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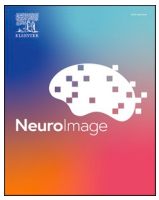

A nap can recalibrate homeostatic and associative synaptic plasticity in the human cortex

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ABSTRACT

Background: Nighttime sleep renormalizes net synaptic strength (homeostatic plasticity) and the inducibility of long-term potentiation (LTP)-like plasticity (associative plasticity) in the cortex. However, whether an afternoon nap is sufficient for this process remains to be characterized.
Methods: Twenty healthy adults participated in a repeated measures sleep laboratory study with an adaptation and two experimental sessions – nap and wake (1:15–2:15 PM). After the nap or wake session, non-invasive indices of net synaptic strength (indexed by transcranial magnetic stimulation, TMS-probed corticospinal excitability and wake EEG theta activity) and inducibility of LTP-like plasticity (indexed by TMS-induced motor evoked potentials, MEPs, following paired associative stimulation, PAS) were assessed.
Results: We observed indices of reduced net synaptic strength after sleep compared to wakefulness, evidenced by a higher TMS intensity needed to induce MEPs and reduced wake EEG theta activity. Additionally, we observed an increase in the inducibility of associative synaptic plasticity after sleep, as evidenced by a greater increase in TMS-induced MEPs in response to PAS.
Conclusions: The study reinforces the restorative effect of sleep for homeostatic and associative synaptic plasticity in the human cortex and demonstrates that even a short nap can promote this process.

1. Introduction

Sleep modulates the strengthening or weakening of connections (synapses) between neurons. This modulation of transmission strength (synaptic plasticity) represents a neural basis for adaptive behavior in a changing environment. Yet the effects of short periods of daytime sleep (naps) on synaptic plasticity remain to be fully characterized.

Sleep modulates the overall weight of synapses (homeostatic synaptic plasticity), which defines the set-point for the inducibility of novel strengthening of synapses through concurrent pre- and postsynaptic activity in response to salient information (associative synaptic plasticity). More specifically, prior work in animals has demonstrated that the encoding of new information during wakefulness leads to synaptic strengthening as evidenced by major markers of synaptic strength including the level of GluR1-containing AMPA receptors in the rat hippocampus and cortex (Vyazovskiy et al., 2008), cortical spine density in adolescent mice (Maret et al., 2011), the number or size of synapses in different neuronal circuits in flies (Bushey et al., 2011; Donlea et al., 2009) and the number of synapses in zebra fish larvae (Suppermpool et al., 2024). Ultimately, increased net synaptic strength, accompanied by enhanced need of space and energy, leads to saturation. Saturation refers to the observation that the state of a neural network with high overall strength of synaptic connections after prolonged periods of wakefulness is less likely to allow for further synaptic strengthening of input-specific activity. For instance in rodents, stimulation of the motor cortex failed to induce consistent increases in local field potentials following prolonged wakefulness, while long-term potentiation (LTP) could be induced again following a period of sleep (Vyazovskiy et al., 2008). Indeed, sleep has been shown to lead to overall downscaling of synaptic strength (Bushey et al., 2011; Donlea et al., 2009; Maret et al.,

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2011; Suppermpool et al., 2024; Vyazovskiy et al., 2008). This desaturation is thought to renew the capacity of neural networks to form and strengthen novel synaptic connections in response to new information (synaptic homeostasis hypothesis; Tononi and Cirelli, 2014, 2006).

In humans, in the absence of direct measures, indirect evidence through non-invasive indices of homeostatic and associative synaptic plasticity are in line with the observations in animal models. We and others have used the intensity of transcranial magnetic stimulation (TMS) to elicit motor evoked potentials (MEPs) as a marker of corticospinal excitability, and found that a lower intensity of TMS was required to produce similar MEP amplitudes after sleep deprivation compared to sleep, suggesting higher synaptic strength (Kuhn et al., 2016; Salehinejad et al., 2022). We also used wake electroencephalography (EEG) theta activity as a non-invasive index of net synaptic strength. Theta activity during wake increases with time awake, correlates with slow wave activity (SWA) at sleep onset, and is reduced after sleep (Cajochen et al., 1995; Finelli et al., 2000; Vyazovskiy and Tobler, 2005). Similar to observations in animal studies (Vyazovskiy and Tobler, 2005), we found that wake EEG theta activity was enhanced after a night of sleep deprivation compared to undisturbed nighttime sleep (Kuhn et al., 2016). Furthermore, we induced associative long-term potentiation-like synaptic plasticity in the motor cortex through paired associative stimulation (PAS) (Stefan et al., 2000) and measured the subsequent TMS-induced MEP amplitudes as an estimation of LTP-inducibility (Kuhn et al., 2016). The PAS protocol involves the paired application of weak electrical stimulation of a peripheral nerve followed shortly by TMS over the corresponding motor cortical area, building on the concept of associativity and coincidence of LTP (Suppa et al., 2017). We showed that the inducibility of LTP-like plasticity through the PAS protocol was occluded after a night of sleep deprivation compared to intact inducibility after undisturbed nighttime sleep (Kuhn et al., 2016). However, whether a short nap is sufficient for renormalization of synaptic plasticity remains to be characterized.

The current study was designed to test whether a brief daytime nap is sufficient to observe a decrease in overall synaptic strength and an increase in the inducibility of novel associative synaptic plasticity, as observed for nighttime sleep. We firstly predicted that we would observe lower indices of net synaptic strength after sleep compared to wake (Kuhn et al., 2016; Maier et al., 2019; Salehinejad et al., 2022). That is, we expected to observe a higher stimulation intensity of TMS to evoke similar MEP amplitudes following the nap compared to the wake session, and a pre- to post-wake increase in wake EEG theta activity. Moreover, we predicted greater LTP inducibility following the nap compared to the wake session. That is, we predicted a larger increase in TMS-induced MEP amplitudes in response to PAS following the nap.

2. Materials and methods

2.1. Participants

Twenty healthy adults participated in the study (9 females, 11 males, mean (SD) age 25.1 (2.9) years; Table 1). All participants were healthy sleepers (Pittsburgh Sleep Quality Index, PSQI, 3.6 ± 1.4) without a past or present sleep, somatic or psychiatric disorder. All participants were right-handed (Edinburgh Handedness Inventory), had a low alcohol and caffeine consumption (caffeine intake < 500 mg/ day or 5 cups of coffee per day), consumed less than five cigarettes per week, and were not pregnant. The sample showed a normal to high estimate of intelligence (MWT-B) (Lehrl, 2005), attention and memory recall. Eleven participants reported taking regular naps (0.6 (0.7) naps per day) at the time of the screening, with an estimated sleep duration of 47.7 (16.2) min. No increased daytime sleepiness was reported based on the Epworth Sleepiness Scale, and subjective nighttime sleep duration prior the nap or wake sessions did not differ. The participants were recruited from the community and were reimbursed for their participation. The study was approved by the ethics committee of the Albert-Ludwigs-University Freiburg and was conducted in accordance with the Declaration of Helsinki. Written informed consent was obtained from all participants prior to participation.

Table 1
Mean (standard deviation) and range of demographics and baseline characteristics of the study population.

Variable	$N = 20$ Mean (SD), range
Age	25.1 (2.9), 20–30
MWT-B	117.8 (11.1), 101–143
PSQI	3.6 (1.4), 2–6
BDI	4.4 (3.5), 0–13
ESS	9.2 (4.0), 4–18
Attention and memory performance	
Number repeated forward	8.2 (2.2), 4–12
Number repeated backwards	7.0 (1.9), 3–10
Total number repeat	15.2, (3.7), 9–22

MWT-B, Mehrfachwahl-Wortschatz-Intelligenztest B; PSQI, Pittsburgh Sleep Quality Index; BDI, Beck Depression Inventory; ESS, Epworth Sleepiness Scale; Attention and memory performance assessment based on Hamburg-Wechsler intelligence for adults. The administered tests and questionnaires are detailed in the supplementary methods.

2.2. Study protocol

Participants underwent a repeated measures protocol consisting of three appointments at the sleep laboratory; one screening visit with an adaptation nap and two experimental sessions – nap and wake – in a counterbalanced order, for one hour in the afternoon (1:15 to 2:15 PM; Fig. 1). The two experimental sessions were 1–3 weeks apart, to prevent carry-over effects. During the screening visit, demographic and baseline characteristics were assessed (Table 1). Participants were also screened for contraindications to transcranial magnetic stimulation (TMS). The possibility to induce a sufficient and consistent motor evoked response over the right primary motor cortex (M1) was assessed. The optimal stimulation position was stored as a reference for the two following experimental sessions. Finally, participants did an adaptation nap to get used to the sleep laboratory environment and setup.

During the experimental sessions, participants arrived at 11:30 AM. The measurement started with a wake EEG theta assessment, followed by the experimental (nap or wake) session. During the nap session, participants were given a sleep opportunity of one hour in the sleep laboratory, between 1:15–2:15 PM. The room was darkened, and any source of disturbance avoided. In the wake session, participants were supervised by study staff and engaged in conversation and short walks. Following each experimental session (nap/wake), corticospinal excitability was assessed using TMS. Following this, the inducibility of LTP-like plasticity was assessed using a combination of TMS and peripheral electrical stimulation (PAS protocol). The TMS assessments started 30 min after the respective session, to minimize possible effects of sleep inertia. Wake EEG theta activity was measured both before and after the sleep or wake session, as well as after the PAS protocol, as an index of net synaptic strength.

2.3. Sleep recordings

Electroencephalography (EEG) was recorded during wakefulness before and after each experimental condition (nap/wake) as well as during the nap period from the channels F3, F4, C3, C4, P3, P4, Oz, A1, A2, referenced to Cz and with Fpz as ground, placed according to the 10–20 system (Jasper, 1958) (SOMNOScreen Plus amplifier; Somnomedics AG, Randersacker, Germany; see Supplementary Methods for details). EEG spectral power densities were calculated for the nap period as well as the wake EEG recordings, across channels (F3, F4, C3, C4, P3, P4, Oz; see Supplementary Methods for details). Polysomnographic recordings were visually scored off-line by an experienced rater according

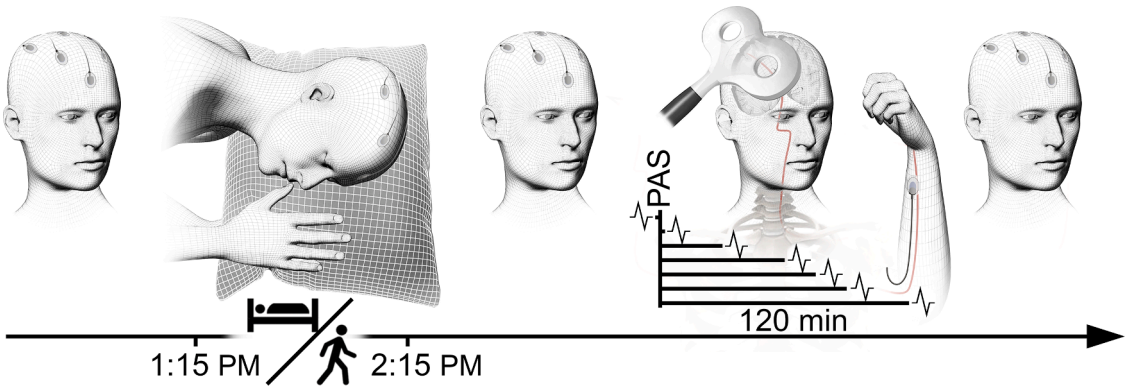

Fig. 1. Study design. Participants underwent a repeated measures protocol with an adaptation nap followed by two experimental sessions with either a wake or sleep period (counterbalanced order). Participants were characterized at the electrophysiological (wake and sleep EEG) and physiological level (MEPs). The effect of PAS (applied for 14 min) on MEP amplitudes was assessed at 6 time-points, up to 120 min post-PAS.

to standard criteria (Berry et al., 2017).

2.4. Indices of net synaptic strength

Single-pulse TMS was applied using a figure-of-eight stimulation coil over the right primary motor cortex (M1) to optimally target the left abductor pollicis brevis (APB) muscle (Fig. 1; The Magstim Company Ltd., Whitland, UK; see Supplementary Methods for details). The intensity of stimulation was calibrated to produce MEPs averaging peak-to-peak amplitudes ranging between 0.6–1.4 mV (SI_{1mV}). In addition to the SI_{1mV}, the resting motor threshold (RMT) was also assessed prior to the PAS protocol as a measure of corticospinal excitability following the nap and wake sessions.

We investigated wake EEG theta activity (3.5–8 Hz) prior to and after the nap and wake session as a non-invasive index of net synaptic strength, using 2.5 min of eyes-closed wake EEG (Kuhn et al., 2016; Fig. 1; see Supplementary Methods for details).

2.5. Indices of LTP-like plasticity

The PAS protocol was used to assess LTP-like plasticity (Kuhn et al., 2016; Stefan et al., 2000), comprising the application of pairs of electrical stimuli targeting the median nerve at the left wrist and cortical stimuli applied over the APB muscle hotspot at M1, for a total of 14 min. To index inducibility of LTP-like plasticity, changes in MEP amplitude in response to TMS (applied at SI_{1mV}) after the PAS intervention were examined across 6 time-points (+2 min, +30 min, +60 min, +75 min, +90 min, and +120 min) in comparison to a pre-PAS baseline (Fig. 1; see Supplementary Methods for details).

2.6. Statistics

MATLAB (R2024b, MathWorks Inc., Natick, MA, USA) was used for statistical analyses (see Supplementary Methods for details). The data are reported as means ± standard deviation, if not indicated otherwise. The level of statistical significance was set at $p < 0.05$ (two-tailed) for all analyses.

3. Results

3.1. Polysomnography

During the 1-h nap opportunity, participants slept on average 43.5 (11.5) min, of which they spent 98.5 % in NREM sleep (Table S1). Specifically, they spent 74.5 % of their sleep, or between 7–54 min, in N2 or N3, indicating a successful implementation of the nap intervention with a well-defined period of NREM sleep. The self-reported sleep duration in the night prior to the test session did not differ between the wake (454.9 (58.3) min) and nap condition (449.0 (52.5) min; $t(19) = -0.40$, $p = 0.69$; Cohen's $d = 0.09$). The EEG spectral band power measured during the nap is reported in Table S2.

3.2. Indices of net synaptic strength

3.2.1. TMS

In line with our hypothesis, we observed a significantly lower excitability after the nap compared to the wake session, as indexed by a significantly increased TMS resting motor threshold (RMT) after the nap versus wake (41.20 (6.20) vs 40.00 (5.47); $t(19) = -2.40$, $p = 0.03$; Cohen's $d = 0.54$; Fig. 2A). SI_{1mV} did not differ significantly following the nap compared to the wake period ($t(19) = -1.46$, $p = 0.16$; Cohen's $d = 0.33$). There was also no difference in the intensity of the electrical stimulation of the median nerve ($t(19) = -0.02$, $p = 0.98$; Cohen's $d < 0.01$) or the thumb ($t(19) = 0.33$, $p = 0.74$; Cohen's $d = 0.07$) between the wake and sleep condition.

3.2.2. Wake EEG theta

We investigated wake EEG theta activity (3.5–8 Hz) prior to and after the nap and wake session as well as after the PAS as a non-invasive index of net synaptic strength. In line with our hypothesis, we observed a significant increase of EEG theta power after the wake session compared to the nap session (Fig. 2B), indicating lower net synaptic strength after the nap. Specifically, the 2 × 2 rm-ANOVA showed no significant main effect for the factor Time ($F(1,18) = 1.23$, $p = 0.28$, $\eta p2 = 0.06$) or Condition ($F(1,18) = 2.08$, $p = 0.17$, $\eta p2 = 0.10$) on wake EEG theta power at electrode C4. However, there was a significant Time x Condition interaction ($F(1,18) = 6.98$, $p = 0.02$, $\eta p2 = 0.28$). Post-hoc comparisons showed a significant difference between conditions both immediately after the nap or wake period ($t(18) = -2.53$, $p = 0.02$; Cohen's $d = 0.81$) as well as after the PAS protocol ($t(17) = -2.59$, $p = 0.02$; Cohen's $d = 0.77$). These differences reflected a selective increase in EEG theta power after the wake period, both for pre-wake vs post- ($t(19) = -2.99$, $p < 0.01$; Cohen's $d = 0.67$) as well as vs after the PAS protocol ($t(18) = -4.20$, $p < 0.001$; Cohen's $d = 0.86$; Fig. 2B) at the electrode closest to the target region for the TMS. No significant difference was found in wake EEG theta activity prior to the nap or wake sessions ($t(18) = 0.07$, $p = 0.94$; Cohen's $d = 0.23$). Wake EEG theta activity also did not increase significantly between the measurements post-session and post-PAS, neither following wake ($t(18) = -1.10$, $p = 0.29$; Cohen's $d = 0.22$) nor following nap ($t(18) = -0.98$, $p = 0.34$; Cohen's $d = 0.22$; Fig. 2B).

The full EEG power spectrum at EEG channel C4 is shown in Supplementary Fig. S1. Exploratory analyses of the power spectrum revealed a reduction in the theta band (3.5–4.5 Hz) after the nap session

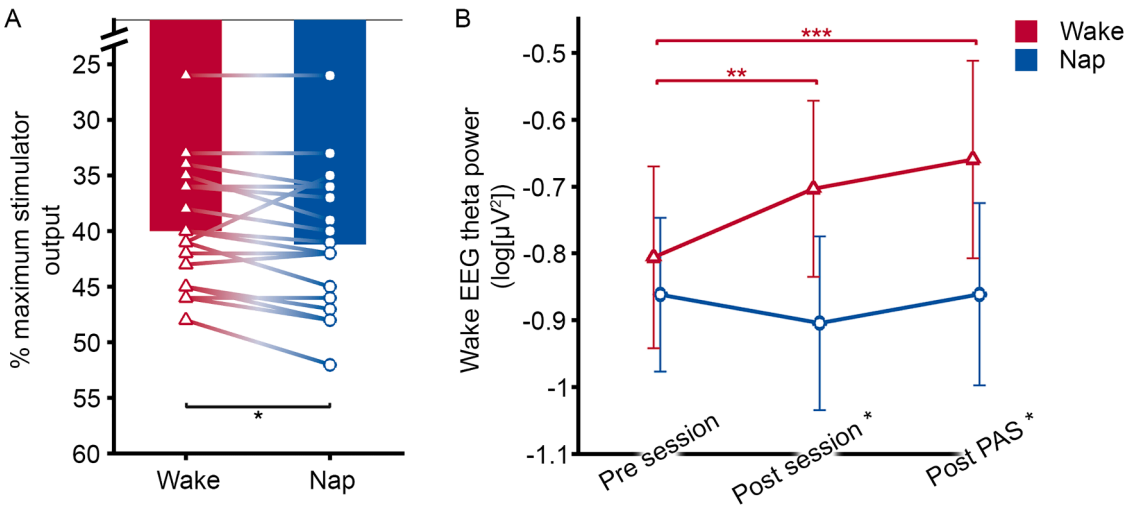

Fig. 2. Indices of net synaptic strength. **A.** The stimulation intensity of transcranial magnetic stimulation (% maximum stimulator output) to induce a threshold level motor response in the resting muscle (RMT) was higher following the nap compared to the wake condition, indicating lower excitability after the nap as compared to wake. **B.** Wake EEG theta power at electrode C4 (channel closest to the TMS target region) was increased following the wake session but not following the nap session. the mean wake EEG theta power ± standard error of the mean is shown per measurement time point. horizontal lines with asterisks indicate a significant difference between time points, while asterisks on the x-axis indicate significant differences between conditions per time points (paired-sample t-test; $*p < 0.05$, $**p < 0.01$, $***p < 0.001$). The full wake EEG spectrum is shown in Supplementary Fig. S1 as well as in Supplementary Fig. S2 (EEG spectrum with aperiodic component subtracted). The wake EEG theta power across the scalp is shown in Supplementary Fig. S3.

while there was an increase within the theta band (5 Hz) after the wake session ($p < 0.05$; FDR corrected). The exploratory analyses did not reveal any differences in the wake EEG power spectrum prior to the respective experimental session. As shown, exploratory analyses revealed condition effects in other frequency ranges. However, we restricted the main analysis and interpretation to the EEG theta range.

To ensure that observed differences in EEG power spectra were not driven by changes in the aperiodic (1/f-like) background activity, we estimated and removed the aperiodic component of the power spectrum using the FOOOF (Fitting Oscillations and One-Over-F) algorithm (Donoghue et al., 2020). The full wake EEG spectrum with the aperiodic component subtracted is shown in Supplementary Fig. S2, and confirms the result pattern seen in Fig. S1, with the notable exception of an absence of increase in broadband activity pre-nap session to post-PAS.

Finally, theta band power across the scalp is shown in Supplementary Fig. S3. Importantly, all our analyses confirm that there were no baseline differences prior to the nap and wake session, neither in the theta band across the scalp, not in any other frequency band or in the aperiodic component of the spectra.

3.2.3. *Indices of inducibility of associative plasticity*

In line with our hypothesis, we observed a significant increase in the inducibility of LTP-like plasticity through PAS after sleep, as indexed by an increase in MEP amplitudes in response to the PAS protocol following

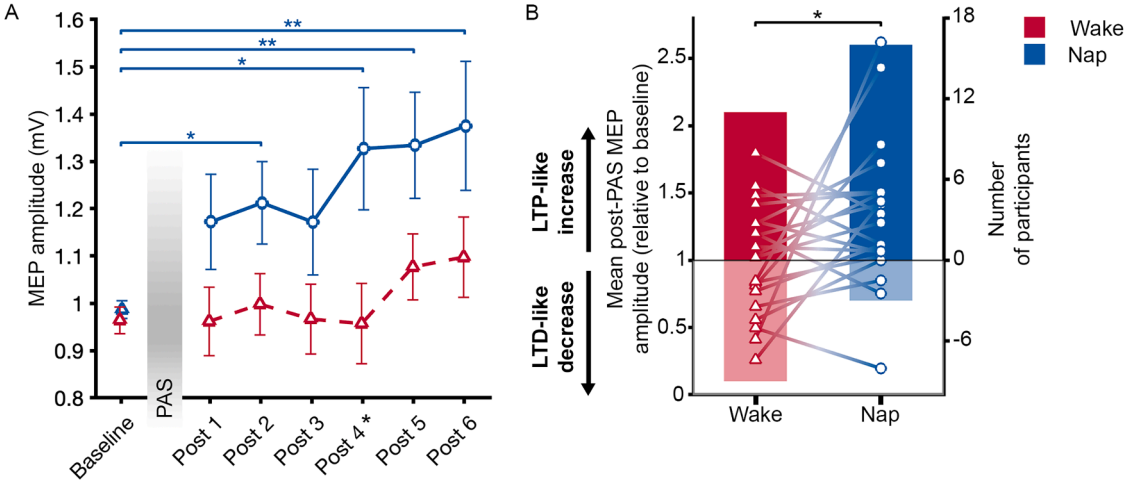

Fig. 3. Indices of inducibility of associative LTP-like plasticity. **A.** Time course of MEPs amplitude, showing an increase in MEP amplitudes in response to PAS following the nap but not the wake condition. The symbols represent the mean MEPs amplitude at the respective time point ± standard error of the mean. Horizontal lines with asterisks indicate a significant difference between baseline and post-PAS measurements, while asterisks on the x-axis indicate significant differences between conditions per time point (paired-sample t-test; $*p < 0.05$, $**p < 0.01$). **B.** Response pattern to PAS protocol at the single-subject level, at post-PAS measurement 4 (75 min), following either the wake or nap condition. Individual markers represent post-PAS MEP amplitudes normalized to pre-PAS baseline for each participant, with lines connecting responses from the same individual across conditions. Values >1.0 indicate an LTP-like increase in corticospinal excitability, whereas values <1.0 indicate an LTD-like decrease. Bars summarize the number of participants exhibiting LTP-like (>1.0) or LTD-like (<1.0) responses in each condition; bar heights are scaled to the right y-axis. Following the nap, a greater number of participants showed LTP-like responses compared to the wake condition (paired-sample *t*-test, $p < 0.05$).

the nap but not the wake session (Fig. 3A). Specifically, the 2 × 2 rm-ANOVA showed a significant effect of the factors Time ($F(1,19) = 7.50$, $p = 0.01$, $\eta p2 = 0.28$; pre vs average of post-PAS measurements) and Condition ($F(1,19) = 4.92$, $p = 0.04$, $\eta p2 = 0.21$), as well as a significant Time x Condition interaction ($F(1,19) = 4.73$, $p = 0.04$, $\eta p2 = 0.20$) on the MEPs amplitudes (Fig. 3A). Post hoc analysis showed that in the nap condition, MEPs were increased following PAS compared to the pre-PAS baseline, at time points 2, 4, 5 and 6 ($p < 0.05$; Fig. 3A). In contrast, MEPs were not significantly increased in the wake condition. Timepoint 4 post-PAS showed a significant difference between conditions ($t(19) = 2.30$, $p = 0.03$; Cohen's $d = 0.51$) and a trend toward significance at time point 5 ($t(19) = 2.05$, $p = 0.05$; Cohen's $d = 0.46$). The baseline MEP amplitude did not differ between the wake and the sleep condition ($t(19) = 0.69$; $p = 0.50$; Cohen's $d = 0.15$). Of note, the stimulation intensity to induce the baseline MEP (SI_{1mV}) did not differ significantly between the wake and the sleep condition ($t(19) = -1.46$, $p = 0.16$; Cohen's $d = 0.33$).

Overall, these findings support the notion that there was renormalization of synaptic plasticity during the nap along with an increased inducibility of LTP-like plasticity lasting for at least 2 h post-PAS (last measurement post-PAS).

To further characterize the response pattern to the PAS protocol at the single-subject level, we calculated the change in MEP amplitudes after the nap/wake session (at timepoint 4 post-PAS (75 min), where we found a significant difference between conditions) referred to baseline (Fig. 3B). After the nap, 80 % of the participants showed an increase (LTP-like response), while 20 % showed a decrease of MEP amplitudes. In contrast following wake, only 55 % of the participants showed an increase (LTP-like response), while 45 % showed a decrease in MEP amplitudes (LTD-like response).

3.2.4. Associations between sleep parameters and indices of synaptic plasticity and renormalization

As exploratory analyses, we assessed the associations between indices of homeostatic and associative synaptic plasticity, as well as their relationship to sleep during the nap. No significant associations were observed between polysomnographic parameters of the nap (shown in Table S1) and PAS-induced MEP potentiation. Likewise, no significant associations were observed between spectral power values during the nap (Table S2) and PAS-induced MEP potentiation. In contrast, a significant positive association was observed between the percentage of wakefulness during the nap and an increase in wake EEG theta from pre- to post-session (Fig. 4A). Note that, associations with REM sleep could not be evaluated, as only three participants exhibited brief periods of REM sleep during the nap. In addition, changes in wake EEG theta power from pre- to post-session were significantly associated with PAS-induced plasticity in a time-dependent manner, with stronger effects at later post-PAS time points (Fig. 4B). Specifically, a larger increase in wake EEG theta pre- to post-session was associated with lower inducibility of LTP-like plasticity, as indexed by PAS.

4. Discussion

This study demonstrates, in line with our hypotheses, indices of decreased net synaptic strength and increased inducibility of LTP-like plasticity in the human cortex following a short afternoon nap. The study is in line with the synaptic homeostasis hypothesis (Tononi and Cirelli, 2014) and prior work on nighttime sleep in humans (Kuhn et al., 2016; Salehinejad et al., 2022). It demonstrates, to the best of our knowledge for the first time in humans, that even a brief afternoon nap can promote the process of recalibration of homeostatic and associative synaptic plasticity in the cortex.

In line with our hypothesis, we observed indices of reduced synaptic strength after a brief period of daytime sleep compared to wake, in line with prior work on nighttime sleep (please refer to the recent meta-analysis by Zhang and colleagues, 2025). More precisely, we observed that a significantly higher intensity of TMS was needed to elicit an MEP (RMT) after sleep compared to wake, which indicates lower cortical excitability. A meta-analysis reported no consistent differences in 7 studies comparing RMT following whole night sleep versus sleep deprivation (small effect size; Zhang et al., 2025), while we observed a difference with a medium effect size. On the other hand, an increase in global corticospinal excitability has been observed as indexed by a lower TMS intensity to induce an MEP of an amplitude of approximately 1 mV (SI_{1mV}; Kuhn et al., 2016; Salehinejad et al., 2022). However, we did not observe differences in the SI_{1mV}. Thus, while observations from both protocols indicate increased global corticospinal excitability during continued wakefulness relative to sleep, the measures appear to differ in a manner that may depend on the phase of homeostatic buildup. In line with our observation of increased corticospinal excitability, we also observed indices of increased cortical net synaptic strength reflected by an increase in wake EEG theta power pre- to post-wake. Exploratory

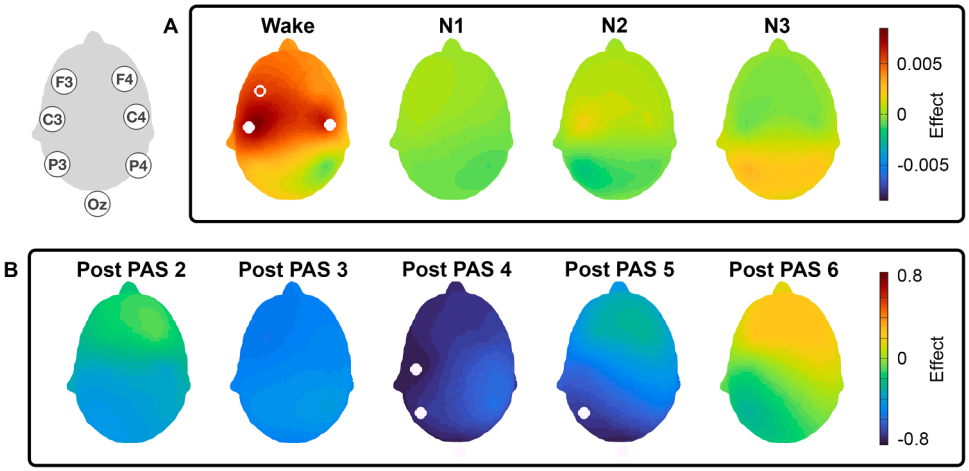

Fig. 4. Channel-wise associations. **A.** Channel-wise associations between polysomnographic parameters of the nap (% of sleep stages referred to total sleep time) and changes in wake EEG theta power (pre- to post-session). A positive association between wake duration and increase in EEG theta power was observed. **B.** Channel-wise associations between changes in wake EEG theta power (pre- to post-session) and PAS-induced changes in motor-evoked potential (MEP) amplitude across post-PAS time points. Associations were assessed using linear mixed-effects models with post-PAS time included as a fixed effect. Filled white circles indicate channels showing statistically significant associations after false discovery rate (FDR) correction across channels, whereas unfilled circles indicate effects that did not survive FDR correction.

analyses of the EEG spectrum revealed specifically an increase at 5 Hz pre- to post-wake, while we observed a decrease at 3.5–4.5 Hz pre- to post-nap session. Wake EEG theta power (3.5–8 Hz) has been proposed to reflect global net synaptic strength and is a marker of synaptic homeostasis. Theta activity also reflects the homeostatic component of sleep pressure (Finelli et al., 2000; Vyazovskiy and Tobler, 2005). Previous studies have shown that wake EEG theta power is reduced across whole night sleep while it is increased after sleep deprivation (Cajochen et al., 1995; Finelli et al., 2000; Kuhn et al., 2016). Recently, we demonstrated that a selective suppression of slow wave sleep inhibits the reduction of theta power. This effect correlated positively with the reduction in SWS and decrease in number of slow waves (Fehér et al., 2023). Theta activity has been observed to be higher in extensively engaged regions, and proposed to reflect an increase in population level synaptic strength (Bernardi et al., 2015; Fattinger et al., 2017; Hung et al., 2013; Vyazovskiy et al., 2011). An increase in net synaptic strength results in an increased tendency of neuronal populations to engage in synchronized activity, which in turn would result in higher amplitude EEG oscillations (Tononi and Cirelli, 2014). In sleep, the downscaling of synaptic strength is accordingly reflected by a gradual decrease in slow wave amplitudes during sleep. Recently, Snipes and colleagues used cycle-by-cycle analysis of wake EEG to show that the gradual increase in synaptic strength and neuronal synchrony throughout wake is reflected by an increase in amplitudes of EEG theta oscillations (Snipes et al., 2023). Together, our results confirm and extend prior results on nighttime sleep and point to a decrease in net synaptic strength across a short period of daytime sleep.

Finally, we observed a trend but not a significant increase in theta power between the post-nap or wake session and post-PAS (Figs. 2, S2 and S3). We speculate that this is consistent with the use-dependent increase in synaptic strength (Bernardi et al., 2015; Huber et al., 2006; Hung et al., 2013) and reflects the relatively controlled condition of the post-session to post-PAS period with restricted input and movement.

Moreover, in line with our hypothesis, we show higher inducibility of LTP-like plasticity after a nap compared to wake, which lasted 2 h after PAS. The PAS protocol has been shown to be a reliable measure for synaptic plasticity, being at least partly independent of motivational, effortful and attentional factors (Player et al., 2013). Our findings parallel prior findings following nighttime sleep versus sleep deprivation (Kuhn et al., 2016) and are in line with the synaptic homeostasis hypothesis, suggesting that sleep shifts the brain into a more favorable window of LTP inducibility. The present study extends our understanding of the restorative role of sleep, showing that a nap is sufficient to observe these processes. Indeed, many studies have assumed that the process of synaptic renormalization can occur also during a short daytime nap (1–2 h). This includes studies experimentally modulating deep sleep with auditory stimulation (Choi et al., 2019; Henin et al., 2019; Ong et al., 2018, 2016; Santostasi et al., 2016), electrical stimulation (Antonenko et al., 2013; Cellini et al., 2019; Garside et al., 2015; Ladenbauer et al., 2016; Julia 2017; Westerberg et al., 2015) or TMS (Manganotti et al., 2013; Massimini et al., 2007). Our study corroborates, to the best of our knowledge for the first time, that short periods of daytime sleep can induce similar effects as nighttime sleep. More precisely, it indicates that the average time spent in sleep stages N2 or N3 of 33.6 min during the nap session (Table S1) is sufficient to observe indices of synaptic renormalization.

It may be argued that our data could be explained by plasticity processes during wakefulness, i.e. that the observed differences in homeostatic and associative synaptic plasticity following nap versus wake may be driven by potentiation during wake rather than synaptic downscaling during sleep. Indeed, sustained wakefulness leads to synaptic potentiation, and synaptic saturation upon deprivation of sleep (Tononi and Cirelli, 2006). The changes in synaptic plasticity may be explained by the Bienenstock-Cooper-Munro theory of a "sliding threshold" of bidirectional synaptic plasticity, which proposes that the threshold for LTP/LTD induction is adjusted to the level of prior postsynaptic activity (Bienenstock et al., 1982). A period of lower synaptic activity during sleep will favor LTP induction, whereas a period of higher synaptic activity during sustained wakefulness will increase the threshold of LTP thus favoring the induction of LTD. However, in addition to an observed increase in wake EEG theta power pre- to post-wake, potentially reflecting synaptic potentiation, we also observed a pre- to post-nap reduction in the wake EEG theta band, in favor of synaptic downscaling during the nap.

Another observation in favor of an active role of sleep would have been an association between sleep macro and microstructure and indices of homeostatic and associative synaptic plasticity. While we observed a positive association between wake duration during the nap session and the increase in wake EEG theta activity pre- to post-session, we did not observe an association with sleep macro and microstructure in our data. It should be noted that we also did not observe such a correlation in a previous nap study with 30 participants (Maier et al., 2019), potentially due to a limited homeostatic build-up in our nap protocol. We also did not observe associations between sleep parameters and PAS indices of associative synaptic plasticity, potentially due to the study not being powered to identify these associations with sufficient reliability due to high variability in PAS response (Suppa et al., 2017). However, we observed an association between changes in wake EEG theta power from pre- to post-session and PAS-induced plasticity in a time-dependent manner, with stronger effects at later post-PAS time points (Fig. 4B). The results indicate that a larger increase in wake EEG theta pre- to post-session, indexing an increase in net synaptic strength, was associated with lower inducibility of LTP-like plasticity, as indexed by PAS.

Suppermpool and colleagues (2024) recently disentangled this inherent interdependency between sleep pressure and sleep duration in zebrafish larvae, showing how pharmacologically induced sleep during periods of low sleep pressure was insufficient to trigger synapse loss. Similarly, pharmacologically induced daytime naps versus wake in humans could more directly demonstrate the role of a homeostatic buildup for the renormalization of homeostatic and associative synaptic plasticity.

There are other protocols for TMS which, beyond providing a global measure of corticospinal excitability, can provide specific information about the cortical systems, including the neurotransmitters and neuromodulators, underlying the changes in homeostatic plasticity. Recently, Salehinejad and colleagues (2022) showed that a whole night of sleep deprivation compared to undisturbed sleep leads to enhanced glutamate-related cortical facilitation and decreases GABAergic cortical inhibition. It would be of interest in future studies to follow up with such an extended assessment following a daytime nap. In particular, a recent meta-analysis found that the short-intracortical inhibition protocol (SICI), provides a particularly sensitive measure of the impact of sleep and sleep deprivation on GABAergic cortical inhibition (Zhang et al., 2025).

Other variables known to account for variance in synaptic plasticity, such as gender, age and time of the day (Fathi et al., 2010; Tecchio et al., 2008) were controlled. Attentional focus, another confounding factor (Stefan et al., 2004), was controlled by having the participants count the number of stimulations on their thumb during the 14 min PAS (Stefan et al., 2004).

The present study indicates a benefit of naps for the inducibility of de novo associative synaptic plasticity. This complements prior work demonstrating that a nap can promote the consolidation of previously induced associative synaptic plasticity (Maier et al., 2019) and various types of memory consolidation, e.g. declarative memory (Piosczyk et al., 2013), texture discrimination (Nissen et al., 2021) and motor memory (Maier et al., 2019; J.G. 2017), with improvement in motor memory also having been shown to correlate with N2 sleep during the nap (Nishida and Walker, 2007). While these studies have demonstrated a benefit of naps for the consolidation of previously induced LTP, the present study shows for the first time in humans a benefit for the *de novo* induction of LTP after a daytime nap. This is also in line with the finding that

boosting SWA using oscillatory transcranial direct current stimulation during an afternoon nap enhances the capacity for encoding of information during subsequent wakefulness (Antonenko et al., 2013). It would be of interest in further studies to demonstrate how specifically changes in plasticity following a nap translate to the behavioral level, which would also be a means to quantify how substantial and beneficial the effect of a short nap is for the de novo induction of synaptic plasticity compared to full nighttime sleep.

A limitation of the present study is the absence of a sham PAS condition. While PAS was applied following both the nap and wake sessions to probe the impact of these states on the inducibility of LTP-like plasticity, the lack of a sham PAS condition prevents the identification of participants who did not exhibit a genuine plasticity response to the PAS protocol. Consequently, it was not possible to distinguish non-responders from responders, and participant responses could only be classified dichotomously as LTP-like or LTD-like based on the direction of MEP amplitude change. This precluded the use of a trichotomous classification scheme (LTP-like, LTD-like, non-response) to better capture inter-individual variability in PAS responsiveness (Nakamura et al., 2016; Tiksnadi et al., 2020). As a result, some observed response patterns may reflect variability in PAS susceptibility rather than state-dependent modulation of synaptic plasticity per se. Future studies incorporating a sham PAS condition would allow for a more precise dissociation of state effects on plasticity induction from individual differences in baseline PAS responsiveness. Furthermore, as suggested by Snipes and colleagues (2023), the analyses of the wake EEG might have benefited from a cycle-by-cycle analysis to disentangle the effect on the amplitude of theta activity which may more closely reflect changes in net synaptic strength, while conventional power spectral analysis does not distinguish between changes in amplitudes from changes in the number of oscillations.

Together, the present work is consistent with the primary hypotheses that a short afternoon nap, which differs in the duration, sleep architecture and timing from nighttime sleep, can lead to a decrease in indices of net synaptic strength and increased inducibility of LTP-like plasticity in the human cortex. These findings indicate that even a short afternoon nap can recalibrate homeostatic and associative synaptic plasticity, suggesting that sleep-dependent synaptic regulation can occur rapidly and outside the canonical context of nighttime sleep. The results provide potential mechanistic links to the beneficial effects of naps observed on various behavioral levels.

Ethical statement

This research was approved by the Ethics Committee of the Albert-Ludwigs-University Freiburg.

Data availability

Data and Code will be made available on request.

CRediT authorship contribution statement

Kristoffer D. Fehér: Writing – review & editing, Writing – original draft, Visualization, Software, Formal analysis. **Pauline Henckaerts:** Writing – review & editing, Writing – original draft, Formal analysis. **Valentin Hirsch:** Writing – review & editing, Investigation. **Ulrike Bucsenez:** Writing – review & editing, Investigation. **Marion Kuhn:** Writing – review & editing, Investigation. **Jonathan G. Maier:** Writing – review & editing, Investigation. **Carlotta L. Schneider:** Writing – review & editing. **Elisabeth Hertenstein:** Writing – review & editing. **Christian Mikutta:** Writing – review & editing. **Dieter Riemann:** Writing – review & editing. **Bernd Feige:** Writing – review & editing, Software, Formal analysis. **Christoph Nissen:** Writing – review & editing, Writing – original draft, Supervision, Resources, Project administration, Methodology, Funding acquisition, Conceptualization.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Supplementary materials

Supplementary material associated with this article can be found, in the online version, at doi:10.1016/j.neuroimage.2026.121723.

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