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Sleep Normalization and Decrease in Dissociative Experiences: Evaluation in an Inpatient Sample

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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.

Keywords: dissociative experiences, unusual sleep experiences, sleep hygiene

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, 2003b).

Relying on two large nonclinical samples, Watson (2001) found that dissociation, as measured by two validated dissociation scales (E. M. Bernstein & Putnam, 1986), correlates with unusual sleep- and dream-related experiences (Watson, 2001, 2003b). 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, Knox, & Lynn, 2006; Giesbrecht & Merckelbach, 2004, 2006; Soffer-Dudek & Shahar, 2009). In a review of 19 studies, van der Kloet, Merckelbach, Giesbrecht, and Lynn (2011) 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 have 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, & Borrow, 2001), yielded correlations in the range of .30–.55. Similarly, Agargun and colleagues (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, Obermeyer, Thisted, & Gillin, 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) than they are correlated with mood and anxiety (Koffel & Watson, 2009b). Koffel and Watson (2009b) aptly concluded that "unusual sleep

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experiences (e.g., nightmares, vivid dreams, narcolepsy symptoms) are associated with symptoms of dissociation in both clinical and nonclinical samples" (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, Giesbrecht, Smeets, Leppink, Jelicic, and Merckelbach (2007) deprived 25 healthy volunteers of 1 night of sleep and determined that sleep loss engenders a substantial increase in dissociative symptoms. An important finding was that this increase could not be attributed to mood or response bias.

Researchers have not, as yet, examined whether promoting healthy sleeping reduces dissociative symptoms. 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 and 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; we 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–posttreatment: Previous studies have revealed a connection between dissociation and anxiety and depression (Giesbrecht, Merckelbach, van Oorsouw, & Simeon, 2010; Sierra et al., 2002), 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, 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) because of somatic or psychiatric complications. Completers versus noncompleters did not differ on any of the baseline measures (e.g., Dissociative Experiences Scale, Beck Anxiety Inventory, Brief Symptom Inventory, Childhood Trauma Questionnaire, and Beck Depression Inventory—II; $ps > .05$) or with respect to age, gender, use of medication, or diagnosis ($ps > .05$).

A psychologist and resident psychiatrist collaborated to determine diagnoses on the basis of 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%); burnout (i.e., mixed anxiety and depression symptoms that fail to reach diagnosis threshold; 5%); attention-deficit/hyperactivity disorder (2%); psychotic symptoms (2%); identity problems (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 differences, 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, mindfulness, daily fitness exercises, and creative work. It is important to note that 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 alpha baseline = .94, follow-up = .94; E. M. 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 et al. (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 eight DES items that constitute the so-called DES-Taxon (DES-T; Waller, Putnam, & Carlson, 1996), which tap more 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 0.90 were assigned to the DES-T taxon group. Psychometric

shortcomings notwithstanding (Watson, 2003a), the DES-T has been considered a useful measure in the dissociation field (Simeon, Knutelska, Nelson, Guralnik, & Schmeidler, 2003).

SLEEP-50 (Cronbach's alpha baseline = .84, follow-up = .93; Spoormaker, Verbeek, van den Bout, & Klip, 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 the *Diagnostic and Statistical Manual of Mental Disorders* (4th ed., text rev.; American Psychiatric Association, 2000): sleep apnea (Cronbach's alpha baseline: .58; follow-up: .69; change score: .52), insomnia (Cronbach's alpha baseline: .87; follow-up: .88; change score: .76), restless legs (Cronbach's alpha baseline: .71; follow-up: .81; change score: .45), circadian rhythm sleep disorder (Cronbach's alpha baseline: .56; follow-up: .55; change score: .27), sleepwalking (Cronbach's alpha baseline: .65; follow-up: .84; change score: .30), nightmares (Cronbach's alpha baseline: .84; follow-up: .90; change score: .93), factors influencing sleep (Cronbach's alpha baseline: .81; follow-up: .82; change score: .53), the impact of sleep complaints on daily functioning (Cronbach's alpha baseline: .66; follow-up: .70; change score: .72), and narcolepsy (Cronbach's alpha 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 Iowa Sleep Experiences Survey (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 alpha 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*, 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 alpha 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 alpha = .93) and modest test–retest reliability (r = .66; Kelett, Beail, Newman, & Frankis, 2003).

Beck Depression Inventory—II (BDI–II; Cronbach's alpha baseline = .92, follow-up = .93; Sprinkle 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 is good (r = .83; Sprinkle et al., 2002).

Childhood Trauma Questionnaire (CTQ; Cronbach's alpha baseline = .90, follow-up = .52; D. P. 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, Bernstein, Lobbestael, & Arntz, 2009).

Results

Individual Differences Measures

Statistical analyses were performed using SPSS 18.0 software. Table 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 of 195 participants (24%) displayed dissociation levels exceeding the clinical cutoff 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 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.

Correlations Between Change Scores of SLEEP-50 Subscales, DES, and Psychopathology Composite

Given their high intercorrelations, 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 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.

Modeling Mood as Mediator of the Dissociation–Sleep Connection

We determined whether the decrease in dissociative symptoms at follow-up was 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)

Table 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**
PLMD	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**
Sleepwalking	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 of 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; PLMD = periodic leg movement disorder; BSI = Brief Symptom Inventory; BAI = Beck's Anxiety Inventory; BDI–II = Beck's Depression Inventory—II; CTQ = Childhood Trauma Questionnaire.
* $p < .05$ (two-tailed). ** $p < .01$ (two-tailed).

as the 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 alphas 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 alpha = .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 nonsignificant predictors by means of backward elimination. We present a hierarchical decomposition in Table 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 was (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 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 ($B = 0.37$, $SE = 0.13$, $p < .01$), with another part of the change in taxon membership being explained by the general psychopathology composite ($B = 0.18$, $SE = 0.08$, $p < .05$).

Childhood Trauma, Improvement in Sleep, and Reduction in Dissociation

We hypothesized that sleep improvement would lead to a reduction in dissociative symptoms. However, because of shortcomings associated with change scores (Peter, Churchill, & Brown, 1993), we tested three theoretically motivated mediation models using structural equation models (see Cole & Maxwell, 2003):

Table 2
Pearson Product–Moment Correlations Between Dissociation, Psychopathology, and Childhood Trauma at Baseline, Follow-Up, and Change Scores

Variable	DES total	DES Amnesia	DES Absorption	DES Depersonalization/ derealization	BSI	BAI	BDI–II	Psychopathology composite	CTQ
DES total									
Baseline	*0.71***	—	—	—	—	—	—	—	—
Follow-up									
Change									
DES Amnesia								—	
Baseline	0.86**	*0.59***	—	—	—	—	—		—
Follow-up	0.86**								
Change	0.82**								
DES Absorption								—	
Baseline	0.93**	0.68**	*0.72***	—	—	—	—		—
Follow-up	0.93**	0.67**							
Change	0.86**	0.51**							
DES Depersonalization/Derealization								—	
Baseline	0.86**	0.65**	0.74**	*0.69***	—	—	—		—
Follow-up	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***	—	—		—
Follow-up	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***	—		—
Follow-up	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***		—
Follow-up	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**			
Psychopathology composite									
Baseline	0.43**	0.28**	0.43**	0.40**	0.96**	0.88**	0.90**	*0.56***	
Follow-up	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***
Follow-up	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: $n = 201$. Numbers in italics display the correlations of the measures between baseline and follow-up. DES = Dissociative Experiences Scale; BSI = Brief Symptom Inventory; BAI = Beck's Anxiety Inventory; BDI–II = Beck's Depression Inventory—II; CTQ = Childhood Trauma Questionnaire.
* $p < .05$ (two-tailed). ** $p < .01$ (two-tailed).

Model 1: no mediation, decrease in narcoleptic symptoms leads directly to a decrease in dissociation; Model 2: partial mediation, decrease in narcoleptic symptoms leads to decreases in both dissociation and general psychopathology, but there is also a direct effect of decrease in narcoleptic symptoms on dissociation; and Model 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: 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 6 gives the fit indices for all three models. As can be seen, both the partial and the full mediation model (Models 2 and 3) fulfilled all criteria for acceptable fit (see also Figure 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 at T1 (follow-up time point) and DES at T1 to zero, and Model 3 restricts sleep at T1 to DES at T1 to zero. Therefore, we tested the differences between models by means of a chi-square test (see, e.g., Schreiber, Nora, Stage, Barlow, & King, 2006). This test showed that Model 2 is statisti-

Table 3
Pearson Product–Moment Correlations Between SLEEP-50 Subscales, Dissociation, and Psychopathology Composite (All Change Scores; n = *195) and Differences Between Correlation Coefficients*

Subscale	DES total	Psychopathology composite	Steiger's *z*	*p*
Sleep Apnea	.15*	.15*	0.05	>.05
Insomnia	.18*	.45**	3.50	<.01
Narcolepsy	.29**	.23**	−0.84	>.05
PLMD	.12	.23**	1.36	>.05
Circadian Rhythm	.14*	.24**	1.17	>.05
Sleepwalking	.07	.13	0.42	>.05
Nightmares	.01	−.06	−0.89	>.05
Factors Influencing Sleep	.08	−.01	−1.15	>.05
Impact of Sleep Complaints on Daily Functioning	.26**	.47**	2.74	<.01

Note. DES = Dissociative Experiences Scale; PLMD = periodic leg movement disorder.
* $p < .05$ (two-tailed). ** $p < .01$ (two-tailed).

cally superior to Models 1 and 3 ($ps < 0.01$). It is interesting that, 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 a small effect size.

Table 4
Summary of Hierarchical Multiple Regression Analysis on the Dissociative Experiences Scale (All Change Scores; n = *195)*

Step	*B*	β	*t*(*df* = 2–6)	*p*	*r*	r^2
1						
Insomnia	−0.05	−.02	−0.29	.78	.43	.19
Narcolepsy	1.08	.20	2.72	.01*		
Nightmares	0.08	.02	0.29	.78		
Daily functioning	0.11	.04	0.47	.64		
Psychopathology	1.21	.29	3.65	.00*		
CTQ	4.85	.10	1.41	.16		
2						
Narcolepsy	1.06	.20	2.72	.01*	.43	.19
Nightmares	0.07	.02	0.26	.80		
Daily functioning	0.10	.04	0.43	.67		
Psychopathology	1.17	.29	3.80	.00*		
CTQ	5.03	.10	1.49	.14		
3						
Narcolepsy	1.07	.20	2.77	.01*	.43	.19
Daily functioning	0.10	.04	0.46	.65		
Psychopathology	1.16	.20	3.80	.00*		
CTQ	4.87	.10	1.47	.14		
4						
Narcolepsy	1.14	.22	3.21	.00*	.43	.19
Psychopathology	1.22	.30	4.41	.00*		
CTQ	4.76	.10	1.44	.15		
5						
Narcolepsy	1.17	.22	3.31	.00*	.42	.18
Psychopathology	1.27	.31	4.61	.00*		

Note. Psychopathology = Psychopathology composite, consisting of total change scores on Beck's Anxiety Inventory, Brief Symptom Inventory, and Beck's Depression Inventory; CTQ = Childhood Trauma Questionnaire; Insomnia = SLEEP-50 Insomnia subscale; Narcolepsy = SLEEP-50 Narcolepsy subscale; Nightmares = SLEEP-50 Nightmares subscale; Daily functioning = SLEEP-50 Impact of Sleep Complaints on Daily Functioning subscale.
* $p < .05$.

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 (Giesbrecht & Merckelbach, 2004, 2006; Soffer-Dudek & Shahar, 2011; Watson, 2001). Our findings are in line with the Giesbrecht et al. (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, Giesbrecht, Knutelska, Sirroff, & Simeon, 2007; Guralnik, Schmeidler, & Simeon, 2000).

An important result of this study was that, 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. It is interesting that, at baseline assessment, 24% of the patients who completed treatment exceeded the clinical cutoff for dissociative disorders (i.e., >30; Bernstein-Carlson & Putnam, 1993); however, only 12% of the "completers" met this cutoff 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 psychopathological 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 psycho-

Table 5
Summary of Hierarchical Multiple Regression Analyses on the Dissociative Experiences Scale Subscales (All Change Scores; n = *195)*

Step	*B*	β	*t*(*df* = 2–5)	*p*	*r*	r^2
			Amnesia			
1						
Insomnia	−0.05	−.02	−0.27	.80	.39	.15
Narcolepsy	1.17	.20	2.59	.01*		
Nightmares	0.06	.01	0.18	.86		
Daily functioning	0.13	.04	0.49	.62		
Psychopathology	1.28	.28	3.40	.00*		
2						
Insomnia	−0.05	−.02	−0.24	.81	.39	.15
Narcolepsy	1.18	.20	2.61	.01*		
Daily functioning	0.14	.04	0.51	.61		
Psychopathology	1.27	.27	3.42	.00*		
3						
Narcolepsy	1.16	.19	2.61	.01*	.39	.15
Daily functioning	0.12	.04	0.48	.64		
Psychopathology	1.24	.27	3.54	.00*		
4						
Narcolepsy	1.24	.21	3.03	.00*	.39	.15
Psychopathology	1.32	.28	4.16	.00*		
			Absorption			
1						
Insomnia	−0.09	−.03	−0.41	.68	.36	.13
Narcolepsy	0.92	.14	1.80	.07		
Nightmares	0.28	.06	0.81	.42		
Daily functioning	0.18	.05	0.61	.55		
Psychopathology	1.44	.28	3.38	.01*		
2						
Narcolepsy	0.89	.13	1.76	.08	.36	.13
Nightmares	0.26	.05	0.76	.45		
Daily functioning	0.16	.05	0.54	.59		
Psychopathology	1.38	.27	3.44	.00*		
3						
Narcolepsy	0.99	.15	2.12	.04*	.36	.13
Nightmares	0.28	.06	0.83	.41		
Psychopathology	1.47	.29	4.07	.00*		
4						
Narcolepsy	1.05	.16	2.27	.02*	.35	.12
Psychopathology	1.44	.28	4.04	.00*		
			Depersonalization			
1						
Insomnia	−0.09	−.04	−0.44	.66	.35	.12
Narcolepsy	1.10	.18	2.34	.02*		
Nightmares	−0.08	−.02	−0.23	.82		
Daily functioning	0.19	.06	0.68	.50		
Psychopathology	1.13	.24	2.88	.00*		
2						
Insomnia	−0.10	−.04	−0.48	.64	.35	.12
Narcolepsy	1.09	.18	2.33	.02*		
Daily functioning	0.18	.06	0.66	.51		
Psychopathology	1.14	.24	2.97	.03*		
3						
Narcolepsy	1.05	.17	2.29	.02*	.35	.12
Daily functioning	0.16	.05	0.59	.56		
Psychopathology	1.08	.23	0.23	.00*		
4						
Narcolepsy	1.15	.19	2.73	.00*	.35	.12
Psychopathology	1.18	.25	3.60	.00*		

Note. Psychopathology = Psychopathology composite, consisting of total change scores on Beck's Anxiety Inventory, Brief Symptom Inventory, and Beck's Depression Inventory; CTQ = Childhood Trauma Questionnaire; Insomnia = SLEEP-50 Insomnia subscale; Narcolepsy = SLEEP-50 Narcolepsy subscale; Nightmares = SLEEP-50 Nightmares subscale; Daily functioning = SLEEP-50 Impact of Sleep Complaints on Daily Functioning subscale.
* $p < .05$.

Table 6
Fit Indices of the Nonmediation Model (1), the Partial Mediation Model (2), and the Full Mediation Model (3)

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

pathology, 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 and 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 posttreatment.

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 was comparable to the correlation between dissociation and narcolepsy. However, when we controlled for changes in general psychopathology, narcoleptic experiences

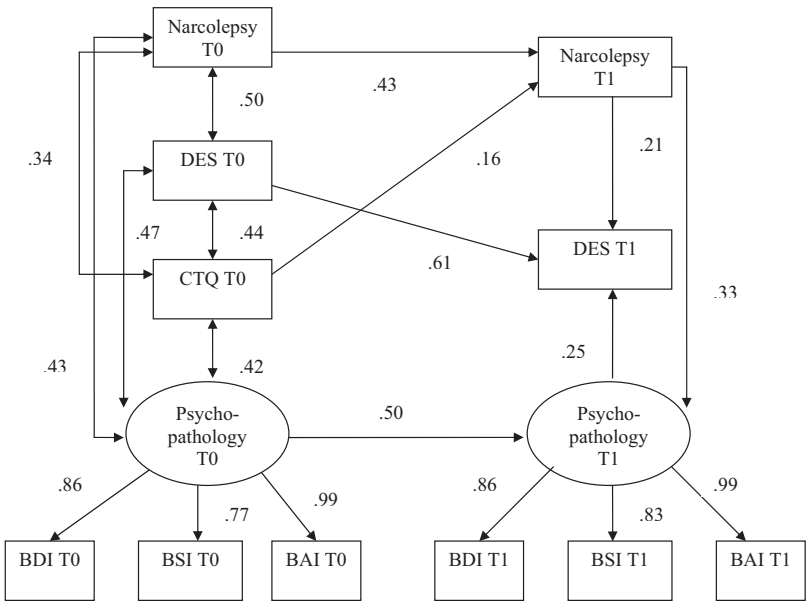

Figure 1. The partial mediation model. Structural equation modeling revealed that a partial mediation model best described the data. Reduction of dissociative symptoms was predicted by decrease in narcoleptic symptoms directly, as well as indirectly via improvement in general psychopathology. Note that 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.

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 affect 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 et al., 1999).

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. An important finding was that 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 (van der Kloet et al., 2011; Watson, 2001, 2003b).

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 multifaceted treatment components (e.g., sleep hygiene vs. 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 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.

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