Dissociative Disorders in DSM-5

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Abstract

The rationale, research literature, and proposed changes to the dissociative disorders and conversion disorder in the fifth edition of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-5) are presented. Dissociative identity disorder will include reference to possession as well as identity fragmentation, to make the disorder more applicable to culturally diverse situations. Dissociative amnesia will include dissociative fugue as a subtype, since fugue is a rare disorder that always involves amnesia but does not always include confused wandering or loss of personality identity. Depersonalization disorder will include derealization as well, since the two often co-occur. A dissociative subtype of posttraumatic stress disorder (PTSD), defined by the presence of depersonalization or derealization in addition to other PTSD symptoms, is being recommended, based upon new epidemiological and neuroimaging evidence linking it to an early life history of adversity and a combination of frontal activation and limbic inhibition. Conversion disorder (functional neurological symptom disorder) will likely remain with the somatic symptom disorders, despite considerable dissociative comorbidity.

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INTRODUCTION

The dissociative disorders are characterized by a disruption of and/or discontinuity in the normal integration of consciousness, memory, identity, emotion, perception, body representation, motor control, and behavior. Dissociative symptoms can disrupt every area of psychological functioning and are usually divided into two types: (*a*) unbidden intrusions into awareness and behavior, with accompanying deficits in continuity of subjective experience, labeled "positive" dissociative symptoms (e.g., identity fragmentation, depersonalization, derealization); and (*b*) inability to access information or to control mental functions, called "negative" dissociative symptoms (e.g., amnesia, aphonia, paralysis). In this review, we discuss three dissociative disorders—dissociative identity disorder (DID), dissociative amnesia, and depersonalization/derealization disorder—and two closely related disorders: posttraumatic stress disorder (PTSD) and conversion disorder (functional neurological symptom disorder). We emphasize changes to these disorders proposed for the fifth edition of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-5) and the rationales for these changes (Brand et al. 2012, Brown & Lewis-Fernández 2011, Spiegel et al. 2011a).

Table 1 DSM-IV-TR diagnostic criteria for dissociative identity disorder

A	The presence of two or more distinct identities or personality states (each with its own relatively enduring pattern of perceiving, relating to, and thinking about the environment and self).
B	At least two of these identities or personality states recurrently take control of the person's behavior.
C	Inability to recall important personal information that is too extensive to be explained by ordinary forgetfulness.
D	The disturbance is not due to the direct physiological effects of a substance (e.g., blackouts or chaotic behavior during alcohol intoxication) or a general medical condition (e.g., complex partial seizures). Note: In children, the symptoms are not attributable to imaginary playmates or other fantasy play.

PTSD and conversion disorder are included in this review because of their close nosological relationship with the dissociative disorders. The dissociative disorders are placed in DSM-5 right after the trauma- and stress-related disorders to indicate the close relationship between them. Both acute stress disorder and PTSD contain dissociative symptoms, such as amnesia, flashbacks, and emotional numbing. A dissociative subtype has been proposed for PTSD, and our review addresses the rationale for this proposal. In DSM-5 conversion disorder is considered a somatic symptom disorder, the disorder grouping that immediately follows the dissociative disorders. These disorders have high comorbidity with dissociative disorders and are characterized by pseudoepilepsy and other pseudoneurological symptoms that are dissociative in nature and that involve interference with sensory and motor function. In fact, the trauma- and stress-related disorders and the somatic symptom disorders both involve failures of integration of experience, traumatic or somatic, into consciousness.

DISSOCIATIVE IDENTITY DISORDER

Symptomatology

Dissociative identity disorder (**Table 1**) is the flagship dissociative disorder, having been described in the nineteenth century by Morton Prince, the founder of the *Journal of Abnormal Psychology* (Prince 1906), and Pierre Janet (Taylor 1984; Janet 1889, 1907). Janet referred to *désaggregation mentale*, a failure of integration of components of mental experience that ordinarily function together, including pronounced alterations in identity and memory. The disorder is typically associated with a childhood history of severe physical or sexual abuse (Dalenberg et al. 2012; Spiegel 1984, 1986, 1997) and presents with inconsistencies in identity, memory, and consciousness. The disorder was popularized in the United States through books such as *The Three Faces of Eve* (Thigpen & Cleckley 1957) and *Sybil* (Schreiber 1973), but this also led to the criticism that psychological fragmentation was being induced in highly suggestible patients by unwitting or inept therapists (Rieber 2006). However, the persistence of solidly grounded clinical description and case series indicates that the disorder is more than an iatrogenic response to maladroit therapeutic suggestion (Spiegel 2006, Spiegel et al. 2011b). The fact that individuals with the disorder typically go five to twelve years before receiving the diagnosis (Putnam et al. 1986, Rivera 1991, Ross et al. 1989) suggests that they are relatively resistant to implicit or explicit suggestions that they do not have the disorder or have a different disorder, such as schizophrenia (Kluft 1987).

The diagnostic criteria for DID have undergone little substantive change since they were set forth in DSM-III, with the exception of the amnesia criterion that was removed from the criteria

in the DSM-III-R but was reinstated in DSM-IV. The fundamental question addressed by the DSM-5 work group involved broadening the cross-cultural reach of the disorder by including reference to pathological possession (Spiegel et al. 2011a). Other changes to the criteria include the following:

- Incorporating in criterion A detailed clinical descriptors of DID to facilitate case detection by clinicians, specifically descriptors that reflect the most highly intercorrelated dissociative symptoms that routinely characterize DID (Spiegel et al. 2011a).
- Clarifying that identity alteration does not have to be directly witnessed by an observer but instead could be reported by the patient.
- Further specifying the amnesia criterion to include inability to recall everyday as well as traumatic information.
- Adding criteria stating that symptoms must be associated with clinically significant distress and functional impairment and cannot be part of a broadly accepted cultural or religious practice.

Below we review recent findings regarding the prevalence, diagnostic features, and treatment of DID, including a discussion of the major proposed change in the definition involving possession-like symptoms.

Addition of Pathological Possession to DID Criteria

Proposed changes. The following revisions are under consideration:

1. Addition of the words "or an experience of possession" to Criterion A.
2. Addition of a new Criterion C that distinguishes pathological from nonpathological forms of apparent DID and also helps to distinguish pathological from nonpathological forms of possession.
3. Addition of a new Criterion D to distinguish pathological forms of apparent DID from normative religious or spiritual practices. The new Criterion D also helps to distinguish pathological from nonpathological forms of possession.

Language on pathological possession has been proposed for the DID diagnostic criteria in order to make these criteria more applicable to diverse cultural groups by identifying a common presentation of DID in Africa, Asia, and other non-Western cultures, as well as in subgroups in Western cultures (e.g., immigrant populations, some conservative religious groups).[1] A new DID specifier was also proposed for the same reason, denoting presentations of DID that are characterized by prominent pseudoneurological symptoms. The specifier is not discussed further in this review.

Rationale for the inclusion of pathological possession. There are two complementary components to the relationship between pathological possession and DID: (*a*) whether the dissociative symptoms of pathological possession are essentially identical to those of DID and, conversely, (*b*) whether the dissociative symptoms of cases of DID across diverse cultures are characterized by pathological possession or possession-like phenomena.

[1]For simplicity, in this review "Western culture" refers to the dominant cultural orientation in North America, Europe, and Westernized, industrialized areas in other parts of the world. "Non-Western" refers to cultures different from those dominant in the Western industrialized world as well as to immigrant groups with non-Western cultural orientation within the industrialized world. Some Western subcultures (e.g., subgroups of evangelical Christian groups) may have belief systems about possession similar to those of non-Western cultural groups and may engage in nonpathological possession rituals as part of their regular worship (Putnam 1991, Spiegel et al. 2011a).

Dissociative symptoms of pathological possession are identical to those of DID. Specifically, cases of pathological possession manifest (*a*) distinct changes in identity and (*b*) amnesia. As noted by Spiegel et al. (2011a):

> It is very important to note that (**pathological possession**) . . . has marked phenomenological similarities to DID. It is **a disorder of identity alteration** that occurs during an altered state of consciousness. Of course, unlike DID, the alternate identity or identities in (pathological possession) are attributed to possession (by an external spirit, power, deity, or other person) rather than to internal personality states. Associated symptoms of (pathological possession) include stereotyped or culturally determined behaviors or movements that are **experienced as being controlled by the possessing agent** and/or **full or partial amnesia for the event . . . These alterations are involuntary, distressing, uncontrollable**, often chronic . . . (pp. 842–843; bold added for emphasis)
>
> Both DID and (pathological possession) manifest **incompatible identities that are separated by an amnestic barrier; these identities** occur during an altered state of consciousness and **display distinct cognitions, affects, and behaviors**. (pp. 844–845)

The above bolded symptoms of pathological possession are very similar to those of DID, with the exception that, in keeping with the patient's cultural background, the identity alteration is attributed to possession by an external spirit, power, deity, or other person.

Cases of pathological possession with the above dissociative symptoms have been reported in many countries (Cardeña et al. 2009): China (Gaw et al. 1998, Ross 2011), India (Adityanjee et al. 1989, Carstairs & Kapur 1976, Castillo 1994, Das & Saxena 1991, Venkataramaiah et al. 1981), Turkey (Şar et al. 1996, 2007), Iran, Republic of Singapore, Puerto Rico (Martinez-Taboas 1991, 2005), and Uganda (van Duijl et al. 2010). Some cases of DID in the United States and Canada are also explicitly attributed by the patient to possession (Bowman et al. 1993, Bull et al. 1998, Ross 2011, Ross & Ness 2010).

Similar to Western patients with DID, patients with pathological possession in Uganda ($N = 117$) report a significantly higher exposure to traumatic events (as assessed on the Harvard Trauma Questionnaire and the Traumatic Experiences Checklist) in comparison with 76 randomly selected mentally healthy inhabitants of the same villages (van Duijl et al. 2010). Scores on both of these trauma scales were strongly correlated with symptoms of pathological possession ($r = 0.51$–0.64) and other dissociative symptoms ($r = 0.64$–0.69). This is consistent with the well-established link between traumatic exposure and DID as well as other types of dissociation. Individuals with pathological possession in Uganda also had significantly higher dissociation scores than did normal controls. Symptoms of pathological possession correlated highly ($r = 0.68$–0.76) with scores on the Dissociative Experiences Scale and the Somatoform Dissociation Questionnaire (van Duijl et al. 2010).

In short, individuals with pathological possession around the world exhibit dissociative symptoms that are essentially identical to those of DID and therefore meet diagnostic criteria: involuntary alterations of identity (DID criterion A) and amnesia for everyday and/or traumatic events (DID criterion B).

Many cases of DID are characterized by pathological possession or possession-like phenomena. Some cases of DID in both Western and non-Western settings (e.g., United States, Canada, Puerto Rico, China, and Turkey) are explicitly attributed by the patient to possession (Bowman 1993b, Bull et al. 1998, Martinez-Taboas 1991, Ross 2011, Ross & Ness 2010, Şar et al. 2010). For example, of 35 consecutive inpatients admitted to a Turkish hospital with DSM-IV DID (Şar et al. 1996), 45.7% attributed their symptoms to possession by a *jinn* or demon, 28.6%

to possession by a dead person, 22.9% to possession by a living person, and 22.9% to possession by another power or source (several participants reported various types of possession).

In Western settings, 58.7% of 303 patients with DID reported that they felt like they were possessed (Ross 2011). In some cases, the phenomenology of the DID was influenced by their participation in religious groups that emphasized normative possession (e.g., Pentecostals, charismatics).

These data show that a substantial proportion of patients with DID conform to the possession-type phenomenology of the disorder. The addition of pathological possession to the definition of DID indicates that, around the world, DID presents in two main forms: as nonpossession-form DID or as possession-form DID. These are not mutually exclusive: individuals in one cultural setting may experience either form, and a single individual may experience both forms. Typically, in nonpossession-form DID the alternate identities are experienced as internal aspects of the person, as other personalities or identity states. In possession-form DID, by contrast, the alternate identities are typically experienced as external possessing agents, usually of supernatural or spiritual origin (e.g., demons, spirits). The main contrast between these two forms of DID appears to be shaped by the patient's sociocultural milieu. That is, possession-form DID arises in contexts where possessing agents are considered to be "real" (e.g., some fundamentalist religions in the United States or in traditional South Asian cultures), as opposed to nonpossession-form DID, which draws its alternate identities from individualistically focused experiences, such as stages of life (e.g., childhood) or biographical roles in the patient's life (e.g., protector, perpetrator).

This does not imply assertion of the objective reality of being possessed by outside agencies any more than it endorses the idea that individuals with nonpossession-form DID have a number of "separate people" within them. Individuals with both possession-form DID and individuals with nonpossession-form DID experience themselves as having alternate identities or possessing entities. This phenomenal experience is a bodily, subjective reality to those affected (Loewenstein & Putnam 2004, Putnam 1997, Spiegel et al. 2011a). Clinical interactions with individuals with DID clearly bring a sense of this experience into the interview. This occurs despite the fact that many individuals with DID struggle with disbelief of their own condition. In contrast, individuals with "imitative" (i.e., factitious) DID generally have no ambivalence about the experience of their purported multiplicity (Draijer & Boon 1999).

The recommendation of the inclusion of pathological possession in criterion A—as well as greater detail on the descriptors of DID and clarification that identity alteration need not be witnessed by an observer—was made because the absence of these elements was judged to contribute to the misdiagnosis of DID. This misdiagnosis has bred an overuse of the diagnosis "dissociative disorder not otherwise specified" (DDNOS). Studies have shown that 40% of dissociative disorder diagnoses are for DDNOS (Dell 2009, Spiegel et al. 2011a), an unacceptably high rate of NOS diagnosis in any DSM diagnostic category (Kupfer et al. 2002). In short, adding language on possession is a key part of a concerted effort by the work group to reduce the misdiagnosis of DID (and the overdiagnosis of DDNOS). This in turn is expected to lead to improved case detection, better treatment outcome, and more culturally relevant research studies of disorder prevalence and treatment outcome (Spiegel et al. 2011a).

Prevalence of pathological possession. With the exception of Turkey and India, there are no data on the community prevalence of pathological possession in most countries. An epidemiological study of a representative sample from a town in central Turkey using the Dissociative Diagnostic Interview Schedule (Ross et al. 1989) found much higher rates of DSM-IV DID (1.1%) and DDNOS-Example 1 (4.1%) than dissociative trance disorder (0.6%) (Şar et al. 2007). Dissociative trance disorder is a diagnosis included in the DSM-IV appendix that includes both

pathological possession and pathological trance without possession (Am. Psychiatr. Assoc. 2000). On the other hand, the period prevalence of pathological possession in rural Indian communities has been estimated at 0.97% (over six months) to 3.5% (over one year), depending on the region, the sample, and the method of assessment (Carstairs & Kapur 1976, Venkataramaiah et al. 1981). The lower number may be a better estimate of endemic prevalence because the higher number (3.5%) was obtained in the midst of an epidemic of pathological possession. The reported prevalence of voluntary (normal) possession trance contributed an additional 0.2% to 1.8% over six to twelve months in the same studies.

Pathological possession is also common among outpatients with dissociative disorder in India. Saxena and colleagues reviewed data on more than 4,000 Indian outpatients; 104 were diagnosed with a dissociative disorder (Das & Saxena 1991). Only 5% to 10% of these received a specified dissociative disorder diagnosis according to DSM-III or DSM-III-R (there were no diagnoses of DID). By contrast, 85.5% of dissociative disorder cases were covered by the specified dissociative diagnoses in the tenth revision of the *International Classification of Diseases* (ICD-10; World Health Org.). Seventy-four percent of patients had dissociative convulsions, 9.5% had trance and possession disorders, and 2% had dissociative movement disorder (Das & Saxena 1991).

Implications for treatment. Inclusion of pathological possession in DID criteria is expected to improve treatment outcome of individuals with possession-form DID who would have received a diagnosis of DDNOS in DSM-IV-TR. The treatment of DID has been well conceptualized and studied, as opposed to that of the more heterogeneous DDNOS group (Brand et al. 2009). Possession-form language in DID criteria may facilitate effective referral and treatment of this type of DID. In addition, linking possession-form and nonpossession-form DID should allow clinicians who have limited experience with possession-form DID to deepen their understanding of DID by acquiring new perspectives on how it is treated in other cultures. This should contribute to the development of novel strategies for treating all DID cases.

Treatment of possession-form DID by indigenous healers frequently resembles the psychotherapy of nonpossession-form DID. In both Western and indigenous therapy, the therapist addresses alternate aspects of the person's identity in order to "give voice" to their point of view and clarify the circumstances of the distress. The evolution of these components of identity over time and their eventual change to a more adaptive configuration or unification constitute one major basis of improvement (Krippner 1987, Martínez-Taboas 2005, Spiegel et al. 2011a). Martínez-Taboas (2005), for example, describes excellent results with a combination of culturally adapted cognitive, behavioral, and experiential psychotherapies.

However, some ritual therapies for possession-form DID are based instead on an attempted eviction of the alternate identity. There have been repeated incidents of fundamentalist clinicians "treating" possession-form DID as a demonic possession that requires an exorcism (Bowman 1993, Fraser 1993). Case series of such incidents report that many of these individuals with DID considered exorcism to be congruent with their religious and cultural belief systems. Despite this congruence, however, their exorcisms produced markedly poor outcomes in more than two-thirds of these individuals. These outcomes included significant worsening of dissociative and PTSD symptoms, suicide attempts, hospitalizations, and significant damage to the individuals' personal religious beliefs and sense of spirituality. Psychotherapeutic treatment of such individuals with possession after failed exorcism has been effective (Spiegel & Fink 1979). Bull and colleagues (1998) found that about half of their DID sample that underwent an exorcism had a more positive outcome, especially if it was undertaken in the context of a culturally congruent psychotherapy for

Table 2 Dissociative amnesia (formerly psychogenic amnesia) in DSM-IV-TR

A	The predominant disturbance is one or more episodes of inability to recall important personal information, usually of a traumatic or stressful nature, that is too extensive to be explained by ordinary forgetfulness.
B	The disturbance does not occur exclusively during the course of dissociative identity disorder, dissociative fugue, posttraumatic stress disorder, acute stress disorder, or somatization disorder and is not due to the direct physiological effects of a substance (e.g., a drug of abuse, a medication) or a neurological or other general medical condition (e.g., amnestic disorder due to head trauma).
C	The symptoms cause clinically significant distress or impairment in social, occupational, or other important areas of functioning.

DID. However, these investigators recruited subjects by asking specifically for those with positive responses to exorcism.

Summary and Conclusions

Cross-cultural data clearly show that persons with pathological possession report the same core dissociative symptoms as do persons with DID (i.e., involuntary alteration of identity and amnesia). Many persons with DID report that they feel or have felt like they are possessed. These data suggest that possession-form DID and nonpossession-form DID are variants of one another.

Including pathological possession in DSM-5 as a variant of nonpossession-form DID is expected to help bridge the counterproductive and culturally insensitive rift between disorders in the DSM and their (unrecognized) counterparts among persons from diverse cultural backgrounds. The alternative is likely treatment delay and the use of ineffective (or sometimes frankly deleterious) approaches to this condition. Moreover, clinicians and researchers unaware of possession-form DID may deepen their understanding of DID by acquiring new perspectives on DID from other cultures, leading to improved treatments.

DISSOCIATIVE AMNESIA

Symptomatology

Amnesia is a diagnostic criterion in four DSM-IV-TR dissociative disorders (dissociative amnesia, dissociative fugue, DID, and DDNOS-Example 1) (**Tables 2** and **3**) and a component diagnostic criterion in acute stress disorder and PTSD. Amnesia involves a deficit in functioning episodic memory such that information that was presumably encoded cannot be retrieved under normal circumstances (Kritchevsky et al. 1997), although it is potentially reversible using

Table 3 Dissociative fugue in DSM-IV-TR

A	The predominant disturbance is sudden, unexpected travel away from home or one's customary place of work, with inability to recall one's past.
B	Confusion about personal identity or assumption of a new identity (partial or complete).
C	The disturbance does not occur exclusively during the course of another dissociative disorder and is not due to the direct effects of a substance or a general medical condition.
D	The symptoms cause clinically significant distress or impairment in functioning.

techniques such as hypnosis (Butler et al. 1996, Yovell et al. 2003). The deficit in retrieval frequently involves memory for stressful or traumatic events but may also involve memory for everyday occurrences.

Dissociative Fugue as a Subtype

The major change regarding dissociative amnesia proposed in DSM-5 is the reclassification of dissociative fugue as a subtype of dissociative amnesia rather than a separate diagnosis. This is due to the prominence of amnesia in dissociative fugue; the fact that other fugue symptoms, confusion about personal identity, assumption of a new identity, and bewildered wandering, do not consistently occur; and the relative rarity of the disorder.

Including fugue as a subtype does create an explicit link between dissociative amnesia and identity disturbance. A consistent sense of personal continuity is achieved through the maintenance of a consistent stream of memory, a kind of smoothing function under which disparate experiences under a common heading of personal integrity and identity are subsumed (Gergen 1991, Spiegel 1990). The accomplished integrated identity is yet subject to disruption through trauma, hypnotic influences, or disjunctions in information-processing strategies (Kihlstrom 1987, Spiegel & Cardeña 1991). However, implicit memories persist across dissociative states that contain different stores of explicit memories (Elzinga et al. 2003). This suggests that information not readily available to consciousness may nonetheless influence it: Out of sight is not out of mind (Spiegel 2006, Spiegel & Cardeña 1991). In addition, some studies show that despite reports of memory gaps, more explicit information is shared across identity states than subjects are aware of (Kong et al. 2008). Thus explicit memory retrieval failures in dissociation likely overrepresent information unavailability. As an example, traumatic experiences not fully available to consciousness may nonetheless exert influence on it: A woman raped in an elevator may still avoid elevators despite an inability to explicitly recollect the assault. Despite the fact that dissociated information is inaccessible to conscious recall, that information still influences the person's emotional reactions and behavior.

This can be mistakenly interpreted as indicating that the person's reported amnesia is feigned or malingered. Indeed, this same phenomenon characterizes some forms of organic amnesia—for example, hippocampal lesions that spare access to information learned long ago and stored in "gist" format in the cortex (Moscovitch et al. 2006, Winocur et al. 2010), and "blindsight," the phenomenon in which people with cortical blindness discriminate among visual stimuli at levels greater than chance despite an inability to provide any conscious description of what they are looking at (Lamme 2001). Similarly, dissociated information may become temporarily available to conscious recall and then redissociated, usually with amnesia for the period that the memory appeared to be consciously available. This has also been thought to indicate factitious or malingered memory impairment. However, dissociative amnesia can be understood as a form of psychological inhibition (Lanius et al. 2010a) that may vary depending on the level of stress facing the individual (Loewenstein 1991).

Dissociative amnesia may also take the form of other dissociative symptoms, such as depersonalization. Individuals with dissociative amnesia may also describe derealized memory, in which they experience some of their memories as if they are detached from a sense of personal ownership; they recall the events, but as if they had been learned in a secondhand way and not personally experienced. Individuals with dissociative amnesia may also report conscious attempts to not think about distressing and/or traumatic events, which over time may lead to more automatic processes of dissociative amnesia (Koutstaal & Schacter 1997, Loewenstein & Putnam 2004). Neurobiological studies of nonclinical samples given amnesia instructions show neural

Table 4 Depersonalization disorder criteria in DSM-IV-TR

A	Persistent or recurrent experiences of feeling detached from, and as if one is an outside observer of, one's mental processes or body (e.g., feeling like one is in a dream; sense of unreality of self, perceptual alterations; emotional and/or physical numbing; temporal distortions; sense of unreality of surroundings).
B	During the depersonalization experience, reality testing remains intact.
C	The depersonalization causes clinically significant distress or impairment in social, occupational, or other important areas of functioning.
D	The depersonalization experience does not occur exclusively during the course of another mental disorder, such as schizophrenia, panic disorder, acute stress disorder, or another dissociative disorder, and is not due to the direct physiological effects of a substance (e.g., a drug of abuse, a medication) or a general medical condition (e.g., temporal lobe epilepsy).

network activation similar to that shown by clinical subjects with dissociative forms of PTSD and dissociative amnesia (Anderson et al. 2004).

DEPERSONALIZATION/DEREALIZATION DISORDER

Depersonalization and Derealization

Depersonalization and derealization (**Table 4**) are typical dissociative symptoms involving a failure of perceptual integration associated with a subjective experience of detachment from one's self and/or the world around (Simeon et al. 2003b). There is evidence that individuals with prominent derealization alone do not significantly differ from those with depersonalization accompanied by derealization in any respect, including demographics, precipitants, illness characteristics, and comorbidity (Simeon 2009). Thus, derealization has been proposed as an equal component of depersonalization disorder in DSM-5, resulting in the new name of depersonalization/derealization disorder. These symptoms are highly prevalent in other dissociative conditions ranging from peritraumatic dissociation to DID, although they also occur in mood, anxiety (e.g., panic attacks), and psychotic disorders. The primary distinctions from other dissociative disorders involve (*a*) the emphasis on perceptual dissociation rather than dissociation of memory or identity and (*b*) the proximity, type, and severity of traumatic antecedents. There is considerable evidence that dissociation occurs at high rates in the peritraumatic period (Koopman et al. 1996, Marmar et al. 1994) and that early life trauma, in particular complex trauma, is strongly associated with later dissociative disorders (Dalenberg et al. 2012). There are three fundamental dissociative ways of responding to a traumatic experience: detach from it (depersonalization/derealization), forget it (amnesia), or separate the memory of the experience from one's present identity (dissociative flashback, DID) (Dalenberg et al. 2012; Koopman et al. 1994, 1995, 1996; Marmar et al. 1994). Depersonalization and derealization provide an immediate means of modulating the acute perceptual impact of a traumatic experience, but if they persist, then over time they can become disturbing persistent or recurrent symptoms that hamper processing of past and present experiences.

Neurobiology

Neuroimaging studies show patterns of brain activation and inhibition consistent with other dissociative disorders. Three consistent findings have emerged: (*a*) altered activation of posterior cortical sensory association areas, primarily the inferior parietal lobe; (*b*) prefrontal activation; and

(*c*) limbic inhibition (Simeon et al. 2003a). These findings are similar to observations regarding the dissociative subtype of PTSD (Lanius et al. 2002, 2005, 2010a, 2012), those reported for the "apparently normal personality" of DID (Reinders et al. 2006), and findings of inferior parietal lobe involvement in simulated out-of-body experiences in healthy volunteers (Arzy et al. 2006). These observations differ from those seen in mood and anxiety disorders. They highlight a neural mechanism common among dissociative disorders—prefrontal activation and limbic inhibition. Suppression of emotion would plausibly inhibit the neural connectivity that would facilitate integration of information about memory, identity, and consciousness.

The neurochemistry of depersonalization disorder is consistent with a pattern of hypothalamic-pituitary-adrenal (HPA) hyperarousal involving higher cortisol levels and reduced feedback inhibition (Simeon et al. 2001, 2007), even when controlling for the presence of depression or depressive symptoms. However, there is blunted cortisol reactivity to psychosocial stress (Simeon et al. 2007), unlike in depression, in which baseline hypercortisolemia is accompanied by heightened HPA axis responsivity to stressors (Giese-Davis et al. 2006), or in PTSD, in which there is generally lower HPA activation but heightened response to stressors (Yehuda et al. 2004, 2006). Although dissociation, especially when accompanied by anxiety, is usually associated with heightened noradrenergic tone, norepinephrine declines as the severity of depersonalization increases (Simeon et al. 2003b). Depersonalization disorder is also associated with blunted skin conductance responses to emotional stimuli (Sierra et al. 2002), consistent with autonomic blunting in depersonalization disorder. Although not directly studied in the disorder, other neurotransmitter systems associated with depersonalization/derealization symptoms include the endogenous cannabinoid, kappa opioid, and N-methyl D-aspartate systems (Sierra 2008). These neurobiological findings are consistent with the phenomenology described above linking depersonalization/derealization to stress buffering by inhibiting limbic arousal, reducing the spread of trauma-related associations, and blocking HPA and noradrenergic arousal.

THE DISSOCIATIVE SUBTYPE OF POSTTRAUMATIC STRESS DISORDER

The following section of the review outlines the rationale and evidence for the dissociative subtype of PTSD with a focus on (*a*) latent class, taxometric, and confirmatory factor analyses; (*b*) neurobiological mechanisms; and (*c*) treatment implications.

Background

Evidence for a dissociative subtype of PTSD has recently been described (Ginzburg et al. 2006; Lanius et al. 2010a, 2012; Steuwe et al. 2012; Wolf et al. 2012a,b). A recent epidemiological study involving 25,018 people from 16 countries in a World Mental Health Survey found that 14.4% of those with PTSD had the dissociative symptoms of depersonalization and derealization. They were characterized by higher levels of re-experiencing symptoms, onset of PTSD in childhood, high trauma exposure and childhood adversities, severe role impairment, and suicidality (Stein et al. 2013). Thus a dissociative subtype of PTSD is currently being considered for inclusion in DSM-5 to address symptoms of depersonalization and derealization exhibited among a subset of patients with PTSD. The definition of dissociation included in the Introduction section of the present review clearly demonstrates that dissociation is a complex phenomenon that comprises a host of symptoms that are usually studied as a single construct (Bryant 2007, Hagenaars et al. 2010). Researchers have therefore argued for the deconstruction of the term "dissociation" into distinct factors, including depersonalization, derealization, time distortion, and dissociative

flashbacks, to name just a few (Bryant 2007). Neurophenomenology is one emerging approach to studying the different facets of dissociation (Frewen & Lanius 2012). The basic concept behind neurophenomenology is that first-person experience is combined with the measurement of neurophysiological processes (Varela 1996). It combines real-time collection and analysis of subjective experiential reports with objective measures of brain activity and thus allows the examination of different dissociative experiences such as depersonalization, derealization, flashbacks, amnesia, and the perception of self.

The proposed dissociative subtype of PTSD has focused on depersonalization and derealization for reasons concerning existing psychological measurement and neurobiological evidence. The majority of studies to date that have investigated evidence for the dissociative subtype (Cloitre et al. 2012; Lanius et al. 2010a; Resick et al. 2012; Steuwe et al. 2012;Wolf et al. 2012a,b) have utilized the Clinician Administered PTSD Scale (CAPS) (Blake et al. 1995) and the Trauma Symptom Inventory Dissociation Subscale (TSI-DIS) (Briere 1995). These scales predominantly focus on symptoms of depersonalization and derealization, although they also assess psychogenic amnesia. Moreover, neurobiological evidence for a dissociative subtype has focused on brain activation patterns underlying symptoms of depersonalization and derealization. However, the relationship of depersonalization and derealization symptoms to specific core PTSD symptoms that are conceptually related to dissociation, including dissociative flashbacks, psychogenic amnesia, and emotional numbing, remains to be elucidated and will need to be an ongoing focus of investigation.

Rationale for the Dissociative Subtype of PTSD

The addition of a dissociative subtype of PTSD in the DSM-5 will be beneficial for several reasons. First, several studies using latent class, confirmatory factor, and taxometric analyses have demonstrated increasing evidence for a dissociative subtype of PTSD in both veteran and civilian PTSD samples (Ginzburg et al. 2006; Lanius et al. 2010a; Putnam et al. 1996; Steuwe et al. 2012; Waelde et al. 2005; Wolf et al. 2012a,b). Second, a neurobiological model has been described in which the dissociative subtype is viewed as a form of emotion dysregulation that involves emotional overmodulation mediated by midline prefrontal inhibition of the limbic regions, including the amygdala. Third, preliminary evidence suggests that individuals with symptoms of depersonalization and derealization can have a differential response pattern to current cognitive behavioral treatments for PTSD as compared to those without these symptoms (Cloitre et al. 2012, Resick et al. 2012). Adding a dissociative subtype of PTSD may therefore allow symptoms of depersonalization and derealization to be considered during treatment planning. Fourth, the addition of this subtype will further much needed research into the etiology, epidemiology, neurobiology, and treatment response of this PTSD subtype. Such research is necessary to deepen our understanding of the environmental and genetic contributions of this subtype and to expand our understanding of its neurobiological characteristics. Moreover, it will be crucial to further elucidate the effects of the subtype on current PTSD treatments.

Evidence from Taxometric, Signal Detection, Latent Class, and Confirmatory Factor Analyses

Current research suggests that dissociation is associated with unresponsive parenting and psychological trauma as well as PTSD (e.g., Dalenberg et al. 2012; Ginzburg et al. 2006; Lanius et al. 2010b; Ogawa et al. 1997; Steuwe et al. 2012; Wolf et al. 2012a,b). Although not all individuals who meet criteria for PTSD have high levels of dissociation, most individuals with high levels of dissociative symptomatology meet criteria for PTSD. For example, taxometric analyses have

demonstrated that in a sample consisting of 316 trauma-exposed male veterans with a current diagnosis of PTSD, 32% belonged to the dissociative taxon. Among those in the dissociative taxon, 80% met criteria for PTSD as compared to only 18.2% of the nontaxon individuals (Waelde et al. 2005). In another study, women seeking treatment for childhood sexual abuse were divided into high-dissociation and low-dissociation groups using signal detection analyses. The group with PTSD included 77% of the high-dissociation subgroup and 30% of the low-dissociation subgroup, whereas the non-PTSD group included 23% of the high-dissociation subgroup and 70% of the low-dissociation subgroup (Ginzburg et al. 2006). Similarly, 89% of a general population sample who showed high levels of dissociation had PTSD as compared to only 22% of individuals with low levels of dissociation (Carlson et al. 2012).

Studies using taxometric analyses have also argued that certain forms of dissociation can be conceptualized as dimensional as opposed to others that are distributed categorically (Waller et al. 1996). Taxometric procedures found a dissociation "taxon" that described a group of individuals reporting pathological levels of dissociation (Waelde et al. 2005) among 316 veterans. One-third of those who had PTSD fell into the dissociative taxon. Individuals in the taxon-positive group were more likely to suffer from PTSD (80% versus 18% in the taxon-negative group) in addition to meeting criteria for dysthymia and major depression as compared to those who were not in the dissociative taxon. The dissociative taxon was also associated with more severe PTSD symptoms. However, two additional taxometric studies examining veteran samples concluded that dissociation was on a continuum rather than being a dimensional, taxonic variable (Forbes et al. 2005, Ruscio et al. 2002).

Wolf and colleagues (2011b) have recently utilized latent class analysis to examine PTSD and dissociative symptomatology in 492 veterans and their partners, not all of whom met criteria for PTSD. Of the individuals who met CAPS criteria for PTSD, 12% formed a dissociative group characterized by high PTSD symptoms and elevated dissociation scores, specifically derealization and depersonalization, as well as significantly more flashbacks. Although the classic PTSD symptom clusters were strongly intercorrelated, they did not correlate as highly with the derealization or depersonalization items (both r's $= 0.27$, p values <0.001). Wolf et al. (2012a) further validated these findings using latent profile analyses on symptoms of PTSD and dissociation (depersonalization, derealization, and reduction in awareness of surroundings) among 360 male Vietnam War veterans with combat-related PTSD and 284 female veterans and active duty service personnel with PTSD and a high base rate of exposure to sexual trauma. Similar to the first study by Wolf and colleagues, the latent profile analysis yielded evidence for a three-class solution in both samples, including moderate and high PTSD classes as well as a class marked by high PTSD severity coupled with dissociative symptoms including depersonalization, derealization, and a reduction in awareness of surroundings. In the latter study, approximately 15% of the male sample and 30% of the female sample were classified into the dissociative subtype. Women (but not men) with the dissociative subtype of PTSD exhibited higher rates of axis II comorbidity, including avoidant and borderline personality disorder. In a civilian PTSD sample consisting predominantly of women with a history of childhood trauma, Steuwe et al. (2012) also demonstrated evidence for a dissociative subtype of PTSD using latent class and confirmatory factor analyses. Similar to the findings of Wolf et al. (2012a), latent class analysis yielded three groups, one of which was uniquely characterized by high derealization and depersonalization symptoms and accounted for 25% of the sample. Individuals in this dissociative subgroup also showed a higher number of comorbid Axis I disorders and a more significant history of childhood abuse and neglect. Furthermore, confirmatory factor analyses suggested the acceptance of a five-factor solution in which depersonalization and derealization symptoms are distinct from but correlate significantly with the core PTSD symptom clusters. Additional research using a wider range of items to assess dissociation is needed

to develop a sophisticated understanding of the relationship between the various dissociative and PTSD symptoms over time and across trauma types.

Relationship of PTSD Dissociative Subtype Symptoms to Flashbacks and Dissociative Amnesia

Studies exploring the relationship between symptoms of depersonalization and derealization to other dissociative PTSD symptoms (e.g., flashbacks and dissociative amnesia) are just emerging. Steuwe et al. (2012) reported results from a confirmatory factor analysis that examined a five-factor model of PTSD, including the well-accepted four factors reported by King et al. (1998) in addition to a dissociation factor consisting of symptoms of depersonalization and derealization. Such a five-factor model showed the best overall fit. Interestingly, fit was only slightly poorer in a model recognizing a third symptom as dissociative, namely PTSD item B3 (flashbacks). The latter supports the notion of flashbacks as dissociative phenomena. On the other hand, models that relegated depersonalization and derealization symptoms to loadings on any of the four previously recognized PTSD factors provided comparably poorer fit. These findings suggest that symptoms of derealization and depersonalization cannot be better explained by any core PTSD cluster than by a dissociative factor and give credence to the importance of distinguishing depersonalization and derealization symptoms as a distinct construct. Nonetheless, it is important to note that the dissociative factor consisting of symptoms of depersonalization and derealization is not independent from the other four core PTSD factors, supporting the assumption of dissociation as a component of PTSD (Carlson et al. 2012). Future research will need to examine the relationships among symptoms of depersonalization, derealization, dissociative amnesia, and emotional numbing.

Neurobiological Evidence

The neurobiology underlying dissociative symptomatology: emotional under- and overmodulation. Research to date has examined the neuronal underpinnings of re-experiencing/hyperarousal and depersonalization/derealization dissociative responses in PTSD using the script-driven symptom provocation paradigm (Bremner 1999; Lanius et al. 2002, 2005, 2006). This paradigm involves patients creating a narrative of their traumatic experience including as many sensory details as possible. During a brain-imaging scan, these narratives are then read back to the patients, who are instructed to recall the traumatic memory as vividly as possible. Researchers have demonstrated that approximately 70% of patients re-experience their trauma and show a concomitant increase in heart rate. The remaining 30%, however, exhibit states of depersonalization and derealization often associated with no significant concomitant increase in heart rate (Lanius et al. 2005, 2010a).

Emotional undermodulation: failure of corticolimbic inhibition. Emotional undermodulation refers to symptoms of re-experiencing and hyperarousal often associated with cluster B symptoms of PTSD. The PTSD patients who responded to hearing their trauma narratives in the functional magnetic resonance imaging (fMRI) scanner with re-experiencing and hyperarousal symptoms, as measured by the Response to Script-Driven Imagery Scale (Hopper et al. 2007), exhibited abnormally low activation in the medial prefrontal and the anterior cingulate cortex. These brain regions play a role in modulating arousal and regulating emotion (Etkin & Wager 2007, Lanius et al. 2006). Consistent with the notion of impaired cortical modulation of affect and arousal, increased activation of the limbic system, particularly the amygdala, has often been demonstrated in PTSD patients in response to exposure to traumatic reminders and to masked

fearful faces (Etkin & Wager 2007). Neuroimaging investigations in PTSD patients have also demonstrated inhibitory influence of the prefrontal cortex on the amygdala. Specifically, studies using positron emission tomography have shown a negative correlation between blood flow in the left ventromedial prefrontal cortex and the amygdala, and negative correlations between medial prefrontal cortex and the amygdala during exposure to fearful faces in individuals with PTSD (Shin et al. 2005). Decreased activation of medial prefrontal regions observed in the re-experiencing/hyperaroused PTSD subgroup is therefore consistent with failed inhibition of limbic reactivity, including the amygdala, and is associated with re-experiencing/hyperaroused emotional undermodulation (for a review, see Francati et al. 2007).

Emotional overmodulation: excessive corticolimbic inhibition. Emotional overmodulation refers to dissociative symptoms, including depersonalization and derealization, which usually involve a distancing from an emotional experience. In contrast to the re-experiencing/hyperaroused group described above, the group experiencing symptoms of depersonalization and derealization, as measured by the Clinician Administered Dissociative Symptom Scale (Bremner et al. 1998) during the trauma script-driven imagery procedure, exhibited abnormally high activation in the anterior cingulate cortex and the medial prefrontal cortex. This subgroup of PTSD patients can therefore be considered as experiencing emotional overmodulation in response to traumatic memory recall accompanied by increased activation of medial prefrontal structures and concomitant hyperinhibition of limbic regions, including the amygdala. It is important to note that individuals with PTSD may show both over- and undermodulated response patterns at different time points, although only patients exhibiting the dissociative subtype show severe and frequent overmodulated emotional responses characterized by impairing levels of depersonalization and derealization. As reviewed above, different traumatic experiences, including childhood trauma and military trauma, have been related to the overmodulated response observed in the dissociative subtype of PTSD.

An investigation by Felmingham et al. (2008) provides further evidence for the corticolimbic inhibition model of dissociation. This study compared brain activation patterns during the processing of consciously and nonconsciously perceived fear stimuli. PTSD patients with high state-dissociation scores during the neuroimaging procedure as measured by the Clinician Administered Dissociative Symptom Scale showed enhanced activation in the ventral prefrontal cortex during conscious fear processing as compared to patients with low state-dissociation scores. During processing of nonconscious fear, high dissociative symptomatology at the time of the scan in PTSD was associated with increased activation in the bilateral amygdala, insula, and left thalamus as compared to low state-dissociation. The authors propose that dissociation, including states of depersonalization and derealization, is an emotion regulatory strategy during conscious processing of threat that is employed to cope with extreme arousal in PTSD through hyperinhibition of limbic regions.

Implications for Treatment

Preliminary evidence suggests that high levels of dissociation appear to be associated with a differential response pattern to some conventional PTSD treatments. It has been suggested that dissociation during traumatic memory processing may interfere with habituation, a critical process for resolving PTSD during exposure-based treatments (Foa & Kozak 1986, Jaycox et al. 1998).

One open trial study of prolonged exposure found that PTSD subjects who exhibited high dissociative symptomatology were more likely to maintain their diagnosis of PTSD at the end

of treatment than were low dissociative symptomatology PTSD patients (69% versus 10%, respectively), even though both groups showed an equal improvement in PTSD symptoms posttreatment (Hagenaars et al. 2010). In another study, women with childhood trauma-related PTSD were randomized to one of three psychotherapeutic interventions (Cloitre et al. 2012). The treatment of principal interest included a form of modified exposure therapy [labeled narrative story telling (NST)] preceded by a cognitive-behavioral intervention intended to facilitate the development of emotion regulation and interpersonal effectiveness skills [labeled skills training in affective and interpersonal regulation (STAIR)], together labeled STAIR-NST. The STAIR-NST model was compared with two other forms of treatment delivery: (*a*) supportive counseling (SC) followed by NST and (*b*) STAIR followed by SC. The STAIR-NST model had already been shown to be the most advantageous delivery model for most patients in a prior report (Cloitre et al. 2010). A reanalysis of the data, however, showed that in women reporting lower pretreatment (baseline) levels of dissociative symptoms, outcomes across the three delivery models in terms of dissociative symptoms were largely equivalent. In comparison, in women with high pretreatment levels of dissociation, dissociation was reduced most effectively by STAIR-NST; the effect was most obvious at six-month posttreatment follow-up. In addition, for those with higher posttreatment dissociation scores, the greatest further decreases in PTSD symptoms over the course of follow-up occurred in the STAIR-NST group.

A study conducted by Resick and colleagues (2012) randomized women with PTSD related to interpersonal violence to one of three treatments: (*a*) cognitive therapy alone, (*b*) written exposure therapy alone, or (*c*) cognitive processing therapy. The latter intervention combines elements of cognitive therapy and written exposure therapy (Resick et al. 2012). Outcomes were determined not only in terms of PTSD symptoms but also in terms of dissociative symptoms. Resick and colleagues found that at high pretreatment baseline levels of dissociation, cognitive processing therapy achieved quicker and overall better outcomes on PTSD symptom severity than did cognitive therapy alone, with outcomes for written exposure therapy alone producing outcomes that did not significantly differ from either of the two conditions. Importantly, group differences were observed only for certain dissociation measures (Multiscale Dissociation Inventory depersonalization subscale but not Trauma Symptom Inventory dissociation subscale), suggesting the need to examine different factors of dissociation on treatment outcome.

Additional evidence for the influence of dissociation, including symptoms of depersonalization and derealization, on emotional learning stems from a study examining a classical conditioning paradigm to investigate the effects of state dissociation on acquisition and extinction processes in patients with borderline personality disorder (Ebner-Priemer et al. 2009). Individuals with borderline personality disorder and high levels of state dissociation did not show differences in skin conductance and arousal during acquisition and early extinction. In contrast, patients with borderline personality disorder and low levels of state dissociation (as well as healthy subjects) showed higher skin conductance and arousal during these processes. These findings suggest that emotional, amygdala-based learning processes appear to be hindered by state dissociation, resulting in the alteration of acquisition and extinction processes. The investigators point out that dissociative patients should therefore be closely monitored in exposure-based psychotherapy since they may respond differently to exposure treatment (Ebner-Priemer et al. 2009). Furthermore, dissociation, including symptoms of depersonalization and derealization, has recently been demonstrated to be a negative predictor of psychotherapy outcome in borderline personality disorder (Kleindienst et al. 2011). The findings described above are consistent with the notion that dissociative symptoms can interfere with the cognitive and affective processes thought to mediate the efficacy of psychotherapy for PTSD (e.g., habituation, cognitive restructuring, and emotion regulation).

Summary and Conclusions

In summary, a subgroup of PTSD patients experiences symptoms of depersonalization and derealization. Taxometric, latent class, and confirmatory factor analyses have revealed a taxon/subtype/group of individuals exhibiting symptoms of depersonalization and derealization in veteran and civilian samples ranging in prevalence from 12% to 30% of individuals with PTSD (Bernstein & Putnam 1986; Steuwe et al. 2012; Waelde et al. 2005; Wolf et al. 2012a,b). Individuals in this group also endorsed elevations on items assessing flashbacks (Wolf et al. 2012b), exhibited greater exposure to childhood abuse and neglect, and showed increased Axis I comorbidity (Steuwe et al. 2012; Wolf et al. 2012a,b). The relationship of depersonalization and derealization symptoms to specific core PTSD symptoms that are conceptually related to depersonalization and derealization, including dissociative flashbacks, psychogenic amnesia, and emotional numbing is largely unknown at this time and should be an ongoing focus of research. Neurophenomenology, an emerging approach that combines real-time collection and analysis of subjective experiential reports with objective measures of brain activity, may aid in determining the mechanisms underlying these conceptually related phenomena (Frewen & Lanius 2013). According to the corticolimbic model of dissociation (Lanius et al. 2010a), the dissociative subtype of PTSD may involve emotion dysregulation in the form of emotional overmodulation mediated by midline prefrontal inhibition of limbic regions, including the amygdala. Research is beginning to suggest that symptoms of depersonalization and derealization can lead to a differential response to current cognitive-behavioral treatment designed for PTSD (Cloitre et al. 2012, Jaycox et al. 1998, Lanius et al. 2010a, Resick et al. 2012). The addition of a dissociative subtype to DSM-5 not only allows a more careful analysis of different phenotypes of PTSD, but should also lead to research that deepens our understanding of the prevalence, symptomatology, neurobiology, and treatment of individuals suffering from the dissociative subtype of PTSD.

CONVERSION, DISSOCIATION, AND SOMATIZATION

The long history of research on the relationship between pseudoneurological symptoms and the dissociative disorders reveals many areas of commonality and overlap (**Table 5**) (Brown 2002, 2004; Brown & Lewis-Fernández 2011; Cardeña 1994; Interian et al. 2004; Kihlstrom 1992; Moene et al. 2000; Nemiah 1991; Nijenhuis et al. 2004). Dissociative processes appear to underlie nonepileptic seizures, which can be reversed using appropriate hypnotic suggestions (Oakley 1999). Individuals with medically unexplained sensory loss (e.g., blindness, deafness) who process information in the affected modality are temporarily unable to access these sensations consciously due to a lack of integration between implicit and explicit perceptual processes, suggesting a dissociative origin (Holmes et al. 2005). As in other dissociative disorders, physical/sexual abuse and other traumatizing events are common among individuals with pseudoneurological symptoms (Bowman & Markand 1996, Reilly et al. 1999, Roelofs et al. 2002, Şar et al. 2000a) and other medically unexplained symptoms (MUS; Brown et al. 2005, Drossman 1996, Labbate et al. 1998, Pribor et al. 1993, Şar et al. 2004). Relatively high levels of hypnotic suggestibility have been found in both dissociative (Frischholz et al. 1992) and pseudoneurological (Roelofs et al. 2002) symptom patients, although not all studies have found such a relationship with pseudoneurological symptoms (Şar et al. 2000b, Tezcan et al. 2003).

Not all researchers, however, find a close relationship between conversion disorder and dissociative processes. An alternative viewpoint emphasizes the overlap between medically unexplained somatic symptoms and anxiety (excessive concern over medical problems) rather than dissociation (Brown 2006). In fact, there is approximately equal overlap between conversion disorder and the

Table 5 Conversion disorder in DSM-IV-TR

A	One or more symptoms or deficits affecting voluntary motor or sensory function that suggest a neurological or other general medical condition.
B	Psychological factors are judged to be associated with the symptom or deficit because the initiation or exacerbation of the symptom or deficit is preceded by conflicts or other stressors.
C	The symptom or deficit is not intentionally produced or feigned (as in factitious disorder or malingering).
D	The symptom or deficit cannot, after appropriate investigation, be fully explained by a general medical condition, or by the direct effects of a substance, or as a culturally sanctioned behavior or experience.
E	The symptom or deficit causes clinically significant distress or impairment in social, occupational, or other important areas of functioning or warrants medical evaluation.
F	The symptom or deficit is not limited to pain or sexual dysfunction, does not occur exclusively during the course of somatization disorder, and is not better accounted for by another mental disorder.
	Specify type of symptom or deficit:
	With motor symptom or deficit (e.g., impaired coordination or balance, paralysis or localized weakness, difficulty swallowing or "lump in throat," aphonia, and urinary retention). With sensory symptom or deficit (e.g., loss of touch or pain sensation, double vision, blindness, deafness, and hallucinations). With seizures or convulsions: includes seizures or convulsions with voluntary motor or sensory components. With mixed presentation: if symptoms of more than one category are evident.

DSM-IV-TR dissociative and somatoform disorders. The link with dissociation is strongest among individuals with nonepileptic seizures, both in terms of comorbid prevalence and symptomatology (e.g., loss of consciousness and memory).

The complex associations between conversion symptoms, somatic symptoms, and dissociative processes have resulted in disagreement over how to classify conversion disorder. DSM-IV-TR considers it a somatoform disorder, emphasizing the common preoccupation with somatic dysfunction. ICD-10 places conversion disorder with the dissociative disorders, emphasizing a common underlying dissociative mechanism and comorbidity. In this review, we describe the prevalence of conversion disorder, its comorbidity with dissociative disorders, somatic symptom disorders, and other psychiatric disorders, and its underlying neurophysiology.

Comorbidity of Conversion Disorder and Dissociative Disorders

Several studies have found substantial comorbidity between dissociative disorder and conversion disorder. Şar and colleagues (Guz et al. 2004), for example, examined 38 consecutive patients with conversion disorder utilizing the SCID for DSM-III-R, the Somatoform Dissociation Questionnaire (SDQ-20), the Childhood Trauma Questionnaire, and the SCID for DSM-IV Dissociative Disorders (SCID-D). They found that 48% of these patients also met diagnostic criteria for a dissociative disorder (Şar et al. 2004). A history of abuse and neglect was significantly more common among the comorbid conversion and dissociation patients. However, it is worth noting that comorbidity with other disorders was even higher: 78.9% had an anxiety disorder, 76.3% had a somatoform disorder, and 71.1% had an affective disorder. Thus, on the basis of comorbidity alone, conversion and dissociation would not necessarily be considered overlapping disorders. Also in Turkey, Tezcan et al. (2003) examined 59 consecutive conversion disorder patients using

the Dissociative Experiences Scale (DES), the Dissociative Disorders Interview Schedule (DDIS), and the SCID-D. By SCID-D criteria, 30.5% of patients also had a dissociative disorder, 50% of them DID, 44.4% of them DDNOS, and 5% dissociative amnesia. Other common comorbidities were major depression and borderline personality disorder.

In Germany, Spitzer et al. (1999) compared 72 patients with conversion disorder to 96 gender- and age-matched psychiatric patients "suffering from various neurotic disorders" (p. 291). Scores on the DES were twice as high in the conversion disorder group ($p < 0.001$), whereas somatization scores on the SCL-90 were 40%, but not significantly higher ($p < 0.055$) than those in the comparison sample. They concluded that their findings support a "reclassification of the conversion disorders with the dissociative disorders" (p. 293).

Nijenhuis postulates two types of dissociation, somatoform and psychological, and proposes that the somatoform type, analogous to conversion, is more likely after physical and sexual trauma (Nijenhuis et al. 1998, 2003, 2004). Bowman (2006), in a thoughtful and careful review of the literature, notes the common co-occurrence of conversion seizures and dissociation in the wake of trauma. She concurs with Nijenhuis that "conversion is a somatoform manifestation of dissociation" (Bowman 2006, p. 198). Brown et al. (2007) make a similar argument, noting as did Bowman and Isaac & Chand (2006) that the ICD links the two disorders, reserving for somatoform disorders the more anxiety-based preoccupations with physical health, but not including conversion, which is characterized by altered sensorimotor experience rather than anxiety.

Studies using the DDIS (Şar et al. 2007) have found that MUS are extremely common in patients with DID, many of whom also meet diagnostic criteria for DSM-IV somatization disorder (Ross 1989; Ross et al. 1990a,b). Studies using the SDQ-20, which mostly comprises items pertaining to pseudoneurological phenomena (e.g., anesthesias, seizures, paralysis, dysphagia), have found that these symptoms are significantly more common in patients with dissociative disorders than in psychiatric controls and that the severity of these symptoms is correlated with the complexity of the dissociative disorder (Nijenhuis et al. 1998, 1999). Other studies have measured dissociative symptoms in patients with well-documented pseudoneurological symptoms. Tezcan and colleagues (2003) used the SCID-D to diagnose dissociative disorders in mixed pseudoneurological samples, obtaining prevalences of 30.5% and 47.4%, respectively. In the latter study, a concurrent dissociative disorder predicted higher psychiatric comorbidity more generally, including somatization disorder, dysthymic disorder, major depression, borderline personality disorder, self-destructive behavior, suicide attempts, and childhood trauma.

Mixed results have been found in studies using questionnaire measures of dissociation, such as the DES and the Dissociation Questionnaire (DIS-Q). Several studies (Goldstein et al. 2000, Spitzer et al. 1999, Tezcan et al. 2003) have found elevated DES scores in patients with medically unexplained seizures compared to controls. Other studies with similar populations have failed to find comparable associations, however (Alper et al. 1997, Kuyk et al. 1999, Prueter et al. 2002). It is likely that these mixed findings are due to the fact that only some DES and DIS-Q items are related to pseudoneurological illness. According to one recent review, the DES and DIS-Q include items pertaining to two qualitatively distinct categories of dissociative phenomena, detachment and compartmentalization; only the latter is directly linked to pseudoneurological symptoms (Brown 2006, Brown et al. 2007, Holmes et al. 2005, Moene et al. 2000).

Patients with pseudoneurological symptoms often experience other MUS. Mace & Trimble (1996), for example, followed up a group of pseudoneurological patients and found that although only 4% had been initially diagnosed with somatization disorder, 64% met criteria for it 10 years later. Similarly, patients with large numbers of MUS across multiple bodily systems have pseudoneurological complaints as their predominant symptoms (Interian et al. 2004).

Neurophysiology

Possible common mechanisms of neural dysfunction may underlie both dissociative and conversion disorders (Black et al. 2004, Isaac & Chand 2006). In particular, conversion disorder has been linked to dysfunction in orbitofrontal cortex and anterior cingulate cortex (ACC), insula, thalamus, and ventrolateral prefrontal cortex (Mailis-Gagnon et al. 2003). Similar brain regions were involved in an fMRI study of hypnotically induced paralysis (right orbitofrontal cortex, right cerebellum, left thalamus, and left putamen) (Halligan et al. 2000). Recently, high hypnotizability has been shown to be associated with functional connectivity during resting states between the dACC and dorsolateral prefrontal cortex (Hoeft et al. 2012). The ACC has been strongly implicated in hypnotic dissociation of pain (Rainville et al. 1997, 1999, 2002). Thus, dysfunction in brain regions that integrate cognition, affect, and sensation are implicated in both conversion and dissociative symptomatology (Spiegel 2008).

Alternatives for DSM-5

Taken together, these findings provide a relatively consistent picture. Many patients with dissociative disorders report phenomena akin to the pseudoneurological symptoms experienced by patients with DSM-IV conversion disorder, as well as many other MUS. Similarly, one-third to one-half of patients with diagnosed pseudoneurological symptoms meet criteria for an additional dissociative disorder. Patients with pseudoneurological symptoms often do not exhibit high scores on measures of dissociation such as the DES, although this may reflect the fact that DES items encompass a wide range of qualitatively distinct phenomena, only some of which are relevant to somatoform and pseudoneurological illness. Indeed, patients with pseudoneurological symptoms typically score high on measures such as the SDQ-20, which is thought to tap the type of dissociation that is most relevant to this group. Many pseudoneurological patients also report histories of other MUS, with pseudoneurological symptoms often being the predominant complaint in patients with the most severe medically unexplained syndromes. A growing body of research and theory also suggests that patients with dissociative and pseudoneurological symptoms suffer from a failure of integration of sensory information and have other important features in common, such as histories of abuse and high suggestibility.

Although the evidence clearly shows that the overlap between the dissociative and pseudoneurological disorders is considerable, the co-occurrence of pseudoneurological illness with other somatic symptoms is just as impressive. Moreover, there is evidence to suggest that pseudoneurological and other somatic symptoms have similar psychosocial precipitants (e.g., potentially traumatizing events), and there are conceptual grounds for assuming common underlying mechanisms. Furthermore, pseudoneurological symptoms are an important aspect of somatization disorder, the paradigmatic example of a somatoform disorder in DSM-IV. Indeed, the most severe cases of somatization disorder appear to be those characterized by numerous pseudoneurological symptoms. It would clearly be unsatisfactory to assume that pseudoneurological symptoms are dissociative disorders when they occur in isolation but somatoform disorders when they occur alongside other MUS. This is particularly true given that many patients with pseudoneurological symptoms often report new MUS over time.

Alternative fates for conversion disorder in DSM-5 include:

1. Recategorizing conversion disorder as a dissociative disorder, perhaps labeled dissociative sensorimotor disorder.
2. Dividing conversion disorder into somatic and dissociative subtypes, based on evidence that pure motor paralysis has more in common with somatization and that sensory/pseudoseizure disorders are more related to dissociation.

3. Leaving conversion disorder among the somatic symptom disorders, the most likely outcome.

Clearly there is evidence that dissociative failure of integration of consciousness plays an important role in at least some types of conversion disorder. Failure of integration may include on the psychological side identity and memory and on the somatic side sensation, perception, and motor function. This framing of conversion disorder has important implications for treatment (Moene et al. 2003, Nash 2005) because techniques useful in diagnosing and treating dissociation apply to conversion disorder as well (Maldonado & Spiegel 2000, Spiegel 2003). Although conversion symptoms clearly are somatic in nature, they are distinct from many of the other somatic symptom disorders in (*a*) presenting with dysfunction to a greater extent than with anxiety about dysfunction and (*b*) representing failures in the integration of sensory and motor function, similar to dissociative failures in integration of memory and identity.

CONCLUSION

This review has focused on the changes to the dissociative disorders proposed for DSM-5 and the rationales for these changes. The following proposed changes were discussed in detail:

- Including language on pathological possession in DID.
- Incorporating derealization into depersonalization disorder in a new combined diagnosis of depersonalization/derealization disorder.
- Reformulating dissociative fugue as a subtype of dissociative amnesia.
- Adding a dissociative subtype to PTSD.
- Reformulating conversion disorder as a dissociative disorder.

The intent of these diagnostic refinements is to more accurately describe the symptomatology of the dissociative disorders in order to (*a*) reduce the proportion of patients who receive a diagnosis of dissociative disorder not elsewhere classified (the DSM-5 equivalent of DSM-IV-TR DDNOS), (*b*) reflect research advances since the publication of DSM-IV-TR, and (*c*) help clinicians maximize the specificity of the treatments at their disposal for each patient's particular presentation.

Overuse of the diagnosis of DDNOS has been considered one of the major nosological problems of the dissociative disorders field that DSM-5 would need to address (Spiegel & Cardeña 1991). The proposed changes—most directly those to DID and depersonalization disorder—directly confront this issue. The clear intent of the work group has been to reduce the years-long delay patients with these conditions encounter before they are identified and offered effective treatment.

Personalizing treatment to the patient's specific phenomenology of illness is ultimately the most important goal behind these nosological refinements. The dissociative subtype of PTSD illustrates clearly how refining diagnostic criteria can help maximize treatment efficacy. The data presented in this review argue forcefully that treatments should be chosen for individuals with PTSD taking into account their level of dissociative symptomatology. Patients with elevated dissociative symptoms may not respond as well to traditional exposure therapy, apparently because dissociation interferes with amygdala-based learning processes. Specific forms of treatment, such as STAIR-NST and cognitive reprocessing, may be necessary for these high state-dissociation patients. Further research is needed to confirm these findings, but they point to the potential usefulness of careful diagnostic evaluation and the need for precise nosological distinctions such as the proposed PTSD subtype.

In sum, cultural and neurobiological research findings are two important engines for the development of the dissociative disorders nosology, as described for pathological possession on the one

hand, and for the PTSD subtype and depersonalization/derealization disorder on the other. The dissociative disorders show marked cultural variation (Lewis-Fernández et al. 2007). Clarifying the extent of this diversity not only facilitates diagnostic accuracy but also may help identify universal social and psychological processes underlying the development of these disorders. Neurobiologically, dissociation appears to be associated with a pattern of altered activation of posterior cortical sensory association areas, prefrontal cortical activation, and limbic inhibition. Future research will show whether these recent but robust findings can continue to refine our nosological categories and the treatments that derive from them.

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Errata

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