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Fragmented Sleep, Fragmented Mind: The Role of Sleep in Dissociative Symptoms

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Abstract

In psychopathology, dissociation typically refers to a disturbance in the normal integration of thoughts, feelings, and experiences into consciousness and memory. In this article, we review the literature on how sleep disturbances relate to dissociative symptoms and memory failure. We contend that this body of research offers a fresh perspective on dissociation. Specifically, we argue that dissociative symptoms are associated with a labile sleep–wake cycle, in which dreamlike mentation invades the waking state, produces memory failures, and fuels dissociative experiences. The research domain of sleep and dissociation can accommodate the dominant idea in the clinical literature that trauma is the distal cause of dissociation, and it holds substantial promise to inspire new treatments for dissociative symptoms (e.g., interventions that focus on normalization of the sleep-wake cycle). We conclude with worthwhile paths for further investigations and suggest that the sleep–dissociation approach may help reconcile competing interpretations of dissociative symptoms.

Keywords

dissociation, sleep, unusual sleep experiences, sleep deprivation, nightmares, memory, commission errors

Dissociative disorders encompass an array of symptoms associated with alterations in consciousness, ranging from profound amnesia for autobiographical events to equally profound changes in identity and the experience of everyday reality (*Diagnostic and Statistical Manual of Mental Disorders,* 4th ed., revised [*DSM-IV-TR*]; American Psychiatric Association [APA], 2000). An impressive corpus of research has succeeded in elucidating the relation between dissociative symptoms and a gamut of psychological disorders, cognitive processes, and behaviors (for reviews, see Benca, Obermeyer, Thisted, & Gillin, 1992; Giesbrecht, Lynn, Lilienfeld, & Merckelbach, 2008).

The dominant perspective on dissociative symptoms is that they reflect a defensive response to highly aversive events, especially psychological trauma during the formative childhood years (Bremner, 2010; Spiegel et al., 2011; Spitzer, Vogel, Barnow, Freyberger, & Grabe, 2007). We will refer to this perspective as the posttraumatic model (PTM) of dissociation.

In this article, we suggest that sleep disturbances play a potentially important role in accounting for dissociative symptoms and how they relate to highly aversive events. We will argue that there now exists a solid foundation of research to contend that dissociative symptoms are associated with a labile sleep–wake cycle in which dreamlike mentation invades the waking state, produces memory failures, and fuels dissociative experiences. Whereas our article builds on previous contributions (e.g., Giesbrecht et al., 2008; Koffel & Watson, 2009a, 2009b; Watson, 2001) indicating that dissociation and sleep disturbances belong to a common domain, we will also provide the most comprehensive analysis of studies on sleep and dissociation to date in both tabular and narrative form. In so doing, we (a) consider important definitional issues and limitations of the prevailing PTM; (b) describe the main findings and clinical ramifications of studies that examine the sleep–dissociation link; (c) discuss evidence pertaining to the causal relation between sleep and dissociation; and (d) suggest that the studies reviewed may provide a basis for not only understanding the association between highly aversive events and dissociation, but also a rapprochement between the PTM and interpretations that emphasize a nontraumatic etiology of dissociation (Lilienfeld et al., 1999; Spanos, 1996). We conclude with suggestions for extending the sleep–dissociation model

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and call for future research on the link between sleep and dissociation.

Defining Dissociation

The DSM-IV-TR defines dissociation as "a disruption in the usually integrated function of consciousness, memory, identity, or perception of the environment" (APA, 2000, p. 519). In the clinical literature, a distinction is often made between dissociative *states* and dissociative *traits* (e.g., Bremner, 2010; Bremner & Brett, 1997). Whereas state dissociation is viewed as a transient symptom (e.g., acute dissociation during a traumatic event), trait dissociation is viewed as an integral aspect of personality. As dissociative symptoms are prevalent in both nonclinical and clinical populations, dissociation has commonly been conceptualized as ranging on a continuum, from nonpathological manifestations of daydreaming to more severe disturbances typical of dissociative disorders (Bernstein & Putnam, 1986). These disorders include dissociative amnesia (extensive forgetting typically associated with highly aversive events), dissociative fugue (short-lived reversible amnesia for personal identity, involving unplanned travel or wandering), depersonalization disorder (DPD; feeling as though one is an outside observer of one's body and feeling like an automaton or like living in a dream or a movie; an experience technically referred to as derealization), and dissociative identity disorder (DID; experiencing two or more distinct identities that recurrently take control over one's behavior, APA, 2000).

Epidemiological studies among psychiatric inpatients and outpatients have yielded prevalence rates of dissociative disorders in the 4%–29% range (Ross, Anderson, Fleisher, & Norton, 1991; Sar, Tutkun, Alyanak, Bakim, & Baral, 2000; Tutkun et al., 1998; for reviews, see Foote, Smolin, Kaplan, Legatt, & Lipschitz, 2006; Spiegel et al., 2011). Although a recent study of women in the general population of Turkey reported a prevalence rate of 18.3% for lifetime diagnoses of a dissociative disorder (Sar, Akyüz, & Dogan, 2009), studies generally find a much lower prevalence in the general population, with rates on the order of 1%–3% (Lee, Kwok, Hunter, Richards, & David, 2012; Rauschenberger & Lynn, 1995; Sandberg & Lynn, 1992). Variability in prevalence across studies is probably due to methodological and perhaps cultural differences, rather than gender differences, as Sar et al.'s (2009) study might suggest. Indeed, Sandberg and Lynn (1992) found that only 6% of female college students who scored in the top 15% on the *Dissociative Experiences Scale* (DES; Bernstein & Putnam, 1986) could be diagnosed with a dissociative disorder, and none of the students who scored below the mean on the measure qualified for a diagnosis of dissociative disorder. Most important, dissociative symptoms are not limited to the dissociative disorders. Certain diagnostic groups, notably patients with borderline personality disorder, posttraumatic stress disorder (PTSD), obsessive–compulsive disorder (Rufer, Fricke, Held, Cremer, & Hand, 2006), and schizophrenia (Allen & Coyne, 1995; Merckelbach, à Campo, Hardy, & Giesbrecht, 2005; Yu et al., 2010) also display heightened levels of dissociation.

Most authors concur that certain clusters of symptoms (e.g., derealization and depersonalization) are core features of dissociation (Holmes et al., 2005). For example, the *Structured Clinical Interview for DSM-IV Dissociative Disorders* (SCID-D, Steinberg, Cicchetti, Buchanan, Rakfeldt, & Rounsaville, 1994) assesses a set of symptom clusters, including depersonalization, derealization, dissociative amnesia, and alterations in identity/identity confusion.

The DES (Bernstein & Putnam, 1986; for more recent versions, see Carlson & Putnam, 2000; Wright & Loftus, 1999) is the most widely used self-report measure of dissociation. This scale measures dissociation with items such as "Some people sometimes have the experience of feeling as though they are standing next to themselves or watching themselves do something and they actually see themselves as if they were looking at another person," and "Some people find that sometimes they are listening to someone talk and they suddenly realize that they did not hear part or all of what was said." Early studies employing the DES concluded that dissociation can best be described as a multifaceted construct composed of three main factors or dimensions: obliviousness/amnesia, depersonalization/derealization, and imagination/absorption (Carlson et al., 1991). Most authors also agree that the first two dimensions—obliviousness/amnesia and depersonalization/derealization—define the more pathological manifestations of dissociation. Accordingly, key items of the DES that refer to such manifestations have been grouped together on an empirical basis as the *dissociative taxon* (DES-T, Waller & Ross, 1997). Although researchers and theorists have proposed different constructs and classification schemes to define or elucidate the complex nature of dissociation and its diverse manifestations, so far these attempts have done little to help us understand the genesis of dissociative symptoms.

Dissociation and Trauma

One prominent interpretation of the origins of dissociative disorders is that they are the direct result of exposure to traumatic experiences, and that dissociative symptoms can best be understood as mental strategies to cope with or avoid the impact of highly aversive experiences (e.g., Spiegel et al., 2011). We refer to this interpretation as the PTM. According to the PTM, individuals rely on dissociation to escape from painful memories (Gershuny & Thayer, 1999). Once they have learned to use this defensive coping mechanism, it can become automatized and habitual, even emerging in response to minor stressors (Van der Hart & Horst, 1989). The idea that dissociation can serve a defensive function can be traced back to Pierre Janet (1899/1973), one of the first scholars to link dissociation to psychological trauma (Hacking, 1995).

The PTM casts the clinical observation that dissociative disorders are linked to a trauma history in straightforward

causal terms (Gershuny & Thayer, 1999). For example, Gast, Rodewald, Nickel, and Emrich (2001) interpreted the positive correlation between self-reported trauma and dissociative disorders in their clinical sample as follows: "These results give further evidence for the posttraumatic model of dissociative disorders, which conceptualizes dissociative disorders as a result of repeated, severe, and overwhelming traumatic experiences during childhood" (Gast et al., 2001, p. 257). Likewise, Vermetten, Schmahl, Lindner, Loewenstein, and Bremner (2006) found that the DID patients in their subsample ($n = 15$) all suffered from PTSD and concluded: "These results are consistent with the conceptualization of dissociative identity disorder as an extreme form of early-abuse-related PTSD" (p. 633).

The empirical support for this causal interpretation, however, is the subject of intense debate (Bremner, 2010; Giesbrecht, Lynn, Lilienfeld, & Merckelbach, 2010; Kihlstrom, 2005). Whereas a comprehensive review of the literature on trauma and dissociation is outside the scope of this article, we will briefly touch on several limitations of studies investigating the link between trauma and dissociation. One major limitation is that the majority of studies reporting links between self-reported trauma and dissociation are based on cross-sectional designs. The correlational data that these designs yield do not allow for strong causal claims (Merckelbach & Muris, 2002). In addition, the reliance on self-report measures of trauma may be problematic, as individuals suffering from dissociative symptoms typically score high on measures of fantasy proneness, a disposition to engage in extensive and vivid fantasizing. The overlap with fantasy proneness may limit the conclusions that can be derived from self-reports of dissociative individuals (Merckelbach et al., 2005), given that the propensity to fantasize may potentially lead to over- or underreporting of traumatic experiences (see Giesbrecht et al., 2008). Furthermore, individuals scoring high on dissociation report more cognitive failures (i.e., everyday slips and lapses) than individuals scoring low on dissociation. People who frequently make such slips and lapses often mistrust their own cognitive capacities and tend to overvalue the hints and cues provided by others (Merckelbach, Horselenberg, & Schmidt, 2002; Merckelbach, Muris, Rassin, & Horselenberg, 2000). This vulnerability to suggestive information, which may bias or distort memory reports, thus limits conclusions that can be drawn from studies that rely solely on self-reports to investigate the trauma-dissociation link (Merckelbach & Jelicic, 2004).

Even more germane to our discussion, the PTM articulates why, but not how, trauma produces dissociative symptoms. Accordingly, researchers and theorists sensitive to the limitations of the PTM have begun to explore other avenues to understand the potentially complex link between dissociation and trauma. More specifically, some investigators (see Giesbrecht et al., 2008; Watson, 2001) have proposed that dissociative symptoms like derealization, depersonalization, and absorption are associated with sleep-related experiences, due to their dreamlike character, and further noted that sleep-related experiences can mediate or moderate the link between highly aversive events and manifestations of dissociative symptoms. In the remainder of this article, we review the available evidence that provides the basis for our perspective on how sleep-related experiences, in concert with aversive events, might produce or exacerbate dissociative symptoms. Our perspective explicitly acknowledges traumatic experiences as potentially distal causes of dissociation, with sleep disturbances acting as the more proximal cause of dissociation, and contributes to a growing literature that addresses the relation between sleep and dissociation (Giesbrecht et al., 2008, 2010; Koffel & Watson, 2009a, 2009b; Watson, 2001, 2003).

Dissociation and Sleep

The idea that dissociative symptoms such as absorption, derealization, and depersonalization originate from sleep is not entirely new. In the 19th century, double consciousness (or *dédoublement*), the historical precursor of DID (formerly known as multiple personality disorder), was often described as *somnambulism*, which refers to a state of sleepwalking. Patients suffering from this disorder were referred to as *somnambules* (Hacking, 1995), and many 19th century scholars believed that these patients were switching between a "normal state" and a "somnambulistic state." Hughlings Jackson, a well-known English neurologist from this era, viewed dissociation as the uncoupling of normal consciousness that resulted in what he termed "the dreamy state" (Meares, 1999). A century later, Levitan (1967) hypothesized that "depersonalization is a compromise state between dreaming and waking" (p. 157), and Arlow (1966) observed that the dissociation between the "experiencing self" and the "observing self" serves as the basis of depersonalized states, as it emphasizes its occurrence, especially in dreams. Likewise, Franklin (1990) considered dreamlike thoughts, the amnesia one usually has for dreams, and the lack of orientation of time, place, and person during dreams to be strikingly similar to the amnesia DID patients often report for their traumas (Franklin, 1990). In addition, Barrett (1994, 1995) described the similarity between dream characters and "alter personalities," reported in conjunction with cases of multiple personality disorder, with respect to cognitive and sensory abilities, movement, amnesia, and continuity with normal waking. Barrett contended that the sequelae of adult trauma act as precursors to REM fragmentation, sleep paralysis, and other unusual sleep experiences. The many similarities between dreaming states and dissociative symptoms are also a recurrent theme in the more recent clinical literature (e.g., Bob, 2004).

Anecdotal evidence supports the idea that sleep disruptions are linked to dissociation. For example, in patients with DPD, symptoms are worse when they are tired (Simeon & Abugel, 2006), and one case study highlighted the comorbidity of dissociative symptoms and sleep problems like cataplexy (a sudden and transient episode of loss of muscle tone, often brought on by strong emotions), which is a hallmark feature of

narcolepsy (a chronic sleep disorder, characterized by an excessive urge to fall asleep at inappropriate times; LaVia & Brewerton, 1996). Among participants who report memories of childhood sexual abuse, experiences of sleep paralysis[1] typically are accompanied by raised levels of dissociative symptoms (Abrams, Mulligan, Carleton, & Asmundson, 2008; McNally & Clancy, 2005a). Finally, Gurstelle and Oliveira (2004) speculated about the existence of a newly identified state of consciousness, *daytime parahypnagogia*, which they described as "a transient and fleeting episode, that is dissociative, trance-like, dreamlike, uncanny, and often pleasurable" (p. 166), and which would be most likely to occur when one is tired or suffering from attention fatigue.

Patients with mood disorders, anxiety disorders (including PTSD), schizophrenia, and borderline personality disorder—conditions with relatively high levels of dissociative symptoms—as a rule exhibit sleep abnormalities. Such abnormalities have been extensively studied in the context of these disorders (see for a review, Benca et al., 1992; Brunner, Parzer, Schmitt, & Resch, 2004), and recent research points to fairly specific associations between certain sleep complaints (e.g., insomnia, nightmares) and certain forms of psychopathology (e.g., depression, PTSD; Koffel & Watson, 2009a). For example, Ginzburg and colleagues (2006) found evidence for a dissociative subtype among PTSD patients, which exhibits a specific constellation of symptoms, including high dissociation levels, high hypervigilance, and sleep difficulties (Ginzburg et al., 2006).

Literature Search

We identified studies on sleep and dissociative tendencies through searches of the PsycINFO and Medline electronic databases. We used the entry terms *dissociation, dissociative, derealization, depersonalization, amnesia, absorption*, and *multiple personality disorder*, in combination with other terms including *sleep, hypnopompic hallucinations, hypnagogic hallucinations*[2], *nocturnal experience, dream, nightmare*, and *insomnia*. We limited our search to articles written in English and published after 1980, the year in which the dissociative disorders were first introduced in the *DSM-III* (APA, 1980). Our literature search yielded 2,696 hits. We then examined titles and abstracts to identify empirical studies using adult samples. The search was not restricted to patient populations with formal diagnoses of dissociative or sleep disorders. We also identified additional articles that might be relevant by examining the references from articles selected during the literature search. This procedure yielded 38 studies. However, to be included in our review, studies were required to include at least one (sub) analog or diagnosed sample of patients with a dissociative disorder or a sleep disorder (with a sample size of 10 or more) or rely on a standardized self-report measure of dissociation (e.g., DES) in a clinical or nonclinical sample (with a sample size of 20 or more). Another requirement was that the article reported statistics directly relevant to the relation between dissociation and sleep. This procedure resulted in the 23 studies listed in Table 1.

In the next section, we summarize research that examines dissociative symptoms and sleep and then discuss how sleep is related to memory. In the final section, we address practical implications of the sleep-dissociation link, proffer suggestions for future research, and argue that the sleep–dissociation perspective points to a mechanism that can help reconcile the PTM- and nontrauma-based interpretations of the origins of dissociation.

Systematic Studies on Dissociation and Sleep Phenomena

In the general population, both dissociative symptoms (e.g., depersonalization, Aderibigbe, Bloch, & Walker, 2001) and sleep problems are highly prevalent. For example, 29% of American adults report sleep problems (National Sleep Foundation, 2005). This high prevalence rate allows researchers to study a variety of sleep experiences and relate these to the severity of dissociative symptoms in general population samples. The *Iowa Sleep Experiences Survey* (ISES; Watson, 2001) is a widely used measure that assesses two categories of sleep experiences: The general sleep subscale relates to hypnagogic hallucinations, recurring dreams, nightmares, and waking dreams (i.e., dreams that are confused with reality), and the lucid dreaming subscale of the ISES relates to dreams people report they can control. Research using longitudinally collected daily ratings of sleep-related variables demonstrated that the ISES is a valid measure (Watson, 2003).

In a pioneering study, Watson (2001) relied on two large samples of undergraduate students and showed that dissociative symptoms—as indexed by the DES—are linked to self-reports of vivid dreams, nightmares, recurrent dreams, hypnopompic imagery, and other unusual sleep phenomena measured by the general sleep subscale of the ISES (Watson, 2001). The correlation of the ISES lucid dreaming subscale with the DES was considerably smaller than the association between the ISES general sleep subscale and the DES.

To investigate whether this specific pattern of findings holds when investigating all evidence available in the published literature, we combined the findings from existing studies that relied on the ISES and the DES (see Tables 1 and 2) by means of a random effects model using the metaphor package (Viechtbauer, 2010). The results of these analyses mirror Watson's (2001) initial findings. Specifically, the DES exhibited an average correlation of r =.41, CI: [.35; .48], with the ISES general sleep subscale and a significantly lower correlation of r =.17, CI: [.11; .23], with the lucid dreaming subscale.

Tables 1 and 2 summarize the results of all studies that examined dissociative symptoms and abnormal sleep phenomena. Taken together, the 23 studies summarized in Tables 1 and 2 support several conclusions. First, as can be seen, with the exception of the study by Hartman, Crisp, Sedgwick, and Borrow (2001), Watson's (2001) basic findings have been

Table 1. Summary of Pearson Product-Moment Correlations Reported by Studies on the Sleep-Dissociation Link, Listed in Alphabetical Order

Study	Dissociation and its correlates
Abrams, Mulligan, Carleton, and Asmundson (2008)	
N = 263 adults reporting childhood sexual abuse	
Sleep paralysis	DES = 0.31–0.35
Agargun, Kara, Ozer, Selvi, Kiran, & Kiran (2003)	
N = 292 students	
VDAS	DES = 0.41
Fassler, Knox, and Lynn (2006)	
N = 163 students	
ISES General Sleep	DES = 0.35, TAS = 0.49, MCSD = −.13
ISES Lucid Dreaming	DES = 0.10, TAS = 0.15, MCSD = −.05
Giesbrecht and Merckelbach (2004)	
N = 94 students	
ISES General Sleep	DES = 0.38, DES-T = 0.38
ISES Lucid Dreaming	DES = 0.23, DES-T = 0.24
MEQ	DES = 0.09, DES-T = 0.12
Giesbrecht and Merckelbach (2006a)	
N = 205 students	
ISES General Sleep	DES = 0.35, DES-T = 0.35, CEQ = 0.38
ISES Lucid Dreaming	DES = 0.08, DES-T = 0.10, CEQ = 0.10
Giesbrecht and Merckelbach (2006b)	
N = 87 students	
ISES General Sleep	DES = 0.37
ISES Lucid Dreaming	DES = 0.01
Giesbrecht, Jongen, Smulders, and Merckelbach (2006)	
N = 67 students	
ISES General Sleep	DES = 0.55, DES-T = 0.47
ISES Lucid Dreaming	DES = 0.09, DES-T = 0.05
Giesbrecht, Smeets, Leppink, Jelicic, and Merckelbach (2007)	
N = 25 students	
SSS	CADSS = 0.51, PDEQ = 0.51
POMS Fatigue–Inertia subscale	CADSS = 0.48, PDEQ = 0.43
Koffel (2011)	
N = 200 patients	
ISDI Unusual Sleep Experiences Subscale	DPS–Imagination = 0.39, DPS–Detachment = 0.35, DPS–Obliviousness = 0.38
Koffel and Watson (2009b)	
N = 376 students	
ISES General Sleep	Dissociation composite (DES, DPS) = 0.45
Levin and Fireman (2002a)	
N = 116 students	
Nightmare frequency	DES = 0.30, DES-T = 0.21
Nightmare distress	DES = 0.30, DES-T = 0.36
Ross (2011)	
N = 303 DID patients	Sleepwalking frequency = 54.3%*
N = 303 psychiatric outpatients	Sleepwalking frequency = 0.7%
N = 502 general population	Sleepwalking frequency = 16.6%
	(Comparison DID patients vs. Chinese psychiatric outpatients: Effect size *r* = 0.60. Comparison DID patients vs. Canadian general population: Effect size *r* = 0.53)
Semiz, Basoglu, Ebrinc, and Cetin (2008)	
N = 88 borderline patients	
VDAS	DES = 0.58

(continued)

Table 1. (continued)

Study	Dissociation and its correlates
Soffer-Dudek and Shahar (2009)	
N = 273 students, retest *N* = 214 students	
ISES General Sleep	DES = 0.33, RTS = 0.35
ISES Lucid Dreaming	DES = 0.16, RTS = 0.19
ISES General Sleep retest	DES = 0.44, RTS = 0.25
ISES Lucid Dreaming retest	DES = 0.38, RTS = 0.26
Soffer-Dudek and Shahar (2011)	
Time 1 *N* = 200 students, Time 2 *N* = 155 students	
ISES General Sleep	DES (Times 1–2) = 0.39–0.41, CADSS (Times 1–2) = 0.28–0.29
ISES Lucid Dreaming	DES (Times 1–2) = 0.15–0.29, CADSS (Times 1–2) = 0.14–0.15
Suszek and Kopera (2005)	
N = 71 medical students	
Dream recall frequency	DES = 0.29
Watson (2001)	
Sample 1 *N* = 482 students, Sample 2 *N* = 466 students	
ISES General Sleep	Dissociation composite (DES, DPS, QED) = 0.53
ISES Lucid Dreaming	Dissociation composite (DES, DPS, QED) = 0.24
ISES General Sleep	Dissociation composite (DES, DPS, QED) = 0.54
ISES Lucid Dreaming	Dissociation composite (DES, DPS, QED) = 0.22
Watson (2003)	
N = 169 students	
ISES General Sleep	DES = 0.30, DPS = 0.52
Yu et al. (2010)	
N = 608 participants	
Dream Intensity Profile	DES = 0.35

Note. DES = Dissociative Experiences Scale; DPS = Dissociative Processes Scale; QED = Questionnaire of Experiences of Dissociation; ISES = Iowa Sleep Experiences Survey; DES-T = DES Taxon; MEQ = Morning-Evening Questionnaire; CEQ = Creative Experiences Questionnaire; SIMS = Structured Inventory of Malingered Symptomatology; TAS = Tellegen Absorption Scale; MCSD = Marlowe-Crowne Social Desirability Scale; CADSS = Clinician-Administered Dissociative States Scale; PDEQ = Peritraumatic Dissociative Experiences Questionnaire; SSS = Stanford Sleepiness Scale; RTS = Revised Transliminality Scale; POMS = Profile of Mood States; DID = dissociative identity disorder; VDAS = Van Dream Anxiety Scale, DIS-Q = Dissociation Questionnaire; ISDI = Iowa Sleep Disturbances Inventory.
*$p < .05$

reproduced time and again. Replications have involved both studies that used sleep measures and samples similar to Watson's (2001), as well as studies that used different instruments and samples, yet produced findings that converge on Watson's (2001) conclusion that unusual sleep experiences and dissociative symptoms are linked. Moreover, the connection between sleep and dissociation is evident when researchers use instruments other than the DES to tap dissociative symptoms (e.g., Koffel & Watson, 2009b; Watson, 2003), and when they assess the more pathological manifestations of dissociation (e.g., the DES-T; Giesbrecht & Merckelbach, 2004, 2006).

Second, the connection between sleep and dissociation is specific in the sense that unusual sleep phenomena that are difficult to control, including nightmares and waking dreams, are related to dissociative symptoms, but lucid dreaming—dreams that are controllable—are only weakly related to dissociative symptoms. In a recent study germane to this issue, 374 participants completed a comprehensive test battery, including measures of nightmares, initial insomnia, fatigue, the *Inventory of Depression and Anxiety Symptoms* (Watson, O'Hara, Simms, Kotov, & Chmielewski, 2007), three dissociation measures, three measures of schizotypy (i.e., a tendency to experience hallucinations, magical thinking, disorganized thoughts, and unstable mood), and the ISES. The results prompted the authors to conclude that "unusual sleep experiences are specific to dissociation and schizotypy, whereas insomnia and lassitude are specific to depression and anxiety" (Koffel & Watson, 2009b, p. 551; see also van der Kloet, Giesbrecht, Lynn, Merckelbach, & de Zutter, 2011). In a sample of 71 medical students, Suszek and Kopera (2005) found dream recall frequency to be related to proneness to dissociation. Levin and Fireman (2002b) found greater levels of dissociation and schizotypy in individuals who reported three or more nightmares over a 3-week period than in individuals reporting two nightmares or less. As the researchers noted, this finding provides "further evidence for continuity between waking psychological dysfunction and dream disturbance" (p. 208). More recently, Yu (2010) found positive and significant correlations between the *Dream Intensity Scale*, the *Boundary*

Table 2. Summary of Cross-Sectional Studies on the Sleep–Dissociation Link Comparing Groups, Listed in Alphabetical Order

Study	Measure	Effect size
Agargun, Kara, Ozer, Selvi, Kiran, & Ozer (2003)		$r = 0.23$
$N = 30$ patients with dissociative disorder		
Patients with nightmare disorder ($n = 17$)	DES *M (SD)* = 48.8 (20.5)	
Patients without nightmare disorder ($n = 13$)	DES *M (SD)* = 36.5 (18.9)	
Hartman et al. (2010)		$r = 0.12$
Parasomnias ($N = 16$)	DIS-Q *M (SD)* = 1.6 (0.4)	
Normals ($N = 378$)	DIS-Q *M (SD)* = 1.5 (0.4)	
Levin and Fireman (2002b)		DES: Comparison high vs. medium nightmare, $r = 0.33$; comparison high vs. low nightmare, $r = 0.42$. DES-T: Comparison high vs. medium nightmare, $r = 0.29$; comparison high vs. low nightmare, $r = 0.31$
$N = 116$ students, 21-day dream log		
High nightmare	DES *M (SD)* = 31.16 (17.91); DES-T *M (SD)* = 18.70 (16.89)	
Medium nightmare	DES *M (SD)* = 20.08 (14.48); DES-T *M (SD)* = 9.74 (13.35)	
Low nightmare	DES *M (SD)* = 18.62 (14.07); DES-T *M (SD)* = 8.90 (13.61)	
Ross (2011)		Comparison DID patients vs. Chinese psychiatric outpatients, $r = 0.60$; comparison DID patients vs. Canadian general population, $r = 0.53$
$N = 303$ DID patients	Proportion of sleepwalking: 54.3%*	
$N = 303$ Chinese psychiatric outpatients	Proportion of sleepwalking: 0.7%	
$N = 502$ Canadian general population	Proportion of sleepwalking: 16.6%	

Note. DES = Dissociative Experiences Scale; DIS-Q = Dissociation Questionnaire; DES-T = DES Taxon; DID = dissociative identity disorder.
*$p < .05$

Questionnaire, and the DES in a group of 608 participants and concluded that "the breakdown in boundaries between different conscious states and the ability to cruise along the continuum of consciousness through, for example, voluntarily altering and self-suggesting dreams and conscious activities... are indicative of dissociative and conversion predispositions" (p. 196).

Third, one could argue that the link between unusual sleep experiences and dissociative symptoms rests on a spurious correlation. That is, due to their fantasy proneness, highly dissociative people might endorse atypical answer options on the ISES, rendering self-reports suspect. However, there is no basis for the contention that the connection between dissociative symptoms and unusual sleep experiences is the byproduct of a reporting bias related to demand characteristics or over-reporting: Studies employing instruments that tap over-reporting (e.g., the *Structured Inventory of Malingered Symptomatology*; Smith & Burger, 1997) and demand characteristics (e.g., the *Marlowe-Crowne Social Desirability Scale*; Ballard, Crino, & Rubenfeld, 1988) have revealed no significant correlations between these scales and the ISES (Fassler, Knox, & Lynn, 2006; Giesbrecht & Merckelbach, 2006).

Fourth, the sleep-dissociation link is evident not only in analogue samples, but also in clinical populations. Accordingly, one group of researchers reported nightmare disorder (ND) in 17 out of 30 DID patients (Agargun, Kara, Ozer, Selvi, Kiran, & Ozer, 2003) and also found a 27.5% prevalence of nocturnal dissociative episodes in patients with dissociative disorders (Agargun et al., 2001). Semiz and colleagues (Semiz, Basoglu, Ebrinc, & Cetin, 2008) found that 49% of patients with borderline personality disorder suffered from ND and displayed significantly higher levels of dissociation, as measured with the DES, than patients not suffering from ND. In addition, Ross (2011) found that patients suffering from DID ($n = 303$) reported higher rates of sleepwalking than did a group of psychiatric outpatients ($n = 303$) and a sample from the general population ($n = 502$).

Fifth, Hartman et al.'s (2001) study stands alone in contradicting the covariance of sleep disturbances and dissociation. Although these authors failed to find heightened dissociation levels in their sample of patients diagnosed with sleepwalking or night terror, their relatively small sample ($N = 16$) suggests that this isolated null finding should be interpreted with caution.

In summary, the studies presented in Tables 1 and 2 document a robust correlation between unusual sleep experiences and dissociative symptoms. Based on the more than 5,600 participants in the studies listed in the tables, the correlation falls in the 0.30–0.55 range, indicating that unusual sleep experiences and dissociative symptoms are discriminable, yet related, constructs.

Apart from the tabulated studies, several other researchers have attempted to assess the dissociative status of participants reporting specific unusual sleep experiences. For example, one study showed that people who experience difficulty discriminating between vivid dreams and reality also report heightened dissociation scores (Rassin, Merckelbach, & Spaan, 2001). Moreover, older findings of a positive correlation between individuals' reports of nightmares and their DES scores (Agargun, Kara, Ozer, Selvi, Kiran, & Kiran, 2003; Agargun, Kara, Ozer, Selvi, Kiran, & Ozer, 2003; Levin & Fireman, 2002a) were recently replicated in a study with school-aged children (Agargun et al., in press). Taken together, the extant research provides strong support for a link between dissociative experiences and a labile sleep–wake cycle that is evident in a range of phenomena, including waking dreams, nightmares, and hypnopompic and hypnagogic hallucinations.

Causality and Temporality

The studies summarized in Table 1 relied on a correlational approach, thereby precluding the determination of causal relations among variables. However, sleep disturbance can be induced reliably in healthy participants by depriving them of normal sleep. If dissociative symptoms were fueled by a labile sleep–wake cycle, then sleep loss would be expected to intensify dissociative symptoms. Tentative evidence for such an effect comes from a study by Morgan et al. (2001) that found an increase in dissociative symptoms in healthy soldiers who underwent a U.S. Army survival training that included sleep deprivation. A more stringent test of the hypothesis was conducted in a pilot study (Giesbrecht, Smeets, Leppink, Jelicic, & Merckelbach, 2007) that tracked dissociative symptoms in 25 healthy volunteers during 1 day and 1 night of sleep deprivation. The investigators quantified both spontaneous dissociative symptoms and those induced by means of dot staring during sensory deprivation (see also Leonard, Telch, & Harrington, 1999). The researchers determined that sleepiness, as well as spontaneous and induced dissociative symptoms, were stable during the first day, but substantially increased after 1 night of sleep loss. It is interesting to note that this increase in dissociative symptomatology was highly specific: Dissociative symptoms were affected by sleep loss earlier in time than mood deterioration, whereas no increase in reports of auditory hallucination reports was evident. If demand characteristics and mood deterioration could account for the increase in dissociation, then changes in mood and auditory hallucination reports would have paralleled changes in dissociative symptoms, but this was clearly not the case (Giesbrecht et al., 2007). Therefore, the researchers concluded that the findings were neither carried by demand characteristics nor by mood fluctuations due to sleep loss.

To further examine the temporal link between dissociative experiences and sleep, we (van der Kloet et al., 2011) conducted a longitudinal study to investigate the relation between unusual sleep experiences and dissociation in a mixed inpatient sample at a private clinic ($N = 195$) evaluated on arrival and at discharge 6 to 8 weeks later. We found a robust link between sleep experiences and dissociative symptoms and determined that sleep normalization was accompanied by a reduction in dissociative symptoms. Although sleep normalization was associated with a general reduction in psychopathological symptoms, this reduction could not account for the substantial and specific beneficial effect of sleep improvement on dissociation. It is interesting that, at baseline assessment, 24% of the patients who completed treatment exceeded the cut-off for clinically significant dissociative symptoms (i.e., DES score of 31 or more; Bernstein-Carlson & Putnam, 1993). However, only 12% of the "completers" met this cut-off at follow-up. Similarly, when DES taxon probability scores, indicative of more serious dissociative pathology, were considered, 24.61% of participants met the criterion for taxon membership at baseline versus only 9.74% at the completion of therapy. As per Koffel and Watson (2009a), we also found support for a specific link between unusual sleep experiences (i.e., narcolepsy/hypnagogic imagery, excessive daytime sleepiness) and dissociation and for an association of insomnia symptoms with a composite measure of psychopathology. Levels of self-reported trauma—which we expected would not change over the test–retest period—remained unaffected by sleep normalization, suggesting that demand characteristics are not a plausible explanation for the results obtained.

An exciting interpretation of the link between dissociative symptoms and sleep-related phenomena (see also, Watson, 2001) can be stated as follows. For some yet to be specified reason—perhaps associated with a genetic propensity or, as we will suggest later, intrusions of trauma-related memories—a certain subgroup of individuals experiences a labile sleep–wake cycle that may have two distinct consequences. First, this labile cycle may promote intrusions of sleep phenomena (e.g., dreamlike experiences) into waking consciousness, which in turn foster fantasy-proneness and feelings of depersonalization and derealization. Second, disruptions of the sleep–wake cycle exert a detrimental effect on memory (Hairston & Knight, 2004) and attentional control (Williamson, Feyer, Mattick, Friswell, & Finlay-Brown, 2001), thereby accounting for, or contributing to, the general attention deficits and elevated cognitive failure scores evidenced by high dissociative individuals (Giesbrecht, Merckelbach, Geraerts, & Smeets, 2004; Merckelbach, Muris, & Rassin, 1999; Merckelbach, Muris, Rassin, et al., 2000) and dissociative patients (Dorahy, McCusker, Loewenstein, Colbert, & Mulholland, 2006; Guralnik, Giesbrecht, Knutelska, Sirroff, & Simeon, 2007).

Indirect support for this sleep-dissociation perspective comes from a correlational study on background EEG in participants high and low in dissociation, in which highly dissociative individuals evidenced a reduced α power. Reduced α power is known to predict a dysfunctional inhibitory capacity, leading to an influx of irrelevant information into consciousness. One might therefore speculate that this influx of information might create feelings of "unreality" in relation to the self and the external world, thereby fueling depersonalization and/or derealization experiences (Giesbrecht, Jongen, Smulders, & Merckelbach, 2006). Studies on nonpharmacological manipulations that induce dissociation in the laboratory are also germane. For example, in one such study, researchers (Leonard et al., 1999) found that people sitting for 10 minutes with their eyes closed while wearing goggles and earphones experienced an increase in dissociative symptoms, an effect especially pronounced in individuals who already were highly dissociative. The authors speculate that stimulus deprivation in these latter individuals promotes an internal orientation towards imaginative mentation. Similarly, sleep-related deficiencies in cognitive control may promote an influx of imaginative, dreamlike mentation in daily life that contributes to dissociative symptoms such as depersonalization and derealization.

A strong and simple version of the sleep-dissociation view proposes that a disturbed sleep–wake cycle is a necessary and sufficient antecedent of dissociative symptoms. Although studies conducted in our laboratory provide support for a causal arrow leading from sleep disruption to dissociative symptoms, the associations between sleep and dissociation may be more complex. For example, causal links may be bidirectional, such that dissociative symptoms may engender sleep problems, and psychopathology may partially mediate the link between sleep and dissociative symptoms (van der Kloet et al., 2011). Indeed, sleep problems may stand in a recursive relation with dissociative symptoms, such that sleep disturbances engender such experiences, and dissociation, in turn, increases the vulnerability to sleep disruptions.

Sleep and Memory

The sleep-dissociation link may help us understand why dissociation is related to certain memory aberrations. Giesbrecht et al. (2008) conducted a comprehensive review of the published literature on dissociation and memory function and reported that dissociation is associated with (a) commission errors in memory, (b) self-reported fragmentation of memory, and (c) a failure to forget emotional material. We will next address these three memory phenomena and how they might relate to sleep and dissociation.

Speculation about the connection between sleep and memory can be traced back to the early 19th century when the British psychologist David Hartley (1801) argued that dreaming might change the strength of associative memory links in the brain. Today, we know that the involvement of sleep in memory is far more complex (for a recent review, see Diekelmann & Born, 2010). Part of this complexity stems from the fact that sleep and its disturbances, just like memory and its failures, are not monolithic entities.

It is tempting to think of memory failures solely in terms of forgetting. However, memory failures involve both forgetting (i.e., omissions, the failure to report information) and pseudomemories (i.e., commissions, reporting items that were not learned). *Prima facie*, one would expect dissociative individuals to produce many omission errors in response to memory tasks. Indeed, the dissociative symptom of amnesia can be conceptualized as an extreme manifestation of memory omission. However, there is now abundant evidence that participants scoring high on dissociation differ from control participants primarily in the heightened number of commission errors they make, rather than in the frequency of omission errors. Indeed, one of the most typical features of highly dissociative people's cognitive architecture is that they tend to produce a relative abundance of pseudomemories (i.e., false alarms; commission errors). This prevalent finding is evident in diverse samples, ranging from undergraduate students scoring high on dissociation (Candel, Merckelbach, & Kuijpers, 2003; Giesbrecht et al., 2007; Merckelbach, Zeles, van Bergen, & Giesbrecht, 2007) to patients with PTSD (Bremner, Shobe, & Kihlstrom, 2000).

There are also good reasons, related to the role of sleep in extracting meaning from encoded material, to assume that sleep disturbances foster commission errors. For example, Blagrove and Akehurst (2000) used the *Gudjonsson Suggestibility Scale* (GSS) to study vulnerability to misleading information in sleep-deprived and control participants. The authors reported that sleep-deprived individuals more readily adopt false information (i.e., make more commission errors) than do control participants. Blagrove and Akehurst point to the integrity of the frontal brain areas for differentiating between accurate and pseudomemories and argue that sleep deprivation deregulates the frontal areas, thereby increasing the probability of commission errors (see also Horne, 1993). Evidence from another research line shows that people scoring high on dissociation make more commission errors on a memory task (Candel et al., 2003) and are more suggestible, as measured by the GSS (Merckelbach, Muris, Rassin, et al., 2000), than are control participants. The available evidence thus supports the hypothesis that sleep disturbances foster both increased suggestibility and the tendency to make commission errors associated with pseudomemories. This hypothesis is consistent with the possibility of a nontraumatic etiology of DID symptoms (e.g., the sociocognitive perspective we refer to below) that arise as a function of exposure to highly suggestive techniques in psychotherapy (e.g., hypnosis, leading questions, naming "alter personalities") and media influences (e.g., dramatic portrayals of DID in movies and television; see Spanos, 1996).

Gomez, Bootzin, and Nadel (2006) tested the role of sleep in integrating information in memory. They provided infants with "phrases" from an artificial language; for example, "pel-wadim-jic." An underlying rule was that the first and

last word formed a relationship (e.g., "pel" predicts "jic"). Infants who did not sleep recognized the phrases they had learned earlier, but those who did sleep displayed a generalization of the predictive relationship, implying that sleep supports the ability to detect general patterns in new information (Gomez et al., 2006; see Diekelmann & Born, 2010, for similar phenomena in adult participants). Apparently, the memory-enhancing effect of sleep is not so much that it strengthens recollection of individual items, but that it plays a crucial role in extracting meaning and in facilitating associative links with existing information (abstraction) to create more adaptive semantic networks (Payne & Kensinger, 2010; Spitzer et al., 2007; Tse et al., 2007). Crick and Mitchison (1995) proposed that a process they dubbed "reverse learning" functions during REM sleep to weaken certain memory traces in order to improve memory by "…separating distinct memories from each other which nevertheless have something in common, so that the system is less confused." In sum, "we dream to reduce fantasy" (p. 150). Crick and Mitchison's proposal might explain why people who report sleep disturbances often score highly on fantasy proneness (Giesbrecht & Merckelbach, 2006). It is interesting that patients with narcolepsy indicate that they can misinterpret their dreamlike hallucinatory experiences as real events—for example, sincerely believing that they have been the victim of sexual assault or another offense (Hays, 1992; LaVia & Brewerton, 1996; Szucs, Jansky, Hollo, Migleczi, & Halasz, 2003).

Sleep disturbances are not only associated with commission errors, but also with memory fragmentation in which memories are stored as fragments rather than as reasonably linear, cohesive chronological narratives. Given the crucial role of sleep in memory encoding and consolidation, it seems logical to assume that sleep loss produces such fragmentation. In fact, a night of sleep deprivation prior to training undermines declarative memory encoding; specifically, memory for temporal relations (Harrison & Horne, 2000). Simeon, Hwu, and Knutelska (2007) investigated the relation between dissociative symptoms of DPD patients and temporal disintegration (i.e., deficits in memory information regarding the chronology and dating of events). The researchers found a significant positive correlation between temporal disintegration, as measured by the *Temporal Integration Inventory*, and total DES scores. Furthermore, Simeon and her colleagues (Simeon et al., 2007) concluded that the dissociative dimension of absorption is a significant predictor of temporal disintegration. Note that fantasy proneness is often conceptualized as the "close cousin" of absorption (Allen & Coyne, 1995). We propose that the extant evidence supports the hypothesis that sleep-related temporal disorganization promotes memory fragmentation that, in turn, engenders depersonalization/derealization and amnesia associated with the failure to develop chronologically sequenced memory schema.

Dissociation is linked to an inability to forget emotional stimulus material. For example, Elzinga, De Beurs, Sergeant, Van Dyck, and Phaf (2000) showed that patients suffering from a dissociative disorder find it difficult to forget emotional stimuli. In their study, patients were asked to either forget or remember neutral words, sex words, and threat words. The instruction to forget was expected to reduce conscious memory performance and enhance nonconscious memory performance. However, the researchers found that the instruction to forget increased patients' overall conscious *and* nonconscious memory performance, particularly for sex words (Elzinga et al., 2000).

Sleep deprivation seems to differentially affect memory for emotional stimuli (Walker, 2009). Phelps (2004) reported that sleep deprivation seriously disrupts encoding and later retention of neutral and especially positive emotional memories. However, negative memories were relatively immune to the effects of sleep deprivation (Phelps, 2004). Accordingly, and clearly germane to our theoretical perspective, sleep deprivation may increase the salience of negative memories relative to neutral and positive memories, setting the stage for the infiltration of negative thoughts into consciousness, further sleep disruption, dissociation, and other manifestations of psychopathology.

Whereas negative waking thoughts might interfere with the sleep–wake cycle, dreamlike mentation might arise in waking life. Currently, literature on dreaming and memory is burgeoning, and one theory that deserves serious attention holds that the progression of waking state to REM sleep is marked by an increase in "fluid" and hyperassociative thinking (Stickgold, Hobson, Fosse, & Fosse, 2001). Accordingly, one possibility is that dreamlike intrusions into the waking state that are typical of dissociation interfere with source-monitoring abilities (Lindsay & Johnson, 2000) and produce commission errors. Hartmann (1991) argues that individuals differ in the thickness of boundaries that segregate dream and wake states. Hartmann assumes that people with so-called "thin" boundaries—a hypothetical trait allowing easy passage between reality-based and fantasy-based states of consciousness—would report more extensive dream recall. There is indeed empirical evidence for this hypothesis (Hartmann, 1991; Yu, 2010).

Similarly, Levin and Nielsen (2007) emphasized the concept of "cross-state continuity," which assumes that "…some structures and processes implicated in nightmare production are also engaged during the expression of pathological signs and symptoms during the waking state" (Levin & Nielsen, 2007, p. 483). A related view is the notion of "transliminality" (Thalbourne & Houran, 2000), which assumes that there are robust individual differences in the extent to which mentation may cross thresholds into and out of consciousness. Using a self-report scale designed to measure this trait—the *Revised Transliminality Scale*—Soffer-Dudek and Shahar (2009) recently showed that people who score highly on transliminality (i.e., who are attuned to their inner fantasy life) subsequently report more unusual sleep experiences (related to dissociation, as noted above) than those who score low on this trait (Soffer-Dudek & Shahar, 2009; see also Table 1). The combined findings on cross-state continuity and transliminality buttress the hypothesis that sleep-related phenomena

infiltrate waking consciousness to produce dissociative symptoms such as depersonalization/derealization.

Implications

The sleep-dissociation approach offers a fresh and integrative perspective on dissociative symptoms. There may appear to be little or no link between studies indicating that fantasy immersion and lack of cognitive control overlap with dissociative symptoms (Giesbrecht et al., 2007; Guralnik et al., 2007; Merckelbach et al., 1999, 2002) and studies that assume a traumatogenic etiology of these symptoms (Holmes et al., 2005). However, both strands of research can be integrated in a single conceptual scheme in which disturbed sleep patterns may be determined to be the final common pathway to dissociative symptoms. Indeed, PTSD patients exhibit an increase in nightmare frequency and REM sleep density, but they also complain of insomnia. Moreover, dissociative symptoms go hand in hand with increased frequencies of nightmare reports (Levin & Fireman, 2002a). Recently, Soffer-Dudek and Shahar (2011) reported that daily stress brings about sleep-related abnormalities, including hypnagogic hallucinations and nightmares, among highly dissociative young adults. Accordingly, the sleep-dissociation perspective may explain both how stressful and highly aversive events and environmental and intrapersonal stimuli disrupt the sleep–wake cycle and increase vulnerability to dissociative symptoms, and why dissociation, trauma, fantasy proneness, and cognitive failures overlap.

Thus, the sleep-dissociation perspective is not at all inconsistent with the possibility that aversive and stressful experiences—via their sleep disturbing effects— play a pivotal role in the genesis of dissociation. If future studies, which rely on objective measures of sleep problems and disruptions, further document that traumatic experiences disrupt sleep, they would provide a basis for a rapprochement between the PTM and the sociocognitive interpretation of dissociation. This latter perspective posits that social and cognitive variables, such as media influences and suggestive therapy, shape patients' autobiographical memories, their definition and construal of the self, and their perception of dissociative symptoms (Lilienfeld et al., 1999; Spanos, 1996, but see also Gleaves, 1996). The sensitivity to suggestive influences may arise from the propensity to fantasize, memory errors, increased salience of negative memories, and difficulties in distinguishing fantasy and reality brought about by disruptions in the sleep–wake cycle.

There are three ways in which the sleep-dissociation approach is relevant to more practical issues. First, patients with clinical levels of dissociation often receive psychotherapeutic treatment. Often such treatments are guided by the implicit assumption that a background of childhood trauma is responsible for patients' dissociative symptoms, and trauma history needs to be a focus of therapy. Unfortunately, studies that have investigated the effectiveness of trauma-based and medication treatments for dissociative disorders have produced mixed results (Lilienfeld, 2007; Simeon, Guralnik, Schmeidler, & Knutelska, 2004, but see Brand, Classen, McNary, & Zaveri, 2009; Ellason & Ross, 1997; Ross, 2005). The sleep-dissociation perspective may inspire new treatment possibilities. In particular, assuming that future studies, using objective, laboratory-based measures of sleep, firmly establish that certain sleep deviations serve as important antecedents of dissociative symptoms, it will be imperative to study the effects of treatment interventions focused on sleep normalization in dissociative patients. Previous studies have already examined the effectiveness of sleep medication in PTSD (van Liempt, Vermetten, Geuze, & Westenberg, 2006; see also Hamner, Brodrick, & Labbate, 2001; Raskind et al., 2007) and DID (Loewenstein, Hornstein, & Farber, 1988), showing promising results.

Future studies can also discern what characteristic sleep signatures or disruptions in the sleep–wake cycle are most reliably associated with different dissociative disorders, and then establish remediation programs, including medication regimens, to address underlying sleep deficits and irregularities. This would constitute an entirely novel and exciting approach to the treatment of dissociative symptoms. An interesting example is that of prazosin, an adrenergic antagonist that has been shown to suppress recurrent distressing dreams (Raskind et al., 2003). Accordingly, it would be interesting to explore whether this drug specifically benefits dissociative symptoms due to its ability to normalize REM sleep. In a study discussed earlier (van der Kloet et al., 2011), we showed that a sleep hygiene program goes hand in hand with a sharp reduction of general psychopathology and dissociative psychopathology in particular. After 6–8 weeks, sleep normalization predicted a decrease in dissociative symptoms, partly mediated by a decrease in general psychopathology.

A second implication of the sleep–dissociation approach relates to schizotypy. On the basis of correlational analyses and structural analyses, Koffel and Watson (2009b) proposed that unusual sleep experiences, dissociation, and schizotypy belong to a common domain. Researchers have found evidence for a nontrivial yet poorly understood correlation between dissociation and schizotypy (e.g., Claridge, Clark, & Davis, 1997; Koffel & Watson, 2009b; Merckelbach & Giesbrecht, 2006) and have established that schizotypy predicts nightmare distress (Claridge et al., 1997; Giesbrecht & Merckelbach, 2006). In a recent review (Giesbrecht et al., 2008), researchers explained how these apparently diverse phenomena might become more comprehensible in terms of the sleep-dissociation approach. What fits well with this approach is the finding that psychomimetic drugs like D-lysergic acid diethylamide (LSD) impair reality testing by promoting REM-like experiences in the stream of consciousness (e.g., Fishman, 1983). Other evidence comes from studies on persons with mystical and anomalistic experiences, which often occur in the context of schizotypy. More specifically, people who report such unusual experiences also exhibit disturbed sleeping patterns (e.g., shorter duration of sleep, hypnopompic hallucinations; Britton & Bootzin, 2004; McNally & Clancy, 2005b), although the patterns of disturbed sleep are far from clear.

Third, perpetrators of serious crimes, such as murder, often claim that they suffer from dissociative symptoms (see Moskowitz, 2004). In such cases, the forensic expert faces the difficult task of determining whether these claims are genuine. Such claims are sometimes framed in terms of parasomnias that Bornemann and colleagues defined as "undesirable behavioral or experiential phenomena arising from the sleep period" (Bornemann, Mahowald, & Schenck, 2006, p. 605). According to these authors, parasomnias include disorders of arousal (i.e., sleep walking), rapid eye movement disorder, nocturnal seizures, and dissociative states.

Over the past few years, several case studies have addressed the "sleep walking" defense in criminal courts. These cases have many similarities. Usually the suspect is a man under the age of 35 years who is accused of sexual assault and/or rape and claims to have no recall of the alleged attack (i.e., crime-related amnesia). Most of the time, defendants have or say they have a history of somnambulism (Pressman et al., 2009). Sleep medicine specialists are regularly asked to serve as expert witnesses in such cases (Pressman et al., 2009). Research on the sleep–dissociation link may inform experts' reports to the court, as this body of research describes the conditions conducive to severe dissociative symptoms such as amnesia (Van Oorsouw & Merckelbach, 2010).

Conclusion and Call for Future Research

At present, the attractiveness of the sleep–dissociation approach hinges on its heuristic value and potential to create order in what previously seemed to be a chaotic pattern of findings. We have reviewed preliminary evidence implying that sleep disturbances may be directly related to dissociative symptoms. However, systematic research is needed to determine whether dissociative symptoms induced by means of sleep deprivation, for example, covary with the physiological peculiarities (e.g., reduced α power in background EEG; Giesbrecht et al., 2006) and cognitive dysfunctions (e.g., the tendency to produce commission errors; Candel et al., 2003) typical of individuals scoring high on dissociation.

Literature demonstrating that dissociation is related to various sleep phenomena suggests several avenues for future research. Studies that examine the sleep–dissociation link in clinical samples are urgently needed insofar as most previous studies are based on undergraduate student samples. To date, two independent lines of research have dominated the empirical literature. One line has addressed unusual sleep experiences and dissociative symptoms, whereas the other line has focused on how sleep disturbances affect memory performance. A convergence of these two lines, tracking sleep disturbances, dissociative symptoms, and memory performance in the context of a single study, would potentially generate new insights.

To be sure, much remains to be done to ascertain the relation between sleep and dissociative symptoms. Future research might profitably address questions like the following: Do dissociative symptoms induced by sleep loss trace changes in sleepiness, throughout the day (see Giesbrecht et al., 2007)? Or are the pathogenic effects of sleep difficulties much more differentiated or specific, as Koffel and Watson (2009a) proposed? Indeed, Koffel and Watson (2009a) found that both anxiety and depression were related to hypersomnia, fatigue, sleepiness, and insomnia; however, the first three sleep-related problems were related more strongly to depression than to anxiety. Relatedly, will research with clinical populations continue to support the observation that sleep-related problems such as hypersomnia are more related to depression and anxiety, whereas sleep paralysis, hypnagogic hallucinations, and narcolepsy are more reliably associated with dissociation (see also van der Kloet et al., 2011)?

Holmes et al. (2005) argued that there are two types of dissociation: compartmentalization phenomena (e.g., dissociative amnesia), which reflect lack of cognitive control (e.g., attentional lapses), and detachment phenomena (e.g., depersonalization, derealization, out-of-body experiences), which may be generated by dreamlike intrusions and flashbacks. A large sleep debt reliably produces attentional lapses and microsleeps that might undermine reality judgment (Coren, 1998). Accordingly, a worthwhile issue to address is whether attentional lapses and microsleeps are the precursors of compartmentalization and detachment symptoms, respectively. Testing this hypothesis would require longitudinal studies of people with sleep debt and enable researchers to elaborate and specify variables associated with the sleep–dissociation approach, especially when studies measure (e.g., MRI) biological parameters. An fMRI study (Yoo, Gujar, Hu, Jolesz, & Walker, 2007) demonstrated that a single night of sleep deprivation intensifies the human amygdala reaction to negative picture stimuli, with amygdala potentiation associated with a loss of top-down medial prefrontal connectivity.

Of course, sleep deprivation is a crude way to disturb the sleep architecture. Accordingly, it would be interesting to study the effects of selective deprivation or enhancement of sleep. Research on memory and sleep suggests that the various sleep stages are differentially involved in memory. Specifically, there are indications that slow wave sleep (SWS) sustains the consolidation of declarative memories, whereas REM sleep primarily sustains the consolidation of procedural memories and weakens interfering memory traces (e.g., Born, Rasch, & Gais, 2006; Crick & Mitchison, 1995; Diekelmann & Born, 2010). Does a shortage of SWS account for the cognitive aspects of dissociation? And are excessive amounts of REM sleep and REM rebound responsible for dreamlike intrusions during waking? Recent developments in pharmacology have made it clear that we now have the tools to specifically enhance SWS (e.g., by administering drugs like eplivanserin) or to specifically disinhibit REM sleep (e.g., by a tryptophan-free diet; see Dijk, 2010; Landolt & Wehrle, 2009). Studies relying on such tools to disentangle the contribution of specific sleep stages to dissociative pathology would greatly advance our understanding of the etiology and dynamics of dissociative symptoms.

Are dissociative symptoms induced or merely increased by sleep disturbances? Disruptive sleep may well be a stage setter rather than the singular cause of dissociative pathology. Disruptions in the sleep–wake cycle might constitute a vulnerability or physiological substrate of dissociation that, in turn, interacts with genetic and environmental factors, including highly aversive events. Indeed, genetic variations may be a third variable that accounts for both poor sleep and the propensity to experience dissociation. Tentative evidence suggests that dissociative symptomatology may partly be heritable. For example, both Becker-Blease et al. (2004) and Jang, Paris, Zweig-Frank, and Livesley (1998) found substantial genetic contributions to dissociation scores (but see Waller & Ross, 1997). Moreover, there is a substantial body of evidence that shows how many aspects of sleep are genetically determined (Andretic, Franken, & Tafti, 2008; Cirelli, 2009). For example, heritability in SWS has been estimated to be 50% (Linkowski, 1999), whereas heritability of other EEG sleep parameters has been determined to be even higher (De Gennaro et al., 2008). Thus, it might be the case that heritable individual differences in sleep patterns predispose individuals to dissociative symptoms. Finally, it will be important for researchers to examine the independent influence of sleep problems in fostering dissociative symptoms versus other manifestations of psychopathology, including depression and anxiety. We anticipate that research that addresses these and other issues will reveal how distal causes, such as childhood trauma, translate into proximal antecedents of dissociation (i.e., sleep abnormalities).

In closing, the sleep–dissociation approach can serve as a heuristic framework for studies that address a wide range of fascinating questions about dissociative symptoms and disorders. We now have good reason to be confident that research on sleep and dissociative symptoms will inform psychiatry, clinical science, and psychotherapeutic practice in meaningful ways in the years to come.

Declaration of Conflicting Interests

The authors declared that they had no conflicts of interest with respect to their authorship or the publication of this article.

Notes

1. Sleep paralysis occurs when the normal paralysis during REM sleep manifests when falling asleep or awakening, often accompanied by hallucinations of danger or a malevolent presence in the room.

2. Hypnopompic hallucinations are hallucinations occurring at the time just before awakening, and hypnagogic hallucinations are hallucinations occurring at the time just before falling asleep.

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