EXPLORING EVIDENCE OF A DISSOCIATIVE SUBTYPE IN PTSD: DIFFERENTIAL
SYMPTOM STRUCTURE AND TREATMENT EFFICACY FOR THOSE WHO DISSOCIATE

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Abstract

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The decision to include a dissociative subtype in the DSM-5 (APA, 2013) is based on three separate lines of research utilizing advanced statistics, brain imaging technology and randomized controlled treatment trials (Carlson, Dalenberg & McDade-Montez, 2012). The current study examined whether a dissociative subtype could be derived using multiple methodological approaches in a sample (N = 200) of individuals undergoing treatment for chronic PTSD. Results indicated that our sample was best represented by four latent classes, as opposed to three, making a subtype difficult to discern. Furthermore, these subtypes did not differ based on symptom structure. Finally, the relationship between dissociation and PTSD was not moderated by treatment type. These findings showed that symptoms of dissociation were not representative of a subtype in our sample. Implications for these findings are discussed in respect to their impact on diagnosis and treatment of PTSD.
Exploring Evidence of a Dissociative Subtype in PTSD: Differential Symptom Structure and Treatment Efficacy for those who Dissociate

Overview

At some point in their lifetime 55.7% to 89.6% of individuals in the U.S. will experience a trauma such as a motor vehicle accident, military combat, sexual assault, or physical assault (Breslau 1998; Kessler, Sonnega, Bromet, Hughes & Nelson, 1995; Resnick, Kilpatrick, Dansky, Saunders & Best, 1993). A majority of these individuals will experience symptoms of PTSD immediately after the trauma (Rothbaum, Foa, Riggs, Murdock & Walsh, 1992). While for most these symptoms will reduce over time, for some (6.8%-12.3%) they will persist, leading to the development of chronic PTSD (Kessler et al., 2005; Resnick et al., 1993). The symptom clusters of PTSD described in the Diagnostic and Statistics Manual, 4th edition (DSM-IV; APA, 2000) include symptoms of re-experiencing, avoidance/numbing, and hyperarousal. In the fifth edition of the DSM (DSM-5; APA, 2013), the symptom structure of PTSD has been altered to include four clusters: re-experiencing, avoidance, negative alterations in cognition and mood, and hyperarousal. The conceptualization of PTSD as an anxiety disorder has also changed in the DSM-5, which now includes PTSD in a separate category of mental disorders called “trauma and stressor related disorders;” a category that includes other maladaptive reactions to trauma and stress such as acute stress disorder (ASD) and adjustment disorders. This new conceptualization groups these disorders based on the common etiology of stressful or traumatic experiences as opposed to anxiety symptoms.

In the DSM-5 (APA, 2013), PTSD has also been revised to include a dissociative subtype. The inclusion of this subtype is designed to identify individuals with PTSD who experience increased dissociative symptoms such as depersonalization and derealization.
Rates of dissociation have been shown to be relatively high in PTSD compared to other psychological disorders (Bernstein et al., 1993). In fact, in their analysis of over 1,000 general psychiatric patients, Carlson et al. (1993) showed that rates of dissociation in PTSD, measured by the dissociative experiences scale (DES- Bernstein & Putnam, 1986), were comparable to some dissociative disorders; with multiple personality disorder being the only disorder with higher rates of dissociation than PTSD. A recent review (Carlson, Dalenberg, & McDade-Montez, 2012) provided additional support for a positive relationship between dissociation and PTSD, but the proposal of a dissociative subtype goes a step beyond this and suggests those with PTSD who dissociate demonstrate a different type of disorder, distinct from classic fear-based PTSD (Dalenberg, Glaser, & Alhasoon, 2012). The addition of a subtype, as opposed to simply adding dissociative symptoms to the PTSD diagnosis, has important implications in terms of how we understand and treat the disorder. For example, a dissociative subtype would suggest that dissociative symptoms are not present for everyone with PTSD and individuals with these symptoms might therefore need a different or more targeted treatment.

In order to validate the existence of a dissociative subtype, data must show that dissociation is related to PTSD and that this relationship is not simply a result of increased PTSD severity (Carlson et al., 2012). To this end, three lines of research have been cited to argue for the existence of a dissociative subtype. These study methods include: brain imaging (Lanius, Brand, Vermetten, Frewen & Spiegel, 2012), advanced statistical analysis (Wolf et al., 2012a; Wolf et al., 2012b; Steuwe, Lanius, & Frewen, 2012), and treatment outcome designs (Cloitre, Petkova, Wang, & Lu, 2012; Resick, Suvak, Johnides, Mitchell & Iverson, 2012). While each of these lines of research
provide at least preliminary support for a distinct dissociative subtype, these few findings are relatively recent and do not yet provide overwhelming support for a subtype. In order to better understand how dissociation should be conceptualized in PTSD, further research is needed that combines multiple analytic methods for identifying a subtype in one sample of individuals with PTSD.

The current study explored the relationship between dissociation and PTSD to attempt to identify a distinct dissociative subtype. The inclusion of a dissociative subtype in the DSM-5 has important implications for how clinicians and researchers view PTSD. If in fact dissociation represents a different type of disorder, it stands to reason that dissociative PTSD might require a different type of assessment and treatment. According to Dalenberg et al. (2012), in order to identify a dissociative subtype in PTSD, the following three criteria should be met. First, the definition of a subtype should be clear and reliably measurable. This is often achieved through statistical analyses such as latent profile analysis (LPA) which retrospectively defines a group after the group has been statistically derived. Second, the subtype should differ in PTSD structure, functional mechanism, or both. A structural difference would include evidence that PTSD symptom presentation is different for those who dissociate, while a mechanism difference would include evidence of differing neurobiological correlates. Third, the subtype should have a clinically meaningful effect, such as leading to differential treatment response. No studies to date have attempted to confirm each of these criteria in a single sample.

The research indicating that dissociation represents a subtype of disorder within PTSD is relatively novel and piecemeal. Furthermore, methodological concerns such as the subjective interpretation of LPA results as well as the small sample sizes and
correlational nature of brain imaging studies, demands that any support for a dissociative subtype incorporate multiple lines of analysis. The current study combined the methods utilized in previous research to explore the evidence of a dissociative subtype in a single sample of individuals diagnosed with PTSD. The goal of this research is to better understand how the phenomenon of dissociation fits into the diagnosis and treatment of PTSD.

**Defining Dissociation**

Dissociation is a broad term encompassing a number of alterations in thoughts, feelings and behaviors that are thought to relate to an inability to integrate objective experience into conscious awareness (Putnam, 1991). This phenomenon includes a number of interpersonal experiences related to loss of conscious awareness. These include feelings of depersonalization, such as out of body experiences; derealization, such as experiencing dream-like states; gaps in awareness of current or past events; and absorption, or “zoning out”, in which awareness of one’s current surroundings is reduced (Waller, Putnam, & Carlson, 1996). The DSM-5 includes a category for dissociative disorders which is comprised of Dissociative Identity Disorder, Dissociative Amnesia, and Depersonalization/Derealization Disorder. These disorders are grouped based on the shared definition of “a disruption of and/or discontinuity in the normal integration of consciousness, memory, identity, emotion, perception, body representation, motor control, and behavior” (APA, 2013, p. 291).

The many definitions of dissociation found in the literature and the DSM suggest that this phenomenon may lie on a continuum that includes both pathological and non-pathological experiences. One measure designed to capture this spectrum of dissociation
is the Dissociative Experiences Scale (DES; Bernstein & Putnam, 1986). The DES is a widely used measure of dissociation with items ranging from common experiences such as distractibility and inattention to extreme alterations in consciousness such as out of body experiences and dissociative flashbacks (Bernstein & Putnam, 1986). Research examining the underlying factor structure of the DES has been mixed with studies supporting two factor (Olsen, Clapp & Parra 2013), three factor (Carlson et al., 1991; Ruiz, Lilienfeld& Douglas, 2008) and four factor (Amdur&Liberzon, 1996) models. The DES has been shown to reliably differentiate pathological and non-pathological dissociation (Bernstein & Putnam, 1986; Waller et al., 1996). The DES has also been shown to predict PTSD diagnosis (Bremner et al., 1992; Waller & Ross, 1997), providing evidence that dissociation plays a role in the reaction to trauma and may need to be further addressed in the diagnostic criteria for PTSD. How to conceptualize dissociation within non-dissociative disorders is the central question in the research on a dissociative subtype within PTSD. It could be that dissociation is simply an additional symptom of PTSD or dissociation could reflect a distinct disorder with differing etiology.

Dissociation has been theorized to relate to trauma for over a century. As described in reviews of the theoretical history of dissociation (Bremner&Marmar, 2002; Van der Hart & Horst, 1989), Pierre Janet was the first to describe dissociation in his doctoral dissertation in 1889 as a reaction to trauma in which one is unable to incorporate the associated memories and feelings from an event into conscious awareness. This idea was echoed in Freud’s “Studies on Hysteria” (Breuer & Freud, 1895), in which it was proposed that dissociation was the result of repression of memories of a traumatic event by the ego in order to protect against their emotionally painful effects. Despite a long
history of theorizing dissociation as a reaction to trauma, the DSM-IV did not emphasize dissociation in its definition of PTSD (APA, 2000), including only two dissociative symptoms: amnesia related to the event and dissociative flashbacks. Trauma related disorders such as PTSD have been historically focused on reactions of fear and anxiety, but reviews of the literature show that symptoms of dissociation are common components of PTSD (Carlson et al., 2012; Gershuny & Thayer, 1999), with as many as 34% to 41% of individuals with PTSD showing pathological levels of dissociation (Bremner et al., 1992; Yehuda et al., 1996).

**Acute stress disorder and peritraumatic dissociation.** PTSD in the DSM-IV (APA, 2000) is categorized as an anxiety disorder and contains few dissociative symptoms. However, a different trauma related diagnosis, acute stress disorder (ASD), is thought of as a precursor to PTSD and includes multiple dissociative symptoms. The focus on dissociation in ASD is controversial (Harvey & Bryant, 2002) and begs the question of why the diagnosis of PTSD contains so few dissociative symptoms. If ASD is in fact a precursor to PTSD, why is dissociation not reflected in both disorders? The answer to this question depends on whether dissociation’s relationship to trauma related pathology changes over time.

ASD in the DSM-IV was characterized by symptoms equivalent to those found in PTSD as well as three out of four dissociative symptoms (numbing/detachment, reduced awareness, derealization, depersonalization, and amnesia). In the DSM-5, dissociative symptoms are not required for a diagnosis of ASD, but are still included as separate symptoms. The presence of dissociation within the diagnostic criteria of ASD has implications for the diagnosis of PTSD given the special relationship between these
disorders within the nosology. Specifically, ASD can only be diagnosed within one month of a trauma due to the fact that symptoms lasting longer than one month are automatically categorized as PTSD. Because PTSD cannot be diagnosed until one month following a trauma, ASD provides a clinical explanation for PTSD symptoms occurring early after a trauma. ASD has thus been criticized as a filler diagnosis that pathologizes normal reactions to trauma (e.g., Harvey & Bryant, 2002). Proponents of the diagnosis contend that in addition to explaining early pathology, ASD is predictive of the later development of PTSD (Koopman, Classen, & Spiegel, 1994). However, in their review, Harvey and Bryant (2002) point out that this effect is largely due to the PTSD symptoms of ASD and not the dissociative symptoms. Given that 94% of individuals meet criteria for PTSD immediately after a trauma, and nearly half of those individuals will recover (Rothbaum et al., 1992), ASD’s predictive ability based on early PTSD symptoms is not surprising, nor particularly informative. In a more recent review, Cardeña and Carlson (2011) critiqued proposed changes to ASD in the DSM-5, suggesting that ASD symptoms should more closely match those of PTSD, and thus be conceptualized as an acute presentation of the chronic disorder. The idea that ASD is simply an early presentation of PTSD brings into question the role of dissociation in both disorders. If dissociation is less emphasized in the diagnostic criteria for ASD (as it is in the DSM-5) should dissociation be less emphasized in the criteria for PTSD? Understanding the temporal nature of the development of dissociative symptoms after a trauma is essential to answering this question.

Dissociation in ASD is described as *peritraumatic dissociation*, or dissociation which occurs during and shortly after a trauma (APA, 2000). This presentation of
dissociation is thought to be more acute than dissociation in PTSD which by definition occurs at least one month after a trauma and has thus been described as persistent dissociation (Briere, Scott & Weathers, 2005). In one meta-analysis, peritraumatic dissociation was predictive of the later development of PTSD (Ozer, Best, Lipsey & Weiss, 2003). These meta-analytic findings are supported by etiological theories of dissociation that posit that dissociative experiences are a defense mechanism employed to avoid emotional distress during a traumatic experience (Bernstein & Putnam, 1986; Bremner, 1992; Spiegel, 1991; van der Kolk, van der Hart & Marmar, 1996; Vanderlinen, Van Dyck, Vandereycken, Vertommen & Verkes, 1993), and emotional processing theories which suggest that this type of early dissociation can lead to fragmented trauma memories and subsequent PTSD (Foa & Kozak, 1986; Foa, Huppert, Cahill, 2006). However, a meta-analysis of prospective studies showed that the effect of peritraumatic dissociation on PTSD loses statistical significance when controlling for other variables such as initial PTSD severity and other mental health problems (van der Velden & Wittmann, 2008). Furthermore, peritraumatic dissociation is frequently measured with retrospective self-reports, such as the widely used Peritraumatic Dissociative Experiences Questionnaire (PDEQ; Marmar, et al., 1997), which have been shown to fluctuate overtime (Zoellner, Sacks & Foa, 2001) and thus may not provide an accurate account of how dissociation is experienced during a trauma.

It may also be that persistent dissociation after the trauma is accounting for the predictive power of peritraumatic dissociation on the later development of PTSD. For example, peritraumatic dissociation has been shown to no longer relate to PTSD when accounting for the influence of persistent dissociation (Briere, et al., 2005). If persistent
dissociation is more important in predicting PTSD than peritraumatic dissociation, then the focus on peritraumatic dissociation in ASD should be reduced, as adopted in the DSM-5. However, the corresponding increased focus on dissociation in the DSM-5 PTSD diagnostic criteria is somewhat confusing. The inclusion of a dissociative subtype suggests a differing etiological mechanism for dissociative PTSD, but if peritraumatic dissociation is not independently predictive of PTSD, a differing etiological mechanism seems less likely. It may be instead that dissociation develops overtime just like other symptoms of PTSD and would be better conceptualized as additional symptoms and not a subtype.

**Dissociation and Chronic PTSD**

While it remains unclear how dissociation manifests early after a trauma and how this impacts the subsequent development of PTSD, it is clear that the tendency to dissociate is higher for those with PTSD compared to other Axis I disorders (Carlson et al., 2012). Furthermore, a high rate of trauma has been found among those with dissociative disorders, with some data suggesting as many as 98% of individuals with dissociative disorders have experienced childhood abuse (Carlson et al., 2001). These findings suggest that persistent dissociation is inherently linked to traumatic experiences.

The notion that traumatic experience is an etiological factor for both PTSD and dissociative disorders has led some to suggest that PTSD should be reclassified with dissociative disorders (Friedman, Resick, Bryant & Brewin, 2011). While the DSM-5 does not include PTSD and dissociative disorders in the same category, it does remove PTSD from the anxiety disorders into a new category called “trauma and stressor related disorders.” This move de-emphasizes the role of fear and anxiety in the nosology, thus
potentially emphasizing other etiological factors such as dissociation. Changing the classification of PTSD from an anxiety disorder to a “trauma and stressor related disorder” has been criticized as an unfounded step, lacking in strong empirical support (Zoellner, Rothbaum & Feeny, 2011).

In addition to prompting changes in the DSM-5, dissociative symptoms have also been cited in support of a new PTSD diagnosis proposed for the World Health Organization (WHO) International Classification of Diseases 11th revision (ICD-11). This diagnosis would be called complex PTSD (Herman, 1997), and would be included as a separate disorder from PTSD in the trauma and stressor related disorders category (Cloitre, Garvert, Brewin, Bryant, & Maercker, 2013). Complex PTSD is conceptualized as a trauma related disorder which in addition to PTSD symptomatology, is characterized by a number of interpersonal and emotion regulation difficulties ( Cloitre et al., 2009). These difficulties are divided into three domains: affective, which includes emotion dysregulation such as dissociation; negative self-concept, like feelings of worthlessness; and relational disturbances, like difficulties feeling close to others (Cloitre et al., 2013). Importantly, these additional symptoms are thought to most often be related to repeated traumatization occurring in childhood (Cloitre et al., 2009).

The increased focus on dissociation in PTSD in the DSM-5 may influence the focus on dissociation in trauma related disorders in the ICD-11 as these two manuals are closely aligned (Kupfer, Regier & Kuhl, 2008). Because complex PTSD is thought to develop from repeated trauma, and is characterized by symptoms such as dissociation, it is important for any study looking at dissociation in PTSD to examine past trauma experience as this could influence the presentation of dissociation in PTSD. While the
DSM-5 did not adopt the new disorder of complex PTSD, the inclusion of a dissociative subtype is in line with a move to emphasize peripheral symptoms, such as dissociation, in the diagnosis of PTSD. However, further research is needed to fully flesh out the relationship between these trauma reactions.

**Dissociative Subtype**

The relationship between PTSD and dissociation can be described in two ways. The first is a continuous relationship in which an increase in dissociation is linearly related to an increase in PTSD. This conceptualization has been labeled the *component model* (Dalenberg & Carlson, 2012) and reflects the idea that dissociation is an additional symptom cluster of PTSD that increases with PTSD severity. The second way to conceptualize this relationship has been labeled the *subtype model* (Dalenberg & Carlson, 2012). This model reflects the idea that individuals either dissociate or do not dissociate after a trauma, and those who do experience a different type of PTSD with distinct symptoms.

The inclusion of a dissociative subtype in the DSM-5 is in some ways a hybrid of these two models in that while dissociation is labeled as a subtype, diagnosing the subtype only requires additional dissociative symptoms, not a distinct change in PTSD presentation or severity. This approach is somewhat confusing given that recent reviews of the research supporting a subtype suggest that in addition to dissociative symptoms, there must also be a corresponding change in PTSD symptom presentation (Dalenberg & Carlson, 2012; Dalenberg et al., 2012; Lanius et al., 2012; Resick et al., 2012). The requirement that there be a corresponding change in PTSD symptomatology, in addition to dissociative symptoms, to evidence a subtype acts as an empirical hurdle which may
prevent the proliferation of unnecessary subtypes throughout the nosology. For example, if a dissociative subtype is nothing more than the addition of dissociative symptoms, could there not also be subtypes representing other associated symptoms of PTSD such as rumination, anger, guilt, or even reckless behavior?

In order to maintain empirical rigor for supporting a subtype, Dalenberg et al. (2012) proposed three categories of evidence which should be met when considering a subtype. First, the criteria for a subtype should be clearly defined and reliably measurable (definitional requirement). Second, the subtype should show a differing symptom structure (structural requirement) or functional mechanism (mechanism requirement) of PTSD. Third, the subtype should be clinically meaningful (clinical requirement). In the case of showing evidence for a dissociative subtype in PTSD, three lines of research have been conducted. 1) research using statistical analysis, such as latent profile analysis (LPA), offers evidence to support both the definitional and structural requirement (Steuwe, Lanius & Frewin, 2012; Wolf et al., 2012a; Wolf et al., 2012b); 2) brain imaging studies offer evidence to support the mechanism requirement (Lanius et al., 2010; Lanius et al., 2012); and 3) treatment outcome studies offer evidence to support the clinical requirement.

**Statistical evidence for a subtype.** Studies utilizing advanced statistical analysis have been referenced to support both the definitional and structural requirements for evidencing a subtype. In the case of dissociation, the definitional requirement is especially important because dissociation is measured differently throughout the literature. For example, some studies looking at statistical evidence for a subtype use the clinician administered PTSD scale (CAPS; Blake et al., 1995) which narrowly defines
dissociation as depersonalization, derealization, and reduced awareness (Steuwe et al., 2012; Wolf et al., 2012a; Wolf et al., 2012b). However, other studies have focused on statistically extrapolating a subtype from the DES which measures dissociation more broadly (Waelde, Silvern & Fairbank, 2005; Waller et al., 1996; Waller & Ross, 1997).

Taxometric statistical analyses have been applied to the DES and reveal that the scale can be used to measure non-pathological, trait-like dissociation as well as pathological dissociation, indicative of dissociative disorders. Specifically, pathological dissociation was reliably measured in a sample of individuals with dissociative disorders using 8 items from the DES. The authors labeled this eight item measure the DES-Taxon or DES-T (Waller, et al., 1996). Taxometric methods have also been used to identify a latent class of dissociation in samples of individuals with PTSD (Waelde et al., 2005). More recently, latent profile analysis (LPA) has been used to examine a dissociative subtype in PTSD. LPA first identifies latent classes and then defines those classes using post hoc analyses. Like taxometric methods, this approach allows for a priori grouping of individuals. However, unlike taxometric methods, LPA is able to group individuals in multiple classes as opposed to only being able to identify data as dichotomous or continuous.

Three recent studies used LPA to identify and define a dissociative subtype in trauma exposed and PTSD samples (Steuwe et al., 2012; Wolf et al., 2012a; Wolf et al., 2012b). Wolf et al. (2012a) analyzed data from a sample of Veterans and their partners all of whom had suffered a trauma. This study was the only one of the three LPA studies to include individuals with and without PTSD. The authors determined that a three class solution was the best fit for the data, meaning that the sample could be divided into three
homogeneous groups that differed on PTSD and dissociation scores. Specifically, the first group (51%) was characterized by low PTSD and low dissociation scores, the second group (43%) was characterized by high PTSD and low dissociation scores and the third group (6%) was characterized by high PTSD and high dissociation scores. This third group was considered by the authors to be a distinct dissociative class. Importantly, these findings do not rule out the possibility that dissociation is simply a reflection of increased PTSD severity as opposed to a distinct type of disorder in that dissociation was only related to high levels of PTSD.

In a replication study, Wolf et al. (2012b) showed similar results to Wolf et al. (2012a); however, because their entire sample was diagnosed with PTSD, the first group was labeled “moderate” as opposed to “low” PTSD. Nonetheless, three distinct groups were still derived from the sample: moderate PTSD/low dissociation, high PTSD/low dissociation, and high PTSD/high dissociation. These findings showed that a three class solution could also be demonstrated in a sample made up entirely of individuals with PTSD. Steuwe et al. (2012) also found a three class solution to be the best fit for a sample of civilians with PTSD. Furthermore, they used confirmatory factor analysis to show that dissociative symptoms were best conceptualized as an additional symptom cluster of PTSD. In other words, dissociation was shown to be a construct distinct from symptoms of re-experiencing, avoidance, emotional numbing, and hyperarousal. While the authors cited this as evidence for a dissociative subtype, these findings could also support a component model in which dissociation is seen as an additional symptom cluster of PTSD. Furthermore, the fact that dissociation was shown to be best represented as an additional symptom cluster, should caution the interpretation of the
LPA as evidence for a dissociative subtype because the LPA essentially removed one of the symptom clusters (dissociation) from PTSD and entered it into the analysis as a separate construct. The resulting dissociative class might simply reflect how the symptoms were entered into the analysis. One could imagine a similar result occurring if avoidance symptoms, for example, were entered as a separate construct. This may result in the appearance of an avoidant latent class.

The presence of a latent class may also reflect an extreme measure of psychopathology as opposed to a truly distinct class of individuals. For example in these LPA studies, the measure of dissociation submitted to the LPA usually consisted of questions regarding depersonalization and derealization only. One study also included reduction in awareness (Wolf et al., 2012b). Depersonalization, derealization, and reduction in awareness are considered more pathological constructs of dissociation (Waller et al., 1996) meaning that it would be more likely that individuals who show high levels of these symptoms would be typologically distinct from other individuals in a sample. In order to reduce the possibility that a latent class is actually the result of a measure biased toward the extreme, it may be beneficial to measure dissociation more broadly in our LPA using a scale such as the DES.

It may also be that the dissociative class found in these studies reflects a transdiagnostic vulnerability to PTSD as opposed to a subtype. Any vulnerability to PTSD could potentially look like a distinct class if submitted to a LPA. For example, rumination, like dissociation, is a phenomenon found in a number of different disorders that negatively relates to PTSD severity (Michael, Halligan, Clark & Ehlers, 2007). Furthermore, rumination predicted PTSD independently of dissociation, indicating
potential divergent mechanisms (Murray, Ehlers & Mayou, 2002). Therefore, it is plausible that rumination could be considered an additional subtype in PTSD. Comparing a dissociative subtype to a ruminative subtype in PTSD would help to clarify the utility of including subtypes based on LPA and potentially caution the unnecessary proliferation of subtypes throughout mental health nosology. Due to some of the insufficiencies of LPA interpretation discussed here, calls for evidencing a dissociative subtype must draw on multiple lines of research. The other main body of research which has been used to support a dissociative subtype in PTSD involves brain imaging studies which suggest that individuals with PTSD who dissociate display opposing neurological mechanisms.

**Brain imaging evidence for a subtype.** Brain imaging studies are referenced to support the mechanism requirement for evidencing a dissociative subtype (Lanius et al., 2012). These studies indicate that those with PTSD who also dissociate display opposing neurological processes compared to those with PTSD who do not dissociate during a trauma reminder task (Lanius, 2010). These authors suggest that these neurological processes manifest in differing PTSD symptomatology. This claim of opposing cognitive mechanisms is helpful to differentiate a component model of dissociation, in which “classic” PTSD symptoms are unchanged, from a subtype model in which PTSD symptoms are changed.

Brain imaging studies (Bremner et al., 1999a; Bremner et al., 1999b; Liberzon et al., 1999; Rauch et al., 1996; Shin et al., 1997; Lanius et al., 2001) generally support a theory of PTSD in which symptoms are related to disinhibition of emotional centers in the brain. However, there is a large degree of heterogeneity in the data which may be
explained by a dissociative subtype. For example, Britton et al. (2005) found that individuals with combat related PTSD showed a deactivation of the left amygdala, an emotional center of the brain, during script driven imagery and a corresponding increase in activity in areas thought to assist with cognitive and emotional regulation. This finding contradicts the fear-based model of PTSD in which trauma reminders lead to activation of emotional centers of the brain. This opposing process of increased emotion regulation has been linked to the presence of dissociative symptoms (Lanius et al., 2002; Lanius et al., 2006) leading to a neurological model for a dissociative subtype that suggests that those who dissociate are experiencing a different type of PTSD (Lanius et al., 2010; Lanius et al., 2012).

The model based on these brain imaging findings (Lanius et al., 2010) suggests that there are two distinct types of PTSD represented by distinct neural processes. The first type, “re-experiencing/hyperaroused PTSD”, is a type of PTSD in which individuals demonstrate an emotion undermodulation. Specifically, these individuals show reduced activation in the ventromedial prefrontal cortex as well as the rostral anterior cingulate cortex which in turn leads to a failure to inhibit corticolimbic regions tied to emotions such as the amygdala. This failure of inhibition leads to the predominance of re-experiencing and hyperarousal symptoms. The second type, “dissociative PTSD”, is a type of PTSD characterized by emotion overmodulation, in which increased activation of the medial prefrontal cortex leads to increased inhibition and subsequent decreased activation of those same corticolimbic regions. This process is thought to lead to dissociative disengagement from trauma-related information.
These brain imaging data are often cited to support a dissociative subtype in PTSD (Dalenberg et al., 2012; Friedman et al., 2011; Lanius et al., 2012). However, methodological issues in these studies should caution proponents of the subtype model from relying solely on these findings. For example, out of the 13 neuroimaging studies reviewed in Lanius et al. (2006), the largest sample size was 36, with only 17 participants in the experimental condition (Shin et al., 2004). The majority of the studies contained less than 10 participants in each condition (i.e., PTSD or control groups). In addition to the small sample sizes, these studies provide mostly correlational data that can be quite heterogeneous. Because of these limitations, it is difficult to determine the existence of a dissociative subtype based solely on neuroimaging data. In order to bolster the claim that these neural correlates reflect a dissociative subtype fundamentally different from the re-experiencing/hyperarousal type, research is needed showing that those who dissociate show a decrease in self-report of these symptoms. The current study will look at self-report PTSD symptoms to see if the biological model described above plays out at the phenotypic level. No studies to date have specifically explored whether Lanius et al.’s (2010) model is reflected in how individuals report symptoms of PTSD. In fact, the LPA studies cited earlier showed little to no change in self-report PTSD symptom structure for those who dissociate.

PTSD symptom structure evidence for a subtype. Some studies have attempted to identify differences in PTSD symptom presentation for those who dissociate. Specifically, the three studies using LPA to identify a subtype also reported differences in PTSD symptoms between the three identified latent classes. All three studies reported that differences mainly occurred between the two high PTSD groups (high and low
dissociation) and the moderate PTSD group (low dissociation) reflecting a difference in PTSD severity. However, there were two instances in which the two high PTSD groups differed, indicating a differing symptom structure for the dissociative subtype specifically. Both flashbacks (Wolf et al., 2012a) and exaggerated startle (Wolf et al., 2012b) were shown to be increased in the dissociative group above the high PTSD/low dissociation group. These findings run counter to the claim made in the neuroimaging studies that dissociative PTSD is separate from re-experiencing/hyperarousal PTSD, and instead suggest that re-experiencing symptoms (flashbacks) and hyperarousal symptoms (startle) are elevated for those who dissociate. Furthermore, Steuwe et al. (2012) showed that avoidance and diminished interest symptoms were increased for those in the high PTSD/low dissociation group compared to both the moderate PTSD and high PTSD/high dissociation group, indicating that the dissociative subtype did not display differing PTSD symptomatology from the low dissociation group. Taken together, these findings do not fulfill the structural requirement that a subtype display differing symptoms of PTSD. Research must attempt to show that the dissociative subtype involves a significant change in PTSD symptomatology that goes beyond a simple increase in severity. Treatment outcome studies may provide the additional piece of evidence that individuals who dissociate are experiencing a different kind of disorder.

**Clinical evidence for a subtype.** The final requirement for supporting a subtype is that those who are categorized in the subtype display differing clinical needs and outcomes. The DSM-5 decision to include a dissociative subtype in PTSD has implications for how clinicians assess and treat PTSD. For example, if it is believed that those who dissociate display a type of PTSD with reduced re-experiencing and
hyperarousal symptoms (Lanius et al., 2010), then a treatment such as prolonged exposure may be less efficacious because it directly targets these symptoms. In order to confirm that dissociation is related to a different type of disorder, it should be shown that those who dissociate respond differently to different treatment modalities (Carlson et al., 2012).

Interventions for PTSD include cognitive behavioral treatments such as prolonged exposure (PE) and cognitive processing therapy (CPT) as well as pharmacotherapy with selective serotonin reuptake inhibitors (SSRIs) such as sertraline and paroxetine. A recent Institute of Medicine (IOM) report (IOM, 2008) found that CBT, specifically exposure therapy, was the only treatment deemed sufficiently valid for the treatment of PTSD based on empirical evidence. In order to understand how dissociation may affect treatment outcome, it is important to understand how different treatments are thought to impact PTSD.

PE was developed based on the emotional processing model of PTSD (Foa & Kozak, 1986; Foa, Huppert, Cahill, 2006) in which emotions are viewed as the result of information structures in memory. In PTSD, the information structure which governs fear is thought to be hyper activated and thus treatment is focused on processing traumatic memories and consequently reducing the fear response. It is theorized that symptoms of dissociation might interrupt the activation of the fear network, interfering with treatment efficacy in exposure therapy (Bryant, 2007; Jaycox & Foa, 1996). However, some treatment trials suggest that dissociation does not predict poor treatment outcome (Hagenaars, van Minnen & Hoogduin, 2010; Speckens, Ehlers, Hackmann & Clark, 2006). Furthermore, findings related to the impact of PE on dissociation are
mixed, with some showing that PE is associated with reduced dissociation (Cloitre, Koenen, Cohen & Han, 2002) and others showing that dissociation does not change during PE (McDonagh et al., 2005; Rothbaum, 1997). It may be that these findings are mixed due to the confounding of dissociation and emotional numbing in this literature, two concepts which have been shown to be separate (Feeny, Zoellner, Fitzgibbons, & Foa, 2000). It may also be that while dissociation plays a role in initial processing of fear during a trauma, it does not prevent one from retrieving the memory and processing the fear during PE.

CPT more heavily emphasizes maladaptive cognitions associated with a traumatic memory (Ehlers & Clark, 2000). However, it is important to note that many aspects of CPT overlap with PE, such as the narration of trauma accounts. The relationship of CPT and dissociation is less studied than with PE, with only one study showing a reduction in dissociation symptoms across treatment (Chard, 2005). However, a recent study has shed light on the impact of dissociation on the efficacy of CPT and is the first to suggest that those who dissociate respond differently to treatment (Resick et al., 2012). Specifically, those with higher levels of dissociation responded significantly better to CPT with written trauma accounts as compared to CPT without written trauma accounts, while those who dissociated less responded better to CPT without written trauma accounts. The authors suggested that those who dissociate do better in treatments that help to reconstruct past trauma memories for which these individuals may have limited cognitive access (Resick et al., 2012).

A second recent RCT (Cloitre et al., 2012) also showed that different psychotherapy treatments were associated with dissociation and PTSD symptoms in
different ways. Specifically, treatment involving emotion regulation skills training followed by exposure was the most effective at reducing dissociation during treatment for PTSD. This was more effective than a treatment involving emotion regulation skills training followed by supportive counseling and supportive counseling followed by exposure. Furthermore, emotion regulation skills training followed by exposure was related to better treatment response during follow up. Taken together with the findings of Resick et al. (2012), this study supports the idea that people who dissociate respond better to a treatment that involves developing the narrative of a trauma through exposure. Furthermore, this study could be interpreted to suggest that there is clinical utility in first addressing dissociative symptoms through emotion regulation training, as opposed to providing general supportive counseling, before beginning exposure therapy.

Importantly, these treatment outcome studies do not rule out the possibility that dissociation is simply a vulnerability to developing PTSD which requires different treatment approaches. One could imagine that individuals who display a large degree of reckless behavior for example, would benefit more from a treatment that directly addressed those behaviors, but this would not provide evidence for a reckless behavior subtype by itself. It would be more compelling to show that these individuals not only responded differently to treatment, but that they also represented a distinct latent class. Incorporating multiple lines of analysis will help to buttress findings concerning a dissociative subtype, and inform how dissociation should be conceptualized within PTSD.

Treatment of PTSD using pharmacotherapy has also been shown to reduce symptoms of dissociation (Marshall et al., 1998; Marshall et al., 2007). Specifically
paroxetine has been shown to reduce dissociation in an open label (Marshall et al., 1998) and a randomized placebo controlled trial (Marshall et al., 2007) among individuals with chronic PTSD. These findings suggest that treatment with SSRIs can reduce core symptoms of PTSD as well as other symptoms such as dissociation, but like therapy trials showing that dissociation changed as a result of treatment, these studies do not clarify whether dissociation is best represented as a subtype which responds differently to treatment. Additional research is needed to understand the impact of medication, specifically SSRI’s, on dissociation. Furthermore, there are no studies to our knowledge that measure the effect of dissociation on pharmacotherapy treatment outcome for PTSD. Comparing the effect of dissociation on treatment outcome between pharmacotherapy and PE will greatly increase our understanding of the clinical impact of a dissociative subtype.

**Aims and Hypotheses**

The inclusion of a dissociative subtype in the DSM-5 represents a shift in how we understand PTSD. The multiple lines of research to support this shift are somewhat compelling, but provide limited support for a subtype when evaluated on their own. It is necessary to combine these lines of research in one sample in order to provide a clearer picture of how dissociation relates to PTSD. This study evaluated whether a dissociative subtype could be derived from a sample of individuals undergoing treatment for PTSD. Specifically, this study used the requirements for a subtype outlined by Dalenberg et al. (2012) as a guide to explore multiple lines of evidence in a single sample.

**Aim 1: Statistically identifying a dissociative latent class.** Based on the requirements that a dissociative subtype should be reliably measurable and structurally
distinct (Dalenberg et al., 2012), the first aim of this study was to identify whether a subtype could be statistically derived based on latent profile analysis (LPA) of our sample. Previous research has identified three latent classes of individuals with PTSD based on LPA of PTSD and dissociative measures (Steuwe et al., 2012; Wolf et al., 2012a; Wolf et al., 2012b). These three latent classes include: individuals with low to moderate levels of PTSD and low levels of dissociation, individuals with high levels of PTSD and high levels of dissociation, and individuals with high levels of PTSD and low levels of dissociation. We hypothesized that our sample would yield a similar pattern of latent classes based on symptoms of PTSD measured by the PTSD Symptom Scale-Interview version (PSS-I; Foa, Riggs, Dancu, & Rothbaum, 1993), and symptoms of dissociation measured by the Dissociative Experiences Scale (DES; Bernstein & Putnam, 1986).

In order to identify whether dissociative symptoms were truly distinct, we re-ran the LPA using rumination, a different transdiagnostic vulnerability, as a potential subtype. For this analysis, we included results from the Ruminative Response Scale (RRS; Nolen-Hoeksema & Morrow, 1991) along with the PSS-I to see if a similar three class solution hypothesized for dissociation would manifest with rumination. This analysis addressed the criticism that the inclusion of a dissociative subtype in the DSM could lead to the proliferation of subtypes (Dalenberg et al., 2012). We hypothesized that an LPA analysis using the RRS would reveal a two class solution with one class showing moderate PTSD/low rumination and the other showing high PTSD/high rumination, indicating that rumination is a vulnerability for PTSD which fluctuates with PTSD severity, as opposed to a subtype.
Aim 2: Examine differences in PTSD symptom presentation between latent classes. Based on the requirement that a subtype show distinct patterns of symptoms of PTSD, the second aim of this study was to examine differences in PTSD symptom presentation between the PTSD latent classes derived from LPA in aim 1. While the first aim was designed to show how those who dissociated differed regarding overall PTSD severity, Aim 2 was designed to examine how dissociation impacted symptoms beyond severity. In line with finding that suggest those who dissociate display a different kind of PTSD characterized by depersonalization and derealization as opposed to re-experiencing and hyperarousal (Lanius et al., 2010), we hypothesized that individuals with high levels of PTSD and dissociation would display decreased re-experiencing and hyperarousal symptoms compared to those with high levels of PTSD and low levels of dissociation.

Aim 3: Examine the impact of dissociation on treatment response. Based on the requirement that a subtype display clinical significance (Dalenberg et al., 2010), the third aim of this study was to examine the relationship of dissociation and PTSD symptoms across two forms of evidence based treatments for PTSD. Previous research suggests that those who dissociate may benefit more in treatment that focuses on reconstructing traumatic memories (Resicket al., 2012; Cloitre et al., 2012). The underlying hypothesis from this treatment outcome literature is that reconstructing traumatic memories through exposure therapy reduces dissociation, allowing for change in PTSD symptoms. In line with these findings, we hypothesized that change in PTSD symptoms would be related to change in dissociation symptoms for those receiving PE, but not for those receiving pharmacotherapy.
Exploratory aim 1: Examine the effect of childhood abuse on dissociation.

Due to the proposal to include complex PTSD in the ICD-11, an exploratory aim for this study was to determine the impact of repeated traumatization on the development of dissociation and PTSD symptoms. Complex PTSD in the ICD-11 would be defined by additional emotion regulation difficulties such as dissociation and is theorized to develop as a result of repeated trauma in childhood (Cloitre et al., 2009). Due to the impact that this diagnosis could potentially have on the future of trauma related disorders, and this study’s unique ability to explore evidence related to complex PTSD, we included this exploratory aim. We hypothesized that repeated past traumatization, specifically childhood sexual and physical assault, would be related to increased dissociation in our sample.

Method

Participants

The sample consists of 200 men and women with chronic PTSD. Participants were recruited through community referrals, fliers and advertisements as part of a randomized controlled treatment trial examining the effect of treatment preference on treatment outcome for PE versus pharmacotherapy with sertraline. Inclusion criteria for the treatment trial included: being between the ages of 18 and 65, and having a DSM-IV diagnosis of primary, chronic PTSD. Exclusion criteria included: a current diagnosis of schizophrenia or other psychotic disorder, medically unstable bipolar disorder, depression with psychotic features or depression severe enough to require immediate psychiatric treatment, substance dependence within the past three months, self-injurious behavior or
suicide attempt within the past three months, or an ongoing relationship with the perpetrator in assault cases.

The sample consisted of 151 women (75.5%) and 49 men (24.5%) with a mean age of 37.41 (SD=11.30) years. The majority of the participants were Caucasian (65.0%) followed by African American (22.0%), and other ethnic minorities (13.0%). In this sample, 70.0% were not college educated and 48.5% had an annual household income of less than $20,000. The most common primary trauma was adult sexual assault (31.0%), followed by childhood physical or sexual assault (24.0%), adult non-sexual assault (22.5%), accident (13.5%), death or violence to a loved one (6.5%), and combat/war (2.5%).

**Interview Measures**

**PTSD Symptom Scale-Interview (PSS-I; Foa, Riggs, Dancu, & Rothbaum, 1993).** The PSS-I was used to assess current PTSD diagnosis and severity. This 17-item interview measure uses DSM-IV symptom criteria. Each item is rated on a four-point Likert scale ranging from 0 (not at all) to 3 (5 or more times per week/ very much) based on frequency and/or severity, with higher levels indicating greater PTSD severity. PTSD diagnosis was attained if at least one re-experiencing, three avoidance, and two arousal items were endorsed. The PSS-I demonstrates good convergent validity and inter-rater reliability, .93-.95 (Foa, Cashman, Jaycox, & Perry, 1997; Foa&Tolin, 2000). In the current study, over 10% of cases were rerated for inter-rater reliability; reliability was high for PTSD severity scores (ICC = .95) and PTSD diagnosis (κ = 1.00).

The PSS-I was used in this study to calculate the dysphoria, re-experiencing, avoidance, and hyperarousal symptom clusters proposed by Simms, Watson, and
Doebbeling (2002). The dysphoria symptom cluster is composed of a mix of symptoms from the current DSM-IV avoidant and hyperarousal clusters. Confirmatory factor analysis show that these symptoms load on to a latent construct thought to represent generalized distress (Simms et al., 2002). Symptoms of the dysphoria cluster include: inability to recall aspects of trauma, loss of interest, detachment, restricted affect, sense of foreshortened future, sleep disturbance, irritability, and difficulty concentrating.

**Structured Clinical Interview for DSM-IV (SCID-IV; First, Spitzer, Gibbon, & Williams, 2002).** The SCID-IV, a semi-structured interview, was used to determine if other Axis I disorders were primary. This measure has good inter-rater reliability (Lobbestail, Leurgans, & Arntz, 2011). In the current study, 10% of the SCID-IVs were rerated for inter-rater reliability; reliability across current diagnoses was acceptable (κ = .80).

**Prior Trauma History (adapted from Resnick, Best, Freedy, Kilpatrick & Falsetti, 1993).** This is a measure of past trauma experiences. Our measure of cumulative trauma exposure was derived from a total score of past trauma experiences which meet criterion-A severity. Our measure of chronic childhood assault (CA) was defined as experiencing physical assault (PA), sexual assault (SA), or both, 5 times or more in childhood. For our analyses the 5 or more occasions of assault did not include the specific index trauma that was the focus of treatment.

**Self-report Measures**

**Dissociative Experiences Scale (DES- Bernstein & Putnam, 1986).** The DES is a 28-item self-report questionnaire designed to measure the continuum of dissociative experiences including disturbances in identity, memory, awareness, and cognitions as
well as experiences of depersonalization, derealization and absorption. Items are presented on a visual analogue scale. Individuals respond by making a slash across a 100 mm long line to indicate agreement with the item. Scores are derived by averaging responses creating a total score between zero and one hundred with higher scores indicating greater pathology. Scores of 10 or below are believed to indicate non-pathological dissociation while scores of 30 or above indicate a dissociative disorder. The DES has good test-retest reliability ($r = .84$), good split half reliability (Bernstein & Putnam, 1986), and adequate convergent validity (Carlson and Putnam, 1993).

**Dissociative Experience Scale- Taxon (DES-T- Waller et al., 1996).** The DES-T includes 8 items from the DES. Taxometric analyses from Waller and colleagues (1996) have shown that these 8 items have a taxometric distribution indicating a dichotomous form of pathological dissociation which is marked by pathological dissociative experiences including amnesia, derealization, depersonalization, and identity alterations. In addition this measure was shown to be predictive of dissociative disorders such as MPD (Waller et al., 1996)

**The Ruminative Response Scale (RRS-Nolen-Hoeksema& Morrow, 1991).** The RRS is a questionnaire assessing ruminative thoughts and behaviors that arise when individuals feel down, sad, or depressed. The 22 item measure is rated on a 4-point scale ranging from a score of 1 (*never*) to 4 (*always*), with higher scores indicating the use of more ruminative responses. Due to error in the reproduction of the RRS given to participants, in this study, the RRS was composed of 10 ruminative responses items. The RRS as a whole ($\alpha = .89$) and the 10 item abbreviated version of the RRS ($\alpha = .87$) have high internal consistency (Nolen-Hoeksema& Morrow, 1991).
Procedure

Participants were initially screened over the phone using a semi-structured phone interview. Those who were potentially eligible then underwent an in-person intake interview to determine eligibility for the study. The intake interview consisted of informed consent and diagnostic assessments conducted by trained independent evaluators. Assessment measures included the PSS-I to diagnose PTSD and the SCID-IV to diagnose other comorbid psychopathology. Those who were eligible after the intake interview were then scheduled for a randomization visit, during which individuals were randomized to preference and treatment groups. Preference groups consisted of the choice group and the no choice group with those in the former group being allowed to choose their treatment and those in the latter group being randomly assigned to a treatment. During the randomization visit, participants were given a battery of self-report questionnaires including the DES. Participants then underwent 10 weekly sessions of PE or sertraline treatment. Participants rated their PTSD (PSS-SR) and depression (BDI) severity at each treatment session. After the 10 weeks of treatment, a post treatment interview was conducted by an independent evaluator blinded to treatment condition during which PTSD and depression diagnoses were assessed. Participants were reassessed at 3 month, 6 month, and 9 month follow up.

Overview of Treatment

Treatment consisted of either 10 weeks of psychotherapy or pharmacotherapy. For psychotherapy, all clinicians had at least Master’s level clinical training. All clinicians received standardized clinical training, through multiple-day initial training workshops and ongoing clinical supervision. For pharmacotherapy, all clinicians were
board certified psychiatrists.

**Psychotherapy Treatment.** Prolonged exposure (PE; Foa, Hembree, & Dancu, 2002b) consisted of 10 weekly, 90-120 min sessions, which included psychoeducation involving common reactions to trauma exposure, breathing retraining, approaching avoided situations outside of therapy (i.e., *in vivo* exposure) starting in Session 2, and approaching the memory of the trauma repeatedly (i.e., imaginal exposure) beginning at Session 3. Clients were assigned weekly homework including listening to their imaginal exposure tapes and practicing *in vivo* exposure exercises.

**Pharmacotherapy Treatment.** Pharmacotherapy consisted of 10 weeks of sertraline, monitored by a study psychiatrist. Each session ranged from 20-30 minutes. Sertraline was adjusted based on a standardized titration algorithm (Brady et al., 2000), starting at 25mg/day and proceeding up to 200mg/day, if indicated. For this sample, the mean dosage at the end of treatment was 135.68 mg/day (SD = 66.80). During visits, the psychiatrist monitored side effects and adjusted medication dosage as well as provided general encouragement and support.

**Results**

**Aim 1: Statistically Identifying a Dissociative Latent Class**

Demographic variables for our sample can be found in Table 1. Latent Profile Analysis (LPA) was conducted using Mplus 7 (Muthen & Muthen, 2012) to identify latent variables based on response patterns within our measures of dissociation and PTSD. LPA is a form of finite mixture modeling (FMM) which not only determines whether a latent class is identifiable, but also determines the best fitting model for the data (Dalenberg et al., 2012). Separate LPA’s were run that included each individual item
from the DES and PSS-I to determine fit for model solutions with two, three, four or five classes. The best-fitting model was determined by comparing fit indices between classes. In line with previous research (Steuwe et al., 2012; Wolf et al., 2012a; Wolf et al., 2012b), reported fit indices (Table 2) include the Bayesian information criteria (BIC), Akaike information criteria (AIC), the bootstrap likelihood ratio test (BLRT) and the Lo-Mendell-Ruben adjusted likelihood ratio test (LMR-A). Overall performance of the model is measured with a statistic called entropy that ranges from 0-1. Previous research suggests that BIC and BLRT are superior fit indices (Nylund, Asparouhov & Muthen, 2007) and thus it is the best approach when interpreting LPA to first identify the lowest BIC value for a model, and then see if the BLRT is significant for that model.

Each individual item from the DES and PSS-I at baseline were entered into separate LPAs testing model fit for two, three, four and five class solutions. Fit statistics for each class solution are presented in Table 2. Results showed that models with two, three, and four classes each demonstrated significant incremental fit compared to the model with one less class, evidenced by a significant p value for the BLRT. The five-class solution did not show significantly better fit above the four-class solution. Counter to our hypothesis, the model with the best fitting class structure based on the lowest BIC value was the four class model. This model also showed high entropy further suggesting good fit. In this model, class 1 contained most of the sample (n = 114), followed by class 2 (n = 53), class 3 (n = 16), and class 4 (n = 6). These results indicated that four underlying latent constructs best represented response patterns for symptoms of dissociation and PTSD in our sample.
Class comparison of four-class model. In line with previous methodological approaches, individuals were grouped based on class membership and then the groups were compared using multivariate analysis of variance (MANOVA). Group comparisons of DES and PSS-I scores can be found in Table 3. Results showed that there was a significant main effect of class membership on both PTSD symptoms and dissociation, Wilks’ Lambda = .09, $F(6, 368) = 138.31, p < .001, \eta_p^2 = .69$. Post hoc analyses were conducted using the Scheffe test for group comparisons. In regards to symptoms of PTSD, results showed higher symptoms in class 1 compared to class 2, ($p < .001$), but class 3 and class 4 did not significantly differ from any other class. In regards to symptoms of dissociation, results showed there was a significant difference between all four classes. Specifically, class 2 showed significantly greater symptoms than class 1, ($p < .001$), Class 3 showed significantly greater symptoms than class 2, ($p < .001$), and class 4 showed significantly greater symptoms than class 3, ($p < .001$). Based on these mean comparisons, the classes were identified as follows: class 1 was characterized by low dissociation and moderate PTSD symptoms; class 2 was characterized by moderate dissociation and high PTSD symptoms; class 3 was characterized by high dissociation and moderate-high PTSD symptoms; class 4 was characterized by very high dissociation and moderate-high PTSD symptoms. These results did not replicate previous findings which identified a three class solution as the best fitting model, with a distinct dissociative subtype class with high dissociation and high PTSD.

Because we did not identify a three-class solution as the best fitting model, it is difficult to clearly delineate a dissociative subtype from our analysis. For example, in our four-class solution, class 1 is analogous to class 1 from previous studies as they are both...
characterized by low dissociation and moderate PTSD. However, unlike previous studies, class 2 in our analysis showed significantly increased dissociative symptoms compared to class 1. This argues against the idea that individuals with high PTSD can have low levels of dissociation, which is a requirement to evidence a subtype. It should be noted that individuals in class 2 still had dissociative symptoms in the lower range of the scale which might suggest that some but not all of the symptoms of dissociation increased as PTSD symptoms increased.

Class 3 and 4 from our four-class solution are also difficult to map on to previous LPA findings. Previous three-class solutions identified a third class characterized by high dissociation and high PTSD. While both class 3 and 4 from our analysis showed high levels of dissociation, these classes did not differ from each other or from class 1 or 2 on PTSD symptoms. Because significantly increased PTSD symptoms coinciding with increased dissociation is required to identify a dissociative subtype, our findings argue against a subtype in our sample. It should be noted that most of our sample (n = 167) fell into the first two classes which were distinguished based on an increase in PTSD symptoms and a corresponding increase in non-pathological levels (< 30) of dissociative symptoms. The pathologically dissociative (> 30) classes (3 and 4) had higher mean PTSD scores than class 1. However, this difference did not meet significance, likely due to the small sample sizes for these classes. Thus class three and four can only be distinguished based on dissociation and therefore, cannot represent a dissociative subtype.

Class comparison of three-class model. Because a three-class solution was a significantly better fit in our analysis than a two-class solution, and because differences
between classes may have been harder to detect in our four-class solution, secondary analyses were run comparing classes from the three-class solution model. Fit statistics and group comparisons for the three class solution can be found in Table 2. The three-class solution showed a lower BIC than the two-class solution, high entropy and it was a significantly better fit than a two-class solution (BLRT \( p < .001 \)). The overall F-test for the MANOVA comparing the three classes was significant for PTSD and dissociation, Wilks’ Lambda = .15, \( F(4, 370) = 146.90, p < .001 \), \( \eta_p^2 = .61 \). Group comparisons using Scheffe’s revealed that class 1 was characterized by low dissociation and moderate PTSD; class 2 was characterized by moderate dissociation and high PTSD; and class 3 was characterized by high dissociation and moderate-high PTSD. As in the four-class solution, the highest dissociation class (class 3) was difficult to interpret because PTSD levels for this class were not significantly higher than for class 1. While the increased PTSD levels in class 3 approached significance, it was not a robust difference as would be needed to identify class 3 as a dissociative subtype. Furthermore, because this was not the best fitting model for our data, it was difficult to justify the interpretation of the three-class solution.

**LPA with DES-Taxon.** In order to determine whether a dissociative subtype is better characterized by more pathological symptoms of dissociation, we conducted a secondary LPA using the DES-Taxon items from the DES. The DES-Taxon is a subset of questions derived from the DES which through taxometric analyses (Waller, Putnam & Carlson, 1996) have been identified as more predictive of dissociative disorders, and thus represent more severe dissociative symptoms. As with the LPA using the DES total score, the LPA using the DES-Taxon items identified a four-class solution as the best
fitting model for the data as measured by lowest BIC values compared to all other class solutions, good entropy, and a significantly p value ($p < .001$) for the BLRT. Post hoc MANOVA showed a significant effect of class membership on DES-Taxon and PSS-I scores Wilks’ Lambda = .08, $F(6, 368) = 158.00$, $p < .001$, $\eta^2_p = .72$. Comparisons using Scheffé’s test showed that like the LPA using DES total scores, the effect was driven by incrementally higher dissociation between all 4 classes and higher PTSD between class 1 and 2. Specifically, dissociation was higher ($p < .001$) for class 2 ($M = 16.34$, $SD = 3.81$) compared to class 1 ($M = 2.82$, $SD = 3.07$), higher ($p < .001$) for class 3 ($M = 30.73$, $SD = 3.40$) compared to class 2, and higher ($p < .001$) for class 4 ($M = 47.64$, $SD = 6.25$) compared to class 3. PTSD scores were higher ($p = .001$) for class 2 ($M = 32.71$, $SD = 5.81$) compared to class 1 ($M = 28.09$, $SD = 6.24$). As with the previous analyses using the DES total score, comparisons with DES-Taxon classes showed that PTSD symptoms did not differ in class 3 ($M = 30.33$, $SD = 5.91$) or 4 ($M = 32.88$, $SD = 9.05$) compared to any other class. These results indicate that a dissociative subtype is not better characterized by severe dissociative symptoms measured by the DES-Taxon.

**LPA with rumination.** Due to the criticism that including a dissociative subtype in PTSD could lead to “bracket creep” in which other peripheral symptoms could be designated as a subtype, we ran a separate LPA using the ruminative response scale (RRS). LPA using rumination provides a good comparison to test the potential for bracket creep because rumination is not explicitly included in the core symptoms of PTSD and has been identified as distinct from dissociation as a predictor of PTSD (Murray et al., 2002). In line with our hypothesis, LPA examining the latent classes underlying symptoms of PTSD and rumination revealed a two-class model to be the best
The two-class solution showed the lowest BIC and AIC values, good entropy and was superior to a three-class solution based on a significant BLRT value ($p < .001$). Post hoc MANOVA showed an overall effect for class membership, Wilks’ Lambda = .31, $F(2, 190) = 209.79, p < .001$, $\eta^2_p = .69$. Comparisons between classes using the Scheffe test showed that class 2 was characterized by significantly higher PTSD ($M = 35.18, SD = 4.13, p < .001$), and rumination ($M = 31.32, SD = 5.24, p < .001$), compared to class 1 PTSD ($M = 24.95, SD = 4.26$) and rumination ($M = 24.08, SD = 4.79$). This coinciding increase in PTSD and rumination between class 1 and class 2 suggests that there is not a ruminative subtype of PTSD. Unlike the results from the LPA using the DES, this model does not include a class with high PTSD symptoms and low symptoms of the corresponding variable (in this case rumination). Without such a class, it is easier to suggest that rumination is better conceptualized as a linearly related symptom of PTSD as opposed to a subtype.

**Aim 2: Examine Differences in PTSD Symptom Presentation Between Latent Classes**

In order to evidence a dissociative subtype, those who dissociate should show a different structure of PTSD symptoms. In line with this requirement, we compared PTSD symptom clusters between the four classes identified in our best-fitting LPA model. Class comparisons of symptom clusters can be found in Table 3. There was an overall effect of class membership on PTSD symptom clusters, Wilks’ Lambda = .82, $F(12, 482) = 3.06, p < .001$, $\eta^2_p = .06$. Post hoc comparisons using the Scheffe test showed that this effect was driven by increased symptoms for class 1 compared to class 2 for both re-experiencing ($p = .001$), and dysphoria ($p = .004$). There were no significant differences between any classes on avoidance or hyperarousal. Contrary to our specific
hypothesis that the high PTSD classes would differ in symptom structure, there were no significant differences found on any of the symptom clusters between our high PTSD classes specifically (class 2, 3 & 4).

Because the four-class solution differs from previous LPA finding, we also compared symptom clusters between classes derived from the three-class model (Table 4). Again, results showed that there was a significant main effect for class membership, Wilks’ Lambda = .83, $F(8, 366) = 4.56$, $p < .001$, $\eta_p^2 = .09$, but that this effect was driven by differences between class 1 and 2. Specifically, class 2 showed higher PTSD symptoms compared to class 1 within the clusters of re-experiencing ($p = .002$), avoidance ($p = .002$), and dysphoria ($p < .001$). Like with the four-class model, differences in specific symptom clusters between classes were the result of increased PTSD symptoms for class 2 compared to class 1, and not the result of different symptom structures between the highly dissociative classes (class 3 and 4), $ps = ns$.

**Aim 3: Examine the Impact of Dissociation on Treatment Response**

Hierarchical Linear Modeling (HLM; Collins & Sayer, 2001; Raudenbush & Bryk, 2002) was used to evaluate the relationship of dissociation and changes in PTSD symptoms across pre-treatment, post-treatment, 3-month follow-up, and 6-month follow-up. Additionally, we aimed to examine the relative impact of treatment type on the relationship between dissociation and PTSD symptoms. Mean scores and standard deviations for the PSS-I and DES for each time point and between each treatment group are provided in Table 5. HLM separates variability in dependent variables into within-participant variance (i.e. change over time) and between participant variance (i.e. treatment condition). This method violates fewer statistical assumptions and is better
able to handle missing data compared to regression approaches by using maximum likelihood estimation (Singer & Willett, 2003). Because the current study did not identify a clear 3-class latent structure of PTSD and dissociative symptoms using LPA, the current HLM analysis examined the linear relationship between dissociation and PTSD across treatment. The coefficients for the fixed effects are provided in Table 6. The analysis included level one fixed effects for our dependent variable intercept (PSS-I score) and slope (rate of change in PSS-I score), as well as our independent variable intercept (DES scores) controlling for PSS-I slope. Treatment condition (PE or sertraline) was included as a level two predictor of both PSS-I slope and DES scores. Additional bivariate growth curve modeling was conducted using Mplus 7 (Muthen & Muthen, 2012) and measured the relationship between DES slope and PSS-I slope across treatment.

The baseline model indicated that the PSS-I slope term was significant, indicating the PTSD symptoms decreased across treatment and follow up, $\beta_{10} = -6.12, p < 0.001$, with 19% of the variance in PTSD symptoms being accounted for by change over time. In addition, dissociation was positively related to PTSD across the repeated measures, $\beta_{10} = 0.36, p < 0.001$, with 4% of the variance in PTSD being accounted for by dissociation symptoms. Level two results indicated that treatment did not moderate PTSD symptom change over time, $\beta_{11} = -0.06, p = 0.92$, or the relationship between dissociation and PTSD across time $\beta_{21} = -0.10, p = 0.21$.

Bivariate growth curve modeling measured the relationship between the slopes of the DES and PSS-I across time. However, our output suggested that this model may have been a poor fit for the data due to a strong relationship between change in dissociation
and change in PTSD symptoms across time. The estimated correlation between these two slopes was greater than 1 ($r = 1.77$) making it difficult to predict one variable from the other. When correlation parameters for the random effects were artificially constrained to 1, the results indicated that changes in dissociation predicted changes in PTSD symptoms across treatment and follow up, $\beta_{11} = 1.37$, $p < 0.001$. Due to the inability for this model to properly fit our data, we were unable to directly test the interaction effect of treatment type on the relationship between PSS-I and DES slopes. However, the lack of a treatment effect in our previous analyses suggested that treatment was not impacting the relationship between dissociation and PTSD.

To further explore the level 1 relationship between dissociation and PTSD, secondary multilevel growth modeling was conducted examining change in DES over time controlling for PTSD symptoms. The coefficients for the fixed effects of this model are presented in Table 7. This analysis showed that while DES scores significantly decreased over treatment and follow-up, $\beta = -2.16$, $p < 0.001$, this effect became non-significant when PSSI slope was added to the model, $\beta = -0.35$, $p = 0.56$. With 13% of the variance in DES slope being accounted for by PTSD symptoms, it is likely that PTSD symptoms were partly driving the change in dissociation across time. Overall, these HLM findings showed that both the random intercepts and slopes of the DES and PSS-I were strongly related, and that changes in PTSD symptoms were largely driving changes in dissociative symptoms across two different treatment modalities. Taken together, these findings suggest that symptoms of dissociation fluctuate linearly with symptoms of PTSD similarly across different treatment modalities.

**Exploratory Aim 1: Examine the Effect of Childhood Abuse on Dissociation**
Hierarchical linear regression was used to test whether chronic childhood abuse (measured as reporting greater than 5 instances of physical assault, sexual assault, or both) was related to the development of dissociative symptoms above and beyond past traumatization. Results showed that a total cumulative score for past criterion A traumas (including both adult and childhood traumas) only peripherally predicted dissociation scores measured by the DES, $\beta = 0.13, p = .07$. When chronic childhood assault was added to the model it did not significantly increase predictability, $\Delta R^2 = .000, p = .78$. Similar null results were found for the experience of both sexual assault and physical assault when they were separately added to the model. These findings suggested that neither past traumatization in general or the experience of chronic childhood abuse predicted dissociative symptoms in our sample of adults with PTSD, refuting claims from the complex PTSD literature (Cloitre et al., 2009) that chronic childhood abuse is related to more dissociative PTSD.

**Discussion**

The goal of this study was to determine whether a dissociative subtype could be identified in a sample of individuals seeking treatment for chronic PTSD. We first attempted to statistically derive an underlying latent construct of dissociative PTSD. We then explored how PTSD symptom structures differed for those who dissociate. Finally, we looked to see how dissociation impacted response to different treatments for PTSD. Each of these attempts yielded little evidence for the existence of a dissociative subtype in our sample of individuals seeking treatment for chronic PTSD.

Our LPA results suggested that four latent classes characterized symptoms of dissociation and PTSD in our sample. Unlike previous findings (Steuwe et al., 2012;
Wolf et al., 2012a; Wolf et al., 2012b), this latent model did not contain one independent class that was distinct from the others based solely on dissociation symptoms. Without this dissociative latent class, it was difficult to interpret the presence of a dissociative subtype in our data. Furthermore, even when we tested the three-class solution, we were still not able to robustly demonstrate the existence of a distinct dissociative class. Taken together, these data showed that there was not a subgroup of individuals in our sample analogous to the dissociative subtype found in previous studies.

Our study differed from prior approaches in some key ways that might explain our inability to replicate previous findings. One major difference was our use of the DES as a general measure of dissociative tendencies. Previous studies had used two single items from the clinician administered PTSD scale (CAPS) that measure depersonalization and derealization specifically as their assessment of dissociation. Our use of the DES provided a more psychometrically sound measure of the broad spectrum of dissociative symptoms. It may be that the use of a broad measure of dissociation made it more difficult to identify a subtype. However, our DES-Taxon findings argue against this position as they showed that even the more severe symptoms of dissociation, like depersonalization and derealization, did not better characterize a dissociative subtype.

It is important to note that the best-fitting model for latent dissociative classes was not a two-class solution as was found for rumination. A two-class solution would suggest that two variables have a more linear relationship given that there is no identified group with low scores of one variable and high scores of the other. Arguments against a dissociative subtype suggest that dissociation may be linearly related to PTSD like other core symptoms of the disorder (Dalenberg, et al., 2012). However, a two-class solution
was not identified for dissociation and PTSD, suggesting dissociation was not better conceptualized as an additional symptom of the disorder like rumination. The fact that dissociation is not better represented in a three-class model (i.e. dissociative subtype) or a two-class model (i.e. additional core symptom), suggests that dissociation may simply be a symptom of PTSD with a low base rate like anger or reckless behavior. In fact, within our sample, 86.2% of individuals had total DES scores lower than 30 which is a clinical cutoff for severe dissociation (Bremner et al., 1992). Given this very low base rate of severe dissociation, it may be too difficult to make meaningful inferences about a dissociative class. Evidence showing that those who dissociate present with a different PTSD symptom profile or respond differently to treatment would bolster the claim that dissociation is not simply an infrequently experienced symptom of PTSD, and is better represented as a subtype.

In our analysis of the impact of dissociative class on PTSD symptom profile, we found further evidence refuting the presence of a dissociative subtype in our sample. Our results from the best-fitting four class solution and the three class solution showed that for individuals in the classes with high levels of PTSD, there were no differences on any of the symptom clusters, regardless of the fact that dissociation varied between all of these groups. These findings are not in line with the neuroimaging literature (Lanius et al., 2010) that suggests different cognitive processes lead to reduced hyperarousal and re-experiencing symptoms for those with PTSD who also dissociate. It is important to note that the neuroimaging studies measured dissociative states acutely while individuals were presented with trauma cues, as opposed to measuring dissociation more broadly as we did in our study. The discrepancy between our results and those of neuroimaging studies
may indicate that while some individuals respond differently to the acute presentation of a trauma reminder, this does not translate to different PTSD symptoms in general, and thus is not indicative of a subtype of PTSD.

Our modeling of the relationship between dissociation and PTSD symptoms across treatment showed that while reduction in dissociation was related to reduction in PTSD across treatment, there was not a differential effect of dissociation on PTSD symptom change between PE and pharmacotherapy, suggesting that regardless of what treatment you give someone, as long as it is effective at reducing PTSD, it will also reduce dissociation. This prediction is strengthened by our finding that the growth curve for dissociation became non-significant when controlling for PTSD symptoms, indicating that any effect of treatment on dissociation is accounted for by the effect of treatment on PTSD symptoms. These findings are in contrast with the theory that exposure therapy has added benefits for those who dissociate (Cloitre et al., 2012), arguing against the clinical utility of including a subtype in the nosology for PTSD. Evidence is needed showing that the relationship between dissociation and PTSD differs depending on treatment modality, in order to support the clinical utility of the dissociative subtype.

Our finding that neither chronic child abuse or cumulative abuse in general was related to dissociation symptoms does not support the developmental theory of PTSD that suggests more complex symptoms of PTSD develop specifically from childhood trauma (Cloitre et al., 2009). However, evidence for a developmental theory of dissociation specifically suggests that parental non-responsiveness, and not the actual experience of trauma, predicts the development of dissociative symptoms in adulthood (Dutra, Bureau, Holmes, Lyubchick & Lyons-Ruth, 2009). It may be that the relationship between the
parent and the child, which was not measured in our study, is carrying the effect of childhood trauma on the development of dissociative symptoms. In regards to complex PTSD, this hypothesis argues for a buffering model in which the experience of childhood trauma leads to more complex symptoms of PTSD only when it is coupled with parental unresponsiveness. Future complex PTSD research should test such a buffering model to better understand the etiology of complex symptoms such as dissociation.

**Limitations**

While our findings inform the debate regarding a dissociative subtype, they are limited in some important ways. First, the use of LPA, while empirically supported, is subjective and somewhat artificial. For example, clear guidelines have not been developed for the interpretation of fit indices, and latent classes are somewhat artificial structures that ignore variability within classes. However, LPA is superior to other analytical approaches such as factor analysis and taxometric methods because it is able to group individuals into multiple classes. Second, our sample consisted only of individuals diagnosed with PTSD. This may have created a suppression effect for PTSD symptoms that prevented our ability to detect a clear subtype. However, the size of our sample, and the diversity of trauma type and PTSD severity within our sample, makes a suppression effect less likely. Furthermore, our methodology was in line with previous research exploring the existence of a dissociative subtype.

**Conclusion**

This study combined different empirically supported methods in one sample and thus provided more in-depth and generalizable results. The inclusion of a dissociative subtype in the DSM-5 and the proposal of Complex PTSD in the ICD-11 encourage
clinicians to identify symptoms of dissociation in order to inform treatment for PTSD.

Our findings suggest that this approach may not be necessary as dissociation was not found to represent a subtype and did not interfere with different treatments in our sample. Instead it seems that the classic fear-based approach to understanding and treating PTSD is still effective even for those who dissociate, and the reconceptualization of PTSD in the nosology may be premature. Future research should continue to integrate methods for studying dissociation in PTSD to develop a more accurate understanding of how these symptoms relate and what etiological factors account for their relationship.
### Table 1

**Demographics and Trauma Type**

<table>
<thead>
<tr>
<th>Variable</th>
<th>N</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gender</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Female</td>
<td>151</td>
<td>76</td>
</tr>
<tr>
<td>Male</td>
<td>49</td>
<td>24</td>
</tr>
<tr>
<td>Education</td>
<td></td>
<td></td>
</tr>
<tr>
<td>College Educated</td>
<td>60</td>
<td>30</td>
</tr>
<tr>
<td>Not College Educated</td>
<td>140</td>
<td>70</td>
</tr>
<tr>
<td>Ethnicity</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Caucasian</td>
<td>130</td>
<td>65</td>
</tr>
<tr>
<td>Minority status</td>
<td>70</td>
<td>35</td>
</tr>
<tr>
<td>Trauma type</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Adult sexual assault</td>
<td>62</td>
<td>31</td>
</tr>
<tr>
<td>Adult non-sexual assault</td>
<td>45</td>
<td>23</td>
</tr>
<tr>
<td>Childhood sexual assault</td>
<td>35</td>
<td>18</td>
</tr>
<tr>
<td>Childhood non-sexual assault</td>
<td>13</td>
<td>06</td>
</tr>
<tr>
<td>Other</td>
<td>45</td>
<td>22</td>
</tr>
<tr>
<td></td>
<td>M</td>
<td>SD</td>
</tr>
<tr>
<td>Age</td>
<td>37.41</td>
<td>11.30</td>
</tr>
</tbody>
</table>

**Note.** M = mean, SD = standard deviation, N = sample size, % = percent of variable within each treatment group.
### Table 2

*Fit of Competing Class Models for DES and PSS-I*

<table>
<thead>
<tr>
<th>Model</th>
<th>Log-Likelihood</th>
<th>AIC</th>
<th>BIC</th>
<th>Entropy</th>
<th>BLRT p-value</th>
<th>LMR-A p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>2 Class</td>
<td>-27,309.534</td>
<td>54,891.069</td>
<td>55,331.946</td>
<td>.99</td>
<td>&lt;.001</td>
<td>.13</td>
</tr>
<tr>
<td>3 Class</td>
<td>-27,051.058</td>
<td>54,466.116</td>
<td>55,056.144</td>
<td>.96</td>
<td>&lt;.001</td>
<td>.64</td>
</tr>
<tr>
<td>4 Class</td>
<td>-26,908.775</td>
<td>54,237.549</td>
<td>55,012.667</td>
<td>.97</td>
<td>&lt;.001</td>
<td>.83</td>
</tr>
<tr>
<td>5 Class</td>
<td>-26,866.561</td>
<td>54,281.121</td>
<td>55,169.360</td>
<td>.97</td>
<td>1.000</td>
<td>.72</td>
</tr>
</tbody>
</table>

*Note:* AIC = Akaike information criteria; BIC = Bayesian information criteria; BLRT = bootstrap likelihood ratio test; LMR-A = Lo-Mendell-Rubin, adjusted likelihood ratio test
Table 3
Mean Differences in Severity of PTSD Total Symptoms, PTSD Cluster Symptoms and Dissociative Symptoms among the 4 Latent Classes of the 4 Class Model

<table>
<thead>
<tr>
<th>Var</th>
<th>Class 1</th>
<th>Class 2</th>
<th>Class 3</th>
<th>Class 4</th>
<th>Comp</th>
<th>Sig</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>n = 114</td>
<td>n = 53</td>
<td>n = 16</td>
<td>n = 6</td>
<td></td>
<td></td>
</tr>
<tr>
<td>PTSD</td>
<td>M 27.54</td>
<td>M 32.91</td>
<td>M 31.56</td>
<td>M 10.42</td>
<td>2 &gt; 1</td>
<td>&lt; .001</td>
</tr>
<tr>
<td></td>
<td>SD 6.12</td>
<td>SD 5.21</td>
<td>SD 6.79</td>
<td>SD 10.42</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Reexp</td>
<td>M 6.82</td>
<td>M 8.70</td>
<td>M 8.06</td>
<td>M 4.65</td>
<td>2 &gt; 1</td>
<td>&lt; .01</td>
</tr>
<tr>
<td></td>
<td>SD 2.44</td>
<td>SD 2.55</td>
<td>SD 3.23</td>
<td>SD 1.98</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Avoid</td>
<td>M 4.39</td>
<td>M 4.98</td>
<td>M 4.81</td>
<td>M 5.17</td>
<td>None</td>
<td>ns</td>
</tr>
<tr>
<td></td>
<td>SD 1.41</td>
<td>SD 1.13</td>
<td>SD 1.31</td>
<td>SD 0.98</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dysph</td>
<td>M 12.65</td>
<td>M 15.02</td>
<td>M 15.38</td>
<td>M 14.50</td>
<td>2 &gt; 1</td>
<td>&lt; .01</td>
</tr>
<tr>
<td></td>
<td>SD 3.95</td>
<td>SD 3.66</td>
<td>SD 3.16</td>
<td>SD 1.57</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hyper</td>
<td>M 3.69</td>
<td>M 4.21</td>
<td>M 3.31</td>
<td>M 3.83</td>
<td>None</td>
<td>ns</td>
</tr>
<tr>
<td></td>
<td>SD 1.71</td>
<td>SD 1.59</td>
<td>SD 2.30</td>
<td>SD 1.76</td>
<td></td>
<td></td>
</tr>
<tr>
<td>DES</td>
<td>M 7.74</td>
<td>M 23.64</td>
<td>M 40.15</td>
<td>M 59.63</td>
<td>4 &gt; 3 &gt;</td>
<td>&lt; .001</td>
</tr>
<tr>
<td></td>
<td>SD 4.25</td>
<td>SD 4.64</td>
<td>SD 3.50</td>
<td>SD 6.31</td>
<td>2 &gt; 1</td>
<td></td>
</tr>
</tbody>
</table>

Note. PTSD = posttraumatic stress disorder; DES = dissociative experiences scale; Var = variable; Reexp = Reexperiencing cluster; Avoid = Avoidance Cluster; Dysph = Dysphoria Cluster; Hyper = Hyperarousal Cluster; Comp = comparison between classes; Sig = significance of differences between classes.
Table 4
Mean Differences in Severity of PTSD Total Symptoms, PTSD Cluster Symptoms and Dissociative Symptoms among the 3 Latent Classes of the 3 Class Model

<table>
<thead>
<tr>
<th>Variable</th>
<th>Class 1 (n = 123)</th>
<th>Class 2 (n = 49)</th>
<th>Class 3 (n = 17)</th>
<th>Comparisons</th>
<th>Significance</th>
</tr>
</thead>
<tbody>
<tr>
<td>PTSD Total</td>
<td>M = 27.67, SD = 5.96</td>
<td>M = 33.55, SD = 5.47</td>
<td>M = 31.47, SD = 7.84</td>
<td>2 &gt; 1</td>
<td>p &lt; .001</td>
</tr>
<tr>
<td>Reexperiencing</td>
<td>M = 6.95, SD = 2.48</td>
<td>M = 8.55, SD = 2.78</td>
<td>M = 8.29, SD = 3.44</td>
<td>2 &gt; 1</td>
<td>p &lt; .01</td>
</tr>
<tr>
<td>Avoidance</td>
<td>M = 4.37, SD = 1.38</td>
<td>M = 5.14, SD = 3.12</td>
<td>M = 4.82, SD = 0.81</td>
<td>2 &gt; 1</td>
<td>p &lt; .01</td>
</tr>
<tr>
<td>Dysphoria</td>
<td>M = 12.63, SD = 3.87</td>
<td>M = 15.63, SD = 3.57</td>
<td>M = 14.76, SD = 3.65</td>
<td>2 &gt; 1</td>
<td>p &lt; .001</td>
</tr>
<tr>
<td>Hyperarousal</td>
<td>M = 3.71, SD = 1.70</td>
<td>M = 4.22, SD = 1.67</td>
<td>M = 3.59, SD = 2.32</td>
<td>No Differences</td>
<td>all ps = ns</td>
</tr>
<tr>
<td>DES Total</td>
<td>M = 8.49, SD = 4.91</td>
<td>M = 25.91, SD = 5.29</td>
<td>M = 48.29, SD = 9.43</td>
<td>3 &gt; 2 &gt; 1</td>
<td>all ps &lt; .001</td>
</tr>
</tbody>
</table>

Note: PTSD = posttraumatic stress disorder; DES = dissociative experiences scale.
Table 5

Descriptive Statistics for each Treatment Condition

<table>
<thead>
<tr>
<th>Measure</th>
<th>Prolonged Exposure</th>
<th>Sertraline</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>M</td>
<td>SD</td>
</tr>
<tr>
<td><strong>Pretreatment</strong> (n = 200)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>PSS-I</td>
<td>29.41</td>
<td>6.90</td>
</tr>
<tr>
<td>DES</td>
<td>15.64</td>
<td>2.71</td>
</tr>
<tr>
<td><strong>Posttreatment</strong> (n = 155)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>PSS-I</td>
<td>10.49</td>
<td>9.41</td>
</tr>
<tr>
<td>DES</td>
<td>10.48</td>
<td>11.57</td>
</tr>
<tr>
<td><strong>3 month follow up</strong> (n = 115)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>PSS-I</td>
<td>8.17</td>
<td>8.82</td>
</tr>
<tr>
<td>DES</td>
<td>8.46</td>
<td>10.20</td>
</tr>
<tr>
<td><strong>6-month follow-up</strong> (n = 119)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>PSS-I</td>
<td>8.36</td>
<td>9.20</td>
</tr>
<tr>
<td>DES</td>
<td>8.36</td>
<td>13.12</td>
</tr>
</tbody>
</table>

*Note. PSS-I = PTSD symptom scale – interview, DES = dissociative experiences scale.*
### Table 6

*Fixed Effects for the Model Examining The Effect of Dissociation on PTSD Growth Curve*

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Posttraumatic stress disorder</th>
<th>$p$-Value</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Fixed Effects</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pretreatment</td>
<td>$\beta_{00}$</td>
<td>20.01</td>
</tr>
<tr>
<td>Treatment rate of change</td>
<td>$\beta_{10}$</td>
<td>-6.12</td>
</tr>
<tr>
<td>Difference between PE &amp; Sertraline</td>
<td>$\beta_{11}$</td>
<td>-0.06</td>
</tr>
<tr>
<td>Dissociation</td>
<td>$\beta_{20}$</td>
<td>0.36</td>
</tr>
<tr>
<td>Difference between PE and Sertraline</td>
<td>$\beta_{21}$</td>
<td>0.06</td>
</tr>
</tbody>
</table>

*Note: PE = prolonged exposure*
Table 7
Fixed Effects for the Model Examining the Effect of PTSD on Dissociation Growth Curve

<table>
<thead>
<tr>
<th>Parameter</th>
<th>dissociation</th>
<th>p-Value</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Fixed Effects</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pretreatment</td>
<td>$\beta_{00}$</td>
<td>6.56</td>
</tr>
<tr>
<td>Treatment rate of change</td>
<td>$\beta_{10}$</td>
<td>-0.35</td>
</tr>
<tr>
<td>Difference between PE &amp; Sertraline</td>
<td>$\beta_{11}$</td>
<td>0.78</td>
</tr>
<tr>
<td>PTSD</td>
<td>$\beta_{20}$</td>
<td>0.31</td>
</tr>
<tr>
<td>Difference between PE and Sertraline</td>
<td>$\beta_{21}$</td>
<td>-0.02</td>
</tr>
</tbody>
</table>
References


