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Sleep loss increases dissociation and affects memory for emotional stimuli

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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

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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