Fragmented sleep - fragmented mind : the role of sleep in dissociative symptoms

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The Role of Sleep in Dissociative Symptoms

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CHAPTER I[1]

General introduction

1 This chapter is an adapted version of the following articles and book chapter:
Van der Kloet, D. (Forthcoming). *Dissociative disorders: The relation between dissociation, trauma, and sleep problems*. In R. Biswas-Diener & E. Diener (Eds), Noba textbook series: Psychology. Champaign, IL: DEF Publishers. DOI: www.nobaproject.com

Van der Kloet, D., Merckelbach, H., Giesbrecht, T., & Lynn, S.J. (2012). Fragmented sleep, fragmented mind: The role of sleep in dissociative symptoms. *Perspectives on Psychological Science, 7*, 159-175.

Van der Kloet, D., & Merckelbach, H. (2010). Waar dissociatie vandaan komt – het schemergebied tussen slapen en waken. *GZ-Psychologie, 7*, 12-21.

Summary

In psychopathology, dissociation typically refers to a disturbance in the normal integration of thoughts, feelings, and experiences into consciousness and memory. In this chapter, we review the literature on how sleep disturbances relate to dissociative symptoms and cognitive aberrations seen in dissociative disorders. We contend that this body of research offers a fresh perspective on the origins of dissociation. Specifically, we argue that dissociative symptoms are associated with a labile sleep-wake cycle in which dreamlike mentation invades the waking state, sleep loss produces memory failures, and fuels dissociative experiences. The research domain of sleep and dissociation does not contradict the dominant idea in the clinical literature that trauma is the distal cause of dissociation, and 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.

 Chapter I

Introduction

Dissociative disorders encompass an array of symptoms associated with alterations in consciousness, ranging from profound amnesia for autobiographical events, to equally profound problems in identity and changes in the experience of everyday reality (DSM-IV-TR; American Psychiatric Association (APA), 2000). 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; Spitzer, Vogel, Barnow, Freyberger, & Grabe, 2007; Spiegel, Loewenstein, Lewis-Fernandez, Sar, et al., 2011). We will refer to this perspective as the posttraumatic model (PTM) of dissociation. 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 a review, see Giesbrecht, Lynn, Lilienfeld, & Merckelbach, 2008). In this chapter, 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 this chapter builds on previous contributions (e.g., Giesbrecht et al., 2008; Koffel & Watson, 2009a,b; Watson, 2001) indicating that dissociation and sleep disturbances belong to a common domain, in the current chapter we 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 non-traumatic etiology of dissociation (Lilienfeld, Lynn, Kirsch, Chaves, Sarbin, Ganaway, & Powell, 1999; Spanos, 1996). We conclude with suggestions for extending the sleep-dissociation model 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 non-pathological 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, Fleischer, & Norton, 1991; Sar, Tutkun, Alyanak, Bakim, & Baral, 2000; Tutkun, Sar, Yargic, Ozpulat, Yanik, & Kiziltan, 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 a measure of dissociation (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.

Importantly, 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, Ross, Keyes, Li, Dai, Zhang, 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, Brown, Mansell, Fearon, Hunter, Frasquilho, 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 *Dissociative Experiences Scale* (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 (1) "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 (2) "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: 1) obliviousness/amnesia, 2) depersonalization/ derealization, and 3) imagination/absorption (Carlson, Putnam, Ross, Anderson, Clark, Torem et al., 1991). Most authors also agree that the first two dimensions—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 will refer to this interpretation as the posttraumatic model (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) interpret 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 (Kihlstrom, 2005; Bremner, 2010; Giesbrecht, Lynn, Lilienfeld & Merckelbach, 2010; Dalenberg, Brand, Gleaves, Dorahy, Loewenstein, Cardeña, et al., 2012). Whereas a comprehensive review of the literature on trauma and dissociation is outside the scope of this chapter, 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) compared with 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, 2000b). 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 due to their dreamlike character, dissociative symptoms like derealization, depersonalization, and absorption are associated with sleep-related experiences, 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 chapter, 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 (a) explicitly acknowledges traumatic experiences as potentially distal causes of dissociation with sleep disturbances acting as the more proximal cause of dissociation, and (b) contributes to a growing literature that addresses the relation between sleep and dissociation (Giesbrecht et al., 2008, 2010; Koffel & Watson, 2009 a, b; 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 dissociative identity disorder (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, which would result in what he termed 'the dreamy state' (Meares, 1999). Interestingly, 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, emphasizing 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). Relatedly, 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), a hallmark feature of narcolepsy , a chronic sleep disorder, characterized by an excessive urge to fall asleep at inappropriate times (LaVia & Brewerton, 1996). Interestingly, among participants who report memories of childhood sexual abuse, experiences of sleep paralysis[2] typically are accompanied by raised levels of dissociative symptoms (McNally & Clancy, 2005a; Abrams, Mulligan, Carleton, & Asmundson, 2008). 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, Obermeyer, Thisted, & Gillin, 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 psycho-pathology (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.

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*[3], *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 (American Psychiatric Association, 1980).

Our literature search yielded 2696 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

2 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.
3 Hypnopompic hallucinations: Hallucinations occurring at the time just before awakening; hypnagogic hallucinations: hallucinations occurring at the time just before falling asleep.

references from articles selected during the literature search. This procedure yielded 38 studies. However, to be included in our review, studies were required to (a) include at least one (sub) analogue or diagnosed sample of patients with a dissociative disorder or a sleep disorder (*sample size* ≥ 10), or (b) rely on a standardized self-report measure of dissociation (e.g., DES) in a clinical or nonclinical sample (*sample size* ≥ 20). 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 1.1.

We first 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 non-trauma based interpretations of the origins of dissociation.

Table 1.1.
Summary of Pearson product-moment correlations reported by studies on the sleep-dissociation link, listed in alphabetical order.

Study	Dissociation and its correlates		
Abrams et al. (2008)	**DES**		
N = 263 adults reporting childhood sexual abuse			
Sleep paralysis	.31 - .35		
Agargun et al. (2003a)	**DES**		
N = 292 students			
VDAS	.41		
Fassler et al. (2006)	**DES**	TAS	**MCSD**
N = 163 students			
ISES General Sleep	.35	.49	-.13
ISES Lucid Dreaming	.10	.15	-.05
Giesbrecht & Merckelbach (2004)	DES	DES-T	
N = 94 students			
ISES General Sleep	.38	.38	
ISES Lucid Dreaming	.23	.34	
MEQ	.09	.12	
Giesbrecht & Merckelbach (2006a)	DES	DES-T	CEQ
N = 205 students			
ISES General Sleep	.35	.35	.35
ISES Lucid Dreaming	.08	.10	.10
Giesbrecht & Merckelbach (2006b)	**DES**		
N = 87 students			
ISES General Sleep	.37		
ISES Lucid Dreaming	.01		
Giesbrecht et al. (2006)	DES	DES-T	
N = 67 students			
ISES General Sleep	.55	.47	
ISES Lucid Dreaming	.09	.05	
Giesbrecht et al. (2007)	CADSS	PDEQ	
N = 25 students			
SSS	.51	.51	
POMS Fatigue–Inertia subscale	.48	.43	
Koffel (2011)	**DPS**	Detachment	
N = 200 patients	Imagination	.35	Obliviousness
ISDI unusual sleep experiences subscale	.39		.38
Koffel & Watson (2009)	Dissociation composite (DES, DPS)		
N = 376 students			
ISES General Sleep	.45		
Levin & Fireman (2002b)	DES	DES-T	
N = 116 students			
Nightmare frequency	.30	.21	
Nightmare distress	.30	.36	
Ross (2011)	Sleepwalking frequency		
N = 303 DID patients	54.3%*		
N = 303 psychiatric outpatients	.7 %		
N = 502 general population	16.6 %		

	Comparison DID vs. Chinese psychiatric outpatients: Effect size r = .60 Comparison DID vs. Canadian general population: Effect size r = .53	
Semiz et al. (2008)	**DES**	
N = 88 borderline patients		
VDAS	.58	
Soffer-Dudek & Shahar (2009)	DES	RTS
N = 273 students		
ISES General Sleep	.33	.35
ISES Lucid Dreaming	.16	.19
N = 214 students (re-test)		
ISES General Sleep	.44	.25
ISES Lucid Dreaming	.38	.26
Soffer-Dudek & Shahar (2011)	DES (time 1-2)	CADSS (time 1-2)
N = 200 students (time 1), *N* = 155 (time 2)		
ISES General Sleep	.39-.41	.28-.29
ISES Lucid Dreaming	.15-.29	.14-.15
Suszek & Kopera (2005)	**DES**	
N = 71 medical students		
Dream recall frequency	.29	
Watson (2001)	Dissociation composite (DES, DPS, QED)	
Sample 1 *N* = 482 students		
ISES General Sleep	.53	
ISES Lucid Dreaming	.24	
Sample 2 *N* = 466 students		
ISES General Sleep	.54	
ISES Lucid Dreaming	.22	
Watson (2003)	DES	DPS
N = 169 students		
ISES General Sleep	.30	.52
Yu et al. (2010)	**DES**	
N = 608 participants		
Dream Intensity Profile	.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; VDAS = Van Dream Anxiety Scale, DIS-Q = Dissociation Questionnaire; ISDI = Iowa Sleep Disturbances Inventory.

[a] Corrected for positive response tendencies

* $p < .05$

Systematic Studies on Dissociation and Sleep Phenomena

In the general population, both dissociative symptoms (e.g., depersonalization, Aderibigbe, Boch, & 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: 1) The General Sleep factor relates to hypnagogic hallucinations, recurring dreams, nightmares, and waking dreams (i.e., dreams that are confused with reality), and 2) The Lucid Dreaming factor 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 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 1.1 and 1.2 summarize the results of all studies that examined dissociative symptoms and abnormal sleep phenomena. Taken together, the 23 studies summarized in Tables 1.1 and 1.2 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 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* (IDAS, 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 et al., 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 (2002a) found greater levels of dissociation and schizotypy in individuals who reported three or more nightmares over a three-week period, compared to 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 in a group of 608 participants positive and significant correlations between the *Dream Intensity Scale*, the *Boundary Questionnaire*, and the DES 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*; SIMS; Smith & Burger, 1997) and demand characteristics (e.g., the *Marlowe-Crowne Social Desirability Scale*; MCSD; Ballard, Crino, & Rubenfeld, 1988) have revealed no significant correlations between these scales and the ISES (Giesbrecht & Merckelbach, 2006; Fassler, Knox, & Lynn, 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 et al., 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. Additionally, Ross (2011) found that patients suffering from DID (*N* = 303) reported higher rates of sleepwalking compared to a group of psychiatric outpatients (*N* = 303) and a sample from the general population (*N* = 502).

Fifth, Hartman et al.'s (2010) study stands alone in not being able to reproduce the relationship between 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 1.1 and 1.2 document a robust correlation between unusual sleep experiences and dissociative symptoms. Based on the more than 5600 participants in the studies listed in the tables, the correlation falls in the .30-.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 et al., 2003a, b; Levin & Fireman, 2002b) 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.

Table 1.2.
Summary of cross-sectional studies on the sleep-dissociation link comparing groups, listed in alphabetical order.

Study	Measure		Effect size
Agargun et al. (2003b)			r = .23
N = 30 patients with dissociative disorder of which	DES (M, SD)		
Patients with Nightmare Disorder (*N*=17)	48.8 (20.5)		
Patients without Nightmare Disorder (*N*=13)	36.5 (18.9)		
Hartman et al. (2010)	DIS-Q (M, SD)		*r* = .12
Parasomnias (*N*=16)	1.6 (0.4)		
Normals (*N*=378)	1.5 (0.4)		
Levin & Fireman (2002a)	DES (M, SD)	DES-T (M, SD)	DES :
N = 116 students, 21-day dream log	31.16 (17.91)	18.70 (16.89)	Comparison high vs. medium nightmare: *r* = .33
High nightmare	20.08 (14.48)	9.74 (13.35)	Comparison high vs. low nightmare: *r* = .42
Medium nightmare	18.62 (14.07)	8.90 (13.61)	
Low nightmare			DES-T :
			Comparison high vs. medium nightmare: *r* = .29
			Comparison high vs. low nightmare: *r* = .31
Ross (2011)	Sleepwalking (proportion of participants)		Comparison DID vs. Chinese psychiatric outpatients: *r* = .60
N = 303 DID patients	54.3%*		Comparison DID vs. Canadian general population: *r* = .53
N = 303 Chinese psychiatric outpatients	.7%		
N = 502 Canadian general population	16.6%		

* $p < .05$

The Causality Issue

The studies summarized in Table 1.2 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, Hazlett, Wang, Richardson, Schnurr, and Southwick (2001) that found an increase in dissociative symptoms in healthy soldiers who underwent a U.S. Army survival training, which 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 one day and one

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 one night of sleep loss. Interestingly, this increase in dissociative symptomatology was highly specific: Dissociative symptoms were affected by sleep loss earlier in time than mood deterioration, while 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 causal link between dissociative experiences and sleep, we (van der Kloet, Giesbrecht, Lynn, Merckelbach, & de Zutter, 2012; see chapter X) 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. Interestingly, at baseline assessment, 24% of the patients who completed treatment exceeded the cut-off for clinically significant dissociative symptoms (i.e., DES score >30; Bernstein-Carlson & Putnam, 1999). 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. Interestingly, 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 et al., 2000b) 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). Germane to this issue are also studies on nonpharmacological manipulations to induce dissociation in the laboratory. For example, in one such study, researchers (Leonard et al., 1999) found that when people sat for 10 minutes with their eyes closed wearing goggles and earphones, they experienced an increase in dissociative symptoms, an effect especially pronounced in individuals who already were highly dissociative. The authors speculate that in these latter individuals, stimulus deprivation 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 bi-directional, 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 to 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 nineteenth 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 pseudo-memories (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 now exists 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) compared with 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 et al., 2000b), compared with 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 non-traumatic 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, 1994,1996).

Gomez and colleagues (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, Bootzin, & Nadel, 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 (Spitzer et al., 2007; Tse Langston, Kakeyama, Bethus, Spooner, Wood, et al., 2007; Payne & Kensinger, 2010). Crick and Mitchison (1995) propose that during REM sleep, a process they dubbed 'reverse learning' functions 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). Interestingly, case vignettes of patients with narcolepsy indicate that they can misinterpret their dreamlike hallucinatory experiences as real events and, for example, come to sincerely believe that they have been the victim of sexual assault or another offense (Hays, 1992; LaVia & Brewerton, 1996; Szucs, Janszky, 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 and colleagues (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). Interestingly, the researchers found a significant positive correlation between temporal disintegration, as measured by the Temporal Integration Inventory (TII), and total DES scores. Furthermore, Simeon and her colleagues (Simeon, Hwu, & Knutelska, 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 and colleagues (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, De Beurs, Sergeant, Van Dyck, & Phaf, 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. This author 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 exist 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 (RTS) – Soffer-Dudek and Shahar (2009) recently showed in a longitudinal study 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). Combined, the 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. 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., 2002; 1999) may seem remote from 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 also complain of insomnia. Moreover, dissociative symptoms go hand in hand with increased frequencies of nightmare reports (Levin & Fireman, 2002b). 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: (1) how stressful and highly aversive events and environmental and intrapersonal stimuli disrupt the sleep-wake cycle and increase vulnerability to dissociative symptoms, and (2) 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 clinical practice. 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., 2012; see chapter X), we showed that a sleep hygiene program goes hand in hand with a sharp reduction of general psychopathology and dissociative psychopathology, specifically. 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. Based on 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), we 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 define 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, Mahowald, Schenck, Bornemann, Montplaisir, Zadra, 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, which tracks 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) argue 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) that 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. Interestingly, 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 (see chapter V). Specifically, there are indications that Slow Wave Sleep (SWS) sustains the consolidation of declarative memories, whereas REM sleep primarily sustains the consolidation of procedural and emotional 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 (see chapters II and V). 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, Deater-Deckard, Eley, Freyd, Stevenson, and Plomin (2004) and Jang, Paris, Zweig-Frank, and Livesley (1998) found substantial genetic contributions to dissociation scores (but see Waller & Ross, 1997). Moreover, a substantial body of evidence has accumulated showing that 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, Marzano, Fratello, Moroni, Pellicciari, Ferlazzo, 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.

Outline of the Present Dissertation

With these fascinating questions about dissociative symptoms, sleep disruption, and memory function in mind, the current dissertation has the following aims: 1) to investigate causality issues with regard to dissociation (i.e., is dissociation caused by sleep problems?); 2) to address the mechanisms that play a role in the causal chain between sleep and dissociation; and 3) to explore new treatment avenues for dissociative symptoms.

Besides the introductory chapter and a general discussion chapter, this dissertation is composed of three main parts. Part I focuses on the causal framework of dissociation and in three chapters the link between sleep and dissociation will be firmly established. In chapter II, a field study will be discussed in which participants suffered from a night of sleep loss, leading to increases in sleepiness and dissociative experiences. In chapter III, the relation between objective sleep parameters and dissociation was explored in a group of insomnia patients. In chapter IV, structural equation modeling analyses lead to the formulation of a tentative causal framework of unusual sleep experiences, dissociation, cognitive failures, and fantasy proneness.

Part II of this dissertation offers a more in-depth discussion of the underlying mechanisms. In chapter V, a sleep deprivation experiment will be described, in which 36 hours of sleep loss led participants to experience dissociative symptoms as well as a number of memory- and executive functioning deficits. In chapter VI, a study exploring the underlying neurocircuitry of dissociative symptoms will be described. We investigated the psychopharmacological effects of a variety of drugs

with known mechanisms of actions: 3,4-methylenedioxy-N-methylamphetamine (MDMA, better known as 'ecstasy' or 'XTC'), benzoylmethylecgonine (cocaine), and tetrahydrocannabinol (THC, better known as cannabis or marijuana), and a placebo on dissociative symptom levels. The idea behind this study was that drugs with stimulating (i.e., anti-sleep) properties are expected to reduce dissociative symptomatology, if the sleep-dissociation framework is correct.

Most studies on sleep and dissociation have been conducted in general population- or undergraduate samples. Their relation has not yet been extensively studied in clinical populations. To remedy this shortcoming, in chapter VII, a study exploring the relation between dissociation and sleep aberrations in a group of patients suffering from dissociative identity disorder, as well patients suffering from posttraumatic stress disorder, will be portrayed.

In part III of this dissertation, in light of previous findings we aimed at reversing the metaphorical arrow from sleep disruption to dissociative symptoms. If poor sleep increases dissociative symptoms, it is worthwhile to study whether *normalizing* sleep would *reduce* dissociative symptoms. Therefore in chapter VIII, a pilot study investigating the effects of a sleep hygiene training will be discussed. Mindfulness-Based Therapy (MBT) has its known beneficial effects on sleep improvement. In chapter IX, the effects of eight weeks of MBT training on sleep improvement and reduction of dissociative symptoms will be described. Furthermore, chapter X describes a longitudinal study in 266 inpatients at a private clinic. Their treatment was based on strict sleep hygiene guidelines, and the effects of these on sleep improvement and dissociative symptom reduction are discussed.

Finally, in the general discussion chapter of this dissertation, our findings from parts I-III are integrated and their contribution to the field of dissociation is highlighted. Theoretical and practical implications are discussed. Attention is given to recent developments and future avenues for research are explored.

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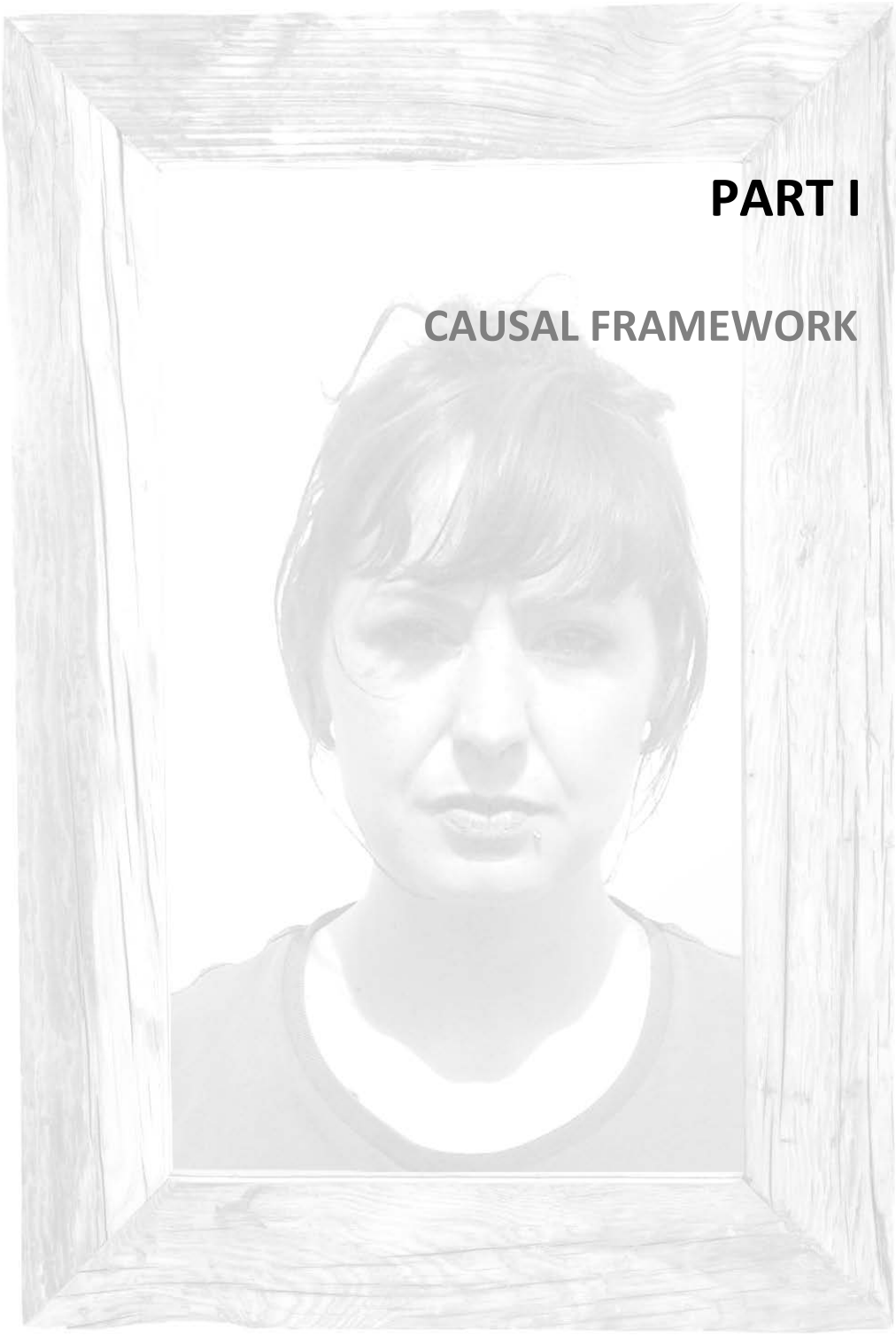


PART I

CAUSAL FRAMEWORK

CHAPTER II[4]

Sleepiness predicts the increase in dissociative symptoms: A field study

4 This chapter is a translated version of the following article:
Van der Kloet, D., Giesbrecht, T., & Merckelbach, H. (2011). Slaperigheid voorspelt dissociatieve symptomen: een veldstudie. *Tijdschrift voor Psychiatrie, 53*, 757-763.

Summary

In laboratory studies, sleepiness has been found to increase dissociative symptoms. We aimed to explore the link between sleepiness and dissociation in a natural setting. We conducted a longitudinal study among volunteers at a pop festival via self-report scales on four successive test moments distributed over a 12 hour period (7 p.m., 11 p.m., 3 a.m., and 7 a.m.). Sleepiness was the only variable that could predict the increase in dissociative symptoms. This effect was not mediated by mood. Our data suggest that also outside a lab environment, sleepiness intensifies dissociative symptoms. Studies focusing on sleep normalization may provide new insights for the treatment of dissociative symptoms.

Introduction

Dissociative disorders rank among the most controversial disorders listed in the *Diagnostic and Statistic Manual of Mental Disorders* (DSM-IV-TR; APA, 2000). They entail a large variety of experiences and complaints, ranging from absorption and memory problems (amnesia) to doubts about the own identity. Such symptoms may present in acute form, i.e., as reaction to an unsettling experience (*acute* or peritraumatic dissociation). However, they may also manifest chronically in which case the literature defines this as *trait* dissociation (Holmes et al., 2005). For decades, clinicians and scientists have discussed the origins of dissociative symptoms (see for a review Giesbrecht et al., 2008).

Many dissociative symptoms possess a dreamlike character (e.g., depersonalization experiences and being engrossed in fantasy). Researchers have therefore speculated about a connection between dissociative symptoms and sleep (see for a review Giesbrecht & Merckelbach, 2006). This line of reasoning is supported by the work of Watson (2001; 2003). In two large studies, he found that dissociative symptoms - as measured by the commonly used scales in research and clinical practice – were related to unusual sleep experiences measured on the *Iowa Sleep Experiences Survey* (ISES; Watson, 2001; see also van der Kloet & Merckelbach, 2010). Unusual sleep experiences refer to experiences such as nightmares, vivid dreams, recurring dreams, lucid dreams, narcoleptic symptoms, hypnagogic and hypnopompic hallucinations, and sleep paralysis. This correlation has since been replicated by various researchers. Based on his findings, Watson proposed that dissociation, schizotypy, and unusual sleep experiences all belong to a common domain. Watson (2001) uses the term *cross-state continuity*, to describe the idea that if one has unusual experiences during the night also tends to experiences these during the day. Since then, several research groups have replicated Watsons findings (Fassler et al., 2006; Giesbrecht & Merckelbach, 2006; Soffer-Dudek & Shahar 2009).

Interestingly, rather specific relations exist between the diverse forms of psychopathology and certain sleep complaints. Depression is related to sleep loss, while posttraumatic complaints are more strongly related to nightmares. Factoranalytic research has clarified that there is a stronger overlap between dissociation and schizotypy and unsusual sleep experiences, than between depressed mood and anxiety and unusual sleep experiences (Koffel & Watsons, 2009). Koffel en Watson (2009) have therefore concluded that “unusual sleep experiences (…) are related to symptoms of dissociation in clinical as well as nonclinical groups” (p.557).

Research of the recent years has thus shown a consistent and robust relation between dissociative symptoms and deviant sleep experiences. However, these findings rest on studies with a majority of cross-sectional designs. Such designs do

not allow for conclusions about cause and effect. To obtain more insight in the causal background, we therefore deprived 25 healthy volunteers from one night of sleep to test this idea that disturbed sleep would lead to an increase of unusual sleep experiences. The volunteers stayed at the laboratory for two days from nine a.m. on day 1, to four p.m. on day 2. During the night, they were kept awake, and every six hours they completed questionnaires to measure dissociation, sleepiness, and mood.

Dissociative symptoms followed a specific temporal pattern. This is to say: dissociation remained stable during the day, but increased during the evening and night. The increase manifested most strongly between 9 p.m. and 3 a.m. Sleepiness also increased during the night, but partly restored itself again during the day. In this study, dissociative symptoms tended to follow the same circadian oscillating patterns as sleepiness. Important here is that the relation was specific, in the sense that only sleepiness and not a deterioration of mood could predict dissociation (Giesbrecht et al., 2007).

These findings are important for their practical implications, because large groups of people suffer from sleep loss and sleepiness. This makes them vulnerable to get involved in accidents. This risk has been described earlier by Jewett and colleagues (1999), although they have related it to a deterioration of concentration and attention due to a shortage of sleep. Our findings suggest that in sleep deprived individuals, acute dissociative symptoms may interfere with cognitive control, making them vulnerable for accidents.

Research in the laboratory has the advantage that many irrelevant factors are under control. Students are easily recruited for this type of research. However, sometimes it is difficult to generalize results to the 'real' world. We therefore wanted to replicate findings obtained under controlled laboratory conditions under real world conditions.

We investigated the relation between sleepiness and dissociative symptoms using a longitudinal design. We studied two groups of volunteers at a pop festival and compared the group that carried out the night shift to another group working the day shift. We hypothesized that sleepiness – as a global index of sleep disruption – would predict an increase in dissociative symptoms especially in the night shift group. Moreover, to tap into the mechanism underlying such a connection, we wanted to know to what degree this relation would be mediated by a deterioration of mood due to sleep loss. Finally, we investigated whether persons scoring high on *trait* dissociation, absentmindedness, and fantasy proneness, were the ones that would eventually depict the strongest increase in dissociative symptoms. Absentmindedness and fantasy proneness are viewed as some of the more benign aspects of dissociation. If the effects of sleep disruption would be limited to people who score high on these traits, then this would tell us something

about the range of the theory that states that sleep disruption contributes to dissociative symptoms.

Participants and procedure

Participants were 36 healthy volunteers (22 women; ± 26 years old, *SD* = 7.94), who were aiding the organization of a large pop festival in The Netherlands. Everybody worked as surveillants for a working shift of 12 hours. The workshift was during the day or during the night. Twentytwo participants worked during the day, 14 participants worked during the night. During work it was prohibited to use alcohol or drugs.

Participants of the study received written and oral information about the study after which they signed an informed consent. They completed questionnaires at 4 times during their 12-hour workshift (at 7 p.m., 11 p.m., 3 a.m., and 7 a.m.). Moreover, participants completed questionnaires at the start of their shift (baseline measures). Participants received a small monetary award upon completion of the questionnaires.

Measures

Dissociative Experiences Scale (DES; Cronbach's α = .90; Bernstein & Putnam, 1986). The DES measures 28 dissociative symptoms using 100mm visual analogue scales (0 = never; 100 = always). It taps into symptoms such as depersonalization experiences and amnestic phenomena. In this study, we computed the total DES score (*range*: 0-100).

Cognitive Failures Questionnaire (*Cognitive Failures Questionnaire*; CFQ; Cronbach's α = .90; Broadbent, Cooper, Fitzgerald, & Parkes, 1982). The 25 items of the CFQ measure absentmindedness (*range*: 0 = never ; 4 = very often).
Creative Experiences Questionnaire (CEQ; Cronbach's α = .73; Merckelbach, Horselenberg, & Muris, 2001). The CEQ measures the tendency to engage in vivid daydreaming and fantasizing (fantasy proneness). The list comprises 25 yes/no items that tap into preoccupation with imaginations and its consequences. Previous research (Merckelbach et al., 1999) has shown that de CFQ and CEQ correlate strongly with the DES. Fantasy proneness tends to be especially related to DES items that tap into absorption while absentmindedness seems most strongly related to DES items that tap into disruptions in cognitive efficiency.

Stanford Sleepiness Scale (SSS; Hoddes, Zarcone, Smythe, Philips, & Dement, 1973). The SSS explores sleepiness with one single item, which is measured with a 7-points scale (1 = I feel active, vital, alert, or very awake; 7 = I no longer fight sleep, I am about to fall to sleep, my thoughts seem like dreams).

Clinician-Administered Dissociative States Scale (CADSS; Cronbach's α = .65 - .82; Bremner et al., 1998). The CADSS measure acute dissociation with 19 items (0 = not present; 4 = extremely present). Example items are "Do objects appear unreal, like you're in a dream?" and "Do you have some experience that separates you from what is happening; for instance, do you feel as if you are in a movie or a play?" Participants were asked to keep in mind the last 3 hours as a reference point when completing the items.

Affect Grid (AG; Russell, Weiss, & Mendelsohn, 1989). The AG measures mood on the dimensions "pleasure" and "energy". The AG is designed as a matrix of 9 x 9 boxes, in which the participants can tick a box.

Data analyses

Statistical analyses were performed using SPSS 18.0 software. Cronbach's α's were used to estimate the internal consistencies of the DES, CFQ, CEQ, and CADSS. Furthermore, we compared CADSS- and SSS scores of the day and night groups at time point 4. Finally, we entered lineair and multiple hierarchical regression analyses to determine which factors – sleepiness, mood, etc. – could predict acute dissociation.

Results and discussion

Against our expecation, the night group did not report more sleepiness (SSS) than the day group: $t(32) = .84$, $p > .05$. Moreover, there were no differences between the groups with regard to acute dissociative symptoms (CADSS): $t(34) = .84$, $p > .05$. The absence of a difference in sleepiness between the day- and night group can be accounted for by the high scores of the day group on this dimension. In retrospect, it is understandable why this group had such high scores: after all, this group had to get up early and had been working for 12 hours at time point 4. Also, it is conceivable that the festival weekend had disrupted the sleep-wake cycle of the day group beforehand. That is why we decided to combine the two groups.

Table 2.1 displays the mean scores on the DES, CFQ, and CEQ and the correlations between these measures. In keeping with the literature, dissociative symptomes (DES), fantasy proneness (CEQ), and absentmindedness (CFQ) were significantly and positively related with each other. Then, we explored the *state* measures. Figure 2.1 illustrates the course of measures over time. The strongest increase in dissociation occurred between the last two time intervals, that coincides with the point at which the sleep-wake cycle usually depicts its energetic low (Coren, 1996). Analyis of Variance with repeated measures showed that

volunteers increasingly became sleepier, experienced less pleasure and energy, and reported more dissociative symptoms (all $F(1,34)$'s > 8.1, all p's < .01).

Table 2.1
Mean scores on *trait* questionnaires and Pearson product-moment correlations during baseline (N = 35).

	Mean (SD)	*DES*	*CFQ*
DES	16.54 (10.72)	-	
CFQ	43.53 (13.36)	.61**	-
CEQ	6.69 (3.92)	.55**	.42*

Note. CEQ = Creative Experiences Questionnaire; CFQ = Cognitive Failures Questionnaire; DES = Dissociative Experiences Scale.
* p < .05
** p < .01

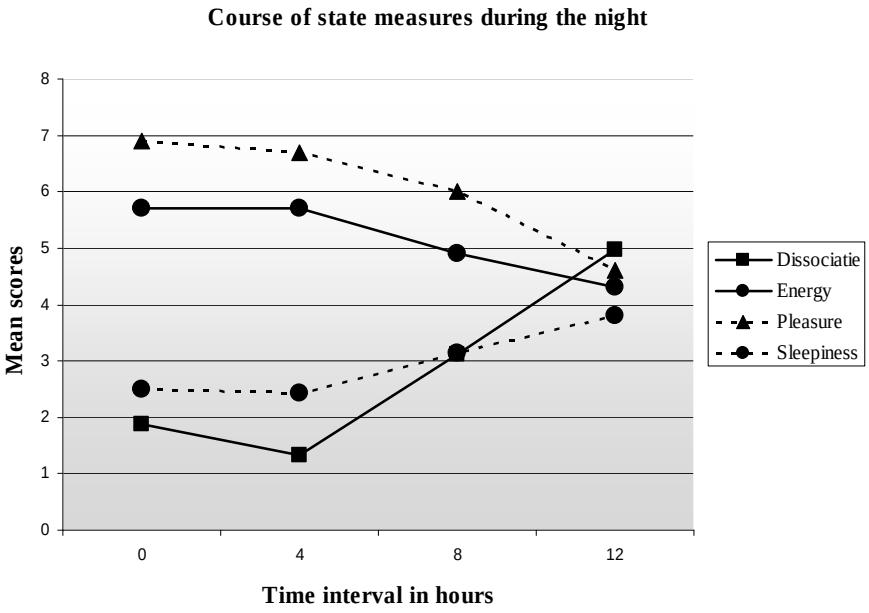


Figure 2.1.
Course of state measures during the night
Note. Data of Affect Grid (energy, pleasure) are transformed.

We wanted to know whether sleepiness, mood, and dissociation were related to each other in a specific way. We were particularly curious whether specifically increased sleepiness would *predict* an increase in dissociation. To this effect, we computed a hierarchical regression with the change scores on the CADSS as dependent variable and the DES, CEQ, CFQ, change scores on the SSS and AG, and the interaction between DES and SSS change score as predictors. A summary of the hierarchical decomposition is depicted in table 2.2. Only increased sleepiness could predict the increase of dissociative symptoms, (t = 7.16, p < .01).

Table 2.2.
Summary of hierarchical regression analyses of the increase of *acute* dissociation (N = 33).

Model	*ß*	*t*	*p*	*r*	r^2
1				.81	.63
SSS change	.72	6.18	< .01		
DES total	.18	1.56	.13		
2				.79	.61
SSS change	.79	7.16	**< .001**		

Note. SSS change = change score Stanford Sleepiness Scale; DES total = mean total score on the Dissociative Experiences Scale.

Conclusion

Our results show that also outside the laboratory walls, sleepiness – as index of sleep disruption – predicts an increase in dissociation. This is in line with previous studies in which a robust connection was found between sleep abnormalities and dissociation (Giesbrecht et al., 2007; Watson, 2001). With an explained variance running into the 60% (see table 2.2), the connection between dissociation and sleepiness in this study is stronger compared to what was found earlier. We believe that this might be due to the fact that in this study dissociation and sleepiness were measured as state and not as trait.

Our analyses showed that specifically the increase of sleepiness predicted acute dissociative symptoms. This effect was not mediated by mood deterioration. It is striking that it does not seem to matter whether sleepiness presents itself during the day or during the night. This is best understood in light of the hectics of the previous days that has propable already disorganized the sleep-wake cycle in both groups. What also points in this direction, is that the day group reported a substantial low point in energy and pleasure. Such findings dovetail with the trio of sleep disruption, poor cognitive efficiency, and risk at accidents (Jewett et al., 1999). Our study suggests that dissociative phenomena fit into that picture as well, and make them relevant for all those groups (i.e., employees in shift work) known to deal with sleep disruption. This is even underlined by at least one EEG-study

showing that people with high dissociation levels display a lower activity in the alpha band of the resting EEG, while we know that such a condition of alpha suppression goes along with a lower cognitive efficiency (Giesbrecht et al., 2006). Even more important is that the results described above, fit rather well with findings from our earlier laboratory study (Giesbrecht et al., 2007). Taken together, these findings support the hypothesis of Watson, namely that sleep-wake disruptions lead to intrusions of dreamlike mentation into the waking consciousness, fostering dissociative symptoms.

Some limitations of our field study merit mention. Our sample of participants was relatively small, a control group (of not-working volunteers) was absent, we relied on self-reports and our measure of sleep disruption was indirect. On the other hand, standardized instruments with a good reputation were used for this study. Another strong point of this study entails the natural environment in which it took place, but in which there was controlled for alcohol- and drug use.

In sum, we can conclude that also in a natural environment, sleepiness – as index for sleep disruption – predicts an increase of dissociative symptoms. Our findings point in the direction of a new approach to the treatment of dissociation symptoms: sleep normalization. With this line of research showing that sleep disruptions is related to an *increase* of dissociative symptoms, it would be interesting to determine whether sleep normalization can contribute to a *decrease* of dissociative symptoms. A first step in this direction has recently been taken with a study in which we found that in a heterogeneous patient group, sleep normalization went hand in hand with a drastic decrease of dissociative symptoms after 6 weeks (Van der Kloet & Merckelbach, 2010). It should be noted that this entailed an intramural institution, hence an environment in which sleep hygiene is easily influenced.

CHAPTER III[5]

Dissociative symptoms and sleep parameters – An all-night polysomnography study in patients with insomnia

[5] This chapter is an adapted version of the following article:
Van der Kloet, D., Giesbrecht, T., Franck, E., Van Gastel, A., De Volder, I., Van den Eede, F., Verschuere, B., & Merckelbach, H. (2013). Dissociative symptoms and sleep parameters - An all-night polysomnography study in patients with insomnia. *Comprehensive Psychiatry, 54,* 658-664.

Summary

Dissociative disorders encompass a range of symptoms varying from severe absent-mindedness and memory problems to confusion about one's own identity. Recent studies suggest that these symptoms may be the by-products of a labile sleep-wake cycle. In the current study, we explored this issue in patients suffering from insomnia (N = 46). We investigated whether these patients have raised levels of dissociative symptoms and whether this is related to objective sleep parameters. Patients stayed for at least one night in a specialized sleep clinic, while sleep EEG data were obtained. In addition, they completed self-report measures on dissociative symptoms, psychological problems, and sleep characteristics. Dissociative symptom levels were elevated in patients suffering from insomnia, and were correlated with unusual sleep experiences and poor sleep quality. Longer REM sleep periods and less time spent awake during the night was predictive of dissociation.This is the first study to show that insomnia patients have raised dissociative symptom levels and that their dissociative symptoms are related to objective EEG parameters. These findings are important because they may inspire sleep-related treatment methods for dissociative disorders.

Introduction

Dissociative symptoms form a heterogeneous class of experiences varying from absent-mindedness, excessive daydreaming, and memory problems to confusion about one's own identity. In their most radical version, such symptoms define conditions like dissociative amnesia and depersonalization disorder. Given their stark heterogeneity, it is not surprising that dissociative disorders are among the most controversial nosological categories listed in the *Diagnostic and Statistic Manual of Mental Disorders* (DSM-IV-TR; APA, 2000). To date, there is no agreed-upon conceptualization of the taxonomy and aetiology of dissociative symptoms (Giesbrecht et al., 2008).

Epidemiological studies among psychiatric inpatients and outpatients have yielded prevalence estimates of severe dissociative symptoms, with rates usually exceeding 10% (Foote et al., 2006), while a recent epidemiological study in the UK general population found a prevalence rate of .95% (Lee et al., 2012). Dissociative symptoms are not restricted to the dissociative disorders. Certain diagnostic groups, notably patients with borderline personality disorder, post-traumatic stress disorder (PTSD), obsessive compulsive disorder, and schizophrenia also display heightened levels of dissociative symptoms (Rufer et al., 2006; Merckelbach et al., 2005; Yu et al., 2010). Prevalence rates of dissociative symptoms may also be raised in certain populations like, for example, homeless and runaway youths (Tyler, Cauce, & Whitbeck, 2004).

A recurrent theme in the clinical literature on dissociative symptoms is that they are caused by aversive experiences. More specifically, the idea is that dissociative symptoms like amnesia and derealisation help individuals to detach themselves from aversive life experiences (Spiegel, 2011). Although there is correlational evidence to back up the link between dissociative symptoms and aversive life experiences (Hagenaars, Fisch, & van Minnen, 2010), the question remains how such experiences might set into motion dissociative symptomatology. An older idea that recently gained momentum focuses on sleep disturbances and how these disturbances might contribute to dissociative symptomatology. Thus, Levitan (1967) hypothesized that "depersonalization is a compromise state between dreaming and waking" (p. 157). Recent studies have found a robust link between dissociation and self-reported sleep disturbances (Watson, 2001; Giesbrecht et al., 2007; van der Kloet et al., 2012; see chapter I). This finding has inspired some authors to speculate that a labile sleep-wake cycle may undermine cognitive efficiency and cause dreamlike mentation to emerge during waking consciousness, thereby fuelling dissociative symptoms (Koffel & Watson, 2009a). There is some indirect evidence to support this hypothesis. For example, McNally and Clancy (2005a) relied on a sample of individuals who reported a history of child sexual abuse. In their sample, dissociative symptoms were more common in

participants who had experienced sleep paralysis compared to those without such experiences. Dissociative symptoms also go along with increased frequencies of nightmare reports (Levin & Fireman, 2002b) and PTSD patients not only have raised levels of dissociative symptoms, but they also exhibit increased nightmare frequency and REM sleep density, and often suffer from insomnia (Raskind et al., 2007).

Particularly interesting but under-researched is the potential link between REM sleep and dissociative symptoms. In her recent review, Llewellyn (2012) stressed that REM sleep is usually accompanied by associative and visual hyperactivity so as to encode episodic memories. This author summarizes evidence to show that during REM sleep, the pre-frontal areas are in a state of de-activation resulting in fluid reasoning and bizarre thoughts. Thus, an excess of REM sleep and/or REM sleep activity throughout the day could lend dissociative qualities to cognitive functioning.

If sleep disturbances are a critical factor in the development of dissociative psychopathology, one would expect that patients with insomnia have heightened levels of dissociative symptoms. After all, the majority of patients who suffer from (primary) insomnia have a disturbed sleep-wake cycle (Harvey, Tang, & Browning, 2005). In this study, we examined whether patients with insomnia have, indeed, raised scores on a validated questionnaire measuring dissociative symptoms. Additionally, and more exploratory, we investigated to what extent these symptoms are related to unusual sleep experiences, sleep quality, and EEG parameters. Previous studies relied on self-reported sleep disturbances. We were interested in whether the sleep–dissociation link is also evident when one looks at objective EEG indices, particularly REM sleep.

Methods and Materials

Patients

The sample consisted of 45 consecutive inpatients (18 men, 28 women), who had been referred to Antwerp University Hospital, Belgium in the period between January 2010 and April 2010. Only patients with a diagnosis of primary insomnia were included. They had a variety of sleep complaints, such as having trouble with falling asleep, night-time awakenings, waking up early, and/or non-refreshing sleep. Mean age of the patients was 41.5 years (*SD* = 13.68; *range*: 17 to 78 years). Most of them were married or lived together with their partner (71.2%). A minority was either divorced (6.5%) or single (19.6%). Educational background ranged from limited (lower secondary education; 10.8%) to extended (higher education / university degree / postgraduate; 39.1%). Thirty-four patients (73.9%) were using

medication during the study. Medication included benzodiazepines (21.7%), melatonine (2.2%), antidepressants (32.6%), pain medication (13%), and neuroleptics (4.3%). Twenty-eight percent of the patients suffered from a (self-reported) psychiatric disorder (e.g., mood-, or anxiety disorder) at the time of the study, and were unable to work due to their complaints for a mean of 22 days (*sd* = 35.9) over the last three months. On average, the current sample had not been able to execute their normal activities for 18.5 days (*SD* = 34.0) during the last three months. Eight patients (17.4%) had been hospitalized (mean duration: 1.98 days; *SD* = 9.01) in the last three months.

All patients had been referred for assessment of their persisting insomnia complaints. As part of the routine procedure, participants completed a number of self-report questionnaires (see Measures) during their stay of one or two nights in the specialized sleep clinic of Antwerp University Hospital. During their stay, data on sleep parameters were collected. For participants who underwent two night sessions (*N* = 13, 28%), data of the second night were employed in analyses. Patients provided written informed consent for the use of their data for the purpose of the present study. The study was approved by the standing ethical committee of the Antwerp University Hospital (B30020107809).

Measures

Dissociative Experiences Scale (DES; Bernstein & Putnam, 1986). The DES is a self-report scale that requires participants to indicate on 100 mm visual analogue scales (anchors: 0 = never; 100 = always) to what extent they experience 28 dissociative experiences in daily life. Examples of such experiences include feelings of depersonalization, derealization, and psychogenic amnesia. Van IJzendoorn and Schuengel (1996) provide meta-analytic evidence for the sound psychometric properties of the DES. In the current study, we calculated a mean total score of the 28 DES items.

Symptom Check List –90 – Revised (SCL-90-R; Derogatis, 1994). The SCL-90-R is a widely used self-report inventory measuring a broad range of psychopathological symptoms (e.g., depression, anxiety, somatic symptoms) in clinical as well as general population samples. In the current study, we used the validated Dutch version of the SCL-90-R (Arrindell, & Ettema, 1981) and calculated the Global Severity Index (GSI), which is the mean total score of the 90 SCL-90-R items.

Iowa Sleep Experiences Scale (ISES; Watson, 2001). The ISES consists of 18 items asking the respondent to rate the frequency of various sleep-related and dream-related experiences on a 7-point Likert scale (anchors: 1 = never; 7 = several times a week). The ISES consists of two separate subscales: general sleep experiences (Cronbach's α = .90, e.g., "I have recurring dreams") and lucid dreaming (Cronbach's α = .83, e.g., "I am aware that I am dreaming, even as I

dream"). The general sleep experiences subscale taps symptoms of narcolepsy, vivid and unusual dreams, and other distinct sleep experiences. The lucid dream subscale consists of several items that refer to the experience that one is aware of dreaming while still being asleep. Previous research by Watson (2001) shows that these two subscales measure distinct constructs. Watson (2001; 2003) obtained evidence for the convergent validity and internal consistency of the ISES.

Pittsburgh Sleep Quality Index (PSQI; Buysse, Reynolds III, Monk, Berman, & Kupfer, 1988). The PSQI is a self-report instrument measuring sleep quality and sleep aberrations during the last month. Consisting of 19 items, it measures seven aspects: subjective sleep quality, sleep latency, sleep efficiency, sleep time, sleep aberrations (e.g., nightmares, snoring, leg movements), medication use, and daytime problems (e.g., problems with staying awake during driving, eating, or social situations). The sum score of the 7 components yields a total score. Buysse and colleagues (1988) examined the psychometric properties of the PSQI using an 18 months longitudinal design to monitor "good" and "bad" sleepers. They found an acceptable test-retest reliability and validity. The PSQI has reasonable sensitivity (89%) and specificity (85.5%) in differentiating between good and bad sleepers, making it a useful tool for clinical use as well as research (Buysse et al., 1988).

Brussels Indices of Sleep Quality (BISQ; van Even, 2004). The BISQ is a 29 items self-report instrument to index subjective feelings of sleep quality, and sleep parameters such as sleep efficiency, night-time awakenings, and sleepiness during the day. The instrument yields total scores for three subscales; 1) subjective sleep quality, 2) whether sleep quality is better or worse than usual, and 3) the subjective tolerance of sleep problems. Psychometric qualities of the measure are unknown as it has not been validated yet.

Traumatic Experiences Checklist (TEC; Nijenhuis, Van der Hart, & Kruger, 2002). The TEC is a reliable and valid self-report instrument to measure physical abuse, emotional abuse and neglect, sexual harassment, and sexual abuse, as well as other traumatic experiences. The TEC has especially been employed with psychiatric outpatients (Nijenhuis et al., 2002), but has also been administered to nonclinical samples in which low and skewed TEC scores are not unusual (Näring, & Nijenhuis, 2005). The correlation between the TEC and the Stressful Life Events Screening Questionnaire (SLESQ; Goodman, Corcoran, Turner, Yuan, & Green, 1998) is relatively high, $r = .77$, $p < .01$. In this study, we used the 26-items version of the TEC, which yields a total score between 0 and 26, with higher scores indicating higher levels of self-reported trauma.

Polysomnographic measures

Polysomnography during the night was performed, using electroencephalogram (EEG), electrooculography (EOG), and electromyogram (EMG) measurements

(BrainRT, OSG, Rumst, Belgium). Electrodes for EEG registration were applied according to standard criteria of the American Academy of Sleep Medicine (AASM, 2007), using frontal, central, and occipital head electrodes with reference electrodes at the mastoids. Central EEG (C4-A1; C3-A2), frontal EEG (C4-F4), and occipital EEG (A1-O2) recordings were obtained. EOG electrodes were placed at the outer canthi of the orbits and EMG was measured at the chin.

Sleep stages were scored according to the rules of the AASM (2007). This allowed determination of total sleep time (TST) defined as the total sleep period minus periods awake during this period, sleep efficiency index (SEI) defined as the ratio of TST/Time in Bed, sleep latency (SL), and relative duration of sleep stages all expressed as percentage of Sleep Period Time (SPT; time from sleep onset to last epoch of sleep). ECG was measured during the whole Time in Bed period. For the purpose of the current study, we focus on % sleep efficiency, sleep latency, % WASO (Wake After Sleep Onset), % REM SPT, % Stage 1 Sleep (S1), and ECG.

Statistical procedure

Statistical analyses were performed using SPSS 18.0 software. Cronbach's α values were used to estimate internal consistency of the DES, SCL-90, ISES, PSQI, BISQ, and TEC. Pearson product-moment correlations were used to examine the links between the psychometric data and sleep parameters.

Results

Table 3.1 shows internal consistency and mean (*SD*) scores of all self-report measures as well as Pearson product-moment correlations between the measures. Mean scores on DES were significantly higher compared to mean DES scores in an American/Canadian community sample ($N = 415$; $t = 4.13$, $p < .001$; Sno, Schalken, op den Velde, & Aarts, 2002). Mean scores on DES did not significantly differ from those obtained in a sample of 386 Dutch outpatients suffering from a variety of psychopathology ($t = .96$, $p = .34$; Sno et al., 2002). Four out of 46 participants (8.7%) displayed dissociation levels exceeding the clinical cut-off score for dissociative disorders (i.e., DES scores > 30; Bernstein-Carlson & Putnam, 1993). SCL-90-R scores were comparable to those of primary care patients ($M = 164.3$, $SD = 49.9$, $t = 0.14$, $p = .89$; Kloens, Barelds, Luteijn, & Schaap, 2012), and significantly differed from normal controls ($M = 123.9$, $SD = 32.5$, $t = 7.16$, $p < .001$). Significant correlations were found between self-reported dissociative symptoms (DES) and the SCL-90-R-GSI Scale, on the one hand, and the two sleep measures (ISES; PSQI), on the other hand, indicating that heightened dissociative levels and general psychopathology were related to poor sleep quality and unusual sleep

experiences. Self-reported traumatic experiences (TEC) were not related to dissociation (DES), but were significantly related to SCL-90-R: GSI, unusual sleep experiences (ISES), and sleep quality (PSQI), suggesting that patients with a history of traumatic experiences showed elevated general psychopathology and unusual sleep experiences, as well as poor sleep quality.

Polysomnographic measures showed that patients had a mean sleep onset of 42 minutes (*SD* = 38; *range*: 10 – 210). Mean sleep onset latency was 23 minutes (*SD* = 21; *range*: 4.2 – 112.3), and mean number of minutes awake after sleep onset was 48 minutes (*SD* = 37; range = 1 – 164). Their subjective estimate of the hours spent asleep was 5.2 hours (*SD* = 1. 9 hours; *range*: 2 – 10 hours).

Table 3.1.
Mean scores and Pearson product-moment correlations between self-report measures (*N* = 45).

	Cronbach's alpha	*Mean (sd)*	*DES*	*SCL-90*	*ISES*	*PSQI*
DES	.94	14.23 (8.56)	-	-	-	-
SCL-90	.98	165.44 (46.43)	.51**	-	-	-
ISES	.90	44.98 (20.01)	.40**	.37*	-	-
PSQI	.63	9.50 (3.06)	.45**	.43**	.26	-
TEC	.82	2.67 (2.46)	.10	.39**	.49**	.39*

Note. DES = Dissociative Experiences Scale; SCL-90 = Symptom Checklist 90; ISES = Iowa Sleep Experiences Survey; PSQI= Pittsburgh Sleep Quality Index; TEC = Traumatic Experiences Checklist

* $p < .05$

** $p < .01$

Table 3.2 displays the Pearson product-moment correlations between self-report measures and objective sleep parameters obtained during polysomnographic recordings. As expected, the DES correlated positively with % REM SPT. Because antidepressants may affect REM sleep, we conducted an independent samples *t*-test to compare the % REM SPT in patients using medication, and patients not on medication. Less time spent in REM sleep was not related to the use of antidepressant medication ($t(42) = -1.07$, $p = .29$). Also, the DES correlated negatively with time spent awake during sleep period time. Finally, ISES correlated significantly and positively with mean ECG rate and with total REM time in minutes.

Table 3.2
Pearson product-moment correlations between self-report measures and sleep parameters (N = 45).

	DES	*SCL-90*	*ISES*	*PSQI*	*TEC*
% sleep efficiency	.27	.14	.21	-.12	.24
Sleep onset	-.05	.02	.04	-.10	-.12
WASO	**-.32***	-.21	-.26	.18	-.26
Total REM	.26	.22	**.34***	.01	.20
% REM SPT	**.31***	.19	.23	.05	.06
% S1 SPT	-.12	-.04	.11	.07	.26
ECG	.22	.26	**.41****	.10	.29

Note. DES = Dissociative Experiences Scale; SCL-90 = Symptom Checklist 90; ISES = Iowa Sleep Experiences Survey; PSQI= Pittsburgh Sleep Quality Index; TEC = Traumatic Experiences Checklist; Sleep onset = sleep onset latency in minutes; WASO = wake after sleep onset, percentage awake during the whole sleep period (SPT = sleep period time); Total REM = total REM sleep in minutes; % REM SPT = percentage REM sleep during the whole sleep period; % S1 SPT = percentage Stage 1 sleep during sleep period time; ECG = mean score on electrocardiogram.
* $p < 0.05$
** $p < 0.01$

Participants rated the sleep quality items of the BISQ on scales ranging from 1 (very poor) to 10 (excellent). Their mean rating was 5.2 (*SD* = 1.82). As shown in table 3.2, the BISQ correlated with several objective sleep parameters, indicating that patients' subjective experience of sleep quality was in line with objective measures such as sleep efficiency, total sleep time (in minutes), time spent awake, time in REM, and time in S1 (van Even, 2004).

Patients were also asked to indicate on a 5-point Likert scale (range: 0 = 'not at all sleepy', to 4 = 'very sleepy') how sleepy they felt upon awakening. Many of them (61 %) scored '2' or more, indicating that they felt 'rather sleepy' to 'very sleepy'. A majority (73%) of the patients said that their sleep complaints were unacceptable to bear. Sleepiness upon awakening was significantly correlated with DES scores, r = .45, $p < .01$. As shown in table 3.3, objective sleep efficiency correlated positively with subjective total sleep time, and subjective sleep quality. Moreover, objective time spent awake during the night correlated negatively with subjective time spent awake and subjective sleep quality.

Table 3.3
Pearson product-moment correlations between subjective sleep measure (BISQ; horizontal row) and objective sleep parameters (polysomnography; left column; *N* = 45).

	Sub. sleep onset	Time wake during night	Subjective TST	Sleep quality (1-10)
Sleep onset	**.34***	.18	-.01	-.25
SOL	**.41****	.01	.03	-.05
SPT	**-.33***	-.04	**.38***	-.04
TST	**-.51****	**-.36***	**.52****	.25
SE %	**-.57****	**-.50****	**.42****	**.42****
SPT wake	**.31***	**.65****	**-.37***	**-.38***
REM TST	-.24	**.47****	**.33***	.09
S1 TST	-.10	-.12	**.45****	**.38***
S2 TST	**-.44****	-.25	**.45****	.17
ECG	-.04	-.10	.18	-.01

Note. SOL = Sleep onset latency; SPT = Sleep Period Time; TST = Total Sleep Time; SE % = Sleep Efficiency in Percentage; SPT Wake = minutes awake during sleep period time; REM TST = minutes of REM sleep during total sleep time; S1 TST = minutes of Stage 1 sleep during total sleep time; S2 TST = minutes of Stage 2 sleep during total sleep time; ECG = mean score on electrocardiogram.
* $p < .05$
** $p < .01$

Discussion

This study is – to the best of our knowledge – the first to examine dissociative symptoms in patients with insomnia. Before we discuss the conclusions that can be drawn from our findings, it is important to emphasize some limitations of the current study. Although our sample consisted of a unique and rather homogenous group of patients with insomnia, its size was relatively small. Thus, future research might want to include a larger and more heterogeneous sample of patients with sleep problems, preferably also outpatients. Furthermore, medication did not seem to have affected our results, as there was no statistically significant difference between patients who used medication and those who did not. Nevertheless, follow-up research should address the issue of medication and how it relates to the sleep-dissociation link in a more systematic way. Most importantly, our study relied on a cross-sectional design that precludes any causal interpretations. Also, all measures were moderately related to each other, with the exception of ISES and PSQI. This pattern makes it difficult to formulate specific hypotheses. Thus, a longitudinal set-up in which patients undergoing targeted treatments for their sleep dysfunctions are followed over time would provide a more optimal starting point for testing causal hypotheses.

With these limitations in mind, the main findings of our study can be catalogued as follows. First, we found that as a group, patients suffering from insomnia had heightened dissociative levels, which is in keeping with research

showing a solid link between sleep experiences and dissociative symptoms in non-clinical samples (Watson, 2001; Giesbrecht & Merckelbach, 2004; 2006; Soffer-Dudek & Shahar, 2011).

Second, dissociative symptoms did not correlate with self-reports of traumatic experiences, but trauma was positively related to the two sleep measures (ISES and PSQI). This is in accordance with the idea that sleep disturbances rather than traumatic experiences per se act as stage setter for dissociative symptoms. Because our study was cross-sectional in nature, it does not allow for strong causal conclusions. However, one distinct possibility is that specifically aversive childhood experiences rather than aversive experiences in general, might lead to sleep disturbances, which in turn might serve as the more proximal antecedents of dissociative symptoms. This interpretation might reconcile seemingly conflicting theories about the origins of dissociative symptoms, with some theories emphasizing the traumatic origins of dissociative symptoms and other theories stressing the etiological role of sleep disturbances (Spiegel et al., 2011; Giesbrecht et al., 2008; Bremner, 2010).

Third, we found that subjective sleep evaluations of insomnia patients often correlate with objective sleep parameters, which supports the integrity of the subjective sleep data obtained in this group.

Fourth and most importantly, our study is the first to document that raised dissociative scores are not only related to subjective sleep parameters, but also to objective sleep indices. More specifically, we found that dissociative symptoms are associated with increased periods of sleep time spent in REM sleep, and with more sleepiness at awakening. This is reminiscent of recent literature about REM sleep and psychopathology. For example, 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" (p. 483). Thus, an influx of dreamlike mentation during the day may fuel dissociative symptoms. A related view is the notion of transliminality (Thalbourne & Houran, 2000), which assumes that there exist robust individual differences in the extent to which mentation may cross thresholds into and out of consciousness. Using a self-report scale that intends to measure this trait – the Revised Transliminality Scale (RTS) – Soffer-Dudek and Shahar (2009) showed in a longitudinal study that people who score high on transliminality (i.e., who are attuned to their inner fantasy life) subsequently report more unusual sleep experiences than those who score low on this trait.

There is consensus that REM sleep is important for emotional memory formation (Walker & van der Helm, 2009). For instance, Crick and Mitchison (1995) emphasized that REM sleep plays a role in memory consolidation. Our finding that dissociative symptoms are related to more intense REM periods might reflect

heightened cognitive effort to cope with irrelevant and noisy memory traces, especially when the memory is emotional in nature (Walker & van der Helm, 2009). Also germane to this issue is the conceptualization of Llewellyn (2012). This author summarizes evidence to show that REM dreaming is more perceptually vivid and hyperassociational compared to the waking state, and argues that during dreaming there is an enhanced access to remote memory material. REM dreaming may therefore provide the ideal state for elaborative encoding of emotional memories. However, when dreaming and waking states become de-differentiated, it is conceivable that the hyperassociational mentation may intrude consciousness and contribute to dissociative experiences. Note that we observed in previous studies (LaVia & Brewerton, 1996; van der Kloet et al., 2012) a connection between dissociative symptoms and narcoleptic experiences, which represent a paradigmatic example of de-differentiation of dreaming and waking. Clearly, disturbed sleep-wake cycles bear relevance to clinical practice, as is also demonstrated by case reports about patients with narcolepsy who misinterpret their dreamlike hallucinatory experiences as real events and sincerely believe that they have been the victim of sexual assault or another offence (LaVia & Brewerton, 1996).

Fifth, dissociation was related to less time spent awake during the night. This contradicts an earlier study that found dissociative symptoms in a healthy sample to be related to more fragmented sleep (Giesbrecht et al., 2007). We have no ready explanation for these conflicting findings but they might be explained by the different samples (patients with insomnia versus healthy individuals), the different methods for measuring fragmented sleep in the two studies, and the different context (psychological laboratory versus sleep laboratory). Also, it is well known among clinicians that patients suffering from insomnia tend to sleep better during recordings in the hospital than they sleep at home.

Finally, a higher heart rate during the night correlated with a higher score on unusual sleep experiences (ISES). This is intuitively plausible because it stands to reason that patients who experience more sleep aberrations during the night react with elevated arousal responses to such episodes, although the causal relation may of course be the other way around. In general, then, it appears that dissociative symptoms are more related to the cognitive aspects of sleep (i.e., REM sleep), while self-reported sleep disturbances are primarily related to the arousal aspects of sleep.

In conclusion, our finding that dissociation levels are elevated in patients suffering from insomnia and are related to various sleep parameters suggests several avenues for future research. Research that addresses the sleep-dissociation link in clinical samples is urgently needed, because most previous studies have relied on student samples. Future studies might elucidate the type of sleep architecture that is most reliably associated with different dissociative disorders in

a longitudinal design, and then establish remediation programs, including medication regimens, to address underlying sleep deficits and irregularities. This would be an entirely novel and exciting lead.

CHAPTER IV[6]

Night-time experiences and daytime dissociation: A structural equation modeling study

6 This chapter is an adapted version of the following article:
Van Heugten – Van der Kloet, D., Merckelbach, H., Giesbrecht, T., & Broers, N. (2013). Night-time experiences and daytime dissociation: A structural equation modeling study. *Psychiatry Research, under revision*.

Summary

Dissociative symptoms may be the by-products of a labile sleep-wake cycle (Koffel and Watson, 2009a). This may help to explain why dissociation overlaps with fantasy proneness and cognitive failures. Using structural equation modeling, we tested to what extent data gathered in a nonclinical sample (*N* = 139) supported two causal models. The first model assumes that unusual sleep experiences increase fantasy proneness and cognitive failures, which in turn encourage trait dissociation and reports of trauma. The second model assumes that trauma leads to dissociative experiences both directly and through its influence on sleep. The data were reasonably well described by both models. Importantly, in both models, unusual sleep experiences serve as antecedents of trait dissociation and reversing the causal direction of this link produced a poor fit with the data. Our analysis underlines the importance of unusual sleep experiences and may inspire treatment intervention focusing on sleep normalization.

Introduction

The *Diagnostic and Statistic Manual of Mental Disorders* defines dissociation as "a disruption in the usually integrated function of consciousness, memory, identity, or perception of the environment" (DSM-IV-TR; APA, 2000, p. 519). Dissociation is often viewed as a continuum ranging from benign to pathological experiences, with depersonalization and dissociative identity disorder situated at the pathological end (Giesbrecht & Merckelbach, 2008; Holtgraves & Stockdale, 1997; Irwin, 2001). Over the past decades, numerous studies have assessed the prevalence of dissociative symptomatology in various populations. General population surveys found that dissociative disorders may affect 3.3% to 11.2% of the population (Loewenstein, 1994; Murphy, 1994; Waller & Ross, 1997). Moreover, dissociative symptoms are common in certain clinical groups, such as mood disorders, post-traumatic stress disorder (PTSD), and persons with schizotypal traits (Brand & Loewenstein, 2010; Galbraith & Neubauer, 2000; Giesbrecht & Merckelbach, 2008). Research has also documented a link between sleep paralysis and dissociative symptoms in people who report childhood trauma experiences (McNally & Clancy, 2005a). This finding dovetails with the work of Watson (2001) and Koffel and Watson (2009a) who presented evidence to the effect that daytime dissociation is a function of problematic sleep–wake state boundaries, which may be precipitated by trauma.

It is well established that good sleep conveys cognitive benefits, while sleep problems may undermine cognitive efficiency (Walker & Stickgold, 2006). Sleep problems are usually defined as having trouble falling asleep, experiencing awakenings during the night, waking up too early, or not feeling rested after a night's sleep. However, sleep problems may also entail unusual sleep experiences such as having nightmares or vivid dreams, recurring dreams, sleep paralysis, and hypnagogic or hypnopompic hallucinations (Watson, 2001). Sleep problems are common in the general population. For instance, it is estimated that a third of the Dutch population now and then experiences sleep problems, with 15% suffering from insomnia (Fonds Psychische Gezondheid, 2009). Most importantly, disruptions in sleep patterns figure prominently in mood and anxiety disorders, schizophrenia, and borderline personality disorder (Benca et al., 1992; Morin & Ware, 1996). For example, 70-91% of the patients diagnosed with PTSD suffer from sleep problems such as falling asleep or staying asleep, and up to 71% of them report nightmares (Maher et al., 2006).

In a pioneering study on dissociation and sleep in two large student samples, Watson (2001) observed that unusual sleep experiences (e.g., sleep paralysis, hypnagogic hallucinations) are related to dissociative symptoms. Since then, a number of studies have replicated this observation (Giesbrecht & Merckelbach, 2004; Watson, 2003; Van der Kloet et al. 2011; see for a review: Van der Kloet et

al., 2012; chapter I). There is also anecdotal evidence that suggests an intimate relationship between dissociation and cataplexy (LaVia &Brewerton, 1996), a hallmark feature of narcolepsy, and people suffering from Depersonalization Disorder often say that their symptoms are most pronounced when they feel tired (Simeon & Abugel, 2006). Furthermore, researchers have found a substantial overlap between dissociative experiences and nightmare frequency (Agargun et al., 2003; Levin & Fireman, 2002b; Soffer-Dudek & Shahar, 2011), as well as dreaming states (Barrett, 1994). Moreover, one sleep deprivation study in healthy people reported that sleep loss intensifies dissociative symptoms (Giesbrecht et al., 2007). Taken together, these studies suggest that dissociative symptoms may be the by-products of a labile sleep-wake cycle (Koffel & Watson, 2009a; Van der Kloet et al., 2012; chapter I). This view assumes thin boundaries between the sleeping and waking states. These thin boundaries would allow for the intrusion of dreamlike content into the waking state, which would set the stage for dissociative symptoms. This conceptualization of dissociation is sometimes referred to as *cross-state continuity* and it is based on the assumption 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). The link between sleep and dissociation appears to be rather specific in that dissociation is related to unusual sleep phenomena that are difficult to control. Thus, nightmares and waking dreams are related to dissociative symptoms and schizotypy, while insomnia and lassitude seem to be related to mood and anxiety (Koffel & Watson, 2009b).

The sleep-dissociation link may help to explain why dissociation overlaps with fantasy proneness and cognitive failures. Fantasy proneness is usually defined as a disposition to engage in vivid and extensive fantasizing. Several studies in clinical (Boom et al., 2010; Kunst et al., 2011) and non-clinical samples (Merckelbach et al., 2000; Giesbrecht & Merckelbach, 2006; Rauschenberg & Lynn, 1995) found a positive correlation between dissociative symptoms, as measured by the *Dissociative Experiences Scale* (DES; Bernstein & Putnam, 1986) or the *Peri-traumatic Dissociative Experiences Questionnaire* (PDEQ; Marmar et al., 1997), and fantasy proneness, as measured by the *Creative Experiences Questionnaire* (CEQ; Merckelbach et al., 2001). Cognitive failures refer to everyday slips and lapses (Broadbent et al., 1982) and are frequently reported by highly dissociative individuals (Giesbrecht et al., 2004; Levin et al., 2004; Merckelbach et al., 2002; Merckelbach et al., 1999) and dissociative patients alike (Dorahy et al., 2006; Simeon et al., 2009).

There is some debate as to whether childhood trauma is a necessary causal antecedent of dissociative symptomatology (Lynn et al., 2012; Giesbrecht et al., 2010; Bremner, 2010; Dalenberg et al., 2012). That fantasy proneness and cognitive failures correlate with dissociation is an important fact to consider for theories on

the origins of dissociative symptoms. Specifically, some authors have argued that the combination of fantasy proneness and cognitive failures may lead to biased reports of traumatic childhood events (i.e., may lower respondents' criteria for reporting ambiguous events as traumatic in nature; e.g., Merckelbach & Jelicic, 2004). Of course, such an interpretation is far removed from the view that dissociation is a defensive response to childhood trauma (Spiegel et al., 2011; Dalenberg et al., 2012).

Germane to this debate is a study by Merckelbach and colleagues (2002) who contrasted a model in which (self-reports of) trauma causally precede dissociation and a concurrent model in which heightened levels of fantasy proneness and cognitive failures constitute dissociation and together precede self-reports of trauma. Their structural equation modeling results performed on data gathered in an undergraduate sample led the authors to conclude that the data provided a similar degree of support to both models (Merckelbach et al., 2002).
The structural equation modeling (SEM) approach of Merckelbach et al. (2002) did not take sleep disturbances as a potential antecedent of dissociation into account. With this in mind, the present study investigated whether data gathered in an undergraduate sample would support a model in which sleep disturbances precede dissociative symptoms. Using structural equation modelling we compared two models. Specifically, we tested whether the data are best described by trauma leading to dissociative experiences both directly and through its influence on sleep (model 1), or by unusual sleep experiences fuelling fantasy proneness and cognitive failures, which in turn constitute trait dissociation and contribute to reports of trauma (model 2).

Method

Participants and Procedure

Participants were 139 undergraduate students (122 women) with a mean age of 21.4 years (*range*: 17 to 32 years). They received written and oral information about the study, after which they gave written informed consent. Participants completed a baseline screening, containing self-report questionnaires, via the user-friendly software program EMIUM (Janssen, 2008). They kept a diary for three weeks, three days per week, twice a day. This diary involved self-report measures on dreams and night-time experiences, but also measures of state dissociation, sleepiness, and mood during the day. After completion of all questionnaires, participants were rewarded with course credits or a small monetary reward. For the purpose of the present chapter, we restrict our analyses to data from the baseline screening (i.e., DES, ISES, CFQ, CEQ, CTQ-SF). The study was approved by

the standing ethical committee of the Faculty of Psychology and Neuroscience, Maastricht University, The Netherlands.

Measures

Dissociative Experiences Scale (DES; Cronbach's α = .93; Bernstein & Putnam, 1986). The DES is a self-report scale that requires participants to indicate on 100 mm visual analogue scales (anchors: 0 = never; 100 = always) to what extent they experience 28 dissociative experiences in daily life. Examples of such experiences include feelings of depersonalization and derealization, and memory difficulties (i.e., psychogenic amnesia). Van IJzendoorn and Schuengel (1996) provide meta-analytic evidence for the sound psychometric properties of the DES. We calculated the DES total score (divided by 28).

*Iowa Sleep Experiences Survey (*ISES; Cronbach's α = .78). The ISES (Watson, 2001) consists of 18 questions asking the respondent to rate the frequency of various sleep- and dream-related experiences on a 7-point Likert scale (anchors: 1 = *never*, 7 = *several times a week*). The ISES consists of 2 separate subscales, which measure general sleep experiences (e.g., "I have recurring dreams.") and lucid dreaming (e.g., "I am aware that I am dreaming, even as I dream"), respectively. Whereas the General Sleep experiences subscale taps into symptoms of narcolepsy, vivid and unusual dreams, and other nocturnal experiences, the Lucid Dreaming subscale consists of several items about knowing that you are dreaming while still being asleep. In the present study, we restricted our analyses to the General Sleep experiences subscale of the ISES. This subscale has consistently been shown to be related to dissociative symptoms, whereas prior studies found the relationship between dissociation and the Lucid Dreaming subscales to be modest at best. To obtain a total General Sleep subscale score, we summed up the ISES items 1 through 15.

Cognitive Failures Questionnaire (CFQ; Cronbach's α = .78). The CFQ (Broadbent, et al., 1982) is a 25-item self-report instrument tapping everyday lapses in perception and attention (e.g., "So you fail to notice signposts on the road?"), memory (e.g., "Do you forget appointments?"), and actions (e.g., "Do you bump into people?"). The CFQ has sound psychometric properties. Participants are requested to indicate on 5-point scales how often they experienced each cognitive failure during the past month (anchors: 0 = *never*; 4 = *very often*). Scores are summed to obtain a total CFQ score, with higher scores indicating a higher frequency of self-reported failures. Merckelbach and colleagues (1996) reported that the Dutch version of the CFQ has adequate psychometrical properties.

Creative Experiences Questionnaire (CEQ; Cronbach's α = .72; Merckelbach et al., 2001). The CEQ is a Dutch instrument to measure fantasy proneness. It consists of 25 yes/no items measuring daydreaming, intense fantasies, and imagination. The

items of the CEQ were derived from extensive case vignettes on fantasy proneness provided by Wilson and Barber (1983). Illustrative items are "I spent more than half of the day on fantasizing or daydreaming" and "Many of my fantasies are as vivid as a good movie". A total CEQ score is obtained by summing the number of items that are endorsed.

Childhood Trauma Questionnaire – Short Form (CTQ-SF; Cronbach's α = .91; Bernstein et al., 2003). The CTQ is a widely used self-report scale designed to assess five types of childhood maltreatment: 1) physical abuse, 2) emotional abuse, 3) sexual abuse, 4) physical neglect, and 5) emotional neglect. In the present study, we employed the Dutch short form, which consists of 28 items. These are scored on 5-point scales anchored 1 (never) and 5 (very often). The Dutch CTQ-SF possesses adequate internal consistency and test-retest reliability and it effectively discriminates between clinical and non-clinical samples (Thombs et al., 2009). Items are summed to obtain a CTQ-SF total score.

Data Analysis

Statistical analyses were performed using SPSS 18.0 and LISREL software. Cronbach's α values were used to estimate internal consistency of the baseline measures. Mean scores and Pearson product-moment correlations between baseline measures were calculated.

Together with standard deviations, correlations served as the input for structural equation modeling which was carried out with LISREL (Student Edition version 8.80). Using SEM, we are able to test how well the empirically derived correlational patterns support the models that we sketched above (Schumacker and Lomax, 1996). Basically, this technique makes it possible to determine whether and to what extent an a priori formulated causal model is consistent with the observed data. Given the relatively small sample size (*N* = 139), we decided to obtain several fit indices for the two models. Thus, the following fit indices are reported (also see Schumacker & Lomax, 1996): 1) the Chi square Goodness-of-Fit value that has to be non-significant for the tested model to be considered a good fit with the observed data; 2) the root mean square error of approximation (RMSEA) that is relatively insensitive to sample size and indicates the fit of the model in relation to degrees of freedom. RMSEA values below 0.05 imply a close fit of the model (Browne & Cudeck, 1993), and 3) the Tucker-Lewis non-normed fit index (NNFI) that according to some authors (e.g., Lawrence et al.,1995) provides a particularly good estimate of how well a model fits when data are based on small sample sizes. NNFI values that exceed the .9 level are indicative of well-fitting models.

Results

Scores on the ISES, CEQ, and CFQ were normally distributed, but DES and CTQ were skewed to the right. For the purpose of the SEM analyses, we decided to perform data transformation on the scores of the latter two scales. After a shift transformation to set the minimum DES score equal to 1, a square root transformation was applied to the DES scores. For CTQ, we used logarithm transformation. First, we subtracted 24, so as to set the minimum equal to 1. This transformation resulted in symmetrically distributed DES and CTQ scores. Transformed scores were highly correlated with the original scores (i.e., *r's* = .85 - .98). Table 4.1 gives mean scores and Pearson product-moment correlations between baseline measures. Both original and transformed scores are shown. With one exception, correlations involving original and transformed measures never differed more than .05. The exception concerns the correlation between CTQ and CEQ, which fell from .32 to .21 after data transformation. Mean scores on dissociation (DES), cognitive failures (CFQ), and fantasy proneness (CEQ), and self-reports of trauma (CTQ) were comparable to results obtained in previous non-clinical studies (Giesbrecht & Merckelbach, 2004; 2006).

Table 4.1.
Mean scores and Pearson product-moment correlations of baseline measures (*N* = 139), transformed scores in *italics*.

	Mean (*SD*)	*1*	*2*	*3*	*4*
1.ISES	45.41 (10.00)	-	-	-	-
2.CTQ *CTQlon*	33.47 (9.72) *1.75 (1.07)*	.26* (*.21**)	-	-	-
3.CFQ	64.81 (9.05)	.23*	.22**(*.23***)	-	-
4.CEQ	6.22 (3.49)	.36*	.32*(*.21**)	.20*	-
5.DES *sqrtDES*	18.49 (12.96) *20.97 (8.21)*	.39**(*.41***)	.35**(*.30***)	.41**(*.43***)	.37**(*.36***)

Note. ISES = Iowa Sleep Experiences Survey; CTQ = Childhood Trauma Questionnaire (Log transformation); CFQ = Cognitive Failures Questionnaire; CEQ = Creative Experiences Questionnaire ; DES = Dissociative Experiences Scale (Square Root transformation).
* $p < .05$
** $p < .01$

Structural Equation Modeling

Table 4.2 shows fit indices for the two models specified in Figures 4.1 and 4.2. Model 1 (the Trauma-Dissociation model) assumes that childhood trauma (CTQ) is a causal antecedent. This model seems to describe the data well and after removal of the non-significant path from unusual sleep experiences (ISES) to cognitive failures (CFQ), the model fit becomes excellent, see table 2 and Fig. 4.1.

Table 4.2.
Fit indices of the Trauma-Dissociation model (model 1) and the Dissociation-Trauma model (model 2), see also Fig. 4.1 and Fig. 4.2.

	Chi Square	*P*	RMSEA	NNFI
Model 1 (df = 4)	3.64	.46	.00	.99
Model 2 (df = 4)	6.26	.18	.07	.95

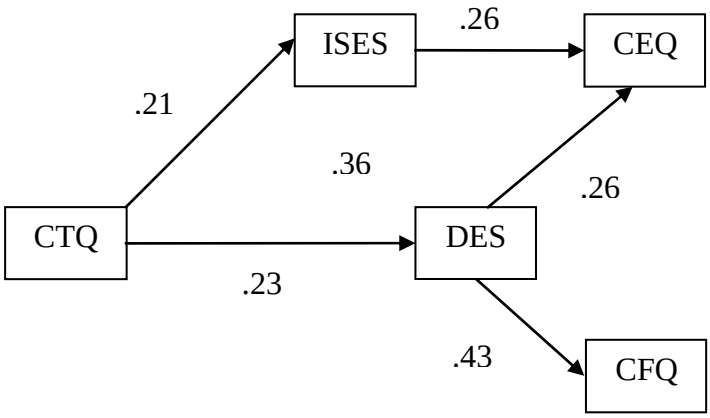


Figure 4.1.

The Trauma-Dissociation model (model 1). Reported are the standardized pad-coëfficients.

Next, we explored model 2 (the Dissociation-Trauma model), which had a problematic fit (Chi square = 29.96, $p < .001$; RMSEA = .13, and NNFI = .77). However, there were only 2 variables larger than 2 in the standardized residuals. More specifically, the correlations between trait dissociation (DES) and unusual sleep experiences (ISES) were poorly reproduced. The overview of modification indices suggested adding a direct effect from unusual sleep experiences (ISES) to dissociation (DES). We incorporated this amendment in a second step. The revised model 2 had a good model fit, see table 4.2 and Fig. 4.2.

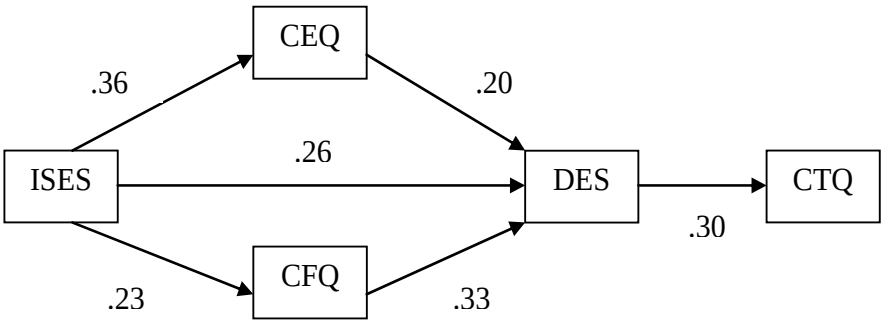


Figure 4.2.

The Dissociation-Trauma model (model 2). Reported are the standardized pad-coëfficients.

To sum up, model 1 gave an acceptable fit of the data without any modifications. Model 2 started out with a poor fit of the data, but modification of the model ultimately led to a reasonable model fit. Thus, both models are consistent with the data. Longitudinal research is warranted to determine which of the two models is closest to the empirical data. Interestingly, in both examined models, unusual sleep experiences (ISES) serve as a determinant of trait dissociation (DES). Importantly, reversing the causal chain by swapping the DES and ISES, led to a substantially poorer fit of both models.

Discussion

This study explored different causal models to describe the relations between dissociation, unusual sleep experiences, self-reports of childhood traumatic experiences, and other related constructs. Our findings can be catalogued as follows.

Replicating previous research (e.g., Giesbrecht & Merckelbach, 2006) we found dissociation to be related to fantasy proneness, and cognitive failures. Also, as was found in many previous studies (for a review, see Dalenberg et al., 2012), dissociation was significantly and positively related to self-reports of childhood traumatic experiences. This is in line with researchers who assume that trauma causes dissociation within clinical and nonclinical groups (Allen et al., 2002). Nevertheless, the link between dissociation and trauma might, at least to some extent, be inflated due to absentmindedness implied by cognitive failures and/or exaggeration implied by fantasy proneness (Merckelbach et al., 2000; Merckelbach et al., 2002).

Second, we also replicated the finding that dissociative symptoms are related to unusual sleep experiences (e.g., Koffel & Watson, 2009a, Giesbrecht et al., 2006, Van der Kloet et al., 2012). Thus, not only trauma reports, fantasy proneness, and cognitive failures can be considered solid correlates of dissociation, the same is true for unusual sleep phenomena such as nightmares, sleep paralysis, or hypnagogic hallucinations. Clearly, unusual experiences deserve a place in causal models that try to explain the origins of dissociative symptoms.

Third, using SEM, we analyzed the goodness of fit for two theoretically motivated causal models. Our analyses did not unambiguously favor any of the two. Thus, while the significant correlations between dissociation, fantasy proneness, cognitive failures, and trauma self-reports are well replicated, it remains difficult to determine the precise causal flow between these variables. Taken together with the findings from Merckelbach et al. (2002), one may be tempted to speculate that both models represent two different potential pathways

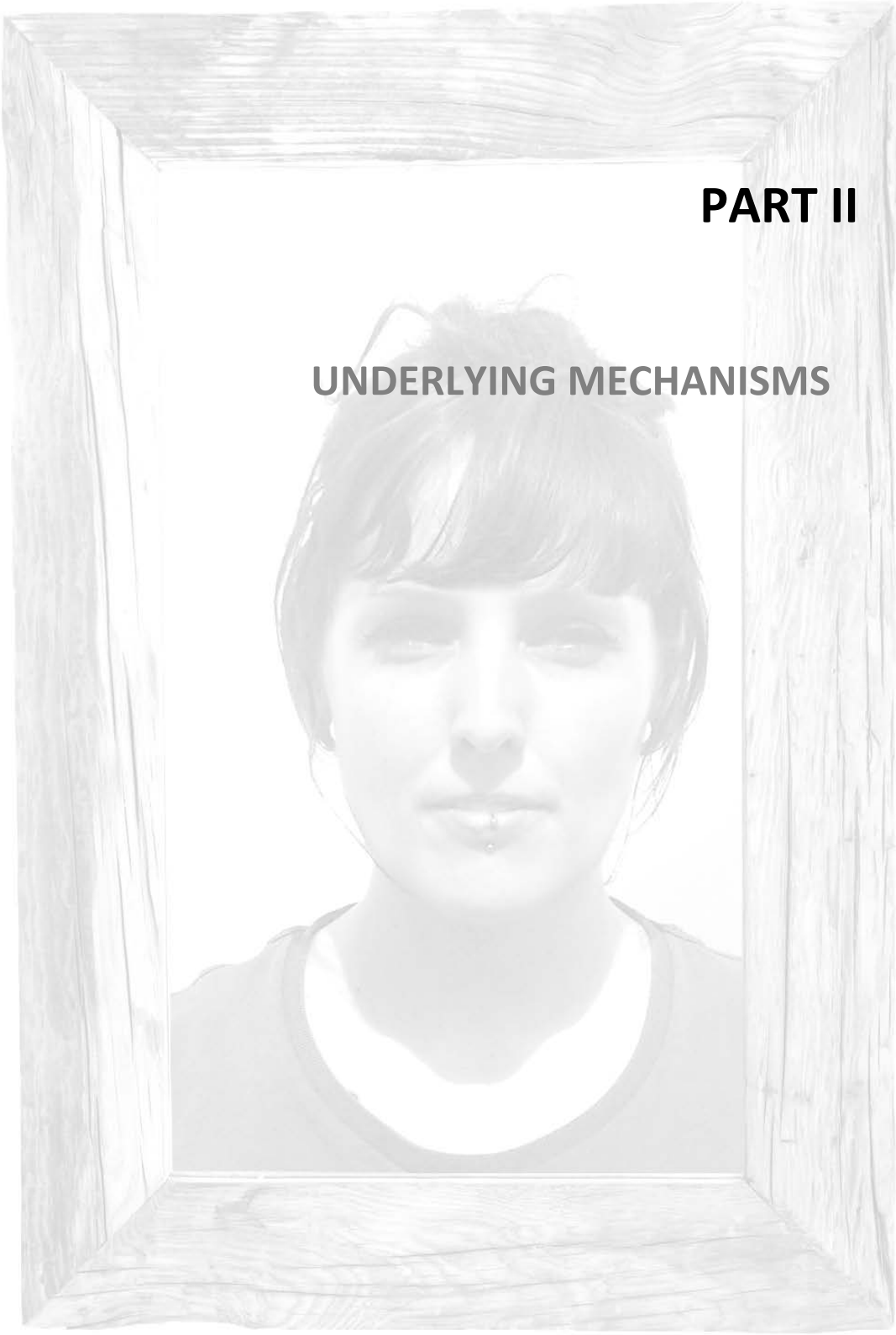
to dissociation. This would explain why there are two seemingly contradictory lines of research in dissociation research with some scientists advocating a clear role of trauma in dissociation (e.g., Dahlenberg et al., 2012), and others emphasizing the substantial heritability of dissociative experiences (Jang et al., 1998).

Fourth, both models clearly imply that unusual sleep experiences seem to precede dissociation. Thus, it seems that unusual sleep experiences can contribute to more sophisticated models about the origins of dissociation. Previous studies leave no doubt that such experiences are highly prevalent in people with dissociative disorders (Van der Kloet et al., 2012) and in trauma survivors (Maher et al., 2006). Interestingly, among participants who report memories of childhood sexual abuse, experiences of sleep paralysis[7] typically are accompanied by raised levels of dissociative symptoms (McNally & Clancy, 2005a; Abrams et al., 2008). While our SEM findings are inconclusive concerning the causal role of trauma, both models clearly suggest that unusual sleep phenomena lead to higher levels of dissociation.

A focus on sleep disturbances might inspire new treatment methods aimed at sleep hygiene interventions for people suffering from dissociative disorders. Germane to this idea, a review of Lamarche (2007) showed that treatments for sleeping difficulties in people with posttraumatic stress disorder (PTSD) significantly improved sleep, but also lead to a significant decrease in other PTSD symptoms, including dissociation. Interventions along these lines might be also be promising for other forms of psychopathology that are accompanied by high levels of dissociation (e.g., eating disorders, schizophrenia). Thus, future research in this area may inspire the development of new treatment methods based on sleep normalization.

Some restrictions of the current study merit attention. Limitations include the cross-sectional design of this study and its relatively small sample size. Also, although we employed well-validated measures, our study heavily relied on self-reports in a nonclinical sample. With such a sample, relatively low rates of childhood trauma are expected. This implies a certain restriction of range, which may have influenced the explanatory power of the models that we evaluated. It may well be the case that in a sample with longitudinally obtained data of verified childhood trauma, the predictory range of childhood trauma is entirely different from the one found in the current study (but see Cima et al., 2001; Sanders & Giolas, 1991). Also, it should be acknowledged that the choice and interpretations of goodness-of-fit data has been the subject of intense debate (Schumacker & Lomax, 1996). Thus, further research is warranted to determine which etiological model is closest to the empirical truth.

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



PART II

UNDERLYING MECHANISMS

CHAPTER V[8]

Sleep loss increases dissociation and affects memory for emotional stimuli

8 This chapter is an adapted version of the following article:
Van Heugten – Van der Kloet, D., Giesbrecht, T., & Merckelbach, H. (Submitted). Sleep loss increases dissociation and affects memory for emotional stimuli. *Psychological Medicine, submitted*.

Summary

Because of the dreamlike character of dissociative symptoms, researchers have started to investigate the role that disturbances in the sleep-wake cycle may play in these symptoms. In the present study, we build on earlier findings of Giesbrecht and colleagues (2007) who found that sleep loss fuels dissociative symptoms. Fifty-six healthy undergraduate students were divided in an experimental group (*N* = 28) and a control group (*N* = 28). The experimental group was deprived of sleep for 36 hours in a sleep laboratory at the university, during which time the control group had a regular night of sleep. Every 6 hours sleepiness, mood, and dissociative symptoms were assessed in the experimental group. Furthermore, several cognitive tasks were administered. Sleep deprivation led to an increase in dissociative symptoms, which was mediated by levels of general distress. Dissociative symptoms, sleepiness, and mood deterioration followed the same oscillating pattern of the sleep-wake cycle, remaining stable during the day and increasing during the night. Feelings of sleepiness preceded an increase of dissociative symptoms and deterioration of mood. Finally, sleep loss also undermined memory of negative material, and reduced the executive control function, especially in highly dissociative individuals.

Introduction

The *Diagnostic and Statistic Manual of Mental Disorders* defines dissociation as "a disruption in the usually integrated function of consciousness, memory, identity, or perception of the environment" (DSM-IV-TR; American Psychological Association, 2000, p. 519). Dissociative symptoms form a heterogeneous group, and many researchers distinguish between three distinct clusters of dissociation: Amnesia, depersonalization/derealization, and absorption (e.g., Koffel & Watson, 2009). Dissociation may manifest itself as state symptoms, but also as trait symptoms. State dissociation is viewed as a transient symptom lasting only minutes, whereas trait dissociation is chronic and is viewed as part of one's personality (Bremner, 2008).

Dissociative symptoms are prevalent in both general and clinical populations (Foote, Smolin, Kaplan, Legatt, & Lipschitz, 2006). In their most radical versions, they characterize the dissociative disorders. However, dissociative symptoms are also common in other diagnostic groups, such as borderline personality disorder (BPD), posttraumatic-stress disorder (PTSD), depression, schizophrenia (Holmes et al., 2005; Yu et al., 2010), and various anxiety disorders such as obsessive-compulsive disorder (Rufer et al., 2006; Watson, Wu, & Cutshall, 2004), panic disorder, and agoraphobia (Cassano et al., 1989).

The etiology of dissociation has been studied for decades by a number of researchers, and has been the subject of intense debate (Bremner, 2010; Giesbrecht et al., 2010). According to the posttraumatic model of dissociation, dissociation originates from the exposure to traumatic experiences. In this view, dissociative symptoms serve a defensive function in that they help traumatized individuals to avoid the memory of aversive events (Spiegel et al., 2011). This coping strategy is hypothesized to become automatized and habitual, such that even minor stressors can provoke dissociative symptoms.

An alternative perspective on the origins of dissociation focuses on the link between sleep and dissociation. The dreamlike character of dissociative symptoms gave rise to the idea that depersonalization and derealization symptoms are linked to sleep. Watson (2001, 2003) provided the first evidence for a relationship between unusual sleep experiences as measured by the *Iowa Sleep Experiences Survey* (ISES; Watson 2001, 2003; Giesbrecht & Merckelbach, 2006) and dissociative symptoms, as indexed by the *Dissociative Experiences Scale* (DES; Bernstein & Putnam 1986). Based on this finding, Watson (2001, 2003) proposed that disruptions in the sleep-wake cycle may intensify dissociative symptoms. Replicating Watson's original observation, a number of studies have found a robust correlation between sleep disturbances and dissociation (r = .31-.55), even when this association was tested with other instruments than the ISES and the DES (van der Kloet et al., 2012; see chapter I).

Adding to these correlational findings, an experimental study showed that dissociative symptoms of undergraduates intensify when their sleep-wake cycle is disrupted (Giesbrecht et al., 2007). Giesbrecht and colleagues found elevated levels of spontaneous dissociative symptoms as well as symptoms induced by dot-staring, after 36 hours of sleep loss. This effect was not mediated by a deterioration of mood, as the participants experienced an increase in sleepiness and dissociative symptoms first, which was then followed by mood deterioration (Giesbrecht et al., 2007). The researchers also noted that the occurrence of dissociative symptoms followed the oscillating pattern of sleep. Thus, dissociative symptoms remained stable during the day and only increased in the evening and night. However, an important limitation of this study was the absence of a control group.
Only recently, scientists have started to investigate the merits of the sleep-dissociation approach in clinical populations. Longitudinal research by van der Kloet, Giesbrecht, and Merckelbach (2011) showed that sleepiness increased dissociative symptoms in a natural environment (i.e., outside the laboratory) as well, an effect that was again not mediated by mood deterioration.

Results collected in inpatients suffering from depression, anxiety, and addiction, showed that normalization of the sleep-wake cycle reduced their dissociative symptoms within 6 weeks (van der Kloet et al., 2012). However, many questions remain regarding the specific link between sleep disturbances, dissociation, and their cognitive concomitants.

The marked influence of sleep disruption on performance and alertness has been shown by numerous studies (Jewett et al., 1999; Williamson et al., 2001). Reduced activity of the frontal lobes and their executive functions due to sleep disruption (Harrison, Horne, & Rothwell, 1997) might explain these effects. Another well-documented consequence of sleep-wake disruptions is their detrimental effect on memory (Hairston & Knight, 2004). Thus, disturbances in the sleep-wake cycle may undermine memory and attention, promoting absentmindedness and a propensity to produce memory commissions, two well-established correlates of people scoring high on dissociation measures, i.e., high dissociators (Giesbrecht et al., 2004; Giesbrecht et al., 2010). An unstable sleep-wake cycle may also promote dream mentation to intrude into waking consciousness, leading to fantasy proneness and feelings of depersonalization and derealization (van der Kloet et al., 2012; see chapter I). In short, the cognitive side-effects of sleep disruptions – attentional lapses, memory commissions, loss of executive control, intrusions of dream mentation - may account for dissociative symptoms and their heterogenous manifestations. So far, however, most studies documenting the relationship between sleep and dissociation rest on correlational data. One inherent limitation of this type of study is that it does not allow the deduction of causal relations between various variables.

Building on earlier findings from Giesbrecht and colleagues (2007), the goal of the present study was to test whether 36 hours of sleep deprivation increases dissociative symptoms, along with memory commission errors, lack of executive control, and transient attentional deficits. We hypothesized that the largest cognitive effects of sleep deprivation would be found in people with increased levels of trait dissociation.

Method

Participants

Participants were 56 healthy undergraduate students (43 women) enrolled at Maastricht University. Their mean age was 20.7 years (*SD* = 2.33, *range* = 18 to 29 years). They received written and oral information about the study, after which they gave written informed consent. This information entailed what they could expect during the night and the restrictions during the experiment (e.g., no smoking, no caffeine or alcohol holding drinks, no chocolate). All participants completed an online baseline screening. Exclusion criteria for both experimental and control group entailed any kind of sleep medication, substance misuse or dependence, nicotine dependence, serious mental disease, or an endocrinological disorder. A good understanding of the Dutch language was necessary for inclusion. After inclusion, participants were subjected to a balanced randomization procedure by order of inclusion to determine their place in either the experimental (*N* = 28, 20 women) or control group (*N* = 28, 23 women). All participants in the experimental group were brought home safely by taxi.

After completion of the experiment, participants received a monetary reward. This study was conducted according to the Medical Research Involving Humans Act (WMO) and the principles of the World Medical Association (WMA) Declaration of Helsinki, 2008, and was approved by the Medical Ethics committee of the Academic Hospital of Maastricht and Maastricht University.

Procedure

The night before the start of the study, participants had slept *M* = 7.96 hours (*SD* = 1.08) at home. Participants woke up at *M* = 8.56 a.m. (*SD* = 1.21). Participants arrived in the lab at 5 p.m. on day 1 and completed a number of tasks and questionnaires to assess sleepiness, mood, and dissociative symptoms at baseline. Next, the experimental group completed the same questionnaires at 8 p.m., 11 p.m., 3 a.m., 9 a.m., and 1 p.m. The control group completed the same questionnaires at 8 p.m., and the next day at 9a.m., and 1 p.m. Sleep deprived

participants stayed until 3 p.m. the next day and were not allowed to sleep. Control participants returned the next morning at 9 a.m. to the laboratory after a regular night of sleep at home (*M* = 7.99 hours, *SD* = 1.05).

Measures

Baseline screening

Dissociative Experiences Scale (DES; Cronbach's α = .89; Bernstein & Putnam, 1986). The DES is a self-report scale of trait dissociation. It requires participants to indicate on 100 mm visual analogue scales (anchors: 0 = never; 100 = always) to what extent they experience 28 dissociative experiences in daily life. Examples of such experiences include feelings of depersonalization, derealization, and psychogenic amnesia. Van IJzendoorn and Schuengel (1996) provide meta-analytic evidence for the sound psychometric properties of the DES.

Cambridge Depersonalisation Scale (CDS; Cronbach's α = .92). The CDS (Sierra & Berrios, 2000) consists of 29 items that ask the respondent to rate depersonalization symptoms over the past six months. The frequency aspect is evaluated on a 0-4 scale (anchors: 0 = never; 4 = all the time) and the duration aspect is rated on 1-6 scale (anchors: 1 = few seconds; 6 = more than a week). Hence, for each individual symptom, scores range from 0-10. Scores are summed to obtain a total CDS score (range: 0-290).

Iowa Sleep Experiences Survey (ISES; Cronbach's α = .83). The ISES (Watson, 2001) consists of 18 questions asking the respondent to rate the frequency of various sleep- and dream-related experiences on a 7 point-Likert scale (anchors: 1 = never, 7 = several times a week). The ISES consists of 2 separate subscales that measure general sleep experiences (e.g., "I have recurring dreams.") and lucid dreaming (e.g., "I am aware that I am dreaming, even as I dream"), respectively.

SLEEP-50 (Cronbach's α = .88; Spoormaker et al., 2005). Sleep experiences were assessed with the 50-item Dutch version of the SLEEP-50, which contains nine subscales that index sleep complaints and sleep disorders listed in DSM-IV (American Psychiatric Association, 2001). Each item is scored on a 4-point Likert scale ranging from 0 (not at all) to 3 (very much). The SLEEP-50 has high internal consistency (Cronbach's α = 0.84) and adequate test-retest reliability (r =0.78; Spoormaker et al., 2005). Analyses were based on the total score.

State measures

Clinician-Administered Dissociative States Scale (CADSS, Cronbach's α = .77-.89). The CADSS (Bremner et al., 1998) is a 27-item scale with 19 subject-rated items, and 8 items scored by an observer. It is a reliable and valid instrument for the measurement of present-state dissociative symptoms (Bremner et al., 1998).

Items are scored on a 5-point scale (0 = not at all, 4 = extremely). For the purpose of this study, only the self-report items were administered. A sample item is: “does it seems to you that things move in slow motion?”

Stanford Sleepiness Scale (SSS). The SSS (Hoddes et al., 1973) is a measure of subjective sleepiness. It consists of a single item that is rated on a 7-point scale with response options ranging from ‘feeling active, vital, alert and aweke’ to ‘I almost fall asleep, I struggle to remain awake’. The SSS is widely used in sleep deprivation research (e.g., Babkoff, Caspy, & Mikulincer, 1991).

Profile of Mood States (POMS, Cronbach’s α = .61-.94). Mood was assessed by the Profile of Mood States - Short Form (POMS; McNair, Lorr, & Droppleman, 1992). The POMS is a self-report measure that is commonly used as a measure of typical and persistent mood reactions to current life situations. Participants indicate to what extent they agree with adjectives (e.g., annoyed, nervous, angry) describing their current mood or feelings on 5-point scales (anchors: 0 = not at all, 4 = extremely). The POMS has excellent psychometric properties (e.g., Lezak, Howieson, & Loring, 2004). The present study employed a Dutch version of the POMS that has been proven to be both valid and reliable (de Groot, 1991).

Memory measures

To measure commission errors (i.e., pseudo-memories), the participants were asked to watch three movie clips. The stimulus material consisted of a fearful clip from the movie “The silence of the lambs” (length 12.30 minutes), a neutral clip (i.e., a fragment from a Discovery Channel documentary), and a positive clip from the movie “When Harry met Sally” of similar durations. These three clips have been shown to elicit the emotional responses that we wanted to study, namely negative, neutral, and positive, respectively (Rottenberg, Ray, & Gross, 2007). After watching each movie clip, the *Self-Assessment Manikin* (SAM; Bradley & Lang, 1994) was administered as a manipulation check on the emotional impact of the video fragment. SAM is “a non-verbal pictorial assessment technique that directly measures the pleasure, arousal, and dominance associated with a person's affective reaction to a wide variety of stimuli” (Bradley & Lang, 1994, p. 49). The following three measures were obtained, from 35 to 60 minutes after stimulus offset, at 8 p.m. and once again after sleep deprivation, at 1 p.m. the following day:

Free recall of the videoclip. Participants were asked to write down everything they could remember about the clip. Their accounts were scored in terms of hits and commission errors by two independent raters who were blind to the experimental or control condition of the participants. Hits and commission were averaged across raters. We hypothesized that most commission errors would be made by people scoring high on dissociation after sleep deprivation (see also Candel et al., 2003).

Subjective memory fragmentation (Kindt & Van den Hout, 2003). This was measured using three 100-mm visual analogue scale (VAS) items. Participants had to indicate to what extent they had 'snap-shot' like recollections of the video clip. The items were as follows: "How much does your memory of the video exist of fragmented pieces as opposed to a whole entity?", "How much does your memory of the video exist of visual images?", and "How emotionally intense are your memories of the video?" Items were summed up to obtain an index of subjective memory fragmentation. We expected people scoring high on dissociation to report a more fragmented memory than people scoring low on dissociation (Bedard-Gilligan & Zoellner, 2012). Furthermore, we expected these results to be most pronounced in the sleep deprivation group.

Objective memory fragmentation. Objective memory fragmentation was measured along the lines of Wegner, Quillian, and Houston (1996). These authors were interested in the effect of thought suppression on memories. To this end, they developed a method which allowed them to investigate disruptions in the temporal organization of memories for a movie clip. More specifically, the Objective Memory Fragmentation Task requires participants to sort 5 different scenes of 4 fragments, each lasting 5 seconds, into the correct order (Kindt & Van den Hout, 2003). Scores for the 5 fragments were summed up to obtain a measure of objective memory fragmentation. We expected high dissociators to display a more fragmented memory than low dissociators, especially after sleep deprivation (see also Giesbrecht et al., 2010a).

Attentional functioning measure

Attention Network Test (ANT; Fan, McCandliss, Somner, Raz, & Posner et al., 2002). The ANT had been devised to measure three different facets of attention within a single task: orienting, alerting component, and executive control. To this end, it combines elements of Posner's (1980) cueing task with elements of the Eriksen flanker task (Eriksen & Eriksen, 1974). The ANT employed in the current study consisted of 3 blocks of 96 trials during each of which participants had to indicate the direction of an arrow that was surrounded by other arrows. Trials were presented in a random order and each block lasted about 5 min. Thus, in total, the ANT lasted about 15 min. We expected that sleep loss would undermine attentional performance, particularly the executive (i.e., frontal) aspect of it (see Harrison et al., 1997).

Data analysis

Statistical analyses were performed using SPSS 18.0 software. Cronbach's α values were calculated to estimate internal consistency of the baseline and state measures. Pearson product-moment correlations between baseline and state

measures were calculated. State data were analyzed using the General Linear Model (GLM). Significant multivariate results were evaluated with univariate tests. Huynh-Feldt or Greenhouse-Geisser corrected *P* values, their corresponding epsilons as well as the original, i.e. uncorrected, degrees of freedom are reported when the sphericity assumption was not met.

Results

Table 5.1 displays mean scores of baseline and state measures and Pearson product-moment correlations between these variables for the full sample. Mean scores were as expected for this population (IJzendoorn & Schuengel, 1996; Giesbrecht & Merckelbach 2004; 2006). We computed changes scores for the state measures, by subtracting each state measure from its previous time-point measure. Then, we correlated baseline measures with the change scores of state measures during the follow-up testing sessions of the experiment. Table 5.1 illustrates three interesting aspects of our data. First, dissociation measures (DES, CDS) were correlated positively with aberrant sleep experiences (ISES) and with sleep disturbances (SL-50). Second, overall, correlations between baseline dissociation measures and (increases in) state dissociation, sleepiness, and mood during the follow-up testing sessions remained non-significant. Third, the exception to this pattern was the CDS that was significantly related to state dissociation (CADSS) between 5 and 8 p.m., $r = -.28$, $p < .05$, the negative correlation meaning that heightened CDS scores were related to decreases in CADSS. Furthermore, a higher score on the Sleep-50 was related to a mood deterioration in the experimental group between 3 and 9 a.m., $r = .50$, $p < .01$. As well, various state measures were related to each other at different time points in the expected way (e.g., mood with state dissociation between 3 and 9 a.m., and sleepiness with state dissociation between 11 and 3 a.m.; see Table 5.1).

Table 5.1
Mean scores and standard deviations of baseline measures and Pearson product-moment correlations between measures in undergraduate sample (*N* = 56).

	Mean (SD)	DES	CDS	ISES	SL 50	CADSS 5-8	CADSS 8-11	CADSS 11-3	CADSS 3-9	POMS 3-9	SSS 11-3
DES	15.08 (9.63)	-	-	-	-	-	-	-	-	-	-
CDS	50.05 (16.65)	**.64****	-	-	-	-	-	-	-	-	-
ISES	51.09 (13.84)	**.55****	**.45****	-	-	-	-	-	-	-	-
SL 50	87.21 (15.70)	**.45****	**.39****	**.62****	-	-	-	-	-	-	-
CADSS 5-8	.00 (2.07)	-.21	**-.28***	-.20	-.10	-	-	-	-	-	-
CADSS 8-11	.11 (2.42)	.06	.12	.27	.34	**-.82****	-	-	-	-	-
CADSS 11-3	1.82 (1.93)	.01	.08	-.21	.01	-.20	.04	-	-	-	-
CADSS 3-9	5.07 (5.95)	.05	.01	.06	.18	.01	.15	.30	-	-	-
POMS 3-9	4.86 (9.51)	.24	.03	.34	**.50****	-.04	.25	-.14	**.54****	-	-
SSS 11-3	.79 (1.17)	.06	.01	-.11	-.03	-.01	-.10	**.58****	.24	-.21	-
SSS 3-9	1.07 (.98)	.13	.12	.08	.22	-.11	.18	-.25	.11	**.51****	**-.50****

Note. DES= Dissociative Experiences Scale; CDS = Cambridge Depersonalization Scale; ISES = Iowa Sleep Experiences Survey; SL 50 = The Sleep-50; CADSS 5-8 = Clinician-Administered Dissociative States Scale, change score 5 p.m. to 8 p.m.; CADSS 8-11 = Clinician-Administered Dissociative States Scale, change score 8 p.m. to 11 p.m.; CADSS 11-3 = Clinician-Administered Dissociative States Scale, change score 11 p.m. to 3 a.m.; CADSS 3-9 = Clinician-Administered Dissociative States Scale, change score 3 a.m. to 9 a.m.; POMS 3-9 = Profile of Mood State, change score 3 a.m. to 9 a.m.; SSS 11-3 = Stanford Sleepiness Scale, change score 11 p.m. to 3 a.m.; SSS 3-9 = Stanford Sleepiness Scale, change score 3 a.m. to 9 a.m.

* $p < .05$

** $p < .01$

Experimental group vs. control group

First, we investigated whether there was a significant difference between the two groups with regard to sleepiness over time. Using repeated measures with SSS as dependent variable, time as within-subjects factor, and group as between-subjects factor, we found a significant interaction effect between time and group, $F(3,53)=39.21$, $p < .001$, partial eta² = .43, due the higher levels of SSS in the experimental group between at 9 a.m. and 1 p.m. (see Figure 5.1). We also found a significant main effect of time, $F(1,54)=13.28$, $p < .001$, partial eta² = .20. The repeated measures analyses revealed a main effect of group due to higher levels of SSS in the experimental group than in the control group, $F(1,53)= 23.17$, $p < .001$, partial eta² = .30.

Second, we looked at CADSS as an index of state dissociation over time. Using repeated measures with CADSS as dependent variable, and time and group as independent variables, we found a significant interaction effect between time and group, $F(3,54)=23.94$, $p < .001$, partial eta² = .31, due to higher CADSS levels in the experimental group than in the control group between 9 a.m. and 1. p.m. (Figure 5.1) We also found a significant main effect of time, $F(3,54)= 18.66$, $p < .001$, partial eta² = .26. Furthermore, the analyses revealed a significant difference between the experimental group and the control group $F(1,54)= 14.83$, $p < .001$, partial eta² = .22.

Finally, we performed similar analyses on the POMS as index of mood deterioration over time. Again, we found a significant interaction effect between time and group, $F(3,54)=19.83$, $p < .001$, partial eta² = .27, with the experimental group displaying stronger mood deterioration than the control group between 9 a.m. and 1. p.m. (Figure 5.1). Furthermore, the ANOVA yielded a significant main effect of time, $F(3,54)=3.51$, $p = .02$, partial eta² = .06, but no significant effect of group, $F(3,54)=2.66$, $p = .11$, partial eta² = .05.

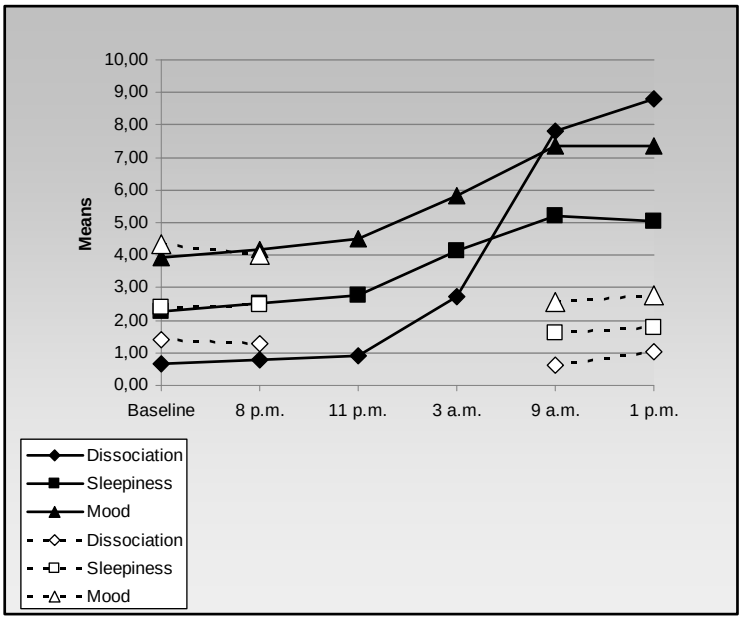


Figure 5.1.

Pattern of dissociation, sleepiness, and mood of the experimental group (*N* = 28) and the control group (*N* = 28; dotted lines) during the test period of the experiment.

During the night, there is an increase of sleepiness and dissociative symptoms, and a deterioration of mood. The largest increase of symptoms takes place between 3 a.m. and 9 a.m.

Time course of acute dissociation, sleepiness, and mood deterioration during sleep loss

Next, we focused on the experimental group. Using repeated measures analyses, we found a significant increase in sleepiness over test sessions, $F(5,26) = 70.51$, $p < .001$, partial eta² = .53. Significant linear main effects of time were also evident for dissociation and mood measures, CADSS: $F(5,27) = 28.73$, $p < .001$, partial eta² = .52, and POMS: $F(5,27) = 16.06$, $p < .001$, partial eta² = .37, respectively.

Because we were interested in the predictive power of mood and sleepiness on dissociation, we divided the subscales of the POMS into a Fatigue-Inertia subscale, and a General Distress subscale in order to avoid an artificial correlation between mood and sleepiness due to the fatigue subscale. We then computed change scores for all variables. Using multiple hierarchical regression analyses, we entered CADDS change score at time 4 (i.e., the increase in dissociation between 3 a.m. and 9 a.m.; see Figure 5.1) as dependent variable and the General Distress and Fatigue-Inertia subscales of the POMS change scores and SSS change score all at times 3 and 4 as independent variables (i.e., increases in sleepiness and mood deterioration between times 11 p.m. and 3 a.m., and 3 a.m. and 9 a.m., respectively). The measures at time 4 were chosen because dissociation levels increased strongest at time 4 (between 3 and 9 a.m.). Table 5.2 gives a summary of the hierarchical regression. As can be seen, the increase in sleepiness between 11 and 3 a.m. as well as increased levels of general distress between 3 a.m. and 9 a.m. accounted for 42% of the variance in CADSS scores.

Table 5.2.
Summary of multiple hierarchical regression analyses with dissociation (CADSS change score at time 4) as dependent variable (N = 28), all change scores.

Model	*B*	*ß*	*t*	*p*	*r*	r^2
1					.65	.42
SSS 11-3	1.82	.37	1.10	.29		
SSS 3-9	.30	.05	.17	.86		
POMS 11-3	-.10	-.04	-.11	.92		
POMS 3-9	2.33	.66	2.98	.01		
POMS Sleep 11-3	.03	.03	.08	.94		
POMS Sleep 3-9	-.16	-.13	-.44	.67		
2					.65	.42
SSS 11-3	1.87	.38	1.24	.23		
SSS 3-9	.31	.05	.19	.85		
POMS 11-3	-.07	-.03	-.08	.93		
POMS 3-9	2.34	.67	3.08	.01		
POMS Sleep 3-9	-.16	-.14	-.50	.62		
3					.65	.42
SSS 11-3	1.77	.36	1.94	.07		
SSS 3-9	.29	.05	.18	.86		
POMS 3-9	2.36	.67	3.32	<.001		
POMS Sleep 3-9	-.16	-.13	-.50	.62		
4					.65	.42
SSS 11-3	1.70	.34	2.09	.05		
POMS 3-9	2.37	.67	3.42	<.001		
POMS Sleep 3-9	-.12	-.10	-.51	.62		
5					.65	.42
SSS 11-3	1.80	.36	2.32	.03		
POMS 3-9	2.16	.61	3.93	<.001		

Note. SSS 11-3 = Stanford Sleepiness Scale change score, increase in sleepiness between 11 p.m. and 3 a.m.; SSS 3-9 = Stanford Sleepiness Scale change score, increase in sleepiness between 3 a.m. and 9 a.m.; POMS 11-3 = Profile of Mood States all subscales except for subscale Fatigue-Inertia change score, decrease in mood between 11 p.m. and 3 a.m.; POMS 3-9 = Profile of Mood States all subscales except for subscale Fatigue-Inertia change score, decrease in mood between 11 p.m. and 3 a.m.; POMS Sleep 11-3 = Profile of Mood States subscale Fatigue; change score, increase in sleepiness between 11 p.m. and 3 a.m.; POMS Sleep 3-9 = Profile of Mood States subscale Fatigue; change score, increase in sleepiness between 3 a.m. and 9 a.m.

Memory functioning after sleep loss

Before and after sleep deprivation, participants watched three movie clips of positive, neutral, and negative emotional content. *Self-Assessment Manikin* (SAM; Bradley & Lang, 1994) analyses revealed that for all movie clips the intended emotions were successful induced (all *p*'s < .001). Two independent raters analyzed the free recall reports of participants. Moderate to strong significant correlations were obtained between the two raters for all measures (r = .57 - .91, *p's* = <.001), and ratings were therefore averaged across the raters.

Mean scores and standard deviations are reported in table 5.3. Repeated measures ANOVA's revealed a significant time x group interaction for the correct free recall of the positive movie clip, $F(1,53) = 10.41$, p = .02, and a main effect of time, $F(1.53) = 7.29$, p = <.01, meaning that after sleep deprivation correct free recall scores were lower than before sleep deprivation. However, there was no significant difference between the experimental group and control group in the level of correct free recall of the positive movie clip, $F(1,53) = .60$, p = .44. For the neutral movie clip, no differences between the groups or within the groups emerged, meaning that they did not differ in levels of neutral free recall before and after sleep deprivation, nor was there a difference between the two groups. For the negative movie clip, a main significant effect of time was found, $F(1,54) = 9.12$, p <.01, but not for group, p > .57. Levels of correct recall for the negative clip were lower after than before sleep deprivation.However, there was no difference in levels of free recall between the two groups.

With regard to commission errors, for none of the three movie clips, differences were found when comparing levels before and after sleep deprivation in the experimental group, as well as when comparing the groups (*p's* = .10 - .80). We analyzed subjective and objective memory fragmentation, using repeated measures analyses. The experimental and control group did not differ with regard to objective memory or subjective memory fragmentation. This lack of effects was evident for analyses involving the two groups and analyses within groups for differences between baseline and follow-up (all *p*'s > .13).
Finally, we performed a median split on the trait dissociation levels (i.e., DES scores) within the experimental group. Using a 2 (high versus low DES) x time repeated measures ANOVA, we investigated whether memory effects were specifically pronounced in participants with relatively high or relatively low trait dissociation levels (DES). This was not the case (all *p*'s > .36).

Executive functioning after sleep loss

Mean scores and standard deviations are reported in table 5.3. First, we compared the experimental group to the control group that completed the ANT twice at times

8.30 p.m. and 11.00 a.m. We performed repeated measures analyses for the three components of the ANT: alerting, orienting, and executive control. We found a significant main effect of time for the alerting component, indicating that there was a difference in scores between the measure at 8.30 p.m. and the measure at 11 a.m., $F(1,52) = 13.84$, $p < .01$, with lower alerting scores at the second time point. There was no significant difference in scores between the experimental and control group, $p = .57$. No differences were found for the orienting component of the ANT, all p's > .08. We found a significant time x group interaction for the executive control component of the ANT, $F(1,52) = 12.58$, $p < .01$, and a main effect of group, $F(1,52) = 7.19$, $p < .01$. Thus, in the control group, reaction times decreased and participants became faster in completing the task compared to baseline, whereas in the experimental group, reaction times increased and task completion took longer.

Second, we analyzed the data from the experimental group that completed the task three times: at 8.30 p.m., 5.00 a.m., and 11.00 a.m. Using repeated measures analyses, we found a main effect of time for the alerting component and executive control ($F(2,54) = 3.57$, $p = .04$; $F(2,54) = 3.78$, $p = .03$, respectively), but not for the orienting component of the ANT ($F(2,54) = 1.09$, $p = .34$). This means that after sleep loss, reaction times were delayed compared to baseline indicating that alertness and vigilance became slower in participants, and executive control of attention deteriorated.

Finally, we explored whether these attentional effects of sleep loss were specifically pronounced in participants with high trait dissociation (i.e., high DES). Using a median split, we carried out a 2 (high versus low DES) x time repeated measures ANOVA on the three ANT parameters. Figure 5.2 shows the time course of these parameters in high and low DES individuals. As can be seen and as was confirmed by the ANOVA, the high DES group tended to deteriorate in executive control of attention more than the low DES group, although this effect only reached borderline significance, $F(1,26) = 3.90$, $p = .06$.

Table 5.3

Mean scores and standard deviations of memory and ANT measures (N = 56).

Measure	Mean (SD) Rater 1	Mean (SD) Rater 2
FR Positive	18.98 (12.04)	15.86 (8.69)
FR Neutral	12.16 (4.36)	10.84 (4.27)
FR Negative	22.77 (11.67)	18.48 (6.98)
Com Positive	.76 (.74)	.64 (.86)
Com Neutral	.82 (.97)	.93 (.91)
Com Negative	1.38 (1.12)	1.21 (.89)

ANT	8.30 a.m. (*N* = 56)		5.00 a.m. (*N* = 28)	11.00 a.m. (*N* = 56)	
	EXP	C	EXP	EXP	C
ANT alerting	44.16 (22.11)	46.91 (18.50)	63.31 (23.08)	56.41 (32.56)	60.61 (24.10)
ANT orienting	39.63 (19.54)	40.85 (22.01)	44.08 (26.06)	50.62 (32.45)	38.55 (25.87)
ANT control	90.45 (25.24)	80.87 (24.23)	89.48 (25.95)	97.99 (43.28)	76.04 (30.28)

Note. FR = Total free recall score of positive, neutral, or negative video clip; Com = total number of commission errors of positive, neutral, or negative video clip; ANT alerting = Attention Network Task, alerting component; ANT orienting = Attention Network Task, orienting component; ANT control = Attention Network Task, executive control component; EXP = experimental group; C = control group.

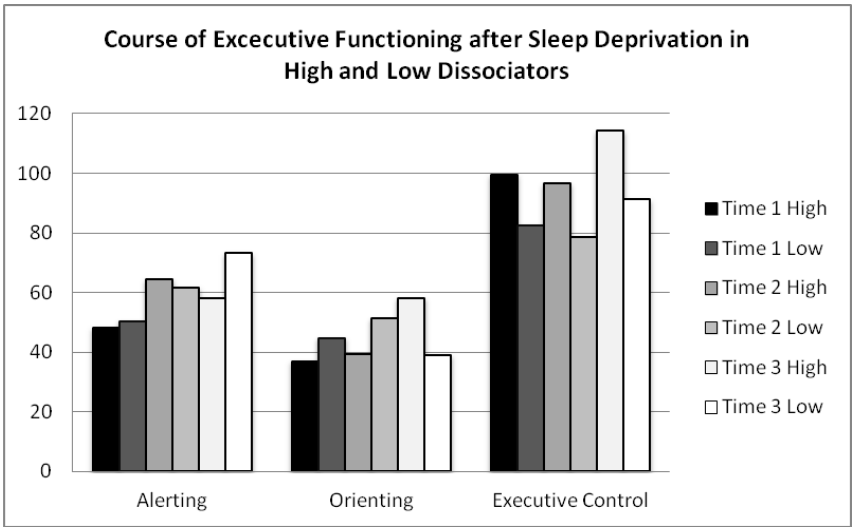


Figure 5.2

Course of executive functioning (as measured by reaction time) after sleep deprivation in the experimental group (n = 28). We performed a median split based on high and low dissociation (DES) participants. The bars depict the three parameters of the ANT subdivided for high and low dissociators, and displayed for all three measure points. The first measure (Time 1) took place at 8.30 p.m., the second measure (Time 2) at 5 a.m., and the final measure (Time 3) at 11 a.m.

Discussion

With the present study, we built on previous findings of Giesbrecht and colleagues (2007), who found suggestive evidence that sleep loss intensified dissociative symptoms, but did not use a control group to correct for repeated testing effects. Thus, the main aim of our study was to investigate whether a 36-hours sleep deprivation would enhance dissociative symptoms, sleepiness, and mood deterioration. Specifically, we hypothesized that sleep deprivation would increase dissociative symptoms, that increases in sleepiness would predict the increase in dissociation, and that this effect would not be mediated by a deterioration of mood.

The most important findings can be catalogued as follows. First, with regard to our baseline and trait measures, we replicated the robust link between dissociative symptoms and unusual sleep experiences that has now been documented in many studies (see e.g., Watson 2001, 2003; Levin & Fireman, 2002; Agargun et al., 2003; 2004; Giesbrecht & Merckelbach, 2004; 2006a; 2006b; Giesbrecht et al., 2006; 2007; van der Kloet et al., 2010; see chapter I). Second, in accordance with Giesbrecht and colleagues (2007), we found that sleep deprivation had significant effects on dissociation, sleepiness, and mood. That is, sleepiness and dissociation increased during the night, while mood deteriorated.

Third, in contrast to our expectations and unlike the study of Giesbrecht and colleagues (2007), we found that changes in mood paralleled changes in dissociative symptoms. Our regression analyses showed that dissociative symptoms were also mediated by a deterioration of mood during 36 hours of sleep deprivation. There might be practical reasons as to why this pattern was not observed by Giesbrecht and colleagues (2007), such as differences in the administration of measures as well as different points of measurement during the night.

Fourth, we found that dissociative symptoms, sleepiness, and deterioration of mood all followed a similar oscillating pattern. That is, they were relatively stable during the day and linearly increased during the night until a plateau was reached. This is in line with the findings of, for example, Babkoff, Caspy, Mikulinger, and Sing (1991) who argued that sleepiness follows diurnal oscillations. However, when we investigated these patterns in more detail during the night, we also found that mood, as indexed by the POMS General Distress subscale, exhibited a slightly different temporal pattern than dissociative symptoms and sleepiness. During the night, significant mood deterioration occurred later in time than the increase in sleepiness and dissociative symptoms. Thus, in our sample, feelings of sleepiness seemed to precede an increase of dissociative symptoms and deterioration of mood.

Fifth, we found that after sleep loss, participants had poorer free recall of positive and negative stimulus material, but not of neutral stimulus material compared to the control group. This accords well with the findings of Walker (2009), who extensively described the role of sleep in cognitive processing of emotional material. However, we did not find an increase in commission errors in the sleep deprived participants, although their state dissociative levels went up. So, in a way, this pattern – stable commission errors and increasing dissociation – contradicts earlier studies that reported that dissociative tendencies are associated with commission errors (Candel et al., 2003; Giesbrecht et al., 2007; Merckelbach et al., 2007; Bremner, Shobe, & Kihlstrom, 2000). Neither did we find that sleep deprived participants in whom state dissociation went up reported more memory fragmentation as compared to controls, a null finding that is in line with the critical review on dissociative encoding of Bedard-Gilligan and Zoellner (2012).

Sixth, we found that sleep loss reduced attention and undermined executive control functioning. The latter effect was particularly pronounced in highly dissociative individuals. In general, our findings in this regard are consistent with studies that noted that sleep deprivation deregulates the frontal areas, thereby undermining cognitive efficiency (Horne, 1993; Harrison et al., 1997) as well as studies that observed executive problems in high dissociative individuals (Cima et al., 2001; Amrhein, Hengmith, Maragkos, & Hennig-Fast, 2008).

At a more general level, the findings of the current study provide support for a view on dissociative symptomatology that stresses the importance of sleep deficiencies (e.g., van der Kloet et al., 2012) and their concomitants. By this view, sleep disruptions fuel distress, but also degrades memory and attentional control. Thus, sleep disruptions undermine cognitive efficiency and it is against this background that dissociative symptoms arise. This may also at least partially account for the cognitive deficiencies in highly dissociative individuals (Amrhein et al., 2008). A labile sleep-wake cycle may stem in part from a genetic propensity (i.e., Lang, Paris, Zweig-Frank, & Livesley, 1998), distressing trauma-related memories, or other unknown causal influences. Consequently, the sleep-dissociation model may explain how traumatic experiences disrupt sleep and increase vulnerability for dissociative symptoms. Moreover, it may explain why unusual sleep experiences, dissociation, and cognitive failures overlap.

Some limitations of our study merit mention. Although we employed an experimental design, our findings may have been influenced by as yet unspecified confounding variables (e.g. willingness to participate in a sleep deprivation experiment). Accordingly, the connections between sleepiness, deteriorated mood, and dissociation should be interpreted with caution. Germane to this is that the reliability (Cronbach's alpha) of the POMS General Distress was medium to low, the inter-rater reliability of the memory measures proved only moderate in some cases, and working with change scores is far from ideal. It is important that future

studies make a clear distinction between mood and sleepiness. Replication of this study is important to investigate the antecedent role of sleepiness, but also mood in fluctuations of dissociative symptoms.

Second, the choice of the precise sleep deprivation manipulation does matter. The literature recommends using 24 hours or a multiple of 24 hours of sleep loss in order to distinguish clearly between sleep loss effects, diurnal effects, and interaction effects (Babkoff et al., 1991). Future studies on sleep and dissociation should therefore use longer periods of sleep deprivation, with equal amounts of peaks and troughs, for a more balanced comparison between the days.
Finally, undergraduate students form a quite homogenous group, and generalization of the results is therefore limited. Replication of this study is warranted in larger and clinically more relevant groups. It would also be interesting to investigate more thoroughly the relation between the sleep-wake cycle and dissociative symptoms, in reversed order. That is, it would be interesting to investigate whether dissociative symptoms decrease after a good night sleep. The first steps have been taken to investigate this effect, with positive results showing that sleep normalization leads to a significant decrease in dissociative symptoms in psychiatric inpatients after 6 to 8 weeks (van der Kloet et al., 2012).

In conclusion, the role of the sleep-wake cycle in fluctuations of dissociative symptoms, memory functions, and executive control remains complex. The boundaries between mood, dissociation, and sleepiness are far from clear. This study established that sleepiness due to sleep deprivation increases dissociative symptoms, an effect mediated by mood deterioration. More insight into the role of the sleep-wake cycle and its influence on dissociative symptoms could provide answers regarding the etiology of dissociative psychopathology, and may even create common ground for researchers investigating the posttraumatic model of dissociation, and researchers who stress the sleep-dissociation-pseudo memory links. Research shows that 70-91% of the people diagnosed with PTSD suffer from sleep disturbances. Moreover, up to 71 % of people suffering from PTSD, depending on the severity of the traumatic event, report nightmares (Maher, Rego, & Asnis, 2006). Thus, it is likely that trauma promotes sleep disturbances and that an increase in dissociative symptoms may arise from such disturbances. Future studies in this domain could inspire new perspectives on treatment methods for dissociative symptoms and disorders. A review from Lamarche (2007) showed that treatments for sleeping difficulties in people with PTSD significantly improved sleep disturbances, and also led to a significant decrease of other PTSD symptoms, including dissociative symptoms. A similar treatment approach might be promising for other forms of psychopathology that are accompanied by high levels of dissociation (e.g., Depression, Eating disorders, Anxiety Disorders and Schizophrenia; see also Koffel & Watson, 2009). Thus, it appears that some structure is to be found in the realm of sleep disturbances. Improving the balance

of the sleep-wake cycle may enlighten patients suffering by reducing one of their most aggravating symptoms.

CHAPTER VI[9]

Recreational doses of MDMA, cannabis, and cocaine produce dissociative symptoms

9 This chapter is an adapted version of the following article:
Van Heugten – Van der Kloet, D., Giesbrecht, T., van Wel, J., Bosker, W.M., Kuypers, K.P.C., Theunissen, E.L., Spronk, D., Verkes, R.J., Merckelbach, H., & Ramaekers, J.G. (Submitted). Recreational doses of MDMA, cannabis, and cocaine produce dissociative symptoms. *American Journal of Psychiatry, submitted*.

Summary

Dissociative symptoms have often found to be refractory to pharmacological interventions. The development of effective pharmacological treatment has been hampered by the fact that the neurocircuitry underlying dissociative symptoms is not well understood. By exploring the effects of three recreational drugs on dissociative symptoms, we aim to further build our understanding of the neuropharmacology of drug induced dissociative symptoms. We conducted two placebo controlled studies in which we employed the Clinician-Administered Dissociative States Scale (CADDS) to monitor dissociative symptoms during intoxication with 25, 50 and 100mg of 3,4-methylenedioxymethamphetamine (MDMA; study 1), cannabis (THC 300 µg/kg; study 2), and cocaine (HCl 300mg, study 2). In addition, trait symptoms of dissociation were measured at baseline using the Dissociative Experiences Scale (DES). Dissociative symptoms as indexed by the CADDS significantly increased under the influence of MDMA and cannabis. A similar finding was obtained for cocaine, but here dissociative effects were less pronounced. Our results suggest that increased serotonine and dopamine neurotransmitter activity may intensify dissociative symptoms. State dissociation levels found in healthy controls when under the influence of MDMA and cannabis were higher than those typically found in patients suffering from schizophrenia and comparable to levels found in posttraumatic stress disorder (PTSD). For cocaine, state dissociation levels were comparable to levels found in schizophrenia and PTSD. These findings illustrate how psychopharmacological research may inform theories about the antecedents of dissociative symptoms.

Introduction

Dissociative symptoms form a heterogeneous class of experiences varying from absent-mindedness, excessive daydreaming, and memory problems to confusion about one's own identity. In their most radical form, such symptoms define conditions like dissociative amnesia and depersonalization disorder. Epidemiological studies among psychiatric inpatients and outpatients have yielded prevalence estimates of severe dissociative symptoms, with rates usually exceeding 10% (Foote et al., 2006), while a recent epidemiological study in the UK general population found a prevalence rate of 0.95% (Lee et al., in press).

Dissociative symptoms are not restricted to the dissociative disorders. Certain diagnostic groups, notably patients with borderline personality disorder, post-traumatic stress disorder (PTSD), obsessive compulsive disorder, and schizophrenia also display heightened levels of dissociative symptoms (Rufer et al., 2006; Merckelbach et al., 2006; Yu et al., 2010).

Dissociative symptoms have been show to be often refractory to pharmacological interventions (Lilienfeld, 2007) and the development of effective pharmacological treatment has been hampered by the fact that the neurocircuitry underlying dissociative symptoms is not well understood. However, initial research has implicated the serotonin, glutamate, and opiate systems (Stein & Simeon, 2009). For example, Hollander and colleagues (1990) observed in their study with 8 patients suffering from depersonalization disorder and co-morbid obsessive-compulsive disorder that the use of selective serotonin reuptake inhibitors (SSRI) may help to decrease depersonalization symptoms. The other way around, Rusconi and colleagues found that patients who suspended their SSRI medication increasingly began to experience depersonalization and derealization symptoms (Rusconi, Carlone, Muscillo, Coccanari de'Fornari, Podda, & Piccione, 2009).

There is little consensus in the literature on the origins of dissociative symptoms, with some authors stressing the antecedent role of traumatic experiences (Foote et al., 2006), while others have suggested a causal for aberrant sleep-wake patterns (Van der Kloet et al., 2012a). Examing the effects of different recreational drugs on dissociative symptoms might help us to elucidate the neurocircuitry underlying these symptoms, which ultimately may promote consensus in this research domain.

Specifically, when a variety of drugs with common mechanisms of action lead to an increase in dissociative symptoms, this may provide us with clues as to the underlying neurocircuitry involved in dissociation. An example is provided by Krystal and colleagues (1994) who administered subanaesthetic doses of ketamine to healthy participants and found that their participants reported experiences that closely resembled dissociative symptoms (Krystal, Karper, Seibyl, Freeman, Delaney, Bremner, et al., 1994; Pomarol-Clotet, Honey, Murray, Corlett, Absalom,

Lee, et al., 2006). Ketamine is a glutamate receptor antagonist, believed to promote the increase of glutamate release in response to NMDA receptor blockade, resulting in an excess of glutamate activity at non-NMDA glutamate receptors (Krystal et al., 1994). Using a facial recognition task, Abel and colleagues (2003) studied the effects of ketamine on the neural responses to emotional stimuli in healthy subjects. Ketamine is also known to produce emotional blunting in normal participants, which mimics the emotional blunting seen in patients with DPD (Abel, Allin, Kucharska-Pietura, David, Andrew, Williams, et al., 2003). Thus, an excess of glutamate activity may be accompanied by dissociative experiences, notably depersonalization/derealisation symptoms, and therefore, drugs inhibiting glutamate release might have positive outcome effects for patients suffering from these symptoms. In line with this hypothesis, Sierra and colleagues (2006) used lamotrigine as an add-on treatment in 32 patients with depersonalization disorder. They found that around 50% of the patients seemed to benefit from lamotrigine in combination with an antidepressant (Sierra, Baker, Medford, Lawrence, Patel, Phillips, et al., 2006).

3,4-methylenedioxymethamphetamine (MDMA) is the main psychoactive substance of the party drug ecstasy (or 'XTC'). Ecstasy is a popular stimulant drug: Lifetime prevalence of ecstasy use among the 15–34 age group ranges from 0.6% to 12.4%, with most estimates for European countries falling in the range of 2.1–5.8%. The lifetime prevalence estimate in the Netherlands is 11.6% in the age group of 15-34 years (Parrott, 2001). MDMA is an indirect mono-aminergic agonist. It stimulates the release and inhibits the re-uptake of serotonine, and to a lesser extent affects other neurotransmitters such as dopamine, noradrenaline, and acetylcholine (Parrott, 2001). Studies have established a connection between regular MDMA use and a broad range of psychopathological symptoms (e.g., anxiety, depression; (Parrott, Sisk, & Turner, 2000)). Thus, it would be informative to test whether MDMA would increase acute dissociative symptoms.

Over the last ten years, cocaine has established itself as the most commonly used illicit stimulant drug in Europe. Cocaine is a natural product extracted from the leaves of Erythroxylon coca Lam (coca leaves). Cocaine stimulates the release of dopamine. It has a similar psychomotor stimulant effect to that of amphetamine and related compounds and likewise produces euphoria, tachycardia, hypertension, and appetite suppression. It is estimated that about 15.5 million Europeans have used cocaine at least once in their life; on average, 4.6 % of adults aged 15–64 (European Monitoring Centre for Drugs and Drug Addiction (EMCDDA), 2012). Especially in higher doses, amphetamine has psychomimetic properties (Griffith, Cavanaugh, Held, & Oates, 1972) and given the overlap between schizophrenia and dissociative symptomatology (Yu et al., 2010), one would expect such doses to enhance state dissociation levels.

Cannabis (i.e., its main substance THC) is known for its acute transient psychotic effects, even in healthy controls. A recent review from Bugra and colleagues even concluded that cannabis might cause psychosis, particularly in individuals with a predisposition to schizophrenia (Bugra, Rapp, Studerus, Aston, Borgwardt, & Riecher-Rossler, 2012). Common primary psychoactive effects of THC include a state of relaxation, and to a lesser degree, euphoria. Secondary psychoactive effects, such as a facility for philosophical thinking, introspection and metacognition have been reported amongst cases of anxiety and paranoia (EMCDDA, 2012). Cannabis increases dopamine activity, especially in mediotemporal and anterocingulate areas of the brain, because these regions have a high density of cannabinoid receptors. It also undermines ventrostriatal activity. These areas are important in learning and memory and their sensitivity to THC might explain why cannabis users often experience memory problems. More importantly, the distorting effect of cannabis on these brain regions may form the substrate for cannabis users' increased risk at schizophrenia. Interestingly, the mediotemporal and anterocingulate cortex, and striatum have been critically implicated in the onset of schizophrenia (Bhattacharyya, Fusar-Poli, Borgwardt, Martin-Santos, Nosarti, O'Carroll, et al., 2009). Again, given the overlap between schizophrenia and dissociative symptoms (Yu et al., 2010; Spitzer, Haug, & Freyberger, 1997), one would expect cannabis, due to its dopamine enhancing effect, to raise dissociative levels.

With this in mind, we conducted two studies exploring the psychopharmacological effects of MDMA (Study 1), and cocaine and cannabis (Study 2) on dissociative symptom levels. We anticipated that MDMA would promote dissociative symptoms, due to its stimulating effect on serotonin, norepinephrine, and dopamine activity. Likewise, we expected an increase in dissociation while under the influence of cocaine and cannabis, because these drugs enhance dopamine through the blockage of the dopamine transporter protein and cannabinoid receptor activation, respectively.

Methods

Measures

Clinician-Administered Dissociative States Scale (CADSS; Cronbach's α = .82; Bremner et al., 1998). The CADSS is an instrument to measure *state* symptoms of dissociation. The scale consists of 19 self-report items and 8 observation items. The intensity of each dissociative symptom can vary from 0 (not present) to 4 (extremely present). Participants are asked to use the last three hours as a point of reference when completing the items. This study only made use of the self-report

items. An illustrative item is: "Do you feel as if you are watching the situation as an observer?" The CADSS is a reliable and valid instrument for the measurement of present-state dissociative symptoms (Bremner et al., 1998). CADSS scores were summed to obtain a total measure of state dissociation (range: 0-76).

Dissociative Experiences Scale (DES; Cronbach's α = .93; Bernstein & Putnam, 1986). The DES is a self-report scale that intends to measure trait dissociation. It requires participants to indicate on 100 mm visual analogue scales (anchors: 0 = never; 100 = always) to what extent they experience 28 dissociative experiences in daily life. Examples of such experiences include feelings of depersonalization and derealization, and memory difficulties (i.e., dissociative amnesia). In the current studies, we calculated total DES scores by summing across items (range: 0-100). Van IJzendoorn and Schuengel (1996) provide meta-analytic evidence for the sound psychometric properties of the DES.

Participants and procedure

Sixteen healthy participants (8 female; *mean age*: 22 years, *SD* = .41) took part in the study 1. Mean lifetime use of MDMA was 27.0 (*SD* = 8.4) times. In study 2, twenty-one healthy volunteers (5 female; *mean age*: 23 years, *SD*= 3.57; *range* 18 to 32 years; with a *mean* weight of 69.9 kilograms, *SD* = 9.95; *range* 53 to 89 kilograms) participated. Participants for both studies were recruited via advertisements at Maastricht University, the Netherlands. Data collection of study 1 was part of a larger sleep deprivation study. Participants were only included if they indicated to have experience with recreational drug use. They all underwent a medical examination before study inclusion. For details regarding the data collection in this study, the reader is referred to Bosker (2010). Study 2 was part of a larger trial on the association between drug use and impulse control (Van Wel, Kuypers, Theunissen, Toennes, Spronk, Verkes, et al., submitted). For details regarding the data collection in this trial, the reader may consult Van Wel (submitted). Both studies were conducted according to the code of ethics on human experimentation established by the Declaration of Helsinki (1964) and amended in Seoul (2008). Approval for the study was obtained from the Medical Ethics committee of the Academic Hospital of Maastricht and Maastricht University. A permit for obtaining, storing, and administering MDMA, THC, and cocaine was obtained from the Dutch drug enforcement administration.
In study 1, we compared three dosages of MDMA and a placebo to explore their effects on dissociative experiences. The study was conducted according to a double-blind, placebo-controlled, randomized, four-way, cross-over design.

Treatments consisted of single doses of placebo, 25, 50, and 100 mg MDMA. Treatment orders were balanced over participants and treatment periods. Placebo and MDMA were administered orally in identically appearing formulations. MDMA

was dissolved in 25 ml bitter orange peel syrup, and placebo consisted of only the bitter orange peel syrup. The syrup was mixed with 200 ml juice before it was given to the subjects. The wash-out period between treatments was at least 1 week. Participants were asked to refrain from any drugs 1 week before the medical examination until 2 weeks after study completion. Subjects were not allowed to drink alcohol and caffeine or smoke tobacco during a 24-h period prior to testing. Subjects were always tested for alcohol and drugs, i.e., tetrahydrocannabinol, opiates, amphetamine/ecstasy, benzodiazepines, cocaine, and methamphetamine/ecstasy, respectively, in breath and urine upon arrival (4:30 p.m.) at the laboratory on test days. At 5:00 p.m. subjects received a light, standard dinner, and at 5:15 p.m., MDMA or placebo was administered. The CADDS was completed at 6.30 p.m.

In study 2, we compared cannabis, cocaine, and a placebo to explore their effects on dissociative experiences in regular cannabis users. Screening and inclusion criteria were similar to those in study 1, with the exception of a minimum of THC use of twice per week during the previous three months, and a minimum of recreational cocaine use of 5 times in the previous year. Participants were asked to refrain from using any drugs except cannabis 1 week before the medical examination until study completion. They were not allowed to drink alcohol during a 24-h period prior to testing. Participants were always tested for alcohol and drugs (tetrahydrocannabinol, opiates, amphetamine/MDA, benzodiazepines, cocaine, and methamphetamine/MDMA/XTC) in breath and urine, respectively, upon arrival at the laboratory on test days. Treatments were only administered when participants were negative for all drugs except cannabis. This is because high lipid-solubility of cannabinoids will be present in the body for long periods of time.

The procedure entailed three test sessions on three separate days. Drugs were administered using a double-blind, placebo-controlled, double-dummy procedure. A double dummy procedure was used to control for differences in Tmax between both drugs. At T1 subjects received a cocaine or placebo capsule. At T2, 45 minutes after T1, subjects received either a single dose of cannabis (300 µg/kg THC) or cannabis placebo. At T3, 1 hr following T2, subject received another THC dose (150 µg/kg THC or cannabis placebo. Cannabis (300 µg/kg) or placebo (a herbal plant mixture (Knaster)) was administered through a vaporizer (Volcano) obtained from Storz & Bickel GmbH & Co (Tuttlingen, Germany) and was used according to the manual provided by the producer. Inhalation took place in a standardized manner (Hazekamp, Ruzaak, Zuurman, van Gerven, & Verpoorte, 2006). Percentage of THC was 11%, a standard potency for marijuana used recreationally and sold at Dutch pharmacies for medical use. Cocaine HCl (300mg) or placebo was administered in an opaque white capsule.

The DES was completed during a training session on a separate day before the placebo or drug sessions. The CADSS was completed 3 hours and 15 minutes after

T1 (i.e., 3.5 hours after cocaine and 1.25 hours after the second cannabis administration).

Data analysis

Statistical analyses were performed using SPSS 18.0 software. Cronbach's α values were used to estimate internal consistency of the measures. Pearson product-moment correlations between baseline and state measures were calculated. State data were analyzed using the General Linear Model (GLM). Significant multivariate results were evaluated with univariate tests. Huynh-Feldt or Greenhouse-Geisser corrected *P* values, their corresponding epsilons as well as the original, i.e. uncorrected, degrees of freedom are reported when the sphericity assumption was not met. We compared our data with findings from a previous study (Bremner et al., 1998), using independent samples t-tests.

Results

Table 6.1 shows mean scores on the DES and the CADSS and Pearson product-moment correlations between both variables in study 1 and 2.
Using one-way ANOVA, we found in study 1 a significant effect of drug dosage on CADSS scores ($F(3,45) = 21.27$, $p < .001$; partial eta² = .59). Post-hoc analyses revealed significant differences between the highest dosage MDMA (100 mg) and the other dosages (placebo, 25 mg, and 50 mg, $p < .05$ for all measures), but no significant differences were found between the placebo, 25 mg, and 50 mg conditions ($p = .22 – .72$).

We compared state dissociation levels in study 1 with those typically seen in patient samples (Bremner et al., 1998). Thus, for example, Bremner and colleagues administered the CADSS to 68 patients with combat-related PTSD ($M = 18.9$, $SD = 118.3$) and 22 patients with schizophrenia ($M = 3.7$, $SD = 5.2$). Our analysis showed that after ingestion of 100mg of MDMA, induced dissociative symptoms in our sample (see table 6.1) exceeded those of patients with schizophrenia ($t = 3.74$, $p < .001$), and were comparable to dissociation levels of patients with PTSD ($t = .20$, $p = .84$).

In study 2, CADSS scores were skewed to the right. We therefore performed a logarithm transformation on CADSS scores. Using repeated-measures ANOVA, we found a significant main effect of drug (THC, cocaine, or placebo) on CADSS scores ($F(2,36) = 14.99$, $p < .001$; partial eta² = .45). Post-hoc pairwise comparisons revealed significant differences between all three groups ($p < .001 – p = .045$), with the THC group exhibiting highest dissociation scores, followed by the cocaine group

(see table 6.1). Both drug groups scored significantly higher on dissociation than the placebo group.

We compared the CADSS scores obtained in study 2 with previous findings of Bremner and colleagues (1998). The induced levels of dissociation in study 2 were strikingly high. That is, induced levels of dissociation under the influence of THC were significantly higher than dissociation levels found in patients suffering from schizophrenia ($t = 2.77$, $p < .01$), and comparable to dissociation levels of patients with PTSD ($M = 18.9$, $SD = 118.3$; $t = .26$, $p = .79$; (Bremner et al., 1998)). Participants on cocaine exhibited dissociation levels comparable to mean dissociation levels in patients with schizophrenia, and PTSD ($t = 1.08$, $p = .28$, and $t = .51$, $p = .61$, respectively).

Finally, we investigated whether participants, who are high on trait dissociation, as measured with the DES, showed a higher sensitivity to drug induced state dissociation (see table 1). There was a significant correlation between the DES and CADSS scores when participants consumed cocaine, but not placebo or THC, indicating that participants displaying higher dissociative traits seem more vulnerable to experience state dissociation when under the influence of cocaine.

Table 6.1.
Mean scores and Pearson product-moment correlations of state dissociation in the MDMA group (Study 1; *N* = 16), and both trait (DES) and state (CADSS) dissociation in placebo, THC, and cocaine groups (Study 2; *N* = 21).

	M (SD)	DES	CADSS Placebo	CADSS THC
Study 1				
CADSS MDMA		-	-	-
0 mg	.69 (1.25)			
25 mg	1.50 (1.41)			
50 mg	2.50 (3.16)			
100 mg	13.06 (10.06)			
Study 2				
DES	17.30 (11.05)	-	-	-
Placebo	13.41 (8.39)			
THC	13.00 (5.29)			
Cocaine	23.93 (13.68)			
CADSS Placebo	1.09 (2.00)	-.02	-	-
CADSS THC	12.30 (13.61)	.33	.36	-
CADSS Cocaine	5.86 (7.77)	.47**	.16	.67**

Note. DES = Dissociative Experiences Scale; CADSS = Clinician-Administered Dissociative States Scale.
** $p < .001$

General discussion

Our findings can be catalogued as follows. First, we found that a high dosis of MDMA (i.e., 100 mg) increases acute dissociative symptoms. This may be due to the hallucinogenic properties of MDMA that potentially may arise from 5HT2 receptor agonism (Parrott, 2001; Parrott et al., 2000). Second, an increase in dissociation was also found when participants were under the influence of cannabis (THC). Again, this may reflect this drugs action on the psychopathological continuum that schizophrenic and dissociative symptoms share. Third, an increase in acute dissociation was also evident for cocaine use, but here effects were less pronounced. Our data are silent as to why this was the case, but one possibility could be that compared to THC, cocaine has stronger stimulant properties and therefore may optimally counteract fatigue and sleepiness, factors that may promote state dissociation (Van der Kloet et al., 2012a). Admittedly, this is a retrospective and speculative explanation, but it is a testable idea that warrants further study. Future studies on the psychopharmacology of dissociation might want to include sleep measures.

The usefulness of the psychopharmacological approach is underpinned by the fact that induced levels of dissociation for all recreational drugs were similar or exceeded the ones in patient groups who are known to exhibit high levels of

dissociation, such as schizophrenia and PTSD. Previous studies have relied on, for example, dot staring (Leonard, Telch, & Harrington, 1999) to induce dissociative symptoms in the lab, but our findings show that recreational drugs like THC may be used for this purpose as well.

At a more theoretical level, our findings suggest that an increased release of serotonine and dopamine neurotransmitter activity may play a role in the pathogenesis of dissociative symptoms. Research showing that dissociative disorders and substance abuse disorders are often co-occurring, are consistent with this view (Somer, Altus, & Ginzburg, 2010). Dual diagnoses patients are known to pose serious treatment challenges due to poor compliance, increased risk of suicidal and violent behaviors, and overall poorer functioning (Schwartz, Swanson, Hiday, Borum, Wagner, et al., 1998). In line with this, Somer and colleagues (2010) pose a "chemical dissociation" hypothesis to explain the link between drug abuse and dissociation. According to this hypothesis, individuals suffering from a traumatic past may use illicit drugs to blunt traumatic feelings, when dissociative pathology is not effective any longer to defend against intrusions of traumatic memories. This hypothesis is supported by studies tracing back the onset of dissociative pathology to substance abuse (Kessler, Sonnega, Bromet, Hughes, & Nelson, 1995; Tamar-Gurol, Sar, Karadag, Evren, & Karagoz, 2008).

Our studies were subject to a number of limitations that restrict the generalizability of the current findings to clinical groups. First, our samples were small and consisted of healthy, recreational users. Second, MDMA affects various neurotransmitter systems. Likewise, THC influences various brain regions. Thus, although we found that both high dosed MDMA and THC are potent drugs in eliciting acute dissociation, we can only speculate about the neurotransmitter systems and brain regions that carry these effects. Furthermore, both studies relied on self-report measures rather than neuroimaging or emotions task.

Nevertheless, our findings help to understand the neurochemical basis of dissociative symptomatology and ultimately, the psychopharmacological analysis of dissociation may point in the direction of pharmacological treatment avenues.

CHAPTER VII[10]

Self-reported sleep disturbances in patients with dissociative identity disorder and posttraumatic stress disorder and how they relate to cognitive failures and fantasy proneness

10 This chapter is an adapted version of the following article:
Van Heugten – van der Kloet, D., Huntjens, R. J. C., Giesbrecht, T., & Merckelbach, H. (2013). Self-reported sleep disturbances in patients with dissociative identity disorder and posttraumatic stress disorder and how they relate to cognitive failures and fantasy proneness. *Frontiers in Psychiatry, under revision*.

Summary

Sleep disturbances, fantasy proneness, cognitive failures, and dissociative symptoms are related to each other. However, the co-occurrence of these phenomena has been primarily studied in non-clinical samples. We investigated the correlations between these phenomena in dissociative identity disorder patients, post-traumatic stress disorder patients, and healthy controls. Both patient groups reported more sleep problems and lower sleep quality and displayed higher levels of fantasy proneness and cognitive failures than controls. However, the two patient groups did not differ with regard to these variables. Moreover, a higher level of unusual sleep experiences tended to predict participants belonging to the DID group, while specifically a lower sleep quality and more cognitive failures tended to predict participants belonging to the PTSD group.

Introduction

In psychopathology, dissociation typically refers to a disturbance in the normal integration of thoughts, feelings, and experiences into consciousness and memory. As dissociative symptoms are prevalent in both normal and clinical populations, dissociation has commonly been conceptualized as ranging on a continuum, from non-pathological manifestations of daydreaming to more severe disturbances typical of dissociative disorders (Bernstein & Putnam, 1986), which encompass dissociative amnesia, dissociative fugue, depersonalization disorder (DPD), and dissociative identity disorder (DID) (APA, 2000).

Epidemiological studies among psychiatric inpatients and outpatients have yielded prevalence rates of dissociative disorders exceeding 10% (Foote et al., 2006; Ross et al., 1991; Sar et al., 2000; Tutkun et al., 1998), and a recent study among women in the general population reported a prevalence rate of 18.3% for lifetime diagnoses of a dissociative disorder (Sar et al., 2009). However, 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 et al., 2006), and schizophrenia-spectrum disorder (Holmes et al., 2005) also display heightened levels of dissociation (Merckelbach et al., 2005; Yu et al., 2010).

A widely held notion about the aetiology of dissociative symptoms is that they serve a defensive function in that they help to cope with traumatic memories. Thus, many clinicians assume that dissociative symptoms are intimately linked to a history of traumatic childhood events (Spiegel, 2011). However, this conceptualization remains silent as to how precisely trauma might contribute to dissociative symptoms.

Recent studies have pointed to the role of sleep disturbances in dissociative symptoms (Koffel & Watson, 2009). This research line is not incompatible with the traumatogenic approach to dissociative symptoms. Thus, traumatic experiences may disrupt sleep, which in turn may contribute to or exacerbate dissociative symptoms. In our recent review, we (van der Kloet et al., 2012, see chapter I) concluded that data from 23 studies of clinical and nonclinical samples provide strong support for a link between dissociative experiences and sleep problems. This link is evident across a range of sleep-related phenomena, including waking dreams, nightmares, and hypnagogic (occurring while falling asleep) and hypnopompic (occurring while awakening) hallucinations. In support of this hypothesis, studies of the association between dissociative experiences and sleep disturbances have generally yielded modest correlations (in the range of .30 to .55), implying that unusual sleep experiences and dissociation are moderately related constructs (see also Watson, 2001).

Apart from their links with traumatic experiences and sleep disturbances, dissociative symptoms are related to cognitive failures and fantasy proneness (Merckelbach et al., 2002). Individuals suffering from dissociative symptoms typically score high on measures of fantasy proneness, which is a disposition to engage in extensive and vivid fantasizing (Merckelbach et al., 2005). Furthermore, individuals scoring high on dissociation report more cognitive failures (i.e., everyday slips and lapses) compared with 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, making them susceptible for suggestive information or manipulation (Merckelbach et al., 2002; Merckelbach et al., 2000).

One interpretation of these associations is that individuals with sleep problems experience intrusions of sleep phenomena (i.e., dreamlike experiences) into waking consciousness, which may foster dissociative symptoms. These sleep problems may have originated from distressing trauma-related memories, a genetic propensity (Lang et al., 1998), or other unknown influences. Furthermore, sleep disturbances may undermine cognitive efficiency by degrading memory and attentional control and fuel imaginative mentation, both of which are important constituents of dissociative symptoms.

Dissociative symptoms have the reputation of being refractory to treatment interventions (Lilienfeld, 2007). The recent research focus on sleep disturbances may contribute to the development of effective sleep hygiene interventions for patients with dissociative symptoms. However, before exploring this research avenue, it is important to ascertain in clinical groups with known dissociative symptomatology that sleep disturbances occur and are linked to dissociative symptoms and its correlates. Therefore, we investigated how cognitive failures, fantasy proneness, and sleep are related to dissociative symptoms in a sample of dissociative identity disorder (DID) patients, patients with post-traumatic stress disorder (PTSD), and healthy controls.

Material and Methods

Twelve female DID patients (*mean age*: 42 years, *SD* = 11.8), 27 female PTSD patients (*mean age*: 42 years, *SD* = 13.1), and 55 healthy female controls (*mean age*: 42 years, *SD* = 13.1) took part in the study. The DID group had a mean of 5.3 years of education (*SD* = 1.47), the PTSD group had studied for a mean of 4.15 years (*SD* = 1.25), and the controls had finished a mean of 5.9 years of education (*SD* = 1.18). Both DID and PTSD patients were recruited from treatment settings in The Netherlands and Belgium, and disorder status was verified with the Structured Clinical Interview for DSM-IV Dissociative Disorders (SCID-D; Steinberg et al., 1994)

by the second author. Mean length of treatment for DID was 8.61 years (*SD* = 5.6). Mean number of reported identities was 28 (range: 4-39, with one outlier of 196). PTSD status was verified with the Dutch version of the Clinician-Administered PTSD Scale (Hovens et al., 2005) by the second author. Mean length of treatment for PTSD was 2.58 years (*SD* = 3.2). DID or PTSD was always a primary diagnosis. Most participants used medication during the time of the study. In the DID group, medications were anxiolytics (i.e., benzodiazepines; *N* = 6), antidepressants (*N* = 8), antipsychotics (*N* = 1), and pain medication (*N* = 3). In the PTSD group, medications were anxiolytics (i.e., benzodiazepines; *N* = 13), antidepressants (*N* = 14), pain medication (*N* = 1), and beta-adrenergic blockers (*N* = 1).

Participants in the control group were matched on gender and age[11]. They were community volunteers. All participants received oral information prior to enrolment in the study, after which they gave written informed consent.

Participants completed a test battery including the Dissociative Experiences Scale (DES; Bernstein and Putnam, 1986); the Iowa Sleep Experiences Survey (ISES; Watson, 2001), the Pittsburgh Sleep Quality Inventory (PSQI; Buysse et al., 1988), the Creative Experiences Questionnaire, which is an index of fantasy proneness (CEQ; Merckelbach et al., 2001), and the Cognitive Failures Questionnaire (CFQ; Broadbent et al., 1982), which is a measure of cognitive efficiency. The current data were collected as part of a larger study (Huntjens, Verschuere, & McNally, 2012).

Results

The groups differed in amount of years of education. Using ANOVA, we found that this difference was significant between all three groups ($F(2,94) = 17.31$, $p < .001$) with the PTSD group scoring lower than the DID and control groups. Table 7.1 summarizes the psychometric test data of the three groups. The groups differed significantly from each other with regard to the total and subscale dissociation (DES) scores, with the exception of the absorption subscale on which the DID group did not score higher than the PTSD group. For all other measures, a significant difference was found between DID and controls and between PTSD and controls, but not between the two patient groups.

11 The control group included in this manuscript partly overlaps with the healthy control group and simulator group as included in Huntjens et al., (2012). The simulators were not instructed to mimic DID on the measures included in the current manuscript. The simulator and control groups did not differ on fantasy proneness, ($t = .03$, $p = .98$), age ($t = .32$, $p = .75$), or level of education ($t = .04$, $p = .40$, for more information see Huntjens et al., 2012).

Table 7.1.
Mean scores (Standard Deviations) on dissociation (DES), unusual sleep experiences (ISES), sleep quality (PSQI), fantasy proneness (CEQ), and cognitive failures (CFQ) of the DID (*N* = 12), PTSD (*N* = 27), and Control (*N* = 55) groups.

	DID	PTSD	CONTROL	*F*	*p*	*C's α*
DES						
Total[abc]	44.99 (13.99)	26.23 (16.55)	7.01 (5.23)	72.73	<.001	.97
Absorption[ac]	43.88 (17.55)	35.64 (20.51)	11.54 (8.97)	40.85	<.001	.91
Amnesia[abc]	37.81 (13.97)	14.03 (14.75)	2.34 (2.90)	71.28	<.001	.92
Depersonalization[abc]	49.44 (18.05)	22.04 (22.14)	2.70 (4.60)	62.08	<.001	.93
ISES						
Total[ac]	69.42 (18.69)	64.85 (21.64)	40.05 (15.07)	25.49	<.001	.93
General Sleep[ac]	62.50 (17.71)	57.93 (19.96)	34.11 (12.48)	30.10	<.001	.93
Lucid Dreaming	6.92 (3.03)	6.93 (3.73)	5.95 (3.72)	.82	.44	.75
PSQI[ac]	13.92 (3.58)	14.74 (3.04)	8.93 (2.47)	45.45	<.001	.80
CEQ[ac]	10.17 (3.66)	8.82 (4.50)	5.64 (3.99)	9.20	<.001	.84
CFQ[ac]	49.75 (16.60)	53.89 (10.99)	41.35 (7.80)	14.63	<.001	.93

Note. Pairwise comparisons significant at the .05 level (2-tailed, Bonferroni corrected) are designated as: [a]DID differs from CONTROL; [b]DID differs from PTSD; [c]PTSD differs from CONTROL.

Table 7.2 displays the Pearson product moment correlations between the measures. Effect sizes were medium to large. We performed backwards binary logistic regression analyses[12] on the DID group and the PTSD group to determine which variable most likely predicted the chance of belonging to one of the psychopathology groups. We entered the DID, or PTSD group as dependent variable, respectively. Then, based on our earlier ANOVA, we entered CEQ, CFQ, PSQI, and ISES as predictors in each analysis. We left out the DES, as it would bias our analyses, because dissociation forms a diagnostic determinant in DID. A summary of the regression analyses are displayed in table 7.3. We found that a high score on the ISES best predicted the chance of belonging to the DID group, while higher scores on the CFQ and PSQI predicted the likelihood of belonging to the PTSD group.

12 We performed analyses both with and without number of education years as possible confounder. Education did not prove to make a significant difference in outcome scores. Therefore, we decided to present only the original analyses without education.

Table 7.2.
Pearson product moment correlations between baseline measures (*N* = 94).

	DES	ISES	PSQI	CEQ
DES	-	-	-	-
ISES	.65**	-	-	-
PSQI	.54**	.49**	-	-
CEQ	.49**	.48**	.26**	-
CFQ	.57**	.46**	.48**	.44**

Note. DES = Dissociative Experiences Scale; ISES = Iowa Sleep Experiences Survey; PSQI = Pittsburgh Sleep Quality Index ; CEQ = Creative Experiences Questionnaire; CFQ = Cognitive Failure Questionnaire.
* $p < .05$;
** $p < .001$

Table 7.3.
Summary of backwards binary logistic regression analyses on DID group (*n* = 12), PTSD group (*n* = 27), and combined Psychopathology group (*n* = 39).

Model	*B* (SE)	Wald	*p*	R^2	χ^2 (*df*)	*p*
DID						
1.				.27	14.74 (4)	.005
CEQ	.15 (.09)	2.93	.09			
CFQ	-.04 (.03)	1.65	.20			
PSQI	.18 (.10)	3.27	.07			
ISES	.03 (.02)	3.31	.07			
2.				.25	13.16 (3)	.004
CEQ	.12 (.08)	2.09	.15			
PSQI	.13 (.09)	2.07	.15			
ISES	.03 (.02)	2.50	.11			
3.				.21	11.03 (2)	.004
CEQ	.10 (.08)	1.76	.19			
ISES	.04 (.02)	4.70	.03			
4.				.17	9.26 (1)	.002
ISES	.04 (.01)	8.39	<.01			
PTSD						
1.				.49	39.09 (4)	<.001
CEQ	.04 (.08)	.23	.63			
CFQ	.04 (.03)	1.66	.20			
PSQI	.33 (.09)	12.04	<.001			
ISES	.02 (.02)	1.12	.29			
2.				.49	38.86 (3)	<.001
CFQ	.05 (.03)	2.35	.13			
PSQI	.32 (.09)	11.95	<.001			
ISES	.02 (.02)	1.32	.25			
3.				.47	37.58 (2)	<.001
CFQ	.06 (.03)	4.09	.04			
PSQI	.34 (.09)	14.21	<.001			

Note. DID = DID group (*N* = 12); PTSD = PTSD group (*N* = 27); ISES = Iowa Sleep Experiences Survey; PSQI = Pittsburgh Sleep Quality Index ; CEQ = Creative Experiences Questionnaire; CFQ = Cognitive Failure Questionnaire

Discussion

This study is – to the best of our knowledge – the first to examine the relation between self-reported sleep disturbances, fantasy proneness, and cognitive failures in patients with DID. Our aim was to ascertain in clinical groups with known dissociative symptomatology that sleep disturbances occur and are linked to dissociative symptoms and its correlates. Before we discuss the conclusions that can be drawn from our findings, it is important to emphasize some limitations of the current study. Although our sample consisted of a unique group of patients with DID, combined with a group of PTSD patients and a healthy group of controls, sample sizes of the patient groups were relatively small and limited to the female gender. Thus, future research might want to include a larger sample of both male and female patients. Furthermore, it would have been superior if we could have conducted mediation analyses that would allow for specific testing within patient groups. Unfortunately, this was not possible due to low power issues. Therefore, using logistic regression analyses, we aimed to provide some information about the separate groups. Most importantly, our study relied on a cross-sectional design based on self-report measures that precludes any causal interpretations. Thus, a longitudinal set-up in which patients undergoing targeted treatments for their sleep dysfunctions are followed over time would provide a more optimal starting point for testing causal hypotheses.

With these limitations in mind, the main findings of our study can be summarized as follows.

The DID group only differed from the PTSD group in the number of dissociative symptoms. Both patient groups reported more unusual sleep experiences (as measured by ISES) and worse sleep quality (as measures by PSQI) than controls, and scored more pronounced on levels of fantasy proneness and cognitive failures. Moreover, a higher level of unusual sleep experiences tended to predict participants belonging to the DID group, while specifically a lower sleep quality and more cognitive failures tended to predict participants belonging to the PTSD group. Our findings are consistent with the hypothesis that sleep problems lead to intrusions of sleep phenomena into waking consciousness, resulting in dissociative experiences (Watson, 2001). Because sleep disruptions exert detrimental effects on attentional control and memory, they may contribute to the attention deficits that are typically found in patients with dissociative disorders (Guralnik et al., 2007). However, our data also indicate that both unusual sleep experiences and sleep problems (i.e., poor sleep quality) are not unique for patients with DID. Patients with DID and PTSD seem to share these sleep-related problems and future studies focusing on the treatment implications are warranted, as the impact of sleep quality on cognitive functioning might be substantial in these patients. The idea

that DID is strongly related to PTSD fits well with our findings (Zlotnick, Zakriski, Shea, Costello, Begin, Pearlstein et al., 1996).

PART III

TREATMENT OF DISSOCIATIVE SYMPTOMS

CHAPTER VIII[13]

Decreasing dissociative symptoms using sleep hygiene recommendations: An exploratory study

13 This chapter is an adapted version of the following article:
Van Heugen – Van der Kloet, D., Soontiëns, L., & Giesbrecht, T. (Submitted). Decreasing dissociative symptoms using sleep hygiene recommendations: An exploratory study. *Journal of Experimental Psychopathology, submitted*.

Summary

Most treatments for dissociative symptoms are predicated on the belief that traumatic childhood experiences are a prominent etiological factor in these symptoms and that the adverse effects of traumatic experiences need to be resolved. The effectiveness of such interventions, however, leaves much to be desired. An alternative approach to the treatment of dissociation is offered by research that suggests that dissociative individuals have a labile sleep-wake cycle that causes them to experience intrusions of sleep phenomena into their waking consciousness. With this in mind, our study explored whether a sleep normalization intervention is followed by a decrease of dissociative symptoms. Young adults (N = 37) with sleep problems strictly committed themselves to sixteen sleep hygiene recommendations for a period of four consecutive days, and completed a test battery containing the Dissociative Experiences Scale, the SLEEP-50 , and the Iowa Sleep Experiences Survey among others. Every day, acute states of dissociation, sleepiness, anxiety, and mood were assessed. Results provide initial support for the idea that sleep hygiene behaviors may contribute to a reduction of dissociative symptoms.

Introduction

In the *Diagnostic and Statistical Manual of Mental Disorders* (*DSM-IV-TR;* APA, 2000), dissociation is defined as "a disruption in the usually integrated functions of consciousness, memory, identity, or perception of the environment" (p. 519). Many clinicians believe that dissociative symptoms like amnesia, derealization, depersonalization, and absorption are invoked as a defense mechanism to psychologically withdraw oneself from the impact of overwhelming traumatic memories (Giesbrecht & Merckelbach, 2008). These clinicians further assume that chronic reliance on this defense mechanism is what constitutes a dissociative disorder (e.g., dissociative identity disorder; van der Hart et al., 2004).

Although several studies have demonstrated a reliable association between dissociative symptoms and retrospective self-reports of traumatic events (Brand et al., 2009; Dalenberg & Palesh, 2004; Nash, Hulsey, Sexton, Harralson, & Lambert, 1993), there is only scarce evidence that trauma directly causes dissociative symptoms (Giesbrecht & Merckelbach, 2008; Kihlstrom, 2005; Giesbrecht et al., 2008). Nevertheless, the most commonly used treatment interventions for dissociative disorders are predicated on the belief that trauma is a direct etiologic factor in dissociative disorders. Such treatments often incorporate techniques like hypnosis, regression therapy, and dream analysis that intend to help the patient cope with post-traumatic psychopathology (Vermetten, Dorahy, & Spiegel, 2007). However, there is little evidence for the effectiveness of these, often costly and time-consuming, interventions (Lilienfeld, 2007).

Unfortunately, patients with dissociative disorders are notoriously resistant to treatment. One reason for this is that patients suffering from a dissociative disorder often have multiple psychiatric diagnoses (Brand et al., 2009). Moreover, patients with dissociative disorders regularly engage in self-destructive behaviors and repeated suicide attempts are not uncommon (Allen et al., 2000; Loewenstein, 1994). Also, it is not uncommon that these patients terminate treatment prematurely or show a worsening of their symptoms over treatment (Brand et al., 2009; Tarrier, Pilgrim, Sommerfield, Faragher, Reynolds, Graham, & Barrowclough, 1999).

An alternative treatment option is suggested by the pioneering work of Watson (2001), who studied individual differences in sleep-related experiences. Sleep-related experiences refer to a variety of nocturnal phenomena, such as nightmares, recurring dreams, dream recall, waking dreams (i.e., dreams confused with reality), vivid dreams, problem-solving dreams, narcoleptic symptoms including sleep paralysis (i.e., a conscious state in which one is unable to move upon wakening or prior to falling asleep), and hypnagogic or hypnopompic hallucinations (i.e., sensory hallucinations occurring at sleep onset or offset; Soffer-Dudek & Shahar, 2009). In his study, Watson (2001) related these self-reported sleep anomalies to

dissociative symptoms and found a significant relationship between both constructs. A number of authors have replicated this finding (Giesbrecht et al., 2006; Giesbrecht & Merckelbach, 2004; Watson, 2003).

Similarly, researchers have noted that there is a substantial overlap between dissociative experiences and nightmare frequency (Agargun et al., 2003; Levin & Fireman, 2002). One sleep deprivation study with undergraduates showed that sleep loss intensifies dissociative symptoms (Giesbrecht et al., 2007). In a clinical study, empirical support was found to suggest that normalization of sleep patterns is accompanied by a specific reduction in dissociative symptoms (van der Kloet et al., 2012; see chapter X). The link between dissociative symptoms and unusual sleep experiences suggests a continuity, such that people who are prone to vivid and unusual experiences during the day also tend to have them at night and vice versa (Watson, 2001). In a similar vein, Mahowald and Schenck (2001) argued that the relationship between dissociation and unusual sleep experiences can be explained in terms of an overlap of daytime and nighttime experiences. There is, indeed, a growing body of evidence suggesting that dissociative individuals have a labile sleep-wake cycle, which causes them to experience intrusions of sleep phenomena in their waking consciousness (Giesbrecht & Merckelbach, 2006). As a result of such intrusions, these individuals may have feelings of depersonalization and other unusual, dreamlike experiences (e.g., memory loss, identity confusion, absorption), which are all key features of dissociation.

Research showing that sleep disruptions intensify dissociation sparked the basic idea that patients with dissociative symptoms may profit from therapeutic approaches directed at normalizing sleep patterns (Giesbrecht et al., 2007). Interestingly, there is a substantial body of research describing effective treatments for sleep problems (Harvey, Sharpley, Ree, Stinson, & Clark, 2007; Ong, Shapiro, & Manber, 2009; Stepanski & Wyatt, 2003). Many of these treatments include sleep hygiene interventions (Stepanski & Wyatt, 2003), which target factors known to affect sleep. The latter include bedtime routine, consistency of bedtimes, abstinence from alcohol and caffeine, refraining from taking naps, and the amount and timing of sports and exercise (Cheek, Shaver, & Lentz, 2004). Specifically, sleep hygiene interventions aim to promote those behaviors, lifestyle practices, environmental factors, and other sleep related factors that are believed to improve quality and quantity of sleep (Stepanski & Wyatt, 2003).

The present exploratory study aimed to examine dissociative symptoms after sleep normalization by means of sleep hygiene instructions. Finding that dissociative symptoms decrease after such instructions, would be consistent with, but by no means prove that sleep normalization reduces dissociative symptoms. On the other hand, finding that dissociative symptom levels remain unaffected by sleep hygiene measures would refute the idea that sleep normalization reduces dissociative symptoms. Thus, the specific aims of this exploratory study were: (1) to

provide further evidence for the relationship between unusual sleep experiences and dissociative symptoms; (2) to gather evidence for a correlation between sleep hygiene measures and a reduction of dissociative symptoms; and (3) to investigate to what extent individuals continue to make use of sleep hygiene instructions after the treatment protocol has ended.

Method

Participants

Thirty-seven young adults (34 women) were recruited from different schools of Maastricht University as well as the surrounding community by means of ads in which we invited people with sleep problems to participate in our research project. The participants' mean age was 21.8 years (*SD* = 3.23, range: 18 – 30). Participants were included when they reported sleep problems, such as problems with falling asleep, staying asleep, early morning awakenings, nightmares, vivid dreams, and hallucinations at night.

The presence of sleep problems was determined by means of a brief interview conducted by e-mail. Prior to their participation in the study, volunteers were screened for the presence of severe psychopathology with the Brief Symptom Inventory (BSI; Derogatis & Melisaratos, 1983), the Beck Depression Inventory, second edition (BDI-II; Beck et al., 1996), and the State-Trait Anxiety Inventory, version DY (STAI-DY; Van der Ploeg, Defares & Spielberger, 1979).

Procedure

Participants were asked to complete questionnaires via internet on a password-protected website, identifying themselves by randomly assigned participant identification numbers distributed at the time they enrolled in the study. At baseline, they completed a battery of questionnaires measuring trait dissociation, sleep quality, and unusual sleep experiences (see *Measures*). After baseline data had been obtained, participants attended a group session during which the importance of healthy sleep hygiene behaviors was explained and participants were motivated to adhere to a number of simple measures. More specifically, participants received a Sleep Hygiene Protocol, consisting of sixteen strategies that are known to promote a stable sleep-wake cycle (e.g., refraining from alcohol and caffeine, promoting relaxing activities before sleeping, providing a dark and quiet room to sleep in). Participants were asked to strictly commit themselves to these strategies for a period of four consecutive days. During this period, each morning,

participants completed a series of state measures to assess sleepiness, state anxiety, state dissociation, and sleep patterns (see *Measures*).

Participants were approached via a surprise follow-up e-mail two weeks later. A total of 26 participants (25 women) took part in this follow-up part of the study. They completed once more the battery of trait measures, and a questionnaire which measured to what extent they still made use of the sleep hygiene recommendations from the Sleep Hygiene Protocol.

Measures

The *Dissociative Experiences Scale* (DES; Bernstein & Putnam, 1986, Cronbach's α = .90) measures to what extent respondents experience 28 dissociative symptoms. Respondents indicate on 100-mm Visual Analogue Scales (anchors: 0 = never; 100 = always) the frequency of these symptoms (e.g., feelings of depersonalization, psychogenic amnesia, and derealization).

The *Iowa Sleep Experiences Survey* (ISES; Watson, 2001, Cronbach's α = .89) is an 18-item self-report questionnaire that measures the frequency of various sleep- and dream-related experiences on a 7-point Likert scale (anchors: 1 = never; 7 = several times a week). Factor analysis yielded two subscales: General Sleep Experiences (GSE) and Lucid Dreaming (LD; Watson, 2001). GSE includes sleep abnormalities that are not under a person's control (e.g., nightmares and sleep paralysis), whereas LD refers to a type of dreaming that the person feels s/he can control.

The *SLEEP-50* (Spoormaker, Verbeek, Van den Bout & Klip, 2005, Cronbach's α = .86) measures the intensity of subjective sleep complaints during the past four weeks with 50 self-report items that are scored on a 4-point Likert scale (anchors: 1 = not at all; 4 = very much).

The *Stanford Sleepiness Scale* (SSS; Hoddes et al., 1973) is a 1-item self-report rating scale to assess subjective perceptions of daytime sleepiness. The item is scored on a 7-point Guttman scale (anchors: 1 = feeling active and vital, alert, wide awake; 7 = almost in reverie, sleep onset appears imminent, lost struggle to remain awake).

The *Clinician Administered Dissociative States Scale* (CADSS; Bremner et al., 1998, Cronbach's α = .95) is a 27-item instrument that measures acute dissociative symptoms on a 5-point Likert scale (anchors: 0 = not at all; 4 = extremely). The instrument contains 19 self-report items and 8 clinician rated items. Only the self-report items were used in the present study (see also Giesbrecht et al., 2007; Morgan, Southwick, Hazlett & Steffian, 2007). The self-report items can be grouped into three subscales, reflecting derealization (DER), depersonalization (DEP), and amnesia (AMN). The CADSS has good discriminant validity (Bremner et al., 1998).

The *sleep diary* is a self-report instrument[14] to examine an individual's sleep pattern and sleep hygiene behaviors. It was designed for the purpose of this study. It contains questions about sleep pattern variables (e.g., time of lights out, number of awakenings during the night, time of final awakening, and time out of bed), and sleep hygiene behaviors (e.g., naps during the day, activities prior to sleep, consumption of alcohol, caffeine, and medicine). Using self-reports from the participants in the sleep diary, sleep pattern variables were computed as follows; Total Wake Time (TWT) was defined as the sum of sleep onset latency (the time it took to fall asleep from the time of lights out), the time awake after sleep onset, and the time between final awakening and time out of bed. Sleep efficiency (SE) was defined as the proportion of time asleep during the sleep period, calculated as the total sleep time divided by the time spent in bed, multiplied by one-hundred. Participants completed the items every morning. Sleep diaries are commonly used and are found to be a valid means of collecting data about daily activities and sleep perceptions (Cheek, Shaver & Lentz, 2004).

Results

Mean scores for DES, SLEEP-50, and ISES for baseline and follow-up are displayed in table 8.1. These scores correspond with values previously found for student samples (Giesbrecht & Merckelbach, 2004, 2006; Giesbrecht et al., 2007). Table 8.1 also presents the Pearson product-moment correlations between these trait measures. In line with previous findings (Fassler et al., 2006; Giesbrecht & Merckelbach, 2004; Giesbrecht et al., 2006; Watson, 2001), positive correlations emerged between dissociative symptoms (DES) and various sleep-related experiences (ISES; SLEEP-50) at baseline. Specifically, dissociation correlated significantly with the GSE subscale of the ISES, whereas the correlation between dissociation and the LD subscale remained non-significant.

[14] A copy of this instrument can be obtained from the first author

Table 8.1.
Mean scores of baseline measures and follow-up measures, and correlations between dissociation, subjective sleep complaints, and unusual sleep experiences at baseline (*N* = 37) and follow-up (*N* = 26).

	Mean (SD)		*DES*	*Sleep-50*	*ISES total*	*ISES GSE*
	BM	*FU*	*BM*	*BM*	*BM*	*BM*
			FU	*FU*	*FU*	*FU*
DES	21.30 (17.50)	22.52 (17.24)	-	-	-	-
Sleep-50	87.41 (14.15)	80.12 (11.26)	.41*	-	-	-
			.42*	-	-	-
ISES total	3.04 (0.93)	2.78 (0.92)	.51**	.36*	-	-
			.64**	.45*	-	-
ISES GSE	3.08 (0.89)	2.82 (0.85)	.52**	.39*	.97**	-
			.68**	.45*	.97**	-
ISES LD	2.83 (1.60)	2.58 (1.71)	.27	.11	.74**	.56**
			.33	.26	.80**	.64**

Note. DES = Dissociative Experiences Scale; ISES = Iowa Sleep Experiences Scale; GSE = General Sleep Experiences subscale; LD = Lucid Dreaming subscale; BM = baseline measurement; FU = follow-up measurement.
* p <.05 level (2-tailed)
** p < .01 level (2-tailed)

Figure 8.1 displays the mean scores of sleep pattern variables and the dissociative state measure (CADSS) for the four consecutive days of measurement and follow-up. Four repeated measures analyses (ANOVA) were conducted with time as within-subjects factor and TWT, SSS, SE, and CADSS as dependent variables, respectively. For none of the variables, significant time effects were found (all F's < 3.13, p's > .09, partial eta² = .04-.08). However, a trend becomes visible when the results are displayed in Figure 8.1. Paired samples tests revealed significant mean differences between day 1 and day 4 for SSS (t = 2.58, p < .01), SE (t = -2.06, p = .02), and the derealization subscale of the CADSS (t = 1.85, p = .04). Also, borderline significance was reached for the mean difference between day 1 and 4 for the TWT (t = 1.47, p = .08). Thus, both sleepiness and dissociation seemed to decrease during the four day period of the Sleep Hygiene Protocol, while sleep efficiency increased. Unfortunately, these beneficial results disappeared at follow-up (Paired differences between day 1 and follow-up for SE: t = 0.57, p = .29; TWT: t = -0.87, p = .20; and CADSS: t = -0.52, p = .60).

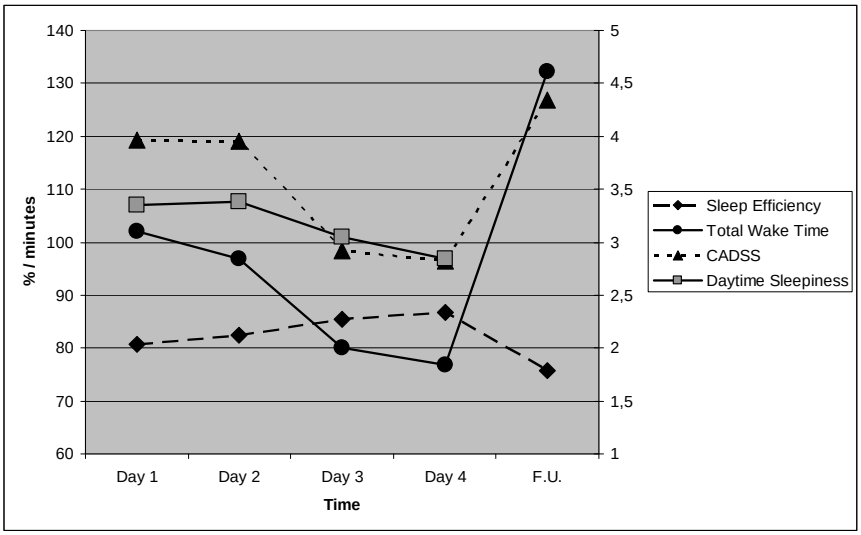


Figure 8.1.

Time course of acute dissociation (right y-axis), daytime sleepiness (right y-axis), sleep efficiency (left y-axis) and total wake time (left y-axis) during the test period. Acute dissociative symptoms, daytime sleepiness, and total wake time decrease during the sleep hygiene protocol, and sleep efficiency improves. At follow-up, sleep efficiency has decreased, while total wake time and dissociative symptoms have increased.

Pearson product moment correlations between total wake time (TWT), sleepiness (SSS), sleep efficiency (SE) and dissociation (CADSS) are presented in table 8.2. There was a significant negative correlation between sleep efficiency and acute dissociation at day 4, and positive correlations between sleepiness and acute dissociation at days 1 to 3. This indicates that higher sleep efficiency was related to lower dissociation scores, and that more sleepiness was related to higher dissociation scores.

We were also interested whether participants would continue to use sleep hygiene strategies voluntarily after completion of the study. Completers versus noncompleters did not differ on any of the baseline measures (all *p*'s > .05). At the surprise follow-up two weeks later, they were asked to what degree they continued to make use of the sleep hygiene recommendations during the preceding week. These scores were dichotomized in a high (*N* = 12) and a low (*N* = 14) compliance group. Independent samples t-tests were performed to compare follow-up data for both groups. The high compliance group scored significantly higher on the SE (*t* = 2.79, *p* < .01), and significantly lower on the TWT (*t* = -2.58, *p* = .01). Mean scores for the high compliance group on the DES, SLEEP-50, and ISES and its GSE and LD subscales did not significantly differ from the low compliance group (*t*'s = .33-.94, *p*'s = .18-.37, respectively).

Table 8.2.
Pearson product-moment correlations between sleep pattern variables and state measures (*N* =37)

	CADSS D1	CADSS D2	CADSS D3	CADSS D4
TWT	.10	.08	.19	-.10
SE	-.03	-.16	-.08	**-.41***
SSS	**.56****	**.49****	**.58****	.28

Note D1 = day one measurement, D2 = day two measurement, D3 = day three measurement, D4 = day four measurement; TWT = Total Wake Time during the night; SE = Sleep Efficiency; SSS = Stanford Sleeping Scale; CADSS = Clinician-Administered Dissociative States Scale total score
* p <.05 level (2-tailed)
** p < .01 level (2-tailed)

Discussion

The present study was an exploratory evaluation of a sleep normalization intervention for treating dissociative symptoms using sleep hygiene recommendations. The main findings can be summarized as follows. First, we found further evidence for the existence of a robust relationship between dissociative symptoms and sleep-related experiences. In line with previous studies (Arargun et al., 2003; Fassler et al., 2006; Giesbrecht & Merckelbach, 2004; Giesbrecht et al., 2006; Watson, 2001), dissociative symptoms were found to positively correlate with several unusual sleep experiences, such as vivid dreams, nightmares, and narcoleptic characteristics. In addition, positive correlations were also found between daytime sleepiness and state dissociation. This is in line with the findings of Giesbrecht and colleagues (2007), who showed that state dissociation is significantly related to sleepiness. Thus, our findings provide further support for the view that disruptions in the sleep-wake cycle may give rise to dissociative phenomena (Giesbrecht et al., 2006; Giesbrecht & Merckelbach, 2006; Mahowald & Schenck, 2001; Watson, 2001, 2003).

Interestingly, no significant correlations were found between dissociative tendencies and lucid dreaming. This finding is in keeping with other research employing the DES and the ISES (Giesbrecht & Merckelbach, 2004; Giesbrecht et al., 2006; Watson 2001, 2003), and suggests that the link between unusual sleep experiences and dissociation is rather specific. That is, dissociation seems to be related to unusual sleep experiences that are uncontrollable (e.g., nightmares), but not to unusual sleep experiences amenable to one's control such as lucid dreams. Second, we found that a period of strict commitment to sleep hygiene recommendations is related to a reduction of sleep problems and daytime sleepiness. In line with studies aimed at examining treatment programs for insomnia (Harvey et al., 2007; Ong et al., 2009; Stepanski & Wyatt, 2003), we were

able to find support for the effectiveness of sleep hygiene advice in treating sleep problems. Although the improvements with respect to sleep problems due to the Sleep Hygiene Protocol were not significant on a day-to-day scale, significant improvements were found between the first and the fourth day of the Protocol. Specifically, participants' subjective perceptions of daytime sleepiness were reduced after only four days of improved sleep hygiene behaviors. Participants felt more awake and able to concentrate after acting upon the recommended sleep-related advice for several days. Moreover, their number of awakenings during the night significantly decreased, their total sleep time significantly augmented, and most importantly, their sleep efficiency significantly improved. Participants' mean proportion of time asleep during the sleep period increased from 80.8% to 86.7% after four days of strict commitment to the Sleep Hygiene Protocol.

At the two week surprise follow-up, little improvements in nighttime symptoms of insomnia and daytime sleepiness were apparent. Aside from a significant reduction in the reported intensity of subjective sleep complaints during the past four weeks, as measured with the SLEEP-50, no significant differences were found between pre-treatment and post-treatment scores for all sleep variables. Thus, the sleep hygiene intervention has little lasting effects, if it is not boosted. These findings emphasize the importance of finding ways to change lifestyle patterns, which are detrimental to sleep, permanently. This is in line with the earlier assumption of Brown, Buboltz, and Soper (2006) that larger changes in overall sleep quality may only occur over longer periods of time.

Third, we found that sleep hygiene recommendations were moderately related to a reduction of dissociative symptoms. Significant improvements were found between the first and the fourth day of the Sleep Hygiene Protocol for the derealization subscale of the CADSS. Participants experienced less state derealization, i.e., their feeling that things around them were unreal decreased. Yet, no significant reductions were found in the frequency with which participants, at follow-up, experienced dissociative phenomena in daily life, compared to their baseline DES scores. Thus, the beneficial effects of sleep hygiene on more trait-like dissociative symptoms remain far from clear. Therefore, further research has to be conducted to obtain clarity concerning possible long term effects of improved sleep hygiene on dissociative symptoms.

In sum, this study broadens our understanding of the link between dissociative symptoms and disruptions in sleep patterns. Moreover, the study was able to replicate positive changes in self-reported sleep quality during a sleep hygiene program. Finally, we found some evidence to suggest that an improvement of sleep quality directly is accompanied by a reduction of state dissociation.

Before closing, a number of caveats in interpreting our findings merit mention. A first limitation of this study is inherent to its design as an uncontrolled treatment development study. Since the study lacked a control group, the possibility that the

observed effects are due to spontaneous recovery or to regression towards the mean cannot be ruled out (Loosveldt, 2005). In future studies, we recommend the use of randomized controlled trials with active control conditions. A second limitation is the small, homogenous and non-clinical sample, with only 37 participants. Future studies should enroll larger and more heterogeneous research samples in order to improve the generalizability of the findings. Third, although we employed clinically well-validated measures, the current data are based entirely on the respondents' self-reports, which may be subject to social desirability and/or recall bias. This is particularly true for the sleep pattern variables derived from the sleep diary. Although sleep diaries represent a core assessment component in studies on sleep problems, polysomnography would be preferred to confirm objective changes in sleep (Ritterband et al., 2009). Moreover, we were unable to monitor and enforce compliance with the sleep hygiene advice. Participants were clearly advised how and when to use the sleep hygiene recommendations, but it remains unclear how many actually did comply with the advice. Finally, participants were merely screened for the presence of sleep problems by a brief e-mail interview. In future studies, objective measures should be used to assess the presence of sleep problems. It would also be interesting to investigate the longitudinal course of dissociative symptoms following sleep hygiene improvements and to conduct further research to test the hypothesis that a labile sleep-wake cycle promotes dissociative symptoms, since this area is still in its infancy. For example, it would be fascinating to experimentally explore with polysomnographic and brain activation measures whether people high on dissociation display an excess of REM or NREM sleep indicators during the day (Koffel & Watson, 2009a).

In conclusion, with these methodological limitations in mind, the findings of the present study should be interpreted with caution. Nevertheless, we were able to find further support for the link between dissociative symptoms and unusual sleep experiences. Moreover, we provided initial support for the assumption that improvements in sleep hygiene behaviors can lead to a reduction of acute dissociative symptoms.

CHAPTER IX[15]

Mindfulness Based Therapy improves sleep, but not dissociative symptoms: A preliminary study

15 This chapter is an adapted version of the following article:
Van Heugten – Van der Kloet, D., Didderen, A., & Giesbrecht, T. (Submitted). Mindfulness Based Therapy improves sleep, but not dissociative symptoms – A preliminary study. *Mindfulness, submitted*.

Summary

Sleep problems and unusual sleep experiences are related to dissociation, while sleep normalization goes hand in hand with a reduction in dissociative symptoms. Mindfulness Based Therapy (MBT) has been demonstrated to have a positive effect on sleep quality. The present study aimed to investigate the effectiveness of 8-week MBT training as a treatment for dissociation by employing pre- and post-questionnaires in participants (*N* = 92) measuring sleep quality, unusual sleep experiences, dissociative symptoms, general psychopathology, and mindfulness skills. After MBT, sleep quality was improved and psychopathology was reduced significantly. However, no reduction of dissociative symptoms nor an increase in mindfulness skills were found after MBT. In sum, the study provides further evidence for the effectiveness of MBT in reducing sleep problems, but not for its usefulness in the treatment of dissociative symptomatology.

Introduction

One third of the Dutch population experiences sleep problems. These problems refer to both problems in sleep quality and quantity, and conditions closely associated with the sleep cycle or the physiological mechanisms of sleep (Ohayon, 2002). For up to 15% of the population, insomnia is a serious problem leading to impaired functioning such as irritability, concentration problems, and a lack of energy (Ohayon, 2002). People who suffer from chronic insomnia often explain their condition as a 'vicious cycle' (Ong & Sholtes, 2010). During the day they feel less able to concentrate and to perform well, which worries them at night. Moreover, these people increasingly dread going to bed, which leads to the development of bad sleeping behavior. The fear of not being able to sleep increases stress and enhances sleep problems. Therefore, chronic insomnia can persist long, even after the original cause had disappeared (Ong & Sholtes, 2010). Sleep loss can cause other diseases or disorders and in turn, diseases and disorders can lead to sleep problems. These entail physical illnesses as well as mental disorders. Disruptions in sleep patterns are particularly well-known in some psychiatric groups. Thus, patients with mood disorders, anxiety disorders, posttraumatic stress disorder, schizophrenia or borderline personality disorder often have serious sleep dysfunctions (Benca et al., 1992; Soffer-Dudek & Shahar, 2011). Extensive research has been conducted to elucidate the role of sleep disorders in mood and anxiety.

However, our understanding of the role sleep plays in dissociation is only starting to be explored since the pioneering study of Watson (2001).

Dissociative symptoms include a wide range of experiences and complaints, ranging from absorption to memory problems, and concerns regarding the own identity (Gershuny & Thayer, 1999). To a mild degree, most people are familiar with these subjective experiences. However, people with more severe dissociative symptoms, are defined in diagnostic groups such as posttraumatic stress disorder, schizophrenia and the dissociative disorders.

As earlier mentioned, a number of studies have investigated the relation between dissociation and sleep problems. For example, a study by Agargun and colleagues (2003) shows that patients diagnosed with dissociative identity disorder suffer from increased rates of nightmare disorders compared to people without a dissociative identity disorder. Other studies (Giesbrecht et al., 2006; Giesbrecht & Merckelbach, 2004; Watson, 2001) have related dissociative symptoms in students to self-reported sleep abnormalities, such as flying dreams or hypnagogic hallucinations. There is also anecdotal evidence that symptoms in patients with depersonalization disorder become worse when they feel exhausted (Simeon & Abugel, 2006). The effect also seems reversible, improvements in sleep quality led to a reduction of dissociative symptoms in a group of over 200 clinical inpatients

(Van der Kloet et al., 2012). These findings support Koffel and Watson's (2009) hypothesis that disruptions in the sleep–wake cycle lead to intrusions of sleep phenomena into waking consciousness, resulting in dissociative experiences. Dissociative disorders have the reputation of being refractory to behavioural therapy and pharmacological interventions such as fluoxetine (Lilienfeld, 2007). It is therefore important to explore other potential treatments for dissociative symptoms. A possible alternative treatment for dissociative symptoms may be sought in the domain of mindfulness treatment. Mindfulness is related to specific levels of awareness that can be developed through meditation. The specific level of awareness is enhanced through concentration on a specific stimulus, for example breathing, while being in the present moment and being nonjudgmental to this unfolding experience (Kabat-Zinn, 2003). According to various studies (e.g., Ong, Shapiro & Manber, 2008; Howell, Digdon & Buro, 2010), mindfulness proves to be effective in improving sleep and might thus potentially be associated with a decrease of dissociative symptoms.

Given previous studies (Ong et al., 2008; Howell et al., 2010), we expect MBT to be positively associated with an improvement in sleep quality. Furthermore, we hypothesize that this improved sleep quality will be positively related with a decrease of dissociative symptoms.

Method

Participants and procedure

92 Clients (16 men, 73 women, 3 unidentified) of 'Indachtig' participated in the study. Their mean age was 45.5 years (*SD* = 10.56; range = 24 to 74 years). Marital status was subdivided into single (18%), living together (14%), married (57%), or divorced (6%).

MBT consists of a combination of Mindfulness Based Cognitive Therapy (MBCT) and Mindfulness Based Stress Reduction (MBSR), and entails a structured program of eight weekly sessions of 2.5 hours in which mindfulness exercises are combined with behavioral therapy interventions. The training took place at 'Indachtig', a privately owned health care facility and was administered by a licensed psychologist. Participants are trained to use these strategies to cope with unwanted conditions (Ong et al., 2008; Ong & Sholtes, 2010). Psycho-education is also a part of the therapy in which participants receive information, talk about earlier notice of alarm bells, about the role of cognitions, and the relations between cognition, emotion, behavior, activation, self-care and acceptance. After each session, a homework form is completed by the participants (Segal, Williams &

Teasdale, 2004). The participants spend every day 30 to 60 minutes of their time to exercises (body exploration, meditation, yoga).

The participants were allowed a two-week waiting period to decide to participate in the study. After registration, the participants were screened by an experienced psychologist with an intake interview in order to exclude severe psychopathology (e.g., psychotic disorders, suicidal ideation), before they were enrolled in the study. At the start of the study, 46 participants had been using medication in the previous three months, and 40 participants were using medication at that present time. At the end of the study, these numbers had dropped to 32 participants using medication at that time.

Before the start of the mindfulness training, participants were provided with oral and written information about the study after which they gave written informed consent and completed a test battery of questionnaires (baseline) at home. After 8 weeks, when the training was finished, they completed the test battery at home again (follow-up). After completion participants received a small monetary award and a written debriefing.

Measures

All questionnaires were validated Dutch translations of the original English questionnaires and have been commonly used in previous studies (Giesbrecht & Merckelbach, 2004; 2006, Giesbrecht et al., 2007; Van der Kloet et al., 2012).

The *Iowa Sleep Experiences Survey* (ISES; Watson, 2001, Cronbach's alpha baseline = .86, follow-up = .88) consists of 18 items requiring participants to evaluate the frequency of diverse sleep- and dream experiences on a 7-point Likert scale (range: 1 = never, 7 = several times a week). Examples of items of the ISES are 'If I lie in bed, I feel the presence of someone who is not there' or 'I experience intense, dreamlike scenes when I fall asleep'. Watson (2003) attained support for the convergent validity of the ISES.

The *SLEEP-50* (Spoormaker et al., 2005, Cronbach's alpha = .86, and .65, respectively) is a 50-items scale which measures sleep experiences in the past four weeks on a 4-point Likert scale (*range*: 1 = never, 4 = very often). The SLEEP-50 is able to identify most prevalent DSM-IV-TR sleep disorders and is a practical universal sleep questionnaire attending the intensity of sleep complaints. Examples of items entail 'During the night I feel little shocks into my legs' or 'I would like to have more energy during the day'. Spoormaker and colleagues (2005) have displayed high internal consistency, good test-retest reliability, and good preliminary predictive validity for the SLEEP-50.

The *Dissociative Experiences Scale* (DES; Bernstein & Putman, 1986, Cronbach's alpha = .89, and .92, respectively) is a 28-items self report questionnaire that asks the participant to estimate the frequency of various dissociative experiences on

100-mm visual analogue scales (VAS), ranging from 0 (*never*) till 100 (*always*). Example items are 'Some people have the experience of finding themselves dressed in clothes that they don't remember putting on' or 'Some people have the experience of looking in a mirror and not recognizing themselves'. Bernstein and Putnam (1993) have validated the construct validity of the DES as satisfying.

The *Brief Symptom Inventory* (BSI; Derogatis & Melisaratos, 1983, Cronbach's alpha = .96, and .96, respectively) is a 53-items scale that assesses experiences of psychopathological symptoms in the past month. The BSI uses a 5-point Likert scale (*range*: *0 = not at all, 4 = very much)* to indicate to what extent participants experienced a particular problem during the past week including today (de Beurs & Zitman, 2006). Examples of items of the BSI are 'How much did you suffer from 'feeling sad' or 'suicidal thoughts'. The convergent and divergent validity of the BSI supports the satisfying validity of most scales, and the discriminant validity of the scales is sufficient. As screener for psychopathology in the population, the BSI proves to be excellent (de Beurs & Zitman, 2006).

The *Kentucky Inventory of Mindfulness Skills* (KIMS; Baer, Smith & Allen, 2004, Cronbach's alpha = .79, and .83, respectively*)* is a 39-items questionnaire which measures the mindfulness skills of the participants on a 5-point Likert scale (*range*: 1 = never or very rarely true, 5 = very often or always true). Examples items of the KIMS are 'I notice changes in my body, such as whether my breathing slows down or speeds' or 'I tell myself that I should not think the way I think'. The content validity of the KIMS has high ratings of item clarity and representation of mindfulness skills (Baer et al., 2004).

Results

Baseline measures were completed by *N* = 92 participants, follow-up measures were completed by *N* = 67 participants (drop-out rate: 27%). Independent samples t-tests revealed no significant differences between the completers and drop-outs on the baseline measures (*p* = .16 - .95). Table 9.1 displays the mean scores and standard deviations of ISES, SLEEP-50, DES, BSI and KIMS at baseline and follow-up, as well as paired samples t-tests. Participants rated the subjective quality of their sleep with a grade (0-10 points), which significantly improved from a mean 6.8 score (*SD* = 1.39) to a grade of 7.2 points (*SD* = 1.26) after the mindfulness training, *t* = 2.04, *p* = .04. Their mean hours asleep during the night remained approximately the same; a mean of 7.38 hours (*SD* = 1.10) at baseline, and a mean of 7.29 hours (*SD* = 1.05) at follow-up. Furthermore, there was a significant improvement in sleep quality (SLEEP-50) after MBT, as well as a decrease of psychopathology. No significant decrease was found for unusual sleep experiences (ISES), nor

dissociative symptoms (DES), and mindfulness skills did not seem to have improved significantly, see also table 9.1.

Table 9.1.
Mean scores and paired samples t-tests of variables at baseline (*N* = 92) and follow-up (*N* = 69).

	Mean (SD)	*t*	*p (2-tailed)*
ISES		1.12	.27
BL	42.30 (13.73		
FU	41.22 (13.22)		
SLEEP-50		3.21	< .01
BL	79.67 (16.45)		
FU	75.07 (16.51)		
DES		1.45	.15
BL	15.40 (8.39)		
FU	14.80 (9.03)		
BSI		4.79	< .01
BL	52.33 (32.51)		
FU	38.38 (28.70)		
KIMS		.36	.72
BL	116.42 (12.46)		
FU	115.90 (12.95)		

Note. ISES = Iowa Sleep Experiences Survey; DES = Dissociative Experiences Scale; BSI = Brief Symptom Inventory; KIMS = Kentucky Inventory of Mindfulness Skills; BL = baseline measures; FU = follow-up measures.

Discussion

The present study aimed to explore whether participation during 8 weeks MBT training would be effective in improving sleep quality and in turn lead to a decrease in dissociative symptoms. Our findings can be catalogued as follows. First, MBT improved sleep quality and reduced general psychopathology. Second, MBT did not prove effective through the increased adequacy in mindfulness skills. Third, dissociative symptoms were not decreased after MBT.

MBT led to improved sleep quality and reduction in psychopathological symptoms, which is in line with other published studies, showing that mindfulness proves effective in improving sleep (Ong et al., 2008; Howell et al., 2010) and reduces treating anxiety and mood disorders in the clinical population and prevents patients from relapse (Hofmann, Sawyer, Witt, & Oh, 2010; Zindel, Williams & Teasdale, 2002).

Mindfulness exercises aim to develop such skills as inner calm, emotional control, perseverance, and a strong sense of self. Interestingly, MBT did not prove effective through the increased adequacy in mindfulness skills (as measured by

KIMS). Thus, mindfulness skills may not have become automatized in participants due to the relatively limited time frame of the intervention and thus, would not have been measurable. This leads to speculation about what factors in MBT did lead to the improvement of sleep quality and decrease of general psychopathology. Possibly, these improvements are due to the cognitive behavioral aspects of MBT, such as the focus on changing maladaptive thinking. In MBT, changing one's relationship to the maladaptive thinking rather than changes in thinking itself may be cause of the positive changes in affect and behavior.

Strikingly and in contrast to the improvements in general psychopathology and sleep, we were unable to find support for our hypothesis that sleep improvement after MBT would lead to a decrease of dissociative symptoms. One might speculate that this specific pattern of findings can be explained by previous studies who have found dissociation related to a specific subset of sleep problems, namely unusual sleep experiences (Giesbrecht et al., 2006; Giesbrecht & Merckelbach, 2004; Watson, 2001, Van der Kloet et al., 2012) and only to a lesser extent to sleep problems in general. In the present study, sleep quality (as measured by SLEEP-50) improved, unusual sleep experiences (as measured by ISES) did not decrease after MBT. Likely, the pathway from sleep improvement to decrease of dissociative symptoms proves to be more complex in the sense that specifically unusual sleep experiences should be changed in order to change dissociative symptomatology. Another interpretation of our null finding could lie in the possible adverse effects of MBT. Although MBT seems appropriate for most people, some adverse effects have been reported (Dobkin, Irving, & Amar, 2011). Specifically, Manocha (2000) states that meditation is contraindicated for people suffering from psychosis. Furthermore, Shapiro (1992) found that in 27 long-term meditators 7.4% of them experienced profound adverse effects, such as: paradoxical increases in tension, relaxation-induced anxiety and panic, less motivation in life, boredom, pain, impaired reality testing, confusion and disorientation, feeling 'spaced out', depression, increased negativity, being more judgmental and feeling 'addicted to meditation' (Shapiro, 1992). Finally, Perez-de-Albeniz and Holmes (2000) cite early work from the 1980s describing mild dissociation as one of the adverse effects of MBT. Possibly, MBT is not useful in reducing dissociation, as dissociative phemomena may arise as its side effects.

Some limitations to this study merit mention, including the absence of a control group, and the reliance on self-report measures. This leaves the possibility that the observed effects are due to regression towards the mean and spontaneous recovery cannot be ruled out. Although this study heavily relied on self-reports, increasing the risk at shared method variance, standardized measurement instruments with good psychometric qualities (validity and reliability) were used. Future studies should examine the possibilities of assessment by multiples, such as structured clinical interviews. The MBT was applied by a single therapist, which

may have advantages as well as disadvantages. We excluded the possibility of reliability and confounding issues and standardize treatment by using only one therapist. However, it could prove beneficial for future research to select multiple therapists and have objective evaluators assess the therapists' functioning. This would also allow for the assessment of common factors in therapy, such as empathy or the work relation. Finally, there may be other variables that could be underlying the relation between sleep, dissociation and MBT which were not evaluated. For example, variables such as daily mood, attention, memory and fantasy proneness should be considered in future studies (Soffer-Dudek & Shahar, 2011).

Despite its limitations, the present study provides new insights in the beneficial effect of MBT on general psychopathology and sleep quality, but not on unusual sleep experiences, and dissociative symptoms. Many people suffer from dissociative symptoms and it is therefore important to explore potential alternative treatments other than MBT.

Conflict of interest

Angelique Didderen is working at 'Indachtig', a privately owned health care facility, which provides Mindfulness Based Therapy (MBCT/MBSR). She is a member of the Dutch Association for Mindfulness trainers (Nederlandse Vereniging van Mindfulnesstrainers), and a licensed ACT (Acceptance Commitment Therapy) trainer.

CHAPTER X[16]

Sleep normalization and decrease in dissociative experiences: Evaluation in an inpatient sample

16 This chapter is an adapted version of the following article:
Van der Kloet, D., Lynn, S.J., Giesbrecht, T., Merckelbach, & de Zutter, A. (2012). Sleep normalization and decrease in dissociative experiences: Evaluation in an inpatient sample. *Journal of Abnormal Psychology, 121*, 140-150.

Summary

We conducted a longitudinal study to investigate the relation between sleep experiences and dissociative symptoms in a mixed inpatient sample at a private clinic evaluated on arrival and at discharge 6 to 8 weeks later. Using hierarchical regression analyses and structural equation modeling, we found a link between sleep experiences and dissociative symptoms and determined that specifically *decreases* in narcoleptic experiences rather than insomnia accompany a *reduction* in dissociative symptoms. Although sleep improvements were associated with a general reduction in psychopathology, this reduction could not fully account for the substantial and specific effect that we found for dissociation. Our findings are consistent with Watson's (2001) hypothesis that disruptions in the sleep-wake cycle lead to intrusions of sleep phenomena into waking consciousness, resulting in dissociative experiences. Accordingly, sleep hygiene may contribute to the treatment or prevention of dissociative symptoms.

Introduction

For decades, clinicians and researchers have studied dissociative symptoms (e.g., depersonalization, derealization, memory lapses, absorption) in a systematic fashion. However, a consensus about their genesis remains elusive. Because of their dream-like character, some authors have recently pointed to and discerned a possible link between dissociative symptoms and sleep (Watson, 2001; 2003). Relying on two large non-clinical samples, Watson (2001) found that dissociation, as measured by two validated dissociation scales (Bernstein & Putnam, 1986), correlates with unusual sleep- and dream-related experiences (Watson, 2001; 2003a). Based on these findings, Watson argued that dissociation, schizotypy, and certain sleep experiences map onto a common domain that encompasses unusual perceptions and cognitions. Watson (2001) referred to the continuity in unusual perceptions and cognitions across the day and night as 'cross-state continuity.'

Several laboratories have replicated Watson's finding of an association between unusual sleep experiences and dissociation (e.g., Fassler et al., 2006; Giesbrecht & Merckelbach, 2004; 2006; Soffer-Dudek & Shahar, 2009). In a review of 23 studies, van der Kloet, Merckelbach, Giesbrecht, and Lynn (2012) concluded that the extant research provides strong support for a link between dissociative experiences and a labile sleep-wake cycle that is evident across a range of phenomena, including waking dreams, nightmares, and hypnagogic and hypnopompic hallucinations. Studies that offered evidence for a link between dissociative experiences and sleep disturbances relied on clinical and nonclinical samples, and, with only one exception (Hartman, Crisp, Sedgwick, & Borow, 2001), yielded correlations in the range of .30-.55. Similarly, Agargun and his colleagues (Agargun et al., 2003a) tested an undergraduate sample and found that chronic nightmare sufferers scored higher on dissociation, compared with controls. These authors also reported an increased prevalence of nightmare disorder among patients with Dissociative Identity Disorder (Agargun et al., 2003b). The link between unusual sleep experiences and dissociative tendencies is further illustrated by dissociative phenomena (e.g., out-of-body experiences), which often accompany hypnagogic and hypnopompic hallucinations (Girard & Cheyne, 2004).

Disruptions in sleep patterns figure prominently in mood and anxiety disorders, schizophrenia, and borderline personality disorder (Benca et al., 1992; Morin & Ware, 1996), with fairly specific associations among discrete sleep complaints and forms of psychopathology (Koffel & Watson, 2009a). For example, insomnia and tiredness appear to be primarily associated with depression and anxiety, whereas unusual sleep experiences (e.g., hypnagogic hallucinations) appear to be primarily related to dissociative symptoms (Koffel & Watson, 2009a). According to factor analytic research, dissociation and schizotypy are more strongly correlated with unusual sleep experiences (i.e., as measured by the *Iowa Sleep Experiences Survey*

(ISES)), than they are correlated with mood and anxiety (Koffel & Watson, 2009b). Koffel and Watson (2009b) aptly concluded that "unusual sleep experiences (e.g., nightmares, vivid dreams, narcolepsy symptoms) are associated with symptoms of dissociation in both clinical and non-clinical samples" (Koffel & Watson, 2009b, p.557).

Although researchers have consistently found a robust correlation between dissociative and sleep experiences, studies have generally relied on cross-sectional designs. To arrive at meaningful causal conclusions, we (Giesbrecht et al., 2007) deprived 25 healthy volunteers of one night of sleep and determined that sleep loss engenders a substantial increase in dissociative symptoms. Importantly, this increase could not be attributed to mood or response bias.

Researchers have not, as yet, examined whether promoting healthy sleeping reduces dissociative symptoms (but see chapters VIII and IX). If such results were secured in a clinical sample, it would generalize previous findings and have important implications for understanding and treating dissociative symptoms. The current study represents the first prospective study to evaluate the hypothesis that improving sleep results in a decrease in dissociative experiences, and the first prospective test of the hypothesis that unusual sleep experiences are associated with dissociation, whereas insomnia is more reliably associated with anxiety/depression (see Koffel & Watson, 2009a).

We evaluated a mixed sample of inpatients treated for 6 to 8 weeks in a private clinic that emphasizes sleep hygiene as a core treatment component, and anticipated that participants with the greatest sleep improvement at retest would display the strongest decrease in dissociative experiences. Moreover, we evaluated whether anxiety and depression could account for the amelioration of dissociative experiences pre-post treatment: previous studies have revealed a connection between dissociation and anxiety/depression (Sierra, Senior, Dalton, McDonough, Bond, Philips, et al., 2002; Giesbrecht, Merckelbach, van Oorsouw, & Simeon, 2010), as well as a connection between both anxiety and arousal and arousal and sleep (Giesbrecht et al., 2010). Finally, we included a measure of childhood traumatic experience. We anticipated that this measure would be related to dissociation levels (e.g., Gast, Rodewald, Nickel, & Emrich, 2001), but that it would remain stable over time.

Method

Participants

Participants were 266 inpatients (132 men, 113 women, 21 not recorded; mean age: 44.2 years, *SD* = 11.5; range: 18 – 74 years) admitted for 6 to 8 weeks to U-

Center in Epen, The Netherlands. U-Center is a private clinic with an eclectic treatment approach. Seventy-one participants did not complete treatment as a result of leaving the clinic prematurely on a voluntary basis or being referred to other clinics (e.g., university hospital) due to somatic or psychiatric complications. Completers versus noncompleters did not differ on any of the baseline measures (see below) (e.g., DES, BAI, BSI, CTQ, and BDI-II; all *p*'s > .05), or with respect to age, gender, use of medication, or diagnosis (all *p*'s > .05).

A psychologist and resident psychiatrist collaborated to determine diagnoses based on test scores, clinical interviews, information from (medical) records and intake, and collateral information. Part of the sample (38%) used medication during the study, predominantly anxiolytics and antidepressants. Participants suffered from alcohol dependence (15%), medication dependence, especially sleep medication (18%), physical complaints (33%), depression (72%), anxiety (15%), burn-out (i.e., mixed anxiety and depression symptoms that fail to reach diagnosis threshold; 5%), ADHD (2%), psychotic symptoms (2%), identity problem (1%), difficulty stopping smoking (1%), and other complaints (12%). A comparison on the primary outcome measures of patients using medication and patients not using medication yielded no significant difference, justifying the inclusion of medication using participants in the analyses.

Procedure

Participants were informed that the scales were part of routine diagnostic testing and that data would be used for study purposes, after which they gave written informed consent. Participants completed questionnaires in a set order during their first days at the clinic (baseline), and 195 participants completed the same measures the day before discharge (follow-up). At baseline and follow-up, computerized measures were completed via the user-friendly software program EMIUM (Janssen, 2008). Participants received instructions from a psychologist who explained how to use the program, and who was available to answer questions. After the baseline measures, patients received Therapy As Usual (TAU) for 6 to 8 weeks. TAU comprised individual and group therapy and included cognitive behavioral therapy (CBT), mindfulness, daily fitness exercises, and creative work. Importantly, the clinic actively encouraged sleep hygiene practices and rules, considered to promote good sleep regulation (Costa e Silva, 2006). Patients were awakened in the morning, denied access to their room during the day to preclude napping, and returned to their rooms at the same time every night. Patients had no access to alcoholic beverages, and caffeine in the evening and night was not permitted. Fitness activities were restricted to the morning and participants had access to relaxing activities such as massages and sauna in the evening. Afternoons

included healthy outdoor activities. Staff, therapists, and patients were naive with respect to the hypotheses under study.

Measures

Dissociative Experiences Scale (DES; Cronbach's α baseline = .94, follow-up = .94; Bernstein & Putnam, 1986; Dutch version: Boon & Draijer, 1995). The DES is a self-report scale that requires participants to indicate on 100 mm visual analogue scales (anchors: 0 = never; 100 = always) to what extent they experience 28 dissociative experiences in daily life. Van IJzendoorn and Schuengel (1996) provide meta-analytic evidence for the sound psychometric properties of the DES. Following the three-factor solution proposed by Carlson and colleagues (1991), in addition to the DES total score, we calculated subscale scores for amnesia, absorption and imaginative involvement, and depersonalization/derealization. Furthermore, we examined the subset of 8 DES items that constitute the so-called DES-Taxon (DES-T; Waller et al., 1996), which tap the pathological symptoms of dissociation (e.g., depersonalization and amnesia). Following Waller et al. (1996), we created a dichotomous measure of taxon-membership versus nontaxon-membership; patients with a taxon probability exceeding .90 were assigned to the DES-T group. Psychometric shortcomings notwithstanding (Watson, 2003b), the DES-T has been considered a useful measure in the dissociation field (Simeon, Knutelska, Nelson, Guralnik, & Schmeidler, 2003).

SLEEP-50 (Cronbach's α baseline = .84, follow-up = .93; Spoormaker et al., 2005)**.** Sleep experiences were assessed with subscales of the 50-item Dutch version of the SLEEP-50, which index sleep complaints and sleep disorders listed in DSM-IV (APA, 2001): sleep apnea (Cronbach's α baseline: .58, follow-up: .69, change score: .52), insomnia (Cronbach's α baseline: .87; follow-up: .88; change score: .76), restless legs (Cronbach's α baseline: .71; follow-up: .81; change score: .45), circadian rhythm sleep disorder (Cronbach's α baseline: .56; follow-up: .55; change score: .27), sleep walking (Cronbach's α baseline: .65; follow-up: .84; change score: .30), nightmares (Cronbach's α baseline: .84; follow-up: .90; change score: .93), factors influencing sleep (Cronbach's α baseline: .81; follow-up: .82; change score:.53), the impact of sleep complaints on daily functioning (Cronbach's α baseline: .66; follow-up: .70; change score: .72), and narcolepsy (Cronbach's α baseline: .51; follow-up:.73; change score: .61). Each item is scored on a 4-point Likert scale ranging from 0 (not at all) to 3 (very much). Spoormaker et al. (2005) have demonstrated adequate test-retest reliability for the Sleep-50 total score (*r* = .78). The SLEEP-50 narcolepsy subscale covers unusual sleep phenomena, including hypnagogic imagery and excessive daytime sleepiness that overlap with the ISES general subscale (Koffel & Watson, 2009b). The use of the SLEEP-50 provides the

opportunity to test predictions derived from Koffel and Watson (2009b) pertaining to unusual experiences versus insomnia.

Brief Symptom Inventory (BSI; Cronbach's α baseline = .97, follow-up = .97; Boulet & Boss, 1991). The 53-item BSI assesses general symptoms and complaints experienced by people with psychiatric problems. Although the BSI comprises nine subscales, analyses were based on the total score. Items are scored on a 5-point Likert scale (anchors: 0 = not at all, to 4 = extremely). The Dutch version of the BSI has good convergent and divergent validity and has proven to be a useful outcome measure for therapy efficiency (de Beurs & Zitman, 2006).

Beck Anxiety Inventory (BAI; Cronbach's α baseline = .93, follow-up = .92; De Ayala, Vonderharr-Carlson, & Doyoung, 2005). The BAI is a 21-item widely used self-report measure of anxiety symptoms. Each item is scored on a 4-point Likert scale (anchors: 0 = not at all bothered by this symptom; 3 = severely bothered by this symptom). The range of total scores is 0 to 63, with higher scores indicating more anxiety symptoms. The BAI has high internal consistency (Cronbach's α = .93) and modest test-retest reliability (*r* = .66; Kelett, Beail, Newman, & Frankis, 2003).

Beck Depression Inventory II (BDI-II; Cronbach's α baseline = .92, follow-up = .93; Sprinkle, Lurie, Insko, Atkinson, Jones, Logan et al., 2002; Dutch version: Van der Does, 2002). The BDI–II is a homogeneous measure of depressive symptoms comprising 21 items. Each item is scored on a 4-point Likert scale ranging from 0 (not at all bothered by this symptom) to 3 (severely bothered by this symptom). The range of total scores is 0 to 63, with higher scores reflecting more depressive symptoms. The BDI has high test-retest reliability (*r* = .96), and convergent validity with the Structured Clinical Interview for DSM Disorders (SCID-I) is good (*r* = .83; Sprinkle et al., 2002).

Childhood Trauma Questionnaire (CTQ; Cronbach's α baseline = .90, follow-up = .52; Bernstein et al., 2003). The CTQ is a widely used self-report scale of traumatic childhood events, such as emotional, physical, and sexual abuse, and emotional and physical neglect. In the present study, we employed the 25-item short form scored on 5-point scales anchored 1 (never) and 5 (very often). The Dutch version of the CTQ possesses satisfactory psychometric properties (Thombs et al., 2009).

Results

Individual differences measures

Statistical analyses were performed using SPSS 18.0 software. Table 10.1 shows mean scores of all measures at baseline and follow-up. With exception of the CTQ (i.e., self-reported childhood trauma experiences), paired-samples *t*-tests revealed significant decreases across the two time points for all measures. This pattern

supports our hypothesis that TAU would lead to an improvement in sleep quality as measured by the SLEEP-50 subscales, as well as a general decrease in psychopathology as measured by the DES, BSI, BAI, and BDI-II. For instance, of the completers, 46 out of 195 participants (24%) displayed dissociation levels exceeding the clinical cut-off for dissociative disorders (i.e.; >30; Bernstein-Carlson & Putnam, 1993) at baseline. This number was reduced to 24 (12%) at follow-up (Fisher's exact p = .005). Table 10.2 presents the Pearson product-moment correlations between all psychopathology and dissociation measures at baseline and follow-up, as well as the correlations among change scores.

Table 10.1.
Mean scores at baseline and follow-up and *t*-statistics of inpatient sample (*N* = 195).

Measure	Baseline *M* (*SD*)	Follow-up *M* (*SD*)	Mean difference (*SD*)	*t* (df = 193-198)
DES total	20.86 (15.32)	13.88 (12.76)	6.98 (10.96)	8.93**
Amnesia	13.82 (12.77)	8.27 (11.24)	5.54 (12.47)	6.24**
Absorption	28.32 (19.24)	19.88 (17.36)	8.44 (13.84)	8.56**
Depersonalization/Derealization	14.13 (17.46)	7.86 (12.14)	6.27 (12.71)	6.93**
SLEEP-50 subscales				
Sleep apnea	4.45 (2.89)	2.91 (2.58)	1.54 (2.41)	8.92**
Insomnia	11.01 (6.02)	6.52 (5.24)	4.49 (5.03)	12.45**
Narcolepsy	1.99 (2.01)	1.43 (2.09)	0.56 (2.08)	3.76**
PLMDª	1.65 (2.08)	1.13 (1.89)	0.52 (1.57)	4.61**
Circadian Rhythm	1.75 (1.84)	0.83 (1.35)	0.92 (1.61)	7.93**
Sleep walking	0.20 (0.70)	0.09 (0.50)	0.11 (0.69)	2.18*
Nightmares	2.28 (2.37)	2.00 (2.59)	0.28 (2.79)	1.42
Factors influencing sleep	1.18 (2.30)	1.22 (4.05)	-0.04 (3.04)	-0.19
Impact sleep complaints on daily functioning	9.23 (4.05)	4.30 (3.32)	4.92 (3.94)	17.39**
BSI subscales	63.29 (37.14)	28.55 (26.81)	34.74 (32.14)	15.31**
Paranoid ideation	5.23 (4.16)	2.57 (2.92)	2.66 (3.41)	10.97**
Psychoticism	6.28 (3.99)	2.86 (2.88)	3.41 (3.71)	12.94**
Somatization	6.14 (5.21)	2.78 (3.41)	3.36 (4.30)	11.00**
Obsession-compulsion	9.72 (5.99)	4.55 (4.47)	5.18 (4.96)	14.68**
Interpersonal sensitivity	5.35 (4.10)	2.54 (2.75)	2.82 (3.53)	11.22**
Depression	9.80 (5.96)	4.08 (4.73)	5.72 (5.64)	14.27**
Anxiety	8.51 (5.87)	3.88 (4.40)	4.63 (5.10)	12.75**
Hostility	3.75 (4.35)	1.78 (2.37)	1.97 (3.88)	7.14**
Phobic anxiety	4.81 (4.31)	1.95 (2.46)	2.85 (3.62)	11.09**
BAI	39.48 (12.05)	30.49 (8,38)	8.99 (10.27)	12.35**
BDI-II	26.86 (11.95)	10.89 (10.10)	15.97 (11.01)	20.46**
CTQ	20.85 (14.32)	21.23 (13.97)	-0,38 (5.56)	-0.97

Note. DES = Dissociative Experiences Scale; BSI = Brief Symptom Inventory; BAI = Beck's Anxiety Inventory; BDI-II = Beck's Depression Inventory II; CTQ = Childhood Trauma Questionnaire; ª PLMD = Periodic Leg Movement Disorder.
* $p < .05$ (2-tailed),
** $p < .01$ (2-tailed)

Table 10.2.
Pearson product-moment correlations between dissociation, psychopathology composite, and childhood trauma at baseline, follow-up, and change scores.

	DES Total	DES Am	DES Ab	DES Dep	BSI	BAI	BDI-II	Psychop.	CTQ
	Baseline	*Baseline*	*Baseline*	*Baseline*	*Baseline*	*Baseline*	*Baseline*	*Baseline*	*Baseline*
	FU	*FU*	*FU*	*FU*	*FU*	*FU*	*FU*	*FU*	*FU*
	Change	*Change*	*Change*	*Change*	*Change*	*Change*	*Change*	*Change*	*Change*
DES total									
Baseline	*0.71***	-	-	-	-	-	-	-	-
FU									
Change									
DES-Am									
Baseline	0.86**	*0.59***	-	-	-	-	-	-	-
FU	0.86**								
Change	0.82**								
DES-Ab									
Baseline	0.93**	0.68**	*0.72***	-	-	-	-	-	-
FU	0.93**	0.67**							
Change	0.86**	0.51**							
DES-Dep									
Baseline	0.86**	0.65**	0.74**	*0.69***	-	-	-	-	-
FU	0.80**	0.65**	0.65**						
Change	0.80**	0.58**	0.60**						
BSI									
Baseline	0.42**	0.29**	0.41**	0.39**	*0.54***	-	-	-	-
FU	0.51**	0.35**	0.50**	0.42**					
Change	0.35**	0.33**	0.29**	0.28**					
BAI									
Baseline	0.35**	0.21**	0.36**	0.35**	0.78**	*0.55***	-	-	-
FU	0.40**	0.22**	0.41**	0.38**	0.84**				
Change	0.30**	0.26**	0.28**	0.25**	0.72**				
BDI-II									
Baseline	0.38**	0.26**	0.31**	0.33**	0.84**	0.63**	*0.51***	-	-
FU	0.39**	0.27**	0.36**	0.33**	0.89**	0.76**			
Change	0.29**	0.27**	0.26**	0.21**	0.77**	0.57**			
Psychop									
Baseline	0.43**	0.28**	0.43**	0.40**	0.96**	0.88**	0.90**	*0.56***	-
FU	0.46**	0.28**	0.45**	0.40**	0.97**	0.92**	0.93**		
Change	0.35**	0.32**	0.31**	0.28**	0.93**	0.86**	0.88**		
CTQ									
Baseline	0.35**	0.27**	0.31**	0.37**	0.35**	0.24**	0.33**	0.33**	*0.93***
FU	0.34**	0.24**	0.30**	0.36**	0.37**	0.29**	0.31**	0.36**	
Change	0.16**	0.10	0.10	0.14	0.16*	0.04	0.16*	0.14	

Note. Baseline: *N* = 256; follow-up: *N* = 201; DES-am = DES amnesia; DES-ab = DES absorption; DES-Dep. = DES depersonalization. The numbers in *italics* display the correlations of the measures between baseline and follow-up (FU).

* $p < .05$ (2-tailed);

** $p < .01$ (2-tailed)

Correlations between change scores of SLEEP-50 subscales, DES, and psychopathology composite

Given their high inter-correlations, BSI, BAI, and BDI-II were collapsed into one psychopathology composite by standardizing the baseline, follow-up, and change scores and summing the standardized values. Table 10.3 displays the Pearson product-moment correlations between the change scores of the SLEEP-50 subscales, change scores of DES, and psychopathology composite. Next, we tested whether the correlations between dissociation and sleep factors were different from the correlations with psychopathology. Whereas differences between the correlations of SLEEP-50 and DES, and SLEEP-50 and the psychopathology composite (BSI, BAI, BDI-II) did not reach significance for most subscales of the SLEEP-50, the correlation between psychopathology and the Insomnia subscale was significantly greater than the correlation between the DES and the Insomnia subscale.

Table 10.3
Pearson product-moment correlations between SLEEP-50 subscales, dissociation, and psychopathology composite (all **change** scores; *N* = 195) and differences between correlation coefficients.

	DES total	Psychopathology composite	Steiger's *z*	*p*
Sleep apnea	.15*	.15*	.05	>.05
Insomnia	.18*	.45**	3.50	<.01
Narcolepsy	.29**	.23**	-.84	>.05
PLMD	.12	.23**	1.36	>.05
Circ. rhythm	.14*	.24**	1.17	>.05
Sleep walking	.07	.13	.42	>.05
Nightmares	.01	-.06	-.89	>.05
Factors infl. sleep	.08	-.01	-1.15	>.05
Daily functioning	.26**	.47**	2.74	<.01

Note. * $p < .05$ (2-tailed), ** $p < .01$ (2-tailed)

Modeling mood as mediator of the dissociation-sleep connection

We determined whether the decrease in dissociative symptoms at follow-up is mediated by a reduction in psychopathology. According to this hypothesis, the relationship between dissociation and sleep scores should be eliminated when general psychopathology is statistically controlled. We subjected change scores to a hierarchical multiple regression analysis with dissociation (DES) as dependent

variable and the Narcolepsy, Insomnia, Nightmares, and Daily functioning subscales of the SLEEP-50, the psychopathology composite, and self-reported trauma (CTQ) as predictors. Only SLEEP-50 subscales with change scores that had Cronbach's α exceeding .60 were included. Because change scores tend to have lowered reliability, we chose a lower bound of acceptability than the commonly recommended Cronbach's α =.80. The analysis consisted of the following steps: First, we entered the SLEEP-50 subscales. Next, we entered other predictors (i.e., psychopathology composite and CTQ). Following this, we removed non-significant predictors by means of backwards elimination. We present a hierarchical decomposition in Table 10.4. Neither changes in insomnia, nightmares, or daily functioning scores could account for the decrease in dissociative symptoms at follow-up. Moreover, we employed a bootstrapping methodology (Preacher & Hayes, 2004) to determine whether the decline in dissociative symptoms is (partially) mediated by a decrease in general psychopathology. We used 10,000 bootstrap resamples of the data with replacement and found a significant mediation effect (bootstrap coefficient = .38, SE=.14), with a 95% confidence interval of .15 to .71 (significance indicated by the 95% confidence interval not crossing zero). Thus, part of the decrease in dissociation was explained by a decrease in narcoleptic symptoms due to a reduction in general psychopathology. However, the effects of sleep improvement on dissociation were only partly mediated and remained significant. We repeated this approach for all three DES subscales. The results are summarized in Table 10.5. Decrease in narcoleptic symptoms was a significant predictor in explaining the decrease in absorption, amnesia, and depersonalization. Again, this effect was partially mediated by a decrease in general psychopathology (i.e., psychopathology composite).

Finally, we conducted a logistic regression analysis with DES-T membership probability as dependent variable. We found a significant decrease in membership from baseline (n = 48; 24.61%) to follow-up (n = 19; 9.74%), Pearson $\chi^2 = 35.67$, $p < .001$. Change scores on Sleep-50 subscales and change in general psychopathology were entered as predictors. Improvements in narcolepsy explained most of the decrease in DES-T membership probability (B = .37, SE = .13, $p < .01$), with another part of the change in taxon membership being explained by the general psychopathology composite (B = .18, SE = .08, $p < .05$).

Table 10.4.
Summary of hierarchical multiple regression analysis on the Dissociative Experiences Scale (all **change** scores, *N* = 195).

Step	B	*ß*	*t*	*p*	*r*	r^2
1						
Insomnia	-.05	-.02	-.29	.78	.43	.19
Narcolepsy	1.08	.20	2.72	.01*		
Nightmares	.08	.02	.29	.78		
Daily function.	.11	.04	.47	.64		
Psychopathology	1.21	.29	3.65	<.001*		
CTQ	4.85	.10	1.41	.16		
2						
Narcolepsy	1.06	.20	2.72	.01*	.43	.19
Nightmares	.07	.02	.26	.80		
Daily function.	.10	.04	.43	.67		
Psychopathology	1.17	.29	3.80	<.001*		
CTQ	5.03	.10	1.49	.14		
3						
Narcolepsy	1.07	.20	2.77	.01*	.43	.19
Daily function.	.10	.04	.46	.65		
Psychopathology	1.16	.20	3.80	<.001*		
CTQ	4.87	.10	1.47	.14		
4						
Narcolepsy	1.14	.22	3.21	<.001*	.43	.19
Psychopathology	1.22	.30	4.41	<.001*		
CTQ	4.76	.10	1.44	.15		
5						
Narcolepsy	1.17	.22	3.31	<.001*	.42	.18
Psychopathology	1.27	.31	4.61	<.001*		

Note. Psychopathology = Psychopathology composite, consisting of total change scores on BAI = Beck's Anxiety Inventory, BSI = Brief Symptom Inventory, and BDI-II = Beck's Depression Inventory; CTQ = Childhood Trauma Questionnaire; Insomnia = SLEEP-50 subscale "insomnia"; Narcolepsy = SLEEP-50 subscale "narcolepsy"; Nightmares = SLEEP-50 subscale "nightmares", and Daily function. = SLEEP-50 subscale "Influence of sleep complaints on daily functioning".
* $p < .05$

Table 10.5.
Summary of hierarchical multiple regression analyses on the DES subscales (all **change** scores, *N* = 195).

Subscales DES	Step	B	*ß*	*t*	*p*	*r*	r^2
Amnesia	1						
	Insomnia	-.05	-.02	-.27	.80	.39	.15
	Narcolepsy	1.17	.20	2.59	.01*		
	Nightmares	.06	.01	.18	.86		
	Daily function.	.13	.04	.49	.62		
	Psychopathology	1.28	.28	3.40	<.001*		
	2						
	Insomnia	-.05	-.02	-.24	.81	.39	.15
	Narcolepsy	1.18	.20	2.61	.01*		
	Daily function.	.14	.04	.51	.61		
	Psychopathology	1.27	.27	3.42	<.001*		
	3						
	Narcolepsy	1.16	.19	2.61	.01*	.39	.15
	Daily function.	.12	.04	.48	.64		
	Psychopathology	1.24	.27	3.54	<.001*		
	4						
	Narcolepsy	1.24	.21	3.03	<.001*	.39	.15
	Psychopathology	1.32	.28	4.16	<.001*		
Absorption	1						
	Insomnia	-.09	-.03	-.41	.68	.36	.13
	Narcolepsy	.92	.14	1.80	.07		
	Nightmares	.28	.06	.81	.42		
	Daily function.	.18	.05	.61	.55		
	Psychopathology	1.44	.28	3.38	.01*		
	2						
	Narcolepsy	.89	.13	1.76	.08	.36	.13
	Nightmares	.26	.05	.76	.45		
	Daily function.	.16	.05	.54	.59		
	Psychopathology	1.38	.27	3.44	<.001*		
	3						
	Narcolepsy	.99	.15	2.12	.04*	.36	.13
	Nightmares	.28	.06	.83	.41		
	Psychopathology	1.47	.29	4.07	<.001*		
	4						
	Narcolepsy	1.05	.16	2.27	.02*	.35	.12
	Psychopathology	1.44	.28	4.04	<.001*		
Depersonalization	1						
	Insomnia	-.09	-.04	-.44	.66	.35	.12
	Narcolepsy	1.10	.18	2.34	.02*		
	Nightmares	-.08	-.02	-.23	.82		
	Daily function.	.19	.06	.68	.50		
	Psychopathology	1.13	.24	2.88	<.001*		
	2						
	Insomnia	-.10	-.04	-.48	.64	.35	.12
	Narcolepsy	1.09	.18	2.33	.02*		
	Daily function.	.18	.06	.66	.51		
	Psychopathology	1.14	.24	2.97	.03*		

Subscales DES	Step	B	*ß*	*t*	*p*	*r*	r^2
	3						
	Narcolepsy	1.05	.17	2.29	.02*	.35	.12
	Daily function.	.16	.05	.59	.56		
	Psychopathology	1.08	.23	.23	<.001*		
	4						
	Narcolepsy	1.15	.19	2.73	<.001*	.35	.12
	Psychopathology	1.18	.25	3.60	<.001*		

Note. Psychopathology = Psychopathology composite, consisting of total change scores on BAI = Beck's Anxiety Inventory, BSI = Brief Symptom Inventory, and BDI-II = Beck's Depression Inventory; CTQ = Childhood Trauma Questionnaire; Insomnia = SLEEP-50 subscale "insomnia"; Narcolepsy = SLEEP-50 subscale "narcolepsy"; Nightmares = SLEEP-50 subscale "nightmares", and Daily function. = SLEEP-50 subscale "Influence of sleep complaints on daily functioning".
* $p < .05$

Table 10.6.
Fit indices of the non mediation model (1), the partial mediation model (2), and the full mediation model (3).

	χ^2	*df*	NFI	CFI	PCFI	GFI	RMSEA	AIC	BCC	BIC
Model 1	80.03	33	.95	.97	.58	.93	.09	146.03	150.38	254.04
Model 2	57.37	32	.96	.98	.57	.95	.06	125.37	129.85	236.65
Model 3	70.80	33	.96	.98	.59	.94	.08	136.80	141.16	244.81

Note. NFI = Bentler-Bonett normed-fit-index; CFI = comparative-fit-index; PCFI = Parsimony Comparative Fit Index; GFI = goodness-of-fit index; RMSEA = root mean-square error of approximation; AIC = Akaike's Information Criterion; BCC = Browne-Cudeck Criterion; BIC = Bayesian Information Criterion.

Childhood trauma, improvement in sleep, and reduction in dissociation

We hypothesized that sleep improvement would lead to a reduction in dissociative symptoms. However, due to shortcomings associated with change scores (Peter, Churchill, & Brown, 1993), we tested three theoretically motivated mediation models using structural equation models (see Cole & Maxwell, 2003): 1) no mediation; decrease in narcoleptic symptoms leads directly to a decrease in dissociation, 2) partial mediation; decrease in narcoleptic symptoms leads to both decreases in dissociation and general psychopathology, but there is also a direct effect of decrease in narcoleptic symptoms on dissociation, and 3) full mediation; the effect of decrease in narcoleptic symptoms on dissociation is fully accounted for by a reduction in psychopathology. The analyses were conducted with AMOS 17 (Arbuckle, 2008).

In all three models, we used the psychopathology composite as a latent variable consisting of BAI, BSI, and BDI-II. The following fit indices were used: 1) the Bentler-Bonett normed-fit-index (NFI), the comparative-fit-index (CFI), the

goodness-of-fit index (GFI), and the root mean-square error of approximation (RMSEA). We assumed in line with Finch and West (1997) that the fit is acceptable if NFI, CFI, and GFI are .90 or greater and RMSEA values of .08 or less indicate adequate fit. Table 10.6 gives the fit indices for all three models. As can be seen, both the partial and the full mediation model (model 2 and 3) fulfilled all criteria for acceptable fit (see also Figure 10.1). However, Akaike's Information Criterion (AIC), the Browne-Cudeck Criterion (BCC), the Bayesian Information Criterion (BIC), and the Parsimony Comparative Fit Index (PCFI) indicated superior fit for model 2. That is, AIC, BCC, and BIC values were all numerically smaller for model 2. Unfortunately, the AIC, BCC, and BIC do not lend themselves to statistical testing (Barrett, 2007). Fortunately, model 1 and 3 are nested in model 2. Specifically, model 1 restricts model 2's connection between Psychopathology T1 and DES T1 to zero, while model 3 restricts Sleep T1 to DES T1 to zero. Therefore, we tested the differences between models by means of a χ^2-test (see, e.g., Schreiber, Nora, Stage, Barlow, & King, 2006). This test showed that model 2 is statistically superior to model 1 and 3 (both *p*'s < .01). Interestingly, in this model, self-reports of childhood trauma at baseline (CTQ) contributed longitudinally to narcolepsy at follow-up. Finally, a model we assessed with sleep as a potential mediator between psychopathology and dissociation proved to be nonsignificant with small effect size.

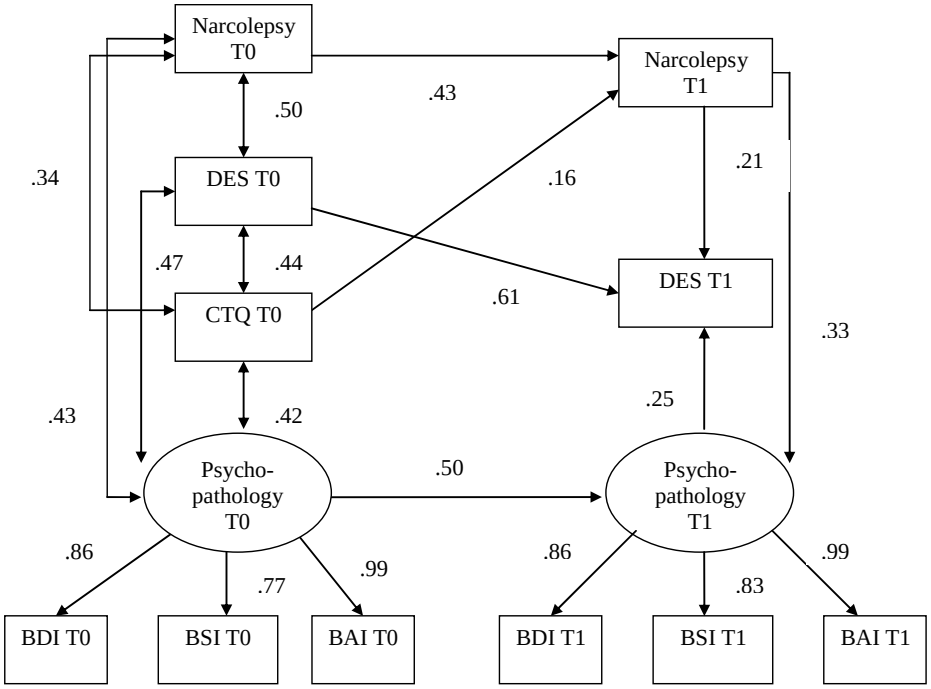


Figure 10.1.

The partial mediation model.

Structural equation modeling revealed that a partial mediation model best described the data. Reduction of dissociative symptoms was predicted by decrease in narcoleptic symptoms directly, as well as indirectly via improvement in general psychopathology. Note that self-reports of trauma at baseline only contributed longitudinally in the model as an influence on narcolepsy at follow-up.

T0 = Time point baseline; T1 = Time point follow-up; Narcolepsy = Narcolepsy subscale of SLEEP-50; DES = Dissociative Experiences Scale; BAI = Beck's Anxiety Inventory, BSI = Brief Symptom Inventory, BDI = Beck's Depression Inventory II, CTQ = Childhood Trauma Questionnaire.

Discussion

Our research replicates and extends previous findings and provides important insights regarding the relation between sleep and dissociation. More specifically, in a mixed inpatient sample, we replicated research showing a robust link between sleep experiences and dissociation (Watson, 2001; Giesbrecht & Merckelbach, 2004; 2006; Soffer-Dudek & Shahar, 2011). Our findings are in line with Giesbrecht et al.'s (2007) study in which sleep deprivation promoted dissociative experiences, an outcome entirely consistent with the hypothesis that disruptions in the sleep-wake cycle lead to intrusions of sleep phenomena into waking consciousness, resulting in dissociative experiences (Watson, 2001). Because disruptions in circadian rhythms exert detrimental effects on attentional control and memory, they may contribute to the attention deficits that are typically found in patients with dissociative disorders (Guralnik, Schmeidler, & Simeon, 2000; Guralnik et al., 2007).

Importantly, using a longitudinal design, we demonstrated that *improvements* in sleep quality and more specifically, decreases in narcoleptic/unusual sleep symptoms, accompany a *reduction* in dissociative experiences, including DES total scores, the three DES subscales, and the DES-T taxon membership. Interestingly, at baseline assessment, 24% of the patients who completed treatment exceeded the clinical cut-off for dissociative disorders (i.e.; >30; Bernstein-Carlson & Putnam, 1993); however, only 12% of the "completers" met this cut-off at follow-up. Similarly, when 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. Improvements in sleep were associated with a general reduction in psycho-pathological symptoms. However, this reduction could not account for the substantial and specific beneficial effect of the decrease in narcoleptic symptoms on dissociation. Although structural equation modeling revealed that narcoleptic symptoms are associated with both decreases in dissociation and general psychopathology, we also found tentative evidence for a specific link between narcoleptic symptoms and dissociation. Multiple regression analyses converged on the conclusion that changes in dissociation, as indexed by DES and taxon probability scores, could not be fully accounted for by global changes in psychopathology. Indeed, a substantial part of the decrease in dissociation was uniquely explained by improvement in sleep, and specifically by the decrease in narcoleptic symptoms. Furthermore, we found support for Koffel and Watson's (2009a) contention that insomnia appears to be associated with depression and anxiety, as measured by the psychopathology composite in our study. Changes in unusual sleep experiences/narcolepsy (e.g., vivid dreams, hypnopompic and hypnagogic hallucinations) were associated with dissociative symptoms as well as

anxiety/depression. However, the regression analyses demonstrated that the other Sleep-50 subscales were unable to explain further variance in dissociative symptoms over and above narcoleptic symptoms, which are a typical manifestation of unusual sleep experiences. In contrast, relative to changes in dissociation, changes in the general psychopathology composite were more strongly related to changes in insomnia from pre- to post-treatment.

Even though decreases in dissociation after treatment could not be fully accounted for in terms of reductions in global psychopathology, we did find that the association between sleep and psychopathology was not specific to dissociation. That is, other measures of psychopathology were, like dissociation, associated with sleep, a finding that is in keeping with the literature (e.g., Benca et al., 1992). Indeed, the measure of psychopathology was correlated not only with insomnia, but with narcolepsy symptoms as well; the latter correlation comparable to the correlation between dissociation and narcolepsy. However, when we controlled for changes in general psychopathology, narcoleptic experiences emerged as a predictor of absorption, amnesia, and depersonalization. Moreover, the fact that changes in narcoleptic symptoms were the most prominent predictor of changes in DES-T probability scores —associated with serious dissociative pathology — suggests that sleep may indeed impact serious dissociative pathology.

A widely held notion about the etiology of dissociative symptoms is that they serve a defensive function in that they help the individual to cope with traumatic memories (e.g., Gershuny & Thayer, 1999). One shortcoming of this conceptualization is that it remains silent as to how trauma contributes to dissociation. In contrast, the sleep-dissociation approach we evaluated suggests that traumatic experiences or the sequelae of trauma disrupt sleep, which contributes to or exacerbates dissociation. As in previous work (e.g., Gast et al., 2001), we found an association between self-reported trauma and dissociation. Finding a correlation between self-reported trauma and dissociation does not constitute proof of a relationship between objectively documented trauma and dissociation, and in no way implies a causal relationship (Giesbrecht et al., 2010). Although sleep did not emerge as a potential mediator between psychopathology and dissociation, highlighting the specificity of our partial mediation model, our structural equation modeling findings are consistent with a causal model in which trauma fuels sleep disturbances that in turn promote dissociation. Thus, we found childhood trauma (CTQ) at baseline to contribute longitudinally to narcoleptic symptoms at follow-up and thus indirectly to dissociation. Our findings suggest a role for trauma — mediated by sleep disturbances—in the genesis of dissociation, possibly by hampering recovery through its impact on sleep. If future studies replicate this pattern, it would provide a possible basis for a rapprochement between the posttraumatic and sociocognitive model of dissociation, which holds

that social and cognitive variables shape dissociative symptoms (Lilienfeld, Lynn, Kirsch, Chaves, Spanos, Ganaway et al., 1999; see also chapter I).

Our findings suggest that research on dissociation might benefit from the literature on the origins of and treatment options for narcoleptic symptoms (e.g., Scammell, 2003). Indeed, there exists an urgent need for fresh treatment ideas, as studies have found dissociative disorders to be recalcitrant to standard therapeutic approaches, including cognitive behavioral therapy and pharmacological interventions such as fluoxetine (Lilienfeld, 2007). Our findings also highlight the importance of sleep problems in clinical settings. The prevalence of sleep problems is often underestimated (Stores, 2007). Whereas they seldom are the primary focus of therapy, sleep problems are known to exert a decided impact on psychological well-being (Kumar, 2008), quality of life (Costa e Silva, 2006), and mental (Wu et al. 2008) and physical stability (Kalia, 2006).

Because sleep abnormalities are concomitants of various psychopathological conditions (Benca et al., 1992; Morin & Ware, 1996), it is not surprising that we found sleep problems to be prominently present in a heterogeneous inpatient sample. Importantly, we determined that a decline in psychopathological symptoms accompanied improvements in sleep. However, because our mixed patient sample was quite heterogeneous, our findings are not specific to a particular diagnostic entity and may not be necessarily generalizable to more discrete manifestations of well-diagnosed psychopathology (e.g., anxiety, depression). Future research should examine which patients benefit most from sleep hygiene programs to explore treatment options and to ascertain the possible role of sleep difficulties in diverse forms of psychopathology.

Before closing, a number of caveats in interpreting our findings merit mention. Although we employed a prospective longitudinal design, our findings may have been influenced by as yet unspecified confounding variables. Accordingly, it is necessary to interpret the direction of the relation between sleep problems and dissociation with caution. Measuring variables of interest over three or more points in time would allow researchers to more finely assess the temporal and causal links between dissociation and sleep problems. Nonetheless, the mere fact that we obtained a substantial connection between sleep problems and psychopathology—notably dissociative experiences — is clinically relevant and theoretically meaningful in terms of the sleep-dissociation hypothesis (Watson, 2001; 2003a; van der Kloet et al., 2012; see also chapter I).

One could argue that the decline in narcoleptic symptoms, dissociative experiences, and general psychopathological complaints reflect report bias. However, given the fact that CTQ scores remained stable, a report bias related to global demands for “positive reporting” over time is unlikely an adequate alternative account of our results. Our finding that narcoleptic symptoms predicted dissociation even when psychopathology was statistically controlled also argues

against a reporting bias interpretation. Moreover, staff, therapists, and patients were naive with respect to the hypotheses, further reducing sources of potential bias. Still, our research does not permit determination of which of the multi-faceted treatment components (e.g., sleep hygiene versus cognitive behavioral therapy) were responsible for symptom reduction and sleep improvement.

Because our study sampled participants at only two time-points, causal statements about the link between sleep and dissociation remain speculative. We suggest that future studies: (a) administer objective measures of dissociation and sleep on multiple occasions, (b) dismantle complex treatments, and (c) control for expectancies and motivation to identify efficacious treatment components and mechanisms of sleep hygiene. Furthermore, future studies should use the Iowa Sleep Experiences Survey (ISES; Watson, 2001) in addition to the SLEEP-50, as the ISES specifically taps unusual sleep experiences. We also recommend that future studies include psychometrically sound measures of quality of life (Krystal, Thakur, & Roth, 2008). Although we found that daily functioning was associated with dissociation and other indices of psychopathology, the measure most closely associated with quality of life was based on a single subscale of our sleep measure.

In closing, our study replicated and extended previous research and implicates sleep hygiene as a means of treating or preventing dissociative symptoms, as well as symptoms of psychopathology more broadly. Studies in which sleep hygiene variables and treatment components of sleep hygiene programs are manipulated, and dissociative and other symptoms of psychopathology are monitored over multiple time points, would be a next logical step. Ultimately, this line of research holds tremendous promise to contribute to our understanding of psychopathology in general, and dissociation in particular, and to the development of effective treatment interventions for people with a broad range of psychological disorders.

GENERAL DISCUSSION[17]

17 This chapter is in part an adapted version of:
Lynn, S.J, Lilienfeld, S.O., Merckelbach, H., Giesbrecht, T., & Van der Kloet, D. (2012). Dissociation and dissociative disorders: Challenging conventional wisdom. *Current Directions in Psychological Science, 21*, 48-53.

Aims of the present dissertation

The studies described in this dissertation aimed to 1) investigate the causal role of sleep in the aetiology of dissociation (i.e., is dissociation caused by sleep problems?); 2) eludicate the mechanisms by which sleep leads to dissociation; and 3) explore new treatment avenues for dissociative symptoms. In the introductory chapter, a review was provided of the recent literature on sleep, dissociation, and memory. In part I of this dissertation, we built upon these findings and further solidified evidence for the link between dissociative symptoms and sleep disruption. Thus, the link was found in participants experiencing sleep loss in our field study, but was also confirmed using objective polysomnographic measures in our sample of insomnia patients. Furthermore, using structural equation modeling, we evaluated the direction of this relation, and found that unusual sleep experiences may lead to dissociative symptoms, but the reverse does not seem to be true.

In part II, we studied possible underlying mechanisms and put our hypotheses to a more stringent test during a sleep deprivation experiment. As was the case in a previous sleep deprivation study carried out in our lab (Giesbrecht et al., 2007), sleepiness tended to precede an increase in dissociative symptoms. Furthermore, we explored the underlying neuropharmacology of dissociative symptoms and observed that dissociative symptom levels increase when participants are under the influence of MDMA, THC, and benzoylmethylecgonine, providing tentative support for the hypothesis that an increased release of serotonine and dopamine neurotransmitter activity may play a role in the pathogenesis of dissociative symptoms. Moreover, we established that the connection between sleep disruptions and dissociative symptoms is not only evident in healthy subjects, but in patients with severe dissociative disorders as well.

Finally in part III of the dissertation, we interrogated our theoretical framework from another angle and investigated whether sleep normalization would lead to a reduction of dissociative symptoms. In three studies, we explored the effects of treatment methods focusing on sleep improvement and found that while sleep hygiene protocols seem to be succesful in reducing dissociative symptoms, Mindfulness Based Therapy was not.

The implications of the research findings will be described below. Furthermore, some directions for future research will be provided. Finally, some concluding remarks will be made.

Implications of the research findings

Taken together, the research findings from studies described in this dissertation have several both theoretical and clinical implications implications.

Theoretical implications

An important contribution to the research field is that we have been able to establish a robust connection between sleep abnormalities and dissociation using a varity of approaches and measures. Table 11.1 provides a summary of the correlations between self-reported sleep experiences and dissociation that we observed in the studies of this dissertation. The weighted average (N = 756) between sleep disruptions and dissociation is r = .45. Thus, there can be no doubt that sleep disruptions and dissociative psychopathology are related to each other.

Furthermore, we provide (tentative) evidence for a directiontionality in the relation between sleep and dissociation, namely that sleep disruption seems to precede dissociative symptoms. This is important because it provides support for a causal framework in which both the posttraumatic view and the sociocognitive view of dissociation can be concealed. Specifically, one could hypothesize that childhood traumatic experiences fuel the likelihood of the development of sleep problems during the adult years, which in turn fosters dissociative symptoms.

Table 11.1.
Summary of Pearson product-moment correlations between DES and sleep measures, including the weighted average.

Chapter / Study	N	r	p value
Chapter II: Field study	36	DES – SSSchange: .35	.05
Chapter III: Polysomnography study	46	DES – ISES: .40	<.01
Chapter IV: SEM study	139	DES – ISES: .31	<.01
Chapter V: Sleep deprivation study	56	DES – ISES: .55	<.01
Chapter VII: DID study	94	DES – ISES : .65	<.01
Chapter VIII: Sleep hygiene study	37	DES – ISES: .65	<.01
Chapter IX: Mindfulness study	92	DES – ISES: .39	<.01
Chapter X: Sleep normalization study	256	DES – SLEEP-50: .44	<.01
Weighted average combined studies	756	.45	<.01

However, the specific influence of sleep on dissociative symptomatology proved to be more complex than initially expected. Sleep is not a unitary entity, and often sleep problems are intertwined with co-morbid psychopathology, such as anxiety and mood disorders. Our studies demonstrated that it is difficult to disentangle the effect of sleep problems on dissociation and the effect of mood on both

dissociation and sleep problems (for examples, see chapters V and X). Furthermore, although the relation between sleep and dissociation seems specific in the sense that dissociation is most strongly related to unusual sleep experiences, but not to lucid dreaming (see chapter I and VIII), a number of questions remain. For example, dissociation was found to be related to nightmares, vivid dreams, and hypnagogic hallucinations (all REM sleep phenomena), but also to sleep walking and night terrors (both NREM sleep phenomena). This seems to point in the direction of an unknown common underlying sleep regulatory mechanism driving both REM and NREM sleep aberrations (Germain, 2013).

Another interesting avenue worth exploring is the underlying neurocircuitry of dissociative symptoms. In chapter VI, we demonstrated that dissociative symptoms increase when under the unfluence of MDMA, cocaine, or THC. This might be related to the dopamine neurotransmitter system, particularly the cannabinoid-1-receptors. A recent study by Neumeister and colleagues (2013) showed that this specific cannabinoid-1-receptor (CB-1 receptor) is highly prevalent in people suffering from posttraumatic stress disorder (PTSD). This is because they have a shortage of the neurotransmitter anandamide, which binds to the CB-1 receptor and sends calming signals to the brain. Consequently to the anandamide shortage, the brain reacts with creating extra CB-1 receptors. Anandamide is known for its role in blocking memory formation (for unpleasant memories, such as during childbirth) and has an important function in memory consolidation. Not only is this an explanation for why people with PTSD respond well to THC, as it temporarily takes over the role of anandamide, but it is also illustrative in underpinning the relation between PTSD, dissociation, and memory problems.

It would be worthwile to study the complex relation between sleep, dissociation, memory, and the processing of emotional information. Although the posttraumatic model of dissociation would predict that individuals with heightened dissociation are inhibiting the impact of threat-related information and therefore exhibit slower or impaired processing of such information, patients with DID and other “high dissociators” display, as a matter of fact, better memory for to-be-forgotten sexual words in directed-forgetting tasks (Elzinga, de Beurs, Sergeant, van Dyck, & Phaf, 2000). This challenges the widespread idea that amnesia (i.e., extreme inhibition) is a core element of dissociation (Giesbrecht et al., 2008).

Alternatively, the sociocognitive model poses that disruptions of the sleep–wake cycle degrade memory and attentional control, thereby accounting for, or at least contributing to, the cognitive deficits of highly dissociative individuals. This disruption of the sleep-wake cycle may also explain why highly dissociative people tend to have a propensity towards false memories. Thus, false memories might be mediated by heightened levels of suggestibility, fantasy proneness, and cognitive failures (e.g., lapses in attention). Chapters I, IV, and VII provide support for this interpretation. Accordingly, the relation between dissociation and fantasy

proneness may explain why individuals with high levels of dissociation are especially prone to develop false memories of emotional childhood events. This dovetails with the analysis of Llewellyn (2013; see also Van der Kloet, Merckelbach, & Lynn, in press), who stated that REM dreaming is more perceptually vivid and hyperassociational compared to the waking state, and that during dreaming there is an enhanced access to remote memories in the presence of recent ones. REM dreaming may therefore provide the ideal state for elaborative encoding of emotional memories. However, when these dreaming and waking states should become de-differentiated, the hyperassociational junctions may intrude consciousness, possibly fostering dissociative symptoms and false memories (see below). Our findings are in keeping with this hypothesis, as we found REM sleep to be more strongly present in highly dissociative insomnia patients than in low dissociative patients (chapter III).

A short note on dreaming and memory[18]

In a recent, thought provoking review, Llewellyn (2013) made a convincing case that REM sleep serves the function of elaborative encoding by following ancient art of memory (AAOM) rules. At the phenomenological level, this becomes manifest in visualizations, bizarre connections, and narrative form during dreams. Llewellyn summarizes research showing that, during REM sleep, the prefrontal areas are in a state of deactivation resulting in fluid reasoning and flexible thought. However, she only briefly touches upon the ramifications of her analysis for understanding the development and perseverance of psychopathological symptoms.

We would like to emphasize the relevance of Llewellyn's analysis for one particular type of psychopathology – namely, *dissociative symptoms* (e.g., derealization, amnesia, and absorption). Dissociative symptoms are common in the healthy population, but disorders such as dissociative identity disorder (DID; formerly known as multiple personality disorder) and depersonalization disorder, represent severe, albeit rare, manifestations of psychopathology (Lynn et al., 2012). The traditional view is that dissociative symptoms reflect (transient) disruptions in memory, perception, and/or consciousness and that these disruptions are causally related to aversive life events (e.g., Dalenberg et al., 2012).

More specifically, the idea is that dissociative symptoms enable individuals to distance themselves from the emotional impact of aversive events. Although there is some indirect evidence – largely correlational in nature – for this trauma

18 This paragraph is an adapted version of:
Van der Kloet, D., Merckelbach, H., and Lynn, S.J. (2013). Dissociative symptoms and REM sleep. Commentary on Llewellyn (2013) Such stuff as dreams are made on? Elaborative encoding, the ancient art of memory and the hippocampus. *Behavioral Brain Sciences, in press*.

hypothesis, it suffers from one important weakness: it remains silent as to *how* aversive events produce dissociative symptoms.

More recently, researchers have proposed that sleep disturbances play an important role in the development of dissociative symptoms. A solid and steadily accumulating foundation of research now exists 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 (Koffel & Watson, 2009; Van der Kloet et al., 2012ab; Watson, 2001; see table 11.1).

The idea that sleep disturbances and dissociative symptoms are related is not new. In the 19th century, double consciousness, the historical precursor of DID, 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." In 2001, Watson investigated two large samples of undergraduate students and showed that dissociative symptoms are linked to self-reports of vivid dreams, nightmares, recurrent dreams, hypnopompic imagery, and other unusual sleep phenomena. His finding has been reproduced time and again. In chapter I, we (Van der Kloet et al., 2012a) summarized the findings of 23 studies and found an average correlation of $r = 0.41$ between dissociative symptoms (as measured by the *DES*) and unusual sleep experiences (collected with measures such as the *ISES*). All studies in this dissertation (table 11.1) replicated this correlation.

The connection between sleep and dissociative symptoms seems 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. Germane to this specificity issue is the study by Koffel and Watson (2009) in which 374 participants completed a comprehensive test battery, including measures of psychopathology and sleep. The authors concluded that "unusual sleep experiences are specific to dissociation and schizotypy, whereas insomnia and lassitude are specific to depression and anxiety" (p. 551).

However, these studies on sleep and dissociation used a correlational approach, which precludes the ability to draw causal conclusions. If dissociative symptoms are, indeed, fueled by a labile sleep-wake cycle, sleep loss would be expected to intensify dissociative symptoms, thereby suggesting a specific temporal pattern. We tested this prediction in a pilot study (Giesbrecht et al., 2007) that tracked dissociative symptoms in 25 healthy volunteers during one night of sleep deprivation. We found that sleepiness, as well as spontaneous and induced dissociative symptoms, were stable during the first day, but substantially increased after one night of sleep loss. Interestingly, the increase in dissociative

symptomatology was highly specific: Dissociative symptoms were affected by sleep loss earlier in time than were mood deterioration. By and large, chapter V replicated this pattern.

The reverse appears to be true, as well. We (chapter X; Van der Kloet et al., 2012a) conducted a longitudinal study to investigate the relation between unusual sleep experiences and dissociation in a mixed inpatient sample (*N* = 195) evaluated on arrival and at discharge 6–8 weeks later. We found a robust link between unusual sleep experiences and dissociative symptoms and determined that sleep normalization was accompanied by a reduction in dissociative symptoms. The link between dissociation and sleep is likely more differentiated, as we observed that decreases in narcoleptic experiences rather than decreases in insomnia accompanied the reduction in dissociative symptoms.

Finally, in a recent study (chapter III; Van der Kloet et al., in press), we measured dissociative symptoms and EEG sleep parameters in patients (*N* = 45) suffering from insomnia. We found that it is lengthening of REM sleep that predicts dissociative symptoms. This finding is consistent with the hypothesis that a disturbed sleep-wake cycle, possibly due to aversive life events, produces excessive or out-of-phase REM activity that, in turn, underlies dissociative symptoms. These dissociative symptoms may, in turn, exacerbate or increase vulnerability to sleep disturbances, engendering a vicious cycle that may be ameliorated with interventions that target dissociation, sleep problems, or both.

Thus, one distinct scenario that warrants further investigation is that excessive REM sleep during the night and/or minor REM sleep episodes during the day fuel the type of fluid and hyperassociative cognition that is typical for dissociative disorders. This research perspective might shed new light on the propensity of dissociative individuals to develop false memories. Even more importantly, it might suggest new treatment options for dissociative patients. Whereas Llewellyn focused on the memory-promoting aspects of REM sleep, we have emphasized the pathological potential of excessive REM. It would be exciting to combine these two lines of research.

Clinical implications

Our findings have implications for the clinical field as well; particularly in the way we think about dissociative disorders and how we should approach this category of psychopathology. Multiple personality disorder, or dissociative identity disorder (DID) - as it is known now –, used to be a mere curiosity. The disorder was rarely diagnosed until the 1980's. In that period multiple personality disorder became an official diagnosis in the DSM-III. From then on, the numbers of 'multiples' increased rapidly. In the 1990s, there were hundreds of people diagnosed with multiple

personality in every major city in the United States of America (Hacking, 1995). How could this 'epidemic' be explained?

One of the possible explanations entails the media attention that was given to the disorder. This started with the The Three Faces of Eve (Thigpen & Cleckler, 1957). This book, and later the movie, was one of the first to speak of multiple personality disorder. However, it was not until years later, when the fictional "as told to" book of Sybil (Schreiber, 1973) became known worldwide, that the prototype of what it was like to be a 'multiple personality' was born. Sybil tells the story of how a clinician – Cornelia Wilbur – unravels the different personalities of her patient Sybil during a long course of treatment (over 2500 office hours). Wilbur was one of the first to relate multiple personality to childhood sexual abuse (but see Nathan, 2011; Rieber, 2006, for evidence that many details of the Sybil story are inaccurate). Probably, establishing this relation between childhood abuse and dissociation has fuelled the increase of numbers of multiples from that time on. It motivated therapists to actively seek for clues of childhood abuse in their dissociative patients. This fitted well with the mindset of the 1980s, as childhood abuse was a sensitive issue then in psychology as well as in politics (Hacking, 1995). From then on, many movies and books focused on the subject of multiple personality, and nowadays we see patients with DID as guests visiting the Oprah Winfrey show, as if they were our modern-day circus acts.

Mainstream treatment techniques for DID often reinforce patients' displays of multiplicity (e.g., asking questions like, "Is there another part of you with whom I have not spoken?"), reify alters as distinct personalities (e.g., calling different alters by different names), and encourage patients to establish contact and dialogue with presumed alters (Lynn, Lilienfeld, Merckelbach, Giesbrecht, & van der Kloet, 2012). These sources of evidence do not imply that DID is typically created de novo by iatrogenic (therapist-induced) or sociocultural influences. Rather, the idea is that iatrogenic and sociocultural influences operate against a backdrop of preexisting psychopathology.

Indeed, most patients with DID, and to a lesser extent other dissociative disorders, meet criteria for borderline personality disorder, a condition marked by extremely unstable behaviors, such as unpredictable shifts in mood, impulsive actions, and self-mutilation (Lilienfeld et al., 1999). Individuals with this disorder are understandably seeking an explanation for their bewildering behaviors. The presence of hidden alters may be one such explanation, one that may appear particularly plausible when suggested by psychotherapists or sensational media portrayals.

Of course, the sensitivity of these individuals to suggestive influences may arise from increased salience of distressing memories (some of which may stem in part from trauma). In previous chapters, we also came across their susceptibility to memory errors (chapter V) and a propensity to fantasize and experience difficulties

in distinguishing fantasy from reality (chapter VII), brought about at least in part by a labile sleep-wake cycle (chapters I, IV, V, VII). Therefore, clinicians should be cautious when they encounter patients with diagnoses of DID or other dissociative disorders, in the way they approach treatment methods and guard against suggestive influences.

The sleep-dissociation view has the potential to reshape the way we think about dissociative disorders and how we should conceptualize this group. The relation between sleep disruption and dissociation is evident in both clinical and general populations. From this perspective, the hypothesis that dissociative disorders can be triggered by (a) a labile sleep cycle that impairs cognitive functioning, combined with (b) highly suggestive psychotherapeutic techniques, warrants further empirical investigation.

Directions for future research

Future studies may discern what types of disruptions in the sleep-wake cycle are most reliably related to dissociative disorders, and then establish training programs, including medication regimens, to address these problems. Some steps have already been taken to this effect (chapters VIII-X).

One possible way to study types of sleep disruptions entails the use of neuroimaging methods. As stated above, it would be of interest to elucidate the specific influences of REM sleep and NREM sleep and their underlying common sleep regulatory mechanism. This could be studied using sleep neuroimaging methods, such as EEG or single photon emission computed tomography (SPECT). REM and NREM sleep provide unique biological states making them suitable to study via the detection of neurobiological changes. REM is characterized by a milieu of heightened activation in the limbic areas, while NREM is an endogenous milieu of attenuated arousal (Germain, 2013). Potential mechanisms can be studied that might contribute to sleep complaints, or may be related to dissociative symptomatology. A conceptual framework building on this line of reasoning is presented in Figure 11.1.

In chapters VIII-X, we described our efforts in exploring sleep normalization as a therapeutic vehicle for decreasing dissociative symptoms. It seems that cognitive behavioral approaches, specifically sleep hygiene recommendations, are more effective in decreasing dissociative symptoms than mindfulness based approaches. Possibly, mindfulness is not useful in reducing dissociation, as it may sometimes have adverse effects, and even mild dissociative phemomena may arise as its side effects (Dobkin et al., 2011). It would be interesting to explore whether a short-term cognitive behavioral treatment for insomnia (CBT-I) would prove effective to decrease the refractory symptoms of dissociative disorders. Hopeful results in this respect were reported by Myers and colleagues (2011), who employed a 4-session

standard-format CBT-I treatment in participants with persecutory delusions and found that sleep improvement lessened the delusions.

Another field worth exploring entails the domain of nightmares. As nightmares seem particularly associated with dissociation (see chapter IX), one would expect a dissociative symptom level decrease when nightmares are treated. Interesting work in this field has been reviewed by Germain (2013), who summarized sleep treatment studies in PTSD. A number of treatment methods have been developed in treating individuals suffering from nightmares using CBT, psychopharmacology, and other therapeutic techniques. Particularly, the use of Imagery Rehearsal and Rescripting Therapy (IRRT) is promising (Schmucker, Dancu, Foa, & Niederee, 1995; Arntz & Weertman, 1999; Davis & Wright, 2007; Cook, Harb, Gehrman, Cary, Gamble, Forbes et al., 2010) and might prove of interest in the field of dissociation as well. This would constitute an entirely novel and exciting approach to the treatment of dissociative symptoms.

Finally, yet another direction may be fruitful as well. Dissociative symptoms are also related to sleep walking and night terrors (both NREM phenomena). These are described as dissociative states during the night and point in the direction of a cross-state continuity of consciousness (Koffel & Watson, 2009), with individuals suffering from dissociation day and night. Studies aimed at exploring a possible common underlying mechanism that could explain both unusual sleep experiences and dissociative symptoms may inform research and clinical practice. One possible avenue might be to study the influence of emotional processing of stimuli, as we know that memory – specifically memory for emotional stimuli – is particularly involved in both dissociation (Elzinga et al., 2000; see also chapter I and V) and sleep (Walker & van der Helm, 2009; Llewellyn, 2013). It would be interesting to explore whether individuals suffering from either excessive REM sleep abnormalities (nightmares, hypnagogic hallucinations) or NREM disruptions (sleep walking, night terrors) show impairments in performing emotional memory tasks. If so, are these impairments predicted by high dissociation scores? And, moreover, would these performance difficulties be resolved (i.e., would memory improve) when individuals are treated with a CBT protocol aimed at reducing sleep disruptions and improving sleep quality? We would hypothesize that a CBT treatment aimed at normalizing sleep would decrease dissociation (see also chapter X) and may improve memory for emotional stimuli.

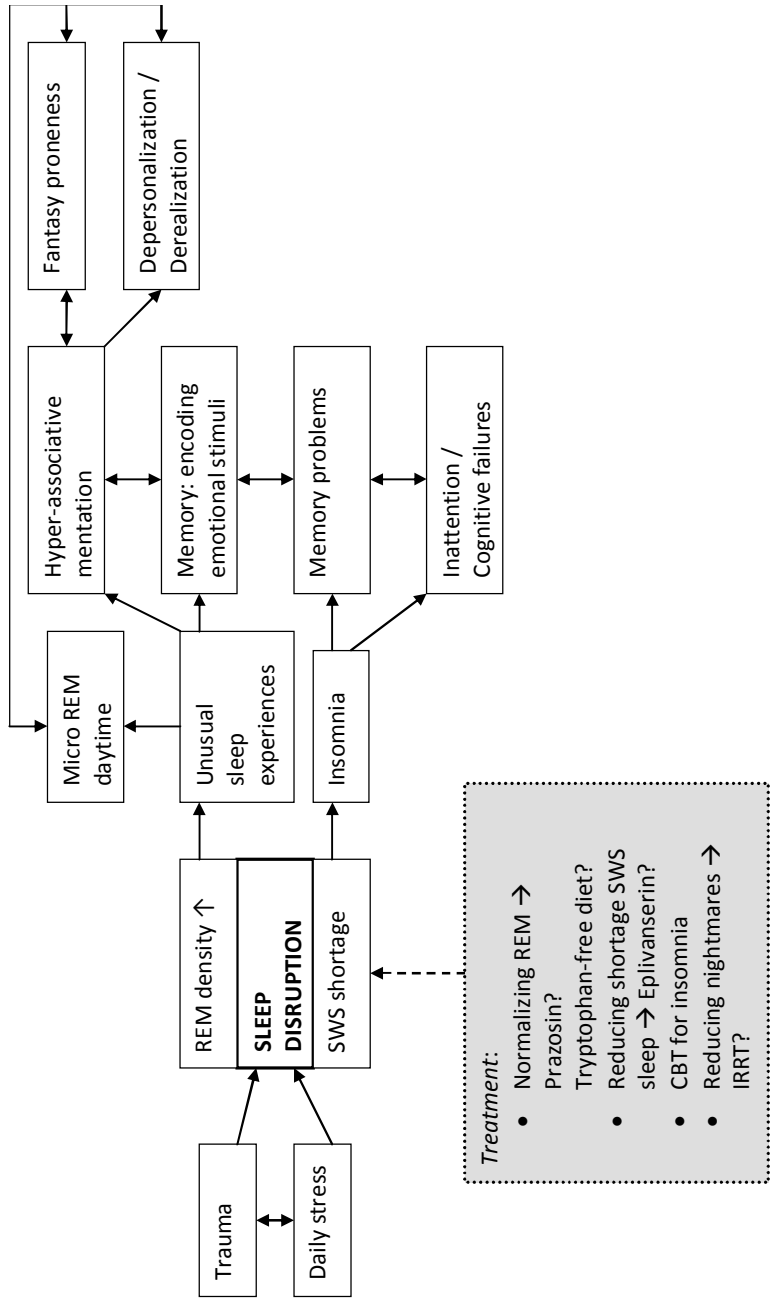


Figure 11.1.
A conceptual framework of the relation between sleep, dissociation, memory, cognitive failures, and fantasy proneness.

Final remarks

In closing, the sleep-dissociation model can serve as a 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.

Summary

The studies described in this dissertation aimed at 1) investigating causality issues with regard to dissociation (i.e., do sleep problems cause dissociative symptoms?); 2) addressing the mechanisms that play a role in the causal chain between sleep and dissociation; and 3) exploring new treatment avenues for dissociative symptoms.

Apart from the introductory chapter and a concluding discussion chapter, the dissertation comprises three parts. The first part consists of three chapters in which the link between sleep and dissociation is firmly established. More specifically, the chapters describe a field study in which participants suffered from a night of sleep loss, a study examining the relation between objective sleep parameters and dissociation in a group of insomnia patients, and a structural equation modeling study leading to the formulation of a tentative causal framework of unusual sleep experiences, dissociation, cognitive failures, and fantasy proneness. Thus, In this first part, we present evidence showing that sleepiness forms a precursor of dissociative symptoms, that both subjective and objective sleep measures are related to dissociation, and that when exploring a causal framework, unusual sleep experiences seem to precede dissociative symptoms rather than vice versa.

The second part of this dissertation offers a more in-depth discussion of the underlying mechanisms. Its three chapters describe a) a sleep deprivation experiment, b) a study exploring the psychopharmacological effects of MDMA, cocaine, and THC, and c) a study exploring the relation between sleep aberrations and dissociation in a group of patients suffering from dissociative identity disorder, as well patients suffering from posttraumatic stress disorder. In this second part of the dissertation, we find that sleep loss induces dissociative symptoms. However, there are other means to increase dissociative symptoms. For example, we show that dissociative symptoms can be readily induced with recreational dosages of MDMA, cocaine, and THC. This points in the direction of the dopamine system as a possible underlying factor for dissociation. Finally, the clinical importance of these studies is underlined by the finding that the relation between sleep disruption and dissociation is also evident in patients with dissociative identity disorder and patients with posttraumatic stress disorder.

In the third part, we investigated whether *normalizing* sleep would *reduce* dissociative symptoms. Its three chapters discuss a sleep hygiene training, a Mindfulness-Based Therapy, and a treatment consisting of strict sleep hygiene guidelines and their positive effects on sleep quality parameters and dissociative symptoms. In this part of the dissertation, we find support for our hypothesis that normalizing sleep will reduce dissociative symptoms. Sleep hygiene programs seem effective in this regard, but mindfulness appears to be less promising.

Finally, in the general discussion chapter of this dissertation, our findings are integrated and their contribution to the research domain of dissociation is highlighted. Theoretical and practical implications are discussed. We review recent developments and future avenues for research are explored.

Samenvatting

Dit proefschrift gaat over drie vragen: 1) leiden slaapproblemen tot dissociatieve symptomen?; 2) welke mechanismen spelen mogelijk een rol in de relatie tussen slaap en dissociatie?; en 3) biedt dit perspectieven voor nieuwe behandelmethoden?

Afgezien van het inleidende hoofdstuk en de uitleiding, bestaat deze dissertatie uit drie onderdelen. We starten met het onderzoeken van het causale model van dissociatie en de relatie tussen slaap en dissociatie. In dit eerste deel bespreken we een veldstudie waarin deelnemers een nacht lang wakker bleven. Hierin vinden we dat slaperigheid een voorbode is van dissociatieve symptomen. Vervolgens onderzoeken we de relatie tussen objectieve maten van slaap (bijvoorbeeld REM-latentie) en dissociatie in een groep van patiënten met slaapklachten (slapeloosheid). We tonen aan dat niet alleen subjectieve, maar ook objectieve maten van slaap verband houden met dissociatie. Tot slot formuleren we een causaal model van ongewone slaapervaringen, dissociatie, cognitieve fouten, en fantasierijkheid. Dat model is gebaseerd op bevindingen van een grote groep studenten van de universiteit Maastricht. Het model zegt met zoveel woorden dat ongewone slaapervaringen vooraf gaan aan dissociatie, en niet vice versa.

Hierna volgt een discussie over de mogelijke mechanismen die een rol spelen in de relatie tussen slaap en dissociatie. In dit tweede deel van de dissertatie beschrijven we drie studies. Met een slaapdeprivatie-experiment laten we zien dat slaapgebrek leidt tot acute dissociatieve symptomen. Maar niet alleen slaperigheid veroorzaakt dissociatieve symptomen. Zulke symptomen kunnen ook worden opgewekt met recreationele doses van MDMA, cocaïne, en THC. Dit laten we zien in een studie waarin deelnemers onder invloed werden gebracht van deze drie soorten drugs. De hoge niveaus van dissociatie die we maten bij de deelnemers, doen vermoeden dat het dopamine- neurotransmittersysteem een rol van betekenis speelt in het tot stand komen van dissociatieve symptomen. Tot slot onderzochten we de klinische betekenis van onze bevindingen. We bestudeerden de relatie tussen slaapverstoringen en dissociatie in een groep van patiënten met dissociatieve identiteitsstoornis en in een groep patiënten met posttraumatische

stress stoornis. Onze resultaten lieten zien dat de relatie tussen slaapverstoring en dissociatie sterk aanwezig is in beide groepen en daarmee klinisch relevant is.

In het derde deel van de dissertatie proberen we, met de eerdere bevindingen in het achterhoofd, het verband tussen slaapverstoringen en dissociatieve symptomen op een andere manier te benaderen. Als slechte slaap ervoor zorgt dat dissociatieve symptomen optreden, dan is het de moeite waard om te onderzoeken of het *normaliseren* van slaap dissociatieve symptomen doet *afnemen*. We hebben drie studies uitgevoerd. Daarbij onderzochten we de effecten van een slaaphygiëne-training, de effecten van een op *mindfulness* gebaseerde therapie, en een behandelmethode gebaseerd op strikte slaaphygiëne richtlijnen. Bij de drie studies keken we steeds wat de effecten waren op slaapverbetering en de afname van dissociatieve klachten. In dit deel van de dissertatie laten we zien dat het normaliseren van slaap dissociatieve symptomen inderdaad doet afnemen. Slaaphygiëne-programma's lijken hierbij vooral effectief te zijn, maar mindfulness niet.

Tot slot bespreken we in het uitleidende hoofdstuk van deze dissertatie onze bevindingen in hun onderlinge samenhang. Bovendien onderstrepen we hun bijdrage aan het onderzoeksdomein van dissociatie. We bespreken welke theoretische en praktische implicaties onze bevindingen kunnen hebben, en we besteden aandacht aan recente ontwikkelingen en nieuwe onderzoeksideeën.

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Curriculum Vitae

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Conference presentations

I presented various papers at the following conferences:

- Research Day EPP, 2012, Maastricht, The Netherlands
- Forensic Psychology Update, 2012, Maastricht, The Netherlands.
- ESSPD 2nd International Congress on Borderline Personality Disorder and Allied Disorders, 2012, Amsterdam, The Netherlands
- Nuffield Department of Clinical Neurosciences Departmental Seminar, 2013, John Radcliffe Hospital, Oxford, United Kingdom
- Psychiatry Congress Berlin, 11-2013, Berlin, Germany.