

Daytime Affect and Sleep EEG Activity: A Data-Driven Exploration

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Conflict of Interests

The authors declare no conflict of interest.

Author contributorship

J.X.Z and M.t.B conceptualized the study. M.t.B. and S.K. performed data collection. J.X.Z. and Y.Y. preprocessed the data. J.X.Z. performed data analysis and data visualization. R.M. and J.J.G supervised the study. J.X.Z. wrote the original draft of the manuscript. All authors reviewed and provided critical revisions to the manuscript.

Abstract

It has long been thought that links between affect and sleep are bidirectional. However, few studies have directly assessed the relationships between (1) pre-sleep affect and sleep EEG activity, and (2) sleep EEG activity and post-sleep affect. This study aims to systematically explore the correlations between pre-/post-sleep affect and EEG activity during sleep. In a community sample of adults ($n=51$), we measured participants' positive and negative affect in the evening before sleep and in the next morning after sleep. Participants slept at their residence for one night of EEG recording. Using Fourier transforms, the EEG power at each channel was estimated during rapid eye movement (REM) sleep and non-REM (NREM) sleep for the full range of sleep EEG frequencies. We first present heatmaps of the raw correlations between pre-/post-sleep affect and EEG power during REM and NREM sleep. We then thresholded the raw correlations with a medium effect size $|r| \geq 0.3$. Using a cluster-based permutation test, we identified a significant cluster indicating a negative correlation between pre-sleep positive affect and EEG power in the alpha frequency range during REM sleep. This result suggests that more positive affect during the daytime may be associated with less fragmented REM sleep that night. Overall, our exploratory results lay the foundation for confirmatory research on the relationship between daytime affect and sleep EEG activity.

Keywords: sleep, affect, EEG power, alpha band, REM sleep, data-driven

It is widely thought that links between daytime affect and nighttime sleep are bidirectional (Baglioni, Spiegelhalder, Lombardo, & Riemann, 2010; Kahn, Sheppes, & Sadeh, 2013; Tempesta, Soccì, De Gennaro, & Ferrara, 2018). According to this view, daytime positive and negative affect exerts an influence on how one sleeps at night, and how one sleeps at night exerts an influence on next-day positive and negative affect. Extensive research has been conducted on this topic (for a recent review, see ten Brink, Dietch, et al., 2022). In particular, studies have found relations between certain sleep stages, which are characterized by different brain activity profiles, and pre-/post-sleep affect (see a brief review below). These previous findings suggest that brain activity during sleep may be related to pre- and post-sleep affect. However, few studies have directly examined the relationship between pre-sleep affect and brain activity during sleep, or between brain activity during sleep and post-sleep affect.

Sleep Stages and Their EEG Characteristics

In humans, sleep occurs in stages that are characterized by their electroencephalography (EEG) profiles (Aeschbach & Borbély, 1993; Armitage, 1995; Fuller, Gooley, & Saper, 2006). EEG waves are typically higher in amplitude and lower in frequency during sleep (below 15 Hz) than during wakefulness (mostly alpha and beta bands). Sleep is broadly separated into non-rapid-eye-movement (NREM) sleep and rapid-eye-movement (REM) sleep. NREM sleep is further separated into three stages. When eyes are closed (during wakefulness), EEG exhibits synchronized waves that are predominantly in the 8-12 Hz range (alpha bands). During NREM stage 1, EEG oscillations slow down to primarily theta waves (4-7 Hz). During NREM stage 2, intermittent sleep spindles (12-15 Hz, sigma bands) and K-complexes appear in EEG (Aeschbach & Borbély, 1993; Armitage, 1995; Fuller et al., 2006). NREM stage 3, also known as slow wave sleep (SWS), typically contains high-amplitude delta waves that are even slower in frequency (< 4 Hz). During REM sleep, EEG transitions to a low-amplitude, mixed-frequency activity similar to NREM stage 1 and wakefulness. In general, the EEG power in beta frequencies (15-30 Hz) is generally low in all sleep stages (Armitage, 1995).

Pre-sleep Affect and Sleep

With the use of EEG recording during sleep, some studies have shown that pre-sleep affect influences or is associated with the architecture of sleep stages, which might have implications for brain activity during sleep. With an induction of negative affect prior to sleep¹, increased arousal and awakenings were observed during REM sleep (Vandekerckhove et al., 2011; Werner, Schabus, Blechert, Kolodyazhniy, & Wilhelm, 2015), and NREM stage 3 was found to be longer in absolute length and greater in proportion (Sopp, Brueckner, Schäfer, Lass-Hennemann, & Michael, 2019; Talamini, Bringmann, de Boer, & Hofman, 2013). In addition, it was reported that alpha activity increased in a nap following negative affect induction, although only for the first 30 minutes (Ackermann, Cordi, La Marca, Seifritz, & Rasch, 2019). One form of positive affect (i.e., relaxation) was found to be associated with longer REM sleep and less awakenings during NREM sleep (Feige, Baumgartner, Meyer, & Riemann, 2019). However, in another study, positive affect was found to be uncorrelated with sleep architecture (Ryff, Singer, & Love, 2004).

Taken together, a few research studies have examined how pre-sleep affect may influence sleep architecture, which suggests that pre-sleep affect might influence sleep EEG activity. However, few studies have directly examined how pre-sleep affect relates to EEG activity during subsequent sleep (e.g., EEG power).

Sleep and Post-sleep Affect

Findings regarding the association between sleep architecture and post-sleep affect have been mixed. On the one hand, some evidence suggests a role of REM sleep in consolidating or retaining negative affect (Lara-Carrasco, Nielsen, Solomonova, Levrier, & Popova, 2009; Wagner, Fischer, & Born, 2002). For example, longer duration of REM sleep was associated with elevated reactivity to negative stimuli (Werner et al., 2015), and higher

¹ These studies used various stimuli to induce negative emotion, such as aversive films and psychosocial stress.

percentage of REM sleep was associated with less attenuated reactivity at re-test after sleep to previously shown negative stimuli (Baran, Pace-Schott, Ericson, & Spencer, 2012). On the other hand, some studies have shown a role of REM sleep in reducing negative affect (Goldstein & Walker, 2014). For example, amygdala reactivity to negative stimuli decreased overnight in proportion to the duration of REM sleep (Wassing et al., 2019), lower gamma activity during REM sleep was associated with decreased reactivity to negative stimuli at retest (van der Helm et al., 2011), and individuals who were selectively deprived of REM sleep had greater reactivity to emotional stimuli compared to control individuals (Rosales-Lagarde et al., 2012). In terms of NREM sleep, existing studies suggest that slow-wave activity during NREM sleep is associated with less post-sleep negative affect. For example, NREM delta activity predicted less anxiety in the morning (Ben Simon, Rossi, Harvey, & Walker, 2020). With the occurrence of NREM stage 3 during a nap, autonomic reactivity to negative stimuli was attenuated while no attenuation was observed without NREM stage 3 (Pace-Schott et al., 2011).

Taken together, these findings on the relationship between sleep architecture and post-sleep affect might suggest relations between sleep brain activity and post-sleep affect, but they are mostly restricted in the domain of (experimentally induced) negative affect. Some studies have reported how sleep EEG activity in certain frequency bands relates to post-sleep affect, and yet a more systematic study exploring the full range of sleep EEG frequencies is needed.

The Present Study

The goal of the present study was to explore the relationship between (1) pre-sleep daytime affect and nighttime sleep EEG activity, and (2) nighttime sleep EEG activity and post-sleep daytime affect. Affect is an umbrella term for psychological states defined by relatively quick good-for-me/bad-for-me discriminations (Scherer, 1984), including emotion, stress, and mood (Gross, 2015). This study sought to extend prior research by examining negative affect, positive affect, and emotional arousal in a naturalistic way, and by exploring the full range of sleep EEG frequencies. In particular, we used a data-driven approach to understand the

correlations between naturalistic daytime affect and sleep EEG power in a community sample. To assess the overall affective tone accumulated during the daytime, we asked participants in the evening to rate their positive affect, negative affect, and emotional arousal during the day. Participants then slept overnight at their residence with ambulatory polysomnography (PSG) that included EEG and other channels. The next morning after getting up, they reported their positive affect, negative affect, and emotional arousal again. For each EEG channel, we quantified their EEG power for the full range of sleep EEG frequencies. We calculated the correlations between EEG power and pre-/post-sleep affect. In particular, we inspected the correlations that were medium-sized or greater to highlight the most robust results. We further used a cluster-based permutation approach to correct for the multiple tests (see Methods for details).

Methods

Participants

This study is part of the Stanford Home Sleep Study, which investigates links among affect, affect regulation, and sleep bruxism. We recruited participants with or without sleep bruxism, who have no other significant movement, dental, psychiatric, or medication conditions, using Facebook/Instagram advertisements, and flyers on campus, local hospitals, and dental clinics. Participants were not enrolled if they reported any medical conditions, medication treatment, dental conditions other than sleep bruxism, psychiatric conditions, sleep disorders, caffeine use disorder, intake of sleep medicines, or significant exposure to tobacco or nicotine products. This research does not focus on the sleep bruxism aspect of the sample.

For present purposes, we considered only those participants who completed the PSG recording of sleep ($n = 68$). Among the participants with usable data for spectral EEG analysis, $n = 51$ completed both pre-sleep and post-sleep affect measures, which was our final sample (age: $M = 29.76$, $SD = 8.01$; gender: 38 female, 12 male, 1 others). In the final sample, there were 22 White participants (43.14%), 22 Asian participants (43.14%), 2 White-Asian mixed-race

participants (3.92%), 1 African American (1.96%), 1 Pacific Islander (1.96%), and 3 others (5.88%). Six participants (11.76%) were Hispanic. There were 20 participants with self-reported sleep bruxism (39.22%), 17 without self-reported sleep bruxism (33.33%), and 14 unknown cases based on self-reports (27.45%). More sample characteristics can be found in the Supplemental materials (Table S1). All participants provided informed consent before participation.

Procedures

The Stanford Home Sleep Study involved participants for two weeks (also see Zhang et al., 2021). At the start of the two-week period, participants' at-home sleep was monitored for a single night of ambulatory PSG. Two trained research assistants visited the participants' home shortly after dinner time (times set relative to participants' dinner schedules) to set up the PSG, which took approximately 120 minutes. Participants reported their affect during the day using their smartphones 40-60 minutes prior to bedtime. Participants slept for one night with the PSG equipment following their usual schedule. In the next morning, participants reported their sleep and affect using their smartphones 40-60 minutes after getting up. The PSG device was removed by the participants themselves and returned to the lab by the participant during their lab visit. The study protocol was approved by the Institutional Review Board at Stanford University. All participants were financially compensated for their participation in the study.

Sleep Measures

Ambulatory PSG was recorded using Siesta802a wireless amplifier and recorder (Compumedics Ltd, Victoria, Australia), to a compact flash card via the Profusion Sleep Software (Compumedics Ltd, Victoria, Australia). The configuration includes 8-channel EEG (Figure 1A; F3, F4, C3, C4, P3, P4, O1, O2, online reference Pz, ground Cz, mastoid channels M1 and M2) with sampling rate at 1024 Hz, as well as EOG, ECG, and EMG. Sleep onset, wake, sleep stages, and events were scored by an AASM-registered PSG technician on 30-second epochs according to AASM guidelines (2017). Each scored PSG record was signed off

by a senior clinician at the Stanford Sleep Clinic. For artifact rejection, a trained research assistant manually marked all artifacts in the EEG signals, including physiological artifacts (facial muscle contraction, eye movements, cardiac artifacts, etc.) and non-physiological artifacts (electrode displacement, impedance problem, temporary disconnection, etc.).

Sleep EEG Data Reduction

The EEG power spectral analysis was conducted in MATLAB 2019a (Mathworks Inc., 2018), using functions from EEGLAB (Delorme & Makeig, 2004) and MATLAB. Data of all EEG channels during the in-bed period went through Butterworth bandpass filtering with order = 4 and range = [0.5, 50] Hz, and a 60-Hz notch filter. Continuous EEG data was then segmented into 5-second epochs. Data of eight EEG channels (F3, F4, C3, C4, P3, P4, O1, O2) were re-referenced to average M1 and M2. Two participants' M2 channel had artifacts throughout and their data were referenced to M1. We computed the spectral power density on artifact-free epochs using Fast Fourier Transform with a Hamming window (5 seconds in length, rolling window with 50% overlap, tapered 50% at both ends) for every 0.2 Hz from 0-45 Hz. Epochs containing artifacts were not included in the calculation of spectral power density for the contaminated channels. We then calculated the relative power by dividing the absolute power of a frequency bin by the total power density across all frequency bins. The relative power of each EEG channel during NREM and REM sleep is presented in Figure 1B. As most of the EEG power during sleep was within 0-15 Hz (97.8%), we present the data of 0-15 Hz in the main text and the data of 15-45 Hz in the Supplemental Materials (Figure S1-S2). In some participants, we observed harmonics of alpha rhythm at near 20 Hz, 30 Hz, and 40 Hz (Cervone & Blum, 2007; Mari-Acevedo, Yelvington, & Tatum, 2019).

Affect Measures

Affective experience was assessed with a 0-100 slider rating in a Qualtrics survey (Qualtrics, Provo, UT) filled out on participants' smartphones. Due to a need to decrease participant burden, the affect measures were based on the single item affect measure used in

Kron et al. (2013). For pre-sleep positive/negative affect and emotional arousal, in order to assess the overall affective tone accumulated during the daytime, we asked “How positive did you feel today?”, “How negative did you feel today?”, and “How emotionally activated or charged did you feel today?” respectively. For post-sleep positive/negative affect and emotional arousal, as participants filled out the surveys right after the potential sleep inertia period in the morning, we asked “How positive do you feel right now?”, “How negative do you feel right now?”, and “How emotionally activated or charged do you feel right now?” respectively. Responses were anchored at either end of the slider by the labels “Not at all” (0) and “Extremely” (100). The distributions of pre- and post-sleep positive affect, negative affect, and emotional arousal are presented in Figure 1C-1D respectively. Consistent with the idea that positive and negative affect are separate constructs, they were only moderately correlated pre sleep ($r = -.48$) and weakly correlated post sleep ($r = -.24$).

Data Analysis

We share the data and analysis script on Open Science Framework (<https://osf.io/42us9/>). Data analysis and data visualization was performed in R 3.5.1 (R Core Team, 2018) and Python 3.7.3. We calculated Pearson’s correlations between (1) pre-sleep positive/negative affect/arousal and sleep EEG power, and (2) sleep EEG power and post-sleep positive/negative affect/arousal, at every EEG channel for every frequency bin. Results were organized into matrices of channel by frequency and were visualized as heatmaps. We present the results of NREM and REM sleep in the main text and the results of individual NREM stages (i.e., NREM 1-3) in the Supplemental Materials (Figure S3-S4). We then applied an $|r| \geq 0.3$ threshold to the raw correlations to highlight clusters with at least medium-sized correlation (i.e., moderate or strong correlation). With this thresholding at a medium effect size, correlations ($|r| < 0.3$), which are considered weak and less reliable, are masked out. In order to correct for multiple statistical tests, we used a cluster-based non-parametric permutation test (threshold $r = 0.3$, 10000 permutations) to derive the probability p of getting the observed or even greater

cluster mass from permuted data (Maris & Oostenveld, 2007), using Python package *Eelbrain* 0.37 (<https://eelbrain.readthedocs.io/>; DOI: 10.5281/zenodo.6434429) which is a toolkit developed for EEG and magnetoencephalography (MEG) data analysis. The connectivity structure of the EEG channels was specified according to the 10-20 system. Clusters with $p < 0.05$ were highlighted. This cluster-based permutation approach has been a common practice in the analysis of multi-channel EEG data (Brodbeck, Gwilliams, & Pylkkänen, 2015; Endevelt-Shapira, Djalovski, Dumas, & Feldman, 2021; Verschueren, Vanthornhout, & Francart, 2020) and MEG data (Hartmann & Weisz, 2019; Li & Pylkkänen, 2021; Matar, Pylkkänen, & Marantz, 2019).

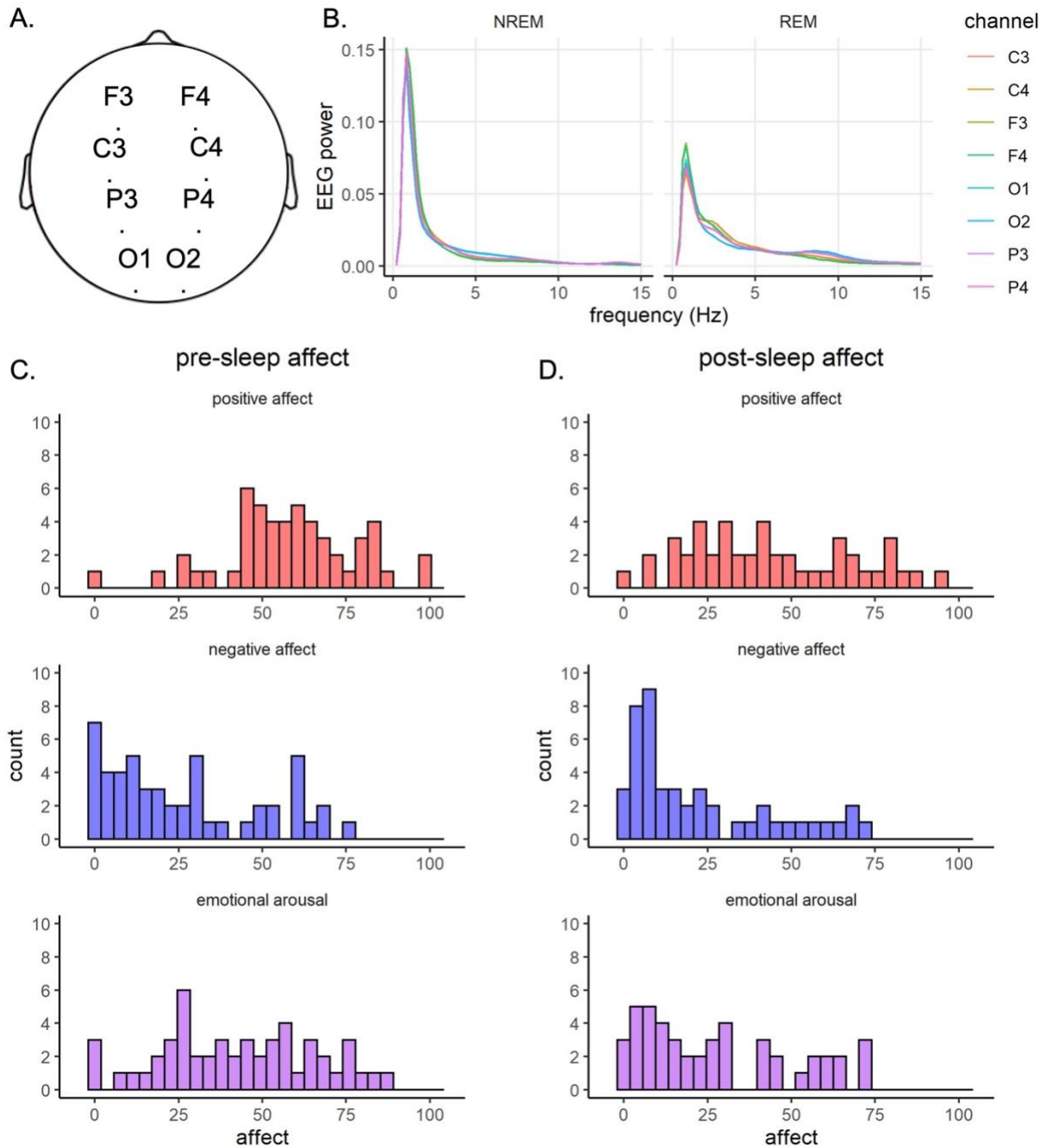


Figure 1. (A) EEG channel set-up. (B) EEG power across frequencies during NREM and REM sleep. (C) Distribution of pre-sleep positive affect (top, in red), negative affect (middle, in blue), and emotional arousal (bottom, in purple). (D) Distribution of post-sleep positive affect (top, in red), negative affect (middle, in blue), and emotional arousal (bottom, in purple).

Results

Pre-Sleep Affect and Sleep EEG Activity

We calculated Pearson's correlations between participants' pre-sleep reports of affect during the daytime and the EEG power during sleep. The correlation results are presented as matrices of EEG channel by frequency bin. We present the raw correlation matrices separately for each combination of sleep stages (NREM and REM) and affect categories (positive affect negative affect, and emotional arousal; Figure 2). We also present correlations thresholded at a medium effect size ($|r| \geq 0.3$) to highlight the most robust results.

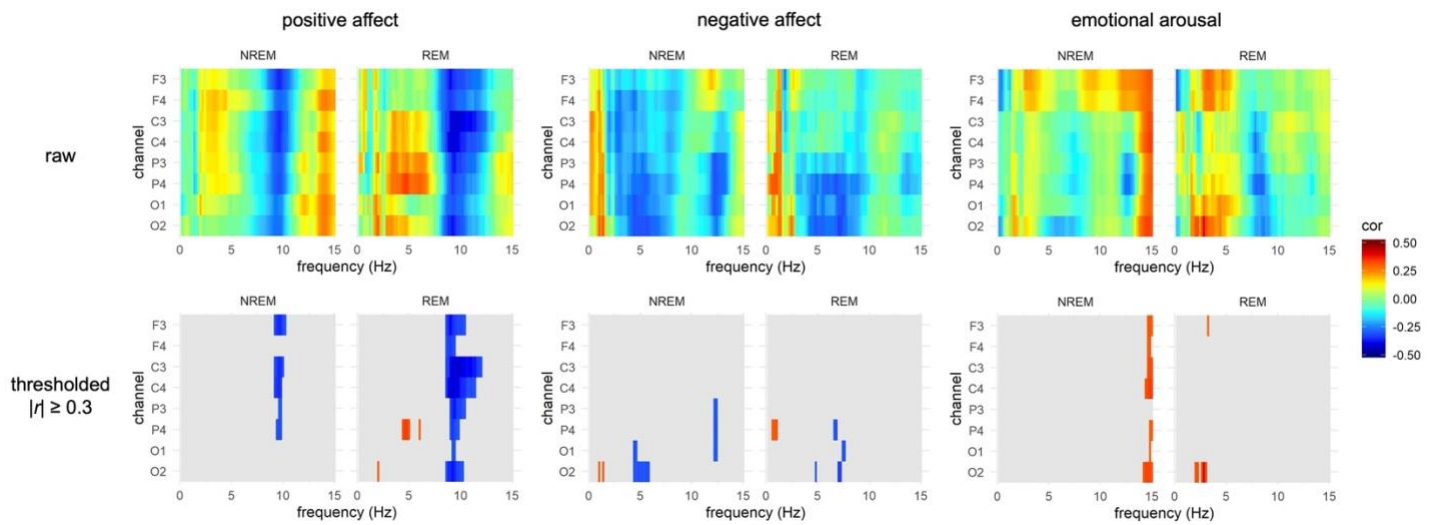


Figure 2. Correlation matrices between pre-sleep affect and EEG power during sleep.

Results are presented for each combination of sleep stages (NREM and REM) and affect categories (positive, negative, arousal). The top row shows the raw correlations and the bottom row shows the correlations thresholded at $|r| \geq 0.3$. Grey cells indicate correlations smaller than 0.3.

In order to correct for multiple statistical tests, we conducted cluster-based permutation tests. Each test was permuted 10000 times. The permutation tests showed one significant negative cluster for the correlation between pre-sleep positive affect and EEG power during REM sleep, permutation corrected $p = .046$ (Figure 3A and 3B). This significant cluster spanned all EEG channels and was in the alpha frequency range (8-12 Hz). We extracted the EEG power within this significant cluster (averaged across channels and frequency bins) and

visualized its correlation with pre-sleep positive affect ($r = -.38$, Figure 3C). We also visualized the correlation between pre-sleep positive affect and the EEG power across the full alpha frequency range (averaged across channels and frequency bins; $r = -.34$, Figure 3D). Follow-up linear regression models confirmed that self-reported sleep bruxism group (positive, negative, and unknown) did not significantly predict EEG power or interact with pre-sleep positive affect for the significant cluster (main effect: $F(2,44) = 0.80$, $p = .457$; interaction: $F(2,44) = 2.04$, $p = .142$) or the full alpha frequency range (main effect: $F(2,44) = 0.79$, $p = .460$; interaction: $F(2,44) = 1.58$, $p = .217$). These results suggest that more daytime positive affect was associated with less alpha activity during subsequent REM sleep. The thresholded cluster at a similar frequency range (around 10 Hz) in the correlation between positive affect and EEG power in NREM sleep (Figure 2, bottom left) did not reach the significance level, permutation corrected $p = .175$ (see Figure S3 for separate NREM stages). No other significant clusters were identified by permutation tests.

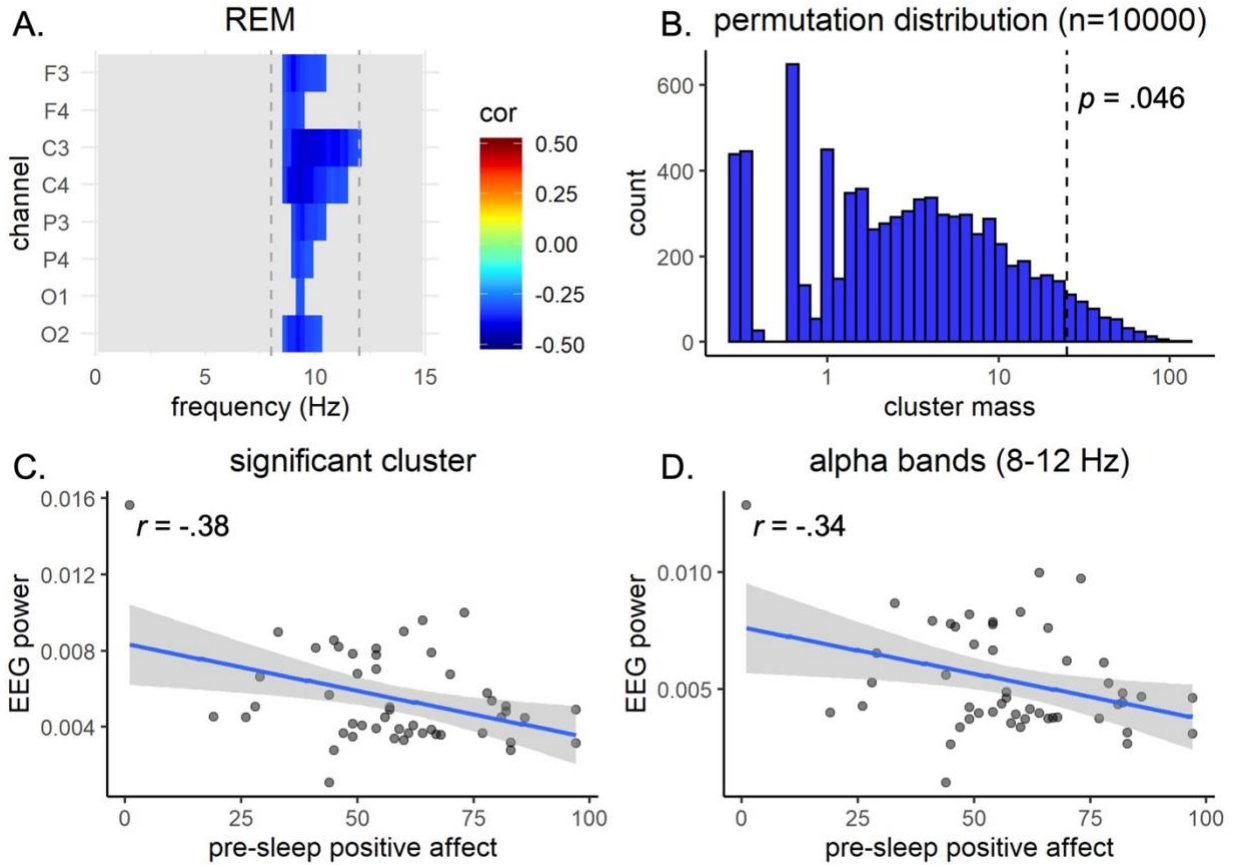


Figure 3. Cluster-based permutation test results. (A) A significant negative cluster was identified for the correlation between pre-sleep positive affect and EEG power during REM sleep. This cluster spanned all EEG channels and was in the alpha frequency range (8-12 Hz, between dashed lines). (B) Distribution of cluster mass in the permutation test. The data were permuted 10000 times and there was a small probability that a thresholded cluster in the permuted data was larger than the observed one, $p = .046$. The dashed line indicates the observed cluster mass in the original data. (C) Scatterplot between pre-sleep positive affect and EEG power within the significant cluster (averaged across channels and frequency bins). (D) Scatterplot between pre-sleep positive affect and alpha-band EEG power (averaged across channels and frequency bins).

Sleep EEG Activity and Post-Sleep Affect

To examine associations between sleep EEG power and post-sleep affect, we calculated Pearson's correlations between the EEG power during sleep and the affect

participants reported the next morning and present them as correlation matrices (Figure 4). We also thresholded the correlations with a medium effect size ($|r| \geq 0.3$) and conducted permutation tests. We found that no clusters survived the cluster-based permutation tests.

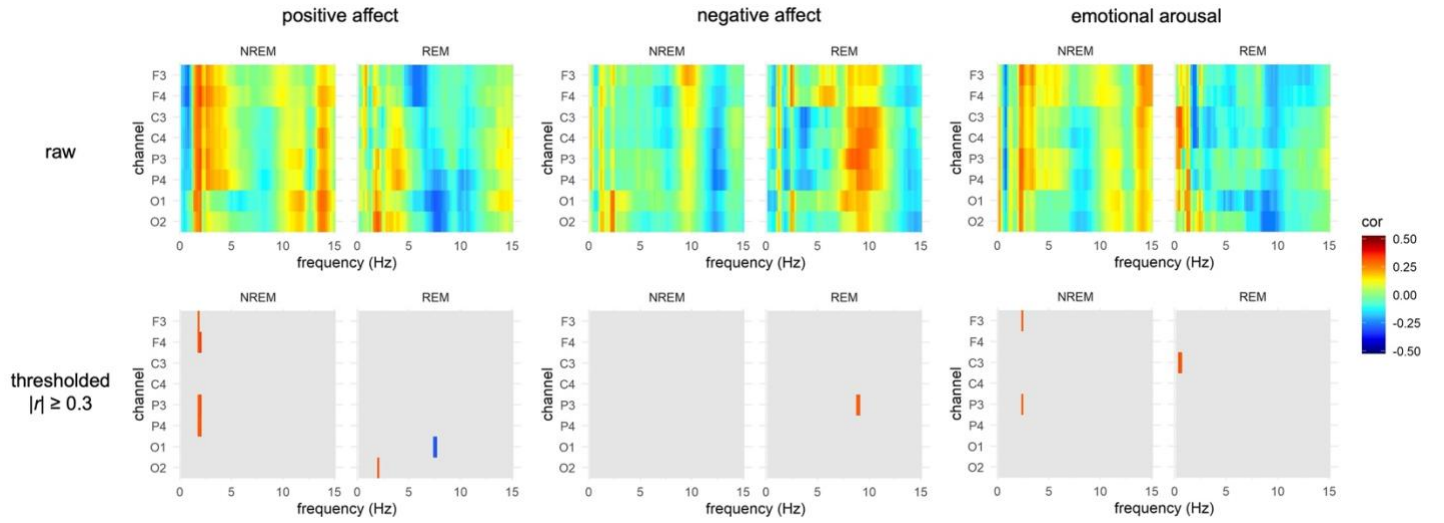


Figure 4. Correlation matrices between EEG power during sleep and post-sleep affect.

Results are presented for each combination of sleep stages (NREM and REM) and affect categories (positive, negative, arousal). The top row shows the raw correlations and the bottom row shows the correlations thresholded at $|r| \geq 0.3$. Grey cells indicate correlations smaller than 0.3.

Discussion

In this exploratory study, we examined the relationships between pre-/post-sleep affect and sleep EEG activity in a community sample. We first inspected the raw correlations between pre-/post-sleep affect and EEG power from 0-15 Hz during NREM and REM sleep. Using a threshold at a medium effect size and cluster-based permutation tests, we found a significant cluster indicating a negative correlation between pre-sleep positive affect and EEG power in the alpha frequency range (8-12 Hz) during REM sleep, such that participants with more pre-sleep positive affect during the daytime had less alpha activity during subsequent REM sleep. We found no significant associations between pre-sleep negative affect and EEG power, or between sleep EEG power and post-sleep negative or positive affect. To our best knowledge, this is the

first to systematically examine the correlations between daytime affect and sleep EEG activity at a full range of EEG frequencies.

Pre-Sleep Affect and Sleep EEG Activity

Alpha activity during sleep is thought to be an indicator of micro-arousals and sleep fragmentation (ASDA, 1992; Cantero & Atienza, 2000; Cantero, Atienza, & Salas, 2002). Our results suggest that more pre-sleep positive affect may be associated with less REM sleep fragmentation and thus more intact REM sleep. This is consistent with a previous study reporting that one form of positive affect (i.e., relaxation) was associated with longer REM sleep (Feige et al., 2019). It bears noting that although there was some indication of an association between pre-sleep positive affect and alpha activity during NREM (Figure 2, bottom left panel), these associations did not pass the cluster-based permutation correction. This relatively weaker association during NREM may be related to the specific role of REM sleep in affective processing (Goldstein & Walker, 2014).

It is noteworthy that, in our study, REM alpha activity was significantly associated with positive affect only, not with negative affect (Figure 2, right panel). We speculate that two factors could account for this. First, pre-sleep negative affect (range = [0, 76]) showed less variability in this sample compared to positive affect (range = [1, 97]) on the 0-100 scale (Figure 1C). The relatively smaller variation of pre-sleep negative affect might have made it more difficult to detect associations with sleep EEG. Second, previous studies reporting effects of negative affect induction on REM sleep typically implemented affect inductions that were high on the arousal dimension (e.g., stress induction, failure experience; Vandekerckhove et al., 2011; Werner et al., 2015). This was not necessarily true in our naturalistic observation of pre-sleep negative affect. With a similar naturalistic observation paradigm, another study also only found an effect of pre-sleep positive affect (but not negative affect) on subsequent sleep (Sin et al., 2017).

Sleep EEG Activity and Post-Sleep Affect

Contrary to prior reports in the literature (e.g., Baran et al., 2012; Ben Simon et al., 2020; Rosales-Lagarde et al., 2012), we failed to find evidence for significant associations between EEG activity during sleep and post-sleep affect the next morning. In fact, even before the multiple-tests correction using cluster-based permutation, the raw correlations between sleep EEG power and post-sleep affect were generally small in magnitude and very few passed the $|r| \geq 0.3$ threshold (Figure 4). One critical difference between the present study and previous studies is that most previous studies measured affective reactivity to experimental presentation of emotional stimuli or the change in affective reactivity after sleep (e.g., Pace-Schott et al., 2011; Wassing et al., 2019; Werner et al., 2015). In the present study, instead, we asked about participants' positive and negative naturalistic affect after sleep, without experimental stimulus presentation, which may explain some of the differences in findings. Although our exploration of the relationship between sleep EEG and post-sleep affect presents null results after thresholding and permutation correction, the raw correlation maps (Figure 4, top row) could serve as estimates of effect sizes for future confirmatory research.

Broader Implications

One implication of our exploratory findings is that there exists heterogeneity of the relationship between daytime affect and sleep EEG activity within the frequency range of traditional EEG bands and across EEG channels. We quantified EEG power across 0-15 Hz, thereby covering delta, theta, alpha, and sigma frequencies, and across frontal, central, parietal, and occipital EEG channels. One general observation is that the correlation between daytime affect and sleep EEG power may vary within traditional bands and across channels. For instance, the correlation between pre-sleep negative affect and NREM EEG power (Figure 2, top right panel) was generally positive in the lower range of delta frequencies (0-2 Hz) and negative in the higher range (2-4 Hz). To take another example, the correlation between pre-sleep negative affect and REM EEG power (Figure 2, top right panel) in the theta range (4-7 Hz) was generally negative in the parietal-occipital channels while it was close to zero in the frontal-

central channels. These results encourage future studies that would investigate the affect-sleep EEG relationship at a finer level across EEG frequencies and channels, rather than commonly used analytic approaches that aggregate data into traditional EEG bands (e.g., delta, theta, alpha, sigma, beta) or derive an “average” EEG channel.

Another implication of our study is that less pre-sleep positive affect during the daytime may be associated with more REM sleep fragmentation (Cantero et al., 2002; Simor, van der Wijk, Nobili, & Peigneux, 2020), although the role of alpha activity in REM fragmentation needs further clarification. Such fragmentation of REM sleep may lead to negative outcomes. It has been suggested that REM sleep plays a role in affect recalibration and that a lack of REM sleep may contribute to affective imbalance and psychopathology (Goldstein & Walker, 2014). In line with this, REM sleep fragmentation has been found to be associated with a genetic risk for depression in adolescents (Pesonen et al., 2019). Furthermore, restless REM sleep was found to contribute to long-lasting distress and further to hyperarousal, which may in turn accumulate to chronic insomnia (Wassing et al., 2016). Given the consequential nature of outcomes associated with REM sleep fragmentation, our preliminary finding of an association between pre-sleep positive affect and EEG power in the alpha band deserves further investigation. However, it should be noted that we did not have a direct observation of an association between REM EEG activity and post-sleep affect in our data.

Limitations and Future Directions

The present study extended prior work by examining bidirectional affect-sleep associations in a community sample with a focus on both negative and positive naturalistic affect, and a consideration of a wide range of EEG frequencies. However, several limitations of this study bear noting. First, as we have indicated in the title and throughout the paper, our study is data-driven and exploratory in nature. Although we did not test specific hypotheses, our results could serve as foundations for future confirmatory hypotheses. Also, our study introduces a data-driven analysis method for future studies in this space to consider using. It

should be noted that our sample size was only moderate for correlational analyses. Future studies are advised to use a greater sample if resources allow. Second, although we distinguished among relationships between pre-sleep affect and sleep and between sleep and post-sleep affect, our study is correlational and cannot support causal claims. Thus, the observed relationship between pre-sleep positive affect and REM alpha activity could be unidirectional (either direction), bidirectional, or due to a common cause. Experimental or longitudinal work is needed for establishing causality. Third, our analysis approach explores only linear relationships (Pearson's correlation) between daytime affect and sleep. Work in our group suggests that there may exist a non-linear relationship between affect and sleep in some cases (ten Brink, Yan, et al., 2022), which deserves more future research. Fourth, although we monitored participants' sleep at their residence to increase ecological validity, we only measured their sleep EEG for one night. The first-night effect may have injected perturbations to the assessment of participants' affect and sleep, even though we had introduced participants to the PSG procedure prior to the home visit to minimize the perturbations. Fifth, it should be noted that we measured pre-sleep and post-sleep affect via single-item questions, which potentially decreased construct validity, even though this has been a long tradition in the literature of affect measures (Bradley & Lang, 1994; Kron et al., 2013), especially in diary studies in order to decrease participants' burden (Quoidbach, Taquet, Desseilles, de Montjoye, & Gross, 2019; Yap et al., 2022). It should also be noted that our pre- and post-sleep affect questions employed different temporal frames. Specifically, we asked about pre-sleep affect across the day, whereas we asked about post-sleep affect at a given moment in the morning.

Conclusion

In this study, we explored the correlations (1) between pre-sleep affect and sleep EEG activity, and (2) between sleep EEG activity and post-sleep affect. We present the correlations in the format of heatmaps which could serve as estimates of effect sizes for future research. Our

results also highlight a negative association between pre-sleep positive affect and REM alpha activity, which deserves future confirmatory research.

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