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A Critical Evaluation of the Complex PTSD Literature: Implications for *DSM-5*

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Complex posttraumatic stress disorder (CPTSD) has been proposed as a diagnosis for capturing the diverse clusters of symptoms observed in survivors of prolonged trauma that are outside the current definition of PTSD. Introducing a new diagnosis requires a high standard of evidence, including a clear definition of the disorder, reliable and valid assessment measures, support for convergent and discriminant validity, and incremental validity with respect to implications for treatment planning and outcome. In this article, the extant literature on CPTSD is reviewed within the framework of construct validity to evaluate the proposed diagnosis on these criteria. Although the efforts in support of CPTSD have brought much needed attention to limitations in the trauma literature, we conclude that available evidence does not support a new diagnostic category at this time. Some directions for future research are suggested.

Complex posttraumatic stress disorder (CPTSD) was first proposed by Herman (1992a) to describe a syndrome observed in survivors of prolonged, repeated trauma. Herman wrote, "the diagnosis of post-traumatic [sic] stress disorder, as it is presently defined, does not fit accurately enough. The existing diagnostic criteria for this disorder are derived mainly from survivors of circumscribed traumatic events. They are based on the prototypes of combat, disaster, and rape" (p. 119). The new diagnosis comprised symptom clusters reflecting alterations in affect regulation, consciousness, self-perception, perception of the perpetrator, relations with others, and systems of meaning. Following this proposal, the posttraumatic stress disorder (PTSD) field trial for the *Diagnostic and Statistical Manual of Mental Disorders* (4th ed.; *DSM-IV*; American Psychiatric Association, 1994) tested this diagnosis as disorders of extreme stress, not otherwise specified (DESNOS), a disorder closely related to CPTSD.

The findings of the field trial indicated that nearly everyone who met criteria for DESNOS met criteria for PTSD (Roth, Newman, Pelcovitz, van der Kolk, & Mandel, 1997). Although the committee believed there was not sufficient evidence to consider DESNOS as an independent diagnosis, they listed symptoms of DESNOS as associated features of PTSD in the *DSM-IV*, along with other comorbid symptoms. Subsequently, other diagnoses have been proposed to capture the phenomena that Herman (1992b) described: personality change after a catastrophic event according to the *International Classification of Diseases, Tenth Revision* (*ICD-10*; World Health Organization [WHO], 1992), developmental trauma disorder (proposed for children who experience prolonged trauma; van der Kolk, 2005), and posttraumatic personality disorder (Classen, Pain, Field & Woods, 2006). We use the term CPTSD both for consistency and because CPTSD is again proposed for inclusion in the *DSM-5*.

This study examines extant research on CPTSD to consider whether there are sufficient data to warrant adoption of a new diagnosis. We searched PsycINFO for the terms "complex PTSD," "complex trauma," "DESNOS," "posttraumatic personality disorder," and "personality change after a catastrophic event." We did not include developmental trauma disorder because we limited our review to studies of adult samples. We employed a snowballing strategy and reviewed the citations and reference sections of relevant manuscripts. Finally, we sought

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in-press articles from experts in the area of CPTSD. We use the framework of construct validity (Cronbach & Meehl, 1955) to evaluate the CPTSD conceptualization, the measures developed to assess its proposed construct, and its distinctiveness from other disorders. Further, we evaluate extant research on treatment for CPTSD to determine whether a different type, sequence, or length of treatment is indicated for survivors of prolonged trauma.

Constructs and Diagnoses

Psychiatric diagnoses are theoretical constructs developed to help understand the co-occurrence of psychiatric symptoms and other psychopathological processes (Borsboom, 2008). For instance, PTSD was codified in the late 1970s to help understand the psychopathological sequelae experienced by large numbers of Vietnam veterans, and developed further by early research on rape, domestic violence, and child abuse (Friedman, Resick, & Keane, 2007).

Construct validity refers to the process of establishing the evidentiary strength and usefulness of unobservable constructs such as psychiatric diagnoses (Cronbach & Meehl, 1955). The first step is "to develop a precise and detailed conception of the target construct and its theoretical context" (Clark & Watson, 1995; p. 310), followed by a description of the construct in terms of a set of operations that can be used to measure and define it, which includes establishing the psychometric properties of the procedures used to measure the construct. This conceptualization is then investigated rigorously, what Cronbach and Meehl described as developing a *nomological network* of research support for the construct. This includes evaluating the convergent and discriminant validity of the construct of interest. Finally, developing construct validity for a new diagnosis requires demonstrating incremental validity and clinical utility, meaning it must provide something over and above already established diagnoses in terms of knowledge about the etiology, course, or treatment of the symptoms. Throughout this article, we evaluate CPTSD using these criteria.

Clinical Descriptions of CPTSD

In an effort to understand the variety of symptoms, traits, and traumas identified as characteristic of CPTSD, we searched the literature for definitions of CPTSD and proposed related disorders. We found significant variability in descriptions of the types of traumatic events that precipitate CPTSD and in core symptoms of the disorder.

Precipitating Traumatic Events

CPTSD was originally conceptualized as the sequela of complex trauma, or trauma that is prolonged in duration and of early life onset (Herman, 1992b). The most common exemplar is prolonged trauma of an interpersonal nature, particularly childhood sexual abuse (CSA; Choi, Klein, Shin, & Lee, 2009; Jackson, Nissenson, & Cloitre, 2010; Roth et al., 1997), or childhood trauma and neglect more broadly (Classen et al., 2006; Dorahy et al., 2009). The criteria for the *ICD-10* diagnosis of personality change following catastrophic experience also includes the qualifier that the stressor resulting in these symptoms must be so severe that considering personal vulnerability is unnecessary to explain its profound effect on personality (WHO, 1992). Subsequently, Courtois (2004) expanded complex trauma experiences to include "other types of catastrophic, deleterious, and entrapping traumatization occurring in childhood and/or adulthood" (p. 412), such as ongoing war, prisoner-of-war, refugee status, human trafficking and prostitution, and acute or chronic illness. The unique trademark of complex trauma, however, has also been described as a compromise in the individual's self-development, which occurs during a critical window of development in childhood, when self-definition and self-regulation are being formed (Courtois & Ford, 2009). Although prolonged trauma has traditionally been considered necessary for the development of CPTSD, Courtois (2004) suggested that CPTSD may also result from a single catastrophic trauma. Variation in descriptions of complex trauma and the proposal that a single trauma might result in CPTSD have led to a lack of clarity regarding how to differentiate simple and complex trauma in some cases. Most recently, however, in a report on an expert clinician survey of best treatment for CPTSD, complex trauma was described as "circumstances such as childhood abuse or genocide campaigns under which they are exposed for a sustained period to repeated instances or multiple forms of trauma," typically of an interpersonal nature, and occurring under circumstances where escape is not possible due to physical, psychological, maturational, environmental, or social constraints (Cloitre, Petkova, Wang, & Lu, 2012).

Further research is needed to determine whether there is a unique relationship between complex trauma and CPTSD. Although one study indicated the number of traumas experienced in childhood predicted problems with disturbed affective and interpersonal functioning (Cloitre, Petkova, Wang, & Lu, 2012), research has not evaluated whether complex trauma necessarily (and specifically) results in CPTSD. Trauma research has revealed that type and amount of trauma exposure can influence the development of PTSD. For instance, two large meta-analyses of risk factors associated with the development of PTSD documented a consistent relationship between exposure to trauma prior to the index event and the development of PTSD (Brewin, Andrews, & Valentine, 2000; Ozer, Best, Lipsey, & Weiss, 2003). Findings from the National Comorbidity Study (Kessler, Sonnega, Bromet, Hughes, & Nelson, 1995) indicated that with regard to trauma exposure, complexity is the norm, not the exception. Of this nationally representative sample, 61% and 51% of males and females, respectively, reported exposure to at least one of 12 types of trauma. Among those exposed, 64% reported more than one trauma exposure with 20% of males and 11% of females reporting experiencing three or more traumatic events. The PTSD rates also varied significantly as a function of type of trauma, with rape being associated with the highest rates of PTSD for both genders of those traumas assessed. Thus,

research has clearly demonstrated that the amount and type of trauma impacts posttraumatic adaptation, even under current diagnostic standards. What has yet to be demonstrated is evidence of a qualitatively different relationship between complex trauma exposure as defined above and the development of a unique symptom pattern that is best captured by an independent diagnosis called CPTSD.

Symptoms

Symptoms of CPTSD include several defining criteria of PTSD (reexperiencing, avoidance, numbing, and hyperarousal), as well as disturbances in self-regulatory capacities that have been grouped into five categories: emotion regulation difficulties, disturbances in relational capacities, alterations in attention and consciousness (e.g., dissociation), adversely affected belief systems, and somatic distress or somatization (Cloitre et al., 2011). There has been, however, variability across descriptions of the specific symptoms proposed for each category. For example, although almost all CPTSD definitions include some form of affect dysregulation as a core feature, some descriptions of affective symptoms were difficult to operationalize (e.g., difficulty managing negative mood, Jackson et al., 2010; anxiety, Herman, 1992b). Further, dissociation, memory disturbance (Ford, 1999; Herman, 1992b; Pelcovitz et al., 1997), and disturbance in attention regulation or concentration (Herman, 1992b; Courtois, 2004; Margolin & Vickerman, 2007) have all been discussed as manifestations of alterations in consciousness in CPTSD. Descriptions of other CPTSD symptoms have been similarly varied. Several additional proposed CPTSD symptoms (e.g., self-harm, hopelessness, change from previous personality, or loss of previously sustaining beliefs) do not fall in the aforementioned symptom clusters. The lack of consistency in symptom descriptions has created challenges in defining and measuring CPTSD. Recent publications indicate that the field may be moving toward consensus on the proposed core and associated symptoms (Cloitre et al., 2011), which can facilitate efforts to develop measures of the construct (Courtois & Ford, 2009).

Measurement of CPTSD

The Structured Interview for Disorders of Extreme Stress (SIDES)

Establishing the construct validity of a diagnosis requires being able to measure the construct reliably. To date, no measure of CPTSD specifically has been established, and only one measure, the SIDES (Pelcovitz et al., 1997; Scoboria, Ford, Lin, & Frisman, 2008) was developed to measure DESNOS. Although DESNOS and CPTSD are often used interchangeably, they are not entirely synonymous: DESNOS represents symptoms not included in the criteria for PTSD (i.e., some of the associated features described in the *DSM-IV*), while definitions of CPTSD generally include PTSD symptoms and associated features.

Lifetime and current diagnoses, as well as a total severity score, can be obtained from the SIDES. The original 48 items included those designed to assess regulation of affect and impulses (e.g., "I find it hard to calm myself down after I become upset and have trouble getting back on track"); attention or consciousness (e.g., "I 'space out' when I feel frightened or under stress"); self-perception (e.g., "I feel chronically guilty about all kinds of things"); perception of the perpetrator (e.g., "I sometimes think that people had the right to hurt me"); relations with others (e.g., "I have trouble trusting people"); somatization (e.g., "I suffer from chronic pain, yet doctors could not find a clear cause for it"); and systems of meaning (e.g., "I believe that life has lost its meaning"). Participants are asked how much each item has been true in the past month; responses range from *none/not at all* to *very much so* (wording is specific to each item) and are rated on a scale from 0 to 4. In the development sample, these major scales had a Cronbach's α ranging from .53 to .90; the internal consistency estimate for the overall scale was .96. The perception of the perpetrator scale had the lowest α value; thus, this scale was excluded from the overall diagnosis.

There are many inconsistencies in the use of the SIDES to diagnose DESNOS because varying scoring formulations have been used (Ford & Kidd, 1998; Pelcovitz et al., 1997; Scoboria et al., 2008; Zlotnick & Pearlstein, 1997). One study evaluated the factor structure of a revised version of the SIDES (Scoboria et al., 2008) in a trauma and substance abuse treatment-seeking sample. A 5-factor model was derived from the retained items: demoralization, somatic dysregulation, anger dysregulation, risk/self-harm, and altered sexuality. These factors do not appear to have been used in subsequent definitions or research on DESNOS or CPTSD. Importantly, although dissociation is considered an important aspect of DESNOS, none of the SIDES dissociation items loaded significantly on these factors. Additional items not related to these factors included those assessing ineffectiveness, guilt, and shame.

Several studies have investigated the validity of the SIDES. Zlotnick and Pearlstein (1997) reported that the various subscales were moderately correlated with the borderline subscale of the Personality Diagnostic Questionnaire-Revised (PDQ-R; Hyler, Skodol, Kellman, Oldham, & Rosnick, 1990), the avoidant and hypervigilance subscales of the Clinician Administered PTSD Scale (CAPS; Weathers, Keane, & Davidson, 2001), the Self-Injury Inventory (Zlotnick, Shea, Pearlstein, & Simpson, 1996), and the hostile and somatization subscales of the Symptom Checklist-90-R (SCL-90-R; Derogatis, 1977). Scoboria et al. (2008) investigated the concurrent, convergent, and discriminant validity of the SIDES. Comparison of individuals with no trauma history, noninterpersonal trauma histories, physical trauma, and sexual trauma indicated that those with sexual trauma had higher scores on all SIDES-R factors as well as the total scale. In addition, participants with recurring interpersonal trauma histories had higher scores on the total scale as well as the somatic dysregulation, anger dysregulation, and risk/self-harm scales.

An important limitation to using the SIDES for diagnostic purposes has been noted. Scoboria et al. (2008) reported that although the interview is administered in the context of discussing past traumatic experiences, the interview questions focus on general symptoms and not those related to a specific event. PTSD, by definition, is diagnosed only if symptom onset is related to a traumatic event. Although the hypothesis is that DESNOS/CPTSD develops in response to trauma, it is notable that the only measure specifically developed to assess this construct does not tie symptoms to traumatic events. Thus, a positive diagnosis based on the SIDES only indicates that symptoms are present. If they are present in individuals who report trauma exposure, then we know only that these symptoms are correlated, but cannot determine whether these symptoms were present before the trauma. In other words, no conclusions of causality can be drawn through the use of this instrument.

Other Measures

Other measures have been used in an effort to assess CPTSD. In a study of treatment of PTSD related to childhood abuse, Cloitre et al. (2010) used a variety of measures to tap into the various constructs representing a range of symptoms associated with PTSD: Negative Mood Regulation Scale (Catanzaro & Mearns, 1990), Inventory of Interpersonal Problems (Horowitz, Rosenberg, Baer, Ureño, & Villaseñor, 1988), Beck Depression Inventory (Beck, Ward, Mendelson, Mock, & Erbaugh, 1961), State-Trait Anxiety Inventory (Spielberger, 1983), and State-Trait Anger Expression Inventory (Spielberger, 1991). Choi et al. (2009) used the Korean versions of the SCL-90-Somatization subscale (Kim, Kim, & Won, 1984), Dissociative Experiences Scale (Park et al., 1995), and the Inventory of Self-Altered Capacities (Park, Suh, & Lee, 2006) to compare PTSD and DESNOS symptoms among women who had experienced CSA and prostitution.

Resick, Nishith, and Griffin. (2003) used the Trauma Symptom Inventory (TSI; Briere, 1995) to measure symptoms associated with CPTSD. Among other symptoms, the TSI evaluates the intra- and interpersonal problems often associated with chronic trauma and DESNOS: dissociation, impaired self-reference, sexual concerns, dysfunctional sexual behavior, and tension reduction behavior. Finally, Zlotnick et al. (1996) used seven self-report measures to assess six key symptoms of DESNOS: somatization (assessed by the Somatization subscale of the SCL-90-R; Derogatis, 1977), dissociation (assessed by the DES; Bernstein & Putnam, 1986), affective symptoms (assessed using the Depression, Hostility, and Anxiety subscales of SCL-90-R; Derogatis, 1977; and the Toronto Alexithymia Scale; TAS; Taylor, 1984), relationship change (measured using the Social Adjustment Scale Self-Report; SAS-SR; Weissman & Bothwell, 1976), identity changes (assessed by the Schema Questionnaire; Schmidt, 1994), and repetition of harm, which was measured with a custom scale (the Self-Injury Inventory) that assessed whether subjects had experienced physical or sexual assault as an adult.

The strategy of using multiple measures of largely nonoverlapping symptoms that were not designed to measure CPTSD is problematic for the diagnosis and measurement of CPTSD symptoms. The measures were not designed for this purpose, their boundaries with each other and with CPTSD have not been evaluated, and they were not designed to link symptoms to experiences of traumatic events. The combination of measures also makes it difficult to establish a cutoff or diagnostic decision rules. Thus, the use of a collection of measures of the various facets of CPTSD complicates interpretation of results and comparisons across studies. Efforts to develop instruments to diagnose and measure symptoms of CPTSD are critical to the advancement of a research agenda to establish construct validity.

The Discriminant Validity Of CPTSD

CPTSD Criteria Overlap

Our review indicated significant overlap between the proposed symptoms of CPTSD and PTSD, borderline personality disorder (BPD), and major depressive disorder (MDD). Figure 1 illustrates that most symptoms of CPTSD are also criteria or symptoms of these other disorders. For example, affect dysregulation is not specific to CPTSD; it is a core feature of BPD, PTSD, MDD, and many other Axis I disorders (Kring, 2008), as is functional impairment, which is required for all *DSM-IV-TR* disorders. Symptoms such as hopelessness and feeling ineffective are cognitive features of depression (Beck, 1967). CPTSD also overlaps with some dissociative disorders (Courtois & Ford, 2009). Of course, the problem of symptom overlap is not unique to CPTSD. For example, Kessler et al. (1995) reported that 47.9% of men and 48.5% of women with lifetime PTSD had a lifetime history of at least one major depressive episode. Grant, Beck, Marques, Palyo, and Clapp (2008) demonstrated that PTSD and depression are related but distinct concepts; however, no similar results have yet been reported for CPTSD and depression. More research has been conducted that is relevant to the overlap with BPD and PTSD, which we consider below in greater detail.

Distinctiveness From PTSD

As Figure 1 illustrates, there is significant overlap between symptoms of PTSD and CPTSD. For example, the symptoms of social isolation, irritability/anger, shame, distrust, avoidance, and features of disturbances in consciousness are listed among criteria for PTSD, including proposed *DSM-5* criteria. Problems with memory and concentration are listed as symptoms of dissociation in CPTSD and are core symptoms of PTSD. Finally, dissociation is also included in PTSD (i.e., flashbacks, dissociative amnesia). Little research has been conducted on

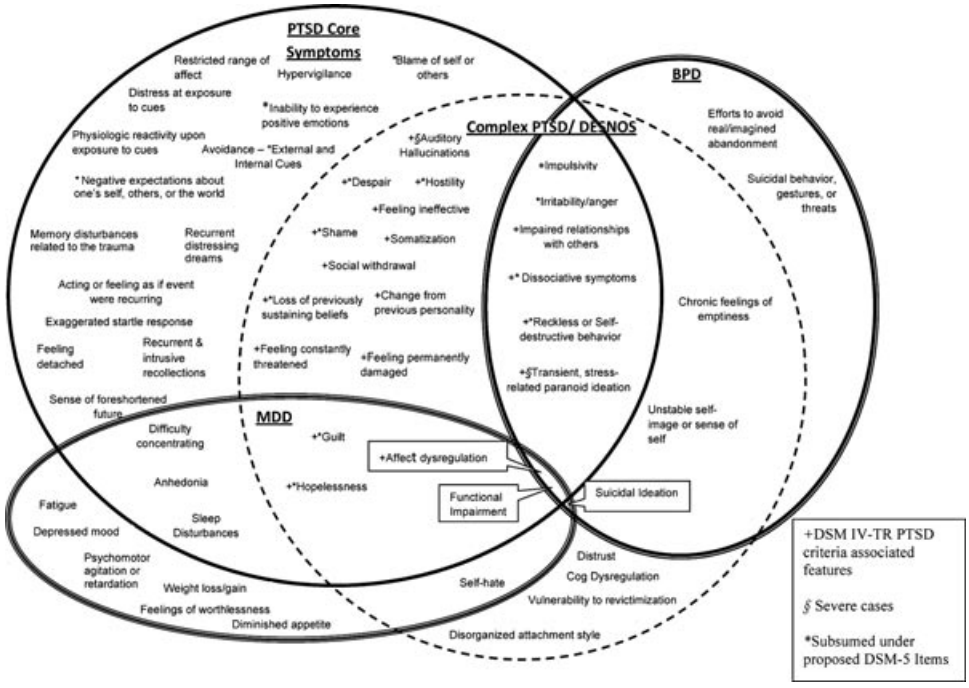

Figure 1. Venn diagram of the overlap between posttraumatic stress disorder (PTSD) core symptoms, PTSD-associated symptoms, disorders of extreme stress not otherwise specified (DESNOS)/complex PTSD, borderline personality disorder (BPD), and major depressive disorder (MDD).

the discriminant validity of CPTSD, i.e., the degree to which it diverges from constructs that are theoretically distinct.

Using the SIDES, Ford (1999) found cases of DESNOS without comorbid PTSD, leading him to conclude that PTSD and DESNOS are qualitatively distinct although often co-occurring. Similarly the CPTSD field trial for *DSM-IV* indicated three symptoms, somatization, dissociation, and affect dysregulation, sometimes occurred independently of PTSD, although were still strongly correlated with it (van der Kolk, Pelcovitz, Roth, & Mandel, 1996). The field trial, however, also found a 92% comorbidity rate between DESNOS and PTSD. This could represent true comorbidity between independent syndromes arising from shared etiologic factors, but empirical work is needed to rule out alternative explanations, including, for example, that CPTSD and PTSD are simply alternate phenotypic expressions of the same disorder process, or that one is a special case of the other (Klein & Riso, 1993).

Other studies have examined the distinctiveness of CPTSD from PTSD. For example, Cloitre, Miranda, Stovall-McClough, & Han (2005) compared symptoms of PTSD versus interpersonal problems and negative mood regulation self-expectancies to determine if problems in the latter two domains in women with histories of childhood abuse were uniquely associated with functional impairment. A hierarchical regression model suggested that interpersonal problems and negative mood regulation expectancies explained an additional 4% and 11% of the variance, respectively, in functioning beyond that attributable to PTSD. Implications of this work, however, with respect to the distinctness of CPTSD from PTSD are limited by several concerns. First, CPTSD was not formally assessed; thus, the extent to which these results are pertinent to CPTSD is unknown. Second, the interpersonal problems and negative mood regulation expectancies measures correlated with each other at $r = -.24$; this weak association would argue against the ability to use these two inventories to assess a broader, cohesive construct, such as CPTSD. Third, the analysis was never reversed (i.e., PTSD was never added as the last step in the regression model) and this makes it impossible to determine if PTSD might explain variance beyond that attributable to these other two symptom dimensions.

Given the established validity of PTSD, we believe that further evaluation of the discriminant validity of the CPTSD construct must come before codifying CPTSD as a diagnosis. If CPTSD and PTSD are not distinct disorders (perhaps instead sharing common underlying dimensions) then introducing the separate diagnosis may impede research efforts aimed at examining the prevalence and course of trauma-related psychopathology, and will complicate clinical decision making (Lilienfeld, Waldman, & Israel, 1994).

Distinctiveness from BPD

Multiple scholars have noted the overlap between CPTSD and BPD, both in terms of symptoms (e.g., impaired interpersonal functioning, impaired sense of self, dissociative symptoms, anger, impulsivity, and self-harm) and theorized causal links to trauma exposure. Some have debated the merits of reconceptualizing BPD as a complex trauma spectrum disorder (Gunderson & Sabo, 1993; Herman & van der Kolk, 1987; Lewis & Grenyer, 2009). In light of this suggestion, one of the

important untested assumptions of CPTSD is that it is distinct from BPD. If it is distinct, trauma exposure (especially early and repeated childhood trauma) should show a stronger magnitude of association with CPTSD than BPD. Complex posttraumatic stress disorder should evidence differential patterns of association with psychosocial correlates (i.e., personality, other diagnoses, coping styles) relative to BPD. For example, BPD should show stronger evidence of other personality pathology relative to CPTSD, while CPTSD should show stronger associations with PTSD relative to BPD. Additionally, CPTSD should differentially predict outcome variables such as response to treatment, functioning and impairment, and quality of life relative to BPD. The longitudinal course of CPTSD should also differ from that of BPD. Complex posttraumatic stress disorder-specific treatment should be necessary for symptom reduction (i.e., treatments originally designed to address symptoms of BPD, such as dialectical behavior therapy [Linehan, 1993], should not be sufficient at reducing CPTSD symptoms). More generally, in predicting important dependent variables such as functioning, quality of life, employability, and response to treatment, CPTSD symptoms should evidence incremental validity over BPD symptoms. Each of these questions await empirical examination. Without this nomological network established, inclusion of CPTSD as a diagnosis risks introducing confusion and redundancy into the diagnostic classification system and may impede research on the constellation of symptoms shared across CPTSD and BPD.

The question of whether CPTSD is likely to be a discrete, categorical construct distinct from BPD can be further explored by examining taxometric evidence. As described, overlap between BPD and CPTSD is primarily in the domains of affective and impulse-control dysregulation and unstable relationships; thus, we can look to these symptoms of BPD as a proxy for CPTSD to evaluate whether they are distributed dimensionally in the population or exist in a discrete subgroup of individuals (i.e., taxon). Current taxometric evidence from *DSM*-based interviews and self-report measures supports a dimensional structure for these symptoms across samples of women and men, psychiatric inpatients, outpatients, and prison inmates (Arntz et al., 2009; Edens, Marcus, & Ruiz, 2008; Rothschild, Cleland, Haslam, & Zimmerman, 2003; Trull, Widiger, & Guthrie, 1990). These findings tend to support a growing consensus that most personality disorders reflect deviation from healthy personality by degree and not by type (Haslam, 2007; Trull & Durrett, 2005), which argues against the existence of discrete disorders with natural boundaries, and to the extent trauma plays a causal role, is consistent with a spectrum of posttraumatic maladjustment.

Importantly, the significant overlap in symptomatology between CPTSD and existing disorders does not in and of itself prove that CPTSD is not an independent entity. As a parallel, the nosology of medical diseases contains many examples of separate classifications of syndromes that symptomatically appear quite similar (e.g., influenza and the common cold). In those cases, however, the classifications are based on empirical evidence that the disease processes differ in etiological agent and/or pathophysiology. With CPTSD, the putative etiological agent is complex trauma, but at this time, it has not been shown to be qualitatively distinct from traumas associated with PTSD. In the absence of unique symptomatology, empirically establishing the uniqueness of the etiology is a necessary precursor to introducing a new diagnosis.

CPTSD Symptoms not Accounted for by Existing Diagnoses

After accounting for symptom overlap with PTSD, BPD, and MDD, two symptoms remain that may set CPTSD apart from other diagnoses: change from previous personality and loss of previously sustaining beliefs, which have received less attention in the literature than other proposed CPTSD features. Prospective research is necessary to understand how such features manifest themselves as a function of trauma history. Particularly with CSA and other childhood traumas, it may be less the case that trauma changes previously held beliefs or personality characteristics, and more that trauma impacts the formation of patterns of behavior and beliefs about the self, world, and others. The concepts of assimilation, overaccommodation, and accommodation have been applied to PTSD (Resick & Schnicke, 1993) in recognition that a traumatic event can alter beliefs (Foa, Cashman, Jaycox, & Perry, 1997) and that these beliefs change as a result of treatment (Owens & Chard, 2003; Resick, Nishith, Weaver, Astin, & Feuer 2002; Resick et al., 2008), but whether qualitatively different alterations to belief systems are experienced by individuals with complex trauma histories has not been established. Resick et al. (2003) compared women with and without a history of CSA on the Impaired Self-Reference Scale of the TSI (Briere, 1995), which measures problems in personal identity and unstable sense of self. Although the CSA group had greater trauma history across a range of events, they did not report differences in impaired self-reference. This lack of an association runs counter to the hypothesis that individuals with CSA histories are more likely than other trauma groups to exhibit these proposed CPTSD symptoms.

Treatment of CPTSD

Other construct issues aside, the clinical utility of CPTSD rests on demonstrating that the diagnosis would make a difference for treatment outcome. Without a uniform definition of CPTSD, and only one measure of DESNOS, the SIDES, which is not anchored to trauma, studying treatment of CPTSD has been challenging. Well-designed clinical trials with appropriate inclusion and exclusion criteria, as well as appropriate measurement and comparisons against standard treatments for PTSD, are needed.

We identified only one study that used CPTSD as an inclusion criterion. Dorrepaal et al. (2010) conducted an open pilot trial of a stabilizing group treatment protocol for CPTSD. Participants included 36 women with a history of childhood abuse

who met criteria for both PTSD and DESNOS (assessed with the SIDES). Participants received concurrent individual therapy during their group participation. At posttreatment, 22% of the sample no longer met criteria for PTSD; at the 6-month follow up, 35% of the sample no longer met criteria. In terms of CPTSD, at posttreatment, 64% no longer met criteria; at the 6-month followup, 78% no longer met criteria.

Another study examined a treatment for CPTSD, although this study did not use CPTSD as an inclusion criterion. Specifically, Zlotnick and colleagues (1997) compared an affect management group to a waitlist control condition; all participants received unspecified individual therapy and pharmacotherapy throughout the duration of the study. Although it was not an inclusion criterion, all individuals met criteria for DESNOS based on the SIDES. The results showed that participants in the affect management group improved more on PTSD and dissociative symptoms than individuals in the waitlist condition.

The results of these two studies suggest that supplemental affect management groups may be somewhat effective in helping to alleviate PTSD and CPTSD symptoms. It is important, however, to note that neither study compared the treatments to either trauma-focused therapies or an active control condition. Furthermore, in both studies, participants received individual unspecified therapy in addition to affective management skills. This is particularly problematic for interpreting the results of the Dorrepaal et al. (2010) study, which did not include a comparison group. It is unclear if participants improved due to the affect management group, individual therapy, or a combination of the two. The absence of well-controlled studies examining the effects of treatment on CPTSD makes it difficult to draw conclusions about appropriate treatments for CPTSD.

A number of other researchers have developed protocols to treat symptoms that may arise from serial traumatization. These include a phase-based protocol (i.e., Skills Training in Affect and Interpersonal Regulation; STAIR) developed by Cloitre and her colleagues (Cloitre, Koenen, Cohen, & Han, 2002; Cloitre et al., 2010) that was designed to treat symptoms that develop in individuals who experienced childhood abuse. The Attachment, Self-Regulation, and Competency (ARC) protocol, was developed to be used with severely traumatized children and adolescents (Kinniburgh, Blaustein, Spinazzola, & van der Kolk, 2005). Narrative Exposure Therapy (Schauer, Neuner, & Elbert, 2005) has been used to treat symptoms that present in asylum seekers and refugees (Robjant & Fazel, 2010). Others have attempted to use existing treatments for PTSD (e.g., Cognitive Processing Therapy; CPT) for CPTSD symptoms (Chard, 2005). These treatments have shown promise in reducing symptoms of PTSD and other trauma-related symptomatology. None of these studies, however, used CPTSD as an inclusion criterion, nor did they explicitly assess CPTSD (i.e., they did not use the SIDES.) Further, although several of these studies used measures that presumably capture some of the symptoms of CPTSD (e.g., Cloitre et al., 2002, 2010; Resick et al., 2003), without employing cut points to distinguish between individuals with and without CPTSD the percentage of their samples who met criteria for CPTSD could not be computed. Therefore, these studies do not provide clear evidence of treatments that are effective specifically for individuals diagnosed with CPTSD. Additional research that explicitly examines these treatments with a population of CPTSD diagnosed individuals is necessary before such claims can be made.

Conclusions

Before making our concluding statements, we would like to explicitly acknowledge that those authors who have proposed a CPTSD diagnosis have brought attention to important unresolved issues regarding adaptation following trauma exposure. First, we appreciate the importance of attempting to understand the heterogeneity in posttraumatic psychological distress. A single diagnosis (i.e., PTSD) cannot adequately capture this heterogeneity, and more research is needed to better account for the heterogeneity within and beyond PTSD. Research examining aspects of CPTSD, from etiology to symptomatology, have also helped elucidate many mechanisms contributing to the very complex and dynamic processes underlying all forms of posttraumatic adaptation, from resilience and recovery to severe and chronic psychological distress. For instance, Ford (2009) provided a sophisticated review of neurobiological processes that are impacted by repeated-trauma exposure early in life. This review elegantly illustrates how brain systems underlying emotion regulation, information processing, healthy attachment, and the development of interpersonal relationships are affected by early and repeated exposure to trauma. This work is important and will contribute to the development of more sophisticated biological/neural models of posttraumatic distress (e.g., Garfinkel & Liberzon, 2009; Suvak & Barrett, 2011). Although important and informative, Ford's review did not include any studies of individuals diagnosed with CPTSD.

We are not aware of any studies that have examined neurobiological mechanisms in individuals diagnosed with CPTSD. Therefore, the extant neurobiological literature is limited in what it can say about CPTSD providing a "coherent formulation of the consequences of prolonged and repeated exposure" (Herman, 2009, p. xiii). Understanding the neurobiological mechanisms contributing specifically to CPTSD requires a concise and coherent formulation and reliable and valid means of assessment. Once this formulation is developed and operationalized, neurobiological investigations can be conducted to help develop a nomological network of research support for the construct validity of CPTSD.

Second, Herman (1992a) brought much needed attention to the social and political influences that impact how the field, and society more generally, conceptualizes responses to trauma. For instance, today there is a tendency for PTSD to be thought as of the "legitimate" response to trauma, whereas diagnoses such as BPD and substance abuse disorders, which are often the result of trauma, are often thought of as deficits of personality or character. Third, discussions of CPTSD have brought attention to

developmental issues related to trauma and we applaud trauma research that adopts this developmental, life span perspective. We hope that this critical review of the CPTSD construct as a psychiatric diagnosis will promote more sound scientific research that addresses these very important issues.

Implications for *DSM-5*

The variety of untested assumptions, existing literature, and dearth of new research on the validity of the DESNOS or CPTSD since the implementation of the *DSM-IV* led us to conclude that there is insufficient evidence to warrant the addition of a CPTSD diagnosis in the *DSM-5.* The *DSM-5*, however, is proposing to add to the PTSD diagnosis symptoms that have frequently been viewed as falling in the range of CPTSD: distorted beliefs about self and others, erroneous blame of self and others, dissociation, reckless behavior, and the full range of negative emotions. As with the *DSM-IV*, functional impairment, including interpersonal functioning, is included.

One untested assumption is the degree to which adoption of the CPTSD diagnosis would bring about greater parsimony to the diagnostic nomenclature. In one view, it would be simpler if an individual were diagnosed with a single disorder (i.e., CPTSD) as opposed to multiple disorders (i.e., PTSD, BPD, and major depression). On the other hand, the introduction of a complex variant of PTSD that shares such significant symptom overlap with other diagnoses does not seem to be parsimonious in solving problems in the classification of mental disorders, because this would add to diagnostic confusion and limit diagnostic reliability. Brett (1996) argued for the need for a more comprehensive list of symptoms that would include the DESNOS/CPTSD symptoms on the grounds that the PTSD criteria "are often used by clinicians as if they were complete descriptions of mental disorders" and because "a clinician may miss the PTSD diagnosis because associated features are more prominent, or the associated features may be overlooked because of the presence of the PTSD" (p. 125). This appears, however, to be a problem that could occur with any disorder and is not specific to PTSD. Better training of clinicians would be more efficient than a change in the diagnostic system.

The magnitude of establishing a new CPTSD diagnosis is reflected by the fact that there is no precedent in the established diagnostic systems for splitting off a more severe form of any disorder. Despite variations in symptom presentations for disorders such as depression and schizophrenia, there is no separate diagnosis of complex MDD or complex schizophrenia. Importantly, though, these diagnoses do include specifiers that reflect important differences in course and symptom presentation. As such, in addition to research on CPTSD as a separate construct, investigation of a possible complex specifier may also be warranted.

A dissociative subtype of PTSD is under consideration for the *DSM-5* due to a convergence of epidemiological, physiological, brain imaging, and treatment study differences between those with severe PTSD with and without dissociation. There is growing consensus that individuals with severe PTSD who dissociate may reflect a discrete group or subtype of individuals with PTSD (Griffin, Nishith, Resick, & Yehuda, 1997; Lanius et al., 2010; Putnam, Carlson, Ross, & Anderson, 1996; Waelde, Silvern, & Fairbank, 2005; Wolf et al., in press) who exhibit a distinct neurocircuitry marked by over-modulation of brain regions governing emotion (Lanius et al., 2010). Such work also suggests that individuals who dissociate may respond differently to PTSD treatment (Cloitre et al., 2012; Resick, Suvak, Johnides, Mitchell, & Iverson, in press). The dissociative subtype (which is a much narrower construct than that of CPTSD) is under consideration for inclusion in the *DSM-5* in an effort to better capture the heterogeneity in the clinical presentation of PTSD. To our knowledge, no study has empirically evaluated the evidence for a similar CPTSD subtype of PTSD. The CPTSD literature would benefit from a similar series of evaluations to determine the nature of its relationship to PTSD and its clinical utility with respect to characterizing distinct responses to trauma.

Complex Posttraumatic Stress Disorder as a Trauma Spectrum Disorder

Even in concluding there is currently insufficient evidence to consider CPTSD a distinct diagnostic category, we do not dismiss or marginalize the putative CPTSD clinical phenomena that are not captured by *DSM-IV-TR* or even proposed *DSM-5* PTSD nosology. We suggest, however, that efforts to explore the structure and boundaries of these phenomena should consider that they may not constitute a discrete disorder at all, but instead the product of extremes on one or more underlying dimensions, perhaps the same dimension(s) underlying PTSD, BPD, and other overlapping conditions. Although proper CPTSD structural work must await the development of reliable and valid measures that can produce a robust factor structure, as an approximation it is noteworthy that pathological reactions to trauma included in PTSD have been found better characterized as a dimension of symptomatic severity rather than a discrete category (Broman-Fulks et al., 2006, 2009; Forbes, Haslam, Williams, & Creamer, 2005; Ruscio, Ruscio, & Keane, 2002). One implication is that PTSD likely has a multifactorial etiology, as latent dimensions are thought to be produced by the small additive effects of multiple risk and protective factors (Meehl, 1992). Indeed, meta-analyses indicate that the specific traumatic stressor is not the only determinant of posttraumatic maladjustment (Brewin et al., 2000; Ozer et al., 2003). Therefore, unless and until complex traumas are shown to have qualitatively different causal effects, the working hypothesis that complex posttraumatic symptomatology also falls on a continuum seems plausible. A dimensional structure for CPTSD would also be more consistent with growing evidence that a small number of internalizing psychopathology dimensions can explain an array of *DSM* categorical diagnoses, including anxiety and mood disorders and BPD (Kotov et al., 2011; Krueger,

1999; cf. Watson, 2005). The dimensional hypothesis seems to warrant serious attention by CPTSD researchers.

Summary

There is need for a great deal of research on all aspects of CPTSD to justify it as a psychiatric diagnosis. First and most important, there is need for a uniform definition of the proposed construct, which is necessary, but not sufficient, for demonstrating that the construct is distinct from other diagnoses, or that CPTSD has a unique etiology. The development of measures that can reliably and validly assess the severity of symptoms of CPTSD is a critical next step. As our review demonstrates, it is important to clearly establish that CPTSD is a separate construct rather than a more severe form of PTSD before it can be recognized as a distinct diagnosis. Additionally, before establishing a CPTSD diagnosis, the incremental clinical benefit of doing so must be established. Many clinical trials have included people who would potentially meet the definition of CPTSD in terms of symptoms and who have complex trauma histories. Many of these individuals have appeared to respond to single-phase treatments that are effective for those with PTSD (e.g., Chard, 2005; Resick et al., 2003). Better characterization of the samples, comparisons of CPTSD treatments with other treatments typically thought of as PTSD treatments (e.g., CPT, PE), and analyses to determine whether CPTSD symptoms improve after treatment are essential to determining whether different treatments are indicated for individuals with CPTSD.

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