



## REGULAR RESEARCH PAPER



# A daytime nap does not increase mnemonic discrimination ability

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**Abstract**

It has been proposed that sleep readies the brain for novel learning, and previous work has shown that sleep loss impairs the ability to encode new memories. In the present study, we examined if a daytime nap would increase mnemonic discrimination (MD) performance. MD is the ability to differentiate between memories that are similar but not identical. Participants performed the Mnemonic Similarity Task (MST) twice, once in the morning and once in the afternoon. The goal of this task is to distinguish stimuli that have been seen before from novel stimuli that are similar but not identical. After the morning MST, participants were randomly allocated into either a sleep or a wake group. The sleep group had a 2-hr nap opportunity, whereas the wake group spent a similar amount of time passively resting. All participants then performed a second MST in the afternoon with a novel set of images. Results did not show any support for increased MD ability after a nap. There was, however, a correlation showing that an increase in sleepiness between sessions predicted a decrease in MD performance. Future work must systematically examine how strong sleep manipulations that are needed for sleep to have an effect on encoding ability, as well as which kind of memory tasks that are sensitive to sleep manipulations. More knowledge about the relationship between sleep and the ability to differentiate similar memories from each other is important because impaired MD ability has previously been reported in various groups in which sleep disturbances are also common.

**KEYWORDS**

encoding, mnemonic discrimination, naps, pattern separation, sleep, sleepiness

## 1 | INTRODUCTION

Sleep deprivation impairs the ability to encode new information (Cousins, Sasmita, & Chee, 2018; Drummond et al., 2000; Harrison & Horne, 2000; Kaida, Niki, & Born, 2015; Yoo, Hu, Gujar, Jolesz, & Walker, 2007). It has further been found that a daytime nap increases learning ability (Mander, Santhanam, Saletin, & Walker, 2011), which

indicates that the ability to encode new information decreases throughout the day, but is “restored” by a nap. A mechanistical explanation for this comes from the Synaptic Downscaling Hypothesis (Tononi & Cirelli, 2006) that stipulates that synapses are built up during learning while we are awake, and then downscaled during sleep, and especially so during slow-wave sleep (SWS). Sleep would then reduce the synaptic load in the brain, and make an organism ready for novel learning.

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In the present study, we examined if a daytime nap would increase memory discrimination (MD) performance. MD refers to the ability to differentiate between memories for events and stimuli that are similar but not identical. This is typically assessed through a task consisting of an initial encoding session where participants view different stimuli, followed by a subsequent memory test where participants are once again presented with the stimuli that were present during encoding, as well as stimuli that are similar, but not identical, to the stimuli presented during encoding. MD is defined as the ability to discriminate the stimuli that are the same as during encoding from the stimuli that are just similar.

Decreased MD performance has previously been associated with ageing, mild cognitive impairment and depression (Déry et al., 2013; Shelton & Kirwan, 2013; Stark, Yassa, Lacy, & Stark, 2013; Yassa et al., 2010). Impaired MD ability, especially in combination with negative affect, has also been suggested to be related to anxiety disorders, perhaps because it results in an inability to discriminate between safe and dangerous stimuli or contexts (Bernstein & McNally, 2018; Kheirbek, Klemenhagen, Sahay, & Hen, 2012; Lange et al., 2017). Considering that all these conditions have also been associated with disturbed sleep (e.g. Baglioni et al., 2016; Moraes et al., 2014; Westerberg et al., 2012), more knowledge of the relationship between MD ability and sleep could, beyond just leading to more information about sleep and memory in general, also potentially be of clinical importance.

Previous experimental work has shown sleep to increase MD performance when the encoding and the memory test is separated by a delay interval containing either sleep or wake (Doxey, Hodges, Bodily, Muncy, & Kirwan, 2018; Hanert, Weber, Pedersen, Born, & Bartsch, 2017). Studies have also found that MD ability is not static, but that it can be increased from interventions such as physical exercise or playing three-dimensional video games (Clemenson & Stark, 2015; Déry et al., 2013; Suwabe et al., 2017; but see also Bernstein & McNally, 2019). In a previous study, Saletin et al. (2016) found impaired MD performance after sleep deprivation, but that it increased again after a recovery daytime nap. The novel contribution of the present study was to test if a daytime nap, as compared to an equal amount of time spent awake, would increase MD performance also in a group that had not been previously sleep-deprived. The research question was if a daytime nap, beyond normalising memory function following sleep deprivation, would be sufficient to facilitate MD performance further. Given the suggested role of sleep in increasing encoding ability, we expected a stronger increase in MD ability in the nap group as compared to in the wake group.

## 2 | METHODS

### 2.1 | Participants

We initially recruited 111 healthy participants between the ages of 18 and 35 years. Inclusion criteria were being free from psychiatric and sleep disorders, and currently not taking any medications known to affect sleep. Participants were further required to have slept for at least 6 hr/night during the 5 nights preceding the study, and for  $\geq 7$  hr the night immediately before the experimental day. The data for this study were

collected as part of two other data collections also including two highly similar fear conditioning paradigms (the results of which have previously been reported in Davidson, Carlsson, Jönsson, and Johansson (2016, 2018)). The only difference between the two data collections was that participants in the first one were required to refrain from all caffeine, alcohol and nicotine during 24 hr before the experimental day, whereas participants in the second data collection only had to refrain from these during the experimental day. Adding dataset as a between-subjects variable in the analyses showed that there were no main or interaction effects of this variable, and thus all analyses were collapsed over datasets.

One participant chose to withdraw during the experimental day, and one testing day had to be aborted because of a power failure in the lab. Four participants had to be excluded because of technical malfunctions during the testing, one because he afterwards reported having used the wrong keys on the keyboard, and additionally one because the experimenter accidentally gave incorrect instructions. Thus, 103 participants remained for the final analysis. There were 55 participants in the wake group (31 women, 24 men, mean [SD] age = 24.5 [4.23] years), and 48 participants in the sleep group (22 women, 26 men, mean [SD] age = 23.9 [4.05] years). The study was approved by the local ethics review board (Lund 2012/73; 2013/96).

### 2.2 | The Mnemonic Similarity Task

Mnemonic discrimination ability was assessed with the Mnemonic Similarity Task (MST; previously known as the Behavioural Pattern Separation Task – Object Version [BPS-O]; Stark et al., 2013). We used the MST version 0.6 for MATLAB (Mathworks). This task starts with an encoding phase in which 128 images of everyday objects are presented for 2 s each, and participants are asked to use the keyboard to indicate if the object is most suitable indoors or outdoors. Typically when administering this task, the encoding is incidental so that participants are not informed about the upcoming memory test. However, given that we were going to test each participant twice, we informed them about the upcoming memory test while instructing them before both encoding sessions. The second part of the task is a memory test, which in our procedure took place 3 min after encoding. During this test, participants view three different kinds of stimuli (64 of each for a total of 192 trials); the same as those that were present during encoding (Targets), completely new items (Foils), and items that are similar, but not exactly the same, as those that were present during encoding (Lures). The task of the participant is to use the keyboard to indicate if an image is 'Old', 'New', or 'Similar'. The items are shown for 2 s each, and responses must be given while the object is still being presented on the screen. The inter-stimulus interval for both encoding and the memory test is 0.5 s.

### 2.3 | Procedure

Prior to the experimental day, all participants completed an online questionnaire that contained the trait part of the State-Trait Anxiety

Inventory-Trait (STAI-T; Spielberger, 1983), the Fear Questionnaire (FQ; Marks & Mathews, 1979), the Penn State Worry Questionnaire (PSWQ; Meyer, Miller, Metzger, & Borkovec, 1990) and the Hospital Anxiety and Depression Scale (HADS; Zigmond & Snaith, 1983).

An overview of the procedure is presented in Figure 1. On the experimental day, participants arrived in the lab at 10:30 hours. After giving informed consent, they first completed the Karolinska Sleepiness Scale (KSS; Åkerstedt & Gillberg, 1990), and then the first MST (MST1). After that, they completed the unrelated fear conditioning paradigm, and were then randomly allocated to either the sleep or the wake group. Randomisation was done by the experimenter rolling a dice. The last four participants were not randomly allocated, but were pre-determined to be part of the wake group (in order to balance the groups for the fear conditioning paradigm, which had different exclusion criteria). These participants were, however, themselves blind to which group they would belong to. Participants allocated to the sleep group then had the polysomnography (PSG) electrodes attached, while participants in the wake group had a 45-min break. All participants were then served a light lunch. The sleep group then had a 2-hr sleep opportunity in the sleep lab, while participants in the wake group spent 2 hr passively resting. We wanted to reduce the amount of outside interference for participants in the wake group as much as possible. For this reason, they were not allowed to read or use their phones or laptops, and were instead asked to just sit still and passively rest. The experimenter checked on them every 15 min to ensure they were feeling alright and had not fallen asleep. After the delay interval, participants in both groups had a 15-min break and then completed the KSS for a second time before they completed the second part of the unrelated fear conditioning paradigm. They then performed the second MST (MST2) at approximately 15:20 ( $\pm 10$  min). We used two independent sets of images for the morning and the afternoon version of the task (Set 'C' in the morning, and Set 'D' in the afternoon).

## 2.4 | Data analysis

The MST yields two different scores: General Recognition (GR) and Mnemonic Discrimination (MD). GR performance is calculated through subtracting the probability of (incorrectly) responding 'Old' to Foils from the probability of (correctly) answering 'Old' to Targets.

MD performance is calculated by subtracting the probability of (incorrectly) responding 'Similar' to Foils from the probability of (correctly) responding 'Similar' to Lures. Thus, MD requires a more

detailed and specific memory than GR. We further wanted to see if the effect of sleep would differ depending on the degree of similarity between the Lures and the images presented during encoding. The Lures have previously been categorised into five different levels based on how difficult they are to separate from the images presented during encoding (Stark et al., 2013). To examine if the effect of sleep would vary depending on Lure similarity, we calculated the MD score separately for each of the five similarity levels. Given that the Foils do not systematically vary in similarity, the same probability of answering 'Similar' to Foils value was subtracted from each Lure Similarity score.

For the correlations, we used the mean MD score across the five levels of Lure Similarity. For correlations involving the change score between the morning and the afternoon sessions, we subtracted the scores on the MST1 from the scores on the MST2.

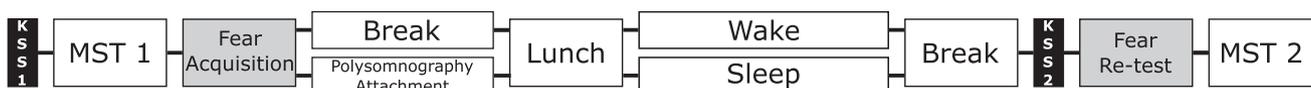
## 2.5 | Polysomnography

Polysomnography was recorded using an Embla Titanium device (Embla Systems) with a montage using F3, F4, C3, C4, O1 and O2 (all referenced to the contralateral mastoid). Electro-oculography was recorded with one electrode above the right ocular canthus, and one below the left ocular canthus. For electromyography, we used two submental electrodes. The sampling rate was 256 Hz. Sleep was scored according to the American Academy of Sleep Medicine manual (Iber, Ancoli-Israel, Chesson, & Quan, 2007). In the first data collection, sleep was scored by the first author, a trained sleep scorer. In the second data collection, sleep was scored by the first author together with a professional sleep technician. The first author was blind to the memory data while scoring the sleep recordings, and the professional sleep technician was blind to the study hypothesis altogether. Epochs with an arousal lasting for a majority of the epoch were scored as wake.

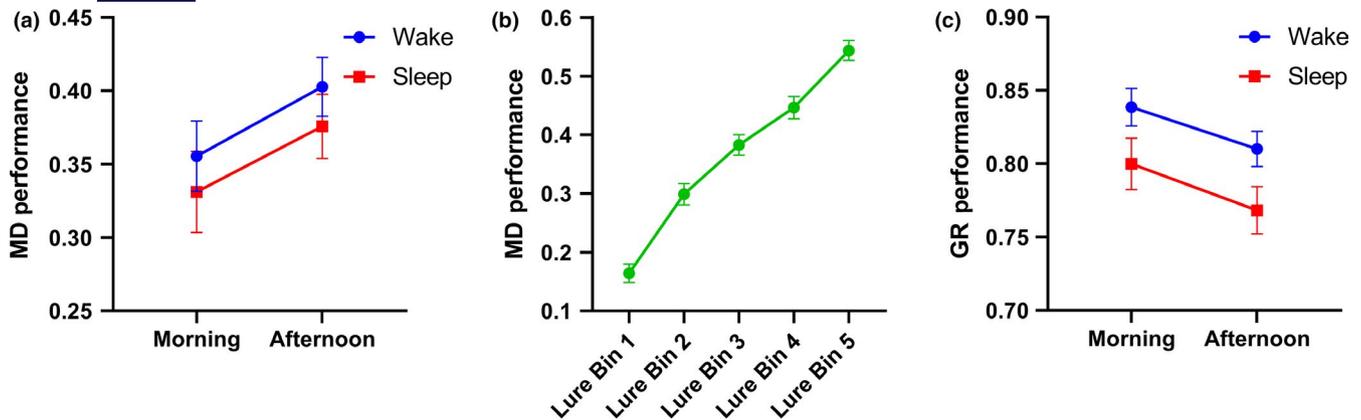
## 3 | RESULTS

### 3.1 | Sleepiness

One participant in the wake group did not complete the KSS1, so all analyses with this variable were based on  $n = 102$ . The mean (*SD*) KSS score for the sleep group was 3.96 (1.49) in the morning, and 5.29 (1.76) in the afternoon. The mean (*SD*) KSS score for the



**FIGURE 1** Overview of the experimental procedure. Participants arrived in the lab at 10:30 hours and first completed the KSS, and then performed the first MST. They then took part in an unrelated fear conditioning task after which they were randomly allocated to either the sleep or the wake group. The wake group then had a break while the electrodes for PSG were attached to participants in the sleep group. Both groups were then served a light lunch, after which participants in the wake group spent 2 hr passively resting, while participants in the sleep group had a 2-hr sleep opportunity. All participants then had a 15-min break, after which they once again completed the KSS, performed another unrelated fear conditioning task, and then finally completed the MST for a second time at approximately 15:20 hours ( $\pm 10$  min). KSS, Karolinska Sleepiness Scale; MST, Mnemonic Similarity Task



**FIGURE 2** (a) Mnemonic discrimination (MD) performance (collapsed over Lure bins) for the sleep and the wake group in the morning and in the afternoon, mean ( $\pm$ SEM). (b) MD performance (collapsed over group and session) as a function of Lure Similarity, mean ( $\pm$ SEM). MD performance increased linearly as the Lures became less similar (Lure Bin 1 being the most similar and Lure Bin 5 being the least similar). (c) General recognition (GR) performance for the sleep and the wake groups in the morning and in the afternoon, mean ( $\pm$ SEM)

wake group was 4.13 (1.40) in the morning, and 5.26 (1.66) in the afternoon. Results from the KSS were analysed with a  $2 \times 2$  mixed analysis of variance (ANOVA) with Time (KSS1/KSS2) and Group (Sleep/Wake). Participants were significantly sleepier after the delay interval as compared to in the morning, as evident by a main effect of Time,  $F(1,100) = 34.51, p < .001$ . There were no differences between the groups, either in sleepiness throughout the day, or in the increase of sleepiness between the first and the second KSS, as evident by the lack of a main effect of Group, and the lack of an interaction between Group and Time, both  $p \geq .63$ .

### 3.2 | Mnemonic discrimination

The effect of sleep on MD ability was analysed with a  $2 \times 2 \times 5$  mixed ANOVA with the factors Group, Time and Lure Bin (the five different levels of Lure Similarity).

We did not find support for our hypothesis that the increase in MD ability would be higher in the sleep group than in the wake group, as evident by a lack of an interaction effect of Group and Time,  $F(1, 101) = 0.01, p = .92$ . These data are displayed in Figure 2a. Bayesian statistics (performed with the JASP version 0.9.2. software [JASP Team, 2019], using the default settings) comparing the change in MD score for the groups between the morning and the afternoon revealed a BF01 value of 4.78 (95 % confidence interval for the Cohen's  $d$  effect size [-0.37, 0.41]), indicating moderate support for the null hypothesis.

Performance decreased as a function of Lure Similarity, as evident by a main effect of Lure Bin,  $F(4, 404) = 210.42, p < .001$ , post hoc contrasts revealed this effect to be linear,  $F_{\text{linear}}(1, 101) = 617.96, p < .001$ . MD performance as a function of Lure Similarity, collapsed over Group and Time, is displayed in Figure 2b.

A main effect of Time indicated that MD performance was higher during the MST2 as compared to during the MST1,  $F(1,101) = 10.94, p = .001$ . The effect of sleep did not differ depending on Lure Similarity, as evident by the lack of a three-way interaction between

Group, Time and Lure Bin,  $F(4, 404) = 0.26, p = .91$ . There was no main effect of Group,  $F(1,101) = 0.72, p = .40$ .

There was no correlation between sleepiness scores when the participants came to the lab and MD performance on the MST 1,  $r = -0.06, p = .53$ . Neither was there a correlation between scores on the KSS2 and MD performance on the MST 2,  $r = 0.04, p = .70$ . This second test should be interpreted with caution given that the MST 2 took place about 30 min after participants completed the KSS2. There was, however, a correlation between the difference between the KSS1 and KSS2 and the change in MD performance between sessions, so that a larger increase in sleepiness predicted a smaller increase in MD ability,  $r = -0.28, p = .005$ . This is consistent with previous findings showing that sleep deprivation impairs encoding ability.

To examine if there were any correlations between the scores on the scales for depression, anxiety, worry and fear and MD ability, we correlated the scores on these scales with MD performance on the MST 1 (because this was measured before the sleep/wake manipulation, and thus represents baseline MD ability). Descriptive data for these scales are presented in Table 1. Considering that these correlations were exploratory, we used a Bonferroni correction so that the  $\alpha$  level for the five tests was set to 0.01. One participant did not complete the online questionnaire, so this analysis was based

**TABLE 1** Descriptive data for the mood scales for all participants combined

Scale	Score, mean (SD)
FQ	21.21 (13.90)
HADS Anxiety	5.80 (3.01)
HADS Depression	3.11 (2.11)
PSWQ	40.95 (12.64)
STAI-T	37.33 (8.32)

Abbreviations: FQ, Fear Questionnaire; HADS, Hospital Anxiety and Depression Scale; PSWQ, Penn State Worry Questionnaire; STAI-T, State-Trait Anxiety Inventory-Trait.

on  $n = 102$ . Additionally, data for the STAI-T were missing for one participant, so all correlations using this scale were based on  $n = 101$ . There was a tendency towards a negative correlation with the FQ,  $r_s = -0.18$ ,  $p = .072$ , so that higher FQ scores predicted lower MD ability. This correlation was, however, no longer significant even at a trend level after applying Bonferroni corrections. MD ability was not correlated with scores on either the PSWQ, STAI-T, HADS Anxiety or HADS Depression, all  $p \geq .25$ . There were no between-groups differences for any of these scales, all  $p \geq .23$ .

### 3.3 | General Recognition

General Recognition performance was analysed with a  $2 \times 2$  mixed ANOVA with Group and Time. This revealed a main effect of Group,  $F(1,101) = 4.72$ ,  $p = .032$ , showing that the wake group overall performed better than the sleep group. There was a main effect of Time,  $F(1,101) = 11.46$ ,  $p = .001$ , indicating lower performance on the second test. There was, however, no interaction between Group and Time,  $F(1,101) = 0.03$ ,  $p = .86$ , indicating that the decrease in performance between sessions was equivalent in both groups. These data are displayed in Figure 2c. Bayesian statistics comparing the change in GR scores between the morning and the afternoon between the groups revealed a BF01 value of 4.73 (95 % confidence interval for the Cohen's  $d$  effect size  $[-0.35, 0.42]$ ), indicating moderate support for the null hypothesis.

There was no correlation between sleepiness when the participants came to the lab and GR performance on the MST1,  $r < 0.001$ ,  $p = .998$ . Neither was there a correlation between scores on the KSS2 and GR performance on the MST2,  $r = 0.03$ ,  $p = .76$ . As mentioned above, this second test should be interpreted with caution given that the MST 2 took place about 30 min after participants completed the KSS2. There was no correlation between the increase in KSS scores during the day and the change in GR performance between sessions,  $r = -0.10$ ,  $p = .32$ .

### 3.4 | Polysomnography results

Due to technical issues, PSG data were missing for three participants in the sleep group, so all correlations with sleep variables were based

**TABLE 2** Sleep statistics

	Time, min, mean(SD)	Percentage of TST, mean(SD)
TST	86.54 (28.91)	
S1	11.16 (8.34)	15.99 (17.32)
S2	28.36 (15.41)	34.24 (17.86)
SWS	36.76 (23.74)	39.33 (21.96)
REM	10.29 (12.85)	10.45 (13.42)

Abbreviations: TST, total sleep time; S1, sleep Stage 1; S2, sleep Stage 2; SWS, slow-wave sleep; REM, rapid eye movement sleep.

on  $n = 45$ . Descriptive data for the PSG are presented in Table 2. The proposed role of SWS in enabling novel learning through promoting synaptic downscaling was tested by correlating SWS duration with the change in MD performance between the morning and the afternoon. Such an association was not supported, as evident by a lack of such a correlation,  $r_s = -0.10$ ,  $p = .53$ . Exploratorily, we also tested correlations with other sleep variables but found no significant correlations between MD change and time spent in either Stage 1, Stage 2, rapid eye movement (REM) sleep, or with total sleep time (TST), all  $p \geq .38$ .

There was no correlation between time spent in SWS and the change in GR performance,  $r_s = -0.16$ ,  $p = .28$ . We also performed exploratory correlations with the other sleep stages, as well as TST. Surprisingly, we found negative correlations between GR change and TST ( $r_s = -0.32$ ,  $p = .034$ ), time spent in Stage 2 ( $r_s = -0.31$ ,  $p = .040$ ), and a tendency towards a negative correlation with time spent in REM sleep ( $r_s = -0.29$ ,  $p = .055$ ). All these sleep variables thus predicted a larger decrease in GR performance. Given that these tests were exploratory, we did apply Bonferroni corrections and set the  $\alpha$  level to 0.0125. None of these correlations were still significant after this, and should thus be interpreted with caution.

## 4 | DISCUSSION

Recent research reported that full sleep deprivation impairs MD ability, and that a daytime nap is sufficient to restore it (Saeitin et al., 2016). In the present study, we set out to investigate if such beneficial consequences of a daytime nap could be extended further, assessing sleep-induced changes in MD performance in a previously well-rested group. Our present data did not support such a facilitatory effect in a relatively large sample, with Bayesian statistics revealing moderate support for the null hypothesis.

One reason for why sleep is expected to increase encoding ability is the Synaptic Downscaling Hypothesis (Tononi & Cirelli, 2006), which suggests that the synaptic downscaling that takes place during sleep "frees up" synapses for novel learning. It has further been found that adenosine, a neurotransmitter that is aggregated in the brain during wake, and cleared out during sleep (Porkka-Heiskanen, Strecker, Thakkar, & McCarley, 2000), affects regions of the brain that have been associated with MD in mice (Florian, Vecsey, Halassa, Haydon, & Abel, 2011). Just a daytime nap, as compared to a similar amount of time spent awake, might however not be sufficient to result in any major group differences in either synaptic density or in the aggregation of adenosine. The relatively mild sleep manipulation in the present study, with two previously well-rested groups where the only difference was an average of about 87 min of sleep, was for example not sufficient to result in a group difference in sleepiness. We did, however, observe that the increase in sleepiness between the morning and the afternoon was correlated with a decrease in MD performance. This suggests that a sleep manipulation large enough to cause a group difference in sleepiness might also impair MD performance. In another previous study, Mander et al. (2011) found learning ability to

decrease during the day. They did, however, have their memory test a few hours later in the evening, which would allow for a larger decrease due to time spent awake. In the future, studies should be conducted where the amount of sleep is systematically manipulated to see how much of a sleep manipulation that is required for a potential impairment of encoding ability to occur. It should, however be mentioned that a previous study found no correlation between MD ability and general sleep quality (Shelton & Kirwan, 2013). For future studies, it would also be interesting to look at if sleep has larger effects in groups that have impaired MD ability to begin with.

Sleep did not affect general recognition ability. This is consistent with some previous studies that have found recognition memory to be intact in subjects having had less sleep, even when performance on other memory tasks requiring more specificity, such as memory for temporal order, free recall of word lists, or face-name associations, has been impaired (Drummond et al., 2000; Harrison & Horne, 2000; Mander et al., 2011). There are, however, studies that have found effects of sleep deprivation also on recognition performance (Cousins et al., 2018; Yoo et al., 2007). Studies manipulating slow-wave activity during sleep have further showed that less slow-wave activity results in impaired subsequent memory in a recognition task (van der Werf et al., 2009), as well as for both recognition and cued and free recall (Antonenko, Diekelmann, Olsen, Born, & Mölle, 2013), and Kaida et al. (2015) found sleep deprivation to impair both recognition and source memory performance. Saletin et al. (2016) found that sleep had a larger effect on MD than on GR, but that sleep deprivation still decreased GR performance, and that this as well increased again after a recovery nap. An interesting feature with the MST is how the different levels of Lure Similarity allowed us to measure if sleep affected MD performance differently depending on similarity level. At the level of the Lures the most different from the stimuli presented during the initial encoding session, this could almost be viewed as an old/new recognition task as well, but with the increasing level of similarity requiring an increasing level of memory specificity. Our present results did, however, not provide any support for sleep having an increasing benefit depending on the extent of memory specificity required by the task.

Another difference between our present design and the Mander et al. (2011) study is that we did not allow the participants in the wake group to go on with their regular daytime activities. Instead, we wanted them to be exposed to as little novel information as the participants in the sleep group. For this reason, they spent the delay interval passively resting, and were not allowed to read or use their phones or laptops. The possibility that relaxed wakefulness of this kind also has a restorative effect on encoding ability should be explored in future studies by comparing both an active and a passive wake condition with a sleep group.

After applying Bonferroni corrections, there were no correlations between scores on the scales for depression, anxiety, worry and fear and MD ability. It should, however, be mentioned that one of the inclusion criteria for participating in the present study was not having any psychiatric disorders, and thus our participants probably scored

lower on these scales compared to what the general population would. It is possible that a larger variance of scores on these scales would have resulted in clearer associations between worry, depression, anxiety, fear and MD ability, even if no such correlations were found in the present relatively psychopathology-free sample.

A limitation of the present study is that we did not collect any data on participants' chronotype. The timing of when during the day one is at one's "peak" in cognitive abilities can vary largely between individuals. In future studies, it will therefore be important to collect data on participants' chronotype to ensure that it does not differ between groups.

Another potential limitation of the present study is that it also included the unrelated fear conditioning paradigm. We do, however, not see any reasons for why the fear conditioning should have affected encoding performance differently in the sleep and the wake group, and this should thus not have affected any potential group differences. Future studies on sleep and MD should also try to separate the encoding and the memory test (as was done in Antonenko et al., 2013; Yoo et al., 2007), and have sleep/wake manipulations before both of these sessions separately. This would make it possible to determine if sleep loss makes one encode memories with less specificity, or if sleep instead mainly affects the ability to retrieve sufficiently specific information during the re-test.

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## CONFLICTS OF INTEREST

None of the authors have any conflicts of interest to disclose.

## AUTHOR CONTRIBUTION

PD, PJ and MJ designed the study. PD collected and analysed the data. PD and MJ wrote the manuscript.

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## REFERENCES

- Åkerstedt, T., & Gillberg, M. (1990). Subjective and objective sleepiness in the active individual. *International Journal of Neuroscience*, *52*, 29–37. <https://doi.org/10.3109/00207459008994241>
- Antonenko, D., Diekelmann, S., Olsen, C., Born, J., & Mölle, M. (2013). Napping to renew learning capacity: Enhanced encoding after stimulation of sleep slow oscillations. *European Journal of Neuroscience*, *37*, 1142–1151. <https://doi.org/10.1111/ejn.12118>
- Baglioni, C., Nanovska, S., Regen, W., Spiegelhalter, K., Feige, B., Nissen, C., ... Riemann, D. (2016). Sleep and mental disorders: A meta-analysis of polysomnographic research. *Psychological Bulletin*, *142*, 969–990. <https://doi.org/10.1037/bul0000053>
- Bernstein, E. E., & McNally, R. J. (2018). Exploring behavioral pattern separation and risk for emotional disorders. *Journal of*

- Anxiety Disorders*, 59, 27–33. <https://doi.org/10.1016/j.janxdis.2018.08.006>
- Bernstein, E. E., & McNally, R. J. (2019). Examining the effects of exercise on pattern separation and the moderating effects of mood symptoms. *Behavior Therapy*, 50, 582–593. <https://doi.org/10.1016/j.beth.2018.09.007>
- Clemenson, G. D., & Stark, C. E. L. (2015). Virtual environmental enrichment through video games improves hippocampal-associated memory. *The Journal of Neuroscience*, 35, 16116–16125. <https://doi.org/10.1523/jneurosci.2580-15.2015>
- Cousins, J. N., Sasmita, K., & Chee, M. W. L. (2018). Memory encoding is impaired after multiple nights of partial sleep restriction. *Journal of Sleep Research*, 27, 138–145. <https://doi.org/10.1111/jsr.12578>
- Davidson, P., Carlsson, I., Jönsson, P., & Johansson, M. (2016). Sleep and the generalization of fear learning. *Journal of Sleep Research*, 25, 88–95. <https://doi.org/10.1111/jsr.12339>
- Davidson, P., Carlsson, I., Jönsson, P., & Johansson, M. (2018). A more generalized fear response after a daytime nap. *Neurobiology of Learning and Memory*, 151, 18–27. <https://doi.org/10.1016/j.nlm.2018.03.005>
- Déry, N., Pilgrim, M., Gibala, M., Gillen, J., Wojtowicz, J. M., MacQueen, G., & Becker, S. (2013). Adult hippocampal neurogenesis reduces memory interference in humans: Opposing effects of aerobic exercise and depression. *Frontiers in Neuroscience*, 7, <https://doi.org/10.3389/fnins.2013.00066>
- Doxey, C. R., Hodges, C. B., Bodily, T. A., Muncy, N. M., & Kirwan, C. B. (2018). The effects of sleep on the neural correlates of pattern separation. *Hippocampus*, 28, 108–120. <https://doi.org/10.1002/hipo.22814>
- Drummond, S. P. A., Brown, G. G., Gillin, J. C., Stricker, J. L., Wong, E. C., & Buxton, R. B. (2000). Altered brain response to verbal learning following sleep deprivation. *Nature*, 403, 655–657. <https://doi.org/10.1038/35001068>
- Florian, C., Vecsey, C. G., Halassa, M. M., Haydon, P. G., & Abel, T. (2011). Astrocyte-derived adenosine and A<sub>1</sub> receptor activity contribute to sleep loss-induced deficits in hippocampal synaptic plasticity and memory in mice. *The Journal of Neuroscience*, 31, 6956–6962. <https://doi.org/10.1523/jneurosci.5761-10.2011>
- Hanert, A., Weber, F. D., Pedersen, A., Born, J., & Bartsch, T. (2017). Sleep in humans stabilizes pattern separation performance. *The Journal of Neuroscience*, 37, 12238–12246. <https://doi.org/10.1523/jneurosci.1189-17.2017>
- Harrison, Y., & Horne, J. A. (2000). Sleep loss and temporal memory. *The Quarterly Journal of Experimental Psychology Section A*, 53, 271–279. <https://doi.org/10.1080/173755870>
- Iber, C., Ancoli-Israel, S., Chesson, A., & Quan, S. (2007). *The AASM Manual for the Scoring of Sleep and Associated Events: Rules, Terminology and Technical Specifications*, 1st ed. Winchester, IL: American Academy of Sleep Medicine.
- Kaida, K., Niki, K., & Born, J. (2015). Role of sleep for encoding of emotional memory. *Neurobiology of Learning and Memory*, 121, 72–79. <https://doi.org/10.1016/j.nlm.2015.04.002>
- Kheirbek, M. A., Klemenhagen, K. C., Sahay, A., & Hen, R. (2012). Neurogenesis and generalization: A new approach to stratify and treat anxiety disorders. *Nature Neuroscience*, 15, 1613–1620. <https://doi.org/10.1038/nn.3262>
- Lange, I., Goossens, L., Michielse, S., Bakker, J., Lissek, S., Papalini, S., ... Schruers, K. (2017). Behavioral pattern separation and its link to the neural mechanisms of fear generalization. *Social Cognitive and Affective Neuroscience*, 12, 1720–1729. <https://doi.org/10.1093/scan/nsx104>
- Mander, B. A., Santhanam, S., Saletin, J. M., & Walker, M. P. (2011). Wake deterioration and sleep restoration of human learning. *Current Biology*, 21, R183–R184. <https://doi.org/10.1016/j.cub.2011.01.019>
- Marks, I. M., & Mathews, A. M. (1979). Brief standard self-rating for phobic patients. *Behaviour Research and Therapy*, 17, 263–267. [https://doi.org/10.1016/0005-7967\(79\)90041-X](https://doi.org/10.1016/0005-7967(79)90041-X)
- Meyer, T. J., Miller, M. L., Metzger, R. L., & Borkovec, T. D. (1990). Development and validation of the penn state worry questionnaire. *Behaviour Research and Therapy*, 28, 487–495. [https://doi.org/10.1016/0005-7967\(90\)90135-6](https://doi.org/10.1016/0005-7967(90)90135-6)
- Moraes, W., Piovezan, R., Poyares, D., Bittencourt, L. R., Santos-Silva, R., & Tufik, S. (2014). Effects of aging on sleep structure throughout adulthood: A population-based study. *Sleep Medicine*, 15, 401–409. <https://doi.org/10.1016/j.sleep.2013.11.791>
- Porkka-Heiskanen, T., Strecker, R. E., & McCarley, R. W. (2000). Brain site-specificity of extracellular adenosine concentration changes during sleep deprivation and spontaneous sleep: An in vivo microdialysis study. *Neuroscience*, 99, 507–517. [https://doi.org/10.1016/S0306-4522\(00\)00220-7](https://doi.org/10.1016/S0306-4522(00)00220-7)
- Saletin, J. M., Goldstein-Piekarski, A. N., Greer, S. M., Stark, S., Stark, C. E., & Walker, M. P. (2016). Human hippocampal structure: A novel biomarker predicting mnemonic vulnerability to, and recovery from, sleep deprivation. *The Journal of Neuroscience*, 36, 2355–2363. <https://doi.org/10.1523/jneurosci.3466-15.2016>
- Shelton, D. J., & Kirwan, C. B. (2013). A possible negative influence of depression on the ability to overcome memory interference. *Behavioural Brain Research*, 256, 20–26. <https://doi.org/10.1016/j.bbr.2013.08.016>
- Spielberger, C. D. (1983). *Manual for the State-Trait Anxiety Inventory*. Palo Alto, CA: Consulting Psychologists Press.
- Stark, S. M., Yassa, M. A., Lacy, J. W., & Stark, C. E. (2013). A task to assess behavioral pattern separation (BPS) in humans: Data from healthy aging and mild cognitive impairment. *Neuropsychologia*, 51, 2442–2449. <https://doi.org/10.1016/j.neuropsychologia.2012.12.014>
- Suwabe, K., Hyodo, K., Byun, K., Ochi, G., Yassa, M. A., & Soya, H. (2017). Acute moderate exercise improves mnemonic discrimination in young adults. *Hippocampus*, 27, 229–234. <https://doi.org/10.1002/hipo.22695>
- Tononi, G., & Cirelli, C. (2006). Sleep function and synaptic homeostasis. *Sleep Medicine Reviews*, 10, 49–62. <https://doi.org/10.1016/j.smrv.2005.05.002>
- Van Der Werf, Y. D., Altena, E., Schoonheim, M. M., Sanz-Arigita, E. J., Vis, J. C., De Rijke, W., & Van Someren, E. J. W. (2009). Sleep benefits subsequent hippocampal functioning. *Nature Neuroscience*, 12, 122. <https://doi.org/10.1038/nn.2253>
- Westerberg, C. E., Mander, B. A., Florczak, S. M., Weintraub, S., Mesulam, M. M., Zee, P. C., & Paller, K. A. (2012). Concurrent impairments in sleep and memory in amnesic mild cognitive impairment. *Journal of the International Neuropsychological Society*, 18, 490–500. <https://doi.org/10.1017/S135561771200001X>
- Yassa, M. A., Stark, S. M., Bakker, A., Albert, M. S., Gallagher, M., & Stark, C. E. L. (2010). High-resolution structural and functional MRI of hippocampal CA3 and dentate gyrus in patients with amnesic Mild Cognitive Impairment. *NeuroImage*, 51, 1242–1252. <https://doi.org/10.1016/j.neuroimage.2010.03.040>
- Yoo, S.-S., Hu, P. T., Gujar, N., Jolesz, F. A., & Walker, M. P. (2007). A deficit in the ability to form new human memories without sleep. *Nature Neuroscience*, 10, 385. <https://doi.org/10.1038/nn1851>
- Zigmond, A. S., & Snaith, R. (1983). The hospital anxiety and depression scale. *Acta Psychiatrica Scandinavica*, 67, 361–370. <https://doi.org/10.1111/j.1600-0447.1983.tb09716.x>

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