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Sleep loss increases dissociation and affects memory for emotional stimuli

Dalena van Heugten – van der Kloet [b, *], Timo Giesbrecht [a], Harald Merckelbach [a]

[a] Maastricht University, The Netherlands
[b] Nuffield Department of Clinical Neurosciences, University of Oxford, Oxford, United Kingdom

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ABSTRACT

Background and objectives: Because of their dreamlike character, authors have speculated about the role that the sleep–wake cycle plays in dissociative symptoms. We investigated whether sleep loss fuels dissociative symptoms and undermines cognitive efficiency, particularly memory functioning.
Methods: Fifty-six healthy undergraduate students were randomly assigned to an experimental group ($n = 28$) and a control group ($n = 28$). The experimental group was deprived of sleep for 36 h in a sleep laboratory; the control group had a regular night of sleep. Sleepiness, mood, and dissociative symptoms were assessed 6 times in the experimental group (control group: 4 times). Several cognitive tasks were administered.
Results: Sleep deprivation led to an increase in dissociative symptoms, which was mediated by levels of general distress. Feelings of sleepiness preceded an increase of dissociative symptoms and deterioration of mood. Finally, sleep loss also undermined memory of emotional material, especially in highly dissociative individuals.
Limitations: Limitations included moderate reliability of the mood scale, limited generalizability due to student sample, and a relatively short period of intensive sleep deprivation rather than lengthy but intermittent sleep loss, representative of a clinical population.
Conclusions: We found that sleep deprivation had significant effects on dissociation, sleepiness, and mood. Specifically, sleepiness and dissociation increased during the night, while mood deteriorated. Our findings stress the importance of sleep deficiencies in the development of dissociative symptoms. They support the view that sleep disruptions fuel distress, but also degrade memory and attentional control. It is against this background that dissociative symptoms may arise.

1. Introduction

Dissociative symptoms refer to disturbances in the integration of thoughts, feelings, and experiences into consciousness and memory. They are prevalent in both general and clinical populations (Foote, Smolin, Kaplan, Legatt, & Lipschitz, 2006). In their most radical versions, they feature as 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 anxiety disorders such as Obsessive-Compulsive Disorder (Rufer, Fricke, Held, Cremer, & Hand, 2006; Watson, Wu, & Cutshall, 2004), Panic Disorder, and Agoraphobia (Cassano et al., 1989).

The etiology of dissociation has been the subject of intense debate (Bremner, 2010; Dalenberg et al., 2012; Giesbrecht, Lynn, Lilienfeld, & Merckelbach, 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).

An alternative perspective on the origins of dissociation focuses on the link between sleep and dissociation. Sleep problems and deficiencies have been implicated in the genesis of a variety of psychological disorders, including PTSD, Depression (Benca, 1996; Breslau, Roth, Rosenthal, & Andreski, 1996), and most recently

* Corresponding author. Nuffield Department of Clinical Neurosciences, University of Oxford, Level 6 West Wing, John Radcliffe Hospital, Headley Way, Oxford, OX3 9DU, United Kingdom.
E-mail address: Dalena.vanheugten@ndcn.ox.ac.uk (D. van Heugten – van der Kloet).

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Obsessive-Compulsive Disorder (Paterson, Reynolds, Ferguson, & Dawson, 2013). Watson (2001, 2003) provided the first evidence for a relationship between dissociation and unusual sleep experiences as measured by the Iowa Sleep Experiences Survey (ISES; Giesbrecht & Merckelbach, 2006; Watson, 2001, 2003) 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 ($r = .31–.55$; Van der Kloet, Merckelbach, Giesbrecht, & Lynn, 2012).

Adding to these correlational findings, an experimental study showed that acute dissociative symptoms of undergraduates intensify when their sleep–wake cycle is disrupted (Giesbrecht, Smeets, Leppink, Jelicic, & Merckelbach, 2007). 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 night. However, an important limitation of this study was the absence of a control group with undisturbed sleep.

Only recently, scientists have started to test the merits of the sleep-dissociation approach in a more rigorous way. For example, a longitudinal field study by Van der Kloet, Giesbrecht, and Merckelbach (2011) showed that in young people, sleepiness preceded an increase in dissociative symptoms, 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, Lynn, Giesbrecht, Merckelbach, & de Zutter, 2012). However, many questions remain regarding the specific links between sleep disturbances, dissociation, and their cognitive concomitants.

The marked influence of sleep disruption on performance and alertness has been documented by numerous studies (Jewett, Dijk, Kronauer, & Dinges, 1999; Williamson, Feyer, Mattick, Friswell, & Finlay-Brown, 2001). Another well-documented consequence of sleep–wake disruptions is their detrimental effect on memory (Frenda, Patihis, Loftus, Lewis, & Fenn, 2014; Hairston & Knight, 2004). Thus, disturbances in the sleep–wake cycle may undermine memory and attention, promoting absentmindedness and a propensity to produce memory commission errors, two well-established correlates of people scoring high on dissociation measures (Giesbrecht, Merckelbach, Geraerts, & Smeets, 2004; Giesbrecht, Merckelbach, Van Oorsouw, & Simeon, 2010; Merckelbach, Zeles, Van Bergen, & Giesbrecht, 2007).

So far, however, most studies documenting the relationships between sleep, cognitive dysfunctions, 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.

With this in mind and building on earlier findings of Giesbrecht et al. (2007), the present study addressed the following questions: 1) Can we replicate the correlations between sleep disturbances and dissociation that have been found in previous work? 2) Do 36 h of sleep deprivation increase dissociative *state* symptoms, along with memory commission errors (2a) and transient attentional problems (2b)? And 3) Is it the case that people with heightened levels of *trait* dissociation are the most vulnerable to the effects of sleep deprivation, relative to those low on trait dissociation?

2. Method

2.1. Participants

Participants were 56 healthy undergraduate students (43 women) enrolled at Maastricht University, with a mean age of 20.7 years (SD = 2.33, range = 18–29 years). 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.

Participants received written and oral information about the study during an intake session, 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 alcoholic drinks, no chocolate). The rationale of the study was not discussed with participants prior to the debriefing, but they were informed that they were going to stay awake the entire night and complete questionnaires every few hours. We confirmed absence of exclusion criteria with a brief interview, and familiarized the participants with the laboratory surroundings and equipment. All participants completed an online baseline screening. 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 (the equivalent of $150). Data collection was carried out by the first author, with help of two graduate students. 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.

2.2. Procedure

The night before the start of the study, participants reported having slept $M = 7.96$ h (SD = 1.08; indicated by their sleep logs) 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 assessing sleepiness, mood, and dissociative symptoms at baseline. The experimental group completed the same measures at 8 p.m., 11 p.m., 3 a.m., 9 a.m., and 1 p.m. Table 1 shows the time schedule for the experimental group. The control group completed the same measures at 8 p.m., and the next day at 9 a.m., and 1 p.m. Sleep deprived participants stayed in the sleep laboratory in groups of up to 6 people until 3 p.m. the next day and were not allowed to sleep. During the night, they were allowed a limited number of activities in between the questionnaires and cognitive tasks to keep themselves awake, such as: conversations, watching movies, reading, and short walks inside the building. They were not allowed to watch scary movies, play computer games, or physical activity as these might influence their test results. Control participants returned the next morning at 9 a.m. to the laboratory after a regular night of sleep at home ($M = 7.99$ h, SD = 1.05).

2.3. Measures

2.3.1. Baseline screening

Dissociative Experiences Scale (DES; Cronbach's $\alpha = .89$; all α's from current study). The DES (Bernstein & Putnam, 1986) is a self-report scale of trait dissociation. It requires participants to indicate

Table 1
Chronology of experimental group ($n = 28$).

Time		Activity
8 a.m.		Estimated wake-up time
5 p.m.	5:10 p.m.	Arrival at University
5:10 p.m.	5:45 p.m.	Baseline state questionnaires (CADSS, SSS, POMS)
5:45 p.m.	6:15 p.m.	Watching movie clips (negative, neutral, positive) + SAM
6:15 p.m.	7 p.m.	Meal
7 p.m.	7:30 p.m.	Free recall task, objective and subjective memory tasks
7:30 p.m.	8 p.m.	Relaxing
8 p.m.	8:30 p.m.	Measure 1 of state questionnaires (CADSS, SSS, POMS)
8:30 p.m.	9 p.m.	Attention Network Task
9 p.m.	11 p.m.	Relaxing
11 p.m.	11:30 p.m.	Measure 2 of state questionnaires (CADSS, SSS, POMS)
11:30 p.m.	0:30 a.m.	Relaxing
0:30 a.m.	1 a.m.	Light meal
1 a.m.	3 a.m.	Relaxing
3 a.m.	3:30 a.m.	Measure 3 of state questionnaires (CADSS, SSS, POMS)
3:30 a.m.	4:30 a.m.	Relaxing
5 a.m.	5:30 a.m.	Attention Network Task
5:30 a.m.	7 a.m.	Relaxing
7 a.m.	8 a.m.	Breakfast
8 a.m.	9 a.m.	Relaxing
9 a.m.	9:30 a.m.	Measure 4 of state questionnaires (CADSS, SSS, POMS)
9:30 a.m.	10:40 a.m.	Relaxing
10:40 a.m.	11:10 a.m.	Attention Network Task
11:10 a.m.	12 p.m.	Relaxing
12 p.m.	1 p.m.	Meal
1 p.m.	1:30 p.m.	Final measure of state questionnaires (CADSS, SSS, POMS)
1:30 p.m.	2:50 p.m.	Free recall task, objective and subjective memory tasks
2:50 p.m.	3:15 p.m.	Debriefing
3:15 p.m.		Participants are brought home by taxi

on 100 mm visual analog scales (anchors: 0 = never; 100 = always) to what extent they experience 28 dissociative experiences in daily life.

Cambridge Depersonalisation Scale (CDS; Cronbach's $\alpha = .92$). The CDS (Sierra & Berrios, 2000) consists of 29 items 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 to 10. Scores are summed to obtain a total CDS score (range: 0–290). Sierra and Berrios (2000) report sound psychometric properties for the CDS.

Iowa Sleep Experiences Survey (ISES; Cronbach's $\alpha = .83$). The ISES (Watson, 2001) consists of 18 questions to rate the frequency of various sleep- and dream-related experiences on a 7 point scale (anchors: 1 = never, 7 = several times a week). The ISES consists of 2 separate subscales that measure general sleep experiences and lucid dreaming.

SLEEP-50 (Cronbach's $\alpha = .88$). Sleep experiences were assessed with the 50-item Dutch version of the SLEEP-50 (Spoormaker, Verbeek, van den Bout, & Klip, 2005), which contains nine subscales that index sleep complaints and sleep disorders listed in DSM-IV (American Psychiatric Association, 2000). Each item is scored on a 4-point scale ranging from 0 (not at all) to 3 (very much). In previous research, the SLEEP-50 showed reasonable predictive validity for a broad range of sleep disorders, with overall sensitivity being .79, and specificity being .77 (Spoormaker et al., 2005). Analyses were based on the total score.

2.3.2. State measures

Clinician-Administered Dissociative States Scale (CADSS, Cronbach's $\alpha = .77–.89$). The CADSS (Bremner et al., 1998) is a 27-item scale with 19 subject-rated items, and 8 items scored by an observer. Items are scored on a 5-point scale (0 = not at all, 4 = extremely). Bremner et al. (1998) found the CADSS to be a highly reliable and valid instrument to measure present-state dissociative symptoms. In this study, only the self-report items were administered.

Stanford Sleepiness Scale (SSS). The SSS (Hoddes, Zarcone, Smythe, Phillips, & Dement, 1973) is widely used in sleep research (e.g., Babkoff, Caspy, Mikulicer, & Sing, 1991). It consists of a single item measuring subjective sleepiness that is rated on a 7-point scale with response options ranging from 'feeling active, vital, alert and awake' to 'I almost fall asleep, I struggle to remain awake'.

Profile of Mood States (POMS, Cronbach's $\alpha = .61–.94$). The Profile of Mood States – Short Form (POMS; McNair, Lorr, & Droppleman, 1992) is a self-report measure that is commonly used for 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). Data collected by De Groot (1991) supports the validity and reliability of the Dutch version of the POMS.

2.3.3. Memory measures

To measure memory functioning (i.e., commission errors), 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 min), 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 negative, neutral, and positive emotional responses that we wanted to study (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 45 min after stimulus offset, at 7 p.m. and once again after sleep deprivation, at 1 p.m. the following day:

2.3.3.1. 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, Merckelbach, & Kuijpers, 2003).

2.3.3.2. Subjective memory fragmentation (Kindt & Van den Hout, 2003). This was measured using three 100-mm visual analog 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 memory fragmentation to be most pronounced in the sleep deprivation group.

2.3.3.3. Objective memory fragmentation. This was measured along the lines of Wegner, Quillian, and Houston (1996). These authors developed a method 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 s, 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 that sleep deprivation would be associated with fragmented memory, especially in high dissociators (see also Giesbrecht et al., 2010).

2.3.4. Attentional problems measure

Attention Network Test (ANT; Fan, McCandliss, Somner, Raz, & Posner, 2002). The ANT has been devised to measure three different facets of attention within a single task: Orienting, alerting, 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. We expected that sleep loss would undermine attentional performance, particularly the executive (i.e., frontal) aspect of it (see Harrison, Horne, & Rothwell, 1997).

2.4. 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, with alpha set at $p < 0.01$ to adjust for the large number of correlations. State data were analyzed using the General Linear Model (GLM). We performed Analyses of Variance (ANOVA) with repeated measures, followed by multiple regression analyses. Significant multivariate results were evaluated with univariate tests. We applied Bonferroni adjustments to correct for the number of ANOVA's within each domain (e.g., we conducted three repeated measures ANOVA's to explore state dissociation and related constructs and therefore set alpha at 0.05/3 = 0.016). 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.

3. Results

3.1. Correlations between sleep and dissociation measures

Table 2 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 (Giesbrecht & Merckelbach, 2004, 2006). We computed change 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 2 illustrates several aspects relevant to our first research question. To begin with, dissociation measures (DES, CDS) were correlated positively with aberrant sleep experiences (ISES) and with sleep disturbances (SLEEP-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. 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 2).

3.2. Sleep deprivation and state dissociation

To address our second research question, we first investigated whether the two groups differed 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 eta$^2 = .43$, due the higher levels of SSS in the experimental group between at 9 a.m. and 1 p.m. (see Fig. 1). We also found a significant main effect of time, $F(1,54) = 13.28$, $p < .001$, partial eta$^2 = .20$. The repeated measures analyses revealed a main effect of group due to higher levels of SSS

Table 2
Mean scores and standard deviations of baseline measures and Pearson product−moment correlations between measures in undergraduate sample ($N = 56$; in bold: significant p-values).

	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 Depersonalisation Scale; ISES = Iowa Sleep Experiences Survey; SL 50 = The SLEEP-50; CADSS 5−8 = Clinician-Administered Dissociative States Scale, change score 5 p.m.−8 p.m.; CADSS 8−11 = Clinician-Administered Dissociative States Scale, change score 8 p.m.−11 p.m.; CADSS 11−3 = Clinician-Administered Dissociative States Scale, change score 11 p.m.−3 a.m.; CADSS 3−9 = Clinician-Administered Dissociative States Scale, change score 3 a.m.−9 a.m.; POMS 3−9 = Profile of Mood State, change score 3 a.m.−9 a.m.; SSS 11−3 = Stanford Sleepiness Scale, change score 11 p.m.−3 a.m.; SSS 3−9 = Stanford Sleepiness Scale, change score 3 a.m.−9 a.m.
*$p < .01$.

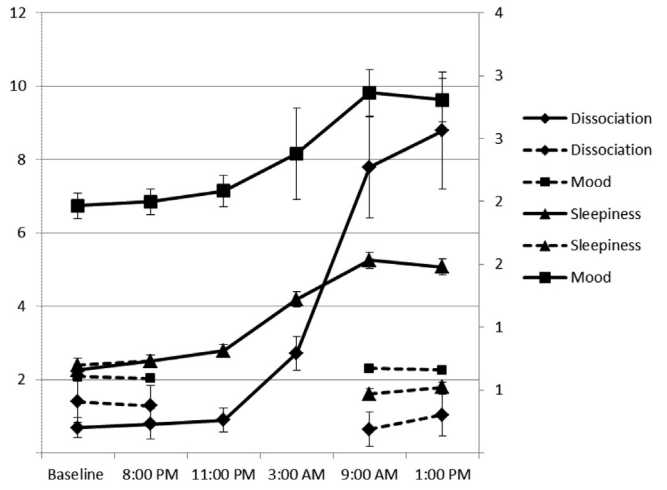

Fig. 1. Pattern of dissociation, sleepiness, and mood of the experimental group ($n = 28$) and the control group ($n = 28$; dotted lines) during the test period of the experiment. Left *Y*-axis depicts raw mean scores on state measures of dissociation and sleepiness. Right *Y*-axis depicts mean scores on state mood measure.

in the experimental group than in the control group, $F(1,53) = 23.17$, $p < .001$, partial eta$^2 = .30$.

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 eta$^2 = .31$, due to stronger increases in CADSS levels in the experimental group than in the control group between 9 a.m. and 1. p.m. (Fig. 1) We also found a significant main effect of time, $F(3,54) = 18.66$, $p < .001$, partial eta$^2 = .26$. Furthermore, the analyses revealed an overall difference in CADSS levels between the experimental group and the control group $F(1,54) = 14.83$, $p < .001$, partial eta$^2 = .22$.

Finally, we performed similar analyses on the POMS as an 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 eta$^2 = .27$, with the experimental group displaying stronger mood deterioration than the control group between 9 a.m. and 1. p.m. (Fig. 1). Furthermore, the ANOVA yielded a borderline significant main effect of time, $F(3,54) = 3.51$, $p = .02$, partial eta$^2 = .06$, but no significant effect of group, $F(3,54) = 2.66$, $p = .11$, partial eta$^2 = .05$.

In sum, we found significant increases in sleepiness and dissociation, as well as some mood deterioration over time, which were specifically pronounced in the experimental group.

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

To address the second research question in more depth, we next focused on the experimental group. Using repeated measures analyses with Bonferroni adjusted alpha's set at $p = 0.016$, we found a significant increase in sleepiness over test sessions, $F(5,26) = 70.51$, $p < .001$, partial eta$^2 = .53$. Significant linear increases were also evident for dissociation and mood deterioration, CADSS: $F(5,27) = 28.73$, $p < .001$, partial eta$^2 = .52$, and POMS: $F(5,27) = 16.06$, $p < .001$, partial eta$^2 = .37$, respectively.

Next, we examined the predictive power of mood and sleepiness on acute dissociation at time 4 (i.e., the increase in dissociation between 3 and 9 a.m.; see Fig. 1). To this end, we selected the Vigor and Tension-Anxiety subscales of the POMS to represent changes in positive and negative affect, respectively (Watson & Clark, 1994; Watson, Clark, & Tellegen, 1998). We used the following approach. We computed change scores for all variables from 3 to 9 a.m. and from 11 p.m. to 3 a.m. Using hierarchical multiple regression analyses, we entered three theoretically motivated blocks of predictors. CADDS change score between 3 and 9 a.m. served as dependent variable. First, we entered changes in sleepiness and mood between 3 and 9 a.m. in the first block. These changes served to control for any fluctuations in correlated variables during the same time frame. Second, we entered changes in mood between 11 p.m. and 3 a.m. This block would effectively control for any changes in mood that would lead to increases in dissociation indirectly. Third, we used changes in self-reported sleepiness to predict changes in dissociation over and above changes due to mood. The measures at time 4 were chosen because dissociation levels increased strongest at time 4 (between 3 and 9 a.m.). Table 3 gives a summary of the hierarchical multiple regression analyses. Altogether, the increase in sleepiness and decrease of positive affect between 11 p.m. and 3 a.m., as well as decreased positive and increased negative affect between 3 and 9 a.m., accounted for 62% of the variance in CADSS scores.

3.4. Memory functioning after sleep loss

Next, we explored the data to address potential memory effects (research question 2a). Before sleep deprivation, control and experimental participants watched three movie clips of positive, neutral, and negative emotional content. They completed memory measures twice; 45–60 min after they watched the three movie clips, and once again the next day. Analyses of the data obtained with the *Self-Assessment Manikin* (SAM; Bradley & Lang, 1994) revealed that for all movie clips the intended emotions were successfully induced (all *p*'s < .001). Two independent raters analyzed the free recall reports of participants. Moderate to strong significant

Table 3
Summary of hierarchical multiple regression analyses with dissociation (CADSS change score at time 4) as dependent variable ($n = 28$), all change scores (in bold: significant *p*-values).

Model	*B*	*β*	*t*	*p*	*r*	r^2
1					.73	.47
SSS 3–9	1.51	.25	1.26	.22		
POMS Vigor 3–9	−.62	−.46	−2.45	**.02**		
POMS Tension 3–9	1.51	.54	3.51	**<.01**		
2					.81	.58
SSS 3–9	1.04	.17	.90	.38		
POMS Vigor 3–9	−.76	−.57	−3.07	**<.01**		
POMS Tension 3–9	1.61	.58	4.06	**<.01**		
POMS Vigor 11–3	−.34	−.35	−2.05	**.05**		
POMS Tension 11–3	.64	.19	1.36	.19		
3					.85	.62
SSS 3–9	1.39	.23	1.25	.23		
POMS Vigor 3–9	−.85	−.63	−3.44	**<.01**		
POMS Tension 3–9	1.47	.53	3.81	**<.01**		
SSS 11–3	2.30	.46	2.06	**.05**		
POMS Sleep 11–3	−.26	−.27	−1.23	.23		
POMS Vigor 11–3	−.49	−.50	−2.45	**.02**		
POMS Tension 11–3	.45	.13	.91	.38		

Note. SSS 3–9 = Stanford Sleepiness Scale change score, increase in sleepiness between 3 a.m. and 9 a.m.; POMS Vigor 3–9 = Profile of Mood States Vigor subscale (positive affect), change score between 3 and 9 a.m.; POMS Tension 3–9: Profile of Mood States Tension-Anxiety subscale (negative affect), change score between 3 and 9 a.m.; SSS 11–3 = Stanford Sleepiness Scale change score, increase in sleepiness 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 Vigor11–3 = Profile of Mood States Vigor subscale (positive affect), change score between 11 p.m. and 3 a.m.; POMS Tension 11–3: Profile of Mood States Tension-Anxiety subscale (negative affect), change score between 11 p.m. and 3 a.m.

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 presented in Table 4.

Repeated measures ANOVA's, with alpha set at p = 0.016, revealed a borderline significant time × group interaction for the correct free recall of the positive movie clip, $F(1,53) = 10.41$, $p = .02$, partial eta^2 = .16, and a main effect of time, $F(1,53) = 7.29$, $p = <.01$, partial eta^2 = .12, together indicating that free recall tended to deteriorate over time in the experimental group. However, there was no overall difference between the experimental group and control group in the level of correct free recall of the positive movie clip, $F(1,53) = .60$, $p = .44$, partial eta^2 = .01. A main significant effect of time was found for the negative movie, $F(1,54) = 9.11$, $p < .01$, partial eta^2 = .14. That is, levels of correct recall for the negative clip were lower at the second measurement. However, there was no overall difference in levels of free recall between the two groups: $F(1, 54) = .33$, $p = .57$, partial eta^2 = .01. For the neutral movie clip, no differences between the groups or within the groups emerged, indicating that they did not differ in levels of neutral free recall (all F's < 2.75, all p's > .10).

With regard to commission errors when recalling the three clips, we found no differences when comparing levels before and after sleep deprivation in the experimental group, and neither were there differences between experimental and control group (all F's < 2.52 and all 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 F's < 2.26; all p's > .14).

3.5. Attentional problems after sleep loss

Then, we examined the attentional effects of sleep loss (research question 2b). Mean scores and standard deviations are presented in Table 4. 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, with alpha set at p = 0.016: alerting, orienting, and executive control. We found a significant main effect of time for the alerting component, indicating that there was a decrease in alerting scores between 8.30 p.m. and 11 a.m., $F(1,52) = 13.84$, $p < .01$, partial eta^2 = .21. The experimental and control group did not differ in alerting, $F(1,52) = 2.46$, $p = .12$, partial eta^2 = .05. No differences were found for the orienting component of the ANT, all F's < 3.18, all p's > .08. We found a significant time × group interaction for the executive control component of the ANT, $F(1,52) = 12.58$, $p < .01$, partial eta^2 = .20, and a main effect of group, $F(1,52) = 7.19$, $p < .01$, partial eta^2 = .12. 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 ANT three times: at 8.30 p.m., 5.00 a.m., and 11.00 a.m. Using repeated measures analyses with alpha set at p = .016, we found borderline significant main effects of time for the alerting component and executive control ($F(2,54) = 3.57$, $p = .04$; partial eta^2 = .12; $F(2,54) = 3.78$, $p = .03$, partial eta^2 = .12, respectively), but not for the orienting component of the ANT ($F(2,54) = 1.09$, $p = .34$). Thus, after sleep loss, reaction times became somewhat delayed compared to baseline indicating that alertness and vigilance became slower in participants, and executive control of attention deteriorated.

3.6. Trait dissociation and effects of sleep loss

Finally, to answer research question 3, we explored whether memory and attentional effects of sleep loss were specifically pronounced in participants with high trait dissociation (i.e., high DES). We performed a median split on DES scores within the experimental group. Using a 2 (high versus low DES) × 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 F's < 1.09, all p's > .36).

Using a median split, we also carried out 2 (high versus low DES) × time repeated measures ANOVA on the three ANT parameters. Fig. 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, $F(1,26) = 3.84$, $p = .06$, partial eta^2 = .13.

Table 4
Mean scores and standard deviations of memory and ANT measures (N = 56).

Measure	Mean (SD) Rater 1				Mean (SD) Rater 2			
	7 p.m.		1.30 p.m.		7 p.m.		1.30 p.m.	
	EXP	C	EXP	C	EXP	C	EXP	C
FR Positive	19.79 (14.67)	18.15 (8.73)	13.54 (9.19)	18.18 (7.63)	35.96 (19.46)	35.25 (13.99)	27.64 (16.51)	36.71 (14.57)
FR Neutral	11.21 (4.40)	13.44 (4.18)	9.46 (4.16)	12.21 (3.99)	20.89 (6.89)	23.21 (7.54)	20.39 (7.37)	22.25 (7.02)
FR Negative	23.07 (13.84)	22.46 (9.24)	16.71 (7.33)	20.25 (6.24)	38.57 (15.59)	38.82 (12.41)	32.46 (10.88)	34.61 (10.62)
Com Positive	.71 (.76)	.81 (.74)	.57 (.79)	.71 (.94)	.36 (.73)	.50 (.84)	.43 (.84)	.46 (.64)
Com Neutral	.71 (.90)	.93 (1.05)	.93 (.94)	.93 (.90)	.61 (1.10)	.32 (.61)	.46 (1.00)	.75 (1.18)
Com Negative	1.00 (1.05)	1.75 (1.08)	.93 (.86)	1.50 (.84)	.79 (1.13)	1.11 (.99)	1.32 (1.39)	1.21 (1.20)

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.

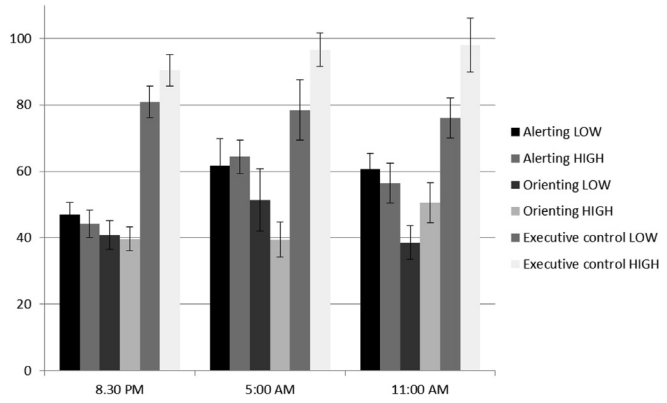

Fig. 2. Course of executive functioning (as measured by reaction time) after sleep deprivation in the experimental group ($n = 28$). We performed a median split based on high (light gray bars) and low dissociative (dark gray bars) participants. The bars depict the three parameters of the ANT subdivided for high and low dissociators (as measured by DES), 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 5.00 a.m., and the final measure (Time 3) at 11.00 a.m.

4. Discussion

The main aim of our study was to investigate whether 36 h sleep deprivation would enhance dissociative symptoms, sleepiness, and mood deterioration. Specifically, we hypothesized that sleep deprivation would increase dissociative symptoms, that sleepiness would predict the increase in dissociation, and that this effect would not be fully accounted for by a deterioration of mood.

The most important findings can be cataloged as follows. First, with regard to our baseline and trait measures (research question 1), we replicated the robust link between dissociative symptoms and unusual sleep experiences that has now been documented in many studies (see e.g., Agargun et al., 2003; Levin & Fireman, 2002; Giesbrecht & Merckelbach, 2004; 2006; Giesbrecht et al., 2007; Van der Kloet, Lynn, et al., 2012; Van der Kloet, Merckelbach, et al., 2012; Watson, 2001, 2003). Second, in accordance with Giesbrecht et al. (2007), we found that sleep deprivation was associated with increases in dissociation, sleepiness, and mood deterioration (research question 2).

Third, in contrast to our expectations and unlike the study of Giesbrecht et al. (2007), we found that changes in mood paralleled changes in dissociative symptoms. Our regression analyses showed that dissociative symptoms were also influenced by a deterioration of mood during 36 h of sleep deprivation. There might be procedural reasons as to why this pattern was not observed by Giesbrecht et al. (2007), such as differences in the administration of measures as well as different points of measurement during the night. Whatever the reasons, our findings do suggest that the dissociative effects of sleep loss have a mood component.

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 et al. (1991) who argued that sleepiness follows diurnal oscillations. Still, when we investigated these patterns in more detail, we 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, mood deterioration seemed to occur later in time than steep increases in sleepiness and dissociative symptoms. This suggests that at least in our sample, sleepiness precedes dissociative symptoms escalation and mood deterioration.

Fifth, we found that after sleep loss, participants in the experimental group tended to display poorer free recall of positive, but not of negative and neutral stimulus material (research question 2a). This accords well with the findings of Walker and Stickgold (2006) who found that after 36 h of sleep deprivation, participants exhibited a 40% memory retention deficit compared to controls who had slept, and this was specifically pronounced for positive emotional words. Our results are also comparable to findings of Walker and van der Helm (2009), who described the enhanced consolidation of emotional stimulus material across periods of sleep (rich in REM), compared with equal periods spent awake. Nishida, Pearsall, Buckner, and Walker (2008) found similar results using a napping paradigm and were able to link their findings to the unique neurobiology of REM sleep and right prefrontal theta power. However, unlike other authors (Frenda et al., 2014), we did not find an increase in commission errors in the sleep deprived participants; although we did find that their state dissociative levels went up. In a way, this pattern – stable commission errors and increasing dissociation – contradicts earlier studies that reported that dissociative tendencies are associated with pseudo-memories (Candel et al., 2003; Giesbrecht et al., 2007; Merckelbach et al., 2007). Perhaps, the link between dissociative tendencies and commission errors after sleep loss becomes only evident when the context includes an element of misinformation (see for an example Frenda et al., 2014). 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 undermined executive control functioning (research question 2b). This is consistent with studies that noted that sleep deprivation deregulates the frontal areas, thereby interfering with cognitive efficiency (Harrison et al., 1997; Horne, 1993). We also found that executive control problems due to sleep loss were somewhat more pronounced in highly dissociative individuals (research question 3). Admittedly, this effect reached only borderline significance and should be interpreted with caution. Future replications in a larger sample will make clear how solid this finding is, but at a theoretical level it is in line with studies that observed executive problems in dissociative individuals (Amrhein, Hengmith, Maragkos, & Hennig-Fast, 2008; Cima, Merckelbach, Klein, Shellbach-Matties, & Kremer, 2001).

Taken together, the results of the current study provide support for the sleep-dissociation model that stresses the importance of sleep deficiencies in the etiology of dissociative symptoms (e.g., Van der Kloet, Lynn, et al., 2012; Van der Kloet, Merckelbach, et al., 2012). By this view, sleep disruptions fuel distress, but also degrade cognitive control, and it is this combination that provides a fertile ground for dissociative symptoms. The sleep-dissociation model may also, at least partially, account for the cognitive deficiencies in highly dissociative individuals (Amrhein et al., 2008). For example, there is good evidence that disturbed sleep–wake cycles, dissociation, and cognitive failures overlap (Van der Kloet, Merckelbach, et al., 2012). A labile sleep–wake cycle may stem from a genetic propensity (Lang, Paris, Zweig-Frank, & Livesley, 1998), distressing trauma-related memories, or other unknown causal influences. Thus, the sleep-dissociation model does not preclude a scenario in which traumatic experiences disrupt sleep, thereby increasing vulnerability for dissociative symptoms.

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 α) 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. Furthermore, it would be worthwhile to look at how specific mood changes, e.g., positive and negative affect as measured by the *Positive and Negative Affect Schedule* (PANAS; Watson et al., 1988), covary with dissociative symptoms.

Second, the choice of the sleep deprivation manipulation does matter. The literature recommends using 24 h or a multiple of 24 h of sleep loss in order to distinguish clearly between sleep loss effects, diurnal effects, and interaction effects (Babkoff et al., 1991). However, to enhance the clinical relevance of sleep deprivation paradigms, it may be informative to study healthy participants who have to go through an extensive period of disrupted sleep (e.g., night shift workers).

Relatedly, our participants were undergraduates, who form a quite homogeneous group. Generalization of our results to the clinical domain is therefore limited. Replication of this study is warranted in larger and clinically more relevant groups. It would also 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 6−8 weeks (Van der Kloet, Lynn, et al., 2012).

In conclusion, the role of the sleep−wake cycle in fluctuations of dissociative symptoms, memory functions, and executive control remains complex. This study suggests that sleepiness due to sleep deprivation contributes to dissociative symptoms, an effect influenced by mood deterioration. Future studies in this domain could inspire new perspectives on treatment methods for dissociative symptoms. Improving the sleep−wake cycle may have considerable psychotherapeutic potential.

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References

Agargun, M. Y., Kara, H., Ozer, O. A., Selvi, Y., Kiran, U., & Ozer, B. (2003). Clinical importance of nightmare disorder in patients with dissociative disorders. *Psychiatry and Clinical Neurosciences, 57*, 575−579.

American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders (text revision)*. Washington, DC: Author.

Amrhein, C., Hengmith, S., Maragkos, M., & Hennig-Fast, K. (2008). Neuropsychological characteristic of highly dissociative healthy individuals. *Journal of Trauma & Dissociation, 9*, 525−542.

Babkoff, H., Caspy, T., Mikulicer, M., & Sing, H. C. (1991). Monotonic and rhythmic influences: a challenge for sleep deprivation research. *Psychological Bulletin, 109*, 411−428.

Bedard-Gilligan, M., & Zoellner, L. A. (2012). Dissociation and memory fragmentation in post-traumatic stress disorder: an evaluation of the dissociative encoding hypothesis. *Memory, 20*, 277−299.

Benca, R. M. (1996). Sleep in psychiatric disorders. *Sleep Disorders II, 4*, 739−764.

Bernstein, E., & Putnam, F. W. (1986). Development, reliability and validity of a dissociation scale. *Journal of Nervous and Mental Disease, 174*, 727−735.

Bradley, M. M., & Lang, P. J. (1994). Measuring emotion: the Self-Assessment Manikin and the semantic differential. *Journal of Behavior Therapy and Experimental Psychiatry, 25*, 49−59.

Bremner, J. D. (2010). Cognitive processes in dissociation: comment on Giesbrecht et al. (2008). *Psychological Bulletin, 136*, 1−6.

Bremner, J. D., Krystal, J. H., Putnam, F. W., Southwick, S. M., Marmar, C., Charney, D. S., et al. (1998). Measurements of dissociative states with the Clinical-Administered Dissociative States Scale (CADSS). *Journal of Traumatic Stress, 11*, 125−136.

Breslau, N., Roth, T., Rosenthal, L., & Andreski, P. (1996). Sleep disturbance and psychiatric disorders: a longitudinal epidemiological study of young adults. *Biological Psychiatry, 39*, 411−418.

Candel, I., Merckelbach, H., & Kuijpers, M. (2003). Dissociative experiences are related to commissions in emotional memory. *Behaviour Research and Therapy, 41*, 719−725.

Cassano, G. B., Petracca, A., Perugi, G., Toni, C., Tundo, A., & Roth, M. (1989). Derealization and panic attacks: a clinical evaluation on 150 patients with panic disorder/agoraphobia. *Comprehensive Psychiatry, 30*, 5−12.

Cima, M., Merckelbach, H., Klein, B., Shellbach-Matties, R., & Kremer, K. (2001). Frontal lobe dysfunctions, dissociation, and trauma self-reports in forensic psychiatric patients. *Journal of Nervous and Mental Disease, 189*, 188−190.

Dalenberg, C. J., Brand, B. L., Gleaves, D. H., Dorahy, M. J., Loewenstein, R. J., Cardeña, E., et al. (2012). Evaluation of the evidence for the trauma and fantasy models of dissociation. *Psychological Bulletin, 138*, 550−588.

De Groot, M. H. (1991). Psychometrische aspecten van een stemmingsschaal (verkorte POMS). *Gedrag en Gezondheid, 20*, 46−51.

Eriksen, B. A., & Eriksen, C. W. (1974). Effects of noise letters upon the identification of a target letter in a nonsearch task. *Perception & Psychophysics, 16*, 143−149.

Fan, J., McCandliss, B. D., Somner, T., Raz, A., & Posner, M. I. (2002). Testing the efficiency and independence of attentional networks. *Journal of Cognitive Neuroscience, 14*, 340−347.

Foote, B., Smolin, Y., Kaplan, M., Legatt, M. E., & Lipschitz, D. (2006). Prevalence of dissociative disorders in psychiatric outpatients. *American Journal of Psychiatry, 163*, 623−629.

Frenda, S. J., Patihis, L., Loftus, E. F., Lewis, H. C., & Fenn, K. M. (2014). Sleep deprivation and false memories. *Psychological Science*. http://dx.doi.org/10.1177/0956797614534694. OnlineFirst, Published on July 16, 2014.

Giesbrecht, T., Lynn, S. J., Lilienfeld, S. O., & Merckelbach, H. (2010). Cognitive processes, trauma, and dissociation − misconceptions and misrepresentations: reply to Bremner (2010). *Psychological Bulletin, 136*, 7−11.

Giesbrecht, T., & Merckelbach, H. (2004). Subjective sleep experiences are related to dissociation. *Personality and Individual Differences, 37*, 1341−1345.

Giesbrecht, T., & Merckelbach, H. (2006). Dreaming to reduce fantasy? − fantasy proneness, dissociation, and subjective sleep experiences. *Personality and Individual Differences, 41*, 697−706.

Giesbrecht, T., Merckelbach, H., Geraerts, E., & Smeets, E. (2004). Disruptions in executive functioning and dissociation in undergraduate students. *Journal of Nervous and Mental Disease, 192*, 567−569.

Giesbrecht, T., Merckelbach, H., Van Oorsouw, K., & Simeon, D. (2010). Skin conductance and memory fragmentation after exposure to an emotional film clip in de derpersonalisation disorder. *Psychiatry Research, 177*, 342−349.

Giesbrecht, T., Smeets, T., Leppink, J., Jelicic, M., & Merckelbach, H. (2007). Acute dissociation after 1 night of sleep loss. *Journal of Abnormal Psychology, 116*, 599−606.

Hairston, I. S., & Knight, R. T. (2004). Neurobiology: sleep on it. *Nature, 430*, 27−28.

Harrison, Y., Horne, J., & Rothwell, A. L. (1997). Sleep loss performance decrements on frontal lobe tasks show no improvement with caffeine. *Sleep Research, 26*, 616.

Hoddes, E., Zarcone, V., Smythe, H., Phillips, R., & Dement, W. C. (1973). Quantification of sleepiness: a new approach. *Psychophysiology, 10*, 431−436.

Holmes, E. A., Brown, R. J., Mansell, W., Fearon, R. P., Hunter, E. C. M., Frasquilho, F., et al. (2005). Are there two qualitatively distinct forms of dissociation? A review and some clinical implications. *Clinical Psychology Review, 25*, 1−23.

Horne, J. A. (1993). Human sleep, sleep loss and behavior − implications for the prefrontal cortex and psychiatric-disorder. *British Journal of Psychiatry, 162*, 413−419.

Jewett, M. E., Dijk, D.-J., Kronauer, R. E., & Dinges, D. F. (1999). Dose-response relationship between sleep duration and human psychomotor vigilance and subjective alertness. *Sleep, 22*, 171−179.

Kindt, M., & Van den Hout, M. (2003). Dissociation and memory fragmentation: experimental effects on meta-memory but not on actual memory performance. *Behaviour Research and Therapy, 41*, 167−178.

Lang, K. L., Paris, J., Zweig-Frank, H., & Livesley, W. J. (1998). Twin study of dissociative experiences. *Journal of Abnormal Psychology, 186*, 345−351.

Levin, R., & Fireman, G. (2002). Nightmare prevalence, nightmare distress, and self-reported psychological disturbance. *Sleep, 25*, 205−212.

McNair, D. M., Lorr, M., & Droppleman, L. F. (1992). *Profile Of Mood States (POMS) manual*. San Diego, CA: EdITS.

Merckelbach, H., Zeles, G., Van Bergen, S., & Giesbrecht, T. (2007). Trait dissociation and commission errors in memory reports of emotional events. *American Journal of Psychology, 120*, 1−14.

Nishida, M., Pearsall, J., Buckner, R. L., & Walker, M. P. (2008). REM sleep, prefrontal theta, and the consolidation of human emotional memory. *Cerebral Cortex, 19*, 1158−1166.

Paterson, J. L., Reynolds, A. C., Ferguson, S. A., & Dawson, D. (2013). Sleep and Obsessive-Compulsive Disorder (OCD). *Sleep Medicine Review, 17*, 465−474.

Posner, M. I. (1980). Orienting of attention. *Quarterly Journal of Experimental Psychology, 32*, 3−25.

Rottenberg, J., Ray, R. R., & Gross, J. J. (2007). Emotion elicitation using films. In J. A. Coan, & J. J. B. Allen (Eds.), *The handbook of emotion elicitation and assessment* (pp. 9−28). New York: Oxford University Press.

Rufer, M., Fricke, S., Held, D., Cremer, J., & Hand, I. (2006). Dissociation and symptom dimensions of obsessive-compulsive disorder − a replication study. *European Archives of Psychiatry and Clinical Neuroscience, 256*, 146−150.
Sierra, M., & Berrios, G. E. (2000). The Cambridge Depersonalisation Scale: a new instrument for the measurement of depersonalization. *Psychiatry Research, 93*, 163−164.
Spiegel, D., Loewenstein, R. J., Lewis-Fernández, R., Sar, V., Simeon, D., Vermetten, E., et al. (2011). Dissociative disorders in DSM 5. *Depression & Anxiety, 28*, 824−854.
Spoormaker, V. I., Verbeek, I., van den Bout, J., & Klip, C. (2005). Initial validation of the SLEEP-50 questionnaire. *Behavioural Sleep Medicine, 3*, 227−246.
Van der Kloet, D., Giesbrecht, T., & Merckelbach, H. (2011). Slaperigheid voorspelt dissociatieve symptomen: een veldstudie [Sleepiness predicts dissociative symptoms: a field study]. *Tijdschrift voor Psychiatrie [Journal of Psychiatry], 53*, 757−763.
Van der Kloet, D., Lynn, S. J., Giesbrecht, T., Merckelbach, H., & de Zutter, A. (2012). Sleep normalization and decrease in dissociative experiences: evaluation in an inpatient sample. *Journal of Abnormal Psychology, 121*, 140−150.
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.
Walker, M. P., & van der Helm, E. (2009). Overnight therapy? The role of sleep in emotional brain processing. *Psychological Bulletin, 135*, 731−748.
Walker, M. P., & Stickgold, R. (2006). Sleep, memory, and plasticity. *Annual Review of Psychology, 57*, 139−166.
Watson, D. (2001). Dissociation of the night: individual differences in sleep-related experiences and their relation to dissociation and schizotypy. *Journal of Abnormal Psychology, 110*, 526−535.
Watson, D. (2003). To dream, perchance to remember: individual differences in dream recall. *Personality and Individual Differences, 34*, 1271−1286.
Watson, D., & Clark, L. E. (1994). *The PANAS-X: Manual for the positive and negative affect schedule-expanded form*. Ames: The University of Iowa.
Watson, D., Clark, L. A., & Tellegen, A. (1988). Development and validation of brief measures of positive and negative affect: the PANAS scales. *Journal of Personality and Social Psychology, 54*, 1063.
Watson, D., Wu, K. D., & Cutshall, C. (2004). Symptom subtypes of obsessive-compulsive disorders and their relation to dissociation. *Anxiety Disorders, 18*, 435−458.
Wegner, D. M., Quillian, F., & Houston, C. E. (1996). Memories out of order: thought suppression and the disturbance of sequence memory. *Journal of Personality and Social Psychology, 71*, 680−691.
Williamson, A. M., Feyer, A. M., Mattick, R. P., Friswell, R., & Finlay-Brown, S. (2001). Developing measures of fatigue using an alcohol comparison to validate the effects of fatigue on performance. *Accident Analysis and Prevention, 33*, 313−326.
Yu, J., Ross, C. A., Keyes, B. B., Li, Y., Dai, Y., Zhang, T., et al. (2010). Dissociative disorders among Chinese inpatients diagnosed with schizophrenia. *Journal of Trauma & Dissociation, 11*, 358−372.