Psychological Trauma: Theory, Research, Practice, and Policy
2012, Vol. 4, No. 6, 551–559

1942-9681/12/$12.00 DOI: 10.1037/a0027900

Dissociation in Posttraumatic Stress Disorder Part II: How Theoretical Models Fit the Empirical Evidence and Recommendations for Modifying the Diagnostic Criteria for PTSD

Constance Dalenberg
Trauma Research Institute, CSPP,
Alliant International University

Eve B. Carlson
VA National Center for Posttraumatic Stress Disorder,
Menlo Park, California

The purpose of this article is to examine how closely six theoretical models fit the empirical evidence reviewed in Part 1 on the relationship between trauma and dissociation and to apply the resulting conclusions to make recommendations about diagnostic criteria for PTSD. We describe six models for the relationships among traumatic stress, dissociation, and PTSD, including models for fantasy-proneness, mediation, comorbidity, interactional comorbidity, component, and subtype. The research reviewed strongly supports component and subtype models of the relationship between dissociation and PTSD, and recommendations are made for including dissociative symptoms in the diagnostic criteria for PTSD in ICD-11 and DSM-5 and adding either a dissociative subtype or a complex PTSD diagnosis in both diagnostic systems.

Keywords: dissociation, trauma, posttraumatic stress disorder, DSM-5, ICD-11

At the time of the preparation of the *Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition* (*DSM–IV*) and ICD-10 in the early 1990s, the idea that dissociation could be a psychological response to traumatic stress had already been discussed in the literature for more than a century (van der Hart & Dorahy, 2009), and clinicians had begun to write and teach about the clinical importance of dissociation as a response to traumatic stress (Briere, 1992; Courtois, 1988; Herman, 1992; Putnam, 1985; Spiegel, 1986; Spiegel & Cardena, 1990; van der Kolk, 1987; van der Kolk, Brown, & van der Hart, 1989; van der Kolk & Van der Hart, 1989). The diagnostic criteria for PTSD in *DSM–IV* included two symptoms that could be considered dissociative: gaps in memory and behavioral or emotional reexperiencing. It did not, however, include a number of other types of dissociative symptoms that are also considered responses to trauma (Ginzburg, Butler, Saltzman, & Koopman, 2009; Steele, van der Hart, & Nijenhuis, 2009; Waelde, Silvern, Carlson, Fairbank, & Kletter, 2009). In the clinical literature, dissociative symptoms have been viewed as responses to trauma that could be extremely distressing for trauma survivors (Briere, 1992; van der Kolk, Pelcovitz, Roth, & Mandel, 1996), These symptoms can interfere with functioning, put clients at risk for further exposure to trauma, have important psychological functions, provide information about clients' central clinical issues, and impede the therapeutic process (Briere, 1992; Courtois, 1988; Foa, Hearst-Ikeda, Michelson, & Ray, 1996; van der Kolk, van der Hart, & Marmar, 1996).

The earliest theories to explain why trauma causes dissociative experiences were published by Janet in the late 1800s. Janet treated patients with very severe and pervasive dissociative symptoms and believed that traumatic experiences could exacerbate divisions or fragmentations of personality or consciousness in those with a lack of capacity for integrated functioning (van der Hart & Dorahy, 2009). Around the same time, before his psychoanalytic theory began to focus on instinctual drives, intrapsychic conflict, and defensive repression, Freud proposed that dissociation was a psychological defense against intense affect related to childhood trauma, particularly sexual abuse (Freud, 1962). Later explanations of how dissociation relates to trauma drew upon both of these models, emphasizing how dissociative symptoms function to provide emotional, cognitive, physiological, and behavioral distance from overwhelming affect for individuals exposed to traumatic stress (Putnam, 1985; Spiegel & Cardena, 1990; van der Kolk, 1987; van der Kolk & Van der Hart, 1989).

Now that a large body of research is available that addresses questions about the relationship between trauma and dissociation, it is possible to examine the fit between theories describing the relationship and findings from empirical work. A number of different models could explain the relationships among traumatic stress, dissociation, and PTSD, each of which is associated with a distinct path model and set of statistical predictions. Below, we review these models and discuss whether the research findings presented in Part 1 (Carlson, Dalenberg, & McDade-Montez, 2012) are consistent or inconsistent with each model. We represent the models in their most fundamental forms for the purpose of evaluating the fit between empirical findings and basic predictions for each model, but we recognize that the true relationship

This article was published Online First May 14, 2012.

Constance Dalenberg, Trauma Research Institute, CSPP, Alliant International University; Eve B. Carlson, VA National Center for Posttraumatic Stress Disorder, VA Palo Alto Health Care System, Department of Veterans Affairs, Menlo Park, California.

Correspondence concerning this article should be addressed to Constance J. Dalenberg, Alliant International University, 10455 Pomerado Road, San Diego, CA 92131. E-mail: cdalenberg2@alliant.edu

between trauma, dissociation, and PTSD may be considerably more complex and include elements of more than one model. It is also possible that different models explain dissociation in different people. Graphic representations of the models are provided in Figure 1. Table 1 shows 12 empirical findings about the relationship between dissociation and PTSD and an indication for each finding of whether the model is consistent or inconsistent with the finding.

Model 1: The Fantasy Proneness Model

According to the Fantasy Proneness Model (Model 1 in Figure 1), some people who are characterologically prone to fantasize and dissociate tend to exaggerate the events they were exposed to or fantasize that they have had traumatic experiences (Dalenberg et al., 2012). A detailed review of predictions and research findings relating to this model is available in Dalenberg et al. (2012). According to this model, retrospective memory for trauma is related to dissociative experiences, but the relationship between actual trauma and dissociation is "weak or nonexistent" (Giesbrecht, Lynn, Lilienfeld, & Merckelbach, 2008). A moderate correlation between PTSD and dissociation is compatible with the model, mediated through a tendency to exaggerate both disorders, but no strong predictions are made as to the relative strength of the PTSD-trauma or PTSD-dissociation relationships.

If the fantasy-proneness model were accurate, the findings related to research questions 1, 2, 3, 4, and 5 reviewed in Part 1 could be the result of a small group of highly dissociative persons in each sample who made erroneous reports of high levels of trauma exposure and high levels of PTSD symptoms. Such a group of outliers could create the appearance of a relationship by inflating correlation coefficients and raising the means for trauma-exposed groups. This explanation is not supported by examination of scatterplots of data in Figure 2 of Part 1 from samples showing that correlations between dissociation and PTSD symptoms reflect shared variance at all levels of dissociation. In addition, the relationship between dissociation and trauma exposure and between dissociation and PTSD symptoms is observed even in samples of persons with relatively low levels of dissociation. Similarly, the findings of elevations in dissociation after trauma exposure in nonclinical samples and the subsequent decline over time (shown in Table 3 of Part 1) do not support the fantasy-proneness model. The model makes no prediction about the relationship between fantasy-proneness and trauma exposure; therefore, there is no prediction about the relationship between dissociation and trauma. Lastly, the fantasy-proneness model would not predict that dissociation and PTSD would be strongly related; therefore, it is inconsistent with the findings of a strong relationship between dissociation and PTSD symptoms presented in Table 5. The plus signs, minus signs, and blank cells for this model in Table 6 indicate that, overall, the research reviewed provides very little support for the fantasy-proneness model of the relationship between trauma and dissociation, and the model does not account for most of the empirical findings reviewed.

Model 2: The Mediation Model

In the mediation model (Model 2), dissociation is a necessary prerequisite to PTSD. Researchers using this model typically assess PTSD in a sample (often of people who have recently experienced a trauma) and show that dissociation increments over prior trauma history in prediction of PTSD. Studying metropolitan New Yorkers about six months after 9/11, Twaite and Rodririguez-Srednicki (2004) found that attachment and dissociation had "mediating effects" on "vulnerability to PTSD" (p. 17). Prior sexual and physical abuse predicted PTSD, but when dissociation and attachment were entered into the model, the beta weight for trauma became nonsignificant. This could, however, be the result of inadequate measurement of trauma severity, which is extremely difficult to quantify.

Some studies have produced results that are consistent with a partial mediation model. In a prospective study of police trainees, both prior trauma and dissociation *before* exposure to stressful incidents experienced as a police officer predicted both dissociation and PTSD symptoms after one year of police work (McCaslin et al., 2008). A study of university women that examined the relationship among dissociation, trauma, and PTSD found that dissociation predicted PTSD and that trauma accounted for additional variance in PTSD once dissociation had already been entered into the model (Gershuny, Najavits, Wood, & Heppner, 2004). It should be noted, however, that the statistical outcome of "partial" mediation does not distinguish between models and could be consistent with any of the models.

Other research that is relevant to this question examined whether there is a dissociation taxon within PTSD. In the Waelde et al. study (described in Part 1), 18% of those in the taxon-negative group had PTSD. Similarly, studies of those with PTSD in general population samples can shed light on whether some with PTSD have low dissociation. In an adult community sample described in detail elsewhere (Carlson, Smith et al., 2011), for example, 15.6% of those with normal or low levels of dissociation had levels of PTSD that were elevated more than 1.5 SDs above the norm (Smith & Carlson, 2011). Because it appears that some with PTSD do not show high levels of dissociation, based on the research reviewed above, a Mediation Model—at least in its strong form—is not supported. Indicators in Table 6 of research findings that were consistent and inconsistent with the Mediation Model indicate weak support for the model, overall.

Model 3: The Comorbidity Model

The Comorbidity Model is the least compatible with the extant data on PTSD and dissociation. The term "cormorbidity" is applied quite loosely in the general clinical and research literature, and at times appears to refer only to the fact that the two disorders often occur together. Thus, many research reports on PTSD refer to the Axis I comorbidity of PTSD with dissociative disorders or dissociative experiences, or the "psychiatric comorbidity" of PTSD and dissociation or dissociative disorders, just as they refer to the "comorbidity" of PTSD and Major Depression.

The source of the comorbidity of two disorders may be an external event, a shared vulnerability, or both. However, researchers addressing comorbidity in PTSD appear to share only a view that trauma might initiate both dissociative and PTSD responses. In pure form, the comorbidity model "posits that traumatic dissociation and PTSD are merely commonly co-occurring nosological entities sharing a necessary prerequisite broadly conceptualized as a traumatic event" (Simeon, 2007, p. 77). The two entities may

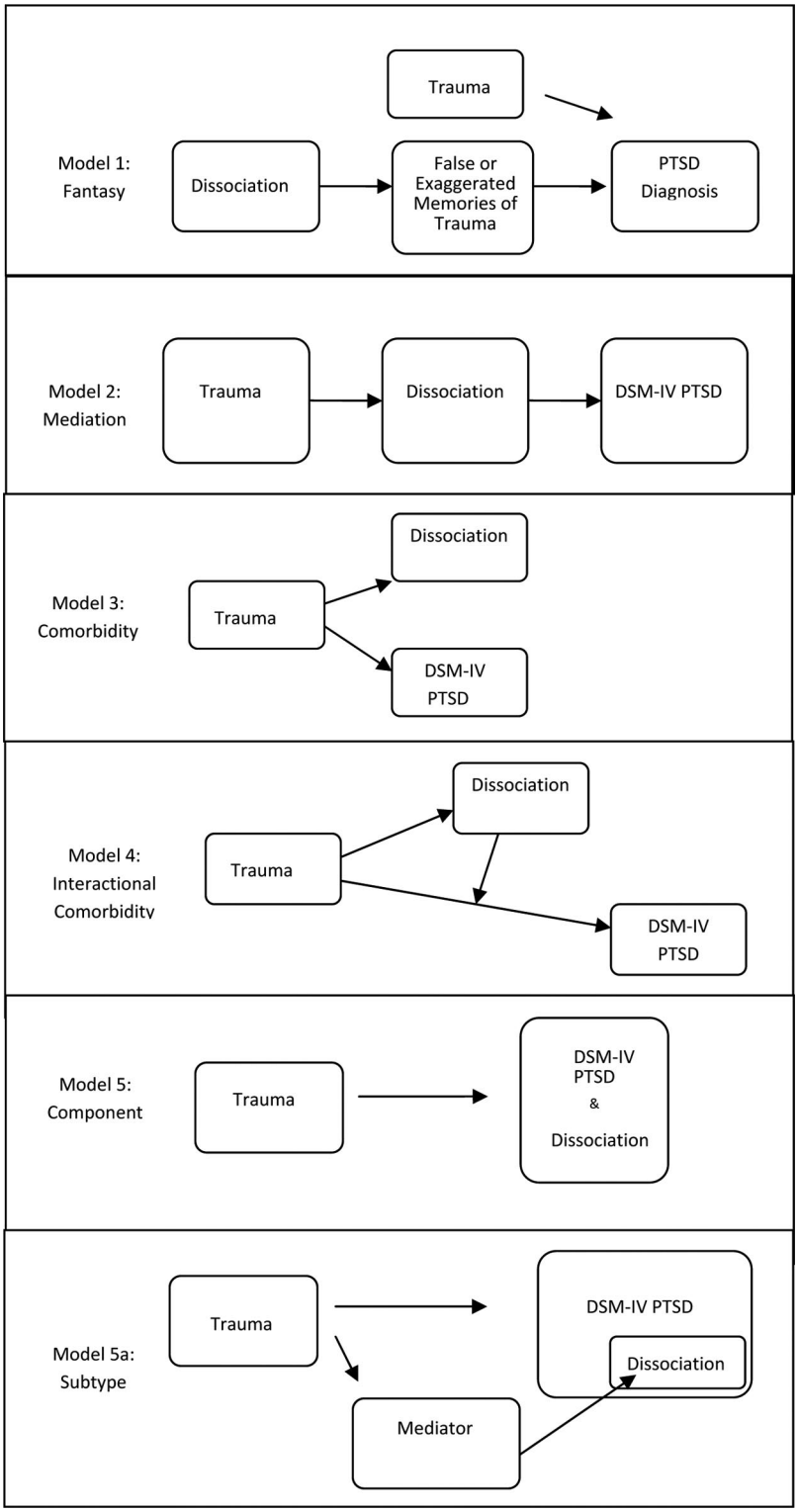

Figure 1. Models of relationships among trauma, dissociation, and PTSD.

co-occur essentially by chance, and the correlations between them may reflect only an artifactual relationship produced by the shared relationship to a third variable (trauma exposure) (Simeon et al., 2007).

The Comorbidity Model predicts that the Dissociation-PTSD relationship will be very weak compared with the strength of the Trauma-Dissociation and Trauma-PTSD relationships. As is shown in Tables 2 and 5 in Part 1, however, the Dissociation-

Table 1
Empirical Findings and Expected Relationships to Theoretical Models for the Relationship Between Dissociation and PTSD

	Fantasy (Model 1)	Mediation (Model 2)	Comorbidity (Model 3)	Interactional comorbidity (Model 4)	Component (Model 5)	Subtype (Model 5a)
1. Dissociation is elevated above norms in populations who have experienced objectively identified trauma and correlates with experience and severity of trauma.	−	+	+	+	+	+
2. Dissociation can be linked in time with an experience of trauma for samples of individuals, showing a pattern of increase after traumatic experience followed by a gradual decrease for most individuals.	−	+	+	+	+	+
3. Dissociation is strongly related to PTSD.	−	+	−	+	+	+
4. Dissociation is related to PTSD within traumatized populations.				+	+	+
5. The presence of dissociation moderates the relationship between PTSD and trauma, such that the relationship between PTSD and trauma is greater for dissociative than for nondissociative individuals.				+		+
6. The relationship of dissociation and PTSD is greater than the relationship of trauma and dissociation.		−	−		+	+
7. The relationship of dissociation and PTSD is greater than the relationship of trauma and PTSD.		+	−		+	+
8. Dissociation increments over trauma in the prediction of PTSD either as a main effect or interactionally.		+	−	+	+	+
9. Trauma increments over dissociation in the prediction of PTSD.	+	−	+	+		+
10. PTSD can occur in the absence of dissociation.	+	−	+	+	+	+
11. The relationship between dissociation and the symptom clusters of PTSD is as strong as the relationship of the clusters of symptoms to each other.		–		–	+	+
12. Dissociative PTSD survivors will show a qualitatively different form of PTSD than will nondissociative PTSD survivors.						+

Note. + = consistent with model; − = inconsistent with model; blank = irrelevant to the model, could be true or false if model is correct.

PTSD relationship (mean $r = .71$) is decidedly stronger than the Trauma-PTSD (mean $r = .39$) *and* the Trauma-Dissociation (mean $r = .34$) relationships. Overall, Table 1 shows that the empirical findings show very poor support for the Comorbidity Model.

Model 4: The Interactional Comorbidity Model

The Interactional Comorbidity (IC) Model, like the Component Model, posits that trauma independently sets in motion reactions that culminate in PTSD and in dissociative symptoms. According to the IC Model, the type of PTSD that emerges after trauma in the presence of dissociation is *different*, perhaps more severe or sharing different dissociative and nondissociative features. Unlike the Mediation and Comorbidity models, the IC Model allows for the possibility that PTSD and dissociation would relate within traumatized survivors, because the dissociative PTSD victims would present with a more severe form of PTSD than would nondissociative survivors. Thus, the strong correlations between PTSD and dissociation within trauma samples (Table 5, part I), are predicted by the IC Model, but not the Mediation or Comorbidity Model. However, the finding that dissociation is as strongly related to the components of PTSD as they are to each other (discussed under Question 7, Part 1), are not positively predicted by IC Model.

The Interactional Comorbidity (IC) Model is consistent with the emerging findings showing that early childhood developmental trauma results in dissociation proneness (Ogawa, Sroufe, Weinfield, Carlson, & Egeland, 1997). Dissociation proneness is then hypothesized to create a vulnerability for future pathological trauma responding. This conceptualization contrasts with the fantasy proneness model, reversing the hypothesized order of trauma and dissociation proneness and proposing that early trauma precedes dissociation proneness, rather than dissociation proneness leading to erroneous reports of early trauma. In Ogawa et al.'s regression analysis, disorganized attachment and psychological unavailability of the caregiver in infancy accounted for approxi-

Table 2
Levels of Dissociation Across Trauma Exposures

Modes of experience	Reexperiencing	Avoidance
Cognitive sensory/perceptual	Trauma-related cognitive or sensory intrusions	Derealization; depersonalization; gaps in awareness & memory
Affective	Anxiety; anger	Emotional numbing
Behavioral	Aggression; escape behaviors	Intentional avoidance behaviors
Physiological	Physiological arousal	Analgesia

mately one quarter of the variance in dissociation at age 19. This diathesis-stress model is proposed by Lyons-Ruth (2003) and other developmental theorists (e.g., Dutra, Bureau, Holmes, Lyubchik, & Lyons-Ruth, 2009) who point to poor maternal affective involvement (rather than maltreatment) as the most common dissociation-relevant developmental trauma. Similarly, Briere (2006) hypothesizes that dissociative symptoms occur after trauma exposure only when emotion regulation capacity is weak. If the IC Model is accurate, then those who are high in dissociation after early attachment problems would be at higher risk for poor prognosis or greater numbers of comorbid problems. A recent study of a general population proposed a variant of an IC Model in which the relationship between trauma and dissociation was mediated by PTSD and by affect dysregulation (Briere, Hodges, & Godbout, 2010). Analysis of data showed that dissociation is part of a latent construct of dysfunctional avoidance that includes tension reduction behaviors, substance abuse, suicidality, and dissociation. Structural equation modeling analysis of data from a general population sample showed support for the mediation parts of the model, but the interaction between PTSD and affect dysregulation was not found to be significant. These findings indicate that there may be two pathways to dissociative symptoms that do not necessarily interact. In summary, as shown in Table 1, the IC Model was more supported than the previous models, but fails to explain a number of findings.

Models 5 and 5a: The Component and Subtype Models

The two models most consistent with the findings in the research reviewed above are the Component Model and the Subtype Model of PTSD, represented in Figure 1 by Model 5 and Model 5a. The Subtype Model is a variant of the Component Model in that both models conceptualize dissociation as one component of the response to traumatic stress. In the Component Model, dissociation symptoms of PTSD include both reexperiencing symptoms (such as trauma-related sensory misperceptions, trauma-related voices, or acting or feeling as if an event were reoccurring) and distancing symptoms (such as derealization, depersonalization, and gaps in awareness and memory). Like other PTSD symptoms, such as problems with sleep, hypervigilence, concentration difficulties, or startle response, dissociative symptoms are not necessarily present in all cases, but are increased in likelihood if PTSD is present.

In previous work, we have proposed a component model for responses to traumatic stress that includes dissociative symptoms and symptoms of PTSD that are currently included in the diagnostic criteria (Carlson & Dalenberg, 2000). Responses to traumatic stress, including dissociation, are included in our conceptual framework (shown in Table 2). When humans are exposed to events that are sudden and subjectively perceived as being uncontrollable and having a high negative valence, they will have responses across cognitive, affective, behavioral, and physiological modes of experience. When confronted with danger, animals may have a basic response that includes fear and aggression (the "fight or flight" response). A third response of immobility (sometimes described in animals as "freeze") is hypothesized in humans to include cognitive experiences that have been labeled dissociative, such as derealization, depersonalization, and gaps in awareness and memory. Very likely, freeze responses occur when an animal is overwhelmed with fear and function to distance the individual from the threatening experience. A similar theoretical framework has also been proposed that includes additional responses of "fright," "flag," and "faint" to further explain dissociative responses occurring at the time of life threat and reexperienced later (Schauer & Elbert, 2010). In those who recover, the fight, flight, or freeze responses gradually fade or become less salient and no longer interfere with functioning. In those who fail to recover, stimuli associated with the traumatic event (including places, people, situations, sensory perceptions, and semantic elements) become conditioned stimuli that continue to elicit responses similar to those at the time of trauma. These conditioned responses constitute the reexperiencing and hyperarousal symptoms of PTSD. Avoidance in all four modes of experience may reflects either intentional efforts or behaviors with no conscious motivations to reduce distress through distancing. Both types of avoidance increase as a result of their reinforcement properties. In other words, the relief provided by intentional or unconscious efforts to avoid reminders of trauma tend to reinforce the efforts, and they increase in frequency. Table 2 shows a matrix of posttraumatic reexperiencing of reexperiencing and avoidance phenomena and includes all symptoms for PTSD included in the *DSM–IV*. Types of dissociative experiences included in the matrix, but not included in the *DSM–IV* PTSD symptom criteria include gaps in awareness, depersonalization, derealization, and somatoform dissociation. It is worth noting that most of the symptoms listed are experienced as nonvolitional with the exception of some behavioral avoidance. Furthermore, the characteristic of operating outside of conscious awareness that is associated with dissociative experiences is not specific to dissociation—many symptoms of PTSD (such as intrusive thoughts, physiological arousal, and distress in response to reminders) are also not experienced as under the conscious control of the individual.

The physiological phenomenology of responses to trauma is hypothesized to be consistent with the psychological phenomenology. That is, when survivors have intrusive thoughts related to a

traumatic stressor, they will feel anxious and be physiologically aroused, and their brains will show increased activity in the regions associated with fear (e.g., the amygdala). When survivors are actively avoiding or experiencing dissociative avoidance, they will feel emotionally distanced from threat, be physiologically numb, and their brains will show increased activity in regions associated with emotion regulation (e.g., anterior cingulated cortex). A review of physiological and neuroimaging studies focused on dissociation is available in Dalenberg et al. (2012). We believe the physiological responses in the body and brain follow, rather than precede, the cognitive and emotional experiences of trauma survivors. The accuracy of this formulation has just recently become the subject of research in which the cognitive, emotional, and physiological responses of survivors are assessed in real time as they react to trauma reminders (Lanius et al., 2010).

In the Component model, because dissociation is conceptualized as part of the disorder itself, the pattern of levels of dissociation in various disorders shown in Figure 1 in Part 1 is predicted. A Component Model also predicts that dissociative symptoms will explain variance in PTSD over and above trauma exposure and will correlate with PTSD both in nonclinical samples and within traumatized subpopulations. The findings that dissociation is as strongly associated with the reexperiencing, avoidance, and hyperarousal clusters (or other components) of PTSD as they are with one another (Question 7 in Part 1) also strongly support the Component Model. Similarly, as shown in Table 3 of Part 1, dissociation appears to be a clear and consistent traveling companion to the other symptoms of PTSD in that it increases immediately after trauma and slowly dissipates after trauma.

A unique prediction of the Subtype Model is that at a high level of dissociation would change the phenomenology of PTSD so that high dissociation would be associated with a different *pattern* of PTSD symptoms or other clinically relevant characteristics or comorbid symptoms. In fact, high correlations between PTSD and dissociation symptoms in the studies shown in Table 5 of Part 1 does provide support for the premise that those with high dissociation have substantially higher PTSD symptoms than those with low dissociation. Furthermore, there is some evidence that those with high dissociation have higher levels of comorbid symptoms than those with low dissociation. In a sample of psychiatric inpatients, those who were highly dissociative showed significantly higher probability of self-harming behaviors, suicidality, and disordered eating than those who were not (Carlson, McDade-Montez, Armstrong, & Loewenstein, 2011). However, because severe comorbid behaviors were also significantly associated with PTSD symptoms, it remains unclear whether these patients are best conceptualized as a subtype of PTSD or the high end of the continuum of PTSD severity.

Further evidence supporting the Subtype Model comes from analyses of the Carlson and Putnam (1993) data shown in Figure 1 of Part 1. In nine diagnostic categories and two general population samples, the high means for dissociation in those with trauma-related disorder did not result from elevations in dissociation in *all* patients in the diagnostic categories, but reflected differences in the proportion of patients in each group with very high dissociation (Putnam, Carlson, Ross, & Anderson, 1996). In the sample of those with PTSD, 46.5% of the patients had high scores on a dissociation scores compared with 75.9% of those with dissociative identity disorder, 25% of those with borderline personality disorder, 20% of those with schizophrenia, 7.2% of those with other anxiety disorders, and 4.4% of those from a nonclinical general population sample. The very high proportion of the PTSD sample who had high levels of dissociation is striking, although it may not be representative of all PTSD patients. This particular sample was of combat veterans with chronic PTSD, and a larger proportion of this group may have high dissociation than groups with PTSD that is less severe and chronic. Still, the finding that almost half of the PTSD sample had high levels of dissociation suggests that there may be fairly high base rates for a dissociative subtype of PTSD in some clinical settings.

One taxometric study of dissociation in PTSD is also relevant to the Subtype model. As described in the section on Question 6 in Part 1, one study has investigated the possibility of a dissociative taxon within a sample of veterans exposed to traumatic stress in combat. The findings supported the existence of a subtype of PTSD with severe PTSD and elevated dissociation, and 32% of those with PTSD in their sample were taxon members (Waelde, Silvern, & Fairbank, 2005). Two studies of PTSD symptoms in trauma-exposed samples concluded that they were dimensional, but these findings do not have a definitive bearing on the question of whether a taxon defined by high dissociation exists (Forbes, Haslam, Williams, & Creamer, 2005; Ruscio, Ruscio, & Keane, 2002).

Recent neuroimaging findings also support the hypothesis that there are important differences between trauma survivors with regard to dissociation. As described under Question 8 in Part 1, a number of studies have found a distinctive pattern of brain activity associated with dissociation in response to trauma reminders in those with PTSD. This finding was observed across trauma types and did not appear to be related to severity of PTSD. Lastly, a recent latent class analysis of data from a sample of veterans with combat-related PTSD and their partners identified a subset of 12% of the sample who endorsed three dissociation items and may constitute a subtype (Wolf et al., 2012). Overall, the predictions made by both the Component Model and the Subtype Model were strongly supported by the empirical evidence as shown in Table 6. The Subtype Model was the most effective in explaining all of the empirical findings reviewed, although there are relatively few studies that address the predictions for items 5, 9, 10, and 12 in Table 6.

Several theoretical frameworks for explaining how trauma relates to dissociation are consistent with component or subtype models. The theory of trauma-related structural dissociation elaborates on Janet's work and integrates theory and research from the fields of neurobiology, learning and memory, ethology, and developmental psychopathology (Steele et al., 2009). According to this theory, trauma causes emotional aspects of personality to separate from the core personality as a means of protecting it (Steele et al., 2009). Other theoretical work has focused on concepts from evolutionary biology and ethology and characterized dissociative symptoms as adaptive and defensive responses to inescapable threat (Perry, 2008; Schauer & Elbert, 2010). One psychobiological theory of traumatic stress characterizes PTSD as a disorder of affect regulation and proposes that negative symptoms of dissociation (such as derealization, depersonalization, gaps in awareness and memory) reflect excessive inhibition of affective arousal that accompanies immobilization in the face of inescapable threat (Frewen & Lanius, 2006). This theory explicitly proposes

that only a subset of those who are trauma-exposed have a dissociative response to trauma reminders.

Discussion and Recommendations

We reviewed the fit between six possible models for the relationships among dissociation, trauma exposure, and *DSM–IV* PTSD symptoms and a large body of research on dissociation and traumatic stress and found strong support for both the Component and Subtype Models. Although it may seem counterintuitive that both models could be accurate, it is possible that dissociative symptoms are a component of the response to traumatic stress and that high levels of dissociation are associated with comorbid psychiatric symptoms that change the nature of the clinical picture in clinically important ways.

Clinical Utility of Dissociation Symptoms

Leaders in the development of *DSM–IV* recommended that any revisions proposed for future versions be considered in light of their clinical utility (First et al., 2004). Adding dissociation symptoms to the PTSD diagnostic criteria would contribute to three aspects of clinical utility in particular: improvement in the conceptualization of PTSD, improvement in accurate diagnosis of PTSD, and improvement in the effectiveness of treatment for PTSD patients with high levels of dissociation symptoms. Conceptualization of PTSD would be improved if dissociation symptoms were included because the diagnosis would represent the diversity of symptoms of PTSD more completely. This change in conceptualization would also lead to new knowledge about traumatic stress as future data collected on those exposed to traumatic stress would include criteria that better reflect the symptoms of most trauma survivors. The diagnosis of PTSD would be more accurate if dissociative symptoms were included because trauma survivors who frequently dissociate may underreport their symptoms because they mislabel or fail to recognize internal states. In fact, research has shown a very high correlation between dissociation and alexithymia (Grabe, Rainermann, Spitzer, Gänsicke, & Freyberger, 2000).

Adding dissociation symptoms to the PTSD diagnostic criteria could also improve the effectiveness of treatment for PTSD patients with high levels of dissociation symptoms, because dissociative symptoms could be taken into account in treatment planning and because specific methods for addressing dissociative symptoms could be used with these patients. There is a need to take dissociation symptoms into account when planning treatment because patients with high levels of dissociative symptoms may respond differently to treatment interventions. In a study of prolonged exposure, dissociation was associated with higher levels of anxiety in pretreatment tests of imaginal exposure (Hagenaars, van Minnen, & Hoogduin, 2010). Also, dissociative symptoms are often experienced as strange and frightening (Dalenberg, 1999) which raises the levels of both stigma and distress in trauma survivors (Courtois & Ford, 2009). Specifically addressing dissociation in treatment is important because dissociation can be quite disruptive to people's lives. Also, trauma survivors with high levels of dissociation are more likely to have self-harming behaviors, suicidality, and disordered eating (Briere et al., 2010; Carlson, McDade-Montez et al., 2011), and it is necessary to address these dangerous behaviors in treatment. Lastly, dissociation may also have an important impact on health-related behaviors such as medication compliance. A recent study of HIV/AIDS patients found that lower adherence to antiretroviral medications was significantly associated with PTSD symptoms in those with high levels of dissociation (Keuroghlian et al., 2011). For all of these reasons, adding dissociative symptoms to the diagnostic criteria for PTSD is expected to increase the clinical utility of the diagnostic criteria.

Adding Dissociative Symptoms to the Diagnostic Criteria for PTSD

The next question is *how* to include dissociative symptoms in the diagnostic criteria for PTSD. One option would be to add a symptom criteria cluster of dissociative experiences and specify some number that must be present for the diagnosis. We see two major problems with this option. First, we know of no empirical studies that support the premise that dissociation is a *necessary* element of PTSD. Dissociative symptoms are clearly present for many who have PTSD and do relate to trauma exposure, but there are certainly people with PTSD who have relatively few dissociative experiences (see Waelde, Silvern & Fairbank, 2005). Second, major changes to diagnostic criteria for a disorder should be made with great caution and only after meeting a high threshold for evidence, particularly changes that make it ambiguous whether people who met criteria for PTSD in past studies also meet the new criteria. Such changes would leave us with very little empirical base for the "new" version of the disorder.

A more appealing option is to incorporate dissociative symptoms into the diagnostic criteria clusters that are already part of the diagnosis. The option fits very well with proposed changes for the DSM-5. The Anxiety, Obsessive-Compulsive Spectrum, Posttraumatic, and Dissociative Disorders Work Group proposed draft criteria that renamed the reexperiencing cluster as intrusions and included "dissociative reactions in which the individual feels or acts as if the traumatic event were recurring" (Friedman, Resick, Bryant, & Brewin, 2010; Phillips, 2009). Elements of past trauma experiences that intrude into consciousness can indeed be considered dissociative reexperiencing. A recent study of dissociative intrusions of voices found that this symptom was reported at high rates and was strongly associated with PTSD in multiple cohorts of combat veterans and in a civilian PTSD sample (Brewin & Patel, 2010). Research on a multidimensional measure of dissociation has reported results that are consistent with placement of dissociative reexperiencing within the reexperiencing cluster. Analyses of scores on a Dissociative Flashback subscale of this measure found it to be highly correlated with the scores for the intrusion scale of a PTSD measure and less strongly correlated with scores for the PTSD avoidance and hyperarousal scales (Dell, 2006). For these reasons, and because of the evidence reviewed here, we recommend adoption of the proposal for DSM-5 to include dissociative reactions in an intrusions criteria cluster. We also recommend that trauma-related sensory misperceptions (including experiences of hearing voices) be added as an example of less extreme forms of dissociative intrusions. We also recommend the specific mention of this symptom in ICD-11.

Some dissociative experiences serve an avoidance function. Experiences of derealization, depersonalization, gaps in aware-

ness, and amnesia can function to distance a person from a negative emotional experience in the moment. Dissociative experiences can also prevent cognitive awareness or divert attention away from stimuli of events that would be distressing, which can be considered a form of experiential avoidance. Inclusion of additional dissociative experiences within the avoidance cluster (or in the new Alterations in Cognition and Mood criterion) would foster measurement of a greater range of symptoms to reflect the experiential avoidance that accounts for a high percentage of variance in PTSD (Boeschen, Koss, Figueredo, & Coan, 2001). The idea that dissociation can serve the purpose of experiential avoidance and contribute to other PTSD symptoms is consistent with the finding of Briere, Scott, and Weathers (2005) that dissociative symptoms that persist for more than two weeks after trauma exposure predict PTSD in a general population sample. This finding was replicated in a sample of motor vehicle accident survivors studied prospectively from the time of trauma exposure (Ehlers, 2006).

We propose, then, that each of these specific, distinct types of dissociative experiences—derealization, depersonalization, gaps in awareness, and amnesia—be listed in the ICD-11 and DSM-5 diagnostic criteria within the Avoidance cluster. Amnesia is already included in the current ICD-10 and *DSM–IV* criteria.

Conclusion

In summary, the empirical evidence and theoretical models reviewed lead us to three specific recommendations for ICD-11 and DSM-5 diagnostic criteria for PTSD.

1. Add mention of trauma related sensory misperceptions as a form of dissociative intrusions. For DSM-5, this would mean the adoption of the proposed language in Criterion B: "Dissociative Reactions (e.g., flashbacks) in which the individual feels or acts as if the traumatic event(s) were recurring" and modification of the parenthetic phrase that follows to "(Such reactions may occur on a continuum, from trauma-related sensory misperceptions to a complete loss of awareness of present surroundings.)"

2. Add "Dissociative avoidance in the form of experiences of derealization, depersonalization, gaps in awareness, or amnesia." In DSM-5, this would be added to Criterion C or D (depending upon whether the new D criterion is adopted).

3. Add a subtype or subtypes to represent a dissociative subtype (and perhaps Complex PTSD) to the ICD-11 and DSM-5 diagnostic criteria.

If these changes are made, dissociative symptoms will be more routinely assessed and attended to clinically within the PTSD population, and their addition to the taxonomy will not have an untoward or distorting impact on past or current diagnoses of PTSD. Adding the symptoms to current or proposed criteria might result in a small number of new cases but would not cause any who currently meet diagnostic criteria to "lose" the diagnosis. In addition, inclusion of dissociative symptoms in the diagnostic criteria would result in an expansion of empirical research that could further clarify the role of dissociative experiences in posttraumatic psychopathology.

References

Boeschen, L. E., Koss, M. P., Figueredo, A. J., & Coan, J. A. (2001). Experiential avoidance and post-traumatic stress disorder: A cognitive mediational model of rape recovery. *Journal of Aggression, Maltreatment & Trauma, 4,* 211–245.

Brewin, C. R., & Patel, T. (2010). Auditory pseudo-hallucinations in Posttraumatic Stress Disorders. *Journal of Clinical Psychiatry, 71,* 419–425.

Briere, J., Hodges, M., & Godbout, N. (2010). Traumatic stress, affect dysregulation, and dysfunctional avoidance: A structural equation model. *Journal of Traumatic Stress, 23,* 767–774.

Briere, J. (1992). *Child abuse trauma: Theory and treatment of the lasting effects*. Thousand Oaks, CA: Sage.

Briere, J. (2006). Dissociative symptoms and trauma exposure: Specificity, affect dysregulation, and posttraumatic stress. *Journal of nervous and mental disease,, 194,* 78.

Briere, J., Scott, C., & Weathers, F. (2005). Peritraumatic and persistent dissociation in the presumed etiology of PTSD. *American Journal of Psychiatry, 162,* 2295–2301.

Carlson, E. B., & Dalenberg, C. J. (2000). A conceptual framework for the impact of traumatic experiences. *Trauma, Violence, & Abuse, 1,* 4–28.

Carlson, E. B., Dalenberg, C. J., & McDade-Montez, E. (2012). Dissociation in Posttraumatic Stress Disorder Part I: Definitions and review of research. *Psychological Trauma, 4,* 479–489.

Carlson, E. B., McDade-Montez, E., Armstrong, J., & Loewenstein, R. J. (2011). *Development and validation of the Structured Interview for Self-Destructive Behaviors.* Manuscript submitted for publication.

Carlson, E. B., & Putnam, F. W. (1993). An update on the Dissociative Experiences Scale. *Dissociation: Progress in the Dissociative Disorders, 6,* 16–27.

Carlson, E. B., Smith, S., Palmieri, P., Dalenberg, C., Ruzek, J. I., Kimerling, R., . . . Spain, D. A. (2011). Development and validation of a brief self-report measure of trauma exposure: The Trauma History Screen. *Psychological Assessment, 23,* 463–477.

Courtois, C. A., & Ford, J. D. (Eds.). (2009). *Treating complex traumatic stress disorders: An evidence-based guide.* New York, NY: Guilford Press.

Courtois, C. A. (1988). *Healing the incest wound: Adult survivors in therapy.* New York, NY: Norton.

Dalenberg, C., Brand, B. L., Gleaves, D. H., Dorahy, M. J., Loewenstein, R. J., Cardena, E. B., et al. (2012). Evaluation of the evidence for the trauma and fantasy models of dissociation. *Psychological Bulletin, 138,* 550–588.

Dell, P. F. (2006). The Multidimensional Inventory of Dissociation (MID): A comprehensive measure of pathological dissociation. *Journal of Trauma & Dissociation, 7,* 77–106.

Dutra, L., Bureau, J., Holmes, B., Lyubchik, A., & Lyons-Ruth, K. (2009). Quality of early care and childhood trauma: A prospective study of developmental pathways to dissociation. *Journal of Nervous and Mental Disease, 197,* 383–390.

Ehlers, A. (2006). More evidence for the role of persistent dissociation in PTSD. *American Journal of Psychiatry, 163,* 1112.

First, M. B., Pincus, H. A., Levine, J. B., Williams, J. B., Ustun, B., & Peele, R. (2004). Clinical utility as a criterion for revising psychiatric diagnoses. *American Journal of Psychiatry, 161,* 946–954.

Foa, E. B., Hearst-Ikeda, D., Michelson, L. K., & Ray, W. J. (1996). Emotional dissociation in response to trauma: An information-processing approach *Handbook of dissociation: Theoretical, empirical, and clinical perspectives.* (pp. 207–224). New York, NY: Plenum Press.

Forbes, D., Haslam, N., Williams, B. J., & Creamer, M. (2005). Testing the

latent structure of Posttraumatic Stress Disorder: A taxometric study of combat veterans. *Journal of Traumatic Stress, 18,* 647–656.

Freud, S. (1962). The aetiology of hysteria (1896). In J. Strachey (Ed.), *The standard edition of the complete psychological works of Sigmund Freud* (Vol. 7, pp. 187–221). London, UK: Hogarth Press.

Frewen, P. A., & Lanius, R. A. (2006). Neurobiology of dissociation: Unity and disunity in mind-body-brain. *Psychiatric Clinics of North America, 29,* 113–128.

Friedman, M., Resick, P. A., Bryant, R. A., & Brewin, C. (2010). Considering PTSD for DSM-5. *Depression and Anxiety, 0,* 1–20.

Gershuny, B. S., Najavits, L. M., Wood, P. K., & Heppner, M. (2004). Relation between trauma and psychopathology: Mediating roles of dissociation and fears about death and control. *Journal of Trauma & Dissociation, 5,* 101–117.

Giesbrecht, T., Lynn, S. J., Lilienfeld, S. O., & Merckelbach, H. (2008). Cognitive processes in dissociation: An analysis of core theoretical assumptions. *Psychological Bulletin, 134,* 617–647.

Ginzburg, K., Butler, L. D., Saltzman, K., & Koopman, C. (2009). Dissociative reactions in PTSD. In P. F. Dell, J. O'Neil & E. Somer (Eds.), *Dissociation and the dissociative disorders: DSM-V and beyond* (pp. 456–470). New York, NY: Routledge.

Grabe, H., Rainermann, S., Spitzer, C., Gänsicke, M., & Freyberger, H. (2000). The relationship between dimensions of alexithymia and dissociation. *Psychotherapy and psychosomatics, 69,* 128–131.

Hagenaars, M. A., van Minnen, A., & Hoogduin, K. A. L. (2010). The impact of dissociation and depression on the efficacy of prolonged exposure treatment for PTSD. *Behaviour Research and Therapy, 48,* 19–27.

Herman, J. (1992). *Trauma and recovery.* New York, NY: Basic Books.

Keuroghlian, A., Kamen, C., Neri, E., Lee, S., Liu, R., & Gore-Felton, C. (2011). Trauma, dissociation, and antiretroviral adherence among persons living with HIV/AIDS. *Journal of Psychiatric Research, 45,* 942–968.

Lanius, R. A., Vermetten, E., Loewenstein, R. J., Brand, B., Schmahl, C., Bremner, J. D., & Spiegel, D. (2010). Emotion modulation in PTSD: Clinical and neurobiological evidence for a dissociative subtype. *American Journal of Psychiatry, 167,* 640–647.

Lyons-Ruth, K. (2003). Dissociation and the parent-infant dialogue: A longitudinal perspective from attachment research. *Journal of the American Psychoanalytic Association, 51,* 883–911.

McCaslin, S. E., Inslicht, S. S., Metzler, T. J., Henn-Haase, C., Maguen, S., Neylan, T., . . . Marmar, C. R. (2008). Trait dissociation predicts Posttraumatic Stress Disorder symptoms in a prospective study of urban police officers. *Journal of Nervous and Mental Disease, 196,* 912–918.

Ogawa, J. R., Sroufe, L. A., Weinfield, N. S., Carlson, E. A., & Egeland, B. (1997). Development and the fragmented self: Longitudinal study of dissociative symptomatology in a nonclinical sample. *Development and Psychopathology, 9,* 855–879.

Perry, B. D. (2008). Child maltreatment: A neurodevelopmental perspective on the role of trauma and neglect in psychopathology. In T. P. Beauchaine & S. P. Hinshaw (Eds.), *Child and adolescent psychopatholgy* (pp. 93–128). Hoboken, NJ: Wiley.

Phillips, K. A. (2009). *Report of the DSM-5 Anxiety, Obsessive-Compulsive Spectrum, Posttraumatic, and Dissociative Disorders Work Group.* Washington, DC: American Psychiatric Association.

Putnam, F. W., Carlson, E. B., Ross, C. A., & Anderson, G. (1996). Patterns of dissociation in clinical and nonclinical samples. *Journal of Nervous and Mental Disease, 184,* 673–679.

Putnam, F. W. (1985). Dissociation as an response to extreme trauma. In R. P. Kluft (Ed.), *Childhood antecedents of multiple personality* (pp. 65–98). Washington, DC: American Psychiatric Association.

Ruscio, A. M., Ruscio, J., & Keane, T. M. (2002). The latent structure of posttraumatic stress disorder: A taxometric investigation of reactions to extreme stress. *Journal of Abnormal Psychology, 111,* 290–301.

Schauer, M., & Elbert, T. (2010). Dissociation following traumatic stress: Etiology and treatment. *Journal for Psychology, 21,* 109–127.

Simeon, D., Knutelska, M., Yehuda, R., Putnam, F., Schmeidler, J., & Smith, L. M. (2007). Hypothalamic-pituitary-adrenal axis function in dissociative disorders, post-traumatic stress disorder, and healthy volunteers. *Biological Psychiatry, 61,* 966–973.

Smith, S., & Carlson, E. B. (2011). *Validation and norms for trauma-related measures in general population samples.* Manuscript submitted for publication.

Spiegel, D., & Cardena, E. (1990). Dissociative mechanisms in posttraumatic stress disorder. In M. E. Wolf & A. D. Mosnaim (Eds.), *Posttraumatic stress disorder: Etiology, phenomenology, and treatment* (pp. 23–34). Arlington, VA: American Psychiatric Association.

Spiegel, D. (1986). Dissociating damage. *American Journal of Clinical Hypnosis, 29,* 123–131.

Steele, K., van der Hart, O., & Nijenhuis, E. R. S. (2009). The theory of trauma-related structural dissociation of the personality. In P. F. Dell, J. O'Neil & E. Somer (Eds.), *Dissociation and the dissociative disorders: DSM-V and beyond* (pp. 239–258). New York, NY: Routledge.

Twaite, J. A., & Rodriguez-Srednicki, O. (2004). Childhood sexual and physical abuse and adult vulnerability to PTSD: The mediating effects of attachment and dissociation. *Journal of Child Sexual Abuse, 13,* 17–38.

van der Hart, O., & Dorahy, M. J. (2009). History of the concept of dissociation. In P. F. Dell, J. O'Neil & E. Somer (Eds.), *Dissociation and the dissociative disorders: DSM-V and beyond* (pp. 3–26). New York, NY: Routledge.

van der Kolk, B. A., Brown, P., & van der Hart, O. (1989). Pierre Janet on post-traumatic stress. *Journal of Traumatic Stress, 2,* 365–378.

van der Kolk, B. A., Pelcovitz, D., Roth, S., & Mandel, F. S. (1996). Dissociation, somatization, and affect dysregulation: The complexity of adaption to trauma. *American Journal of Psychiatry, 153,* 83–93.

van der Kolk, B. A., van der Hart, O., & Marmar, C. R. (1996). Dissociation and information processing in posttraumatic stress disorder In B. van der Kolk (Ed.), *Traumatic stress: The effects of overwhelming experience on mind, body, and society* (pp. 303–327). New York, NY: Guilford Press.

van der Kolk, B. A., & Van der Hart, O. (1989). Pierre Janet and the breakdown of adaptation in psychological trauma. *The American Journal of Psychiatry, 146,* 1530–1540.

van der Kolk, B. A. (1987). The psychological consequences of overwhelming life experiences. In B. A. van der Kolk (Ed.), *Psychological trauma* (pp. 1–30). Washington, DC: American Psychiatric Association.

Waelde, L. C., Silvern, L., Carlson, E. B., Fairbank, J. A., & Kletter, H. (2009). Dissociation in PTSD. In P. F. Dell, J. O'Neil & E. Somer (Eds.), *Dissociation and the dissociative disorders: DSM-V and beyond* (pp. 447–456). New York, NY: Routledge.

Wolf, E. J., Miller, M. W., Reardon, A. F., Ryabchenko, K. A., Castillo, D., & Freund, R. (2012). A latent class analysis of dissociation and PTSD: Evidence for a dissociative subtype. *Archives of General Psychiatry, 69,* 698–705.

Received January 17, 2012
Accepted March 1, 2012 ■

See page 577 for a correction to this article.

Correction to Dalenberg and Carlson (2012)

In the article, "Dissociation in Posttraumatic Stress Disorder Part II: How Theoretical Models Fit the Empirical Evidence and Recommendations for Modifying the Diagnostic Criteria for PTSD," (*Psychological Trauma: Theory, Research, Practice, and Policy*. Advanced online publication. May 14, 2012. doi:10.1037/a0027900) there was an error in the section, "Models 5 and 5a: The Component and Subtype Models." The paragraph that began, "Taxometric studies of dissociation in PTSD are also relevant to the Subtype model . . ." has been replaced with a paragraph that begins, "One taxometric study of dissociation in PTSD is also relevant to the Subtype model . . ." All versions of this article have been corrected.

DOI: 10.1037/a0030647