTSD. Models predicting BPD and PTSD were tested separately. For both models, predictor variables were added in the following blocks in order to observe improvements in fit: (1) age, sex, marital status, race, current socioeconomic status, childhood socioeconomic status; (2) affective and cognitive averages from the SCORS; and (3) trauma severity. Final coefficients for both models are presented in Table 5.
In the logistic regression model predicting diagnosis of BPD, the omnibus tests were significant for the first block, $\chi^2(6) = 30.230$, $p < .001$, nearing significance for the second block, $\chi^2(2) = 5.547$, $p = .062$, and significant for the third block, $\chi^2(1) = 15.433$, $p < .001$. The first and third blocks reliably distinguished between participants diagnosed with BPD and those not diagnosed with the disorder. The third model successfully predicted the presence of BPD in 77.8% of participants and the absence of BPD in 59.3% of participants. According to the Wald tests, age ($\beta = -0.077$, $p < .01$), sex ($\beta = 1.155$, $p < .05$), marital status ($\beta = 0.960$, $p < .05$), and trauma severity ($\beta = 0.530$, $p < .001$) contributed significantly to prediction of a BPD diagnosis. The likelihood of having a BPD diagnosis was greater among participants who were younger, women, married at least once, and more severely traumatized.

In the logistic regression model predicting diagnosis of PTSD, the omnibus tests were significant for the first block, $\chi^2(6) = 31.341$, $p < .001$, not significance for the second block, and significant for the third block $\chi^2(1) = 6.242$, $p < .05$. The first and third blocks reliably distinguished between participants diagnosed with PTSD and those not diagnosed with the disorder. The third model successfully predicted the presence of PTSD in 90.1% of participants and the absence of PTSD in 14.8% of participants. According to the Wald tests, sex ($\beta = 2.335$, $p < .01$) and trauma severity ($\beta = .338$, $p < .05$) contributed significantly to prediction of a PTSD diagnosis. The likelihood of having a PTSD diagnosis was greater among participants who were women and more severely traumatized. Table 6 demonstrates these results by presenting the predicted likelihood that four hypothetical participants would be diagnosed with BPD and PTSD.
CHAPTER IV
DISCUSSION

A. **Summary and Discussion of the Results**

The purpose of the present study was to examine the relationship between childhood trauma and response to treatment during adulthood. Participants in the Austen Riggs Center (ARC) Follow-Along Study (FAS) provided longitudinal symptom severity data following psychiatric hospitalization, which I used to test three primary hypotheses: (1) that symptom severity would follow a trajectory of slowing quadratic decline; (2) that earlier, more chronic, more varied, and more severe childhood trauma would predict slower rates of decline in symptom severity; and (3) that greater impairment in object relations would predict slower rates of decline in symptom severity. I sought to establish relationships between childhood trauma and later impairment through two secondary hypotheses: (4) that more severe trauma would be associated with greater impairment in object relations, and (5) that more severe trauma would be associated with greater likelihood of being diagnosed with PTSD and BPD. I found support for only the fifth hypothesis.

Contrary to Hypothesis 1, the results demonstrated linear and not quadratic decline in symptom severity over time. I had presumed that symptom change following hospital admission would be bounded, such that recovery would slow as an individual approached a stable level of either baseline pathology or health. Given that symptoms cannot decline indefinitely, my failure to find evidence for such boundaries was likely due to limitations in measurement. FAS participants provided symptom data every six to eight months over about a two-year period following intake. Non-linear symptom change
may have been detectable had I measured symptom severity more frequently in the days following intake, or had I continued to measure symptom severity over many more years. Previous research has shown similar linear trajectories of symptom decline during treatment, but with more rapid symptom decline in the days and weeks before treatment begins (Puschner, Kraft, Kächele, and Kordy, 2007). It may be that non-linear patterns of symptom change occur rapidly around the start of treatment and then gradually over many years.

Despite evidence for overall linear decline in symptom severity, I found that individual participants’ symptom trajectories varied considerably. Significant variance remained in individual rates of symptom change even after successful modeling of the overall trend showing linear decline. In fact, some participants in the study showed no symptom improvement over time, and a few became increasingly symptomatic. The presence of significant remaining variance was important because it suggested that factors other than time accounted for variability in symptom trajectories. In other words, symptom change depended on more than just the passage of time.

Contrary to Hypothesis 2, I did not find evidence that childhood trauma predicted rates of symptom change. This was the case for each of four features of childhood trauma: age of onset, chronicity across three periods of childhood, variety among nine forms of trauma, and overall severity. I based Hypothesis 2 on the argument that people who had experienced more severe childhood trauma tend to recover more slowly from acute psychiatric symptoms due to their difficulty engaging with treatment providers. As discussed in the Introduction, there is ample evidence in the literature that chronic trauma causes lasting and pervasive impairments (e.g., Cloitre et al., 2009), and that trauma
survivors have greater difficulty in treatment (e.g., Courtois, 2008). Given such
evidence, my failure to demonstrate a link between childhood trauma and response to
treatment was likely the result of shortcomings in the present study. For example, it is
possible that changes in symptom severity do not fully represent response to treatment.
As discussed in the Strengths and Limitations section below, more proximal measures of
treatment response may have been needed to demonstrate the hypothesized effects.

My inability to demonstrate a relationship between childhood trauma and
symptom change may have also been due to excluded moderating variables. For
example, evidence has suggested that adult women who report having childhood
friendships suffer less severe symptoms of depression than women without early
friendships, and that this protective effect remains when controlling for the consequences
of childhood trauma (Powers, Ressler, and Bradley, 2009). It may be that protective
factors not assessed in the present study, such as social support, moderated the
relationship between childhood trauma and symptom change.

Another potential moderator of the proposed relationships could have been the
responsiveness of the ARC treatment providers. As Stiles (1988) argued, effective
therapeutic treatment may obscure the statistical relationship between process and
outcome variables. Stiles explained that therapists adjust treatment to match their clients’
needs. As a result, variability in the therapeutic process will be associated with client
need rather than treatment outcome. In other words, by adapting to their clients’ needs,
therapists decouple treatment process and outcome by varying the former to maximize
the latter. To the extent that the responsiveness of the ARC providers enabled
participants to engage in treatment despite barriers, the relationship between childhood trauma and response to treatment may have been obscured.

Contrary to Hypothesis 3, I did not find evidence that greater impairment in object relations predicted slower symptom recovery. This was the case for all composite indices of object relations (i.e., overall, affective, and cognitive) and for both symptom levels and rates of change. I found this result particularly surprising given that object relations functioning should affect the therapeutic process. For example, a patient who struggles to understand social causality will have difficulty representing the effects of his or her behavior on others. The shortcomings of the present study described thus far may explain this contrary finding. In addition, my method for deriving SCORS ratings may have been flawed. I used interpersonal anecdotes to generate SCORS ratings in a manner similar to other studies (e.g., Peters et al., 2006). Yet I observed that, due to idiosyncrasies in collection, these anecdotes seemed to be inconsistent in their reflection of participants’ object relations functioning. It seemed that the content and quality of the resulting narratives were highly sensitive to idiosyncrasies in the stories being told, the skill of the interviewer, dynamics between the interviewer and interviewee, and situation factors such as the participant’s emotional state.

Treatment duration, diagnosis of BPD, and variety of childhood trauma emerged as significant predictors of symptom severity in the final model. Each variable contributed significantly to the prediction of overall level of symptom severity. The model predicted more severe symptoms one year after hospital admission for participants who: (1) had longer stays at the ARC, (2) had been diagnosed with BPD, and (3) had experienced more categories of childhood trauma. These findings conform to clinical
and empirical expectations. Previous research has demonstrated a small but consistent association between symptom severity and length of inpatient hospital stay (Hugo, 2000). While research has shown high symptom severity among people diagnosed with BPD, it has also demonstrated that features of PTSD contribute uniquely to the prediction of emotional problems (Marshall-Berenz, Morrison, Schumacher, & Coffey, 2011). Finally, as discussed in the Introduction, clinical wisdom and research findings suggest that greater exposure to trauma in childhood predicts more severe impairment in adulthood (e.g., Streeck-Fischer & van der Kolk, 2000).

Notably, treatment duration, diagnosis of BPD, and variety of childhood trauma were significant predictors of symptom severity while controlling for sex, age, and one another. Each of these characteristics contributed uniquely to the prediction of more severe symptoms one year after hospitalization, and no one variable fully accounted for the effect of another. Remaining in treatment longer, being diagnosed with BPD, and having experienced more varied childhood trauma predicted more severe psychiatric symptoms. Also, biological sex appeared to be an important factor in predicting symptom severity levels, though it dropped below significance by the final model.

The final growth curve model demonstrated that a participant’s age predicted his or her rate of change in symptom severity. According to the observed effect, older participants had slower rates of decline in symptom severity, while younger participants experienced more rapid symptom decline. In fact, age was the only variable found to predict the rate of change in symptom severity. This finding regarding age and may point to other factors that affect response to treatment. If rates of symptom change slow as a person ages, then factors related to age likely account for that slowing. Age related
factors may be psychological (e.g., improving coping skills), environmental (e.g., increasing social and occupational stability), or biological (e.g., decreasing physiological reactivity to stress).

Figure 4 indicates another possible pattern in symptom levels relative to age. While age did not predict symptom levels one year after hospitalization, the age-focused graph suggests that older and younger participants experienced differences in symptom trajectory that go beyond distinct rates of change. Though not tested statistically, younger participants appeared to enter the hospital with slightly more severe symptoms than older participants, and progress to less severe symptom over time. Expressed in terms of variability, younger participants appeared to experience a wider range of symptom severity than older participants. Although merely speculative, the possibility that fluctuations in symptom severity narrow as a person ages merits further research.

Finally, with regard to age, the finding that rates of symptom decline were slower for older participants may suggest quadratic change in symptoms over time. Though the observed trend in symptom decline was linear, continued measurement years into the future may have demonstrated a curvilinear trend. It is possible that the effect of age on rate of symptom change reflects a greater slowing in symptom recovery which takes place over the course of one’s lifetime. If this were the case, then quadratic symptom change would become apparent only over decades. Although rare, longitudinal research spanning decades would be necessary to test for such extended patterns in symptom change.

Contrary to Hypothesis 4, more severe childhood trauma was not associated with greater impairment in object relations. This was true for overall object relations
functioning as well as for the affective and cognitive dimensions alone. Despite the absence of significant findings, some of the hypothesized effects did approach significance. The analysis showed non-significant trends relating more severe childhood trauma to greater impairment in both overall and affective object relations functioning. While these non-significant trends must be interpreted cautiously, they offer tentative evidence for a relationship between the severity of childhood trauma and some features of object relations functioning.

It is possible that the study failed to predict object relations ratings because the analyses were not targeted enough. I tested for relationships that were broad in scope, between overall, cognitive, and affective SCORS composites and four aspects of childhood trauma (i.e., onset, chronicity, variety, and severity). In contrast, previous research has shown relationships between more severe childhood trauma and one SCORS dimension (i.e., understanding of social causality; Slavin, Stein, Pinsker-Aspen, & Hilsenroth, 2007), or between childhood sexual trauma and affective and cognitive SCORS ratings (Kernhof, Kaufhold, & Grabhorn, 2008).

Sex and diagnosis of BPD emerged as possible predictors of the affective and cognitive dimensions of object relations. Women obtained significantly higher ratings on the cognitive dimensions (i.e., complexity of representations and understanding of social causality), indicating that women had more accurate and nuanced representations of themselves, others, and events. Further research is needed on sex differences in SCORS ratings given that the present study had relatively few men. In addition, analyses revealed a non-significant trend such that participants diagnosed with BPD were rated as more impaired on the affective dimensions of object relations (i.e., affective quality of
representations, emotional investment in relationships, and experience and management of aggression), indicating that those diagnosed with BPD expected more pain, isolation, and conflict in their relationships. While the latter, non-significant result concurs with existing research on BPD and object relations (Ackerman et al., 1999), my failure to establish a more clear relationship may be due to previously discussed flaws in the SCORS rating technique.

Finally, the study findings supported Hypothesis 5 in that participants who reported more severe childhood trauma were more likely to be diagnosed with PTSD and BPD. More specifically, a one standard deviation increase in trauma severity was associated with about a 51% increase in the odds of being diagnosed with PTSD and a 90% increase in the odds of being diagnosed with BPD. These findings correspond with conceptualizations of both disorders. By definition, a diagnosis of PTSD requires the history of some traumatic experience (APA, 2000), and theory suggests that exposure to an abusive or neglectful environment contributes to the development of BPD (Sansone & Sansone, 2007). These findings also fit with prior research, reviewed in the Introduction, demonstrating higher rates of reported trauma among people diagnosed with PTSD and BPD. While not surprising, these findings display the predictive utility of the trauma severity variable.

In summary, the study findings supported Hypothesis 5 but not Hypotheses 1 through 4. Despite successful modeling of symptom change, I found little support for the argument that childhood trauma leads to poor treatment response in adulthood. Rather, after admission to the ARC for intensive psychiatric treatment, participants experienced variable symptom trajectories that followed an overall trend of linear decline. Some
participant characteristics (e.g., age, sex, marital status, and trauma severity) predicted participants’ overall symptom severity and the likelihood that they were diagnosed with PTSD and BPD. Nonetheless, I failed to find support for the hypothesized relationships between childhood trauma, object relations, and symptom change.

B. **Strengths and Limitations of the Study**

The strengths of the present study included its sample characteristics, extensive data set, and application of advanced statistical techniques. The characteristics of the sample ensured that the resulting data addressed the phenomena of interest. The present study aimed to examine relationships between childhood trauma, poor response to treatment, and recovery from heightened psychiatric symptoms. The ARC provided a patient population that had been known to experience a high base-rate of complex trauma, identified as treatment resistant, and hospitalized following some psychiatric crisis. The study sample lent strength to the statistical analyses by containing variability in these phenomena.

The extensive data set was another asset of the study. The FAS included a large number of participants who provided extensive data at hospital intake, through treatment, and into the following years. The FAS data set included a wide range of variables, of which I used only a subset for the present study. The level of detail in the data enabled me to the compare outcomes based on specific features of participants’ childhood trauma histories (i.e., the onset, chronicity, variety, and severity). In addition, owing to the 20-year scope of the FAS, the data set contained baseline and follow-up measurements for over 200 participants. The size and extent of this longitudinal sample enabled me to model the nested structure of the data.
Building on the strengths of the sample and data set, I was able to employ advanced statistical techniques including HLM and item analysis. These techniques are capable of modeling latent and complex phenomena more accurately than less advanced techniques. HLM provided a method for accurately modeling variability in the outcome nested between and within participants over time. Item analyses enabled the dynamic aggregation of 27 dichotomous trauma variables into one overall measure of trauma severity with vastly improved statistical properties. These advanced techniques generated more mathematically true representations of the data.

The present study had notable limitations including its unique sample, lack of variables measuring treatment process, and failure to control for potential moderators of the proposed relationships. While the sample characteristics strengthened the analyses, they also limited generalizability of the findings. The ARC draws a unique population of patients who are wealthy and treatment resistant, and then engages them in an intensive psychodynamic treatment milieu. Conclusions based on these findings must therefore be constrained to similar populations. The uniqueness of the sample may present another limitation. The relationship between childhood trauma and response to treatment may have been obscured if all ARC patients were similarly treatment resistant regardless of their histories of childhood trauma. The impact of this latter issue could not be assessed in the absence of a direct measure of treatment resistance.

In addition, the study lacked measures of relevant treatment processes. The basic argument of the study was that childhood trauma causes lasting impairments that inhibit response to treatment by interfering with a person’s ability to engage with treatment providers. While the hypotheses did not address engagement in treatment directly,
measures of treatment processes could have been used to check the assumptions of the
study argument. Such measures might have assessed the patients’ trust in their therapists
or the degree to which defensiveness complicated sessions.

Lastly, the study failed to measure potential moderators of the consequences of
childhood trauma. For example, as discussed in the preceding Summary, the negative
effects of childhood trauma are likely tempered by protective factors such as the presence
of childhood friendships. The failure to control for important moderating variables can
reduce the sensitivity of statistical tests and blur the relationships between observed
variables. While a common limitation in research, the omission of moderating variables
limits the present study nonetheless.

In summary, the findings of the present study are supported by its methodological
strengths, which include a relevant sample, an extensive data set, and the use of advanced
statistical techniques. The limitations of the study constrain generalization of the
findings, leave certain assumptions untested, and may have obscured the hypothesized
relationships.

C. Implications for Research

Despite little evidence supporting its hypotheses, the present study carries a
number of implications for future research. Ongoing efforts to study the consequences of
childhood trauma and the predictors of treatment response can benefit from attention to
five issues raised by the study findings: (1) rates of symptom change may vary across the
lifetime, (2) the most powerful predictors of symptom change may have yet to be
identified, (3) a person’s response to treatment may not be evident in his or her rate of
symptom change, (4) controlling for certain moderating variables may clarify the lasting
consequences of childhood trauma, and (5) advanced statistical techniques such as HLM are powerful tools in the assessment of change processes.

One of the more intriguing findings of the present study was that symptom trajectories varied with participant age but not with time. This finding may indicate that symptom patterns are relatively stable, as evidenced by linear symptom decline, and change more gradually over the course of a person’s lifetime. People may recover from acute psychiatric symptoms more rapidly in their youth, and more slowly as they age. Though further research is needed to examine this possibility, the present findings offer evidence that rates of symptom change are affected by factors that endure over many years.

Despite the inclusion of a number of predictors, individual symptom trajectories varied significantly. Some participants experienced steady symptom decline while others experienced intensification or wide fluctuation in their symptoms. The presence of significant unexplained variability suggests that factors beyond those addressed in this study determine rates of symptom change following hospital admission. Future research may explore the specific variables that predict individual symptom trajectories both following crisis and over the lifetime.

The present study operationalized response to treatment as rate of symptom change during and after psychiatric hospitalization. Given the failure to predict response to treatment using factors identified in the literature, it is possible that symptom change is either an incomplete or a poor measure of response to treatment. Perhaps response to treatment is evident in other variables and is not directly related to symptom change. A patient who has suffered a loss, for example, could respond to treatment effectively by
grieving the loss, and may experience episodes of intense pain and sadness before achieving stable reductions in symptoms. This possibility evokes a long-standing and unresolved debate regarding the importance of symptoms and symptom change in psychotherapy. In light of the present findings, future research should include both symptom and process measures of treatment response.

The enduring consequences of early, chronic, varied, and severe childhood trauma were not apparent in the present study. As discussed in the Introduction, prior research has demonstrated that childhood trauma can result in lasting impairment. However, the existing research does not indicate the conditions under which such impairment occurs. The present failure to observe direct effects of childhood trauma suggests that the developmental ramifications of these experiences are moderated by other factors. By identifying and controlling for such moderators, future studies will be better equipped to examine the effect of childhood trauma on treatment in adulthood.

Finally, the present study demonstrates the power of advanced statistical techniques. The use of HLM enabled more accurate modeling of variance in symptom severity between participants and over time. The HLM platform provided more conceptually and mathematically precise estimations of nested variance that lent confidence and flexibility to the analyses. The application of such advanced statistical techniques can produce more accurate and reliable models of complex processes as they unfold over time.

D. Implications for Practice

The present study carries notable implications for clinical practice. The findings highlight a number of issues relevant to conducting therapy with survivors of childhood
trauma and with treatment-resistant populations, including the following: (1) age may be a factor in determining the speed of recovery following crisis, (2) clinicians may find it difficult to anticipate a patient’s particular symptom trajectory, (3) symptom decline may not be a valid indicator of response to treatment, (4) object relations functioning may not predict response to treatment, and (5) the developmental consequences of childhood trauma may depend on a collection of risk and resilience factors.

The finding that older individuals recover more slowly from acute psychiatric symptoms could be used to inform expectations for treatment. All parties involved in clinical practice would benefit from greater awareness of lifetime patterns in symptom change. First, therapists could adjust the time frames of their treatment plans to reflect differences between older and younger patients. Next, patients could be given more accurate estimations of the course of their symptoms. Finally, third party payers and systems of care could tailor outcome benchmarks to the characteristics of their clients.

Nevertheless, the study demonstrates that rates of symptom change vary widely from person to person. Despite evidence for overall trends, it is difficult to anticipate the course of a single person’s symptoms. This important counter-point reminds all parties involved in treatment to expect uncertainty. While further research is needed to accurately predict symptom trajectories, providers could look to a person’s past symptom patterns for some indication of what to expect in the future.

One challenging implication of the present study is that symptom decline may be a poor indicator of response to treatment. While this conclusion may seem counter-intuitive, it could free providers to identify meaningful treatment goals beyond the alleviation of the most apparent symptoms. This may be particularly relevant for patients
considered treatment-resistant, for whom treatment has failed to provide substantial or lasting gains. For example, rather than focusing on a particular patient’s apparent affect dysregulation, a therapist might choose instead to help the patient learn to tolerate intense affect. Such a change in strategy could lead to more realistic goals that address the very problems that interfere with lasting symptom change.

The present study indicates that patient characteristics such as trauma history and object relations functioning do not predict rates of symptom change. While this may appear to suggest that these characteristics do not affect recovery, a likely alternative merits consideration. The therapist is perhaps the most likely moderator of barriers to treatment, including the consequences of childhood trauma. Therapists adapt their interventions actively to meet their patients’ abilities and limitations. For example, when a patient becomes dysregulated in session due to activation of traumatic memories, the therapist can help to regulate the experience so that he or she can process the trauma. Thus, a history of childhood trauma may not predict poor response to treatment because therapists are able to moderate the consequent difficulties. This possibility suggests it is important for therapists to remain mindful of the ways they accommodate patients’ needs.

Finally, the findings failed to demonstrate the consequences of childhood trauma, suggesting that these consequences may depend on risk and resilience factors not accounted for in the analyses. Such factors may include the variety of traumas a person has experienced, the degree of dysfunction in his or her family system, and the presence of safe and supportive relationships. The developmental consequences of childhood trauma appear to be extremely complex, and it is unwise to assume that the presence of more early, chronic, varied, or severe trauma alone will account for problems seen in
adulthood. Rather, therapists must assess developmental context to understand the lasting implications of a person’s trauma history.

E. Conclusions

Although the present study failed to demonstrate a relationship between childhood trauma and the rate of change in psychiatric symptoms, it did reveal an overall pattern of linear decline in symptom severity following hospital admission. The findings indicated that individual rates of symptom change varied considerably. Contrary to the study hypotheses, only participant age predicted differences in the rate of symptom decline, with younger participants experiencing more rapid decline in symptom severity than older participants. Follow-up analyses did not demonstrate the hypothesized relationship between childhood trauma and impairments in object relations functioning. A number of factors were found to predict the likelihood that a person had been diagnosed with PTSD and BPD. Shortcomings of the present study included the absence of a process measure of treatment response and the failure to include potential moderators of the consequences of childhood trauma. Protective factors in childhood and within treatment merit attention in future empirical and clinical examinations of the consequences of childhood trauma.
Statistics

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|     | 193 | 4.31  | 1.25  | 1.00  | 7.00  |
|     | 193 | 3.98  | 1.11  | 1.00  | 7.00  |

Included in the present study, 191 provided symptom severity ratings at intake and 193 provided responses to the (RAP) used to create Social Cognition and Object Relations (SCORS) ratings. The following variables were ordinal scales: socioeconomic status from 1 (upper class) to 5 (unskilled laborer); trauma onset from 0 (no reported in early childhood), trauma chronicity from 0 (no trauma reported) to 3 (trauma reported in each of CORS dimensions from 1 (extremely pathological) to 7 (ideally healthy).
### Correlations among Predictor Variables

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Correlations are generated at the start of each row, and are identified at the heading of each column by their associated number. *p < .05. **p < .01.
at Each Step in the Construction of the Growth Curve Model

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Conditional model predicting symptom severity over time began with the construction of a basic unconditional model through a series of conditional models (Steps 2 through 5) in which predictors were included and then removed model fit. The coefficients reported here are averages derived from multiple imputations of missing SCORS data. *p < .01, **p < .001.
Multiple Hierarchical Regression Analyses Predicting SCORS Composites

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<th>Cognitive SCORS</th>
<th>Affective SCORS</th>
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<td>β (SE)</td>
<td>β (SE)</td>
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<td>-0.10 † (0.05)</td>
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Separate analyses predicting different composites of the five SCORS dimensions: (1) the overall (2) an average of the two cognitive dimensions, and (3) an average of the three affective dimensions. Standardized regression coefficients accompanied by standard errors. All analyses were conducted with 9 participants (N = 202). *p < .05. **p < .01. ***p < .001. † nearing significance.
Logistical Regression Analyses Predicting BPD and PTSD Diagnoses

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<td>Exp($\beta$)</td>
<td>$\beta$ (SE)</td>
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<td>0.96*</td>
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<tr>
<td>-0.15 (0.71)</td>
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<td>0.10 (0.16)</td>
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<td>-0.40† (0.23)</td>
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<tr>
<td>0.11*** (0.18)</td>
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<td>0.53* (0.14)</td>
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<td>2.60*** (1.02)</td>
<td>7.84</td>
<td>-2.90*</td>
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</table>

Separate analyses predicting the likelihood that participants carried one of two disorders: (1) Borderline Personality Disorder (BPD), and (2) Posttraumatic Stress Disorder (PTSD). The beta ($\beta$) coefficients are accompanied by standard errors and odds ratios. All analyses were conducted on SCORS values for 9 participants (N = 202).

† nearing significance.
Probability of Being Diagnosed with BPD or PTSD for Four Hypothetical Participants

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<td>Female</td>
<td>Married</td>
<td>1.21 (+1 SD)</td>
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Hypothetical participants were chosen in order to represent the range of predicted probabilities of being high and low age and trauma severity values were selected at one standard deviation above and below the
Examining the shape of a logarithmic item characteristics curve. The curve was used to model the presumed relationship between latent trait levels and the likelihood (log-odds) that a person would endorse types of traumatic events either absent or present on the Traumatic Antecedents Scale.
Figure: Change in Symptom Severity over Time for Participants Selected Randomly from the Sample

The figure depicts the change in symptom severity over time for participants selected randomly from the sample.
Figu 1: Change in Symptom Severity over Time predicted by the Final Conditional Model.

The graph shows the decreasing change in symptom severity over time as predicted by the conditional model. The estimated symptom change is based on a female participant of average age (31.2 years), not taking any medication, and with average treatment duration (11.1 months), and no traumatic experiences (2.5 types).
Contrasting Symptom Trajectories across Age and Diagnosis of Borderline Personality Disorder (BPD)

The graph shows estimated symptom trajectories for exemplar older (+1 SD or 41.7 years) and younger (-1 SD or 20.7 years) participants. The BPD-focused graph shows estimated symptom trajectories for participants with (present) and without (absent) a BPD diagnosis. Unless otherwise specified, all estimates are for female participants of average age, not diagnosed with any other psychiatric disorder, with average treatment duration and variety of traumatic experiences.
Contrasting Symptom Trajectories across Trauma Variety and Treatment Duration

![Graph showing symptom trajectories across trauma variety and treatment duration.](image)

Contrasting symptom trajectories across trauma variety and treatment duration. The trauma-focused graph shows estimated symptom trajectories for participants who reported experiencing greater (5) or fewer (1) distinct types of trauma. The treatment-focused graph shows estimated symptom trajectories for participants whose treatment duration was for longer (24 months) or shorter (2 months) periods of time. Unless otherwise specified, all estimates are of average age, not diagnosed with BPD, and with average treatment duration and variety of traumatic experiences.
A Normal Q-Q plot is used to assess normality in the level-1 residuals. Level-1 residuals represent variance in symptom severity for by time. The first graph presents the distribution of level-1 residuals in histogram form. The second graph shows the distribution as a plot of the values that would be expected for a normal distribution across the actual observed values.
To assess variance in the level-1 residuals. The first graph presents a plot of level-1 residuals across the fitted values (y-hat) by the model. The second graph presents a plot of level-1 residuals across time. Note that the level-1 residuals vary more at higher fitted values and less across time. The presence of non-uniform variance is known as heteroscedasticity and suggests that an assumption of the model has been violated.
Plot Used to Assess Normality in the Level-2 Intercept Residuals

The plot used to assess normality in the level-2 intercept residuals. Level-2 intercept residuals represent symptom severity not accounted for by participant characteristics. The first graph presents the distribution of residuals in histogram form. The second graph presents the same distribution as a plot of the values that form a normal distribution across the actual level-2 intercept residuals.
plot used to assess normality in the level-2 slope residuals. Level-2 slope residuals represent variance in
growth not accounted for by participant characteristics. The first graph presents the distribution of level-2
residuals in histogram form. The second graph presents the same distribution as a plot of the values that would be
expected under normality, thus providing a visual assessment of normality in the level-2 slope residuals.
To Assess Bivariate Normality in the Level-2 Residuals

The plot presents a chi-square distribution of the Mahalanobis distance. The observed (Mahalanobis Distance) order was compared to the expected (Chi-Square) distribution.
To assess variance in the level-2 intercept residuals across age and diagnosis. The first graph presents a plot residuals across participant age. The second graph presents a plot of level-2 intercept residuals across evidence (1) of a diagnosis of Borderline Personality Disorder (BPD). There is no apparent indication of a either graph.
to assess variance in the level-2 intercept residuals across treatment duration and trauma variety. The first graph shows a plot of level-2 intercept residuals across the duration of participants’ hospitalizations. The second graph depicts the level-2 intercept residuals across the number of different types of trauma reported by participants. There is no indication of a linear relationship in either graph.
to assess variance in the level-2 slope

The graph presents a plot of level-2 slope participant age. There is no apparent indication in the graph.
APPENDIX A

TRAUMATIC ANTECEDENTS INTERVIEW (TAI) SCORING FORM

Traumatic Antecedents Interview (TAI) Scoring Form-#3 Revised

<table>
<thead>
<tr>
<th>Subject</th>
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<tbody>
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<table>
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<table>
<thead>
<tr>
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**DOMAIN:**

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<tr>
<th>Variable</th>
<th>CHILDHOOD AGE PERIOD</th>
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<tbody>
<tr>
<td></td>
<td>I. 0-6</td>
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<td></td>
<td>II. 7-12</td>
</tr>
<tr>
<td></td>
<td>III. 13-18</td>
</tr>
<tr>
<td></td>
<td>[values]</td>
</tr>
</tbody>
</table>

**A. ABUSE:**

1. Physical [0,1,2,3...]
2. Sexual [0,1,2,3...]
3. Witnessing Violence [0,1,2,3...]

**B. NEGLECT:**

1. Physical [0,1]
2. Emotional [0,1]

**C. SEPARATIONS/LOSSES:**

1. Significant Separations [0,1,2,3...]
2. Losses [0,1,2,3...]

**D. DOMESTIC CHAOS** [0,1]

**E. VERBAL ABUSE** [0,1,2,3...]

**F. CARETAKERS/CONFIDANTES** [0,1,2]

**G. PARENTAL MARITAL RELATIONSHIP**

1. Discord/Frequent Arguments [0,1]
2. Mutual Affection Displayed [0,1]
APPENDIX B

SOCIAL COGNITION AND OBJECT RELATIONS SCALE (SCORS)

Social Cognition and Object Relations Scale (SCORS)
Global Rating Method

Raters score narrative data with respect to the following domains on a seven point Likert scale.

**Complexity of representation of people:**
1. Is egocentric, or sometimes confuses thoughts, feelings, or attributes of the self and others
2. Tends to describe people’s personalities and internal states in minimally elaborated, relatively simplistic ways, or splits representations into good and bad
3. Representations of the self and others are stereotypical or convention, is able to integrate both good and bad characteristics of self and others, has awareness of impact on others
7. Psychologically minded, insight into the self and others, differentiated and shows complexity

**Affective quality of representations** (i.e., expectations and experiences in relationships):
1. Malevolent, abusive, caustic
3. Largely negative or unpleasant, but not abusive
4. Affective quality of the narrative is absent, bland or limited
5. Mixed, neither primarily positive nor primarily negative (some positive)
7. Generally positive expectations of relationships (but not pollyannish), a favorable and affirmative view of relationships

**Emotional investment in relationships:**
1. Tends to focus primarily on his or her own needs in relationships, has tumultuous relationships, or has few if any relationships
2. Only one character is described and no relationship is depicted
3. Somewhat shallow relationships, or only alludes to others
5. Demonstrates conventional sentiments of friendship, caring, love, and empathy
7. Tends to have deep, committed relationships with mutual sharing, emotional intimacy, interdependence, and respect, positive connectedness and appreciation of others

**Understanding of social causality:**
1. Narrative accounts of interpersonal experiences are confused, distorted, extremely sparse, or difficult to follow, limited awareness and coherence
2. Describes interpersonal events as if they just happen, with little sense of why people behave in the way they do (i.e., alogical stories that seem to lack a causal understanding)
3. Understands people in relatively simple, but sensible ways, or describes interpersonal events in ways that largely make sense but may have a few gaps or incongruities
5. Tends to provide straightforward narrative accounts of interpersonal events in which people’s actions result from the way the experience or interpret situations
7. Tends to provide particularly coherent narrative accounts of interpersonal events, and to understand people well, understands the impact behavior on others

**Experiences and management of aggressive impulses:**
1. Physically assaultive, destructive, sadistic, or in poor control of aggressions, impulsive
3. Angry, passive-aggressive, denigrating, or physically abusive to self (or fails to protect self)
4. No anger content in the story
5. Avoids dealing with anger by denying it, defending against it, or avoiding confrontations
7. Can express anger and aggression and assert self appropriately
APPENDIX C

RELATIONSHIP ANECDOTE PARADIGM (RAP) INTERVIEW FORM

Relationship Anecdote Paradigm (RAP)

Interview Form

“Now I would like to you to tell me about some stories of your interactions with others. I’m interested in interactions with others that you found important, interesting, or troublesome. They may be day-to-day interactions or interactions that are more unusual or unique.” The emphasis is on eliciting specific interactions (e.g., what the subject said, what the other person said, etc.), and the subject is asked to tell what happened, when it occurred, how it started, and how it ended. In addition, subjects are asked why they did or said that they did, why they think the other person responded as they did, how they felt about how things came out, how the interaction affected them, what they wanted differently, and what they wished they had said or done differently.
APPENDIX D

DATA SHARING AND CONFIDENTIALITY AGREEMENT

RELATED STUDY
DATA SHARING AND CONFIDENTIALITY AGREEMENT

This Related Study Data Sharing and Confidentiality Agreement (the “Agreement”) between Austen Riggs Center, Inc. (“Riggs”) and Sandro Piselli made as of March 26, 2010.

Background

A study utilizing data from the Austen Riggs Center databank was duly approved by an Independent Review Board (“IRB”) on August 6, 2010 (the “Related Study”). The IRB’s approval is attached hereto as Exhibit A. The estimated completion date of the Related Study is August 6, 2011.

Sandro Piselli, MA has been appointed by the Director of Research of Riggs to work in collaboration with J. Christopher Fowler, PhD on the Related Study for a one-year term as a Research Associate-Level 2.

This Agreement sets forth the confidentiality, security and other obligations of Sandro Piselli respecting the data and other records related to the Austen Riggs Center databank and the Related Study.

Agreement

1. Obligation to Maintain Confidentiality and Security of Data. Each participant in the Austen Riggs Center databank was provided with written assurance that all information gathered from them would be held strictly confidential. Sandro Piselli is bound by this assurance to participants in the Study.

1.1 Privacy and Security Rules Respecting PHI. To ensure that such confidentiality and security is maintained, and to ensure compliance with federal and state law respecting use of protected health information (“PHI”) (as that term is defined by the implementing regulations, 45 C.F.R. Part 160 and Part 164, Subparts A and E, Standards for Privacy of Individually Identifiable Health Information (Privacy Rule) and Subpart C, Security Standards for the Protection of Electronic Protected Information (Security Rule) of the Health Insurance Portability and Accountability Act of 1996), Sandro Piselli agrees as follows:

(a) to use or further disclose PHI only as permitted or required under the auspices, and according to the rules of the Austen Riggs Center databank or as required by law;
(b) to use appropriate safeguards to prevent use or disclosure of PHI other than as provided for by this Agreement;

(c) to the extent practicable, to mitigate any harmful effect that is known to Sandro Piselli of a use or disclosure of PHI in violation of this Agreement;

(d) to immediately report to Riggs any use or disclosure of PHI not provided for by this Agreement of which Sandro Piselli becomes aware;

(e) to make PHI available to employees, independent contractors or other agents of Sandro Piselli only (i) with Riggs’ prior written consent, and (ii) after any such employees, contractors or agents have executed an agreement containing the same restrictions and conditions that apply to Sandro Piselli under this Agreement, copies of which agreement(s) shall be delivered to Riggs prior to any such dissemination of PHI;

(f) upon request, to respond to any inquiry from governmental authorities respecting Sandro Piselli practices relating to the use or disclosure of PHI;

(g) within ten (10) days of receiving a request from Riggs, to make available the information necessary for Riggs to make an accounting of disclosures of PHI as may be required by law;

(h) to implement administrative, physical and technical safeguards that reasonably and appropriately protect confidentiality, integrity and availability of any electronic PHI in accordance with the provisions of 45 C.F.R. §164.314;

(i) to report promptly to Riggs any security incident that Sandro Piselli becomes aware of in accordance with the provisions of 45 C.F.R. 164.314; and

(h) to otherwise promptly comply with reasonable requests from Riggs concerning Sandro Piselli’s use of any PHI obtained through the Study, including, without limitation, promptly returning to Riggs upon its request all copies of any data and records that contain PHI.

1.2 **Adherence to Technical Procedures.** Without limiting the foregoing, Sandro Piselli agrees to comply with the Technical Procedures for Assuring Confidentiality and Security of Study Data attached as Exhibit B, as such Technical Procedures may be amended from time to time, in Riggs’ reasonable discretion.

2. **Rights Respecting Use and Ownership of Data/Work Product**

2.1 **Ownership and Preservation of Austen Riggs Center databank/Work Product.** Unless otherwise agreed by Riggs, all data relating to the Austen Riggs Center databank shall remain at Riggs and shall be maintained in a secure location as long as practically possible. Such data shall include, without limitation, the audio and audiovisual tapes, and transcripts of all Austen Riggs Center databank participants. Any
and all copyright in such materials, as well as existing arrangements, ratings and scalings of any such data, in whatever medium, (including, without limitation, the raw data computer files) shall belong to Riggs.

2.2 Access to Austen Riggs Center databank / Work Product. Sandro Piselli shall be allowed access to Austen Riggs Center databank and work product solely for purposes of the Related Study.

2.3 Publication of Related Study. Sandro Piselli may publish findings of the Related Study only with the prior written consent of the Director of Research of Riggs and in collaboration with Riggs, and provided that Riggs is appropriately acknowledged by stating that such work was “Supported by a grant from the Austen Riggs Center” or such other words of acknowledgement as the parties may agree. In any such publication, Sandro Piselli shall strictly adhere to all applicable law, professional standards, and ethical principals so as not to risk disclosure of any information that might identify any subject of the Austen Riggs Center databank or Related Study without their express consent.

2.4 Use by Riggs of Related Study Materials. Copies of all new ratings that may be completed by Research Associate shall be provided to Riggs following completion of the Related Study, together with any manuals used in connection with those ratings. Sandro Piselli shall provide Riggs with copies of any new ratings completed by Sandro Piselli, together with any manuals used in connection with those ratings. Riggs shall have the right to use all ratings completed in any Related Study, including, without limitation the right to include such ratings in published research or presentations, subject to any rights of first publication of the author of such ratings.

2.5 Professional Standards. Nothing herein is intended to relieve Sandro Piselli of any ethical obligations that may be imposed upon Sandro Piselli by any review or licensure board that may have jurisdiction over Sandro Piselli respecting the conduct of the Research Study.

The parties have signed this Agreement as of the date shown above.

RESEARCH ASSOCIATE-LEVEL 2

______________________________
Sandro Piselli, MA

DIRECTOR OF RESEARCH

______________________________
J. Christopher Fowler, Ph.D.
REFERENCES


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