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The Impact of Change in Self-Compassion, Psychological Inflexibility, and Interpersonal
Courage in PTSD Treatment

by

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A Dissertation submitted to the Faculty of the Graduate School,

Marquette University,

in Partial Fulfillment of the Requirements for

the Degree of Doctor of Philosophy

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ABSTRACT

The Impact of Change in Self-Compassion, Psychological Inflexibility, and Interpersonal Courage in PTSD Treatment

Peter P. Grau, B.A., M.S.

Marquette University, 2021

Despite numerous advances in the field's understanding of the development and maintenance of posttraumatic stress disorder (PTSD), current treatments are often limited in effectiveness due to a narrow focus on symptom reduction (Cloitre, 2015; Yehuda et al., 2015). One reason for this issue is that the impact of PTSD extends into areas such as interpersonal relationships, pursuit of valued activities, and acceptance of the self (Cloitre, 2015). These processes appear to be especially relevant in chronic/complex PTSD, which is rarely represented in controlled trials (Cloitre, Miranda, Stovall-McClough, & Han, 2005). As such, PTSD research with an expanded focus beyond symptom reduction, especially research conducted in clinical settings, is likely to be especially relevant to both PTSD researchers and clinicians working in the field. To that end, this study examined the impact of change in self-compassion, psychological inflexibility, and interpersonal courage on PTSD symptom reduction, trauma-related shame, quality of life, and valued living in an exposure-based PTSD partial hospitalization program ($n = 537$; 75% White; 83% Female; mean age = 36). Latent growth curves, including intercepts and slopes (linear, quadratic, cubic, and spline), were estimated for all variables under study. All measures except for interpersonal courage showed clinically meaningful change over the course of the program. At $p < .001$, latent regressions showed that only admission psychological inflexibility scores predicted discharge PTSD symptoms and that only admission interpersonal courage ($p = .032$) was related to discharge quality of life. For the latent growth curves, three-piece spline models were retained for PTSD symptoms, self-compassion, and valued living. Linear models were retained for psychological inflexibility, quality of life, interpersonal courage, and trauma-related shame. For the three PTSD slopes and three valued living slopes, only the three self-compassion slopes were significant predictors ($p < .001$). The psychological inflexibility slope predicted both the quality of life slope ($p < .001$), while the interpersonal courage slope predicted the trauma-related shame slope ($p < .001$). Results demonstrate that focusing on processes such as psychological inflexibility, self-compassion, and interpersonal courage might increase the efficacy of PTSD treatment in addressing the broad range of concerns present in the disorder.

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Peter P. Grau, B.A., M.S.

As I finished this project, it occurred to me that I have (hopefully) produced something that demonstrates the importance of compassion, connection with others, and values-driven action in trauma treatment. As a particularly lovely parallel process, I realized that my friends and family have embodied these concepts throughout my doctoral education; I cannot imagine this journey without them. I want to thank my parents for their unending support, all of my mentors for seeing my potential and pushing me to find myself, and my wonderful friends for allowing me to be the person I found. Finally, I want to thank my wife, Rachel, for continuing to be my inexhaustible spark.

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The Impact of Change in Self-Compassion, Psychological Flexibility, and Interpersonal Courage in a PTSD Treatment Program

Chapter One

Introduction

The study and treatment of posttraumatic stress disorder (PTSD) is a complex and ever-evolving topic. While the diagnosis of PTSD originated only 39 years ago in the third edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-III), human beings have dealt with the impact of experiencing extremely stressful, traumatic events throughout history (Pomerantz, 2017). In fact, the experience and threat of trauma has played an important role in human evolution, affecting the sympathetic nervous system and related defense strategies (e.g., avoidance, attentive immobility, aggressive defense, appeasement; Gilbert, 2010).

Historically, symptom clusters now contained under the PTSD diagnostic umbrella went by many names. Terms like soldier's heart, war neurosis, shell shock, and battle fatigue were all common during various historical periods, though conceptually they all shared the assumption that experiencing psychological and physiological distress in response to extreme stress was due to an innate characterological weakness (Pomerantz, 2017). In the years leading up to the publication of the DSM-III, researchers began to challenge this assumption, and with the creation of the PTSD diagnosis, focus shifted to the traumatic event as the etiological basis for subsequent symptoms (Keane, Wolfe, & Taylor, 1987). Within this diagnostic framework, the first criterion for a PTSD diagnosis was clinician assessment of whether a client experienced one of several events

that were determined to be traumatic by the authors of the DSM (e.g., war, natural disasters, sexual assault; Pomerantz, 2017).

Since 1980, there has been a large shift away from experts' definitions of what is "traumatic" to a focus on clinician assessment of whether an individual experienced, witnessed, or learned about real or threatened danger stemming from an event (Pomerantz, 2017). Because of the inclusion of threatened danger into the DSM-IV diagnostic criteria, it is now accepted that a person's subjective experience of a potentially traumatic event is a major determinant of the development of PTSD-related symptoms (Creamer, McFarlane, & Burgess, 2005). As a result, in trauma-focused treatments, the subjective experience of a traumatic event is placed at the core of the cognitive and emotional processing (Powers, Halpern, Ferenschak, Gillihan, & Foa, 2010). Over the past 39 years, research has allowed for more targeted treatments by including anhedonic and dysphoric symptom clusters under the PTSD diagnostic umbrella, as well as unique presentations related to one's culture (Armour et al., 2015; Rasmussen, Keatley, & Joscelyne, 2014). Ultimately, the field's understanding of PTSD has evolved, culminating in a shift away from a strict classification as an Anxiety Disorder into a new, separate diagnostic category in the fifth edition of the DSM (DSM-5; Friedman, 2013).

Clinical Outcomes

In the face of significant advances in the field's understanding of PTSD, current conceptualizations of PTSD etiology, course, and appropriate treatments are hotly debated (Ehlers et al., 2010; Wampold et al., 2010). Despite the longstanding history of the field of psychology acknowledging the deleterious impacts of trauma on

psychological well-being (van der Kolk, 2000), many researchers have noted a lack of outcome data from PTSD treatment programs across trauma-affected populations (McGuigan, 2013; Najavits & Hien, 2013; Steenkamp & Litz, 2013; Cloitre, 2015).

A lack of clinical outcome data is concerning for multiple reasons. First, the prevalence rates of PTSD across the world are higher than previously anticipated. The National Comorbidity Survey Replication (NCS-R; Kessler et al., 2005) is a nationally representative, face-to-face, epidemiological, diagnostic survey of English-speaking United States residents. Using the World Mental Health (WMH) survey initiative version of the World Health Organization (WHO) Composite International Diagnostic Interview (CIDI; Kessler & Üstün, 2004), interviewers from the University of Michigan found the prevalence of PTSD to be approximately 3.6% in men and 9.7% in women (Kessler et al., 2005). Given the current prolonged state of warfare engagement, incidence rates of PTSD are expected to increase among members of the United States military in the coming years (Bardhoshi et al., 2016). In conflict zones, this rate increases dramatically, as can be seen in the epidemiological, diagnostic study conducted by De Jong, Komproe, Spinazzola, van der Kolk, and Van Ommeren (2005) with participants from Ethiopia (n = 1,200), Algeria (n = 652), and Gaza (n = 585). In these regions, scores on the Structured Interview for Disorder of Extreme Stress indicated that 37% of Algerian participants, 18% of Gazan participants, and 16% of Ethiopian participants would likely meet criteria for a diagnosis of PTSD. These numbers indicate large numbers of people across the world are in need of effective treatment, especially given the low rate of naturalistic PTSD remission and the large cost burden of chronic PTSD to both the individual and society (Galatzer-Levy et al., 2013; Zatzick et al., 1997).

The second reason a lack of outcome data for PTSD treatment programs is concerning are the well-documented consequences of failing to inform clinical practice with clinical data (Spinazzola, Blaustein, & van der Kolk, 2005). Without consistently gathered patient outcome data, the quality, consistency, and accountability of care environments cannot be reliably determined (Brown, Burlingame, Lambert, Jones, & Vaccaro, 2001). There is also evidence that without measuring clinical effectiveness, therapist skill can deteriorate, which results in poor delivery of service and, potentially, the inability to demonstrate value to customers (e.g., taxpayers and patients; Fortney et al., 2017). Conversely, the benefit of conducting comprehensive outcome research and using that research to inform practice is clear (Prescott, Maeschalck, & Miller, 2017). Measurement-based care (MBC) can help providers identify high-risk patients, foster coordination between care team members, and improve the therapeutic relationship (Fortney et al., 2017). Most importantly, the collection of appropriate outcome data can be used to inform treatment results in both symptom reduction (Prescott, Maeschalck, & Miller, 2017) and improve quality of life (Scott & Lewis, 2015).

Processes of Well-being Over Time

While it is essential that PTSD research continue to assess both symptom reduction and quality of life, an important area of emerging research concerns the impact of change in core processes of well-being on PTSD outcomes. It is not enough to understand whether PTSD treatment impacts both symptom severity and quality of life; research also needs to explore how change in processes of well-being over the course of treatment impact relevant outcomes (Hofmann & Barlow, 2014). Identifying which processes to examine requires a thorough conceptualization of well-being broadly and an

explication regarding how these processes function to promote or restrict the cultivation of well-being in people with PTSD. A full discussion of relevant literature is included in the next chapter, though it is clear that more work needs to be done before any firm conclusions can be reached confidently.

In the absence of definitive research, several findings have emerged that suggest processes of well-being that appear to be prime candidates for further exploration. These processes include psychological flexibility, emotion regulation, present-moment awareness, self-compassion, and interpersonal vulnerability and connection (Boyd, Lanius, McKinnon, 2018; Gu et al., 2015; Hoffart, Økstedalen, & Langkaas, 2015). The evidence in support of present-moment awareness (e.g., Boyd et al., 2018) and emotion regulation (Barlow, 2014) as core processes of well-being in PTSD is established and strong. There is also evidence that exposure-based treatments are especially effective at addressing emotion regulation and present-moment awareness, as the movement towards willingly tolerating distressing emotions in the moment builds up emotion regulation skills behaviorally and neurobiologically (Frank et al., 2014; Gratz & Tull, 2010; Yehuda et al., 2015).

The importance of psychological flexibility, self-compassion, and vulnerability and connection in interpersonal relationships on PTSD outcomes is less clear. Evolutionary psychologists have hypothesized that compassion towards others, compassion towards self, and receiving compassion from others (Gilbert, 2010) are core components of multi-level evolutionary selection that allows human beings to successfully function in groups (Hayes, 2019). From this perspective, creating a context in which interpersonal connection can occur is likely to facilitate the cultivation of well-

being. This view has been supported by findings that people with PTSD have experienced improvements in quality of life following an explicit focus on positive interpersonal interactions (e.g., Markowitz et al., 2015). Importantly, there is also evidence that for positive interpersonal interactions to occur, a willingness to engage in interpersonal interactions characterized by vulnerability must be present (Jordan, 1990; Kohlenberg et al., 2015; Reis & Shaver, 1988). Within this evidence is a pattern that suggests courage to engage in interpersonal relationships might be a key process of well-being, an approach which is integrated in evidence-based treatment (e.g., addressing intimacy and trust in Cognitive Processing Therapy [CPT]), yet not fully explored in the current PTSD literature (Cloitre, 2015).

This focus on interpersonal connection and interpersonal courage has often been neglected in traditional treatment models for PTSD, as the need to reduce core PTSD symptoms (e.g., avoidance, hyperarousal) has taken precedence (Cloitre, Miranda, Stovall-McClough, & Han, 2005). Indeed, this symptom reduction approach targets essential components of the PTSD experience, and contemporary evidence-based approaches (e.g., Prolonged Exposure for PTSD, CPT) have considerable evidence supporting their ability to reduce PTSD symptomatology (Barlow, 2014; Cusack et al., 2016). However, these evidence-based approaches focus on restructuring dysfunctional beliefs about self, others, and the world, whereas altering relationships between fear-triggering stimuli and associated networks via habituation have struggled to consistently provide positive results with respect to quality of life for people with PTSD who endorse high levels of shame and difficulties connecting with others (Au et al., 2017; Cloitre, 2015; Green et al., 2017).

The experience of shame inherently limits the ability of the individual to connect with others, as it is based in strongly held belief that one's identity is flawed, wrong, or broken (Lee, Scragg, & Turner, 2001). One of the most promising interventions for targeting shame in PTSD is the cultivation of self-compassion. The definition of self-compassion most commonly seen in the literature originated from Neff (2003). In this definition, self-compassion comprises three components: (1) self-kindness vs. self-criticism, (2) common humanity vs. isolation, and (3) mindfulness vs. overidentification. According to Neff and Dahm (2016), "In order to give oneself compassion, one must be able to turn toward, acknowledge, and accept that one is suffering" (p. 2). In some ways, this definition of self-compassion provides an alternative definition for shame, and validity comparison of self-compassion scales have demonstrated the link between the "negative" aspects of self-compassion (e.g., self-criticism) and shame (Kelly, Carter, & Borairi, 2014).

Evidence in support of interventions designed to increase self-compassion and decrease shame is moderate in strength, yet growing (Neff & Dahm, 2016). These approaches fill an important gap in the literature; shame has been effectively targeted cognitively and behaviorally in empirically-supported treatments such as CPT with relatively consistent levels of success (Schoenleber & Gratz, 2018), though the reduction of reported levels of shame is not necessarily equivalent to active engagement in connection with self and others. Additionally, there is some evidence to suggest that change in these processes may be best explained by change in psychological flexibility (e.g., Levin, Hildebrandt, Lillis, & Hayes, 2012; Meyer et al., 2018; Seligowski, Miron, & Orcutt, 2015).

Given the previous discussion, clarifying the relationships between psychological flexibility, self-compassion, and interpersonal courage in people with PTSD is an important empirical and clinical goal. As has been noted by Cloitre (2015), these variables should not only be assessed by their relationship to PTSD symptom severity. Quality of life (Steenkamp & Litz, 2013) is an important clinical target, as is trauma-related shame (Neff & Dahm, 2016) due to evidence that suggests high levels of shame are often a barrier to successful PTSD treatment (Kelly et al., 2014). Finally, recent evidence suggests that evidence-based treatments for PTSD can benefit from including core values (e.g., connection with others, fulfilling family relationships) as a motivational and behavioral target (Donahue, Huggins, & Marrow, 2017). A focus on these outcome variables provides a comprehensive and holistic list of clinical targets that can be explored in treatment-seeking populations.

The Present Study

The present study attempted to explore how self-compassion, psychological flexibility, and interpersonal courage functioned as predictors of PTSD symptom severity, quality of life, trauma-related shame, and valued living using clinical outcomes data obtained from participants who completed treatment at a Prolonged Exposure-based partial hospitalization program for PTSD. Using latent growth curve analyses (Little, 2013; Grimm, Ram, & Estabrook, 2016), this study examined if within-person rates of change in self-compassion, psychological flexibility, and interpersonal courage predicted the within-person rates of change in PTSD symptom severity, quality of life, trauma-related shame, and valued living. To complete this task, we tested the null hypothesis that there was no difference between the slopes of the rates of change for each predictor

variable (Grimm, Ram, & Estabrook, 2016). We also assessed the ability of baseline scores for predictor variables to predict scores on our outcome variables at discharge. Additionally, reliable change estimates were calculated for the variables under study. Type of trauma (impersonal vs. interpersonal) was explored as a covariate. Finally, we completed an intent-to-treat analysis to compare (via demographic characteristics and scores on the study measures) participants who completed at least two timepoints with participants who dropped out of treatment before the second timepoint. A full literature review focused on the relationships between predictor and outcome variables is included in Chapter 2, though studies that have explored these relationships were cited for each hypothesis.

PTSD Rate of Change Hypotheses (H1)

We hypothesized that the within-person rate of change (increase) in self-compassion would predict the within-person rate of change (decrease) in PTSD symptom severity (Au et al., 2017; H1a). We hypothesized that the within-person rate of change (decrease) in psychological inflexibility would predict within-person rate of change (decrease) in PTSD symptom severity (Meyer et al., 2018; H1b). We hypothesized that within-person rate of change (increase) in interpersonal courage would predict within-person rate of change (decrease) in PTSD symptom severity (Jordan, 1990; H1c).

Quality of Life Rate of Change Hypotheses (H2)

We hypothesized that the within-person rate of change (increase) in self-compassion would predict the within-person rate of change (increase) in quality of life (Neff & Dahm, 2016; H2a). We hypothesized that the within-person rate of change (decrease) in psychological inflexibility would predict within-person rate of change

(increase) in quality of life (Levin et al., 2012; H2b). We hypothesized that within-person rate of change (increase) in interpersonal courage would predict within-person rate of change (increase) in quality of life (Jordan, 1990; H2c).

Trauma-related Shame Rate of Change Hypotheses (H3)

We hypothesized that the within-person rate of change (increase) in self-compassion would predict the within-person rate of change (decrease) in trauma-related shame (Au et al., 2017; H3a). We hypothesized that the within-person rate of change (decrease) in psychological inflexibility would predict within-person rate of change (decrease) in trauma-related shame (Levin et al., 2012; H3b). We hypothesized that within-person rate of change (increase) in interpersonal courage would predict within-person rate of change (decrease) in trauma-related shame (Kelly, Carter, & Borairi, 2014; H3c).

Valued Living Rate of Change Hypotheses (H4)

We hypothesized that the within-person rate of change (increase) in self-compassion would predict the within-person rate of change (increase) in valued living (Meyer et al., 2018; H4a). We hypothesized that the within-person rate of change (decrease) in psychological inflexibility would predict within-person rate of change (increase) in valued living (Gloster et al., 2011; H4b). We hypothesized that within-person rate of change (increase) in interpersonal courage would predict within-person rate of change (increase) in valued living (Levin et al., 2012; H4c).

Treatment Completion Hypotheses (H5)

We hypothesized that treatment completion rates would not vary based on admission symptom measure scores or demographic variables including race, gender, marital status, or education level.

Interpersonal Trauma Hypotheses (H6)

We hypothesized that people who experienced interpersonal trauma would experience smaller effect size change in PTSD symptom severity than people who did not experience interpersonal trauma (e.g., motor vehicle accident, natural disaster, industrial accident; Cloitre, 2015; H6a). We hypothesized that people who experienced interpersonal trauma would experience smaller effect size change in trauma-related shame than people who did not experience interpersonal trauma (Cloitre, 2015; H6b). We hypothesized that people who experienced interpersonal trauma would experience smaller effect size change in quality of life than people who did not experience interpersonal trauma (Cloitre, 2015; H6c). We hypothesized that people who have experienced interpersonal trauma would experience smaller effect size change in valued living than people who did not experience interpersonal trauma (Levin et al., 2012; H6d).

Discharge Scores Hypotheses (H7)

We hypothesized that admission psychological inflexibility scores would be the only significant predictor of discharge PTSD symptom severity (Meyer et al., 2018; H7a). We hypothesized that admission psychological inflexibility scores would be the only significant predictor of discharge quality of life scores (Shenk et al., 2014; H7b). We hypothesized that admission self-compassion scores would be the only significant predictor of discharge trauma-related shame scores (Au et al., 2017; H7c). We

hypothesized that admission psychological inflexibility scores would be the only significant predictor of discharge valued living scores (Levin et al., 2012; H7d).

Chapter 2

Literature Review

PTSD Etiological Considerations

PTSD is one of the few disorders in which an etiological agent can be identified (Cloitre, 2015). This is represented in criterion “A” for in the DSM-5 diagnostic criteria: “exposure to actual or threatened death, serious injury, or sexual violence...” (American Psychiatric Association, 2013, p. 271). The ways in which one can be exposed to actual or threatened death, serious injury, or sexual violence have expanded with each iteration of the DSM (Cusack et al., 2016), and now include directly experiencing the traumatic event, witnessing the traumatic event in person, hearing about a violent or accidental traumatic event occurring to close family member or friend, or experiencing repeated exposure to details concerning a traumatic event (American Psychiatric Association, 2013).

The presence of an identifiable etiological basis for PTSD allows for targeted treatments (e.g., processing the specific way in which someone encountered the traumatic event), yet, as can be seen in the wide variety of ways in which someone can encounter a traumatic event, the symptom presentation of PTSD is complex and difficult to predict (Cloitre, 2015). How to deal with this complexity has been and continues to be a point of contention in the PTSD literature (Ehring et al., 2014). One proposed differentiating line can be found in the complex PTSD (cPTSD) literature, which has attempted to create a new diagnostic category (cPTSD) that expands beyond classic PTSD symptoms and includes “additional symptoms including disturbances of affect, self, and interpersonal relationships” (De Jongh et al., 2016, p. 361). Importantly, one of the core assumptions of

the cPTSD diagnostic category is that specific traumatic stressors (prolonged traumatic exposure in the case of cPTSD) result in different causal impacts (Resick et al., 2012).

This belief, though common, has not held up under scrutiny, but only in terms of creating a new diagnostic category (De Jongh et al., 2016). Impacts beyond disorder-specific symptoms have been shown to be different according to the type of traumatic exposure. One such example is that survivors of sexual assault experience a slower return to baseline functioning following a sexual assault than disaster survivors, single-incident physical injury victims, and first responders (Steenkamp et al., 2012). However, the identified symptom presentation across a range of traumatic exposures consistently fall within the umbrella of the PTSD diagnostic criteria (Cloitre, 2015). A review of the cPTSD literature completed by Resick et al. (2012) concluded that, while there is not currently enough research to warrant the creation of a new diagnostic category for cPTSD, the heterogeneity of symptoms of functional impacts within the disorder suggests the need for better assessment and study of the impact of experiencing different types of trauma.

In terms of PTSD outcomes assessment, one of the messages emphasized in Resick et al.'s (2012) and later De Jongh et al.'s (2016) reviews of the cPTSD literatures is that new developments in etiological understanding of PTSD, especially in differentiating different courses based on predisposing factors or type of trauma encounter, must be taken into account when assessing PTSD outcomes. Failure to contextualize PTSD outcomes through the person's trauma history and demographic characteristics ignores a large and growing literature base that shows the dynamic nature of trauma impact across the lifespan (Resick et al., 2012).

This etiological discussion, while not the focus of this paper, is important to place several assessment trends into context. Currently, the heterogeneous nature of PTSD presentation and course can be overwhelming in terms of treatment and outcome assessment (Cloitre et al., 2005) and many studies have failed to provide basic methodological information (trauma exposure history, demographics, study exclusion criteria) that could be useful in helping to understand the course of PTSD across a variety of treatment modalities and pre-trauma factors (Spinazzola, Blaustein, & van der Kolk, 2005). While an in-depth discussion of the etiology of PTSD is beyond the scope of this paper, reviewing the evidence behind the etiological ambiguity of the disorder clarifies the criticisms of reporting methodological information in the PTSD treatment outcomes literature.

Models of PTSD Development

In addition to a brief overview of the literature on PTSD etiological considerations, it is important to review the major theoretical models from which empirically supported PTSD treatments are derived. In this section, conditioning models, cognitive models, and emotional processing models will be reviewed with respect to the development and maintenance of PTSD.

Conditioning Model

The conditioning model is one of the first models of PTSD development and maintenance and is a strictly behavioral theory based on the Mowrer's (1960) two-factor theory in which an aversive stimulus (i.e. a traumatic event) is classically conditioned to be associated with environmental stimuli, which are then avoided (Pomerantz, 2017). The temporary reduction in anxiety via the avoidance of trauma-related stimuli then

reinforces avoidance, which maintains the fear of trauma-related stimuli (Green et al., 2017). Higher-order conditioning and stimulus generalization then form new trauma-related associations that branch off the original conditioned reactions. The literature suggests the conditioning model accurately portrays the maintenance of avoidance in anxiety, though its applicability for PTSD is questionable (Barlow, 2014; Green et al., 2017). Specifically, it is not able to account for many of the cognitive or emotional elements of PTSD, including dissociation, flashbacks, and negative alterations in cognitions and mood (Green et al., 2017; De Jongh et al., 2016).

Emotional Processing Model

Following in the footsteps of the conditioning model, the emotional processing model (Foa & Kozak, 1991) asserts that PTSD develops when fear networks, which are adaptive in many instances (e.g. avoiding dangerous stimuli), are generalized to non-threatening stimuli and become maladaptive (Green et al., 2017). Maladaptive fear structures can include physiological reactivity (e.g., racing heart), behavioral responses (e.g., exaggerated startle response), reexperiencing (e.g., flashbacks to the traumatic event), and negative appraisals of self, others, and the world (Ehring et al., 2014; Green et al., 2017). In the emotional processing model of PTSD, attentional bias towards negative appraisals of neutral stimuli lead to the reinforcement of anxiety and avoidance, which is a core mechanism of PTSD development and maintenance (Barlow, 2014). Treatment then requires modification of both dysfunctional schematic beliefs about the world and maladaptive fear networks (Foa et al., 2008). These fear networks are modified through integrating new, safe learning that is incompatible with the preexisting maladaptive associations, which is one of the core components of PE for PTSD (Barlow, 2014), the

evidence for which will be discussed in a later section. While this model represented a more sophisticated view of PTSD symptom development and maintenance, it did not adequately address flashbacks, dissociation, or nightmares (Green et al., 2017) and as such, another PTSD model was developed.

Cognitive Model

To address the limitations of the conditioning model and the emotional processing model, Ehlers and Clark (2000) suggested a model of PTSD based on the individual cognitively appraising a potentially traumatic event as a serious, continued threat. In the cognitive model, this sense of threat evolves based on negative appraisals related to the traumatic event (e.g. negative views of self, others, and the world) and inability to integrate trauma-related memories into autobiographical memory (Ehlers & Clark, 2000). Trauma-related memories are fragmented, and thus are difficult to recall in some contexts but are easily triggered when faced with trauma-related stimuli (i.e. intrusive thoughts, nightmares, and flashbacks; Green et al., 2017). This theory is widely influential in the literature and has a great deal of evidence to support its validity, especially with respect to the development and maintenance of trauma-related cognitions and memories (Green et al., 2017; Ehlers et al., 2010). At this point in time, the cognitive model of PTSD is incorporated into most effective models of PTSD treatment, most notably CPT (Green et al., 2017; Powers et al., 2010).

Common Features and Shortcomings

Together, all three models allow for an increasingly complete understanding of PTSD, and in many respects, are compatible and complementary (Green et al., 2017; Karlin et al., 2010). However, one of the main elements that each theory fails to fully

address, despite the findings from the PTSD etiology literature, is how to account for different levels of vulnerability to PTSD across individuals (Green et al., 2017).

Prognostic Indicators and Risk Factors Assessment

As can be seen from this discussion, to place treatment outcome results in a proper context, it is important to assess identified risk factors for PTSD development and ongoing symptom severity. One important gray area to note in the literature is the discussion surrounding the impact of traumatic exposure versus other risk factors. It should be noted that it is widely accepted that certain factors increase the likelihood that someone will develop PTSD. For example, there is a direct, dose-response relationship between an individual's perceived severity and duration/prolongation of a traumatic exposure and the risk for PTSD development (Sareen, 2014). It is unclear if this is the causal mechanism behind the higher incidence of PTSD development in people with a history of childhood sexual abuse (CSA). Regardless, experiencing CSA is one of the top prognostic indicators of developing PTSD later in life (De Jongh et al., 2016).

Peri-traumatic Risk Factors

Generally, combat exposure, traumatic injury, and rape have been identified as types of trauma exposure associated with the highest rates of future PTSD development (Briere & Jordan, 2004; Kessler et al., 2017; Sareen, 2014). Additionally, experiencing personal versus impersonal trauma is associated with higher risk of PTSD development, as is the experience of physical injury during the trauma, particularly a mild traumatic brain injury (Briere & Jordan, 2004; Cloitre, 2015; Cusack et al., 2016). During the traumatic event, dissociation is associated with increased risk for PTSD development, and research into the impact of dissociation on PTSD maintenance is one of the core

elements of the proposed cPTSD diagnosis that was integrated into the current DSM-5 diagnostic framework for PTSD (De Jongh et al., 2016).

Personality Risk Factors

Research into personality factors that present risk factors for the development of PTSD is relatively weak, as the clear majority of people who present with comorbid personality disorders and PTSD have experienced significant CSA (Resick et al., 2012). As such, with respect to CSA, researchers have posited that personality factors are risk factors for the development of PTSD (Connor, Davidson, & Lee, 2003) and that they can be altered and shaped by the experience of CSA (Resick et al., 2012). For non-CSA related trauma exposures, there is some evidence that neuroticism and avoidance coping styles are risk factors for PTSD (Sareen, 2014), though this finding has not been widely replicated or accepted.

Genetic and Neurobiological Risk Factors

Another area that warrants a brief discussion is the impact of genetic and neurobiological risk factors on the development of PTSD. While the breadth of this discussion is outside of the scope of this literature review, there is considerable research being conducted into the neurobiological and genetic risk factors for PTSD. Neurobiological abnormalities often found in people with PTSD include excessive amygdala activation, impaired hippocampal and prefrontal cortex activity, and hypothalamic-pituitary-adrenocortical dysfunction, which has been linked specifically to the overgeneralization of threats (Bernardy & Friedman, 2015). Much of this research has taken place post hoc to the development of PTSD, though there is some evidence that early identification of some of these neurobiological abnormalities and immediate

intervention might help reduce the risk for PTSD development (Kelmendi et al., 2016). However, this avenue is only available to those with the measurement tools necessary to detect neurological anomalies. As this research base continues to grow, understanding how to assess outcomes related to neurobiological changes following a traumatic event may become increasingly important.

Epigenetics

The current limitations of the research base preclude fully integrating a discussion of genetic and epigenetic factors into this review, yet it is important to note that the next frontier for the understanding of PTSD risk factors may be epigenetics and intergenerational trauma (Zannas, Provençal, & Binder, 2015). Epigenetics is particularly important because studies investigating genetic predictors of PTSD have not been successful (Bernardy & Friedman, 2015; Zannas et al., 2015). Epigenetics is a promising avenue of research that has corroborated evidence that CSA and multiple traumatic experiences are correlated with PTSD development and HPA-axis dysfunction and have extended those findings across multiple generations (Zannas et al., 2015).

Epigenetic research has shown the multigenerational impacts of trauma on genetic expression in an increasingly diverse range of samples (Yehuda et al., 2014), and has been posited as a lens through which intergenerational, community trauma can be explained and ideally, acted upon (Perroud et al., 2014; Zannas et al., 2015). The relevance of this emerging line of research to PTSD outcomes research is the potentially crucial collaboration between geneticists and PTSD researchers to obtain consent from PTSD patients to give peripheral blood tests in large treatment systems and integrate epigenetic data into understandings of desired treatment outcomes, including resilience

and reduced autonomic activation (Zannas et al., 2015). This research area is still in early stages and is not ready to be integrated into clinical feedback. However, the potential is immense, and PTSD outcomes researchers should be aware of this potential when considering areas for future work. It should be noted that a hypothesized limitation of this approach is a decrease in patient rating of treatment acceptability, as blood-draws provoke anxiety and discomfort for many people (Zannas et al., 2015).

Summary of PTSD Risk Factors Literature

The first-order risk factor for PTSD (and in severe cases, chronic PTSD) appears to be continuous exposure to traumatic events (Kessler et al., 2017; Resick et al., 2012). Epigenetic research conceptualizes intergenerational trauma as the impact of profound prenatal stress on the development of the HPA-axis, which closely resembles the course of early-childhood trauma (Perroud et al., 2014). Conceptually, this still places repeated traumatic exposure, pre, post, or peri-natal, as the single greatest risk factor in developing PTSD. The literature has not yet differentiated the specific time-points during which risk is the greatest, though this area is the subject of proposed future research (Zannas et al., 2015).

For PTSD outcomes research, the literature on the risk factors for PTSD emphasizes the need to collect data on early childhood experiences (among other variables) for inclusion in analysis. However, an important element that is missing from PTSD outcomes research is the inclusion and integration of many of the risk-factors discussed above, including past treatment engagement, comorbidity, and trauma exposure history (Najavits & Hien, 2013). This trend is partially due to a lack of acknowledgement of the complexity and heterogeneity of symptom presentations in PTSD, which led to

simplistic study designs that did not measure or account for many of the variables discussed above (Cloitre, 2015; Wampold et al., 2010). As such, the research on potential risk factors for PTSD and the history of non-inclusion in PTSD outcomes studies is a concerning gap in the current literature.

PTSD Symptom Severity Reduction Rationale

As has been discussed previously in this paper, a focus on symptom severity reduction or elimination is an incomplete outcome focus for a variety of mental health conditions (Gilbody et al, 2002). While this is still the case in PTSD (as will be discussed in later sections), symptom severity is a core predictor of a multitude of other identified outcomes, including occupational functioning (Schnurr & Lunney, 2016) and health-related quality of life (HRQoL), measures of which assess vitality, pain, general health perceptions, emotional, physical, and social role functioning (Pagotto et al., 2015, p. 70). The discussion of PTSD symptom reduction is complex, requires a review of identified symptom clusters, diagnostic understanding of PTSD, and the ways in which symptoms have been effectively targeted in PTSD treatment.

Measuring PTSD Symptom Severity

Before discussing specific symptom severity reduction methods, it is important to include a discussion of how those symptoms are typically measured and how best practice recommendations suggest they should be measured. To that end, Bardhoshi et al. (2016) conducted a comprehensive analysis of the most commonly used diagnostic tools for PTSD. In conjunction with findings from Erford et al. (2016), it was found that the Clinician-Administered PTSD Scale (CAPS), the Impact of Event Scale–Revised (IES-R), the Posttraumatic Stress Diagnostic Scale (PDS), the PTSD Checklist (PCL), and the

Structured Interview for PTSD (SI-PTSD), and the Mississippi Scale for Combat-Related PTSD (M-PTSD) are the most commonly used instruments, with the CAPS used the most frequently. Each scale has demonstrated adequate reliability and validity, though the CAPS, PDS, and PCL have demonstrated the strongest reliability, validity, specificity, and sensitivity evidence (Bardhoshi et al., 2016; Erford et al., 2016; Evans, 2012; Green et al., 2017).

Additionally, this study assessed effect size comparisons between outcome measures. After this analysis, four instruments stood out as having the highest validity, reliability, and consistent effect size evidence: the CAPS, the PCL, the SI-PTSD, and the M-PTSD. Among these instruments, the CAPS, PCL, and M-PTSD yielded the strongest evidence overall, though the use of the M-PTSD is typically only appropriate for veterans or people who have experienced combat-related trauma. As such, the PCL and CAPS have the greatest utility across populations, which is reflected in the results from meta-analyses (discussed at length later in this paper) that show that these two instruments have strong psychometric properties in civilian, military, and refugee samples (e.g., Cusack et al., 2016; Kline et al., 2018). Given the strong suggestion that effective outcome research is cost-effective for patients, providers, and the general population (Fortney et al., 2017), it is worth noting that these two instruments are naturally complementary, free to administer (Bardhoshi et al., 2016), help clinicians track specific symptom cluster changes through factor analytic assessment (Armour et al., 2015), and allow for diagnostic evaluation and continuous assessment (Barlow, 2014).

One major limitation of the PTSD-specific measures literature is that PTSD assessments do not focus on quality of life (Cloitre, 2015) and measures that include

validity are largely absent. Assessing quality of life and other appropriate measures of functionality for PTSD populations will be discussed later in this review, and the literature base on this topic has a good deal of depth and breadth. However, measuring validity of symptoms in PTSD, especially on a wide scale, is challenging. This is likely due to a cost barrier, as clinicians are much more likely to administer free-to-use assessments (Bardhoshi et al., 2016). This likely accounts for the widespread use of the PCL and the CAPS. Unfortunately, assessments with both validity measures and strong psychometric properties (e.g., the Trauma Symptom Inventory [TSI] and the Detailed Assessment of Posttraumatic Stress [DAPS]) are not free to use and are thus underutilized by clinicians and underrepresented in the research literature (Evans, 2012). While it is encouraging that the most widely used assessments for PTSD have strong psychometric properties and can be used together to establish convergent validity, symptom validity measures, which may include personality tests with validity measures (e.g., MMPI-2, PAI) are important to include to adhere to best practices in PTSD assessment (Bardhoshi et al., 2016; Cloitre, 2015; Evans, 2012).

PTSD Symptom Severity Reduction Methods

The clear majority of the initial literature found for this paper revolved around methods of symptom severity reduction in PTSD, specifically the efficacy of different treatment modalities. This is not surprising, as controversy and debate surrounding appropriate treatments for PTSD has been prevalent since the introduction of the disorder into the DSM (Pomerantz, 2017). As was mentioned in the previous section, reducing specific PTSD-related symptoms is an important part of PTSD treatment, and as such, it is necessary to review the treatment effectiveness literature for PTSD. After reviewing

the literature, cognitive processing therapy (CPT; Resick & Schnicke, 1992), eye movement desensitization and reprocessing (EMDR; Shapiro, 2001), cognitive therapy (CT; Ehlers & Clark, 2000), and prolonged exposure (PE) for PTSD (Foa, Hembree, & Rothbaum, 2007) are consistently named as the most efficacious treatments for PTSD, which is reflected in the American Psychological Association's Clinical Practice Guideline for the Treatment of PTSD (CPG-PTSD; 2017), the findings from which will be discussed in greater detail in an upcoming section.

Other Common Treatment Approaches

Before fully reviewing the evidence for the PTSD treatments with the strongest support, it is important to note other widely-used treatments that have not demonstrated strong effect size reductions in symptom severity or other outcome measures. Generally, pharmacological treatments alone have failed to demonstrate even medium effect sizes (Green et al., 2017), as have non-trauma-focused CBT methods, coping skills training, and brief eclectic therapy (Barlow, 2014; Cusack et al., 2016; Kline et al., 2018). One controversial approach is stress inoculation training (SIT), which has demonstrated mixed efficacy results due to methodological research issues (Green et al., 2017). SIT has, in the past, been an EST for PTSD (Barlow, 2014), though multiple meta-analyses conducted in the past ten years have not included it in effect size analysis because the strength of the evidence is too weak (e.g., Cusack et al., 2016; Ehring et al., 2014; Powers et al., 2010). Another modified protocol with a growing body of evidence is skills training in affective and interpersonal regulation (STAIR), which was developed for comorbid PTSD and substance use (Cloitre et al., 2005; Green et al., 2017). However, the core mechanism of change in STAIR appears to be exposure-based (Barlow, 2014), and it is unclear if the added

components of affective and interpersonal regulation are necessary for symptom reduction. Currently, the evidence is not sufficient to discuss STAIR as stand-alone empirically supported treatment for PTSD, though the exposure component is highly evidence-based (APA, 2017).

PE for PTSD

PE for PTSD has one of, if not the strongest evidence bases in support of its use in the treatment of PTSD (Barlow, 2014; Karlin et al., 2010). PE is based on the emotional processing theory of PTSD development and maintenance and involves engagement in both in-vivo and imaginary exposure. In-vivo exposure refers to encountering traumatic reminders in a safe, therapeutic context, while imaginal exposure refers to processing traumatic memories through reading, listening to, or reciting a traumatic memory narrative (Foa et al., 2007). PE for PTSD is typically conducted in an outpatient setting, but recent evidence has demonstrated medium to large effect sizes in community clinics (Wade, Lau, & Nixon, 2018), psychiatric hospital settings (Cusack et al., 2016), and in primary care (Cigrang et al., 2017; Pomerantz, 2017).

Additionally, according to an RCT from Rothbaum et al. (2012), when PE for PTSD and a comparison group of breathing and relaxation training were delivered to emergency room survivors within half a day of experiencing a single-event trauma, only PE for PTSD appears to function as a protective factor against the future development of PTSD. PE for PTSD reports dropout rates (20-21%) similar to the rates of other trauma therapies and much lower than rates found in pharmacological interventions (Olatunji, Deacon, & Abramowitz, 2009). It should be noted that some have suggested that this number is grossly deflated due to the reliance on RCT design for meta-analysis of

dropout rates (Najavits, 2015), though the strength of this evidence is not strong enough to assert that dropout rates for PE for PTSD are substantially higher in community settings than in RCTs (e.g., Wade et al., 2018).

While the strength of the evidence in favor of patient preference for PE for PTSD was not strong enough for inclusion in the 2018 CPG-PTSD, there is evidence that patients find PE tolerable and are not caused harm by engaging in the treatment (Farrell, Deacon, Dixon, & Lickel, 2013), despite many the prevailing belief among many clinicians that PE for PTSD is intolerable to many patients (Hipol & Deacon, 2013; Olatunji et al., 2009). PE is best delivered in individual therapy, as meta-analyses of PE for PTSD in group therapy have shown discouraging results (Haagen, Smid, Knipscheer, & Kleber, 2015; Murphy & Busuttil, 2015).

Finally, PE has a strong evidence for treating PTSD with complex (Karlin et al., 2010) and comorbid presentations. In a pilot RCT measuring the effectiveness of combining PE for PTSD and Dialectical Behavioral Therapy (DBT) for women with comorbid borderline personality disorder, PTSD, and self-injury, moderate to large effect sizes were found for the combined treatment with respect to global functioning, depression, anxiety, shame, posttraumatic cognitions, and shame (Harned, Korslund, & Linehan, 2014). These findings have not been replicated extensively (Green et al., 2017), though they are encouraging given that approximately 25%-60% of patients with borderline personality disorder have comorbid PTSD (Pagotto et al., 2015). Moderate to large effect sizes have been demonstrated when using PE to treat PTSD and comorbid psychosis, major depression, and substance use (McGuigan, 2013), although many RCTs

reviewed in meta-analyses exclude patients with comorbid substance use or suicidality (Cusack et al., 2016).

CPT

Based on Ehlers and Clark's (2000) cognitive model, CPT targets cognitive appraisals of the traumatic event, specifically maladaptive beliefs about self, others, and the world (Green et al., 2017). Skills such as thought evaluation and thought challenging are taught through the use of Socratic questioning in session, which is then continued by the client outside of session (Resick et al., 2012). During treatment, the client writes an impact statement that summarizes the ways in which the trauma has affected functional areas of life, which are then targeted for intervention (Resick et al., 2012). While the original form of CPT included writing an account of the trauma and processing it with the therapist and outside of session as needed, the treatment is often delivered without this component (Resick et al., 2008). Interestingly, while component analyses of cognitive behavioral therapy have failed to demonstrate the efficacy of thought challenging and modification (Longmore & Worrell, 2007; Green et al., 2017), components analyses of CPT have demonstrated that the modification of trauma related thoughts is both necessary and sufficient for change in PTSD (Ehlers et al., 2010). These components analysis findings have not been replicated adequately, though the average effect size evidence for CPT is medium to large (Ehring et al., 2014; Powers et al., 2010). Like PE for PTSD, CPT has been shown to be more efficacious when delivered in an individual, rather than group format (Resick et al., 2017). The effect-size evidence for CPT and comorbid conditions is mixed, and the evidence base lacks enough depth and breadth to

make broad-base conclusions, though results appear to be mostly positive (Ehring et al., 2014).

In some guides to effective PTSD treatments (e.g., APA, 2018), cognitive therapy without specific PTSD focus has been determined to be an empirically validated approach to reducing PTSD symptoms, especially posttraumatic cognitions (Barlow, 2014, Green et al., 2017). However, the PTSD-tailored form of cognitive therapy found in CPT is thought to be a more efficacious method, though a component analysis focused on examining these two related approaches has not yet been conducted. Information on whether or not each study reviewed contained a writing component was not provided, so it is unclear whether meta-analytic results support Ehlers et al.'s (2010) component analysis findings. It should be noted that the core mechanism of change in CPT has been purported to be exposure, as processing cognitions related to the traumatic event can be viewed as a form of exposure to that traumatic event, especially given the complex connectivity of trauma-related memories to the event itself (Green et al., 2017). Nevertheless, the strength of the evidence for CPT is strong, overall.

EMDR

EMDR (Shapiro, 2001) is widely-used treatment for PTSD that was developed after Shapiro discovered that lateral eye movements were able to help facilitate the processing of her own difficult thoughts. She then extrapolated this personal discovery to other forms of difficult thoughts, specifically posttraumatic cognitions in PTSD (Green et al., 2017). EMDR typically consists of eight phases of treatment that consists of, “history taking, client preparation, target assessment, desensitization, installation, body scan, closure, and reevaluation of treatment effects” (Barlow, 2014, p. 76). The model of

EMDR treatment is based on a modified Cognitive Processing model that links past cognitive appraisals of traumatic events, current avoidance of traumatic triggers, and the presence of maladaptive neural networks to the development and maintenance of PTSD (Shapiro, 2001). In EMDR, the goal of treatment is to reframe negative cognitions surrounding the trauma, decrease avoidance of trauma-related triggers, develop adaptive responses in response to trauma-related triggers, and, most importantly, allow for the reintegration of newly learned responses into adaptive neural networks via rapid eye-movement (Shapiro, 2001).

EMDR is controversial in that evidence is conflictual at best concerning the mechanism of change, with some researchers suggesting that rapid, lateral eye-movement is a key component of the treatment (e.g., Pitman et al., 1996; Shapiro, 2001), and others noting the similarity of the treatment to exposure and the lack of component analysis evidence to suggest rapid eye movement is necessary (Barlow, 2014; Green et al., 2017). While there are some studies that have suggested a neurobiological mechanism linking rapid eye movement in EMDR to REM sleep cycles and the co-occurring priming of the hippocampus to integrate traumatic memories into larger, less activating semantic networks (e.g., Stickgold, 2002), there is not enough evidence to definitively state that the rapid eye movement component of EMDR is necessary to achieve positive clinical outcomes. In terms of overall effectiveness, most meta-analyses yield medium effect sizes versus control and little difference between EMDR and active treatments (Ehring et al., 2014; Powers et al., 2010). As such, EMDR appears to be an effective treatment for PTSD, though slightly less effective than PE for PTSD (Kline et al., 2018) and with little

evidence in support of the necessity of the rapid eye movement component of the treatment protocol.

Meta-analytic Discussion Rationale

Meta-analytic studies provide the most comprehensive review of the comparative effectiveness of PTSD treatment approaches (Barlow, 2014). While many meta-analyses for PTSD have been conducted (McKay et al., 2017), the following four recent meta-analyses provide a recent and comprehensive view of PTSD treatment effectiveness in both symptom severity and several other notable outcomes. These meta-analyses will be discussed in order of publication date, beginning with the least recent study.

Powers et al., (2010). This meta-analysis was conducted in 2010 and included a total of 13 studies and 675 participants. The purpose of the study was to assess the strength of the RCT evidence for PE for PTSD. Primary outcome measures were focused on PTSD symptom severity and included the CAPS and the PCL, while secondary outcome measures included the Beck Depression Inventory II (Beck, Steer, & Brown, 1996) and the State/Trait Anxiety Inventory (Spielberger, 1972), among other instruments with established reliability and validity (Bardhoshi et al., 2016). Hedge's g was utilized due to the small sample size of several of the RCTs reviewed, which is in accordance with recommendations from Ferguson (2009). Publication bias was tested using the fail-safe N and heterogeneity among studies was accounted for appropriately using the Q statistic, which are both methods recommended for use in meta-analysis by Schmidt and Hunter (2014). Overall, the methodological and statistical rigor appears to be strong.

Results demonstrated large effect sizes for PE for PTSD versus control on both primary (Hedges's $g=1.08$, $SE=.20$, 95% CI[.69, 1.46]) and secondary outcome measures (Hedges's $g=.77$, $SE=.12$, 95% CI[.53, 1.01]). The effect size of PE for PTSD versus waitlist was large (Hedges's $g=1.51$, $SE=.21$, 95% CI[1.12, 1.90]). The effect size of PE for PTSD versus psychological placebo was medium to large (Hedges's $g=.65$, $SE=.19$, 95% CI[.29, 1.01]). Only 7 of the original 13 studies included follow-up data. At follow-up (between 1 and 12 months depending on the study), the effect size for PE for PTSD versus control was medium to large (Hedges's $g=.68$, $SE=.21$, 95% CI[.27, 1.10]). Despite the strong evidence found in support of PE for PTSD, comparison analysis for six studies including PE, CPT, EMDR, CT, and SIT did not find a difference between PE for PTSD and the other active treatments (Hedges's $g=-.07$, $SE=.18$, 95% CI[-.42, .28]). Given this evidence, the authors concluded that PE for PTSD is an effective treatment, though the lack of effect size differentiation between PE and other active treatments is puzzling. The authors suggested that a single process, exposure, is common across treatments, and might account for the lack of variation in results.

Ehring et al., (2014). In a later meta-analysis, the necessity of trauma-focused treatment for PTSD was examined in adult survivors of childhood sexual abuse. Most studies reviewed were RCTs, but controlled research that did not meet RCT criteria was also included, in large part due to the limitations of the current research base on this population. This meta-analysis is inherently limited by its inclusion criteria (only adult survivors of childhood sexual abuse), though it adds to the literature by addressing a group that previously had only been systematically examined in the cPTSD research (Cloitre, 2015). This meta-analysis also sought to “investigate whether trauma-focused

treatments differed from non-trauma-focused interventions regarding the relative efficacy” (p. 647). Finally, this meta-analysis compared individual and group treatments across multiple treatment modalities.

Methodologically, this analysis is highly rigorous. As was in the case in the meta-analysis from Powers et al. (2010), all instruments used (e.g., CAPS, PCL variants) had established reliability and validity (Bardhoshi et al., 2016). Additionally, Hedge’s g was appropriately utilized due to the inclusion of studies with small sample sizes (Ferguson, 2009). Publication bias was tested using the funnel plot method and heterogeneity was addressed using the Q statistic, which are both methods recommended for use in meta-analysis by Schmidt and Hunter (2014). A priori planning rigor was strong in this study as well, as it also included a pre-established study selection and data analytic plan. Sixteen total studies were reviewed. Most studies were manualized and ($k = 13$) and used a structured clinical interview ($k = 13$). Follow-up data (6-12 months) was better than was seen in Powers et al. (2010), though still underwhelming ($k = 11$). Dropout was also assessed. This meta-analysis did not directly compare different types of cognitive-behavioral treatments designed for PTSD and collapsed PE for PTSD, CPT, and STAIR under a cognitive-behavioral intervention for PTSD (CBT-P) umbrella.

The results from this meta-analysis clarify the benefits of trauma-focused treatments for adult survivors of childhood sexual abuse. Between-groups effect sizes at post-treatment versus waitlist were larger for CBT-P (Hedges's $g=.88$, $SE=.30$, 95% $CI[.30, 1.47]$) than for non-trauma-focused CBT (Hedges's $g=.48$, $SE=.39$, 95% $CI[-.28, 1.23]$), and EMDR (Hedges's $g=.76$, $SE=.34$, 95% $CI[.38, 1.71]$). Between-groups effect sizes at post-treatment versus treatment-as-usual (TAU) / placebo were again larger for

CBT-P (Hedges's $g=1.72$, $SE=.58$, 95% CI[.58, 2.87]) than for non-specific CBT (Hedges's $g=-.12$, $SE=.12$, 95% CI[-.37, 0.12]), and EMDR (Hedges's $g=.39$, $SE=.38$, 95% CI[-.36, 1.13]). Effect sizes at follow-up were almost identical, with CBT-P outperforming non-trauma-focused interventions. Dropout for all treatments were assessed to be approximately 20%, which is consistent with rates seen across the PTSD literature (Olatunji et al., 2009). Group treatments (Hedges's $g=.73$, 95% CI[.60, .86]) were found to be less efficacious than individual treatments (Hedges's $g=1.18$, 95% CI[.99, 1.37]).

Notably, non-specific CBT was not effective in treating PTSD based on the confidence interval values. The lack of ability to compare across specific treatment modalities within CBT-P is indicative of a dearth of RCTs for this population, though the result that trauma-focused interventions are more effective in treating PTSD than non-trauma-focused interventions is important, especially given the assertion that all PTSD treatments are equivalent (e.g., Wampold et al., 2010). Inclusion of outcome measures beyond symptom severity was lacking, which is a common shortcoming of RCT designs (Gilbody et al., 2002).

Cusack et al., (2016). While most treatment guidelines for PTSD (e.g., APA, 2018; Foa, Keane, Friedman, & Cohen, 2008) recommend trauma-focused treatments as first-line interventions and require at least one RCT that meets strict methodological criteria (missing data analysis, measures of dropout, blinding assessors; c.f. Powers et al., 2010), there is still a great deal of variance in inclusion criteria and methodological approaches across treatment guidelines. Additionally, there is a lack of direct comparison of trauma-focused treatments, specifically cognitive therapies and exposure therapies. In

response to this gap in the literature, Cusack et al. (2016) conducted the largest meta-analysis of psychotherapeutic interventions for PTSD to date, reviewing 64 trials published between 1980 and 2014.

Methodological rigor in this study was extremely strong, as it utilized essentially the same methods as Ehring et al. (2014) while adding several important modifications. First, two reviewers independently reviewed articles for inclusion criteria, and all disagreements were arbitrated by a third party experienced with meta-analytic methods. Second, “studies were required to assess at least one of the following outcomes: PTSD symptoms, remission (no longer having symptoms), loss of PTSD diagnosis, quality of life, disability or functional impairment, return to work or active duty, or adverse events” (p. 131). Third, bias was assessed more strictly than in previous meta-analyses by assessing selection bias, potential confounds, detection bias, and attrition bias (p. 131). Sample sizes in this analysis were generally larger due to the inclusion of more recent studies, with the largest sample being 563 and the smallest being 10. Most studies analyzed psychological treatment alone, and the majority of participants experienced severe PTSD (CAPS total score = 60-79).

The full extent of the findings from the meta-analysis is too great to include in this review, though several findings are notable. CPT, cognitive therapy, PE for PTSD, and EMDR all demonstrated at least medium effect size symptom reductions, while narrative exposure therapy and brief electric psychotherapy showed small effect sizes. Because symptom reduction was measured across a variety of sample sizes, standard mean difference (SMD), which is equivalent to Cohen’s d (1992) but able to compare across different sample sizes, was reported for PTSD symptoms and weighted mean

difference (WMD) was reported to represent change from baseline data. The only two large effect sizes in this meta-analysis were both found for PE for PTSD. Specifically, PE for PTSD showed large effect sizes for PTSD symptom reduction as measured by the CAPS (SMD=-1.27, 95% CI[-1.54, -1.00], N = 387) and depression symptoms as measured by the BDI (WMD=-8.2, 95% CI[-10.3, -6.1], N = 363).

Echoing findings from the Ehring et al. (2014) paper, comparison findings were largely inconclusive, as the head-to-head evidence was insufficient. The only notable finding here was that PE for PTSD is moderately favored over relaxation training. These comparison findings are interesting, as relaxation training is included in the PE for PTSD protocol (Foa et al., 2007). Although the strength of this evidence is not strong enough to assert any definitive conclusions, it is worth exploring in future dismantling research. Although adverse effects of treatment engagement (e.g., suicidality, dropout, self-harm) were assessed, evidence was not sufficient to draw any strong conclusions. As was the case in the Powers et al. (2010) meta-analysis, the exclusion of comorbid suicidality and substance use confounded analysis to a certain extent, although more studies were assessed that included that information than in previous meta-analyses. Comorbid substance use and PTSD is an area of expanding research that still needs to grow before analyses of this magnitude can be undertaken. Evidence was insufficient to draw strong conclusions about long-term efficacy of any treatments. As has been noted in previous discussions of CPT and cognitive therapy, the distinction between the two approaches appears to be largely arbitrary, and little evidence suggests that they differ. It should be noted that the majority of results were drawn from outpatient settings, and there is a need for additional research across various settings (Cloitre, 2015). Most of the studies

reviewed assessed PTSD and/or depression symptoms, and other outcomes were not utilized enough to include in analysis. Ultimately, this meta-analysis presents the strongest evidence in support of PE for PTSD over other treatments in terms of PTSD and depression symptom reduction, though the lack of head-to-head trials limits the ability to make comparative conclusions.

Kline et al., (2018). The most recent meta-analysis reviewed attempted to expand beyond the limited timeframes often seen in RCTs and assess the long-term efficacy and follow-up evidence for psychological treatments for PTSD. This meta-analysis built upon and cited the previous meta-analyses discussed in this paper and was most methodologically similar to Cusack et al. (2016). Specifically, the approach to inclusion criteria, study bias, and multiple rater methodology was adopted from Cusack et al. (2016). Additionally, to account for the specific focus on long-term follow-up data, Cohen's d was modified to include the pretreatment standard deviation, which is an appropriate statistical modification (Schmidt & Hunter, 2014). As such, methodological rigor was high. Additionally, this study included information concerning population (e.g., civilian, military, refugee), type of trauma, and primary outcome measures, which allowed for increased specificity in analysis.

In total, the 32 studies reviewed comprised 3399 patients with sample sizes ranging from 30 to 284 (median = 92, SD = 57). Seventy-two conditions met full inclusion criteria, and the mean effect size across conditions was large (Cohen's $d = 1.88$, 95% CI[1.68, 2.08]) for pretreatment to follow-up and small (Cohen's $d = .17$, 95% CI[.10, .23]) for posttreatment to follow-up. All follow-up data was collected at least six months post-treatment, which is long enough to reliably determine lasting treatment

effects (Trauer, 2010). Most conditions obtained follow-up data at six months ($k = 48$, 67%) and one year ($k = 19$, 26%), with several studies obtaining follow-up data at nine ($k = 3$, 4%) and 20 months ($k = 2$, 2,8%). Dropout rates in the studies reviewed were 22%, which is consistent with findings from previous meta-analyses (e.g., Ehring et al., 2014; Cusack et al., 2016; Powers et al., 2010). In terms of population studied, this analysis reviewed 23 civilian studies (9 female-specific), 7 military studies, 1 study focused on Cambodian refugees, and 1 study focused on Sudanese refugees. Most studies assessed mixed trauma, which was not further defined, and utilized the CAPS as the primary outcome measure.

As has been shown in previous meta-analyses (e.g., Cusack et al., 2016; Ehring et al., 2014), active treatments (Cohen's $d = 2.14$) performed substantially better than control conditions (Cohen's $d = 1.04$; $p < .01$) in the pretreatment to follow-up period. However, long-term effects were less encouraging, with no significant differences noted between active treatments and control in the posttreatment to follow-up period. The only treatments that demonstrated long-term efficacy compared to other conditions were exposure-based therapies (largely PE for PTSD; Cohen's $d = .27$). Importantly, population type, number of sessions, and type of trauma did not predict effect size differences for active treatments during the pretreatment to follow-up period. In the posttreatment to follow-up period, an index trauma of childhood abuse was related to greater gains at follow-up (Cohen's $d = .46$) than traumas that occurred in adulthood (Cohen's $d = .12$) or mixed trauma focus (Cohen's $d = .14$). Additionally, there was evidence that attrition effects did not impact results if individuals were randomized immediately as opposed to only being randomized at follow-up. Finally, effect sizes

began to increase with longer follow-up times, suggesting the need to assess beyond 6 months posttreatment, especially given research that suggests PTSD can lead to long-term residual symptoms and decreased quality of life (Bryant et al., 2016; Cloitre, 2015). The long-term efficacy of PE for PTSD is especially relevant to assess in the light of these findings, as there is very little evidence beyond this meta-analysis concerning which treatments are effective at maintaining treatment gains long-term.

PTSD Symptom Severity Reduction Summary

While multiple treatments appear to be efficacious for PTSD (e.g., CPT and PE for PTSD), PE for PTSD has the strongest research base, albeit by a slim margin. Specifically, PE for PTSD shows the largest effect size gains of all trauma-focused treatments across meta-analyses, has the strongest evidence with respect to comorbid conditions, is not significantly different from other active treatments in terms of dropout, and is the only active treatment to demonstrate long-term (6 months and beyond) efficacy in terms of PTSD symptom severity reduction. There is evidence that PTSD-specific cognitive restructuring found in CPT focused on safety, trust, power/control, esteem, and intimacy is an effective component to include in trauma treatment, though there is still debate as to the underlying mechanism of change with respect to this approach.

Assessments including measures of validity (e.g., TSI, DAPS, MMPI-2) were largely absent from the symptom severity review, which is a major oversight given the findings from several studies that have emphasized the evidence that instances of PTSD are overreported and occasionally malingered (Evans, 2012). While there is not a consensus in the literature about the severity of this problem (e.g., Poyner, 2010), best practices of obtaining collateral information such as medical and mental health records

and including at least one measure of validity were not followed in the studies reviewed. While this does not discount the findings from these meta-analyses, it does require additional replication of these findings in conjunction with collateral information and a measure of malingering. Evidence suggests the Personality Assessment Inventory (PAI), the DAPS, Morel Emotional Numbing Test-Revised (MENT-R; Morel, 1998) and the TSI to be particularly effective at detecting PTSD malingering when given in some combination (Evans, 2012; Messer & Fremouw, 2007; Wooley & Rogers, 2015).

Additional findings from this section of the review emphasize the utility of the CAPS as a primary symptom outcome measure, especially when measurement only occurs at admit, discharge, and follow-up. Symptom specific measures of depression and other comorbid disorders specific to populations under study are recommended as well.

Functional Impacts of PTSD on Quality of Life

PTSD has been conceptualized as a whole-life disruption (Levers, 2012), as evidence shows experiencing PTSD symptoms is linked to spiritual (Kopacz, Currier, Holland, Drescher, & Pidgeon, 2015), family and marital (Eisenman et al., 2008), occupational (Schnurr & Lunney, 2016), physical health (Agorastos et al., 2014), and interpersonal (Cloitre et al., 2005) impairment. These domains are often assessed together under the umbrella term “quality of life.” There is also evidence that even in early-remitting cases of PTSD (i.e. naturalistic loss of diagnosis within five months), residual functional impacts persist (Bryant et al., 2016). As has been discussed throughout this review, thorough, individualized assessment is especially important in PTSD given the heterogeneity of symptom and functional impairments (Cloitre, 2015). Appropriate methods for assessing functional impairment include Melchert’s (2011) biopsychosocial

assessment, global functioning measures (Brown et al., 2001), domain-specific self-report measures (Fortney et al., 2017), and medical / occupational records to assess concrete functional impairments (Thornicroft & Slade, 2014). In the next section, the literature on functional impairment and quality of life in PTSD will be reviewed with the intention of identifying important areas of outcomes assessment.

Spiritual Functioning

Spirituality in PTSD has been proposed as a potentially important protective factor, especially for veterans experiencing moral injury (Kopacz et al., 2011) and survivors of sexual assault (Resick et al., 2012). However, there is currently a dearth of literature on spirituality and PTSD specifically, as it is typically represented as a single question in the construct of posttraumatic growth (Frazier, Conlon, & Glaser, 2001) and is more loosely connected to moral injury (Kopacz et al., 2011). Additionally, there does not appear to be consistent evidence of spiritual change that predicts improved PTSD outcomes, which may be due to the heterogeneity of religious beliefs and behaviors held across individuals (Chen & Koenig, 2006). As such, the discussion (and this review) of the relationship between spirituality and PTSD is often centered around moral injury and posttraumatic growth.

Moral Injury. The moral injury literature is quickly expanding and warrants a larger discussion beyond the scope of this literature review. However, it is important to note that moral injury is a concept that is increasingly important in the veteran population (McGuigan, 2013). Moral injury can be defined as “enduring distress and alterations in functioning following events in which combatants perceive themselves to violate, through action or inaction, their own moral codes” (Nash et al., 2013, p. 647).

The Moral Injury Events Scale (MIES; Nash et al., 2013) is the most widely used measure of moral injury and has demonstrated strong psychometric properties, including multiple confirmatory factor analyses validating the proposed factor structure (Bryan et al., 2016). The link between moral injury and spirituality is strong in the conceptual literature (Kopacz et al., 2016), yet the MIES does not contain any questions explicitly addressing spirituality. As such, it is difficult to extrapolate the preliminary results from this scale to the assessment of spirituality in PTSD, especially given the lack of replication of initial findings at this time (Bryan et al., 2016).

In the studies that have explicitly assessed the relationship between spirituality and PTSD in military populations, the results are mixed. Two recent studies of veterans (Currier, Holland, & Malott, 2015; Kopacz et al., 2016) found that high levels of spirituality appear to serve as a protective factor against PTSD and suicidality while struggles with spirituality are related to worse treatment outcomes. However, another study of veterans linked higher levels of spirituality to increased PTSD symptom severity, which, in the moral injury framework, is explained by the violation of closely held moral or spiritual beliefs by the experience of combat-related trauma (Hourani et al., 2012). Given the stage of research for moral injury and the lack of research for spirituality, it appears as if it may be useful to continue assessing for moral injury in a veteran population, though it appears to be appropriate to reserve these measures for specific instances in which clinical judgement suggests an area of need.

Posttraumatic Growth. As is the case for moral injury, an in-depth discussion of posttraumatic growth is beyond the scope of this review. With respect to PTSD, the conceptual literature is extensive, though the empirical evidence is mixed (Mancini,

Littleton, & Grills, 2016; Steenkamp et al., 2012). Posttraumatic growth is defined as “the experience of positive change that occurs as a result of the struggle with highly challenging life crises” (Tedeschi & Calhoun, 2004). It is most often measured using the Posttraumatic Growth Inventory (PTGI; Tedeschi & Calhoun, 1996), which has been shown to have adequate psychometric properties across a variety of studies, including successful confirmatory factor analyses of the originally proposed five-factor model (Ramos, Leal, Marôco, & Tedeschi, 2016). The five factors are: Relating to Others; New Possibilities; Personal Strength; Spiritual Change; and Appreciation of Life (Tedeschi & Calhoun, 2004).

It is difficult to ascertain the strength of the literature for PTSD specifically, as much of this literature does not differentiate between trauma and PTSD (Mancini et al., 2015). However, in the studies that have measured posttraumatic growth in PTSD populations, results are mixed. Bryant et al. (2016) found that, unlike the evidence from non-clinical trauma-exposed samples, PTSD results in ongoing functional impairment following symptom remittance. This is compounded by findings from Frazier et al. (2009) that, in a sample of undergraduates who experienced a traumatic event at some point over the course of a semester ($n = 122$), perceived posttraumatic growth (as measured by the PTGI) did not match measured PTSD symptom reduction and did not predict future PTSD symptom reduction. Similarly, in a sample of survivors of the 2011 Oslo bombing, Blix et al. (2016) found that survivors with PTSD who reported early posttraumatic growth experienced greater PTSD symptoms long-term than those who did not report early posttraumatic growth. A large-scale analysis has not been conducted on posttraumatic growth, as the concept is still being refined and has not been rigorously

studied (Blix et al., 2016). There is cause for optimism, as a new update of the PTGI called the PTGI-X was developed to enhance the ability of the scale to assess spiritual change cross-culturally, and preliminary evidence suggests this updated scale has better sensitivity and factor analytic properties than the original scale (Tedeschi, Cann, Taku, Senol-Durak, & Calhoun, 2017). While this may change with more widespread use of this new scale, there appears to be a significant gap between the conceptual literature and the trends seen in the evidence, and as such, no strong conclusions about the relationship between posttraumatic growth, spirituality, and PTSD can be made at this time.

Family Functioning

The relationship between family functioning and PTSD is more straightforward than was the case for spirituality, though there are still several areas in need of further exploration. It has been widely established that PTSD is linked to acute and longitudinal impairments in marital and familial functioning in multiple populations, most notably in veterans, survivors of sexual assault, and refugees across the world (Bryant et al., 2016; Evans, Cowlshaw, & Hopwood, 2009; Silove, Ventevogel, & Rees, 2017; Vogt et al., 2017). Specific areas of impairment include “poorer family adjustment, more relationship problems, more problems with intimacy, higher levels of relationship distress, more parental problems, lower family cohesiveness, and less constructive communication behaviors” (Vasterling et al., 2015, p. 150). The mechanisms of this relationship are unclear, with some positing that specific symptom clusters (most commonly numbing / avoidance) predict functional impairments (e.g., LeBlanc et al., 2016), others who propose family discord contributes to the development and maintenance of PTSD (Evans et al., 2009), and still others who conclude that the relationship is bidirectional (Vogt et

al., 2017). A definitive conclusion cannot be drawn given the state of the research evidence at this time. However, across populations, research supports the assertion that, for many people suffering from PTSD, assessing and treating familial functional impairments can lead to improved treatment outcomes (Evans et al., 2009; LeBlanc et al., 2016; Rodriguez, Holowka, & Marx, 2012). As such, it is important to know best practices for family functioning measurement used in PTSD.

Measuring the full range of the relationship between PTSD and family functioning is incredibly complex, as each family unit is a unique culture situated within a larger culture, the expectations and norms from which impact the specific course of functional outcomes (Silove et al., 2017; Vasterling et al., 2015). However, a general biopsychosocial assessment is recommended to determine if the client perceives family functioning to be an issue, after which specific measures can help clarify identified issues, as well as if family currently serves as a protective factor (Evans et al., 2009; Melchert, 2015). When conducting this assessment, it is important to include culturally-specific functional measures, as “Failure to include indigenously derived measures that capture local expressions or idioms of distress also can lead to the under-enumeration of mental health problems” (Silove et al., 2017, p. 137).

Examples of specific outcome measures that have demonstrated strong utility for PTSD include the Dyadic Adjustment Scale (DAS; Spanier, 1976), the Relationship Assessment Scale (Hendrick, 1988), and the Deployment Risk and Resilience Inventory-2 (Vogt et al., 2013), which measures a range of perceptions of family discord and support. When assessing family functioning, it is crucial to obtain collateral information from family members instead of purely relying on self-report data (Rodriguez et al.,

2012). Multi-dimensional, collaborative assessments such as the McMaster Family Assessment Device (Epstein et al., 1983) and the DAS have been shown to be especially effective in helping clinicians corroborate client reports of familial impairment and can easily be ported to a couples and family setting, should the focus of treatment shift to family functioning (Vasterling et al., 2015). Additionally, effective PTSD treatments (e.g., PE for PTSD, CPT) require work to be done in the home environment, and success in treatment may depend on the stability of the home and family relationships (Green et al., 2017). Finally, if someone has been abused by a member of their family, the quality of relationships with other family members and ability to forgive the person who abused them has been linked to improvements in PTSD symptoms, though balancing this finding with potential safety concerns is a clinical priority (Baldry, Cinquegrana, Regalia, & Crapolicchio, 2017).

In sum, there appears to be an important relationship between PTSD and family functioning, though the directionality of this relationship is unclear. As such, in PTSD populations, it is important to include family functioning in a general biopsychosocial approach to understand the specific relationship between PTSD symptoms and family functioning in the individual. If problems are identified, assessments should be used to inform greatest treatment need, the evidence from which should be corroborated with data from family members obtained through assessment or clinical interview. If necessary, family therapy is indicated and can be an essential treatment component for people with PTSD experiencing familial impairment (Rodriguez et al., 2012; Vasterling et al., 2015; Vogt et al., 2017)

Interpersonal and Social Functioning

Long-term social and interpersonal impairments are well-established in PTSD and are especially connected to avoidance and intrusion symptoms (Kashdan, Morina, & Priebe, 2009; Solomon & Mikulincer, 2007; Tsai, Harpaz-Rotem, Pietrzak, & Southwick, 2012). Impairment in social functioning is a focal point in the cPTSD literature, though the previous focus on phase-based treatments beginning with building up social skills has not been shown to be effective in the literature (De Jongh et al., 2016; Rothbaum et al., 2012). However, there is growing evidence that addressing interpersonal functioning and building interpersonal skills is an essential component of effective trauma treatment that can address symptoms of avoidance and reduce dropout rates (Eidhof et al., 2019; Markowitz et al., 2015). While the evidence is not strong enough to suggest interpersonal therapy or interpersonal skill building alone is a consistently effective PTSD treatment, focusing on interpersonal functioning is undeniably effective in increasing quality of life during and after treatment (Cloitre et al., 2005).

There are many measures of interpersonal functioning that have been utilized in PTSD outcomes assessment (e.g., Inventory of Interpersonal Problems; Horowitz et al., 1988) that are typically focused on perceived quality of interpersonal relationships. However, another perspective on interpersonal functioning in PTSD posits that transdiagnostic intimacy (Wetterneck & Hart, 2012) might be an especially effective treatment target, as people with PTSD tend to struggle to develop close and lasting relationships with others. People who have experienced repeated interpersonal trauma have an especially difficult time forming close bonds with others and are less likely to engage in interpersonal behaviors (e.g., being vulnerable in relationships) that promote

feelings of connection with others (Bistricky et al., 2017; Eidhof et al., 2019). Assessing intimacy (defined here as emotional and interpersonal vulnerability with others) via measures such as the Awareness, Courage, and Love scale (Kuczynski, 2016) can help focus assessment and intervention on the ability to build meaningful relationships with others, which has been posited as a core process of change in PTSD, especially in cases with multiple comorbidities (Cloitre, 2015).

In addition to encouraging the development of close and meaningful relationships with safe others, it is helpful to understand how social networks and social support contribute to the development and maintenance of PTSD. The relationship of social connection to PTSD is complex, and often differs between populations. For instance, in military populations, there is evidence PTSD is associated with high levels of stigma, which is often seen in peers fearing and avoiding the soldier with PTSD (Sayers, Farrow, Ross, & Oslin, 2009; Sripada et al., 2015). This is particularly problematic in military populations, as the military culture often serves as both a crucial social support network and as a barrier to seeking help for mental health problems (Hoge et al., 2004; Michalopoulou, Welsh, Perkins, & Ormsby, 2017). However, research has consistently shown that non-military family and friends are not viewed as being particularly stigmatizing, and instead serve as supports for veterans with PTSD (Hamilton et al., 2015; Hoge et al., 2004).

Additionally, there is preliminary evidence that the perceptions of stigmatizing beliefs for seeking help for mental health issues within the military is decreasing. In a military sample ($n = 601$), Bein, Grau, Saunders, and deRoon-Cassini (2019) found that, while soldiers still perceive military peers, subordinates, and superiors to be more

stigmatizing than non-military family and friends, the overall rates of perceived stigma were much lower than has been seen in previous literature. Additionally, soldiers reported seeking help at higher rates than had been seen in previous literature, which is consistent with other recent trends noted in new generations of veterans (Kulesza et al., 2015). This trend is encouraging and suggests the general finding that social support predicts more willingness to seek out and engage in PTSD treatment (Platt, Keyes, & Koenen, 2014) holds in military populations, especially given the observed decrease in perceived stigma from military others. Given the unique circumstances surrounding the military and social support, it appears to be best practice to tailor interviews with veterans to inquire as to sources of social support and perceived stigma from these sources (Hamilton et al., 2015).

There are several other interesting findings concerning the relationship between social functioning and PTSD that are important to note and integrate into outcome assessment. It appears as if perceived social support is less important than having a diverse social network (i.e., engaging in multiple social groups with different people) in the development and maintenance of PTSD (Platt, Keyes, & Koenen, 2014). Findings were consistent across racial, ethnic, gender, and socioeconomic groups. However, these results are not consistent in the literature. A large epidemiological study assessing PTSD in non-Hispanic White, non-Hispanic Black, and Hispanic women in the United States (Lipsky, Kernic, Qiu, & Hasin, 2016) found that perceived social support inconsistently functions as a protective factor, but that diversity of social networks consistently predicts better treatment outcomes. In a meta-analysis from Brewin, Andrews, and Valentine (2000), the strongest predictor of PTSD development was lack of social support, and

analyses showed relative consistency between perceived social support and diversity of social networks. The importance of diverse social networks is also seen in recent study from Schnurr and Lunney (2016) in which CAPS rating of social impairment and not self-rated social functioning was related to the loss of a PTSD diagnosis.

Taken together, these results suggest that perception of the quality of social functioning and engaging in a diverse range of social activities are potentially important areas of focus, which is an important nuance to note when assessing social functioning. Most of the studies reviewed utilized a variety of metrics to assess perceived social support, although the Interpersonal Support Evaluation List (ISEL12; Cohen et al., 2008), a self-report questionnaire that measures perceptions of the availability of social support, was used most commonly. Measuring social network diversity is typically achieved through use of the Social Network Index (Cohen et al., 2008), a self-report questionnaire that measures participation in 12 different types of social networks over the past two weeks. These measures have substantial evidence in support of their validity and reliability (Lipsky et al., 2016), and appear to appropriate measures for outcomes research. It is also important to keep track of new research designed to increase the availability for culturally-appropriate measures. One potential explanation for the lack of predictive power for perceived social support is the failure to measure culturally-specific forms of social and cultural support (Lipsky, Kernic, Qiu, & Hasin, 2016), which is problematic given the consistent usage of the ISEL12, which was used in all papers assessing social support in this review. For a PTSD program, it appears as if cross-referencing any stand-alone social functioning measure with clinician-rated social

functioning on the CAPS is best practice, as there is research evidence in support of measuring both perceived social functioning and social network diversity.

Occupational Functioning

The literature surrounding occupational functioning in PTSD comprises two general areas: occupational impairment and assessing PTSD-related disability. In terms of the impact of PTSD, Breslau, Lucia, and Davis (2004) found that severity of PTSD was linked to work-loss days and lack of employment. These findings have been seen in multiple populations, so much so that occupational impairment is one of the potential functioning impairments listed in the DSM-5 (De Jongh et al., 2016). It has been suggested that focusing on increasing engagement in work-related activities can be beneficial for people with PTSD, which has minor support in the literature (Levers, 2012; Taylor, Wald, & Asmundson, 2006). However, evidence suggests that the best method to help patients increase occupational functioning is to decrease their symptom severity through evidence-based practice (Schnurr & Lunney, 2016).

Engaging in evidence-based practice is also the recommendation from the literature surrounding assessment of occupational impairment (McNally & Frueh, 2013), though for a slightly different reason. There is mounting evidence that, especially when seeking disability, large numbers of people may malingering PTSD symptoms (Hall & Hall, 2007; McNally & Frueh, 2013; Messer & Fremouw, 2007). A brief discussion of malingering in PTSD can be found in a previous section of this review, including appropriate measures (e.g., DAPS, TSI, MENT-R, PAI, MMPI-2) to assess the validity of PTSD symptoms, specifically. Best practice literature indicates that at least one of these measures should be given during a diagnostic assessment, and ability to detect

malingering increases dramatically with the use of more than one validity measure (McNally & Frueh, 2013).

While some have asserted that malingering is better explained by delayed onset PTSD (e.g., Marx et al., 2012), the evidence does not appear to support this claim. Instead, it is likely that making success of disability claims dependent on a PTSD diagnosis has increased the number of malingering cases (McNally & Frueh, 2013). Additionally, there is evidence that long-term psychiatric disability related to PTSD can be reversed through engagement in evidence-based practice (Marx et al., 2012) and, conversely, that disability payments were linked to lower outcomes of returning to work (Siminski, 2013). While this debate is not likely to be settled in the near future, it is evident that an accurate assessment of occupational functioning, especially when a potential disability claim is involved, should involve validity measures. Finally, with the intention of improving functional outcomes, recommending engagement in ESTs for all people diagnosed with PTSD, regardless of disability claim, is indicated.

Physical Functioning

Across military, civilian, and refugee populations, PTSD is linked to impairments in physical functioning and an increase in the likelihood of greater healthcare utilization, healthcare costs, chronic illness (e.g., fibromyalgia, cardiovascular disease, stroke, and obesity), prescription medication misuse, and decreased involvement in physical activity (Agorastos et al., 2014; McFarlane, 2010; Steinert, Hofmann, Leichsenring, & Kruse, 2015; Trost et al., 2015; Zatzick et al., 1997). The consistency of the negative health-related impacts of PTSD is particularly concerning, because despite the well-established finding that PTSD is linked to negative health outcomes, there is little long-term outcome

research that addresses the relationship between engaging in PTSD treatment and physical health outcomes (Steinert et al., 2015). However, there are several relationships that have been explored in the literature. First, research suggests that untreated pain and physical health problem severity is linked to the development and exacerbation of PTSD, especially when the trauma involves a physical component (e.g., interpersonal violence, workplace injury, mild traumatic brain injury; Archer et al., 2016; Hoge & Engel, 2008). Simultaneously, a challenging trend seen in the development of physical health issues after trauma is a gradual increase in physical health symptom severity that does not match the emergence of PTSD symptomology (Murphy & Busuttil, 2015).

An increasingly accepted explanation of this trend is the cumulative effect of stress on the HPA-axis and autonomic nervous system, which is known as “allostatic load” (McFarlane, 2010). Allostatic load has been proposed as one of the most important concepts for understanding the relationship between PTSD and physical health, as previous theories suggesting that health-related behaviors (e.g., smoking, drug use, and poor exercise) explain this relationship have not been supported in the literature (Shipherd, Clum, Suvak, & Resick, 2014). Additionally, the literature has shown the relationship between allostatic load and PTSD is not mediated by health-related behaviors (Agorastos et al., 2014). Appropriate measurement of allostatic load is well-established, though it requires access to medical testing and diagnostics. Specifically, allostatic load is measured through analyzing nine indicators obtained via a blood test and medical examination including systolic blood pressure, C-reactive protein, and Albumin levels (Shipherd et al., 2014).

While the relationship between allostatic load and PTSD is relatively clear, allostatic load is rarely assessed, especially in the context of outcomes research (McFarlane, 2010). Currently, there is almost no data focused on the relationship between PTSD treatment engagement and allostatic load. Instead, there is considerable evidence that engaging in evidence-based PTSD treatment, especially PE for PTSD, leads to increased HRQoL, specifically general health perceptions, physical role functioning, and bodily pain (Dolezal, Lum, Bentley, & Zoellner, 2018; Pagotto et al., 2015). In the literature, HRQoL is most often assessed using the Short-Form Health Survey (SF-36; Ware & Sherbourne, 1992), which has considerable evidence in terms of reliability and validity (Pittman et al., 2012). However, from the little data available concerning the long-term ability of PTSD treatment to increase HRQoL, it does not appear that improvements are maintained post-treatment (Dolezal et al., 2018). As such, as has been suggested in other measurement domains in this review, it appears to be crucial to assess long-term outcomes to determine if current treatment modalities have HRQoL benefit after treatment. In settings able to complete medical testing, it appears as if measuring allostatic load in conjunction with HRQoL at pre-treatment, post-treatment, and follow-up can provide valuable insight into the ability of PTSD treatment to impact the relationships seen in the literature.

Well-being & Quality of Life

The sections of this review on spiritual functioning (including moral injury and posttraumatic growth), interpersonal and social functioning, occupational functioning, and physical functioning comprise most of the component evidence for quality of life. Additionally, in many ways, these sections provide the building blocks for understanding

quality of life and well-being, as the concepts are intrinsically linked conceptually and in the specific domains they assess (Zatzick et al., 1997). In fact, there is considerable debate concerning the benefits of separately measuring well-being and quality of life, with the consensus being that due to large overlap between the constructs, it is most beneficial to create robust measures that assess domains germane to both constructs (Medvedev & Landhuis, 2018). This followed a long-standing divide between measures of well-being and quality of life that began to converge after the following definition was proposed: “The theoretical formulation of well-being was thus supported as a multifaceted domain encompassing positive self-regard, mastery of the surrounding environment, quality relations with others, continued growth and development, purposeful living, and the capacity for self-determination” (Ryff & Keyes, 1995, p.724). The extent of this debate is too broad to cover in this review, yet the combination of well-being and quality of life into a larger, comprehensive metric is notable.

A large survey-based study ($n = 2533$) conducted using data from 11 countries from Skevington and Böhnke (2018) assessed the relationship between subjective well-being and quality of life using the World Health Organization Quality of Life measure assessing Spirituality, Religious, and Personal Beliefs (Skevington, Gunson, & O’Connell, 2013). Analyses suggested the benefit of combining subjective well-being and quality of life into a single, larger metric that does explicitly focus on subjective well-being. The areas discussed in the sections above, including physical health, social support, familial functioning, spirituality, and resilience, are all represented conceptually, suggesting the benefit of assessing quality of life via the best practice assessments discussed in the previous sections or using a larger, comprehensive metric.

Processes of Well-being in PTSD

When viewed through the process-based lens discussed earlier in this review, the relationship between quality of life and PTSD becomes slightly less clear than might be indicated in the literature. Quality of life is conceptualized as an outcome or outcomes rather than a process or processes (Howgego et al., 2005). While previous research has shown the ability of PTSD treatment, specifically PE for PTSD, to improve quality of life (Dolezal et al., 2018), the processes by which this improvement is achieved are not as well understood. One biological process, allostatic load, has already been shown to be a potentially important moderating variable in the relationship between PTSD and quality of life, and current research on quality of life has been focused on identifying mediating and moderating variables to explain the relationship between treatments for DSM disorders and quality of life (Gu, Strauss, Bond, & Cavanagh, 2015). In PTSD, this area of research is relatively new, and there is still a substantial amount of work that needs to be done to make any strong conclusions. However, evidence does exist that several psychological processes are important mechanisms of change in PTSD outside of symptom severity. These processes with the most empirical support in the literature are: mindful awareness, compassion, emotion regulation, acceptance, and psychological flexibility. These processes, when targeted in conjunction with empirically supported treatments focused on symptom reduction (e.g., PE for PTSD, CPT), can facilitate improved quality of life beyond the effect of empirically supported treatments alone (Boyd, Lanius, & McKinnon, 2018; Gu et al., 2015; Gratz & Tull; Hoffart, Økstedalen, & Langkaas, 2015; Kashdan et al, 2009). It is important to note that more research is necessary to tease apart these processes of change, as core exercises (e.g., mindfulness

meditation) involve the cultivation of multiple processes. In the following sections, the evidence in support of these processes will be reviewed.

Psychological Inflexibility and Experiential Avoidance

The psychological flexibility model, proposed by Hayes, Strosahl, and Wilson (1999), is the foundation for Acceptance and Commitment Therapy. The model includes six core processes: “(1) cognitive fusion / defusion, (2) experiential avoidance / acceptance, (3) loss of flexible contact with the now / present moment focus, (4) attachment to a conceptualized self/ self as context, (5) values problems / chosen values, and (6) inaction, impulsivity, or avoidant persistence / committed action” (Levin et al., 2012, p. 5). In this theoretical model, each of these core processes contribute to the construct of psychological flexibility.

Overall, the evidence for the utility of the AAQ-II, the main measure used to assess psychological inflexibility (the opposite of psychological flexibility), is strong. Psychological inflexibility (as measured by the AAQ-II) has been shown to mediate the relationship between trauma exposure and PTSD symptom severity in prolonged childhood abuse (Shenk et al., 2014), veterans (Meyer et al., 2018), and refugees (Kashdan et al., 2009). However, there is considerable concern about the construct validity of the AAQ-II, although these concerns focus on the assertion that the AAQ-II measures psychological inflexibility, as opposed to the assertion that it measures acceptance versus experiential avoidance (Rochefort, Baldwin, & Chmielewski, 2018; Wolgast, 2014). Experiential avoidance can be defined as “an unwillingness to experience psychological events even when attempts to escape or avoid such events have caused behavioral harm” (Hayes, Wilson, Gifford, Follette, & Strosahl, 1996, p. 1158).

Specifically, it is unclear if the AAQ-II is an appropriate measure of psychological flexibility vs. experiential avoidance, or if it is a measure of acceptance vs. experiential avoidance (Gloster, Klotsche, Chaker, Hummel, & Hoyer, 2011). This confusion can be seen throughout the literature, with some studies referring to the AAQ-II as measuring acceptance (e.g., Kotsou, Leys, & Fossion, 2018), and others referring to the AAQ-II as measuring psychological flexibility (e.g., Meyer et al., 2018). This is an issue that requires additional convergent and divergent validity research.

One example of research in this vein is a meta-analysis from Levin et al. (2012) in which 66 component analyses were evaluated. Results were favorable for the overall construct of psychological inflexibility, though a component analysis revealed a large effect size for acceptance and medium to large effect sizes for cognitive defusion, values, and present moment components. As such, it appears to be most helpful to assess these constructs both individually and in relation to one another. In the PTSD literature, there is substantial support for acceptance, defusion, and present moment awareness as processes of well-being, and the evidence for each will be discussed in the following subsections. Because there is ample evidence that the AAQ-II measures experiential avoidance, acceptance, and aspects of valued living (Gloster et al., 2011; Meyer et al., 2018), which are all components of the psychological flexibility model, it appears acceptable to view the AAQ-II as a measure of psychological flexibility (with a strong emphasis on the acceptance component) vs. psychological inflexibility.

Acceptance vs. Experiential Avoidance. Experiential avoidance is thought of as being central to the development and maintenance of many DSM disorders, including PTSD (Orsillo & Batten, 2005). As has been emphasized in this review, the problem of

comorbidity in PTSD treatment is still central in the development of effective treatments. What is important to note about experiential avoidance is that it has been shown to be a core process in the development and maintenance of the most common comorbid disorders for PTSD, including personality disorders (Iverson et al., 2012), substance use disorders (Levin et al., 2012), panic disorder (Barlow, 2014), social anxiety disorder (Kashdan et al., 2009), and major depressive disorder (Spinhoven et al., 2016). As such, experiential avoidance appears to be a transdiagnostic process that, if targeted effectively, can address PTSD and comorbid disorders simultaneously.

In PTSD specifically, the avoidance of distressing thoughts through attempted thought suppression, the avoidance of trauma-related reminders via physical avoidance, and the avoidance of distressing emotions via numbing are all central factors that contribute to the development and maintenance of PTSD (Orsillo & Batten, 2005; Powers et al., 2010). Exposure-based treatments for PTSD are centered around the concept of re-engaging with previously avoided stimuli in a safe context, especially those that have become trauma-related (Green et al., 2017). It has been proposed that CPT and PE for PTSD, the two PTSD treatments with the strongest evidence base, are both addressing EA, albeit through different approaches (Green et al., 2017). CPT addresses experiential avoidance by way of changing posttraumatic cognitions and PE for PTSD addresses it by way of in vivo and imaginal exposure. As has been discussed in this review, the evidence base favors PE for PTSD by a narrow margin. However, the evidence suggesting experiential avoidance as a shared mechanism of change between treatments begs the question: How can treatments for PTSD actively target experiential avoidance? Given the

conceptualization of acceptance and experiential avoidance as opposite ends of a spectrum, one answer appears to be an explicit focus on acceptance in psychotherapy.

When behaviorally defined, acceptance is “approaching (often distressing) psychological events and related situations, without trying to change, avoid, suppress, escape from, or prolong them” (Forsyth & Ritzert, 2018, p. 663). PE for PTSD and CPT explicitly target the reduction of experiential avoidance and both treatment approaches indirectly target acceptance of trauma-related cognitions, emotions, and reminders (Vujanovic et al., 2009). The process of a patient accepting, rather than approving of, their trauma has been linked to an increase in quality of life after engaging in evidence-based trauma treatment (Cloitre, 2015; Harned et al., 2014; Walser, 2006). Preliminary trials of ACT for PTSD (e.g., Land, 2010; Orsillo & Batten, 2005), which explicitly targets acceptance, have suggested the benefit of accepting internal events and treating experiential avoidance through the cultivation of acceptance and movement towards values. One of the proposed benefits of this approach is increased treatment adherence and lowered dropout rates (Land, 2010), which is a problem in all treatments for PTSD (Ehring et al., 2014). The evidence suggesting ACT as a comprehensive intervention for PTSD is incomplete, though growing (Bluett, 2017). However, the evidence supporting the role of core processes of change in ACT, including acceptance, is strong. As such, the combination of intentional acceptance and engagement in exposure-based treatment for PTSD appears to provide an avenue to address EA in a more robust manner than has been seen in manualized treatment alone.

It should be noted that acceptance is viewed as a behavioral skill that requires practice (Forsyth & Ritzert, 2018). The most common method to practice acceptance is

through mindfulness meditation, though experiential activities from the ACT literature have shown promising preliminary results (Hayes & Hofmann, 2018). Consistently, mindfulness meditation shows robust effects on the increase of acceptance. For example, in a recent study from Boyd, Lanius, and McKinnon (2018) even a novice meditator showed small to medium effect sizes increases in acceptance of distressing mental events. It is important to note that evidence suggests a specific, validated measure of acceptance and related constructs such as the AAQ-II might be better at measuring acceptance than a measure designed to assess mindfulness such as the Mindful Attention and Awareness Scale (MAAS; Brown & Ryan, 2003; Boyd et al., 2018). However, this is not a long-term solution for measuring acceptance, as the AAQ-II has been suggested as best measuring psychological inflexibility (Ong, Pierce, Woods, Twohig, & Levin, 2019).

Clinically, there is some evidence that group therapy with sessions explicitly focused on acceptance (e.g., DBT or ACT groups) can help to build acceptance skills (Becker & Zayfert, 2001; Harned et al., 2014). Finally, a core focus on acceptance as a healing process appears to be effective across cultures, including in non-Western cultures in which the validity of the PTSD construct has been hotly debated (Hinton, Pich, Hofmann, & Otto, 2013; Marsella, 2010). Ultimately, it appears as if acceptance is a potentially important process of change in PTSD, especially with respect to quality of life. In treatment, it also appears as if dedicating time towards building acceptance and psychological flexibility skills through mindfulness mediation and assessing those skills using appropriate measures (e.g., the AAQ-II) is appropriate and beneficial.

Present-moment Awareness

While mindfulness meditation can facilitate an increase in acceptance, there is also evidence that mindfulness awareness, or attention flexibility, is an essential process of wellness in PTSD. In a systematic review of mindfulness-based interventions for PTSD, Boyd et al. (2018) found medium to large effect size increases in attentional control and HRQoL that coincide with decreases in PTSD symptom severity. This may be partially due to a lack of component analysis of mindfulness measures, yet the current literature clearly supports the benefits of increased attentional control in PTSD. In both PTSD and depression, the combination of mindful awareness and acting with awareness is linked to decreased rumination and increased quality of life (Costa, Gouveia, & Marôco, 2018, Land, 2010; Levin et al., 2012). These findings are corroborated by emerging neurobiological evidence that engagement in mindfulness meditation and acting with mindful awareness modulates limbic system activation in PTSD patients, which is linked to a decrease in anhedonia and externalizing behaviors and an increased ability to effectively engage in PE for PTSD (Boyd et al., 2018; Fonzo et al., 2017). Additionally, engaging in mindfulness meditation has been shown to decrease alexithymia in PTSD patients by way of increasing right insula cortical thickness, which is correlated with greater emotional awareness and regulation (Lanius et al., 2015). This may be the most encouraging finding regarding mindful awareness, as people with PTSD typically have a great deal of difficulty identifying and engaging with difficult emotions, and alexithymia is a negative prognostic factor for treatment (Twohig, 2009).

Many measures include components of mindfulness, including the AAQ-II. A systematic review of mindfulness instruments from Park, Reilly-Spong, and Gross (2013)

assessed 46 articles that evaluated 10 different mindfulness measure. Their analysis was conducted using the Consensus-based Standards for the Selection of Health Status Measurement Instruments checklist, which assesses reliability, validity, and responsiveness. From this strong methodological framework, the review found that the MAAS (Brown & Ryan, 2003) had the strongest evidence base, but that additional research is needed on all instruments reviewed, especially factor analytic and convergent validity evidence. As such, in a PTSD outcomes program, it appears to be best practice to perform confirmatory factor analysis on any mindfulness scale administered, which is in conjunction with best-practice statistical recommendations (Chan, 2009). Measurement of neurobiological changes is beyond the abilities of most outcomes research programs, yet this evidence (present and future) is important to note for any PTSD outcomes research program.

Cognitive Fusion and Rumination

Two constructs that are closely related but purport to measure distinct processes are cognitive fusion and rumination. Cognitive fusion occurs when “a person acts on thoughts as though they are literally true, cognitive events come to dominate behavior and experience over other sources of behavioral regulation, and he or she becomes less sensitive to direct consequences” (Gillanders et al., 2014, p. 84). Much like the relationship between experiential avoidance and acceptance, cognitive fusion is thought of as being on one end of a spectrum with cognitive defusion, or “unhooking” oneself from the belief that cognitive events are literally true representing the other end (Levin et al., 2012). Cognitive fusion is most often measured using the Cognitive Fusion Questionnaire (Gillanders et al., 2014), which has demonstrated strong reliability,

validity, and factor analytic properties across treatment populations (Bardeen & Fergus, 2016; Nieuwsma et al., 2015). The definition of rumination, a related, yet theoretically distinct construct, is typically based on the Response Style Theory (RST; Nolen-Hoeksema, 1991), which defines rumination as repetitively thinking about the causes, consequences, and symptoms of a person's negative affect. This model is widely used throughout the literature, though it has mixed empirical support and, in some studies (e.g., Smith & Alloy, 2009), has been found to have poor stability and discriminant validity with measures of depression or anxiety. In PTSD, rumination is thought to fall under the symptom cluster of "negative alterations in cognitions and mood" and is often assessed using the IES-R or factor analytically using the PCL-5 (Armour et al., 2015; Green et al., 2017; Smith & Alloy, 2009). Cognitive fusion is distinguishable from rumination in that the focus is on the relationship to mental events rather than the repetition of those events. The initial scale analysis supported this distinction and found that cognitive fusion added additional variance above and beyond rumination in several large community samples (Gillanders et al., 2014).

Targeting both cognitive fusion and rumination has been shown to decrease PTSD symptom severity and increase quality of life (Bardeen & Fergus, 2016; Green et al., 2017; Jones, 2016), and both constructs have been suggested as core processes that influence the maintenance and development of PTSD (Ehring et al., 2014; Walser, 2006). What is currently unclear in the literature is how these two constructs interact with respect to PTSD, specifically. There is evidence that in PTSD, only specific types of rumination (e.g., focusing on how the trauma shapes one's identity) significantly impact the development and maintenance of negative cognitive alterations in PTSD (Bardeen &

Fergus, 2016), which is conceptually linked to the fusion of self-concept with the impacts of trauma seen in cognitive fusion models (Boals & Murrell, 2016). However, this evidence base is just beginning to take shape, especially in PTSD. In depression, there is growing evidence that cognitive fusion, while related to rumination, should be a central target in treatment due to the unique ability of cognitive defusion to decrease depressive symptoms beyond typical cognitive restructuring methods (e.g., thought challenging; Costa et al., 2018). Even these results are preliminary, and the evidence in support of explicitly changing cognitions, not necessarily the relationship to those cognitions, in PTSD is uniquely strong (Green et al., 2017; McLean et al., 2015). More research is necessary to determine the relationship between rumination and cognitive fusion in PTSD, especially because decreases in both cognitive fusion and rumination have been linked to decreases in PTSD symptoms and quality of life. It appears to be important to replicate the model from McLean et al.'s (2015) study of cognitive fusion, rumination, and depressive symptomology. Translated to PTSD, this design allows the mechanisms of change (changing trauma-related thoughts vs. changing relationships to trauma-related thoughts) to be elucidated.

Emotion Regulation

Another proposed mechanism of change in PTSD, emotion regulation, has a large amount of evidence in support of its transdiagnostic validity. Specifically, emotion regulation has been shown to underlie psychological distress in substance use, depression, generalized anxiety disorder, personality disorders, and PTSD (Barlow, 2014; Gratz & Tull, 2010; Hayes & Hofmann, 2018; Hofmann & Barlow, 2014). Additionally, there is meta-analytic neurobiological evidence that engaging in ESTs, including PE for

PTSD, inhibits emotional reactivity via the amygdala and the specific cortical regions involved with emotion regulation (Frank et al., 2014; Hofmann & Barlow, 2014; Yehuda et al., 2015). Exposure-based treatments are especially effective at addressing emotion regulation, as the movement towards willingly tolerating distressing emotions builds up emotion regulation skills behaviorally and neurobiologically (Frank et al., 2014; Gratz & Tull, 2010; Yehuda et al., 2015).

In a recent study from Cox, Motl, Bakker, & Lunt (2018), a meditation model found that addressing emotion regulation allowed for cognitive defusion in a veteran sample with PTSD. This in turn led to PTSD symptom reduction and increased quality of life (as measured by life satisfaction). This study utilized the PCL-5, the CFQ, and the Difficulties with Emotion Regulation Scale (Gratz & Roemer, 2004), which is a commonly used measure of emotion regulation with strong validity and reliability evidence (Cox et al., 2018). The study re-evaluated each scale for internal consistency in their sample, which adds strength to the findings. The results mirror findings from Gratz and Tull (2010) that suggest emotion regulation is a key process that contributes to the success of mindfulness and acceptance-based interventions. Additionally, while engaging in exposure-based PTSD treatments improve emotion regulation skills, populations with more severe emotion regulation difficulties (e.g., patients with personality disorders) have been shown to benefit from modified PTSD treatment protocols with an increased focus on building up emotion regulation skills such as DBT and ACT (Harned et al., 2014; Shenk et al., 2014). As such, it appears as if emotion regulation is a core process of well-being in PTSD that can be targeted by ESTs and adjunctive treatment approaches (e.g., DBT & ACT).

Compassion and Shame

The final proposed process of well-being in PTSD is compassion. Compassion can be viewed as one end of a spectrum with the other end being shame (Gilbert et al., 2017). Compassion research in PTSD is almost uniformly focused on self-compassion, and most of this section of the review will focus on this research. However, it is helpful to note that compassion has also been conceptualized as a tri-fold flow consisting of compassion towards others, compassion towards self, and receiving compassion from others (Gilbert, 2010). This conceptualization of compassion is measured through the Compassionate Engagement and Action Scales for Self and Others (Gilbert et al., 2017). Theoretically, this approach to measuring compassion has great utility in PTSD, as people with PTSD typically have difficulty feeling connected to self and others (Foa et al., 2008), which would likely lead to deficits in all areas measured by the scale. Additionally, as has been discussed previously in this review, strengthening and broadening social support networks and reducing emotional avoidance is linked to increased quality of life, so targeting feelings of connection with self and others via compassionate meditation and experiential exercises, as is the approach in compassion-focused therapy (CFT; Gilbert et al., 2017), would theoretically increase quality of life in people with PTSD. However, the only aspect of compassion with significant research evidence in PTSD populations is self-compassion, which will be discussed in the following paragraphs.

To understand self-compassion's role as a process of improving well-being in PTSD, it is helpful to explore the ways in which shame impacts PTSD development and maintenance. Shame is defined as the belief that one's identity is flawed, wrong, or

broken (Lee, Scragg, & Turner, 2001). Shame is a particularly important construct for treatment conceptualization (and part of the diagnostic criteria for PTSD), as high levels of shame has been linked to decreased engagement in therapy and reticence to disclose trauma-related cognitions to the treating therapist (Jones, 2016; Steinmetz, Gray, & Clapp, 2019). As a result, failure to address shame has been generally linked to treatment failure across modalities and populations, even more than peri-traumatic fear, which was long seen as the greatest predictor of dropout (Crocker, Haller, Norman, & Angkaw, 2016; Green et al., 2017). This is an especially relevant issue in military and civilian populations, as returning veterans who exhibit strong endorsement of traditional gender norms and people who have experienced sexual assault are two populations with the highest likelihood of developing trauma-related shame (Beks, 2016; Cloitre, 2015; Harned et al., 2014).

In mediation studies, shame has been linked to decreased quality of life, including poorer social, physical health, and occupational domains (Au et al., 2017; Badour, Resnick, & Kilpatrick, 2017). Additionally, shame has been linked to the development of EA in PTSD, especially with respect to avoidance of difficult emotions (Gratz & Tull, 2010). It has been suggested that the explicit targeting of shame in CPT leads to increased emotion regulation, which may be the primary mechanism by which the treatment approach is efficacious (Yehuda et al., 2015). However, there is also evidence that PE for PTSD can reduce shame across a wide range of traumatic exposures and civilian, military, and refugee populations (Crumlish & O'Rourke, 2010; Langkaas et al., 2017), which again suggests the possibility that reducing experiential avoidance of difficult emotions is a core process of change in evidence-based therapies for PTSD. However,

this evidence is preliminary, and is not as strong in survivors who have experienced multiple traumas or prolonged interpersonal trauma (Cloitre, 2015). Conceptually, it has been suggested that shame replaces a previously positive identity with a shame-based identity (Lee et al., 2001), and in subsequent traumas, activates and reinforces those maladaptive schematic representations of self (Au et al., 2017). These shame-based schemas are exceptionally difficult to target, and there is no consensus concerning the most effective way to target shame across disorders, including PTSD (Au et al., 2017).

Different measures of shame are common in the literature, although several self-report questionnaires are commonly used in PTSD, specifically: The Internalized Shame Scale (ISS; Cook & Coccimiglio, 2001), and the Experiences of Shame Scale (ESS; Andrews, Qian, & Valentine, 2002). In a meta-analysis from Saraiya and Lopez-Castro (2016), these measures were shown to have strong reliability and validity evidence, although treatment approaches addressing shame remained unclear. An alternative measure that may be helpful in assessing shame and other schematic representations of self is the Young Schema Questionnaire (YSQ; Schmidt, Joiner Jr., Young, & Telch, 1995). Originally developed by Young (1991) to assess early maladaptive schemas in depression and borderline personality disorder, there is a growing evidence base that the YSQ (which has strong evidence in support of its reliability and validity) allows for the identification of numerous important schematic representations of self in PTSD, including shame-based schemas (Karatzias et al., 2016; Taylor, Bee, & Haddock, 2017). This may be a helpful alternative to a strictly shame-focused measure, as a more broad-based assessment of maladaptive schemas can help tailor treatment (including treatment for shame) to the client's unique negative alterations in cognition.

Self-compassion. Self-compassion is measured using the Self-Compassion Scale (SCS; Neff, 2003), which also exists in a short form (SCS-SF; Raes, Pommier, Neff, & Van Gucht, 2011). While there is considerable debate concerning the appropriate factor analytic structure of the SCS (e.g., Neff, 2016), the psychometric properties of the scale have been replicated numerous times in international samples (Au et al., 2017), the findings from which have also been replicated in PTSD populations (Hoffart et al., 2015). Ultimately, as it the case for most scales, it appears to be vitally important to test the factor analytic structure of the scale in the specific population in which analysis takes place, as no definitive conclusions concerning the factor structure has been reached.

The evidence in support of cultivating self-compassion as a core process of well-being in PTSD is growing, though incomplete. In a review of the self-compassion literature, Au et al. (2017) found medium to large effect size reductions following compassion-focused interventions (e.g., loving-kindness meditation and compassionate mind training). This study also assessed the ability of a six-week compassion-focused intervention (i.e., combining elements of compassionate mind training and loving-kindness meditation) to reduce PTSD symptom severity. The study was well-designed and assessed PTSD symptom severity (as measured by the PCL-5), self-compassion (as measured by the SCS) and shame (as measured by the ISS) weekly, starting at baseline (baseline was randomized to two, four, or six-weeks). Importantly, follow-up data was also collected. The effect size reductions in PTSD symptom severity (Cohen's $d = 1.10$) and shame were large (Cohen's $d = 1.03$), as were increases in self-compassion (Cohen's $d = 1.46$). While these large effect sizes are encouraging, the follow-up data suggests these gains were not only maintained but improved upon. Additionally, this study

experienced a very low dropout rate, and the rated acceptability of the interventions was high. These findings mirror those of Thompson and Waltz (2008), Dahm et al. (2015), Kearney et al. (2013), and Hoffart et al. (2015), who have all found medium to large effect size decreases in PTSD symptom severity (and, when measured, shame) that coincide with medium to large effect size increases in self-compassion following treatment programs either exclusively focused on self-compassion or for PE for PTSD augmented by compassion-focused exercises.

The limitations of this line of research are, in some ways, reflective of the limitations of process-based research in PTSD. Many studies (e.g., Au et al., 2017) are preliminary examinations, meaning they have small sample sizes, are primarily conducted with participants who are White and female or male veterans, and exclude participants with comorbidities. Only a few studies (e.g., Hoffart et al., 2015) have examined the impact of cultivating self-compassion while concurrently engaging in an evidence-based treatment program for PTSD. These limitations (as well as the noted cautious optimism regarding study findings) have been reiterated in recent reviews of the role of self-compassion in PTSD treatment (e.g., Braehler & Neff, 2020; Winders, Murphy, Looney, & O'Reilly, 2020). Additionally, recent evidence has shown that self-compassion might be a more important treatment target in cPTSD than in non-cPTSD (Karatzias et al., 2019) due to the presence of disturbances of self in cPTSD. According to this new research, self-compassion's role in PTSD treatment may be helping those patients who have experienced complex, repeated trauma develop a kind and caring relationship with the self, which can offset this population's oft-reported experience of unstable identity. These results are preliminary, yet promising. As such, the

preponderance of the evidence suggests cultivating self-compassion in PTSD could be a core process of well-being, especially for those with high levels of shame or identity disturbances. However, given the lack of evidence from active treatment programs, it is difficult to make any definitive conclusions.

Gaps in Process-based PTSD Research

Currently, it is unclear how these processes of well-being are interrelated in PTSD, as mediation studies that assess multiple process measures have produced contradictory results. Acceptance has been found to mediate the relationship between PTSD symptoms and both emotion regulation and present-moment awareness (Kotsuo, Leys, & Fossion, 2018). Emotion regulation has been found to mediate the relationship between PTSD symptoms (and to a lesser extent, quality of life) and cognitive fusion (Cox et al., 2018). Experiential avoidance has been found to mediate the relationship between PTSD symptom severity (and to a lesser extent, quality of life), emotion regulation (Shi, Zhang, Zhang, Fu, & Wang, 2016), self-compassion (Seligowski, Miron, & Orcutt, 2015) and cognitive fusion (Jones, 2016). This research is even more limited in treatment-seeking individuals with a PTSD diagnosis. To our knowledge, only one study (Grau, McDonald, Clark, & Wetterneck, 2020) has explored the relationship between experiential avoidance, cognitive fusion, and valued living in this type of sample, finding that experiential avoidance was a more robust predictor of admission PTSD symptom severity than cognitive fusion and valued living.

Adding to the confusion is the theoretical connection between the discussed processes of well-being in PTSD. For example, it has been suggested that cultivating self-compassion positively impacts emotion regulation (Neff & Dahm, 2015), which has been

posited to improve the ability to contact the present moment (Au et al., 2017), although mindfulness is also a core component of self-compassion (Neff, 2003). Additionally, cultivating self-compassion has been suggested as a process that can help increase the efficacy of cognitive defusion exercises by allowing patients to approach difficult thoughts mindfully and with a sense of shared human experience of suffering (Jones, 2016).

This may be a measurement issue in the current literature yet is it perfectly plausible that these processes are all uniquely helpful in facilitating the reduction of PTSD symptoms while improving quality of life. Examining ways in which multiple processes can be targeted at once and measuring the processes according to the best practice guidelines discussed in this review would likely be helpful and has been suggested by process-based researchers (e.g., Hayes & Hofmann, 2018). Augmenting traditional ESTs to explicitly target these processes and measuring how these processes interact to contribute increases in quality of life and decreases in PTSD symptom severity in these types of programs (e.g., PE for PTSD + DBT, ACT, or CFT) appears to be the best way to reach that goal.

Integration

Over the course of this review, best practice recommendations for engaging in PTSD outcomes research have been analyzed and discussed. Due to the heterogeneity of PTSD risk factors, development, and course, more research is needed in all areas, especially with traditionally underserved and underrepresented populations (e.g., REM clients). However, there are several strong lines of evidence with respect to symptom

severity reduction, quality of life, and processes of wellness that have emerged from this literature review.

The ability to focus on well-being and improving patient functional outcomes is particularly important in treatment for patients with complex, repeated trauma. For these patients, the experience of trauma does not appear to be fully captured by the symptom checklists available for PTSD. Effective outcomes research must move beyond measures of symptom reduction and assess quality of life across multiple domains. However, symptom reduction is still an essential component of treatment, and there is a substantial (and ever-growing) evidence base with respect to the most effective approaches to treating PTSD. Based on this review, it appears that ESTs, especially PE for PTSD and CPT, are best able to reduce all clusters of PTSD symptoms, with PE for PTSD having slightly stronger evidence with respect to long-term outcomes than CPT. Overall, despite some variant findings, it appears as if trauma-focused treatment is most effective in reducing PTSD symptoms and has been shown to facilitate improvements in quality of life. Multiple valid and reliable assessments exist for this purpose, including the PCL-5 and the CAPS, and the literature supports the concurrent use of either personality measures with validity indexes (e.g., MMPI-2-RF or PAI) or PTSD measures with embedded validity indexes (e.g., TSI).

The assessment of quality of life in PTSD is complex, and, when possible, should be somewhat individualized for each client. This domain is closely linked to functional impairment, which can be assessed using biopsychosocial measures, global functioning measures, and with corroborating records. There are several potential approaches to assessing quality of life that have support in the literature, including assessing HRQoL or

administering individual measures of physical health, familial functioning, spirituality, resilience, and social support. It appears that the Q-LES-Q-SF measure has strong utility in assessing quality of life and can be helpful in focusing in quality of life assessment to essential ingredients. Additionally, for specific populations that experience unique challenges to quality of life (e.g., moral injury in military populations), it might be important to include relevant measures.

The discussion in this literature review surrounding processes of well-being in PTSD is particularly important for the future of effective PTSD treatment. In particular, the findings from this literature review have shown that the syndromal model of PTSD, while appropriate in some ways, is incomplete, especially with respect to quality of life. While several processes (e.g., self-compassion, interpersonal connection, and psychological flexibility) appear to be crucial in improving quality of life in people with PTSD, it is unclear how these processes interact, and what methods of targeting them are most effective. As such, it is important to examine these processes in clinical populations to help clinicians and researchers design and implement more efficient and expansive approaches to treatment. For reference, a list of acronyms discussed in this review is included in Table 1.

Table 1

Acronym Reference List

PTSD	Posttraumatic Stress Disorder
FIT	Feedback-Informed Treatment
MBC	Measurement-Based Care
AAQ	Acceptance and Action Questionnaire
ROM	Routine Outcome Monitoring

RCT	Randomized Controlled Trial
cPTSD	Complex Posttraumatic Stress Disorder
ESTs	Empirically-Supported Treatments
RCI	Reliable Change Index
REMs	Racial Ethnic Minorities
PE	Prolonged Exposure
CPT	Cognitive Processing Therapy
EMDR	Eye-movement Desensitization and Reprocessing
CAPS	Clinician-administered PTSD Assessment

Chapter 3

Method

Participants

Data were obtained from 537 adult patients who enrolled in an exposure-based PTSD partial hospitalization program (PHP), or intensive outpatient program (IOP) located in 8 Rogers Behavioral Health hospital satellite locations across the Midwest. Full demographic data is presented in Table 2. 75% of patients identified as White and 83% identified as female. The average age in the dataset was approximately 36 (min = 17, max = 79). 78% of our sample had at least one comorbid diagnosis, with Major Depressive Disorder (48%), Bipolar Disorder (17%), Generalized Anxiety Disorder (13%), and Alcohol Dependence (9%) being the most common comorbid conditions. Other comorbid diagnoses included Borderline Personality Disorder (6%), Panic Disorder (5%), and Opioid Dependence (5%).

Table 2

Sample Demographics

Variables	N (%) or <i>M (SD)</i>
<i>N</i>	537
Female	446 (83%)
Male	90 (17%)
Race	
White	400 (75%)
Black	41 (8%)
Pacific Islander	4 (1%)
Native American	8 (2%)
Asian	6 (1%)

Multiple	1 (<1%)
Unknown	46 (9%)
Ethnicity	
Hispanic or Latino	27 (5%)
Not Hispanic or Latino	457 (85%)
Did Not Indicate	30 (5.6%)
Age	36.10 (12.7); (Range: 17-79)
Education	
Some College	156 (29%)
Bachelor's Degree	90 (17%)
High School Degree	50 (9%)
Other	142 (26%)
Associate's Degree	32 (6%)
Master's Degree	43 (8%)
Marital Status	
Married	137 (26%)
Divorced	62 (12%)
Single	261 (49%)
Separated	24 (5%)
Widowed	11 (2%)
Unknown	19 (3%)
Sexual Orientation	
Heterosexual	183 (80%)
Gay, Lesbian, or Bisexual	31 (14%)
Did Not Indicate	14 (6%)

Trauma exposure was assessed using the Life Events Checklist (Weathers, Blake, et al., 2013), a self-report measure designed to allow participants to report lifetime exposure to a wide variety of traumatic events. 14% of participants did not indicate trauma exposure, so these data are not available for analysis. In this sample, total trauma exposure ($M = 12.05$, $SD = 4.05$) was approximately four times higher than rates found in the general population (cf. Benjet et al., 2016). The most common trauma exposures in

this sample were physical assault (77%), sexual assault (74%), and transportation accidents (72%), while the least common trauma exposures were causing serious injury to someone else (15%) and combat/warzone exposure (25%). In this sample, only 3% of participants indicated experiencing non-interpersonal traumas (e.g., transportation accident, natural disaster) without experiencing interpersonal trauma (e.g., sexual assault, physical assault, emotional abuse). As such, 97% of this sample experienced at least one interpersonal trauma. It should be noted that the LEC checklist does not measure total number of exposures, so it is likely that the vast majority of patients in the program experienced more than one instance of an indicated trauma (i.e., multiple assaults).

Procedure

The treatment in these evidence-based PTSD programs focuses concurrently on symptom reduction (via Prolonged Exposure for PTSD) and improving quality of life via the pursuit of meaning and values and engagement in contextual behavior treatment components (e.g., Acceptance and Commitment Therapy, Dialectical Behavioral Therapy, Compassion-Focused Therapy). Patients are screened by a psychologist and psychiatrist for diagnostic fit (i.e., patients have a primary diagnosis of PTSD) with the program. While PTSD must be the primary diagnosis for program admission, most patients present with at least one comorbid condition and have been in previous psychotherapy. Outside of having a primary diagnosis of PTSD, there are no strict exclusionary criteria for program participation, as the supervising psychologist and psychiatrist have final authority with respect to admission. However, active psychosis, significant cognitive impairment, and untreated substance use disorder generally result in admission to alternative treatment programs. All patients are given the Clinician-

Administered PTSD Scale and the Mini-International Neuropsychiatric Interview (MINI) upon admission to further validate a primary diagnosis of PTSD, as well as the PTSD Checklist for DSM-5. Data from these assessments are not integrated into the aggregated master database available for research purposes, so they were not available for analysis. The program does not administer a dedicated measure of malingering, though in accordance with recommendations from Marx et al. (2012), supervising psychologists consider patient records, clinical interview results, and the malingering index of the PAI during the admissions process. Additionally, because the treatment program is structured, evidence-based, and requires consistent treatment engagement for continued enrollment, empirical studies have demonstrated that the obtained results have a low likelihood of bias from malingering (Marx et al., 2012). Unfortunately, at the time of this project, malingering data from the PAI was not fully integrated into the aggregated master database available for research purposes, so it was not available for analysis.

Data were collected at three time points during patients' first week in the program: at the step-down transition from partial hospitalization to intensive outpatient programming, and upon discharge. These timepoints are approximately 6 weeks apart, though there is variation due to differing individual patient needs. Additionally, weekly data measuring self-compassion, PTSD, and valued living is collected and was included in their respective growth curve analyses. Assessments are given in a uniform assessment battery administered on a HIPPA-compliant tablet. Patients' received and signed informed consent papers prior to beginning the program indicating their consent to have their deidentified assessment data utilized for research purposes. As only consenting participants are included in databases available for analysis, no data were available to

contrast those who did not consent to engage in research with those who did. IRB approval was obtained before any analyses were conducted.

Measures

Standard deviations and mean scores were calculated for all scales. Because Cronbach's alpha is a measure of inter-item consistency and not reliability (Sijtsma, 2009), Omega and maximal reliability (Peters, 2014) were also calculated to demonstrate scale reliability.

PTSD Checklist (PCL-5)

The PCL-5 ($M = 54.17$, $SD = 12.70$) is a 20-item self-report measure used to examine symptoms of PTSD severity by mapping on to PTSD criteria from the DSM-V (Weathers, Litz, et al., 2013). The measure is scored using a 5-point Likert scale (1 = Not at all, 4 = Extremely) where higher scores indicate greater symptom severity. The PCL-5 has demonstrated strong convergent validity, discriminant validity, and adequate inter-item correlation (Blevins, Weathers, Davis, Witte, & Domino, 2015). In our sample, inter-item correlation ($\alpha = .88$) and reliability results for the PCL-5 ($MR = .91$, $\omega = .88$) indicate a high proportion of shared variance that defines the underlying construct.

Quality of Life Satisfaction Questionnaire – Short form (Q-LES-Q-SF)

The Q-LES-Q-SF ($M = 42.10$, $SD = 16.13$) is a 16-item self-report measure examining quality of life and satisfaction (Endicott, Nee, Harrison, & Blumenthal, 1993). The measure is scored using a 5-point Likert scale (1 = very poor, 5 = very good) where higher scores suggest greater life satisfaction and quality of life. The Q-LES-Q-SF has demonstrated strong convergent validity, discriminant validity, and inter-item correlation (Riendeau et al., 2018). In our sample, inter-item correlation ($\alpha = .86$) and reliability

results for the Q-LES-Q-SF ($MR = .90$, $\omega = .84$) indicate a high proportion of shared variance that defines the underlying construct.

Acceptance and Action Questionnaire (AAQ-II)

The AAQ-II ($M = 39.49$, $SD = 6.99$) is a 7-item self-report measure of psychological inflexibility, or the attempt to alter the form, frequency, or situational sensitivity of negative thoughts and emotions (Bond et al., 2011). The measure is scored using a 7-point Likert scale (1 = never true, 7 = always true) where higher scores indicate elevated levels of psychological inflexibility. The AAQ-II has demonstrated convergent and discriminant validity, as well as adequate inter-item correlation (Gloster et al., 2011). In our sample, inter-item correlation ($\alpha = .89$) and reliability results for the AAQ-II ($MR = .90$, $\omega = .89$) indicate a high proportion of shared variance that defines the underlying construct.

Awareness, Courage, and Responding (ACR)

The “Courage” subscale ($M = 26.34$, $SD = 6.34$) of the ACR (ACR-C; Kanter et al., in press) is a 7-item measure of interpersonal courage, or willingness to be vulnerable in interpersonal interactions. The measure is scored using a 7-point Likert scale (1 = never true, 7 = always true) where higher scores indicate higher levels of interpersonal courage. Initial validation of the scale shows convergent validity, discriminant validity, and adequate inter-item correlation (Kanter et al., in press). In our sample, inter-item correlation ($\alpha = .84$) and reliability results for the ACR-C ($MR = .85$, $\omega = .84$) indicate a high proportion of shared variance that defines the underlying construct.

Valued Living Questionnaire (VLQ)

The VLQ is a two-part self-report measure of Valued Living and Committed Action assessed across 10 domains (Wilson et al., 2010). The measure is scored using a 10-point Likert scale (1 = not at all important, 10 = extremely important) where higher scores reflect greater valued living. The “Importance” scale asks the participant to rank 10 life domains based on the importance of each value, while the “Consistency” scale asks the participant to report how consistent their actions have been with those same values. A composite score ($M = 40.67$, $SD = 19.02$) is calculated by taking the mean of the products of corresponding importance and consistency scores for each domain. This score indicates total valued living.

Life Events Checklist (LEC-5)

The LEC-5 is a 17-item self-report measure that allows participants to indicate trauma exposure history (Weathers, Blake, et al., 2013). In addition to indicating exposure to an unspecified stressful event or experience, 16 events that have been shown to potentially result in the development of PTSD events (e.g., sexual assault, combat or exposure to a war-zone) are rated using 6 nominal responses (Happened to me; Witnessed it; Learned about it; Part of my job; Not sure; Doesn't apply). The LEC-5 is designed to be administered directly before the Clinician-Administered PTSD Scale for DSM-5 as the identifying mechanism for a person's most prominent criterion A traumatic experience.

Self-Compassion Scale (Short Form; SCS-SF)

The SCS-SF ($M = 26.33$, $SD = 7.38$) is a 12-item self-report measure assessing self-compassion, i.e., showing self-kindness vs. self-judgment, being mindful vs. fused

with thoughts/emotion, and connecting with others vs. isolating (Neff, 2003; Raes et al., 2011). Responses are given on a 5-point Likert-type scale (1 = almost never, 5 = almost always) where higher scores indicate elevated levels of self-compassion. Neff (2003) demonstrated that the SCS-SF has a “near-perfect correlation with the long form SCS” and has almost no loss in inter-item correlation for total scores. The SCS-SF has also demonstrated strong convergent and divergent validity (Castilho, Pinto-Gouveia, & Duarte, 2015). In our sample, inter-item correlation ($\alpha = .80$) and reliability results for the SCS-SF ($MR = .82$, $\omega = .80$) indicate a high proportion of shared variance that defines the underlying construct.

Trauma-related Shame Inventory (TRSI)

The TRSI ($M = 41.84$, $SD = 18.30$) is a 24-item self-report measure of internal and external trauma-related shame (Øktedalen, Hagtvet, Hoffart, Langkaas, & Smucker, 2014). The measure is scored using a 4-point Likert scale (0 = never true, 3 = always true) where higher scores indicate elevated levels of trauma-related shame. The TRSI has demonstrated convergent and discriminant validity, as well as adequate inter-item correlation (DeCou, Mahoney, Kaplan, & Lynch, 2019). In our sample, inter-item correlation ($\alpha = .96$) and reliability results for the PCL-5 ($MR = .96$, $\omega = .95$) indicate a high proportion of shared variance that defines the underlying construct.

Mini-International Neuropsychiatric Interview (MINI)

The MINI is a structured diagnostic interview developed by Sheehan et al. (1998) for DSM-IV. The MINI has been shown to have strong evidence in support of its validity (Lecrubier et al., 1997) and has been used widely as a diagnostic tool.

Data Analytic Strategy

Change in Symptom Measures Over Time

First, descriptive statistics were calculated to show symptom change over time (i.e., admission to discharge) on the variables under study. Reliable change indices (RCIs; Jacobson & Truax, 1991) were then calculated as function of mean admission scores, mean discharge scores, standard deviation at intake, and scale inter-item correlation, as measured by Cronbach's alpha. Alphas are presented in the Measures section. The resulting RCI provided an absolute cutoff value that allowed us to determine if change in mean scores from admission to discharge is more than could be accounted for by measurement error. If the difference between the RCI and the change in mean scores was more than 1.96, which established a 95% confidence interval, we determined that we observed reliable change in the variables under study (Jacobson & Truax, 1991).

Intent-to-treat Analysis

Next, an intent-to-treat analysis compared participants who dropped out of treatment to those who completed treatment. Logistic regression was used to compare participants who dropped out of treatment against participants who completed treatment with respect to PTSD symptoms, quality of life, self-compassion, interpersonal courage, valued living, trauma-related shame, and psychological inflexibility, as well as demographic variables including race, gender, marital status, and education level.

Discharge Model Confirmatory Factor Analysis

As a precursor to a discharge latent regression model (discussed in the next section), a Confirmatory Factor Analysis (CFA) was conducted. This model contained a 7-factor model for the PCL-5 (Grau, Garnier-Villarreal, & Wetterneck, 2019), a single

factor model for the Q-LES-Q-SF (Riendeau et al., 2018), a single factor model for the VLQ (Wilson et al., 2010), a 4-factor model for the TRSI (Øktedalen et al., 2014), a 2-factor model for the SCS-SF (Seligowski et al., 2015), a single factor model for the AAQ-II (Bond et al., 2011), and a single factor model for the ACR-C (Kanter et al., in press). The 7-factor model for the PCL-5 comprised reexperiencing, avoidance, negative alterations (in cognition and mood), anhedonia, externalization, dysphoria, and anxious arousal factors, which were theoretically derived from previous research on the latent factor structure of the PCL-5 (e.g., Armour et al., 2015; Grau et al., 2019). The 4-factor model for the TRSI comprised internal condemnation, external condemnation, internal affective/behavioral, and external affective/behavioral factors, which is the original factor structure proposed by the scale authors (Øktedalen et al., 2014). The 2-factor model for the SCS-SF comprised positive self-compassion and negative self-compassion factors, which have been tested as an appropriate underlying factor structure for the scale (Seligowski et al., 2015). Factor reliability was assessed using Omega and maximal reliability (Peters, 2014). Overall and local model fit were estimated via absolute and relative fit indices as well as an examination of factor loadings.

Discharge Latent Regression Model

After estimating the CFA, a latent regression model was estimated with PTSD symptom severity (as measured by the 7-factor model for the PCL-5), quality of life (as measured by the single factor model for the Q-LES-Q-SF), valued living (as measured by the single factor model for the VLQ), and trauma-related shame discharge scores (as measured by the 4-factor model for the TRSI) set as outcomes and self-compassion (as measured by the 2-factor model for the SCS-SF), psychological inflexibility (as measured

by the single factor model for the AAQ-II), and interpersonal courage (as measured by the single factor model for the ACR-C) admission scores set as predictors. Race and gender were included as demographic covariates.

Latent Growth Curve Models

Next, latent growth curve analysis was used to assess if the rate of change in designated predictor variables predicted the rate of change in designated outcome variables (Grimm, Ram, & Estabrook, 2016). This was achieved using a structural equation modeling (SEM) approach, which allows for non-linear change models (Grimm et al., 2016). In this study, our unit of time was one week, which reflected programmatic administration of assessment batteries. For measures that were only given at stepdown to intensive outpatient programming and discharge from the program, the time interval is approximately 6-8 weeks. In addition to linear growth models that estimated a change matrix for the variables under study, SEM allowed for the estimation of multiple forms of growth models (i.e., quadratic, cubic, and spline models) that could be tested against one another to ascertain the best-fitting growth model for each individual longitudinal measure. Quadratic models adjust the change matrix by adding an additional growth factor that squares the linear values from the original linear model. Cubic models adjust the change matrix by adding another growth factor to a quadratic model that cubes the linear values from the original linear model. Finally, spline growth models adjust the change matrix at knot points that represent theoretical or data-driven turning points between distinct, time-based linear slopes (Grimm et al., 2016).

The large number of latent regressions required for each dependent variable generated estimation problems for the full model, and as such, four separate models were

estimated to test each outcome variable separately (i.e., PTSD symptoms, valued living, quality of life, and trauma-related shame, respectively). All analyses were conducted in R 3.5.0 (R Core Team, 2017) using the *lavaan* (Rosseel, 2012) package. Latent growth curve analysis can be conducted with three timepoints, so it is an appropriate method of analysis for our data (Little, 2013). While it would be possible to assess longitudinal variable change using other statistical methods (e.g., longitudinal panel modeling), latent growth curve analysis allowed us to assess intraindividual growth trends (Little, 2013), which is the focus of the research questions in this study. In clinical settings, this intraindividual focus can help identify core processes of change that can become treatment targets in efficient, transdiagnostic approaches (Hayes & Hofmann, 2018).

Up to four separate growth curve models were estimated for each outcome variable. Linear, quadratic, cubic, and spline slopes were estimated and compared via Likelihood Ratio test (LRT, χ^2) model comparison to retain the best-fitting model to the data (Little, 2013). The AAQ-II, ACR-C, TRSI, and Q-LES-Q-SF contained three timepoints for all included participants. The PCL-5, SCS-SF, and VLQ were administered weekly and, as such, contained a variable range of timepoints. The largest number of timepoints available for analysis was 25. Model fit statistics, including absolute and relative fit approximate indices, were calculated for all estimated latent growth curves. In line with recommendations from Beauducel and Wittmann (2005), fit indices were not interpreted as strict cutoffs for model interpretation. Instead, multiple fit indices, including Gamma Hat, were estimated to provide an overall view of the fit of each model to the data (Cheung & Rensvold, 2002). For the linear models, the majority of models displayed poor relative fit indices. This may be a result of model complexity or

elements of misspecification; however, the Gamma Hat index, which is a more robust fit index than the TLI and CFI (Fan & Sivo, 2007), is above .90 for all models. Based on recommendations from Marsh, Hau, and Wen (2004), a Gamma Hat value above .90 suggests acceptable model fit.

Latent Growth Curve Regressions

From the estimated latent growth curve models, latent regressions were estimated to predict the intercept and slope of the PCL-5, Q-LES-Q-SF, VLQ, and TRSI, respectively. The intercepts and slopes of the AAQ-II, SCS-SF, and ACR-C were set as predictor variables in the latent regression models. In line with recommendations from Little (2013), pruned models, in which non-significant relationships are set to zero, were generated and compared to the initial models via LRT (χ^2). Model pruning continued until a non-significant chi-square result was found. Final, pruned model results are reported.

Missing Data

Missing data were handled using Full Information Maximum Likelihood, which increases power and reduces the chance for bias in parameter estimation due to missing data (Enders, 2010). Growth curves were calculated using all available data with at least two primary (i.e., Admission, Stepdown, and Discharge) timepoints. If a participant did not have at least two timepoints, they were considered to have dropped out of treatment. Because this was a secondary data analysis, a power analysis was not conducted, and all available and qualified subjects were included.

Chapter 4

Results

Results will be discussed first in terms of change in symptom measures over time, including a discussion of reliable change within the sample. This is followed by the results from the intent-to-treat analysis to assess predictors of dropout. Next, we will discuss the estimated discharge latent regression model and associated confirmatory factor analysis. Finally, full latent growth curve model analysis, as well as latent growth curve regression results, are presented.

Change in Symptom Measures Over Time

Additional descriptive statistics, symptom change from admission to discharge and reliable change indices (RCIs), were computed to display the effectiveness of treatment. Clinical or non-clinical cutoff scores have not been determined for the Q-LES-Q-SF, ACR-C, VLQ, or TRSI. Of the 343 patients who completed discharge PCL-5 assessments, 185 (54%) reported scores below 33, which is the cutoff score recommended for inferring a PTSD diagnosis by Blevins, Weathers, Davis, Witte, and Domino (2015). For the SCS-SF, an average level of self-compassion in a non-clinical population is a total score of 36 (Neff, 2003). In our sample, out of the 352 patients who completed discharge SCS-SF assessments, 186 (53%) reported scores at or above the non-clinical population average levels of self-compassion. Of the 274 patients who completed discharge AAQ-II assessments, 146 (47%) reported scores below 28, which is the recommended cutoff score for problematic psychological inflexibility in non-clinical samples (Ong, Pierce, Woods, Twohig, & Levin, 2019). 54% of treatment completers indicated sub-clinical PTSD symptoms, 53% of treatment completers indicated levels of self-compassion at or above the estimated non-clinical average, and 47% of treatment

completers indicated levels of psychological inflexibility below recommended non-clinical cutoff levels.

Importantly, treatment effectiveness cannot be measured by comparing discharge scores to non-clinical cutoff scores, as it is possible to achieve significant clinical change while still maintaining elevated symptom profiles as compared to non-clinical populations (Jacobson & Truax, 1991). To address this issue, RCIs were calculated for each variable under study, as the RCI has been shown to measure whether an individual's change is more than could be accounted for by measurement error (Jacobson & Truax, 1991). Reliable change was observed for the PCL-5, Q-LES-Q-SF, SCS-SF, TRSI, and the AAQ-II. We did not observe reliable change in the ACR-C. An RCI was not calculated for the VLQ, as reliability estimates were not calculated (see the Measures section for reference). Full results for RCIs are included in Table 3.

Table 3

Reliable Change Indices

	Admission Scores (SD)	Discharge Scores (SD)	Mean Difference	RCI	Reliable Change
PTSD Symptoms	54.17 (12.70)	32.43 (18.05)	21.74	17.33	Yes
Quality of Life	42.10 (16.14)	59.72 (18.86)	17.62	15.50	Yes
Self- Compassion	26.33 (7.38)	36.38 (9.85)	10.05	7.91	Yes
Interpersonal Courage	26.34 (6.34)	29.68 (5.66)	3.34	7.03	No

Trauma-related Shame	41.84 (18.30)	20.14 (17.43)	21.70	10.15	Yes
Psychological Inflexibility	39.49 (6.99)	27.05 (10.55)	12.44	6.43	Yes
Valued Living	40.66 (19.02)	50.56 (19.11)	9.90	N/A	N/A

Note. Admission scores are obtained prior to engagement in active treatment.

Intent-to-treat Analysis

The intent-to-treat analysis compared participants who dropped out of treatment with participants who completed treatment based on demographic variables (i.e., age, race, educational status) and baseline scores on the predictor and outcome variables under study. Of the 537 participants with baseline data, 170 were categorized as having dropped out of treatment. A logistic regression was conducted to predict the change in probability of dropout based on PTSD symptoms, quality of life, self-compassion, interpersonal courage, valued living, trauma-related shame, and psychological inflexibility.

Demographic variables, specifically race, gender, marital status, and education level were also entered into the model. No p values were below .05, and as such, we fail to reject the null hypothesis that dropout does not vary based on scores on the variables under study.

This result means that it is unlikely that any of the symptom measures or demographic variables predict the likelihood of completing treatment. Full results for symptom severity and demographic variables are included in Table 4.

Table 4

Logistic Regression for Dropout Based on Symptom Severity and Demographic Information

	Estimate (log odds)	SE	p value	CI 95%	Odds Ratio
Intercept	2.38	1.40	.09	-.33 – 5.15	10.86
PTSD Symptoms	-.00	.01	.78	-.03 - .02	.99
Quality of Life	.01	.01	.47	-.01, .02	1.01
Self-Compassion	-.03	.02	.14	-.07, .01	.97
Interpersonal Courage	-.02	.02	.37	-.06, .02	.98
Valued Living	.00	.01	.49	-.01, -.02	1.00
Trauma-related Shame	-.02	.01	.05	-.03, .00	.98
Psychological Inflexibility	-.01	.02	.78	-.06, .04	.99
Gender (Female)					
Male	.35	.33	.28	-.26, 1.02	1.42
Race (Black)					
White	.69	.42	.10	-.15, 1.52	1.99
Unknown	.96	.55	.08	-.11, 2.07	2.62
Other	.86	.74	.25	-.54, 2.40	2.36
Education (Some College)					
Bachelor's Degree	-.01	.36	.97	-.72, .71	.99
High School Degree	-.29	.44	.50	-1.14, .59	.75
Other	-.50	.30	.09	-1.09, .08	.61
Associate's Degree	.39	.61	.52	-.72, 1.71	1.48

Master's Degree	-.64	.47	.17	-1.56, .29	.52
Marital Status (Single)					
Married	-.15	.43	.74	-.49, .63	1.07
Divorced	.53	.41	.18	-.23, 1.37	1.70
Separated	.17	.56	.76	-.88, 1.37	1.19
Widowed	1.50	1.13	.18	-.35, 4.49	4.46
Unknown	-.26	.62	.67	-1.45, 1.02	.77

Discharge Model Confirmatory Factor Analysis

A CFA was estimated for the discharge latent regression model. This model contained a 7-factor model for the PCL-5 (Grau, Garnier-Villarreal, & Wetterneck, 2019), a single factor model for the Q-LES-Q-SF (Riendeau et al., 2018), a single factor model for the VLQ (Wilson et al., 2010), a 4-factor model for the TRSI (Øktedalen et al., 2014), a 2-factor model for the SCS-SF (Seligowski et al., 2015), a single factor model for the AAQ-II (Bond et al., 2011), and a single factor model for the ACR-C (Kanter et al., in press). The 7-factor model for the PCL-5 comprised reexperiencing, avoidance, negative alterations (in cognition and mood), anhedonia, externalization, dysphoria, and anxious arousal factors.

Based on a test of modification indices that was informed by scale theory, five residual correlations were included in the model. For the residual correlations, two were found between TRSI external condemnation items, two were found between psychological inflexibility items and one was found between valued living items. For the AAQ-II, the first residual correlation was between “My painful experiences and

memories make it difficult for me to live a life that I would value” and “My painful memories prevent me from having a fulfilling life” ($r = .38, p < .001$). The second residual correlation was between “I’m afraid of my feelings” and “I worry about not being able to control my worries and feelings” ($r = .88, p < .001$). For the TRSI, the first residual correlation was between “If others knew what happened to me, they would not like me” and “If others knew what happened to me, they would find me unacceptable” ($r = .42, p < .001$). The second residual correlation was between “I am ashamed of the way I behaved during my traumatic experience” and “If others knew how I behaved during my traumatic experience, they would be ashamed of me” ($r = .38, p < .001$). For the VLQ, the residual correlation was between “Friends / social life” and “Recreation / fun” ($r = .48, p < .001$).

Model fit for the discharge latent regression model was acceptable ($\chi^2 (2941) = 5323.06$, RMSEA = .04 CI[.04, .04], CFI = .89, TLI = .88, Gamma Hat = .90). For the PCL-5, the factor loadings for the 7-factor solution were strong, overall. Standardized factor loadings ranged from .76 to .90 for the Re-experiencing factor, .86 to .92 for the Avoidance factor, .82 to .90 for the Negative Alterations factor, .87 to .89 for the Anhedonia factor, .57 to .83 for the Externalizing Behaviors factor, .65 to .77 for the Dysphoric Arousal factor, and .83 to .93 for the Anxious Arousal factor. R^2 values have a mean of .70, and a range of .42 to .86, which indicates the shared item variance for the respective factor is high. Full reliability results for the CFA are included in Table 5.

Table 5

CFA Reliability Estimates

	Omega	AVE	MR
PCL-5 Model			
Re-Experiencing	.90	.73	.91
Avoidance	.88	.79	.88
Negative Alterations	.89	.73	.89
Anhedonia	.90	.78	.91
Externalizing Behaviors	.70	.56	.70
Dysphoric Arousal	.67	.51	.67
Anxious Arousal	.86	.77	.86
Factor Model	.96	.71	.97
Q-LES-Q-SF			
Factor Model	.90	.53	.90
VLQ			
Factor Model	.85	.47	.86
TRSI			
Internal Condemnation	.89	.64	.91
Internal Affective-behavioral	.89	.64	.90
External Condemnation	.91	.66	.92
External Affective-behavioral	.92	.65	.92
Factor Model	.95	.64	.96
SCS-SF			
Positive	.76	.36	.77
Negative	.75	.39	.75

Factor Model

AAQ-II

Factor Model	.88	.56	.88
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ACR-C

Factor Model	.84	.52	.84
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Note. MR = Maximal Reliability. AVE = Average Variance Extracted.

Based on an upper reliability threshold (represented by maximal reliability) and a lower reliability threshold (represented by Omega), for the PCL-5, the “Dysphoric Arousal” factor appears to be the least precise construct measure with “Anhedonia” and “Re-experiencing” being the most precise construct measures. For the TRSI, the “Internal Affective-behavioral” factor appears to be the least precise construct measure with “External Affective-behavioral” being the most precise construct measure. It should be noted that these differences are extremely small and that all reliability measures for the TRSI are largely consistent. For the SCS-SF, the “Negative” factor appears to be a slightly less precise construct measure than the “Positive” factor. Overall, all reliability estimates are strong.

For the Q-LES-Q-SF single factor solution, standardized factor loadings ranged from .59 to .89. R^2 values have a mean of .55, and a range of .35 to .80, which indicates the shared item variance for the respective factor is high.

For the VLQ single factor solution, standardized factor loadings ranged from .60 to .73. R^2 values have a mean of .47, and a range of .37 to .53, which indicates the shared item variance for the respective factor is high.

For the TRSI, the factor loadings for the 4-factor solution were strong, overall. Standardized factor loadings ranged from .72 to .86 for the Internal Condemnation factor, .68 to .88 for the Internal Affective-behavioral factor, .63 to .89 for the External Condemnation factor, and .74 to .89 for the External Affective-behavioral factor. R^2 values have a mean of .62, and a range of .40 to .80, which indicates the shared item variance for the respective factors is high.

For the SCS-SF, the factor loadings for the 2-factor solution were strong, overall. Standardized factor loadings ranged from .60 to .67 for the Positive Self-compassion factor to .64 to .71 for the Internal Negative Self-compassion factor. R^2 values have a mean of .44, and a range of .36 to .51, which indicates the shared item variance for the respective factor is high.

For the AAQ-II single factor solution, standardized factor loadings ranged from .63 to .84. R^2 values have a mean of .56, and a range of .39 to .71, which indicates the shared item variance for the respective factor is high.

For the ARC-C single factor solution, standardized factor loadings ranged from .66 to .79. R^2 values have a mean of .52, and a range of .43 to .63, which indicates the shared item variance for the respective factor is high.

Discharge Latent Regression Model

PTSD symptom severity (i.e., PCL-5), quality of life (i.e., Q-LES-Q-SF), valued living (i.e., VLQ), and trauma-related shame (i.e., TRSI) discharge scores set as outcomes and self-compassion (i.e., SCS-SF), psychological inflexibility (i.e., AAQ-II), and interpersonal courage (i.e., ACR-C) admission scores set as predictors.

Discharge PTSD Symptoms

Discharge PTSD Reexperiencing Factor. Admission self-compassion, psychological inflexibility, and interpersonal courage explained 8% of the variance in the discharge PTSD reexperiencing factor ($R^2 = .08$). At $p < .001$, only psychological inflexibility ($\beta = .38$, $SE = .11$) was significantly related to the PTSD reexperiencing factor. As admission psychological inflexibility scores increased by one standard deviation, discharge PTSD reexperiencing symptoms increased by .37 standard deviations. Admission positive self-compassion ($\beta = .06$, $SE = .16$, $p = .577$), negative self-compassion ($\beta = .17$, $SE = .12$, $p = .152$), and interpersonal courage ($\beta = .10$, $SE = .09$, $p = .239$) were not related to discharge PTSD reexperiencing symptoms. Gender ($\beta = .07$, $SE = .15$, $p = .665$) and race ($\beta = -.04$, $SE = .02$, $p = .050$) were not significant predictors.

Discharge PTSD Avoidance Factor. Admission self-compassion, psychological inflexibility, and interpersonal courage explained 8% of the variance in the discharge PTSD avoidance factor ($R^2 = .06$). Only admission psychological inflexibility ($\beta = .32$, $SE = .10$, $p = .002$) was significantly related to discharge PTSD avoidance symptoms. As psychological inflexibility scores increased by one standard deviation, PTSD avoidance symptoms increased by .31 standard deviations. Admission positive self-compassion ($\beta = .07$, $SE = .12$, $p = .564$), negative self-compassion ($\beta = .15$, $SE = .12$, $p = .206$), and interpersonal courage ($\beta = .03$, $SE = .08$, $p = .741$) were not related to discharge PTSD avoidance symptoms. Gender ($\beta = .14$, $SE = .16$, $p = .378$) and race ($\beta = -.01$, $SE = .02$, $p = .52$) were not significant predictors.

Discharge PTSD Negative Alterations Factor. Admission self-compassion, psychological inflexibility, and interpersonal courage explained 7% of the variance in the

discharge PTSD negative alterations (in cognitions and mood) factor ($R^2 = .07$). Only admission psychological inflexibility ($\beta = .28, SE = .10, p = .006$) was significantly related to discharge PTSD negative alterations (in cognition and mood). As psychological inflexibility scores increased by one standard deviation, PTSD negative alterations (in cognitions and mood) increased by .27 standard deviations. Admission positive self-compassion ($\beta = .02, SE = .11, p = .849$), negative self-compassion ($\beta = -.01, SE = .11, p = .944$), and interpersonal courage ($\beta = .05, SE = .08, p = .540$) were not related to discharge PTSD negative alterations (in cognitions and mood). Gender ($\beta = -.04, SE = .15, p = .812$) and race ($\beta = -.01, SE = .02, p = .813$) were not significant predictors.

Discharge PTSD Anhedonia Factor. Admission self-compassion, psychological inflexibility, and interpersonal courage explained 8% of the variance in the discharge PTSD anhedonia factor ($R^2 = .08$). Only admission psychological inflexibility ($\beta = .36, SE = .10, p < .001$) was significantly related to discharge PTSD anhedonia. As admission psychological inflexibility scores increased by one standard deviation, discharge PTSD anhedonia increased by .35 standard deviations. Admission positive self-compassion ($\beta = -.01, SE = .11, p = .943$), negative self-compassion ($\beta = .11, SE = .12, p = .321$), and interpersonal courage ($\beta = .07, SE = .08, p = .432$) were not related to discharge PTSD anhedonia. Gender ($\beta = .29, SE = .16, p = .057$) and race ($\beta = -.02, SE = .02, p = .532$) were not significant predictors.

Discharge PTSD Externalization Factor. Admission self-compassion, psychological inflexibility, and interpersonal courage explained 3% of the variance in the discharge PTSD externalization factor ($R^2 = .03$). Admission psychological inflexibility ($\beta = .15, SE = .13, p = .195$), positive self-compassion ($\beta = .10, SE = .13, p = .965$),

negative self-compassion ($\beta = -.01$, $SE = .13$, $p = .885$), and interpersonal courage ($\beta = .11$, $SE = .09$, $p = .235$) were not related to discharge PTSD externalization. Gender ($\beta = .26$, $SE = .17$, $p = .130$) and race ($\beta = -.02$, $SE = .02$, $p = .532$) were not significant predictors.

Discharge PTSD Dysphoria Factor. Admission self-compassion, psychological inflexibility, and interpersonal courage explained 7% of the variance in the discharge PTSD dysphoria factor ($R^2 = .07$). Only admission psychological inflexibility ($\beta = .30$, $SE = .12$, $p = .012$) was significantly related to discharge PTSD dysphoria. As admission psychological inflexibility scores increased by one standard deviation, discharge PTSD dysphoria increased by .09 standard deviations. Admission positive self-compassion ($\beta = .09$, $SE = .13$, $p = .484$), negative self-compassion ($\beta = -.01$, $SE = .13$, $p = .963$), and interpersonal courage ($\beta = -.02$, $SE = .10$, $p = .873$) were not related to discharge PTSD dysphoria. Gender ($\beta = -.16$, $SE = .18$, $p = .364$) and race ($\beta = -.02$, $SE = .03$, $p = .471$) were not significant predictors.

Discharge PTSD Anxious Arousal Factor. Admission self-compassion, psychological inflexibility, and interpersonal courage explained 9% of the variance in the discharge PTSD anxious arousal factor ($R^2 = .09$). At $p < .001$, only psychological inflexibility ($\beta = .39$, $SE = .11$) was significantly related to the discharge PTSD anxious arousal factor. As admission psychological inflexibility scores increased by one standard deviation, discharge PTSD anxious arousal increased by .37 standard deviations. Admission positive self-compassion ($\beta = .11$, $SE = .12$, $p = .345$), negative self-compassion ($\beta = .07$, $SE = .12$, $p = .534$), and interpersonal courage ($\beta = .12$, $SE = .09$, p

= .181) were not related to discharge PTSD anxious arousal. Gender ($\beta = .27, SE = .16, p = .091$) and race ($\beta = -.00, SE = .02, p = .919$) were not significant predictors.

Discharge Quality of Life

Admission self-compassion, psychological inflexibility, and interpersonal courage explained 4% of the variance in discharge quality of life ($R^2 = .04$). Admission positive self-compassion ($\beta = .03, SE = .11, p = .793$), negative self-compassion ($\beta = .05, SE = .11, p = .649$), psychological inflexibility ($\beta = -.17, SE = .10, p = .085$), and interpersonal courage ($\beta = -.08, SE = .08, p = .354$) were not significant predictors of discharge quality of life. Gender ($\beta = .06, SE = .17, p = .961$) and race ($\beta = -.03, SE = .02, p = .287$) were not significant predictors.

Discharge Trauma-related Shame

Discharge Trauma-related Shame Internal Condemnation Factor. Admission self-compassion, psychological inflexibility, and interpersonal courage explained 7% of the variance in the discharge TRSI internal condemnation factor ($R^2 = .07$). Admission positive self-compassion ($\beta = .05, SE = .11, p = .694$), negative self-compassion ($\beta = -.15, SE = .11, p = .194$), psychological inflexibility ($\beta = .18, SE = .10, p = .069$), and interpersonal courage ($\beta = .02, SE = .08, p = .796$) were not significant predictors of discharge TRSI internal condemnation. Gender ($\beta = .01, SE = .15, p = .975$) and race ($\beta = -.04, SE = .02, p = .059$) were not significant predictors.

Discharge Trauma-related Shame Internal Affective-behavioral Factor.

Admission self-compassion, psychological inflexibility, and interpersonal courage explained 6% of the variance in the discharge TRSI internal affective-behavioral factor ($R^2 = .06$). Admission positive self-compassion ($\beta = .15, SE = .11, p = .201$), negative

self-compassion ($\beta = -.17, SE = .12, p = .144$), psychological inflexibility ($\beta = .19, SE = .10, p = .060$), and interpersonal courage ($\beta = -.01, SE = .08, p = .923$) were not significant predictors of discharge TRSI affective-behavioral shame. Gender ($\beta = -.01, SE = .15, p = .975$) and race ($\beta = -.03, SE = .02, p = .206$) were not significant predictors.

Discharge Trauma-related Shame External Condemnation Factor. Admission self-compassion, psychological inflexibility, and interpersonal courage explained 3% of the variance in the discharge TRSI external condemnation factor ($R^2 = .03$). Admission positive self-compassion ($\beta = .05, SE = .11, p = .659$), negative self-compassion ($\beta = -.06, SE = .11, p = .606$), psychological inflexibility ($\beta = .17, SE = .10, p = .103$), and interpersonal courage ($\beta = .02, SE = .08, p = .853$) were not significant predictors of discharge TRSI external condemnation. Gender ($\beta = .14, SE = .15, p = .346$) and race ($\beta = -.02, SE = .02, p = .429$) were not significant predictors.

Discharge Trauma-related Shame External Affective-behavioral Factor.

Admission self-compassion, psychological inflexibility, and interpersonal courage explained 4% of the variance in the discharge TRSI external affective-behavioral factor ($R^2 = .04$). Admission positive self-compassion ($\beta = .04, SE = .11, p = .718$), negative self-compassion ($\beta = -.13, SE = .11, p = .256$), psychological inflexibility ($\beta = .12, SE = .10, p = .242$), and interpersonal courage ($\beta = .00, SE = .08, p = .970$) were not significant predictors of discharge TRSI external affective-behavioral shame. Gender ($\beta = .10, SE = .15, p = .531$) and race ($\beta = -.01, SE = .02, p = .744$) were not significant predictors.

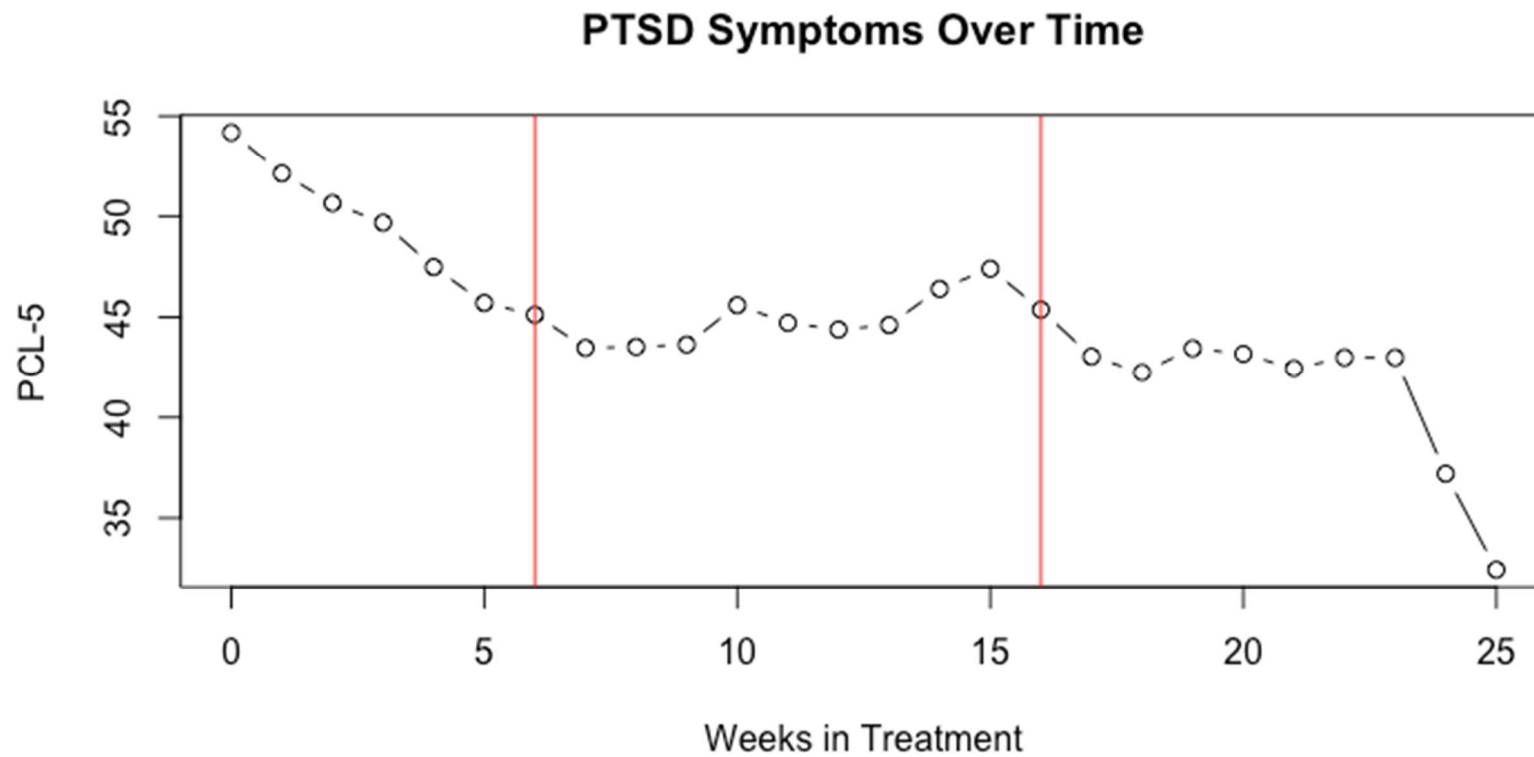
Discharge Valued Living

Admission self-compassion, psychological inflexibility, and interpersonal courage explained 14% of the variance in discharge valued living ($R^2 = .14$). Only admission

interpersonal courage ($\beta = .19, SE = .09, p = .032$) was significantly related to the discharge valued living. As admission interpersonal courage scores increased by one standard deviation, discharge PTSD anxious arousal increased by .18 standard deviations. Admission positive self-compassion ($\beta = .15, SE = .13, p = .224$), negative self-compassion ($\beta = .07, SE = .13, p = .578$), and psychological inflexibility ($\beta = -.13, SE = .10, p = .228$) were not related to discharge valued living. Gender ($\beta = -.01, SE = .17, p = .961$) and race ($\beta = -.03, SE = .02, p = .287$) were not significant predictors.

Latent Growth Curve Models

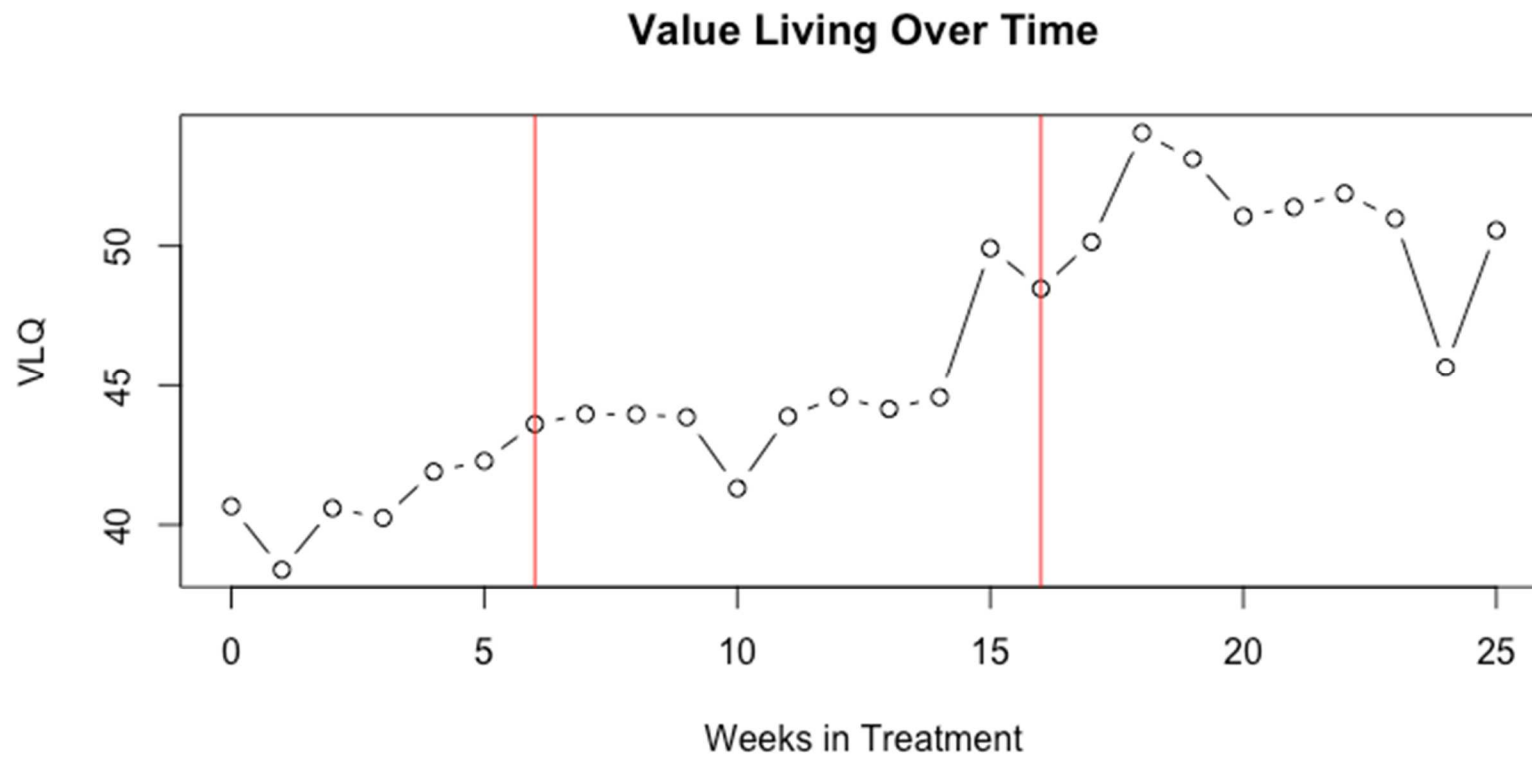
Latent growth curve models were estimated for PTSD symptom severity, self-compassion, valued living, psychological inflexibility, interpersonal courage, quality of life, and trauma-related shame. Plots of these growth curves are contained in Figures 1 through 7.

Figure 1*PCL-5 Means Over Time*

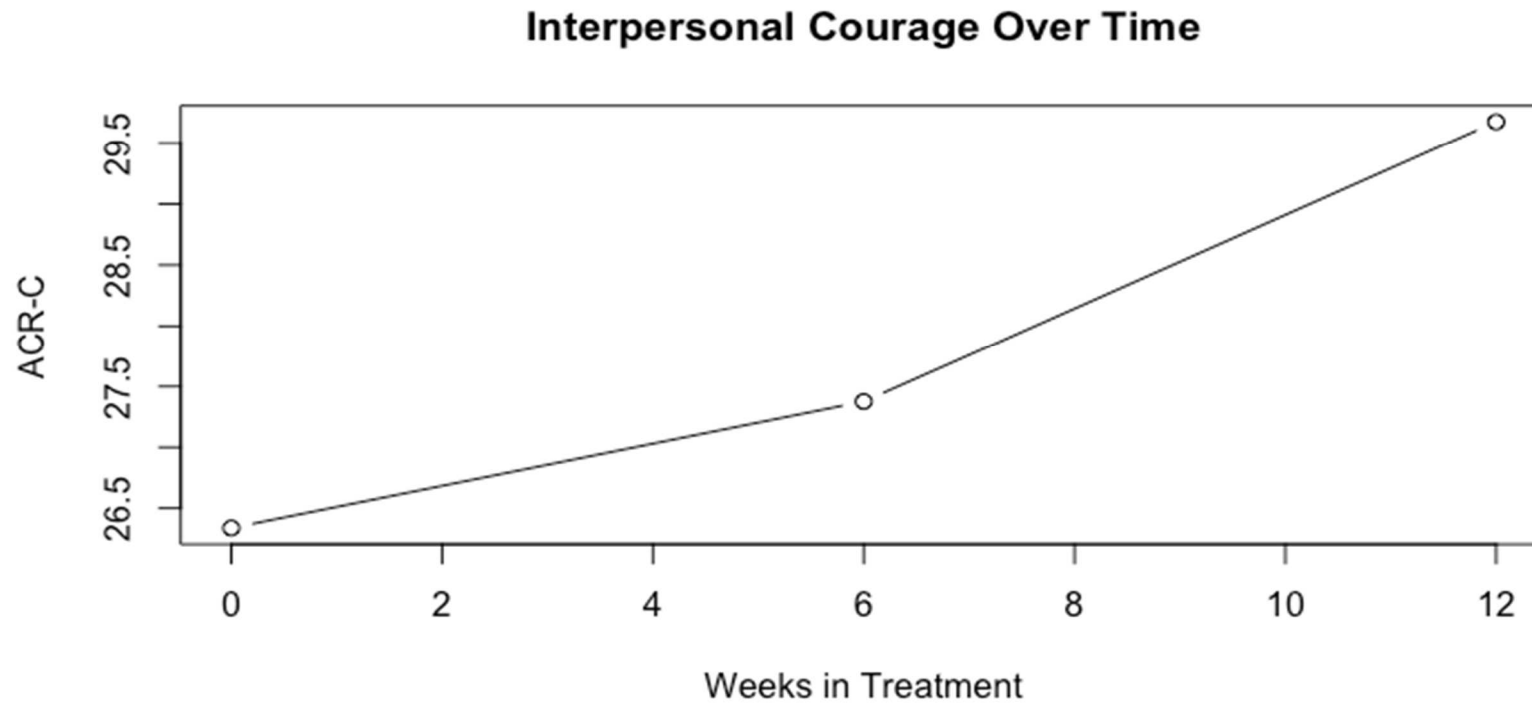
Note. Red lines indicate knot points that separate linear curves within the spline model.

Figure 2*SCS-SF Means Over Time*

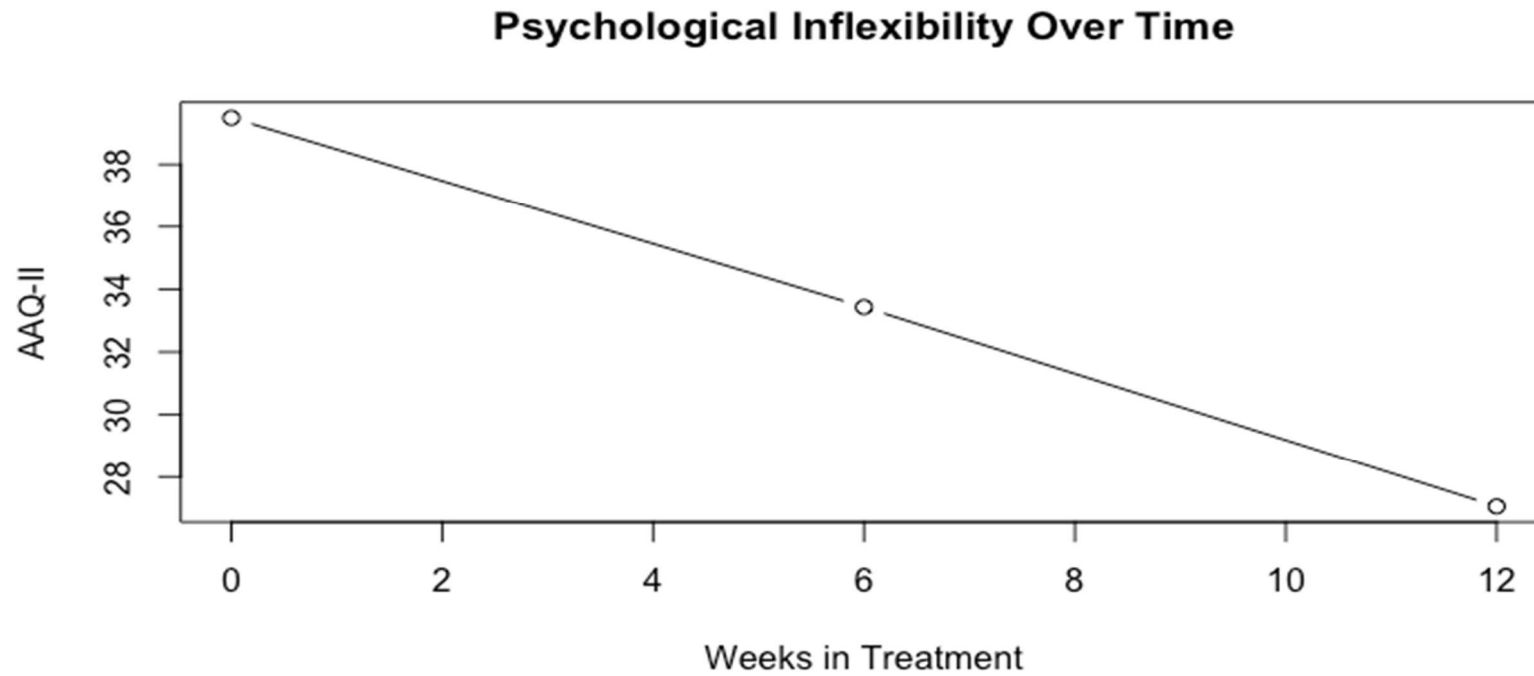
Note. Red lines indicate knot points that separate linear curves within the spline model.

Figure 3*VLQ Means Over Time*

Note. Red lines indicate knot points that separate linear curves within the spline model.

Figure 4*ACR-C Means Over Time*

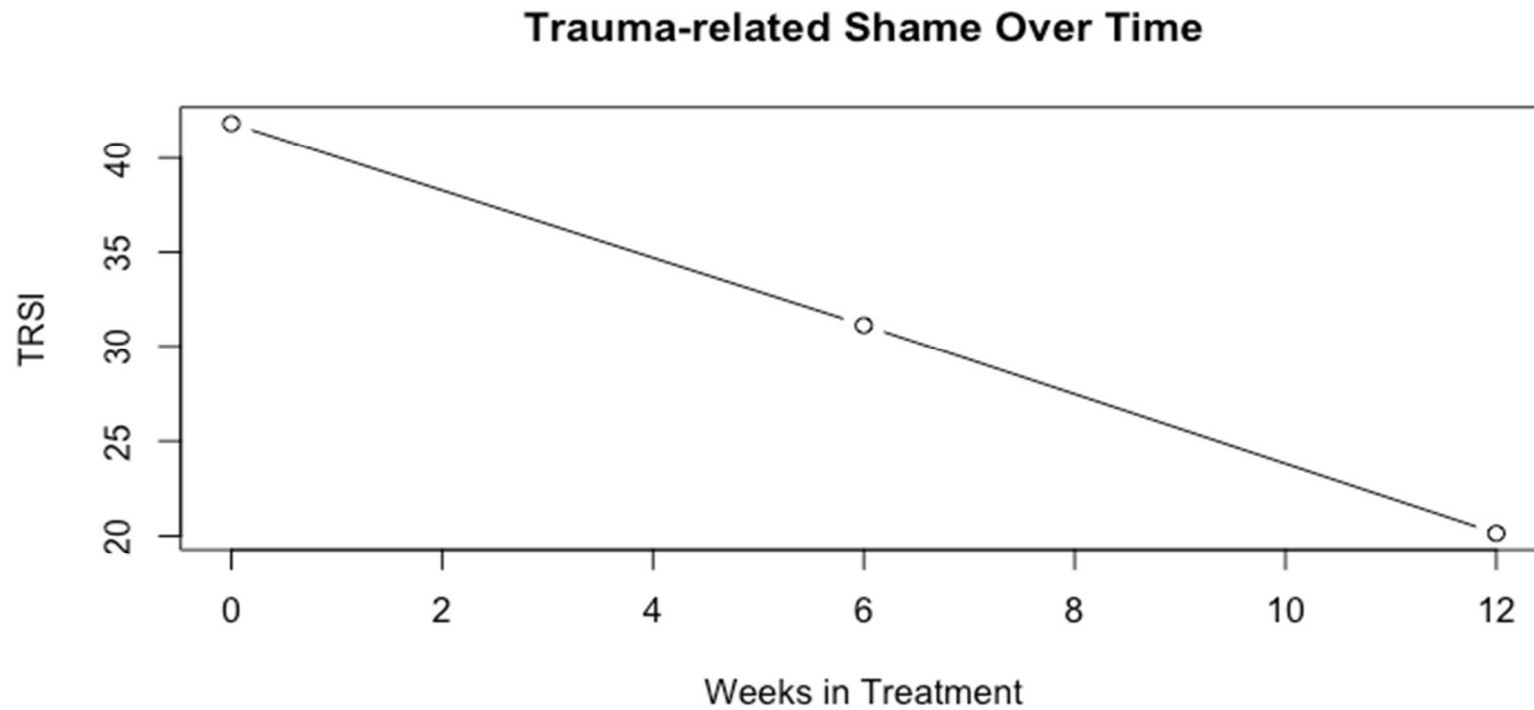
Note. 6 weeks represents the average time in partial hospitalization treatment. 6-12 weeks represents the average time spent in intensive outpatient treatment. These are not exact timeframes and vary between participants.

Figure 5*AAQ-II Means Over Time*

Note. 6 weeks represents the average time in partial hospitalization treatment. 6-12 weeks represents the average time spent in intensive outpatient treatment. These are not exact timeframes and vary between participants.

Figure 6*Q-LES-Q-SF Means Over Time*

Note. 6 weeks represents the average time in partial hospitalization treatment. 6-12 weeks represents the average time spent in intensive outpatient treatment. These are not exact timeframes and vary between participants.

Figure 7*TRSI Means Over Time*

Note. 6 weeks represents the average time in partial hospitalization treatment. 6-12 weeks represents the average time spent in intensive outpatient treatment. These are not exact timeframes and vary between participants.

PCL-5 Growth Model

First, linear, quadratic, and cubic slopes were estimated for the PCL-5. Only the linear slope model converged, preventing model comparison via LRT. Upon examination of plotted values, a three-piece spline model was estimated. Spline models are especially useful when each knot point is associated with a discernable reason for the change in slope (Grimm et al., 2016). For the PCL-5, knot points were discovered after week 6 of treatment and after week 16 of treatment. Week 6 is especially meaningful, as it represents a full course of partial hospitalization programming and the stepdown to intensive outpatient programming. Week 16 is approximately 4 weeks after the intended discharge date from intensive outpatient, though given the variable length of treatment for participants, this knot point represents data from patients who required additional treatment time to complete the program. This spline model was compared to the linear model via LRT ($\Delta X^2 = 659.33$, $\Delta df = 9$, $p < .001$), after which the spline model was retained as the best-fitting model to the data. Model fit for the PCL-5 spline model was also acceptable ($X^2(337) = 882.28$, RMSEA = .05 CI[.05, .06], CFI = .86, TLI = .86, Gamma Hat = .93). The estimated PCL-5 intercept (at admission) was 54.39 ($SE = .56$, $p < .001$). The first two linear slopes were significant at alpha level $p < .001$. However, the third linear slope was not significant ($p = .068$). For the first linear slope, on average, PCL-5 scores decreased 2.01 units ($SE = .15$, $p < .001$) per week. For the second linear slope, on average, PCL-5 scores decreased 1.34 units ($SE = .20$, $p < .001$) per week. Finally, for the third linear slope, PCL-5 scores increased .38 units ($SE = .21$, $p = .068$) per week.

SCS-SF Growth Model

First, linear, quadratic, and cubic slopes were estimated for the SCS-SF. Only the linear slope model and the quadratic slope model converged. These models were compared using LRT ($\Delta X^2 = 462.32$, $\Delta df = 4$, $p < .001$), after which the quadratic model was retained. However, upon examination of plotted values, a three-piece spline model appeared to be a better fit to the data and was therefore estimated. Mirroring results for the PCL-5, knot points were discovered after week 6 of treatment and after week 16 of treatment. This spline model was compared to the quadratic model via LRT ($\Delta X^2 = 140.21$, $\Delta df = 5$, $p < .001$), after which the spline model was retained as the best-fitting model to the data. Model fit for the SCS-SF spline model was acceptable ($X^2(337) = 944.12$, RMSEA = .05 CI[.05, .06], CFI = .86, TLI = .87, Gamma Hat = .92). The estimated SCS-SF intercept (at admission) was 26.13 ($SE = .35$, $p < .001$). The first two linear slopes were significant at alpha level $p < .001$. However, the third linear slope was not significant ($p = .580$). For the first linear slope, on average, SCS-SF scores increased .73 units ($SE = .08$) per week. For the second linear slope, on average, SCS-SF scores increased .46 units ($SE = .10$) per week. Finally, for the third linear slope, SCS-SF scores increased .06 units ($SE = .10$) per week.

VLQ Growth Model

First, linear, quadratic, and cubic slopes were estimated for the VLQ. Only the linear slope model and the quadratic slope model converged. These models were compared using LRT ($\Delta X^2 = 215.97$, $\Delta df = 4$, $p < .001$), after which the quadratic model was retained. However, upon examination of plotted values, a three-piece spline model appeared to be a better fit to the data and was therefore estimated. Mirroring results for

the VLQ and SCS-SF, knot points were discovered after Week 6 of treatment and after Week 16 of treatment. This spline model was compared to the quadratic model via LRT ($\Delta X^2 = 462.32$, $\Delta df = 4$, $p < .001$), after which the spline model was retained as the best-fitting model to the data. model fit for the VLQ spline model was also acceptable ($X^2(337) = 942.56$, RMSEA = .06 CI[.06, .07], CFI = .86, TLI = .87, Gamma Hat = .91). The estimated VLQ intercept (at admission) was 38.62 ($SE = .17$, $p < .001$). The first two linear slopes were significant at alpha level $p < .05$. However, the third linear slope was not significant ($p = .589$). For the first linear slope, on average, VLQ scores increased .85 units ($SE = .17$) per week. For the second linear slope, on average, VLQ scores increased .53 units ($SE = .15$) per week. Finally, for the third linear slope, VLQ scores increased .09 units ($SE = .16$) per week.

AAQ-II Growth Model

Linear, quadratic, and cubic slopes were estimated for the AAQ-II. Only the linear model converged, which prevented model comparison via LRT. Based on recommendations from Marsh, Hau, and Wen (2004), model fit for the AAQ-II linear model was acceptable ($X^2(475) = 35.88$, RMSEA = .27 CI[.20, .35], CFI = .71, TLI = .13, Gamma Hat = .95). Scores decreased consistently from admission to discharge. The estimated AAQ-II intercept (at admission) was 25.73 ($SE = 5.96$, $p < .001$). The linear slope was significant at alpha level $p < .001$. On average, AAQ-II scores decreased .36 units ($SE = .06$) per six weeks.

ACR-C Growth Model

Linear, quadratic, and cubic slopes were estimated for the ACR-C. Only the linear

model converged, which prevented model comparison via LRT. Based on recommendations from Marsh, Hau, and Wen (2004), model fit for the ACR-C linear model was acceptable ($\chi^2 (484) = 2.97$, RMSEA = .06 CI[.00, .15], CFI = .99, TLI = .98, Gamma Hat = .99). Scores increased gradually from admission to discharge. The estimated ACR-C intercept (at admission) was 21.99 ($SE = 2.94$, $p < .001$). The linear slope was not significant ($p = .918$). On average, ACR-C scores increased .002 units ($SE = .02$, $p < .001$) per six weeks.

TRSI Growth Model

Linear, quadratic, and cubic slopes were estimated for the TRSI. Only the linear model converged, which prevented model comparison via LRT. Based on recommendations from Marsh, Hau, and Wen (2004), model fit for the TRSI linear model was acceptable ($\chi^2 (515) = 23.21$, RMSEA = .21 CI[.14, .29], CFI = .87, TLI = .62, Gamma Hat = .97). Scores decreased gradually from admission to discharge. The estimated TRSI intercept (at admission) was 41.01 ($SE = .85$, $p < .001$). The linear slope was significant at alpha level $p < .001$. On average, TRSI scores decreased .77 units ($SE = .04$, $p < .001$) per six weeks.

Q-LES-Q-SF Growth Model

Linear, quadratic, and cubic slopes were estimated for the AAQ-II. Only the linear model converged, which prevented model comparison via LRT. Based on recommendations from Marsh, Hau, and Wen (2004), model fit for the Q-LES-Q-SF linear model was acceptable ($\chi^2 (512) = 23.68$, RMSEA = .21 CI[.14, .29], CFI = .92, TLI = .75, Gamma Hat = .97). Scores increased gradually from admission to discharge. The estimated Q-LES-Q-SF intercept (at admission) was 42.62 ($SE = .73$, $p < .001$). The

linear slope was significant at alpha level $p < .001$. On average, Q-LES-Q-SF scores increased .62 ($SE = .18$) units per six weeks.

Latent Growth Curve Regressions

After calculating intercepts and slopes for each variable under study, latent regressions were estimated to predict the intercept and slope of the PCL-5, Q-LES-Q-SF, VLQ, and TRSI, respectively. Per recommendations from Little (2013), because the tested models are theory-based and not exploratory, non-significant relationships were pruned, or set to zero, after which the pruned models were compared to previous models via LRT (χ^2). Model pruning continued until a non-significant chi-square result was found. Final, pruned model results are reported.

PCL-5 Latent Growth Curve Regression Model

For the first model, the PCL-5 intercept and the three PCL-5 linear slopes set as outcome variables. The SCS-SF intercept, the three SCS-SF linear slopes, the AAQ-II intercept, the AAQ linear slope, the ACR-C intercept, and the ARC-C linear slope were set as predictor variables.

PCL-5 Final Latent Growth Curve Regression Model. A pruned PCL-5 model was compared to the original model via LRT ($\Delta X^2 = 47.12$, $\Delta df = 14$, $p < .001$), after which the pruned model was retained. After model pruning, only the AAQ-II intercept ($\beta = 1.98$, $SE = .18$, $p < .001$) and the ACR-C intercept ($\beta = .36$, $SE = .15$, $p = .018$) were significant predictors of the PCL-5 intercept. Thus, we reject the null hypothesis of these intercept relationships being equal to zero. As the AAQ-II intercept increased by 1 standard deviation, the PCL-5 intercept increased by .97 deviations. As the ACR-C intercept increased by 1 standard deviation, the PCL-5 intercept increased by .16 standard

deviations. For the first PCL-5 linear slope, after model pruning, only the first SCS-SF linear slope ($\beta = -1.58$, $SE = .13$, $p < .001$) was a significant predictor. Thus, we reject the null hypothesis of this regression slope being equal to zero. As the first SCS-SF linear slope increased by 1 standard deviation, the first PCL-5 linear slope decreased by .91 standard deviations. For the second PCL-5 linear slope, after model pruning, only the second SCS-SF linear slope ($\beta = -1.60$, $SE = .21$, $p < .001$) was a significant predictor. Thus, we reject the null hypothesis of this regression slope being equal to zero. As the second SCS-SF linear slope increased by 1 standard deviation, the second PCL-5 linear slope decreased by .87 standard deviations. For the third PCL-5 linear slope, after model pruning, only the third SCS-SF linear slope ($\beta = -1.81$, $SE = .61$, $p = .003$) was a significant predictor. Thus, we reject the null hypothesis of this regression slope being equal to zero. As the third SCS-SF linear slope increased by 1 standard deviation, the third PCL-5 linear slope decreased by 1.02 standard deviations.

VLQ Latent Growth Curve Regression Model

For the second model, the VLQ intercept and the three VLQ linear slopes set as outcome variables. The SCS-SF intercept, the three SCS-SF linear slopes, the AAQ-II intercept, the AAQ linear slope, the ACR-C intercept, and the ARC-C linear slope were set as predictor variables.

VLQ Final Latent Growth Curve Regression Model. A pruned VLQ model was compared to the original model via LRT ($\Delta X^2 = 66.58$, $\Delta df = 20$, $p < .001$), after which the pruned model was retained. After model pruning, only the first SCS-SF linear slope ($\beta = 4.81$, $SE = 1.85$, $p = .009$) was a significant predictor of the VLQ intercept. Thus, we reject the null hypothesis of these intercept relationships being equal to zero. As

the first SCS-SF linear slope increased by 1 standard deviation, the VLQ intercept increased by .47 standard deviations.

For the first VLQ linear slope, after model pruning, the first SCS-SF slope ($\beta = .62, SE = .15, p < .001$), the AAQ-II intercept ($\beta = -.07, SE = .03, p = .026$), and the AAQ-II linear slope ($\beta = -1.49, SE = .60, p = .013$) were significant predictors. Thus, we reject the null hypothesis of these regression slopes being equal to zero. As the first SCS-SF linear slope increased by 1 standard deviation, the first VLQ linear slope increased by .41 units. As the AAQ-II intercept increased by 1 standard deviation, the first VLQ linear slope decreased by .18 standard deviations. As the AAQ-II linear slope increased by 1 standard deviation, the first VLQ linear slope decreased by .29 standard deviations. For the second VLQ linear slope, after model pruning, only the second SCS-SF linear slope ($\beta = .96, SE = .11, p < .001$) was a significant predictor. Thus, we reject the null hypothesis of this regression slope being equal to zero. As the second SCS-SF linear slope increased by 1 standard deviation, the second VLQ linear slope decreased by .78 standard deviations. For the third VLQ linear slope, after model pruning, only the third SCS-SF linear slope ($\beta = .96, SE = .12, p < .001$) was a significant predictor. Thus, we reject the null hypothesis of this regression slope being equal to zero. As the third SCS-SF linear slope increased by 1 standard deviation, the third VLQ linear slope increased by .79 standard deviations.

Q-LES-Q-SF Latent Growth Curve Regression Model

For the third model, the Q-LES-Q-SF intercept and the Q-LES-Q-SF linear slope were set as outcome variables. The SCS-SF intercept, the three SCS-SF linear slopes, the

AAQ-II intercept, the AAQ linear slope, the ACR-C intercept, and the ARC-C linear slope were set as predictor variables.

Q-LES-Q-SF Final Latent Growth Curve Regression Model. A pruned PCL-5 model was compared to the original model via LRT ($\Delta X^2 = 40.66$, $\Delta df = 10$, $p < .001$), after which the pruned model was retained. After model pruning, only the SCS-SF third linear slope ($\beta = 2.08$, $SE = 1.04$, $p = .045$) and the AAQ-II intercept ($\beta = -2.08$, $SE = .23$, $p < .001$) were significant predictors of the Q-LES-Q-SF intercept. Thus, we reject the null hypothesis of these intercept relationships being equal to zero. As the third SCS-SF linear slope increased by 1 standard deviation, the Q-LES-Q-SF intercept increased by .24 standard deviations. As the AAQ-II intercept increased by 1 standard deviation, the Q-LES-Q-SF intercept decreased by .93 standard deviations.

For the Q-LES-Q-SF linear slope, after model pruning, only the AAQ-II linear slope ($\beta = -1.00$, $SE = .20$, $p < .001$) was a significant predictor. Thus, we reject the null hypothesis of this regression slope being equal to zero. As the AAQ-II linear slope increased by 1 standard deviation, the Q-LES-Q-SF linear slope decreased by .62 standard deviations.

TRSI Latent Growth Curve Regression Model

For the fourth model, the TRSI intercept and the TRSI linear slope were set as outcome variables. The SCS-SF intercept, the three SCS-SF linear slopes, the AAQ-II intercept, the AAQ linear slope, the ACR-C intercept, and the ARC-C linear slope were set as predictor variables.

TRSI Final Latent Growth Curve Regression Model. A pruned PCL-5 model was compared to the original model via LRT ($\Delta X^2 = 14.67$, $\Delta df = 10$, $p = .145$), after

which the original model was retained. In the original model, the second SCS-SF linear slope ($\beta = -8.49$, $SE = 4.07$, $p = .037$), the third SCS-SF linear slope ($\beta = -9.39$, $SE = 4.15$, $p = .024$), and the AAQ-II intercept ($\beta = 2.10$, $SE = .20$, $p < .001$) were significant predictors of the TRSI intercept. Thus, we reject the null hypothesis of these intercept relationships being equal to zero. As the second SCS-SF linear slope increased by 1 standard deviation, the TRSI intercept decreased by .84 standard deviations. As the third SCS-SF linear slope increased by 1 standard deviation, the TRSI intercept decreased by .91 standard deviations. As the AAQ-II intercept increased by 1 standard deviation, the TRSI intercept increased by .74 standard deviations.

For the TRSI linear slope, only the SCS-SF intercept ($\beta = .01$, $SE = .01$, $p = .010$), AAQ-II linear slope ($\beta = .88$, $SE = .16$, $p < .001$) and the ACR-C linear slope ($\beta = -2.50$, $SE = .61$, $p < .001$) were significant predictors. Thus, we reject the null hypothesis of these regression slope being equal to zero. As the SCS-SF intercept increased by 1 standard deviation, the TRSI linear slope increased by .13 standard deviations. As the AAQ-II linear slope increased by 1 standard deviation, the TRSI linear slope increased by .40 standard deviations. As the ACR-C linear slope increased by 1 standard deviation, the TRSI linear slope decreased by .28 standard deviations. For reference, a full summary of hypothesis testing results can be found in Table 6.

Table 6

Hypothesis Testing Results

Hypotheses	Null Results (Reject or Fail to Reject the Null Hypothesis)	Summary of Results
H1 (PTSD Rate of Change Hypotheses)		
H1a (Self-Compassion)	Reject	1) First PCL-5 linear slope: The first SCS-SF linear slope ($\beta = -1.58, SE = .13, p < .001$) was a significant predictor. 2) Second PCL-5 linear slope: The second SCS-SF linear slope ($\beta = -1.60, SE = .21, p < .001$) was a significant predictor. 3) Third PCL-5 linear slope: The third SCS-SF linear slope ($\beta = -1.81, SE = .61, p = .003$) was a significant predictor
H1b (Psychological Inflexibility)	Fail to Reject	The AAQ-II linear slope was not a significant predictor of any PCL-5 linear slopes
H1c (Interpersonal Courage)	Fail to Reject	The ACR-C linear slope was not a significant predictor of any PCL-5 linear slopes
H2 (Quality of Life Rate of Change)		

Hypotheses)

H2a (Self-Compassion)	Fail to Reject	The SCS-SF linear slope was not a significant predictor of the Q-LES-Q linear slope
H2b (Psychological Inflexibility)	Reject	The AAQ-II linear slope was a significant predictor of the Q-LES-Q linear slope ($\beta = -1.00, SE = .20, p < .001$)
H2c (Interpersonal Courage)	Fail to Reject	The ACR-C linear slope was not a significant predictor of any PCL-5 linear slopes

H3 (Trauma-related Shame Rate of Change**Hypotheses)**

H3a (Self-Compassion)	Fail to Reject	The SCS-SF linear slope was not a significant predictor of the TRSI linear slope
H3b (Psychological Inflexibility)	Reject	The AAQ-II linear slope was a significant predictor of the TRSI linear slope ($\beta = .88, SE = .16, p < .001$)
H3c (Interpersonal Courage)	Reject	The ACR-C linear slope was a significant predictor of the TRSI linear slope ($\beta = -2.50, SE = .61, p < .001$)

H4 (Valued Living Rate of Change**Hypotheses)**

H4a (Self-Compassion)	Reject	1) First VLQ linear slope: The first SCS-SF linear slope ($\beta = .62, SE = .15, p < .001$) was a significant predictor.
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H4b (Psychological Inflexibility)	Partially Reject (1st linear slope only)	<ul style="list-style-type: none"> 2) Second VLQ linear slope: The second SCS-SF linear slope ($\beta = .96, SE = .11, p < .001$) was a significant predictor. 3) Third VLQ linear slope: The third SCS-SF linear slope ($\beta = .96, SE = .12, p < .001$) was a significant predictor 1) First VLQ linear slope: The AAQ-II linear slope ($\beta = -1.49, SE = .60, p = .013$) was a significant predictor. 2) Second VLQ linear slope: The AAQ-II linear slope was not a significant predictor. 3) Third VLQ linear slope: The AAQ-II linear slope was not a significant predictor.
H4c (Interpersonal Courage)	Fail to Reject	The ACR-C linear slope was not a significant predictor of any VLQ linear slopes
H5 (Treatment Completion Hypothesis)		Reject Dropout rates did not vary based on the variables under study or demographic variables.
H6 (Interpersonal Trauma vs. Non-Interpersonal Trauma)		Fail to reject Due to an extremely high rate of interpersonal (97%) vs. non-interpersonal (3%) trauma, comparative analyses between broad trauma categories was not feasible.
H7 (Predictor of Discharge Scores Hypotheses)		
H7a (Admission Psychological Inflexibility Predicts Discharge PTSD symptom severity)	Partially Reject	<ul style="list-style-type: none"> 1) Discharge PTSD Reexperiencing Factor: Admission psychological inflexibility ($\beta = .38, SE = .11, p < .001$) was a significant predictor. 2) Discharge PTSD Avoidance Factor: Admission psychological inflexibility ($\beta = .32, SE = .10, p = .002$) was a significant predictor.

H7b (Psychological Inflexibility Predicts Quality of Life)	Reject	<p>3) Discharge PTSD Negative Alterations Factor: Admission psychological inflexibility ($\beta = .28, SE = .10, p = .006$) was a significant predictor.</p> <p>4) Discharge PTSD Anhedonia Factor: Admission psychological inflexibility ($\beta = .36, SE = .10, p < .001$) was a significant predictor.</p> <p>5) Discharge PTSD Externalization Factor: Admission psychological inflexibility ($\beta = .15, SE = .13, p = .195$) not a significant predictor.</p> <p>6) Discharge PTSD Dysphoria Factor: Admission psychological inflexibility ($\beta = .30, SE = .12, p = .012$) was a significant predictor.</p> <p>7) Discharge PTSD Anxious Arousal Factor: Admission psychological inflexibility ($\beta = .39, SE = .11, p < .001$) was a significant predictor.</p> <p>Admission psychological inflexibility ($\beta = -.17, SE = .10, p = .085$) not a significant predictor of discharge quality of life.</p>
H7c (Self-Compassion Predicts Trauma-related Shame)	Fail to Reject	<p>1) Discharge Trauma-related Shame Internal Condemnation Factor: Admission positive self-compassion ($\beta = .05, SE = .11, p = .694$) and negative self-compassion ($\beta = -.15, SE = .11, p = .194$) were not significant predictors.</p> <p>2) Discharge Trauma-related Shame Internal Affective-behavioral Factor: Admission positive self-compassion ($\beta = .15, SE = .11, p = .201$) and negative self-compassion ($\beta = -.17, SE = .12, p = .144$) were not significant predictors.</p>

- 3) Discharge Trauma-related Shame External Condemnation Factor: Admission positive self-compassion ($\beta = .05, SE = .11, p = .659$) and negative self-compassion ($\beta = -.06, SE = .11, p = .606$) were not significant predictors.
- 4) Discharge Trauma-related Shame External Affective-behavioral Factor. Admission positive self-compassion ($\beta = .04, SE = .11, p = .718$) and negative self-compassion ($\beta = -.13, SE = .11, p = .256$) were not significant predictors.

Admission psychological inflexibility ($\beta = -.13, SE = .10, p = .228$) was not related to discharge valued living.

H7d (Psychological Inflexibility Predicts
Valued Living)

Reject

Chapter 5

Discussion

Summary of Study Aims, Symptom Change, and Dropout

As its main aim, this study explored how baseline scores and rates of change of self-compassion, psychological inflexibility, and interpersonal courage predicted discharge scores and rates of change of PTSD symptom severity, quality of life, trauma-related shame, and valued living in participants who sought treatment at an exposure-based partial hospitalization program for PTSD. Several additional aims were undertaken. First, descriptive statistics were estimated to demonstrate symptom change over time. These results were further defined via the use of RCIs, which allowed us to determine if change in mean scores from admission to discharge was more than could be accounted for by measurement error. Next, this study attempted to assess how treatment completion might vary depending on trauma type (interpersonal vs. non-interpersonal). Finally, an intent-to-treat analysis was conducted to compare participants who completed at least two timepoints with participants who dropped out of treatment before the second timepoint on the measures on interest and demographic variables.

A discharge latent regression model was estimated with a 7-factor model of discharge PTSD symptom severity, single factor model of discharge quality of life, single factor model of discharge valued living, and 4-factor model of discharge trauma-related shame discharge scores set as outcomes and a 2-factor model of admission self-compassion, single factor model of admission psychological inflexibility, and single factor model of admission interpersonal courage admission scores set as predictors. Race and gender were included in the model as covariates.

With respect to latent growth curve models, the sheer number of analyses, in addition to the variable treatment length between participants, necessitated the estimation of four separate latent growth curve models to predict each outcome variable (i.e., PCL-5, Q-LES-Q-SF, TRSI, VLQ). Within these models, the variables that were assessed on a weekly basis (i.e., SCS-SF, PCL-5, VLQ) were found to be best represented by a three-piece spline curve, which estimated three separate linear growth curves based on identified points at which the slope of the data's growth curve changed. Each of these models converged, which allowed for meaningful interpretation of model parameters. Unfortunately, due to an extremely high rate of interpersonal (97%) vs. non-interpersonal (3%) trauma, comparative analyses between broad trauma categories was not feasible. Subset analysis of trauma exposure (e.g., rape vs. physical assault) was also not possible due to participants experiencing a vast and varied trauma exposure history. As such, it is important to contextualize our findings as occurring within a population with wide-ranging trauma exposure, multiple comorbid conditions, and an almost universal experience of interpersonal trauma.

Before discussing latent regression and latent growth curve model results, it is important to acknowledge the overall effect of treatment on the variables under study. From admission to discharge, participants experienced reliable change in the expected directions on all but one (Interpersonal Courage) of the variables under study. That is to say, beyond what might be accounted for by measurement error, PTSD symptoms decreased, trauma-related shame decreased, psychological inflexibility decreased, quality of life increased, valued living increased, and self-compassion increased. This shows that this treatment program is, according to the measures administered in the program, effective for those who complete it. Additionally, significant percentages of patients who completed treatment showed levels of PTSD symptom

severity, psychological inflexibility, and self-compassion comparable to those found in non-clinical populations. For those who did not complete, the intent-to-treat analysis revealed small, yet statistically non-significant elevations in admission PTSD symptoms, trauma-related shame, and psychological inflexibility scores, as well as statistically non-significant deficits in self-compassion, quality of life, valued living, and interpersonal courage. While the direction of these results was expected, the non-significant result suggests it is inappropriate to draw broad conclusions about dropout from the program based on measure elevations or deficits.

Additionally, the intent-to-treat analysis found similar non-significant results with respect to demographic variables and dropout, which is likely due to the homogenous nature of the sample. A more statistically nuanced examination of dropout will likely be an important future study, though it is beyond the scope of the current project. However, based on the findings from this study, it appears that this exposure-based treatment program for PTSD (augmented with elements of ACT and DBT) is effective in reducing PTSD symptoms, trauma-related shame, and psychological inflexibility, while improving quality of life, valued living, and self-compassion.

Summary of Latent Regression and Latent Growth Curve Results

Latent Regression Results

The estimated latent growth curve showed adequate model fit, which allowed for further interpretation of parameters. Admission psychological inflexibility was the only significant predictor for the discharge PTSD reexperiencing, avoidance, negative alterations (in cognition and mood), anhedonia, dysphoria, and anxious arousal factors. None of the tested predictors were significantly related to the discharge PTSD externalization factor. As will be discussed in the discussion section focused on latent growth curves, these results do not suggest that psychological inflexibility is the only important variable with respect to PTSD treatment, but

they do highlight the relative weight of psychological inflexibility upon admission to the program. Outside of externalizing behaviors, a wide range of elevated PTSD symptom clusters were predicted by admission psychological inflexibility, which suggests it is an essential construct to measure and address early in treatment.

While the robust findings with respect to admission psychological inflexibility and discharge PTSD symptom clusters are important to note, these findings do not apply to discharge trauma-related shame, quality of life, or valued living. No discharge trauma-related shame factors were related to the predictor variables, which is not consistent with findings from the literature (e.g., Au et al., 2017). One potential explanation for these findings might be extrapolated from the high prevalence of alexithymia in PTSD (Twohig, 2009) and the tendency for those with high levels of shame to struggle to engage with emotionally-laden content (Lanius et al., 2015). For patients who experience these internal experiences, emotionally-laden questions, such as those focused on self-compassion and shame, may skew admission assessment results. As such, the importance of the predictor variables with respect to trauma-related shame may be better understood by examining rates of change over time, as engagement in PTSD treatment can help alleviate alexithymia and improve patients' abilities to engage with positive (e.g., self-compassion) and negative (e.g., shame) emotions (Lanius et al., 2015).

Finally, with respect to valued living, none of the predictor variables were significantly associated to valued living at discharge. While surprising at first glance, this finding is consistent with theoretical foundations of valued living. The concept of values-driven action may or may not be clear to patients upon entering treatment, though values clarification is a core component of treatment. For example, some patients may view entering treatment as a values-driven action, while other patients may have avoided considering their values as a function of their PTSD. For

these patients, an assessment focused on rating the importance of various values as well as the consistency with which they have pursued these values may result in a wide range of scores, depending on their interpretation and reaction to the questions. Values incongruence can be disturbing for patients (Wilson et al., 2010), which can then be discussed and addressed early in treatment. As such, mirroring the discussion concerning trauma-related shame, valued living is likely to grow and change over time, which might account for our null findings.

Latent Growth Curve Results

Four latent growth models were estimated to predict the within-person rate of change in PCL-5 scores (PTSD symptom severity), Q-LES-Q-SF scores (quality of life), TRSI scores (trauma-related shame), and VLQ scores (valued living). For each model, the intercepts and slopes of the SCS-SF (self-compassion), AAQ-II (psychological inflexibility), and ACR-C (interpersonal courage) were set as predictors. For the PCL-5, SCS-SF, and VLQ, three-piece splines models were estimated and retained after LRT comparisons to linear, quadratic, and cubic models. For all three spline models, the first two linear slopes were significant, yet the third linear slope was not. For the AAQ-II, ACR-C, Q-LES-Q-SF, and TRSI, linear models were retained after LRT comparisons to quadratic and cubic models.

With respect to the latent growth models, contrary to our hypotheses, only the within-person rate of change (increase) in self-compassion predicted the within-person rate of change (decrease) in PTSD symptom severity. This finding was consistent for the entire spline model. In other words, the first linear slope of the SCS-SF predicted the first linear slope of the PCL-5, the second linear slope of the SCS-SF predicted the second linear slope of the PCL-5, and the third linear slope of the SCS-SF predicted the third linear slope of the PCL-5. For valued living, all self-compassion linear slopes (increase) predicted all valued living linear slopes (increase),

mirroring the results for PTSD symptom severity. Interestingly, the within-person rate of change for psychological inflexibility predicted the first, but not the second or third linear slope of the valued living spline model. In contrast, only the within-person rate of change (decrease) in psychological inflexibility predicted the within-person rate of change (increase) in quality of life. Finally, for trauma-related shame, the within-person rates of change for both psychological inflexibility (increase) and interpersonal courage (decrease) predicted within-person rate of change over the course of treatment.

The growth curve results, especially from the three-piece spline models, provide additional information concerning the time frames during which patients achieved the most significant symptom changes. For PTSD symptom severity, valued living, and self-compassion, the greatest symptom change occurred during the first six weeks of treatment. For valued living and self-compassion, the first week to two weeks of treatment resulted in little to no improvements, though these trends reversed dramatically in the next three to four weeks of programming. Treatment results from weeks 6 through 16 showed consistent, yet much more gradual symptom change on all three measures.

Because the third linear slope within each spline model was non-significant, it is difficult to draw any conclusions concerning these results. This is likely because the typical length of stay in the program is approximately 12 weeks, and sample size decreases when approaching week 16. However, an examination of plotted values allows for some tentative observations. After week 16, PTSD symptoms appeared to continue declining gradually, but self-compassion and valued living both increased at a steeper rate than was seen between week 6 and week 16. On the whole, while it is difficult to draw conclusions concerning the reasons behind these trends, it is possible that, with respect to PTSD symptoms, a high dose of treatment engagement (i.e., 6-7 hrs

of programming per day) leads to near-immediate benefit that continues over the entire course of partial hospitalization programming. In exposure-based therapy for PTSD, substantial early gains have been linked to improved treatment outcomes and engagement (Doane, Feeny, & Zoellner, 2010), which may help to explain our study's similar dropout rate to those found in other exposure-based studies, despite the exceptional symptom severity and disorder comorbidity represented in the sample. These early gains were then translated into gradual, consistent improvements in PTSD symptoms, self-compassion, and valued living over the rest of treatment. The late (after week 16) spikes in self-compassion and valued living are intriguing, as they do not coincide with a marked decrease in PTSD symptom reduction. One explanation for this pattern might be extrapolated from work by Schnurr and Lunney (2016), who found that the best predictor of quality of life improvements for people who engaged in exposure therapy for PTSD was the remission of the PTSD diagnosis. Based on these findings, as well as the reliable clinical change in PTSD symptoms and relatively large percentage of patients who discharged with PCL-5 scores below the diagnostic cutoff score of 33, it may be that the late gains in self-compassion and valued living are representative of patients defining themselves beyond the context of their trauma and beginning to translate treatment gains into their everyday lives. This underscores the need for follow-up (e.g., after 2 months and 6 months) research to assess how this treatment impacted patients' long-term wellbeing. It is important to note that these are tentative implications that cannot be tested within the scope of this project.

In addition to our findings concerning the rate of change in our sample, several broad findings deserve mention. The surprising robustness of the effect of self-compassion is an important result, as is the confirmed strength of the relationships between baseline psychological inflexibility and our outcome variables. While we expected self-compassion and psychological

inflexibility to be strongly related to our outcome variables, the lack of effect from interpersonal courage was somewhat surprising. While the research literature supports the importance of interpersonal courage and connection in healing from trauma, it may be that strengthening psychological flexibility and self-compassion plays a more prominent role in successful treatment. The finding that the within-person rate of change in interpersonal courage predicted the within-person rate of change in trauma-related shame is encouraging, as interventions focused on trauma-related shame have emphasized the importance of sharing seemingly “broken” or “wrong” parts of themselves with trusted others (Lee et al., 2001). It should be noted that these assertions are tentative because the scale used to collect these data is new and its use has not been evaluated extensively to date. It is difficult to draw such construct-based conclusions from a new scale, as additional research using the measure is needed before making strong conclusions.

Taken together, these findings suggest that psychological inflexibility, self-compassion, and interpersonal courage appear to be important in trauma-focused treatment, albeit in different ways. At the outset of treatment, psychological inflexibility provides valuable information concerning future treatment course with respect to symptom severity and pursuing a meaningful life defined beyond trauma. Over the course of treatment, self-compassion appears to play a prominent role in reducing PTSD symptom severity and pursuing valued living, while psychological inflexibility continues to be important with respect to change in quality of life and trauma-related shame. When working specifically with trauma-related shame, interpersonal courage also appears to play an important role. These results paint the picture of trauma treatment characterized by compassionate and flexible connections with the self and others while simultaneously engaging in evidence-based mechanisms of PTSD symptom reduction (i.e.,

exposure). In the future, it appears the definition of evidence-based trauma treatment may well expand to include constructs such as the ones under study in this paper.

Limitations

Although there are multiple strengths of this study, there are also several important limitations to discuss. The data analyzed in this study were obtained from a clinical treatment program, and as such, there was no control group. This limits our ability to make causal inferences about the impact of various treatment elements (e.g., PE vs. DBT vs. ACT) and to formally assess treatment fidelity or consistency across program sites. Another limitation related to the clinical setting is the near-exclusive use of self-report instruments without a measure of malingering, which may bias our findings. It should be noted that completing an evidence-based treatment is a strong recommendation for patients who may be pursuing a PTSD diagnosis for secondary gain, so our analytic approach of only including participants with at least two primary timepoints has been empirically shown to be a strong (yet, incomplete) safeguard against biased results (Hall & Hall, 2007; Marx et al., 2012). In terms of the generalizability of this study, our results were obtained in a PTSD partial hospitalization with largely White and female patients who indicated severe levels of symptom severity and comorbidity. As such, results may not be generalizable to PTSD treatment delivered in a weekly outpatient format, trauma-exposed samples with less severe symptomatology, or more demographically diverse samples.

In terms of analysis, while this study benefits from the use of longitudinal analysis using multiple timepoints, the asymmetry between the analyzed timepoints is an additional limitation. Ideally, all measures would contain the same number of timepoints to assess growth over the exact same amount of time. Because this study utilized data from a clinical sample, treatment duration varied between patients. Additionally, the combination of the spline models calculated

for weekly measures and the single linear models calculated for the non-weekly measures may have resulted in a loss of specificity in analysis. While this does not prevent us from drawing inferences from these data, future studies would likely benefit from confirming our results using both controlled studies and symmetrical data points for each growth curve.

Suggestions for Future Research, Practice, and Policy

The limitations of this study provide several concrete directions for future research. While our findings are encouraging, they should be replicated in more diverse samples. Future controlled replication attempts might also utilize component analyses to assess which elements of treatment (i.e., PE vs. DBT vs. ACT) significantly contribute to change in the variables under study. Statistically, future analyses would benefit from more consistency in data points available for growth curve analysis.

This study's implications for future practice are particularly intriguing. Because this study assessed rate of change over time using multiple timepoints, we are able to make several cautious assertions about implications for treatment. Given our findings concerning the importance of both self-compassion and psychological inflexibility in evidence-based trauma treatment, recommendations for future practice are partially in line with existing recommendations and partially novel. The finding that within-person rate of change in psychological inflexibility is a significant predictor of quality of life and trauma-related shame shows the importance of actively targeting this construct in PTSD treatment, especially when considering outcomes beyond symptom reduction. This is, in many ways, in line with current and evolving directions in evidence-based PTSD treatment. The inhibitory learning model of exposure (Craske, Treanor, Conway, Zbozinek, & Vervliet, 2014) has gained momentum in recent years as a method of maximizing exposure therapy across multiple disorders, including

PTSD (Lebois, Seligowski, Wolff, Hill, & Ressler, 2019). Several recommendations from the inhibitory learning model that overlap with the psychological flexibility model include expectancy violation, variability in exposure, contextual variation, and affect labeling. Our findings suggest the importance of not only measuring psychological inflexibility at the beginning of treatment, but also working to integrate flexibility into exposure-based treatment.

A relatively novel recommendation that can be gleaned from our findings is the integration of compassion-focused elements into evidence-based PTSD treatment. While other studies (e.g., Dahm et al., 2015; Hiraoka et al., 2015) have provided preliminary results suggesting the importance of self-compassion in PTSD treatment, this is, to our knowledge, the first study to have assessed the impact of change in self-compassion in a treatment-seeking PTSD population. Previous research from Grau, Larsen, Lancaster, Garnier-Villarreal, and Wetterneck (in press) has demonstrated the importance of cultivating an expansive sense of self in PTSD treatment, and the results from this study help to clarify that expansion. This study suggests that the process of developing a sense of self that is characterized by mindful awareness, a sense of connection with others, and compassionate engagement with difficult experiences is predictive of decreases in PTSD symptoms and increases in the pursuit of meaning and values. Traditionally, PTSD treatments do not explicitly target self-compassion (Au et al., 2017). Given the findings from this study, one way to increase the efficacy of PTSD treatment might be to build in exercises designed to cultivate compassion such as compassionate imagery or compassion-focused meditation (Gilbert, 2010).

On a broader scale, our results support the assertions from Cloitre (2015) and other researchers focused on complex and repeated interpersonal trauma: PTSD treatment should expand beyond a singular focus on symptom reduction. The inclusion of non-symptom-focused

measures in this study allowed for a more nuanced understanding of the processes of change over time in PTSD treatment. That we failed to reject several hypotheses demonstrates the limitations of the current research base for PTSD treatment, especially with respect to constructs that are not traditionally assessed for in PTSD trials (e.g., self-compassion, valued living). While we did find evidence in support of recent advances in exposure therapy, there is also a need to explore programmatic shifts to incorporate compassion-based interventions in trauma-focused treatment. Based on these results, policy makers might consider supporting funding for augmented PTSD treatments to further explore ways in which evidence-based PTSD interventions might be improved. Additionally, our results would not have been possible without the implementation of a thorough, measurement-based approach to clinical research. This is additional evidence in support of systematic collection of data in treatment programs. As was shown in this paper, clinical outcomes research presents a unique opportunity to provide novel information concerning change over time, and policymakers would do well to allocate support and funding to these types of research endeavors.

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