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Declarative memory consolidation during the first night in a sleep lab: The role of REM sleep and cortisol

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Summary While the consolidation of declarative memory is supported by slow wave sleep (SWS) in healthy subjects, it has been shown to be associated with rapid eye movement (REM) sleep in patients with insomnia. Sleep during a subject's first night in an unfamiliar environment is often disturbed, and this so-called first-night effect (FNE) has often been used as a model of transient insomnia. Additionally, sleeping for the first time in an unfamiliar environment can lead to increased cortisol secretion, and declarative memory consolidation likely depends on low cortisol levels, especially during the early part of the night. Accounting for intersubject variability in the FNE, we examined the relationship between sleep stages, cortisol secretion and declarative memory performance in 27 healthy young men. Declarative memory performance improved significantly after sleep. Whereas memory performance during the learning session and retrieval testing was strongly associated with cortisol secretion, the overnight gain was not. Post hoc analyses indicated that the overnight gain appears to be modulated by the extent of the FNE: a significant overnight improvement in memory performance was found only in subjects with a weak FNE ($n = 12$). In these subjects, no association was found between any sleep stage and the improvement observed in their memory performance. In subjects with a strong FNE ($n = 12$), however, the overnight change in memory performance was associated with the proportion of REM sleep and the total number of REMs. Disturbed sleep in an unfamiliar environment therefore appears to affect the memory consolidation process.

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1. Introduction

There is growing evidence that sleep plays a crucial role in memory consolidation (Diekelmann and Born, 2010). For declarative memory, which involves the conscious recall of facts and events, non-REM sleep (sleep stages 2—4) and especially slow wave sleep (SWS, sleep stages 3 and 4) appear to be of great importance. Taking advantage of the uneven distribution of sleep stages throughout the night, several studies have shown that subjects performed better on a word pair association task after sleeping the first half of the night, which is rich in SWS, than they did after sleeping the second half of the night, in which REM sleep predominates (Yaroush et al., 1971; Barrett and Ekstrand, 1972; Fowler et al., 1973; Plihal and Born, 1999a). Given that the proportion of time spent in stage 2 sleep is comparable in both halves of the night, it was possible in these studies to rule out a primary role for stage 2 sleep (Fowler et al., 1973; Plihal and Born, 1999a). Along these lines, performance on a word pair association task improved in subjects who slept only the first half of the night to the same extent as it did in subjects who had a full night of sleep, indicating that the first hours of sleep confer the greatest memory improvement (Tucker and Fishbein, 2009). Moreover, transcranial direct current stimulation, which enhances the slow oscillations that characterize SWS, has been found to improve recall in a word pair association task (Marshall et al., 2004, 2006). In contrast, a significant correlation between declarative memory performance and the proportion of REM sleep in insomnia patients was demonstrated by Backhaus et al. (2006), who suggest that this may be due to a compensatory mechanism in disturbed sleep.

The results of half-night experiments, however, may be influenced by factors other than sleep, such as those subject to circadian organization. Importantly, declarative memory consolidation is also modulated by the secretion of cortisol (Het et al., 2005; Wolf, 2009). The activity of the hypothalamic-pituitary-adrenocortical (HPA) axis displays a circadian rhythm, which is reflected in high levels of cortisol secretion in the early morning, a decline throughout the day, a prolonged quiescent period of low levels centered around midnight, and a rapid rise during the second half of the night (Weitzman et al., 1971; Van Cauter and Refetoff, 1985). The circadian rhythm of cortisol secretion is also influenced by sleep. Sleep onset appears to have an inhibitory effect on cortisol secretion that persists for one to two hours (Van Cauter and Refetoff, 1985; Born et al., 1988), and low cortisol levels have been shown to be associated with a greater proportion of SWS (Follenius et al., 1992). It is still a matter of debate, however, whether SWS inhibits the activity of the HPA axis or whether decreased HPA tone promotes deep sleep (Balbo et al., 2010). Nocturnal awakenings are related to pulsatile releases of cortisol, followed by a temporary inhibition of cortisol secretion (Späth-Schwalbe et al., 1991; Follenius et al., 1992), whereas the final morning awakening elicits a marked and rapid rise in cortisol levels persisting for about 60 min independent of whether the awakening occurs spontaneously or is triggered externally (Pruessner et al., 1997).

In recent studies, the interaction between cortisol secretion and sleep has also been investigated in relation to memory consolidation. Enhancing glucocorticoid activity during the first half of the night by intravenously administering hydrocortisone has been shown to impair the consolidation of declarative memory for word pairs (Plihal and Born, 1999b). Correspondingly, administering the glucocorticoid receptor agonist dexamethasone blocked the beneficial effect of early, SWS-rich sleep on the recall of word pairs (Plihal et al., 1999). Moreover, in patients suffering from primary insomnia, several studies have found that cortisol levels are elevated, especially in the early part of the night (Vgontzas et al., 2001; Rodenbeck et al., 2002) and that word pair recall after sleep was poor (Backhaus et al., 2006).

A subject's first night in a sleep laboratory differs from subsequent nights in terms of sleep architecture: total sleep time (TST), time spent in REM sleep, and sleep efficiency are diminished, whereas REM latency and intermittent wake time are increased (Agnew et al., 1966; Browman and Cartwright, 1980; Toussaint et al., 1995). This well-known first-night effect, which appears to vary in magnitude between individuals, has often been used as a model of transient insomnia (Roehrs et al., 1990; Roth et al., 1995; Erman et al., 2004; Rosenberg et al., 2007; Zammit et al., 2009). In addition to sleep, other biological systems, such as the HPA axis, may also be altered by the first-night effect. The HPA axis is one of the most important mediators of an organism's response to acute physical and psychological stress, which leads to increased cortisol secretion. In addition to varying degrees of sleep disturbance, sleeping for the first time in a sleep laboratory may lead to increased cortisol secretion in some subjects, resulting in substantial variability both in sleep parameters and cortisol levels. The aim of the present study was to examine the interaction between sleep stages, cortisol secretion and sleep-dependent memory consolidation during the first night in a sleep lab. We hypothesized that declarative memory performance would be associated with the proportion of time spent in REM sleep rather than with the proportion of time spent in SWS. We also expected that high cortisol levels would be associated with worse performance on the memory task.

2. Methods

2.1. Subjects

A total of 32 healthy male subjects aged 18—39 years (mean: 27.3 years) were included in the study. The study protocol was approved by the local ethics committee. All subjects provided written informed consent and underwent physical and mental health examinations before participating. None had a history of drug or alcohol abuse, or of neurological, psychiatric, or sleep disorders. As confirmed by the Pittsburgh Sleep Quality Index (Buysse et al., 1989), no subject had suffered from poor sleep within four weeks prior to study entry (mean Pittsburgh Sleep Quality Index: 2.9). As confirmed by the Munich Chronotype Questionnaire (Roenneberg et al., 2003), none of the subjects demonstrated an extreme chronotype (mean Munich Chronotype Questionnaire score: 3.9). All subjects were accustomed to going to bed between 2200 h and 2400 h and to rising between 0600 h and 0800 h. Of the 32 subjects, 28 were non-smokers and four were smokers. Subjects were instructed to abstain (a) from taking any medication during, and at least eight days prior to, study