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Perspectives on the Conceptualization of the Dissociative Subtype of PTSD and Implications for Treatment

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Abstract

A dissociative subtype of PTSD was added to the fifth edition of the *Diagnostic and Statistical Manual of Mental Disorders (DSM-5)* to denote a distinct presentation of PTSD marked by symptoms of derealization and depersonalization. The subtype has brought renewed interest in the conceptualization of dissociation in PTSD, and raised questions about its treatment. We review the evidence for two models of PTSD-related dissociative symptoms and the treatment approaches that align with each. We find support for the contributions of biologically-based trait-level factors to dissociation, and suggest that empirically supported treatments for PTSD demonstrate efficacy for patients with the subtype.

Keywords

posttraumatic stress disorder (PTSD); dissociative subtype; trauma; treatment; conceptual model

The dissociative subtype of posttraumatic stress disorder (PTSD) was added to the fifth edition of the *Diagnostic and Statistical Manual of Mental Disorders (DSM-5)*. This subtype characterizes individuals who meet criteria for PTSD and experience significant symptoms of *derealization* (feeling unreal or detached from the world), or *depersonalization* (feeling that one's body is detached from the self). The addition of this subtype was prompted, in part, by studies demonstrating markedly elevated symptoms of dissociation in a subset of individuals with PTSD. Specifically, latent profile analyses of the core *DSM-IV* PTSD and three dissociation symptoms (reductions in awareness, depersonalization, and derealization) revealed three classes characterized by: (a) moderate PTSD severity and no dissociation; (b) high PTSD severity and no dissociation; and (c) high PTSD severity and significant dissociation [1]. This last group comprised 15% of those with PTSD. Subsequent studies yielded similar findings, estimating the prevalence of the dissociative subtype of PTSD at approximately 10–30% [2–6].

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Comprehensive literature reviews of the dissociative subtype of PTSD can be found elsewhere [7–9]. Here, we discuss two prominent models of dissociation in PTSD and implications for treatment. The first conceptualizes dissociation as excessive trauma-related downregulation of emotion that serves as a form of conscious and/or conditioned avoidance of trauma-related emotions, cognitions, and memories [7, 10]. The second suggests that biologically-based variability in the capacity to integrate sensory and cognitive processes predisposes to dissociation, particularly under stress [11–15].

The Trauma/Avoidance Model

One prominent view suggests that dissociation occurs as a consequence of trauma exposure, largely due to psychological processes that aide in avoidance of trauma-related emotions, cognitions, and physical sensations [10, 16–17], a perspective rooted in the early writings of Pierre Janet [18]. Recent papers [10, 19–20] highlight associations between trauma and dissociation as evidence that “the relationship between traumatic experience and dissociation is, at least in part, causal” [10, p. 553]. This conclusion by Dalenberg et al. [10] was based on a review of 38 studies that yielded an average moderate effect size ($r = .32$) for the association between trauma and dissociation [10]. Studies of the role of early life experiences have suggested that avoidant or disorganized attachment styles in childhood are also predictive of dissociation [21]. Proponents of the Trauma/Avoidance Model suggest that dissociation is a form of self-hypnosis developed to defend against these traumatic and stressful experiences and memories [22] and conclude that “research supports the Trauma Model of dissociation as a regulatory response to fear or other extreme emotion” [10, p. 578].

Concerns have been raised about the Trauma/Avoidance Model [13–15]. First, it does not address dissociation that temporally precedes trauma. Second, associations between trauma and dissociation are often weaker when objective, compared to self-report, trauma measures are employed [13], raising the possibility of symptom-related bias in self-report trauma measures. Third, studies evaluating motivated forgetting (i.e., avoidance) among those with dissociation have not found evidence for this effect and instead suggest that memory for emotionally evocative words is actually enhanced [23].

Recent neuroimaging research has investigated the neural correlates of dissociation. Consistent with the Trauma/Avoidance Model, Lanius and colleagues conceptualized dissociation as downregulation of internal experiences, mediated by prefrontal neural circuits, when confronted with trauma-related stressors [7, 24–25]. Across a number of PTSD neuroimaging studies, Lanius and colleagues used a trauma script-driven imagery paradigm to provoke trauma-related symptoms during functional neuroimaging [26]. Dissociative reactions were associated with increased activation in prefrontal cortical regions implicated in emotion and arousal regulation [27–28], and concurrent inhibition of limbic regions [25, 29]. This pattern contrasted with that of individuals who showed emotional arousal and anxiety, who showed greater limbic activation [28, 30]. A resting state functional connectivity study [31] found that the dissociative subtype was associated with greater functional connectivity between amygdala and prefrontal regions (important for affect regulation), as well as regions associated with consciousness, awareness, and proprioception

(e.g., superior parietal lobe, precuneus). While these results are intriguing and could provide evidence of neuronal signatures of the dissociative subtype, they are drawn from a small number of studies with relatively small samples (10 cases/controls, except [31]). Thus, replication of these findings is needed.

The Trauma/Avoidance Model acknowledges a role for some biological factors in determining risk for dissociative symptoms following trauma [10], but prioritizes the etiologic role of trauma. However, existing data do not directly address whether trauma exposure is the primary etiological factor in dissociation. Moreover, it does not address whether dissociation should be conceptualized as avoidance. Below, we describe an alternative perspective in which trauma is less central to the development of dissociative symptoms.

The Cognitive/Sensory Disintegration Model

A second model views dissociation as stemming from biological, cognitive, and psychological trait-like factors that reduce the capacity to maintain sensory and cognitive integration under stress [13–15]. This perspective stems from a diverse body of empirical research. Specifically, Jang et al. [32] found that genetic influences accounted for 48% of the variance in pathological dissociation, and a genome-wide association study found ten single nucleotide polymorphisms nominally associated with derealization and depersonalization [33]. Two of the implicated genes (*ADYC8* and *DPP6*) are important for synaptic integration, learning, memory, and long-term potentiation, suggesting that dissociation could arise from genetic variability influencing sensory/cognitive processing and integration.

Consistent with this, cognitive factors including perception, distractibility, memory errors, and attention deficits have been associated with dissociative experiences [13–14, 34–37]. Traits such as absorption (tendency to become absorbed in mental imagery), fantasy proneness (tendency to become engrossed in imaginative experiences), and suggestibility (inclination to accept suggestions and respond accordingly) have also been associated with dissociation, suggesting that these traits may predispose to dissociation or influence reporting of such symptoms [13, 15]. That said, proponents of the Trauma/Avoidance Model suggest that fantasy proneness can be traced to trauma exposure, as elaborate fantasies may develop to cope with early trauma [10].

A number of experimental paradigms and medical disorders are associated with dissociation and would be difficult to conceptualize from a Trauma/Avoidance perspective. First, simple exercises that tax sensory processes can elicit dissociative symptoms: in exposure-based treatments for panic disorder, symptoms of depersonalization, which can occur during panic attacks, are often intentionally induced by having patients stare at their own eyes in a mirror [38]. Second, experimental research has suggested that sleep deprivation can lead to dissociative experiences [39–41], consistent with the general finding that poor sleep exacerbates many stress-sensitive psychiatric symptoms [42]. Finally, dissociation is associated with brain disorders defined by sensory and cognitive processing disruptions such as epilepsy [43] and has been linked to focal lesions in neural regions critical to multisensory integration [44].

The Cognitive/Sensory Disintegration Model does not exclude trauma as a contributing factor to the emergence of dissociative symptoms [14]. Rather, trauma is viewed as one type of stressor that may overtax a vulnerable system. This is consistent with the finding that trauma is not necessary for dissociation, as its prevalence is approximately 3.3% in the general population [45].

Optimizing Treatment

The two models carry different implications for treating both PTSD and dissociation. From the perspective of the Trauma/Avoidance Model, dissociation interferes with the processing of trauma-related memories, emotions, and cognitions (e.g., via avoidance), essential features of empirically supported trauma-focused therapies. Given this, staged approaches to treatment have been suggested to first target dissociation through emotion and interpersonal regulation strategies, followed by trauma-focused therapy [46–47]. In contrast, if dissociation is conceptualized as arising from vulnerabilities in cognitive and sensory integration capabilities and the influence of stress on these vulnerable systems, then reducing stress and distress may reduce dissociation severity.

The growing literature on the effects of dissociation on PTSD treatment response may inform treatment decisions. The majority of studies to date suggest that evidence-based treatments for PTSD are effective in reducing dissociative symptoms [47–50] and that dissociation does not robustly attenuate the PTSD symptom treatment response [47–51]. Specifically, a number of studies [47–50] found that dissociative symptoms tend to remit with PTSD treatment, including Prolonged Exposure (PE), Cognitive Processing Therapy, and Present-Centered Therapy; one study [47] further suggested that such symptoms responded best to Skills Training in Affective and Interpersonal Relationships (STAIR) followed by Narrative Story Telling (NST) compared to supportive therapy paired with STAIR or NST alone.

Studies investigating possible effects of dissociation on PTSD treatment response have generally found little to no effect. One trial of narrative exposure therapy vs. treatment-as-usual found no effect of dissociation on PTSD symptom change [51] and in a separate study, pretreatment dissociative symptoms yielded no effect on PTSD symptom reduction or treatment dropout during PE [48]. A study of Cognitive Processing Therapy (CPT) found no effect of dissociation on PTSD change, but showed that among those with substantial dissociative symptoms, PTSD symptoms declined faster during standard CPT compared to cognitive therapy alone [50]. Another study [47] reported no global effect of baseline dissociation on PTSD symptom change, but showed that among an apparently small group of patients with relatively higher dissociation scores at post-treatment, those who were assigned to STAIR/NST had continued PTSD symptom improvement at follow-up. We are aware of only one study that reported a global effect of dissociation on PTSD symptom change in response to PE and Present-Centered Therapy [50], but this effect was small and yielded minimal differences in final PTSD symptoms across the subtype and high PTSD groups.

Additional studies have reported effects for dissociation but upon closer inspection, analytic concerns raise questions about these interpretations. Specifically, two recent studies found that pre-treatment [52] and first session [53] dissociative symptoms were associated with greater PTSD symptoms at post-treatment. These results suggest a main effect of group (high vs. low dissociation) but cannot address if dissociation differentially impacted treatment response because the group $\times$ time effect was not evaluated. Moreover, given that dissociation tends to be correlated with PTSD severity, and that this association was not controlled for, results may be due to greater baseline PTSD severity as opposed to dissociation. Finally, a study of dissociation during psychotherapy sessions (“state dissociation”) in a modified version of Dialectical Behavior Therapy (DBT), found that patients with less session-to-session dissociation were more likely to demonstrate PTSD symptom reductions [54]. That study used a change-score analytic design and additionally controlled for baseline PTSD symptoms. Distinguishing effects of dissociation from those of baseline PTSD is critical, however this analytic approach was problematic because baseline PTSD contributed both to the exogenous covariate and to the endogenous change score—a form of criterion contamination that complicates interpretation of results. Nonetheless, studies of acute (state) dissociation [53–54] raise the possibility that such symptoms may evidence differential effects on PTSD treatment response relative to trait dissociation, suggesting that additional research on this topic is necessary. This is particularly true given experimental evidence that acute dissociation may impede emotional learning [55], a critical component of PTSD treatment.

Conclusions and Future Directions

The odd, unusual, and highly individual experience of dissociative symptoms makes the construct challenging to measure and limits the ability to determine the etiological factors that give rise to such symptoms and the basic processes underlying their expression. It is likely that there is substantial individual variability in the factors that influence and mediate dissociation, such that aspects of the Trauma/Avoidance and Cognitive/Sensory Disintegration models may both be critical to understanding dissociation for the individual patient. That said, there is currently stronger evidence for the notion that dissociative symptoms arise in the context of an underlying biological vulnerability to suboptimal attentional, cognitive, and sensory integration processes. When these processes are taxed by a stressor, be it trauma exposure, PTSD symptoms, sleep deprivation, or mind wandering, the system fails to operate efficiently and dissociative symptoms emerge.

Additional research is necessary to understand the mechanisms underlying the dissociative subtype. For example, current laboratory approaches to inducing dissociation have relied heavily on trauma scripts [26, 30], which are not specific to dissociation given that they are standard primes for core reexperiencing symptoms [56]. Therefore, it would be beneficial to examine alternative methods for eliciting symptoms, such as hypnosis [22], mirror staring [38], and sleep deprivation [39–41] and to study the neuropsychological and functional imaging correlates of dissociative symptoms arising from these paradigms. Experimental protocols could also be designed that alter the contingencies surrounding dissociation to shed light on whether dissociation reflects a reinforcing form of avoidance. It would be premature at this point to suggest that the dissociative subtype necessitates a different type

or length of treatment, however, additional research is required before final conclusions can be drawn. We welcome research that addresses these questions empirically to better understand the etiology, nature, and course of the dissociative subtype and improve the health and wellness of those who experience these symptoms.

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Highlights

- A new dissociative subtype of PTSD has been added to DSM-5
- Perspectives differ on the centrality of trauma to development of dissociation
- Trait-level factors may interact with stress to increase vulnerability to dissociation
- Existing evidence-based treatments for PTSD have demonstrated efficacy for the dissociative subtype