The effects of sleep restriction and sleep deprivation in producing false memories

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Abstract
False memory has been claimed to be the result of an associative process of generalisation, as well as to be representative of memory errors. These can occur at any stage of memory encoding, consolidation, or retrieval, albeit through varied mechanisms. The aim of this paper is to experimentally determine: (i) if cognitive dysfunction brought about by sleep loss at the time of stimulus encoding can influence false memory production; and (ii) whether this relationship holds across sensory modalities. Subjects undertook both the Deese-Roedigger-McDermott (DRM) false memory task and a visual task designed to produce false memories. Performance was measured while subjects were well-rested (9h Time in Bed or TIB), and then again when subjects were either sleep restricted (4h TIB for 4 nights) or sleep deprived (30h total SD). Results indicate (1) that partial and total sleep loss produced equivalent effects in terms of false and veridical verbal memory, (2) that subjects performed worse after sleep loss (regardless of whether this was partial or total sleep loss) on cued recognition-based false and veridical verbal memory tasks, and that sleep loss interfered with subjects’ ability to recall veridical, but not false memories under free recall conditions, and (3) that there were no effects of sleep loss on a visual false memory task. This is argued to represent the dysfunction and slow repair of an online verbal associative process in the brain following inadequate sleep.

1. Introduction
Sleep is vital for optimal functioning during wakefulness, and sleep loss has wide ranging effects on overall neurocognitive performance (Ratcliff & Van Dongen, 2009). Numerous studies have shown that sleep loss has a deleterious impact on basic elements of cognitive functioning, such as attentional processing (Van Dongen, Maislin, Mullington, & Dinges, 2003), response inhibition (Drummond, Paulus, & Tapert, 2006), and working memory (Drummond, Anderson, Straus, Vogel, & Perez, 2012; Turner, Drummond, Salamat, & Brown, 2007); as well as elements of higher cognitive functioning, such as rule based learning (Whitney, Hinson, Jackson, & Van Dongen, 2014), memory encoding (Drummond et al., 2000; Mander, Santhanam, Saletin, & Walker, 2011; Saletin & Walker, 2012; Yoo, Hu, Gujar, Jolesz, & Walker, 2007) and the ability to plan and implement intentions (Diekelmann, Wilhelm, Wagner, & Born, 2013a, 2013b). Sleep has also recently been suggested to play a role in associative memory formation (Lewis & Durrant, 2011; Payne, 2011; Stickgold & Walker, 2013); for instance, in the selective learning of relevant (as opposed to irrelevant) information (van Dongen, Thelen, Takahama, Barth, & Fernández, 2012; Wilhelm et al., 2011), rules governing sets of stimuli (Durrant, Taylor, Cairney, & Lewis, 2011) and the generalisation of specific learning for general application (Lau, Alger, & Fishbein, 2011). The impact of sleep loss on these latter functions is, however, relatively unknown.

It must also be acknowledged that the impacts of sleep on cognitive functioning are not always obvious (Killgore, 2010). For instance, studies have found differential effects of sleep loss on different components of both working memory (Drummond et al., 2012; Turner et al., 2007) and executive functioning (Tucker, Whitney, Belenky, Hinson, & Van Dongen, 2010). This is likely due to the fact that the cognitive systems of the brain are comprised of interacting subcomponents, and rarely does a cognitive
have used the DRM false memory task (Roediger & McDermott, 1995). While this is a valid and reliable method of eliciting false memories, false memories can occur in nonverbal domains as well (Frenda et al., 2014). In all, there are still several issues that need to be addressed in the false memory, sleep and memory, and sleep research literatures before a comprehensive understanding of false memory in the context of sleep is available.

Here, we address some of these issues by assessing the impact of both TSD and the purportedly equivalent period of PSD (i.e., sleep loss (SL) of either 30h time awake; and 4h TIB for four nights; Van Dongen et al., 2003), in comparison to a well-rested baseline (WR), on false memory generation in both the verbal and visual domains. This allows us to test: whether false memory generation is differentially impacted by PSD and TSD; whether different modalities of false memory are influenced in the same manner as one another; and whether different types of memory (free recall and recognition) are differentially effected by PSD and TSD. We hypothesised that: (i) PSD and TSD will not significantly differ in their effects on veridical and false memory; (ii) sleep loss (PSD and TSD) will increase the rates of false memory production; and that (iii) sleep loss will increase false memory in both verbal and visual modalities.

### 2. Methods

#### 2.1. Participants

44 healthy, normally functioning individuals (25F, 19 M; Mage = 24.9 ± 5.29y) gave informed consent and participated in the study. Subjects were screened for sleep disorders, drug use, axis I psychiatric conditions and medical disorders through a combination of structured interview and laboratory testing. To be included in the study, subjects had to report maintaining regular sleep-wake schedules (7–9h TIB, with bed times of 0000-0600 and wake times of 0600-0800).

#### 2.2. Procedure

Subjects maintained their normal sleep schedules for one week prior to participation. Adherence to at-home schedules was verified with wrist actigraphy, voicemail call-ins and sleep diaries. They then undertook both a well-rested condition (9h TIB for six nights; 4 at home followed by 2 in the laboratory) and either a total sleep deprivation (30h total) or sleep restriction (4h TIB for four nights) condition. Participation in the partial or total sleep loss conditions was randomised, and order of condition (rested or deprived) was counterbalanced across subjects. The well-rested sleep schedule was based on each subject’s habitual sleep schedule at home. If they did not report normally spending 9h in bed per night, sleep time was extended from their habitual schedule equally in the evening and morning to achieve a 9-h window. Similarly, the sleep restriction schedule was determined by shrinking time in bed equally in the evening and the morning. While in the laboratory, subjects were monitored with actigraphy throughout the day and night, and were monitored with polysomnography during sleep periods. Wakefulness during total sleep deprivation (TSD) and partial sleep deprivation (PSD) was guaranteed through staff interaction, and subjects were allowed to play games, watch television and browse the internet. Stimulant and alcohol consumption was prohibited 48h before entering the laboratory and during the lab stay. Test administration was scheduled at 5h post habitual wake time in the TSD condition and 7h post habitual wake time in the PSD and well-rested conditions. This variation was due to constraints imposed by other aspects of the study design. Given the relatively neutral circadian time represented by the mid-day
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