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Reconsidering the Role of Sleep for Motor Memory

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Previous studies suggest that sleep may play an important role in memory consolidation of motor skills. It has been difficult, however, to tease apart the effect of sleep from circadian and homeostatic factors. We examined the effect of sleep on a popular motor sequence task, utilizing a design that controlled for time of day and time since sleep between wake and sleep groups. When these factors were controlled, there was no benefit of sleep to motor memory, suggesting that previous work may have been influenced by circadian and homeostatic confounds.

Keywords: motor skill, sleep, consolidation, learning

Sleep has been postulated to play an active role in the consolidation of motor memories (Stickgold, 2005), although not without controversy (for a review, see Siegel, 2005; Vertes, 2004; Vertes & Siegel, 2005). Most studies use a task in which subjects repeatedly tap out an explicit, known sequence. The typical design involves a wake group in which subjects are trained in the morning and tested 12 hr later and a sleep group in which subjects are trained at night and tested 12 hr later. Both groups are trained and tested by interleaving 30-s blocks of tapping followed by 30 s of rest, with 12 blocks of training and 2 blocks during the test. The sleep group typically has faster and more accurate responses on the test than at the end of training, whereas the wake group does not. The reported improved performance after a sleep period (i.e., sleep enhancement) has been taken as evidence for sleep-dependent consolidation, an active replay process that occurs solely during sleep (Walker, 2005).

Rickard, Cai, Rieth, Jones, and Ard (2008), however, argued that the sleep enhancement effect in the explicit sequence task is instead the result of data averaging and perhaps also time-of-day (circadian) and time-since-sleep (homeostatic) confounds. They showed that there are strong reactive inhibition effects (Hull, 1943) that build up during each 30-s block, the result of which is a roughly linear worsening of performance within each block. This effect is pronounced at the end of 12 blocks of training, perhaps because of a gradual build-up of fatigue over the training session. When those reactive inhibition effects are reduced by either limiting analysis to the first few sequences of each block or by experimentally reducing the build-up of fatigue during training, the sleep enhancement effect was eliminated.

However, the sleep group still performed better than the wake group at test. Rickard et al. (2008) advanced two candidate accounts of that effect. First, sleep consolidation may yield protection from forgetting rather than performance enhancement. Second, the group differences may reflect circadian and/or homeostatic confounds.

Researchers have tried different methods to get around the circadian and homeostatic confounds when comparing awake versus sleep groups. One method is testing and training at the same time of day, with one group having nocturnal sleep between sessions and the other group being sleep deprived. With this design, the sleep-deprived group showed worse performance than the sleep group at 48 hr from training (with a night of recovery sleep; Fischer, Hallschmid, Elsner, & Born, 2002; Fischer, Nitschke, Melchert, Erdmann, & Born, 2005). This design effectively controls for circadian and homeostatic effects. However, the effects may be caused by nonsleep-related factors associated with the method of deprivation, such as stress (Campbell, Guinan, & Horowitz, 2002; Plihal, Krug, Pietrowsky, Fehm, & Born, 1996). Moreover, the fatigue associated with sleep deprivation may produce performance deficits, even after a night of recovery sleep. One recent imaging study found that it takes more than one night of recovery sleep for the human brain to return to normal use of its neural network for a declarative task (McKenna, Meloy, Wetherell, Stricker, & Drummond, in press). This suggests that sleep-deprivation produces changes in brain state that could, in theory, impair normal time-based consolidation that can occur in wake or sleep.

Another method to control for circadian rhythm (but not homeostatic factors) is to use a daytime training-test interval containing either a nap or a rest period. The results from studies using the nap paradigm are inconsistent (Korman, Doyon, Doljansky, Carrier, Dagan, & Karni, 2007; Nishida & Walker, 2007; Tucker, Hirota, Wamsley, Lau, Chaklader, & Fishbein, 2006; Mednick, Cai, Kanady, & Drummond, 2008), with only some showing better test performance after a nap. Even given positive results, it is difficult to know whether the sleep advantage is due to sleep-specific processing, passive reduction of interference while sleeping, or increased general alertness during test compared with the non-nap group. Last, an alternative design that controls circadian factors is to test subjects 24, 48, or 72 hr after training (Walker, Brakefield, Hobson, & Stickgold, 2003; Walker, Brakefield, Seidman, Morgan, Hobson, & Stickgold, 2003). Although this controls

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for time of day, this does not allow for comparison between wake and sleep groups.

Given that there appears to be no ideal design for comparing waking and sleeping groups, it is prudent to use a variety of approaches with different strengths and weaknesses. A successful theory of sleep consolidation must then account for the results from all of the different approaches. The present study was conducted in this spirit. We used a new design that controls for circadian and homeostatic factors at the cost of possible time duration effects.

Method

Subjects

A total of 79 undergraduate students participated for course credit. All subjects were right-handed. Nineteen subjects reported practicing between sessions, 7 additional subjects reported napping between sessions, and 10 additional subjects reported not getting at least 6 hr of sleep the previous night. All of these subjects were eliminated from the primary data analyses described later, leaving data from 43 subjects.

There were three groups: a wake ($n = 15$) group, a 1-night ($n = 17$) group, and a 2-night ($n = 11$) group. All groups were trained within an hour and half of 9:30 a.m. The wake group returned for testing at 5:30 p.m. of Day 1 (8-hr delay). The 1-night group was tested at 5:30 p.m. of Day 2 (32-hr delay) after a night of sleep posttraining. The 2-night group was tested at 5:30 p.m. of Day 3 (56-hr delay) after 2 nights of sleep posttraining. Hence, whereas the time of training and testing was the same among the groups, eliminating circadian and homeostatic effects between groups, the delay interval differed.

It is important to note that there is no a priori reason to believe that our approach of controlling for circadian and homeostatic factors at the cost of differing delay intervals is more problematic than the usual design that controls for delay interval at the cost of circadian and homeostatic differences. The main potential problem of allowing delay duration to differ between groups is that forgetting (i.e., worsening of task performance at test) may increase with a longer delay, an effect that would tend to mask any enhancement effect for the sleep groups. However, work to date clearly indicates no forgetting effect for delay durations of 24 to 72 hr that include sleep (Walker, Brakefield, Hobson, & Stickgold, 2003; Walker, Brakefield, Seidman, et al., 2003).

Procedure

We adapted the sequential finger tapping task from Walker, Brakefield, Morgan, Hobson, and Stickgold (2002), which required subjects to repeatedly complete, with their left (nondominant) hand, the sequence 4–1–3–2–4. The numeric sequence (4–1–3–2–4) was displayed at the top of the screen at all times to exclude any working memory component to the task. Each key press produced a white dot below the correct digit, forming a row from left to right over the course of each key press sequence. Both the training session and the testing session consisted of 20 blocks, with each followed by a 30-s rest period. Each block ended after 20 sequences (100 key presses), rather than after 30 s, as in previous studies. Fixing the number of sequences rather than the time per block simplifies data analysis because each subject has the same number of key press trials in each block, and it may motivate subjects to perform faster so that they can finish the experiment sooner. We included 20 blocks in the testing session to explore whether sleeping versus waking intervals have any effect on rate of subsequent learning. Immediately after the test session, subjects were administered the Stanford Sleepiness Scale (Hoddes, Zarcone, Smythe, Phillips, & Dement, 1973) and completed a questionnaire in which they reported hours slept on the night before the testing session, whether they napped between sessions, and whether (and how long) they practiced between sessions.

Results

In the 1-night group, the mean reported number of hours slept on the night after training was 7.47 ($SD = 1.46$). In the 2-night group, the mean reported number of hours slept after training was 6.82 ($SD = 1.12$). There were no significant differences across groups for the Stanford Sleepiness Scale; means were 2.87 ($SD = 1.36$), 3.06 ($SD = 1.30$), and 2.91 ($SD = 1.58$) for the wake, 1-night, and 2-night groups, respectively.

To roughly equate the amount of data averaging in the analyses of this experiment with that of previous experiments (Walker, Brakefield, Seidman, et al., 2003), which compared groups with 24-hr differences in delay interval, we compared error and response time (RT) data averaged over the last four training blocks with data averaged over the first four test blocks. Critical results were not specific to this amount of averaging.

Accuracy was consistently high for all groups. Mean key press accuracies for the last four blocks of training were .97, .98, and .98 for the wake, 1-night, and 2-night groups, respectively. For the first four blocks of the test session, these values were .98, .99, and .99, respectively. Before conducting the RT analyses described later, errors were removed, as were a small number of extreme outlier trials with RTs of greater than 3,000 ms (0.2% of the data).

The mean key press RTs are plotted as a function of session, block, and group in Figure 1. A between-subjects analysis of variance (ANOVA) on the grand mean key press RTs from the training session showed no significant group differences, $F(2, 40) = 0.96$. Figure 2 shows the RT difference scores (the mean of the last four training blocks minus the mean of the first four test block) for each group. A one-way ANOVA on the difference scores did not approach significance, $F(2, 40) = 0.45$, suggesting that there were no differences between the wake and sleep groups.

To ensure that we had enough power to detect the sleep enhancement effect, we performed a retrospective analysis of the statistical power. We first combined the 1-night and 2-night groups into a single group, yielding two groups—a wake group and a sleep group—and framing these two sets of difference scores in the form of a t test for two independent samples. For the 43 subjects, power to detect an effect in which there is no improvement between sessions for the wake group but a 20% speedup between sessions for the sleep group (approximately the effect size observed in the literature) is greater than .98. The power to detect 10% speedup is .79.

To explore whether sleep enhances rate of speedup in the test session, we compared RTs for the last four blocks of the training session to the last four blocks of the test session. There was again no effect across groups in an ANOVA of the difference scores,

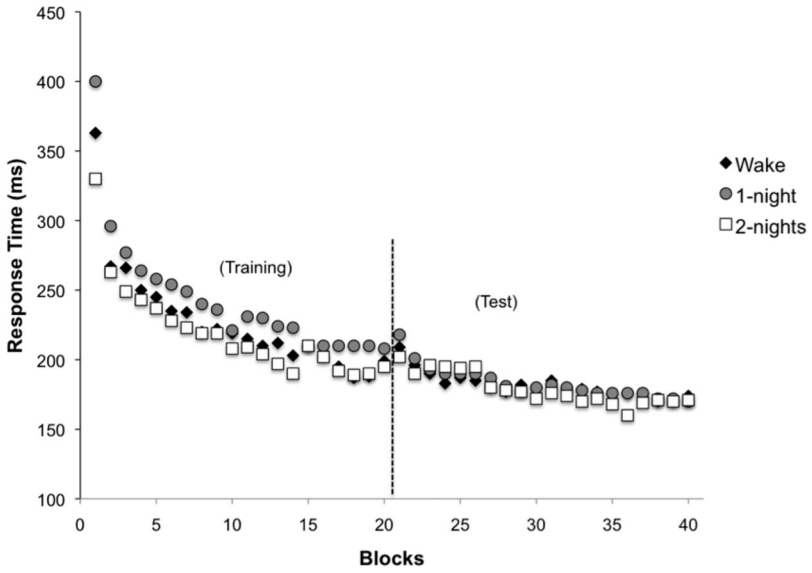

Figure 1. The mean response time per key press as a function of block, session, and group.

$F(2, 40) = 1.54$, $p = .23$, indicating that posttraining sleep does not increase the rate of performance at test. However, the difference scores within each group were significantly greater than zero, $t(42) = 7.12$, $p < .001$, confirming the expected learning due to practice during the testing session.

Discussion

In this study, the first one to control for circadian and homeostatic factors when comparing wake and sleep groups, we found that posttraining sleep does not enhance motor sequence performance. It is possible that the lack of a sleep effect in this study was due to relatively minor procedural differences between our version of the motor sequence task and the task used by Walker et al. (2002). Most significantly, we defined a block in terms of the number of required key press sequences rather than by time duration. There is no theoretical reason, however, to expect this procedural difference to be pertinent. If it is an important factor underlying the sleep effect, then this finding alone should reduce our confidence in the notion that sleep plays a significant role in consolidating motor memory.

In our view, the more plausible account for the lack of sleep enhancement is our novel design, in which we control for circadian and homeostatic factors. This result is consistent with the observations of Rickard et al. (2008), suggesting circadian and perhaps homeostatic differences between morning and evening motor performance. Keisler, Ashe, and Willingham (2007) have also replicated the apparent sleep enhancement in an implicit motor sequence task but demonstrated that time of day, and not sleep, accounted for the enhancement.

The reader may question why the Walker et al. studies (Walker, Brakefield, Hobson, & Stickgold, 2003; Walker, Breakfield, Seidman, et al., 2003) demonstrate a 20–30% improvement after a 24-,

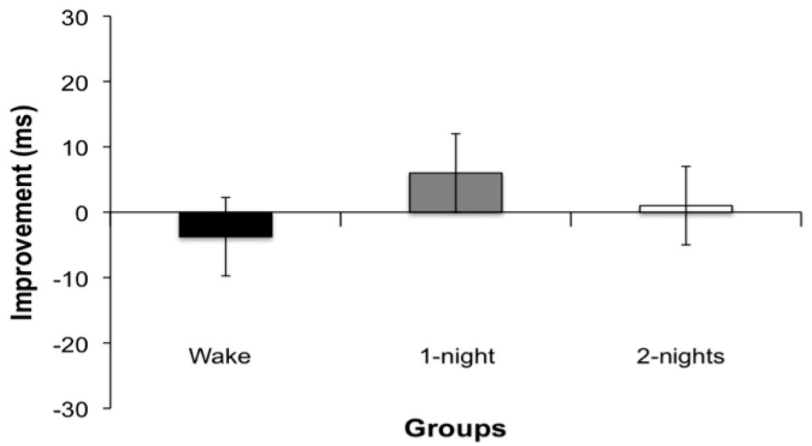

Figure 2. The response time difference scores (the mean of the last four training blocks minus the mean of the first four test block) for each group.

42-, and 72-hr delay, whereas we observe no difference after a 32- or 56-hr delay while using roughly the same amount of data averaging that they did. One possibility is that the Walker et al. studies (Walker, Brakefield, Hobson, & Stickgold, 2003; Walker, Breakfield, Seidman, et al., 2003) train and test subjects at the same time of day, whereas subjects in the present design trained in the morning and tested in the early evening. Although Walker et al. (2002) did not find differences between morning and evening performance, other studies suggest that the morning is better than later afternoon (Payne, 1989; Wright, Hull, & Czeisler, 2002) or evening (Keisler et al., 2007; Rickard et al., 2008) for motor performance. If there is sleep consolidation and a circadian influence on motor sequence performance, we expect that our sleep groups would have a smaller effects size as compared with Walker, Brakefield, Hobson, and Stickgold (2003), as was observed, but that we would still observe a difference between the wake and sleep groups. Our results therefore are consistent with circadian influences on performance in the absence of a sleep consolidation effect.

The literature is also inconsistent with respect to which sleep components contribute to motor memory consolidation. Stage II (Nishida & Walker, 2007; Walker et al., 2002) and rapid eye movement (REM) have been found to correlate with motor sequence performance. Spindle density during Stage 2 and slow-wave sleep (SWS) have also been reported to correlate with motor sequence performance (Nishida & Walker, 2007; Rasch, Pommer, Diekelmann, & Born, 2008). Furthermore, studies using other motor tasks have found SWS (Huber, Ghilardi, Massimini, & Tononi, 2004) or REM sleep (Plihal & Born, 1997) to be associated with performance. To date, it is unclear as to what sleep components are associated with motor memory consolidation.

Our goal in this study was to advance a new method for exploring behavioral sleep consolidation effects that, when considered in combination with other methods in the literature, can yield new insights. Any successful theory of sleep consolidation must ultimately explain this full set of results. At present, four general accounts seem viable. First, it may simply be the case that sleep plays no role in motor sequence memory and that previous findings suggesting otherwise were driven by circadian and homeostatic factors or by data averaging and reactive inhibition effects as discussed in Rickard et al. (2008). By this account, the test advantage for the sleep group in the Fischer et al. (2002, Fischer et al., 2005) deprivation studies would be interpreted as reflecting impairment of a purely time-based consolidation process in the sleep-deprived group.

A second intriguing account of the broad pattern of results over various studies is that sleep only benefits consolidation of motor memory if training occurs within a critical time window before sleep, as suggested by some rodent studies (Smith, 1985). The morning training of our subjects may have been outside of that time window.

Third, sleep might play a permissive role of retroactive facilitation (Ellenbogen, Payne, & Stickgold, 2006), which protects memories from being forgotten by reduction of retroactive interference, similar to the effect of benzodiazepines (Wixted, 2004). It has also been demonstrated that sleep may stabilize motor memory, making it more resistant to interference (Korman et al., 2007). According to this theory, there may have been forgetting across the wake period that followed training in the wake and sleep groups of the present design. The sleep period may then have stabilized the memory from further deterioration, resulting in the lack of difference between the wake and sleep groups.

Our findings should not be taken to mean that sleep does not play any role in the consolidation of motor memories. However, the present results, in combination with recent work (Keisler et al., 2007; Rickard et al., 2008; Song, Howard, & Howard, 2007), do call into question previous evidence of sleep-dependent enhancement of motor performance. To the extent that circadian, homeostatic, data averaging, and reactive inhibition influences have not been adequately controlled, we cannot be sure that a relative sleep advantage reflects an active consolidation process unique to sleep. Future research can benefit from use of multiple approaches to tease apart the interaction of sleep, circadian effects, and memory.

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