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Research article

Memory deficits link trait-like EEG spectral profiles during REM and slow-wave sleep with shared symptoms of depression and anxiety

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ABSTRACT

Emerging evidence suggests that trait-level cortical hyperarousal during sleep–indicated by heightened EEG activity in higher frequency bands–disrupts memory processing and contributes to depression and anxiety, particularly during rapid eye movement (REM) and slow wave sleep (SWS). Given that memory deficits are putative risk factors for depression and anxiety, we hypothesized that memory deficits might mediate the association between elevated high-frequency EEG activity and greater symptom severity. A sample of 149 healthy adults (ages 18–59) encoded scenes featuring either negative (e.g., snake) or neutral objects (e.g., chipmunk) placed on neutral backgrounds (e.g., forest). Participants indicated whether an object or a background was the same, similar or new compared to what they encountered during encoding. Participants also underwent one night of laboratory-monitored polysomnography to assess trait-level spectral bandpower. General distress (a broad underlying factor of negative affect common to both depression and anxiety) was assessed using questionnaires. Results indicated that elevated beta and gamma power during REM sleep were significantly associated with false memory for negative objects (i.e., mistakenly recognizing negative objects not presented during encoding), which was in turn linked to greater general distress. During SWS, higher beta power was associated with reduced overall memory accuracy across all scene components, which was in turn related to greater general distress. However, mediation effects were not significant after controlling for age and sex. These results suggest that memory impairments may serve as a pathway linking trait-like, hyperarousal-related physiological profiles during REM and SWS to the increased comorbidity of depression and anxiety.

1. Introduction

Depressive and anxiety disorders are two of the most frequently diagnosed psychiatric conditions in the United States, with an estimated lifetime prevalence of 17 % for major depressive disorder and 31 % for any anxiety disorder [87]. These disorders frequently occur comorbidly [25,85,86] and share overlapping genetic factors [102]. Thus, it may be more appropriate to consider depression and anxiety as latent dimensions of internalizing symptoms rather than discrete psychiatric disorders [72,88].

Clark and Watson [35] proposed the Tripartite Model of Anxiety and Depression to distinguish depression-specific, anxiety-specific, and shared internalizing symptoms. In this framework, general distress (e.g., fatigue, irritability) reflects high negative affect and represents a *shared* component between depression and anxiety, while anhedonia (e.g., unhappiness, low pleasure) reflects low positive affect and is *specific* to depression, and anxious arousal (e.g., sweating, trembling, rapid heart rate) reflects physiological hyperactivation and is *specific* to anxiety [35]. This tripartite model has been widely researched and is very well supported by the existing literature [3,20,45,59,106]. Internalizing symptomatology can negatively affect different domains of daily living, including increased suicidal thoughts or behaviors and impaired academic or occupational performance [57,158]. Given growing evidence that alterations in memory processes and sleep physiology contribute to emotional dysregulation, it is important to understand how these factors might underlie the general distress that characterizes high internalizing

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symptomatology [34,111].

1.1. Memory and internalizing symptoms

One key vulnerability marker of internalizing symptoms involves memory, including improved memory for negative information [9,53]. Negative memories are generally remembered better than neutral ones [34,100] – a form of 'negativity bias' that can become excessive in conditions like anxiety and depression. Emotional arousal during encoding triggers the release of stress-related neuromodulators (e.g., norepinephrine and cortisol), which may in turn "tag" emotional content as significant and important to remember later [11,19,89,118]. Evidence suggests that individuals with more severe internalizing symptoms tend to show heightened stress and emotional reactivity to negative events and perceive them as more salient [9,52]. This is partly due to amygdala hyperactivation in individuals with high internalizing symptoms, which contributes to the tendency to focus on negative thoughts and feelings [15,81,107,108].

While the association between internalizing symptoms and negativity is well established, the role of memory remains unclear. Some studies show that individuals with higher depression and anxiety symptoms remember more negative than neutral stimuli compared to those with less severe symptoms [36,40,60,65,71,76,97,103]. However, other research finds no such evidence depressed or anxious individuals compared to healthy controls [58,78,90,94].

There are several potential explanations for these inconsistent results. Although negative information is often prioritized in memory processing, it is not always accurately remembered [39,131]. Internalizing symptoms are often associated with negative overgeneralization, a cognitive distortion in which emotional responses or interpretations of specific negative situations (e.g., witnessing a plane crash on the news) are extended to more general, innocuous events (e.g., perceiving any flight as dangerous; [101,123]). As a result, individuals with high internalizing symptoms may be more susceptible to negative false memories, which are inaccurate recollections of negative events that never occurred [101,127].

Relatedly, some studies demonstrate that individuals with higher internalizing symptoms have worse memory performance across all types of information regardless of its emotional valence [54,71,78,90,109]. These memory deficits possibly arise from diminished cognitive control, reduced motivation, and impaired pattern separation abilities due to hippocampal atrophy [53,70,137]. Thus, it is unclear whether individuals with high internalizing symptoms selectively remember the emotional aspects of memories due to extended negative emotional processing, or if they exhibit equally poor memory performance for both negative and neutral information. Therefore, to investigate memory features of emotionally salient events in relation to internalizing symptoms, the current study considers not only negative memory bias, but also assesses overall memory performance and the presence of negative false memories.

1.2. Sleep and memory

Emotionally negative memories with the highest degree of salience tagging during the encoding stage are also given priority during memory consolidation, including post-encoding sleep [12,13,89,118]. During Slow-Wave Sleep (SWS; also known as Stage 3 Sleep), memory traces that undergo repeated reactivation are integrated into coherent representations with pre-existing schemas in the neocortex, which is critical for long-term memory consolidation [16,17,66,92]. During Rapid Eye Movement (REM) sleep, increased levels of acetylcholine further reinforce newly formed neocortical representations through increased synaptic plasticity within the emotional memory circuit [12,55,92]. Although it seems likely that aspects of both SWS and REM sleep enhance memory consolidation, REM sleep, characterized by increased information flow between the hippocampus and amygdala, may be more pertinent for emotional memory consolidation than SWS [12,55,92].

Although theories suggest that REM sleep may be more relevant to the selective consolidation of negative memories than SWS, empirical findings have been inconsistent. Recent reviews and meta-analyses indicate that while REM-rich sleep may enhance memory performance for negative information compared to SWS-rich sleep, correlations between REM or SWS duration and negative memory performance have generally not been replicated [41,130]. In addition to limited statistical power in prior studies due to small sample sizes and methodological differences in memory assessment [37], these inconsistent findings may also stem from limitations in traditional sleep staging methods. These methods, based on American Academy of Sleep Medicine guidelines, categorize sleep into broad stages (e.g., REM, SWS) and provide information on the duration of each sleep stage and their proportion of the total sleep period [14]. However, the traditional sleep staging approach overlooks finer-grained physiological features in sleep microarchitecture.

Spectral power across different electroencephalogram (EEG) frequency bands reflects rhythmic brain activity during sleep and is highly heritable, as shown in twin studies [2,135]. Studies comparing spectral bandpower across multiple nights within the same individuals have found high test-retest reliability, even when sleep opportunity was restricted or during daytime naps [23,43,114]. These findings indicate that spectral bandpower is a robust, trait-like characteristic of sleep physiology with strong within-individual stability. Yet, it shows substantial between-individual variability and serves as a reliable predictor of age, sex, and cognitive profiles [33,117,140,146,157].

For example, SWS is marked by the temporal coupling of slow oscillations in the [0.1–1.25 Hz] frequency range and sleep spindles in the sigma range [12–16 Hz], a process strongly linked to memory reactivation [128,47–50]. In comparison, REM sleep is characterized by theta oscillations in the 4–8 Hz range, which, in conjunction with changing acetylcholine levels, further integrates previously consolidated memory traces during SWS [12,79].

While the protective role of lower frequency brainwave activity during sleep in memory consolidation is relatively well-understood, the impact of higher frequency activity, including beta [16–30 Hz] and low gamma [30–40 Hz], remains less clear. Beta and low gamma activity are typically prominent during quiet wakefulness, but tend to decrease during restorative sleep [22]. In conditions like insomnia and PTSD, elevated beta and gamma activity during sleep is often observed, suggesting a link between disrupted sleep and increased cortical hyperarousal [125,150].

This elevated cortical hyperarousal may interfere with neural activities necessary for optimal memory consolidation processes [96,156]. A recent study demonstrated that elevated beta power during sleep is associated with worse memory for extinguished fear [46]. One possible explanation is that heightened cortical hyperarousal may weaken the top-down control of the medial prefrontal cortex over the amygdala, resulting in disinhibited emotional responses [61,156]. Additional research is clearly needed to investigate how elevated beta and gamma activity during sleep may affect episodic memory for emotionally negative information.

1.3. Sleep, memory, and internalizing symptoms

Given the crucial role of sleep in memory consolidation [12,13,89,118,120] and the established link between memory and internalizing psychopathology [9,53], recent studies have begun to investigate the intricate relation between sleep, memory, and internalizing symptoms. For example, theories propose that increased REM sleep, such as longer REM sleep duration and higher REM sleep percentage, provides additional opportunities for the excessive salience tagging of negative events and feelings [74,75,147,148]. Consequently, these negative memories are preferentially and maladaptively consolidated during REM sleep, which in turn contributes to the initiation and maintenance of mood

disturbances [74,75,147,148]. In support of this, one study found that individuals reporting high depression symptoms exhibited better memory for negative stimuli following a 3-hour sleep period in the latter half of the night (e.g., 3:30–6:30 AM), a time dominated by REM sleep [73], although another study did not find such effects [68].

The current literature suggests that individuals with increased REM sleep, reflected in longer duration or higher percentage, may be more susceptible to developing internalizing symptoms partly due to their negatively biased memory representations. Although increased REM sleep is considered a characteristic of depressive disorders (e.g., Major Depressive Disorder) rather than anxiety disorders, the high comorbidity and overlapping genetic factors between depressive and anxiety disorders highlight the need for more research on memory as a pathway linking REM sleep to anxiety-related internalizing symptoms [25,29,85,86,102]. In addition, since internalizing symptoms are linked to general memory impairments [53,137], impoverished SWS may also increase vulnerability to internalizing symptoms by limiting opportunities to stabilize long-term neocortical storage of newly encoded memories [27,55,92]. Finally, beyond the duration and proportion of REM and SWS, more evidence is needed to explore the role of spectral power during sleep within different frequency bands (e.g., delta, theta, beta, gamma) in relation to memory and internalizing symptoms.

1.4. The current study

Our first aim was to identify memory features associated with different dimensions of internalizing symptoms, as outlined in the Tripartite Model of Anxiety and Depression [35]. We distinguished between a) common internalizing symptoms—general distress, or high negative affect experienced in both depression and anxiety, b) depression-specific symptoms—anhedonia, or low positive affect that characterizes depression but not anxiety, and c) anxiety-specific symptoms—anxious arousal, or physiological hyperactivation experienced only in anxiety. Past studies have linked internalizing symptoms to negative memory bias, negative false memory, and overall memory deficits [9,53,123,137]. Based on these findings, we hypothesized that three sets of memory features would predict more severe internalizing symptoms: a) negative memory bias, reflected by enhanced memory for emotionally negative content, b) negative false memory, indicated by mistakenly identifying negative items that are never presented during encoding, as previously encountered, and c) overall memory deficits, measured by worse memory across both negative and neutral information.

The second aim was to examine how trait-like microarchitectural features during REM and SWS influence negative memory bias, negative false memory, and overall memory deficits. We predicted that increased theta activity during REM sleep, given its emotion-specific consolidation effects, would be associated with increased negative memory bias [147,148]. Additionally, as elevated beta and gamma power during sleep–markers of cortical hyperarousal–contribute to impairments in memory and emotional processing [46,61,156], we explored whether these higher frequency bands during REM and SWS would be linked to overall memory deficits and negative false memory.

The third aim was to assess whether memory features mediate the effects of REM and SWS on internalizing symptoms. Specifically, we investigated the following mediating paths of sleep microarchitectural features predicting internalizing symptoms: a) increased theta power during REM sleep via negative memory bias, and b) elevated beta and gamma power through negative false memory and overall memory deficits. We explored whether these mediating effects influenced common, depression-specific, and anxiety-specific internalizing symptoms in similar or different manners. In this preregistered study (https://doi.org/10.17605/OSF.IO/8YVC5), we tested these aims in a community sample of young and middle-aged adults (18–59 years of age).

2. Methods

2.1. Participants

We recruited 161 eligible participants via flyers, newsletters and social media advertisements in South Bend, Indiana. To be eligible, participants needed to meet all of the following criteria: 1) be between 18 and 59 years of age, 2) have no history of diagnosed sleep, psychotic, and neurological disorders, 3) have no current use of sleep aid medications, 4) have no history of prescription medications for psychiatric disorders or have taken the same dosage for at least a year, and 5) have normal or corrected vision. Of the 161 eligible participants, four chose to drop out from the experiment, five had incomplete memory data, and additional three had incomplete survey data, leaving 149 participants included in behavioral analyses (M_{age}=36.58, SD_{age}=12.75). More than half of the sample reported biological sex as female (65.8 % female, 34.2 % male, and 0.0 % intersex). The majority identified as white (84.6 % White, 6.6 % Black/African American, 8.8 % other; 10.7 % Hispanic/Latino/Spanish). The procedures were approved by the Institutional Review Board at the University of Notre Dame. Participants provided written informed consent and received financial compensation for their participation.

The 149 participants with complete behavioral data (i.e., memory and internalizing symptoms) were assigned to one of two conditions, in which the retention interval between encoding and recognition memory testing spanned either nighttime sleep (n = 89) or daytime wake (n = 60). All 89 participants in the nighttime sleep condition completed an overnight laboratory polysomnography (PSG) following the encoding task, while 41 participants in the daytime wake condition underwent PSG after the recognition task. Data from the two conditions were collapsed in analyses to maximize statistical power, as we aimed to use spectral bandpower as a trait-like characteristic of sleep physiology, rather than to examine mechanistic memory processing during post-encoding sleep.

Among the 130 participants who completed laboratory PSG, data were either not collected or consisted primarily of pervasive noise or flatlining throughout the night for 16 participants due to technical issues during data acquisition. Of the remaining 114 participants whose overall sleep architecture was preserved, additional exclusions were made for poor data quality during the first half of the night that primarily interfered with SWS recording (n = 6), or during the second half of the night that affected REM sleep (n = 12), This resulted in 108 participants included in SWS analyses and 102 in REM analyses. See Fig. 1 for participant flow chart for each condition.

2.2. Materials

2.2.1. Mental health surveys

2.2.1.1. Individual questionnaire. The Beck Depression Inventory (BDI) is a 21-item questionnaire measure of depression [8]. Each item presents a group of four-point Likert scale variables (e.g., 0–"I do not feel sad", 1–"I feel sad", 2–"I am sad all the time and I can't snap out of it", and 3–"I am so sad and unhappy that I can't stand it"). Participants choose the statement that best describes how they have been feeling over the past week from each group of four statements. The BDI has good internal consistency and convergent validity [124].

The Beck Anxiety Inventory (BAI) is a 21-item questionnaire designed to assess anxiety symptoms [7]. For each item, participants rate how much they have been bothered by a symptom (e.g., "numbness or tingling") over the past month, on a four-point Likert scale from [not at all] to [severely—it bothered me a lot]. The BAI is a reliable and valid measure of anxiety symptoms [42,83].

The trait form of the State-Trait Anxiety Inventory (STAI) measures anxiety symptoms across typical situations that individuals commonly

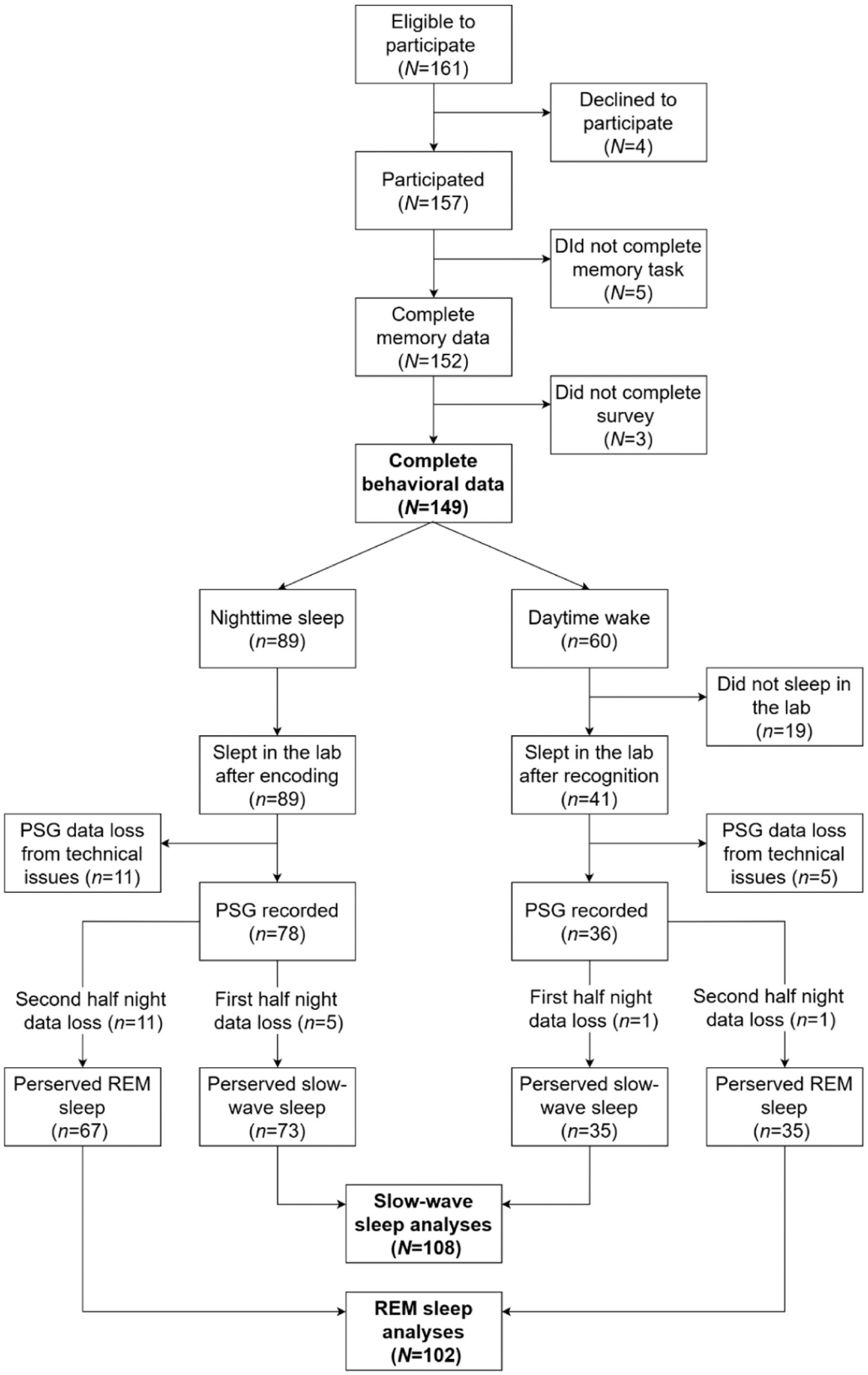

Fig. 1. *Participant Flow Chart Note*. Behavioral analyses of memory and internalizing symptoms included 149 participants with complete data. Of these, 89 completed an overnight laboratory polysomnography (PSG) session following encoding, 41 completed PSG after recognition, and 19 did not sleep in the lab. Using all available PSG data and excluding recordings with poor data quality, REM sleep analyses included 102 participants and slow-wave sleep analyses included 108 participants.

experience on a daily basis [139]. Participants rate a total of 20 statements (e.g., "I feel nervous and restless") based on how they generally feel, using a four-point Likert scale from [almost never] to [almost always]. The STAI has demonstrated good internal consistency and construct validity [83].

2.2.1.2. Dimensions of internalizing symptoms. Internalizing symptoms in the current study were assess using a total of 60 items from The BDI[1] [8], BAI [7], and the trait form of the STAI [139]. Table 1 shows examples, definitions, and interpretation of five dimensions of internalizing symptoms based on the Tripartite Model of Anxiety and Depression [35,151]. See Table S33 for a full list of items within each dimension. Since each item was rated on a scale from 0 to 3, total scores ranged from 0 to 39 for mixed general distress (13 items), 0–30 for depressive general distress (10 items), 0–39 for anxious general distress (13 items), 0–27 for anhedonia (9 items), and 0–45 for anxious arousal (15 items).

2.2.2. Emotional memory trade-off task

2.2.2.1. Encoding. During incidental encoding of the Emotional Memory Trade-Off (ETO) task, participants viewed scenes on a 53.1 × 30.2 cm desktop monitor (1920 × 1080 resolution; 60 Hz refresh rate). Scene stimuli (700 × 700 pixels) appeared at the center of a light gray background. Participants were presented with a series of 32 negative and 32 neutral scenes in a random order, with interstimulus interval varying randomly from 2 to 4 s. Negative scenes featured a negative object placed on a neutral background, while neutral scenes depicted a neutral object placed on a neutral background (Fig. 2A). Participants rested their left hand on the desk and used their right hand to respond via a button box (1 =thumb, 2 =thumb, 3 =index finger, 4 =middle finger, 5 =ring finger). After viewing each scene for 5 s, participants rated the scene for its valence (1 =very negative, 5 =very positive), and arousal (1 =highly calming/subduing, 5 =highly agitating/exciting) at their own pace. Prior studies that used the same stimuli consistently have found that, on average, negative scenes were rated as more negative and arousing compared to neutral scenes [51,109,110].

Table 1
Definitions, examples, and interpretation of five dimensions of internalizing symptoms based on the tripartite model of anxiety and depression.

	Examples	Definition	Interpretation
Mixed general distress	1) Irritability 2) Indecisiveness 3) Tiredness or fatigue	A component of negative affect equally associated with depression and anxiety	Shared symptoms of depression and anxiety
Depressive general distress	1) Sadness 2) Guilty feelings 3) Self-dislike	A component of negative affect more associated with depression than anxiety	Non-specific symptoms of depression
Anxious general distress	1) Nervousness 2) Worry 3) Disturbing thoughts	A component of negative affect more associated with anxiety than depression	Non-specific symptoms of anxiety
Anhedonia	1) Pessimism 2) Loss of pleasure 3) Loss of interest	Low positive affect, only associated with depression	Specific symptoms of depression
Anxious arousal	1) Hands trembling 2) Felling of choking 3) Difficulty breathing	Physiological hyperactivation, only associated with anxiety	Specific symptoms of anxiety

2.2.2.2. Recognition. In a surprise recognition test conducted approximately 12 h later, participants viewed a series of 192 items where objects and backgrounds were presented separately and one at a time in a random order (Fig. 2**B**). Each item was shown for up to five seconds. Participants were instructed to indicate: 1) "same" for objects or backgrounds that were identical to components of scenes that they viewed in the previous session, 2) "similar" for objects and backgrounds similar but not identical to previously viewed ones, as they might be the same type of object/background but differed in visual details ("For instance, you may have seen a bag of popcorn before, but not the particular bag of popcorn shown here"), and 3) "new" for entirely novel objects or backgrounds not previously viewed ("For example, you never saw a bag of popcorn in a scene presented in the previous session"). Participants indicated their response using a button box with their right hand (1 ="same" with index finger; 2 ="similar" with middle finger; 3 ="new" with ring finger). Among the 192 items, 64 were *same* to components of scenes during encoding, 64 were *similar* but not identical, and the rest 64 were entirely *new*. We counterbalanced the presentation of the *same* and *similar* versions of each item so that no participant viewed both. All task instructions and stimuli were presented using MATLAB R2021b [142].

2.2.2.3. Key memory variables. We scored hit rates as the proportion of trials where participants responded "same" or "similar" to a *same* item. We scored false alarm rates as the proportion of trials where participants responded "same" to a *new* item.[2] We then derived a corrected recognition memory score for each scene component by subtracting false alarm rates from hit rates [136]. For hits, false alarms, and corrected recognition, we compared performance of negative objects with both neutral objects on separate scenes (*difference* score), and neutral backgrounds on same scenes (*trade-off* score). Negative memory bias was operationalized as higher hits for negative objects, higher hit difference scores (negative vs. neutral objects across different scenes), or higher hit trade-off scores (negative objects vs. neutral backgrounds within the same scenes). Negative false memory was indicated by higher false alarms for negative objects, higher false alarm difference scores, or higher false alarm trade-off scores. Overall memory deficits were reflected in worse recognition memory, lower hits, or higher false alarms averaged across all scene components.

2.2.3. Overnight PSG recordings

2.2.3.1. Data acquisition. Polysomnography (PSG) is a safe, noninvasive procedure that uses an array of electrodes to measure multiple physiological signals during sleep and wakefulness [91]. In this study, PSG data were collected using the actiCAP Electrode Cap for LiveAmp developed by BrainVision (Brain Products, Gilching, Germany) at a sampling rate of 500 Hz. The recording montage included 10 electroencephalography (EEG) electrodes (F3, F4, C3, C4, P3, P4, O1, O2, Cz, Pz) positioned on the scalp according to the standardized International 10–20 System to monitor brain activity. In addition to EEG, two electromyography (EMG) electrodes were affixed two centimeters to the left and right of the underside of the chin to detect muscle tone changes. The system also included two electrooculography (EOG) electrodes: one positioned one centimeter to the right and above the right eye, and the

[1] Two items were excluded from the BDI (loss of interest in sex and suicidal thoughts) due to Institutional Review Board restrictions, resulting in the inclusion of 19 BDI items in our analyses.

[2] More stringent measures of hits ("same" to a *same* item) and false alarms ("same" and "similar" to a *new* item) showed similar associations with other key study variables but weaker effect sizes so we sticked with more lenient measures (Figure S15).

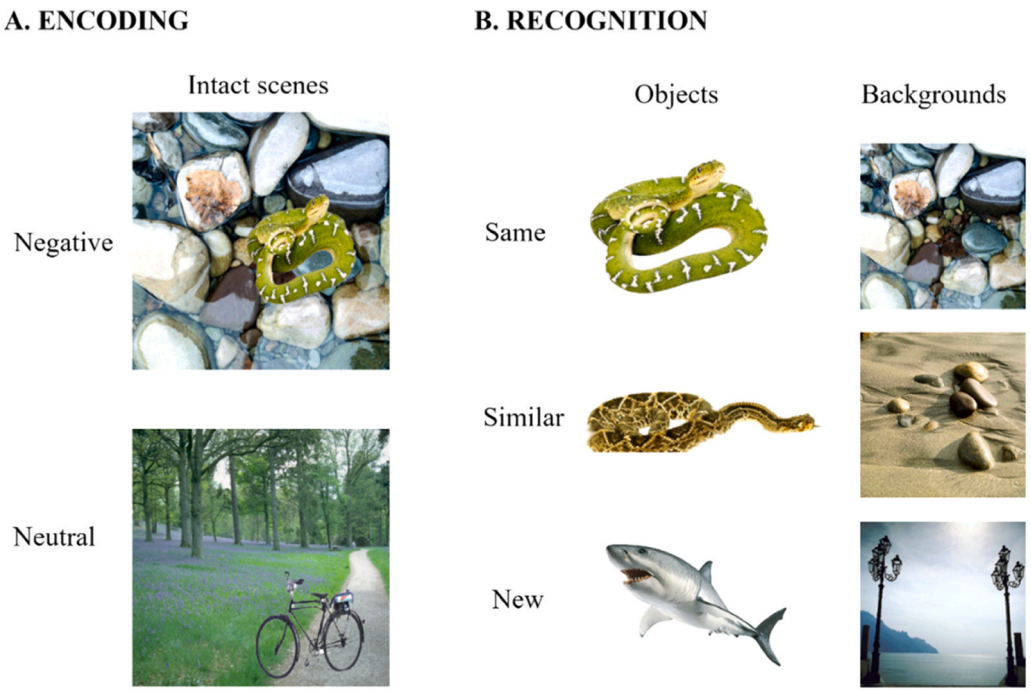

Fig. 2. *The Emotional Memory Trade-Off Task, Note.* (A) Encoding. A negative scene may include a snake placed on wet pebbles, whereas a neutral scene might feature a bicycle in a woodland setting. (B) Recognition. A *same* item would be an exact match to components of scenes viewed during encoding. A *similar* item would share the same verbal label as a viewed scene component but differ in specific visual details. A *new* item would not have been seen during encoding.

other one centimeter to the left and below the left eye to capture eye movement. Two additional electrodes were attached on the right and left mastoids.

Participants wore an appropriately sized EEG cap, which was then connected to a wireless EEG amplifier. All sensors were filled with abrasive conductive gel. Before bedtime, additional gel was applied as needed, and each sensor was secured with tape to prevent drying overnight. For added comfort and stabilization, the EEG cap was wrapped with gauze. All electrodes achieved good (<10 kΩ) to acceptable (<15 kΩ) contact impedance before data acquisition. EEG data were collected using BrainVision Recorder.

2.2.3.2. Preprocessing. Following data collection, data preprocessing was conducted in MATLAB using the EEGLAB toolbox [44]. Six EEG channels (F3, F4, C3, C4, O1, O2), two EMG channels (EMG1, EMG2), and two EOG channels (EOG1, EOG2) were selected for analyses. Signals were down-sampled to 250 Hz, and a notch filter (58–62 Hz) was applied to the EEG channels. Bandpass filtering was performed for each signal type: EEG (0.3–35 Hz), EOG (0.3–35 Hz), and EMG (10–100 Hz). All EEG were re-referenced to the contralateral mastoid electrodes (e.g., F4-M1, F3-M2). The two EOG channels were bipolar-referenced and then re-referenced to contralateral mastoid (i.e., EOG1-M2, EOG2-M1). The two EMG channels were bipolar-referenced, resulting in a single channel (EMG1-EMG2) representing the voltage difference.

2.2.4. YASA sleep analyses

2.2.4.1. Sleep staging. We implemented the Yet Another Spindle Algorithm (YASA), a command-line toolbox in Python, for automated sleep staging and analyses [145]. For automated sleep staging, we determined the specific sleep stage for each 30-s epoch of PSG recordings, assigning scores for Wake (W), Stage 1 Sleep (N1), Stage 2 Sleep (N2), Slow Wave Sleep (SWS; also known as Stage 3 Sleep or N3), and Rapid Eye Movement (REM) sleep. YASA's detection required one EEG channel and optionally one EOG and one EMG channel. By default, we selected C4-M1in 86.0 % of the 114 files with overnight EEG recordings. If C4-M1 had poor signal quality, we selected the next best available channel in the following order: C3-M2 (8.8 %), F4-M1 (3.5 %), F3-M2 (0.9 %), and O1-M2 (0.9 %). For the EOG reference channel, EOG-M2 was used in 71.9 % of files, EOG2-M1 in 11.4 %, and no EOG reference was used in 16.7 %. Lastly, 97.4 % of files used the EMG reference channel, while 2.6 % did not.

YASA's automated sleep staging has been validated across PSG data from heterogeneous populations worldwide and shown comparable performance to human scoring accuracy [145]. In our sample, YASA's performance was evaluated by comparing it with manual staging from an experienced sleep scorer (Author K.E.G.S.) for 67 participants. YASA demonstrated good to acceptable performance for both SWS (sensitivity: 78.1 %, specificity: 97.1 %) and REM sleep (sensitivity: 84.5 %, specificity: 97.4 %). For comparison, human interrater agreement among over 2500 experts with three or more years of experience has been reported at 67.4 % for SWS and 90.5 % for REM [129]. Further details are provided in Supplementary Materials (Section I: YASA's Performance).

2.2.4.2. Artifact rejection. After sleep staging, we implemented artifact rejection separately for each sleep stage, as applying a single set of artifact rejection criteria across all sleep stages can lead to the erroneous rejection of epochs that simply reflect characteristics of another sleep stage [145]. We employed a multivariate approach that estimated the variance within each channel and the covariances between all available channels, providing more robustness to signal noise and isolated artifacts in individual channels [6]. Epochs that were three standard deviations away from the expected variance and covariance patterns for a given sleep stage were automatically rejected.

2.2.4.3. Spectral analyses. With artifact-free data, we employed the Welch's sliding periodogram [152] to calculate power spectral density with a 4-second sliding window and 50 % overlap. To mitigate the influence of signal noise, we used the median approach, rather than the traditional mean, for measuring the average of all resulting sliding windows [80]. Absolute power in specific frequency bands was then calculated by the area under the curve of power spectral density,

separately for each EEG channel during REM and SWS stages. To account for between-subject variability in signal amplitude caused by factors such as scalp shape or cap positioning, absolute power within each frequency band was divided by the sum of absolute power across all frequency bands. This normalization produced relative spectral power values for the following frequency bands: delta [0.5–4 Hz], theta [4–8 Hz], alpha [8–12 Hz], sigma [12–16 Hz], beta [16–30 Hz], and gamma [30–40 Hz].

We visually inspected each EEG channel to assess data quality and exclude channels with pervasive noise or flat signals. Among the 108 participants included in SWS analyses and 102 in REM analyses, the number of exclusions per channel were as follows: F3-M2 (4 for SWS, 5 for REM); F4-M1 (4 for SWS, 8 for REM); C3-M2 (6 for SWS, 9 for REM); C4-M1 (7 for SWS, 12 for REM); O1-M2 (6 for SWS, 8 for REM); O2-M1 (8 for SWS, 14 for REM). After excluding any channels with poor signal quality, we averaged relative spectral power across available channels. Most participants had at least one usable channel in each of the frontal, central, and occipital regions (SWS: $n = 107$; REM: $n = 100$). Fig. 3 shows the average relative spectral bandpower across all available channels separately for REM and SWS stages in our sample. This decision was made because the interaction between spatial locations and frequency bands was beyond the scope of the current study, and associations between spectral bandpower and key study variables showed consistent patterns across frontal, central and occipital regions (see Supplementary Materials, Section II: Spatial Location Analysis).

2.3. Procedure

Participants signed up for a study titled "Emotional reactivity at different times of day", which was part of a larger project comparing memory performance following post-encoding sleep vs. wake. Eligible participants were consented and randomly assigned to one of the two 12-hour delay conditions between the encoding and recognition phases of the Emotional Memory Trade-Off (ETO) Task: nighttime sleep (n_{sleep}=60) or daytime wake (n_{wake}=60). We then intentionally oversampled the nighttime sleep condition by recruiting an additional 29 participants (n_{sleep}=89, n_{wake}=60) to ensure at least 60 high-quality recordings for the larger project. However, because the present spectral bandpower analyses excluded individual bad channels rather than entire participants, we ultimately retained 29 more nighttime sleep participants than planned in the larger project. For the current analyses, data were collapsed across the nighttime sleep and daytime wake conditions to maximize statistical power for examining spectral bandpower as a trait-like marker of cortical hyperarousal during sleep, rather than to investigate sleep's mechanistic role in memory processing.

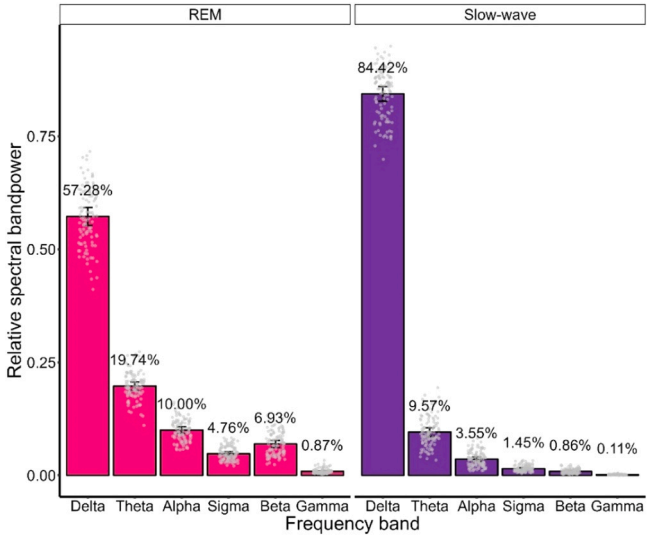

Fig. 3. *The Average Relative Spectral Bandpower During REM (N = 102) and SWS (N = 108), Note.* Error bars represent ± 3 standard errors of the average relative spectral bandpower. Frequency bands are defined as follows: delta [0.5–4 Hz], theta [4–8 Hz], alpha [8–12 Hz], sigma [12–16 Hz], beta [16–30 Hz], and gamma [30–40 Hz].

In the nighttime sleep condition, participants completed the incidental ETO encoding task at around 8:30 PM in the evening, had approximately 8.5 h of sleep opportunity in the lab ($M_{TimeInBed}$=8.52, $SD_{TimeInBed}$=0.54) between 10:30 PM ($M_{BedTime}$=22:53:33, $SD_{BedTime}$=1:05:35) and 7 AM ($M_{BedTime}$=7:25:19, $SD_{BedTime}$=0:52:00), and a surprise ETO recognition task at 8:30 AM the following morning. In comparison, participants in the daytime wake condition completed ETO encoding at around 8:30 AM, left the lab to pursue their regular daily activities with the exception of napping, and returned to the lab again for ETO recognition around 8:30 PM. Following recognition, participants in the daytime wake condition also had the option to sleep in the laboratory overnight ($n = 41$), with also approximately 8.5 h of sleep opportunity ($M_{TimeInBed}$=8.32, $SD_{TimeInBed}$=0.55) between 10:30 PM ($M_{BedTime}$= 22:47:04, $SD_{BedTime}$= 0:51:47) and 7 AM ($M_{BedTime}$=7:07:22, $SD_{BedTime}$=0:41:36). In both conditions, after participants were woken up, the EEG cap and sensors were removed. To reduce potential sleep inertia, they were given time to shower, have a snack, or drink coffee.

Fig. 4 illustrates the typical timeline and procedures for both conditions. Based on data quality screening of all participants who slept in the lab (N = 130), 102 were included in the REM sleep analyses and 108 in the SWS analyses (see Fig. 1 for more detailed information).

2.4. Analyses

Mediation, or indirect *ab* effects, typically have smaller effect sizes because they are estimated by the product of the *a* and *b* paths, and thus require larger sample sizes to achieve adequate statistical power [62, 133]. Therefore, our analyses were collapsed across the nighttime sleep and daytime wake conditions to maximize statistical power, for several reasons. First, most key study variables did not vary significantly between the two delay conditions (Table S2-S5). Second, the bivariate correlations of key study variables showed consistent patterns across conditions (Figure S6-S11). Third, we tested for scalar invariances in our Structural Equation Models by constraining the intercepts to be equal across the daytime wake and nighttime sleep conditions. Scalar invariance was established across all models as constraining intercepts did not result in significantly worse model fits (ps>.8, Table S19). See Supplementary Materials (Section III: Results by Delay Conditions) for full results.

All data analyses were conducted in R [122]. See Supplementary Materials (Section IV: Descriptive Statistics) for means, standard deviations, age correlations, and sex comparisons of key study variables. Individual data points that were more than three standard deviations away from the mean were considered outliers and excluded pairwise from analyses. Analyses that include the outliers are largely consistent with those excluding them and are reported in the Supplementary Materials (Section V: Outlier Analysis). The threshold for statistical significance is set to $p < .05$, two-tailed. The two-stage sharpened method [10] was used to control the false discovery rate (FDR) in mediation and path analyses across all direct, indirect, and total effects of Structural Equation Modeling (SEM). All SEMs were fitted using bootstrap-based standard errors with 1000 replications for robustness against non-normality.

2.4.1. Variable reduction

We conducted bivariate correlations among variables of interest to identify the most relevant ones for inclusion in subsequent SEM analyses. First, to identify memory features that could characterize internalizing symptoms, we tested the correlations between the five dimensions of internalizing symptoms (mixed general distress,

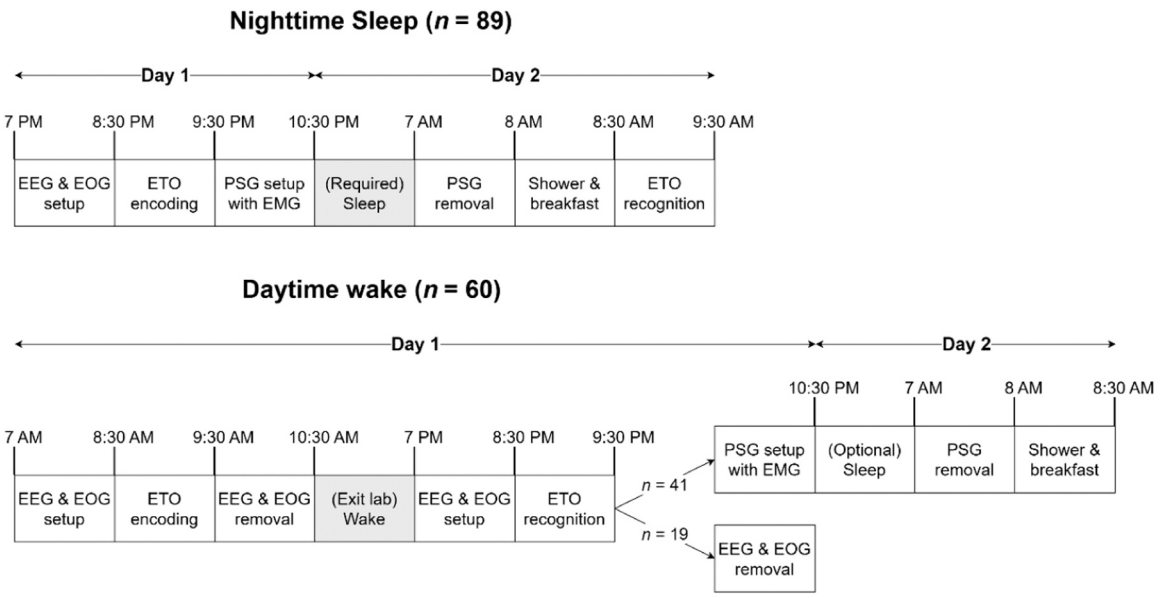

Fig. 4. *Typical Timeline and Study Procedures for Nighttime Sleep and Daytime Wake Conditions, Note.* As an example of the typical timeline and study procedure, participants completed EEG and EOG setup (7:00 PM for the nighttime sleep condition; 7:00 AM for the daytime wake condition) while filling out questionnaires assessing mental health and well-being (BDI, BAI, STAI; [7,8,139]) during setup. They then completed the ETO encoding task (8:30 PM for the nighttime sleep condition; 8:30 AM for the daytime wake condition). After encoding, the procedures differed between conditions. In the nighttime sleep condition, all 89 participants underwent full PSG setup with EMG, turned off all distractions (e.g., phones, reading materials), and attempted to sleep after lights out at approximately 10:30 PM. The following morning, they were awakened around 7:00 AM, given time to shower, eat breakfast, and drink coffee, and then completed ETO recognition at around 8:30 AM. In the daytime wake condition, all 60 participants had their cap and sensors removed after encoding and left the lab to pursue normal daily activities (no napping). They returned at 7:00 PM for EEG/EOG setup and completed ETO recognition at 8:30 PM. Following recognition, 19 participants left the lab, and 41 completed an optional overnight PSG. Session times varied slightly to accommodate participants' schedules, with pre-sleep and post-sleep sessions generally occurring between 6 and 12 AM or PM. PSG, Polysomnography. EEG, electroencephalography. EOG, electrooculography. EMG, electromyography. ETO, emotional memory trade-off.

depressive general distress, anxious general distress, anhedonia, and anxious arousal) and indicators of key memory features. These features included negative memory bias (hits for negative objects, hit difference, and hit trade-off), negative false memory (false alarms for negative objects, false alarm difference, and false alarm trade-off), and overall memory deficits (average corrected recognition, average hits, and average false alarms). Memory features that were significantly correlated (based on original, uncorrected *p*-values) with at least one dimension of internalizing symptoms were selected for further analyses.

In the next step, we assessed the correlations between the selected memory features and spectral bandpower during REM and SWS. We did not examine bivariate correlations between sleep and internalizing symptoms, as the current study focused on testing the indirect associations between sleep and internalizing symptoms via the mediating role of memory. Only memory features that exhibited significant correlations with both sleep microarchitectural features and internalizing symptoms were included as mediators, with the corresponding sleep feature as the predictor and internalizing symptom as the outcome, in subsequent SEM analyses.

2.4.2. Mediation models

Based on the outcomes of the variable selection process, we performed a series of SEM mediation analyses to examine the indirect effects (*ab*) of sleep on internalizing symptoms mediated by memory features. Each model assessed the mediating role of a single memory feature on a single dimension of internalizing symptom, but multiple sleep predictors might be included if they were significantly correlated with the same memory mediator. For models with more than one sleep predictor, we compared a constrained model, which assumed equal effects on the memory mediator (*a*) and equal indirect effects on the internalizing symptom mediated by memory (*ab*), to a unconstrained model, which allowed *a* and *ab* to freely vary for each sleep predictor. Because the unconstrained model estimated all possible parameters, it was saturated with zero degrees of freedom, providing a perfect fit to the data. We freed the parameters if constraining them resulted in significantly worse model fits based on chi-square (χ^2) difference tests.

Each model was also conducted with age and sex included as a covariate for memory and internalizing symptoms to control for their potential effects on memory and emotional processing [67,106,110]. To account for the bidirectional relationship between sleep and internalizing symptoms [1,107], each model was also tested with internalizing symptoms as the predictor and sleep as the outcome. Our manuscript focused on models examining sleep as a predictor for internalizing symptoms, as these effects were generally larger compared to models where internalizing symptoms predicted sleep (Table S29-S32).

3. Results

3.1. Bivariate correlations

3.1.1. Memory and internalizing symptoms

Fig. 5 presents a heatmap showing the correlations between dimensions of internalizing symptoms and indicators of negative memory bias, negative false memory, and overall memory deficits. Notably, mixed general distress was associated with indicators of negative false memory and overall memory deficits, as shown by higher false alarms for negative objects ($r = 0.21$, $p = .013$) and lower corrected recognition scores averaged across all scene components ($r = -0.19$, $p = .018$). Additionally, anhedonia was associated with a reduced negative memory bias, indicated by a smaller difference in hit rates between negative objects and neutral objects ($r = -0.19$, $p = .022$). This was driven by higher hit rates for neutral objects ($r = 0.20$, $p = .013$), rather than lower hits for negative objects ($r = 0.09$, $p = .297$). Finally, anxious arousal was correlated with lower corrected recognition for negative

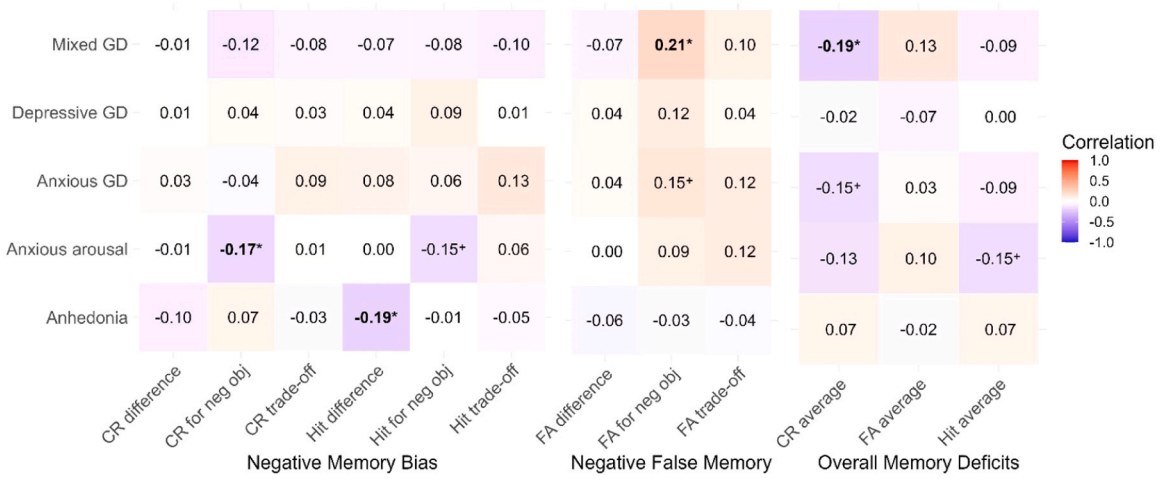

Fig. 5. *The Correlation Heatmap Between Internalizing Symptoms and Key Memory Features (N = 149), Note.* The depth of the color reflects the magnitude of the correlation. Warmer colors indicate stronger positive correlations, while cooler colors indicate stronger negative correlations. Levels of significance are denoted as: +, $p < .100$, *, $p < .050$. GD, general distress. CR, corrected recognition. FA, false alarms. Neg obj, negative objects. Difference, comparing memory for negative objects with neutral objects on separate scenes. Trade-off, comparing memory for negative objects with neutral backgrounds on same scenes. Average, memory scores averaged across negative objects and paired backgrounds, as well as neutral objects and paired backgrounds. Mixed general distress, a component of negative affect equally associated with depression and anxiety, represents shared symptoms of depression and anxiety. Depressive general distress, a component of negative affect more associated with depression than anxiety, represents non-specific symptoms of depression. Anxious general distress, a component of negative affect more associated with anxiety than depression, represents non-specific symptoms of anxiety. Anhedonia, low positive affect only associated with depression, represents specific symptoms of depression. Anxious arousal, physiological hyperactivation only associated with anxiety, represents specific symptoms of anxiety.

objects (r = -0.17, p = .049). However, this likely reflected a general tendency towards overall memory deficits instead of a specific reduction in negative memory bias, as anxious arousal was also numerically correlated with lower hit rates on average (r = -0.15, p = .076).

Based on bivariate correlations between memory and internalizing symptoms, we selected the following memory parameters to test for correlations with spectral bandpower during REM sleep and SWS: 1) negative memory bias, measured by the difference in hit rates between negative and neutral objects, 2) negative false memory, measured by false alarms for negative objects, and 3) overall memory performance, measured by average corrected recognition across all scene components. See Fig. 6 for a visualization of the memory features associated with internalizing symptoms.

3.1.2. Memory and sleep

Fig. 7 presents a heatmap illustrating the correlations between spectral power within specific frequency bands during REM and SWS and key memory features linked to internalizing symptoms. No significant correlations were observed between spectral bandpower within any frequency bands and negative memory bias, as measured by the difference in hit rates between negative and neutral objects (*ps*>.8). For negative false memory, higher beta (r = 0.23, p = .024) and gamma spectral power (r = 0.22, p = .032) during REM sleep were correlated with increased false alarm rates for negative objects. For overall memory performance, increased beta (r = -0.33, $p < .001$) and gamma spectral power (r = -0.23, p = .021) during SWS were correlated with poorer corrected recognition averaged across all scene components.

See Fig. 8 for a visualization of the associations between memory features that characterized mixed general distress and beta and gamma power during REM sleep and SWS. Based on these bivariate correlations, we tested two mediating effects: 1) false alarms for negative objects as a mediator of the associations between beta and gamma spectral power during REM sleep and mixed general distress, and 2) corrected recognition average across all scene components as a mediator of the associations between beta and gamma spectral power during SWS and mixed general distress.

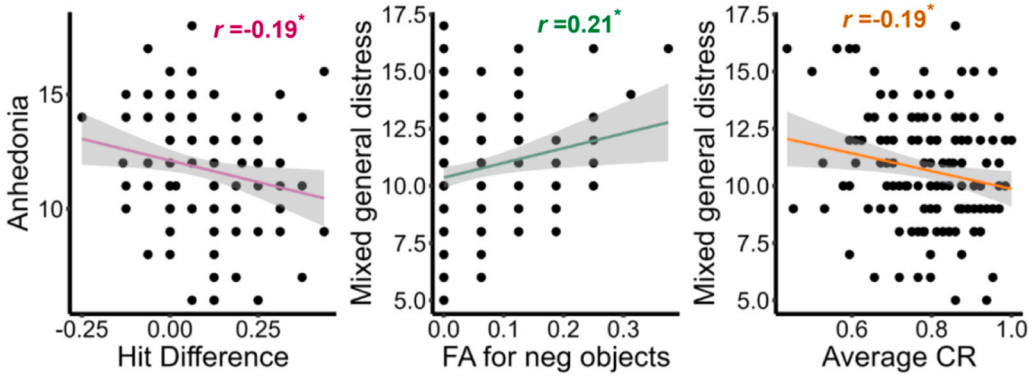

Fig. 6. *Correlation Scatterplots Between Internalizing Symptoms and Key Memory Features (N = 149), Note.* Hit Difference, the extent to which negative objects had higher hits compared to neutral objects in separate scenes. FA for neg objects, false alarms for negative objects. Average CR, corrected recognition averaged across all scene components. Anhedonia, low positive affect only associated with depression, represents specific symptoms of depression. Mixed general distress, a component of negative affect equally associated with depression and anxiety, represents shared symptoms of depression and anxiety. *, $p < .050$.

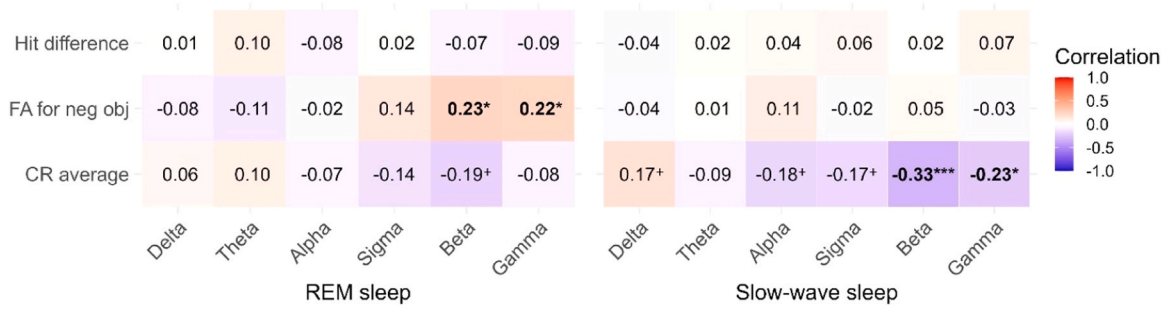

Fig. 7. *Correlations Between Memory Features That Characterize Internalizing Symptoms and Spectral Bandpower During REM (N = 102) and SWS (N = 108). Note.* The depth of the color reflects the magnitude of the correlation in the total sample ($N = 102$ for REM sleep, $N = 108$ for slow-wave sleep). Warmer colors indicate stronger positive correlations, while cooler colors indicate stronger negative correlations. Levels of significance are denoted as: ⁺, $p < .100$. *, $p < .050$. ***, $p < .001$. Hit difference, comparing hit rates for negative objects with neutral objects on separate scenes. FA for neg obj, false alarms for negative objects. CR average, corrected recognition scores averaged across negative objects and paired backgrounds, as well as neutral objects and paired backgrounds. REM, rapid eye movement. SWS, slow-wave sleep. Delta, 0.5–4 Hz. Theta, 4–8 Hz. Alpha, 8–12 Hz. Sigma, 12–16 Hz. Beta, 16–30 Hz. Gamma, 30–40 Hz.

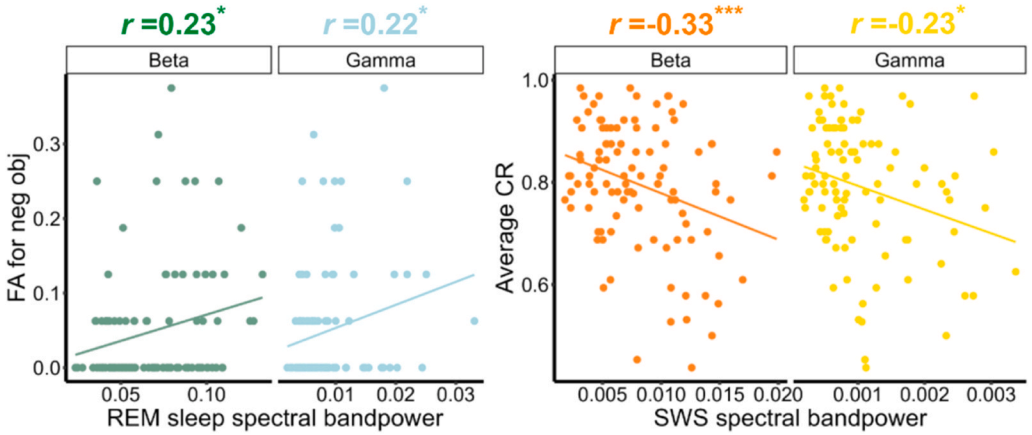

Fig. 8. *Scatterplots for the Correlations Between Beta and Gamma Spectral Bandpower During REM (N = 102) and SWS (N = 108) with Negative False Memory and Overall Memory Accuracy. Note.* FA for neg obj, false alarms for negative objects. Average CR, corrected recognition averaged across negative objects and paired backgrounds, as well as neutral objects and paired backgrounds. REM, rapid eye movement. SWS, slow-wave sleep. Beta, 16–30 Hz. Gamma, 30–40 Hz. *, $p < .050$. ***, $p < .001$.

3.2. SEM mediations

3.2.1. Negative false memory

First, we ran a mediation analysis to examine whether beta and gamma spectral power during REM sleep were linked to mixed general distress indirectly via false alarms for negative objects. The constrained model was retained, because constraining the indirect effects of beta and gamma power to be equal did not hurt model fit ($\Delta\chi^2(1) = 0.00$ $p = .983$; Table S20). Adjusted *p*-values were reported after applying FDR correction across eight effects.

Results indicated that elevated beta (a=0.16, $p = .004$) and gamma spectral power (a=0.12, $p = .004$)[3] during REM sleep were significantly associated with higher false alarm rates for negative objects, which, in turn, were associated with more severe mixed general distress (b=0.25, $p = .013$). Thus, false alarms for negative objects significantly mediated the effects of elevated power in beta (ab=0.04, $p = .041$) and gamma bands (ab=0.04, $p = .041$) during REM sleep, on mixed general distress (Fig. 9). Interestingly, only beta power (c'=0.34, $p = .004$), but not gamma power (c'=-0.16, $p = .063$), was directly linked to higher levels of mixed general distress. When age and sex were included as covariates for mixed general distress and false alarms for negative objects, the same patterns were observed. However, all of these effects were no longer significant ($ps > .2$), which could be related to the association between older age and higher mixed general distress (β=0.22, $p = .057$). See Table S21 & **S22** for full model results.

A post-hoc power analysis was assessed using Monte Carlo simulations with 1000 iterations and a sample size of 98. The estimated statistical power (i.e., the percentage of significant coefficients) for the equally constrained beta and gamma spectral power during REM sleep was 66.1 % for a= 0.16, 78.7 % for b= 0.25, and 29.4 % for ab= 0.04 (Table S34).

3.2.2. Overall memory deficits

Next, a mediation analysis was conducted to examine whether beta and gamma spectral power during SWS predicted mixed general distress indirectly via average corrected recognition. The indirect effects were allowed to vary between beta and gamma bands, as constraining them to be equal significant hurt model fit ($\Delta\chi^2((1) = 4.23$, $p = .040$; Table S20). Adjusted *p*-values were reported after applying FDR correction across nine effects.

For beta activity during SWS, elevated spectral power was linked to poorer average corrected recognition (a=-0.45, $p = .015$), which, in turn, was linked to higher levels of mixed general distress internalizing symptoms (b=-0.27, $p = .020$). Consistent with these results, poorer average corrected recognition mediated the relation between beta spectral power during SWS and the severity of internalizing symptoms, although not significantly after adjusting for multiple comparisons

[3] Although the effects beta and gamma power on false alarms for negative objects were constrained to be equal, their standardized estimates differed slightly due to differences in the variances of the outcome variables.

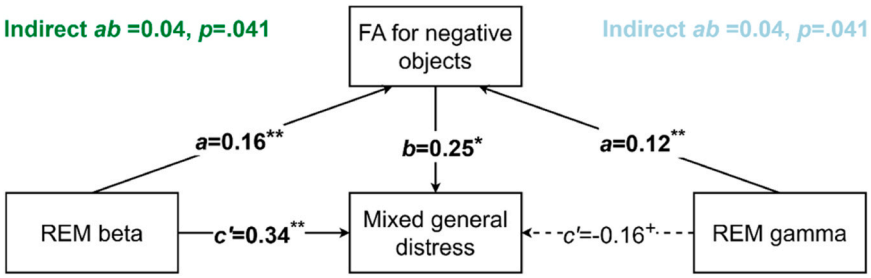

Fig. 9. *Standardized Results of Beta and Gamma Spectral Power during REM Sleep Predicting Mixed General Distress Via False Alarms of Negative Objects (N = 98). Note*. Of the 102 participants included in REM analyses, one was identified as an outlier (>3 standard deviations from the mean) on beta power, two were outliers on FA for negative objects, and additional one was an outlier on both gamma power and FA for negative objects, resulting in a final sample of 98 participants in all paths. REM, rapid eye movement sleep. Beta, 16–30 Hz. Gamma, 30–40 Hz. FA, false alarms. Mixed general distress, a component of negative affect equally associated with depression and anxiety, representing shared symptoms of depression and anxiety. a, direct effects of sleep on memory. b, direct effects of memory on internalizing symptoms. c', direct effects of sleep on internalizing symptoms. ab, indirect effects of sleep on internalizing symptoms mediated by memory. Levels of significance after controlling for false discovery rates are denoted as: $^{+}$, p < .100, *, $p < .050$, **, p < .010.

(*ab*=0.12, $p = .070$; Figure 10). However, the association between higher beta spectral power and poorer average corrected recognition (*a*=-0.41, $p = .072$), or between poorer corrected recognition and greater severity of internalizing symptoms (*b*=-0.26, $p = .055$), was no longer significant after controlling for the effect of age and sex on mixed general distress and average corrected recognition.

In contrast, gamma spectral power during SWS was not indirectly associated with mixed general distress via average corrected recognition (*ab*=-0.04, $p = .280$), as gamma power was not significantly related to average corrected recognition (*a*=0.16, $p = .280$). Neither beta (*c'*=0.20, $p = .197$) or gamma spectral power (*c'*=-0.23, $p = .092$) was directly associated with the severity of internalizing symptoms (Figure 10). None of these effects were significant after controlling for age and sex either (*ps*>.5), which could be related to the association between older age and higher mixed general distress (β=0.28, $p = .055$). See full model results in Table S23 & **S24**.

A post-hoc power analysis was assessed using Monte Carlo simulations with 1000 iterations and a sample size of 104. The estimated statistical power for beta spectral power during SWS was 100 % for *a*= -0.45, 28.4 % for *b*= -0.27, and 25.2 % for *ab*= 0.12. Power for SWS gamma power was 61.9 % for *a*= 0.16, 28.4 % for *b*= -0.27, and 5.9 % for *ab*= -0.04 (Table S35).

4. Discussion

The goal of the current study was to examine how memory deficits may link microarchitectural features of REM and SWS to internalizing symptoms across common, depression-related, and anxiety-related dimensions. Despite the substantial comorbidity and overlapping genetic factors between depression and anxiety, previous research has often focused on effects unique to depression or anxiety [25,85,86,102]. Our findings suggest that, rather than depression-specific or anxiety-specific symptoms, sleep is associated with internalizing symptoms common to both depression and anxiety, through its relation to memory disruption. These common internalizing symptoms are reflected in mixed general distress, representing broad negative affect equally associated with depression and anxiety.

Extending prior work that emphasized the restorative or protective functions of lower-frequency activity during sleep [12,128,47–50,79], the current study identifies beta and gamma spectral power as relatively stable indicators of cortical hyperarousal during REM and SWS. Specifically, false memory for negative information, or misremembering negative events that did not actually happen, is a potential mechanism through which higher beta and gamma spectral power during REM sleep are associated with mixed general distress. In addition, elevated beta spectral power during SWS is indirectly associated with more severe mixed general distress via its link to overall memory deficits.

4.1. Negative false memory

Building upon prior work showing that individuals with elevated depression or anxiety symptoms tend to remember negative events differently from how they actually happened [82,101,127,143,144], the current study highlighted that increased negative false memory, or incorrectly identifying a novel negative object as previously encountered, was uniquely associated with mixed general distress, a dimension of internalizing symptoms thought to be equally relevant to both depression and anxiety [35,151]. These findings suggest that negative

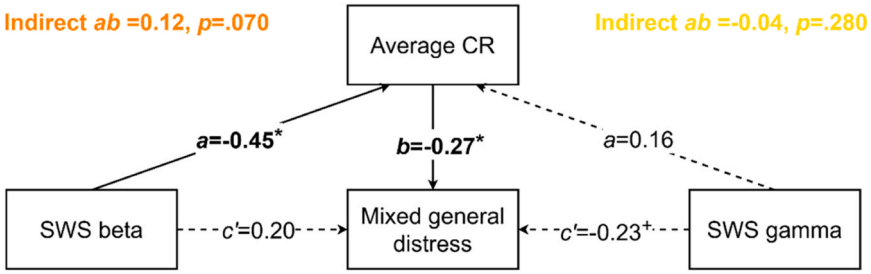

Figure 10. *Standardized Results of Beta and Gamma Spectral Power during SWS Predicting Mixed General Distress Via Average Corrected Recognition (N = 104). Note*. Of the 108 participants included in SWS analyses, one was identified as an outlier (>3 standard deviations from the mean) on beta power, two were outliers on gamma power, and additional one was an outlier on both gamma power and CR average, resulting in a final sample of 104 participants in all paths. SWS, slow-wave sleep. Beta, 16–30 Hz. Gamma, 30–40 Hz. CR, corrected recognition. Mixed general distress, a component of negative affect equally associated with depression and anxiety, representing shared symptoms of depression and anxiety. a, direct effects of sleep on memory. b, direct effects of memory on internalizing symptoms. c', direct effects of sleep on internalizing symptoms. ab, indirect effects of sleep on internalizing symptoms mediated by memory. Levels of significance after controlling for false discovery rates are denoted as:: $^{+}$, p < .100, *, $p < .050$.

false memory may play a critical role in the comorbidity between depression and anxiety.

This association may be partly explained by negative overgeneralization, a common cognitive distortion in both depression and anxiety, characterized by drawing broad conclusions from isolated negative incidents [101,123]. Negative overgeneralization can increase the accessibility of negative false information within the memory system [101,127]. For instance, witnessing a plane crash on the news might provoke an intense and generalized fear of flying, and this fear can contribute to the recall of exaggerated or inaccurate details of the plane crash that were not originally presented in the news report or inaccurate recall for the frequency at which similar news reports occur.

While negative false memory may partly reflect the formation of a distorted memory representation, it could also stem from heightened familiarity and a liberal response bias to negative information [32]. Specifically, emotionally charged negative stimuli often evoke a stronger subjective sense of familiarity among individuals with higher depression symptoms [32]. This effect may be driven by increased connectivity between the amygdala and sensory regions at retrieval, which enhances the perceptual vividness and emotional salience of negative information [18]. Combined with greater impulsivity and reduced cognitive control, individuals with higher internalizing symptoms may reply on automatic emotional response that misattributes vividness and salience as familiarity, rather than engaging in deliberate evaluation of memory traces [31].

REM sleep has been proposed to strengthen the associative networks of emotionally negative information, such that encountering one negative experience can activate related ones, making negative false information more accessible within the memory system [26,30,104]. This process may be linked to selective synaptic downscaling, a hallmark of REM sleep, during which emotionally salient synaptic connections are strengthened while less relevant ones are weakened [92,121]. By reinforcing emotionally charged memories, this process makes these memories more robust within the semantic network and simultaneously facilitates the spreading activation of related negative concepts, even if they are not part of the original encoding experience [26,30,104].

Additionally, while REM sleep is generally associated with cortical inhibition, it also involves increased functional connectivity among the hippocampus, amygdala, and medial temporal regions [12,55,92]. This increased information flow within the emotional memory circuit likely amplifies the spreading activation of negative false memories [101,127]. Consistent with these theoretical frameworks, we observed that elevated beta and gamma spectral power during REM sleep may reflect trait-like markers of cortical hyperarousal [22,149], which are associated with increased negative false memory.

While there is prior evidence demonstrating associations between REM sleep microarchitectural features and negative false memory [26,30,104], as well as between negative false memory and internalizing symptoms [82,101,127,143,144], our findings provide the first evidence that negative false memory mediates the relationship between elevated beta and gamma spectral power during REM sleep and the severity of mixed general distress. These results suggest that individuals who show consistent patterns of heightened cortical activity during REM sleep may be more susceptible to forming negative false memories, and these inaccurate memory representations of negative experiences–perhaps reflecting early signs of negative memory generalization–may be associated with comorbid depression and anxiety symptoms.

The results of these mediation effects are in line with a previous study showing that elevated beta power during REM sleep is linked to difficulties in recalling extinguished fearful memories, a crucial memory process for reducing PTSD symptoms [46]. In addition to its indirect effect through negative false memory, beta spectral power during REM sleep was also found to have a direct association with more severe mixed general distress. This extends prior research linking elevated beta spectral power to various clinical conditions, including PTSD, depression, and insomnia [46,112,125,132]. Together, these results highlight the role of heightened cortical hyperarousal during REM sleep in the persistence of distress across internalizing disorders, possibly occurring with or without the formation of negative false memories.

4.2. Overall memory deficits

We found that overall memory deficits, reflected by poorer corrected recognition averaged across all scene components, were associated with mixed general distress. This suggests that impaired general memory function may play a critical role in the comorbidity between depression and anxiety. Our findings support the idea that memory deficits observed in past studies of depression may, in part, reflect the high comorbidity between depression and anxiety [54,71,78,90,109]. These memory impairments could stem from deficits in cognitive control allocation due to reduced motivation and active engagement, as well as deficits in pattern separation abilities due to hippocampal atrophy [53,70,137].

Consistent with prior research highlighting the role of SWS for memory performance regardless of emotional valence [16,17,66,92], we found that individuals whose SWS was characterized by reduced spectral power in beta and gamma bands tend to exhibit better overall memory accuracy. This lower activity in higher-frequency beta and gamma bands is indicative of stable, trait-level reductions in cortical hyperarousal during SWS. Therefore, our findings further support the hallmark minimal cholinergic activity in hippocampal and neocortical regions during SWS, creating an optimal environment for memory processing [63,115].

Our findings provide novel evidence that (descriptively speaking) overall memory deficits mediated the relation between increased beta spectral power during SWS and elevated mixed general distress. Individuals with heightened cortical hyperarousal during SWS tend to exhibit impaired memory performance for both negative and neutral information, and these memory impairments could be linked to comorbid depression and anxiety symptoms. However, memory deficits did not mediate the association between gamma power and mixed general distress. This is potentially because gamma activity, compared to beta activity, may be less effective at promoting synchronized activity across distant brain structures involved in memory processing [22,93].

4.3. Effects of age

Importantly, when controlling for age and sex in our models, the same patterns of mediations were observed, although effects were no longer statistically significant. While no sex-related effects were found, older age was modestly associated with greater mixed general distress, which may have contributed to the attenuation of other effects. This contrasts with prior work on improved mental well-being with increasing age [5,77]. The discrepancy may arise because mixed general distress reflects broader internalizing symptoms equally relevant to both depression and anxiety, capturing aspects not assessed by depression- or anxiety-specific scales [35]. Alternatively, our sample included individuals aged 18–59, a period in which age-related improvements in mental well-being may not yet have emerged, while health decline and reduced social support may begin to appear [69].

In addition, older age is associated with increased EEG activity in higher frequency bands across all sleep stages, but particularly during SWS, due to more frequent awakenings and weakened endogenous circadian control [56,157]. Aging is also linked to memory disruption due to structural and functional decline of the hippocampal system [38]. Despite age's known effects on sleep and memory, the associations between REM beta/gamma power and negative false memory, SWS beta power and overall memory impairments, and these memory features with mixed general distress, remained moderately sized. Overall, these findings suggest that the contribution of memory disruption to the relationship between cortical hyperarousal during sleep and shared internalizing symptoms is relatively robust across adulthood, from

young to middle age. These findings suggest that targeting memory-related impairments could be an effective strategy for reducing internalizing symptoms in individuals with disrupted sleep.

4.4. Negative memory bias

We found that depression-specific anhedonia symptoms were associated with a reduced negativity bias, indicated by a smaller difference in hit rates between negative and neutral objects. This finding contrasts with prior research, which has reported either a positive association between depression and increased negative memory bias [36,40,60,65,71,76,97,103], or no significant relations between depression and negative memory bias [58,78,90,94]. One possible explanation for this unexpected finding is that the negative stimuli used in our study (e.g., snake, blood, vomit) were intended to be universally aversive and might not have been mood-congruent with the specific negative affect experienced by individuals with depression. However, it is important to note that this finding is based on simple bivariate correlations rather than more robust SEM testing and should be interpreted with caution.

Our findings revealed a non-significant correlation between theta spectral power during REM sleep and negative memory bias. This is inconsistent with previous studies showing the involvement of theta activity in emotional memory processing [12,79,105,138]. It is possible that the impact of theta activity on emotional memory processing during REM sleep is modulated by the interplay of neurotransmitters, such as acetylcholine and norepinephrine. On one hand, increased acetylcholine levels may suppress the hippocampal feedback to the neocortex, potentially promoting the synaptic pruning of no-longer-relevant information [79,92]. On the other hand, reduced norepinephrine levels may facilitate information flow within the neocortex, which further integrates previously consolidated memory traces during SWS [12,79]. However, in the absence of direct neurotransmitter measurements, it is possible that the effects of theta activity alone were not prominent enough to drive the selective processing of emotionally negative memories.

4.5. Limitations and future directions

The current study has several limitations. First, our sample primarily consisted of White participants from the South Bend community in Indiana, which limits the generalizability of our findings to young and middle-aged adults from more diverse racial and ethnic backgrounds. Relatedly, our sample reported a restricted range of mild to moderate internalizing symptoms (see Table S3). This is particularly relevant as increased measures of REM sleep, decreased measures of SWS, and enhanced negativity bias are well-established clinical markers of internalizing symptoms [9,53,116,126]. Consequently, studying primarily healthy adults from a community sample may have constrained our ability to detect more pronounced effects in the relationships among sleep, memory, and internalizing symptoms that may be apparent in more clinically severe psychiatric patients.

Second, the current study did not differentiate between phasic (i.e., active and sympathetically driven) and tonic (i.e., quiet and parasympathetically driven) periods of REM sleep [134]. Prior research suggests that tonic REM sleep may be more relevant for overall memory accuracy due to reduced gamma activity, whereas phasic REM sleep may be more involved in emotional memory processing due to increased theta activity [4,28,79]. Future research should therefore examine the specific contributions of tonic and phasic periods of REM sleep to better understand their differential roles in memory and internalizing symptoms. Third, participants in our study only completed one night of laboratory-monitored polysomnography without an adaptation night. First-night sleep in a laboratory setting is often shallower and more fragmented [99]. Therefore, although spectral bandpower generally shows greater within-individual stability than other sleep metrics [99,114], the increased beta and gamma activity we observed could partially reflect adjustment to the novel sleep environment, rather than trait-like cortical hyperarousal.

Next, memory performance was assessed only once 12 h following encoding. Although single testing avoids potential contamination from repeated retrieval, it limits our ability to evaluate the initial strength of memory at encoding, before consolidation or reconsolidation processes occur [98]. Therefore, we cannot rule out the possibility that individual differences in encoding contributed to the memory performance observed after the 12-hour retention interval. Future studies should consider including both immediate and delayed memory tests to provide a more complete characterization of memory changes over time.

Finally, given the exploratory nature of the study, we conducted mediation analyses on only a small subset of sleep, memory, and internalizing symptom variables that showed statistically significant bivariate correlations. Therefore, the findings should be interpreted with caution and will require replication in future pre-registered studies with *a priori* hypotheses [95].

4.5.1. Collapsing analyses across delay conditions

Importantly, we collapsed analyses across the nighttime sleep and daytime wake conditions to maximize statistical power in our mediation models that typically require large sample sizes to detect true population effects. Even after combining the delay conditions, statistical power for detecting the mediating effect of memory remained below the conventional threshold (i.e.,.80). Insufficient power can lead to both overestimation of effect sizes (false positives) and reduced reproducibility (false negatives; [24]). This highlights the importance of maximizing sample size, particularly in neuroscience research that often suffers from low statistical power [24], though collapsing across conditions may come with other drawbacks and potential confounds.

For one, nighttime sleep participants slept in the lab between encoding and recognition, whereas daytime wake participants remained awake after encoding and only slept following recognition. Descriptively, both negative false memory and overall memory impairments were lower in the nighttime sleep condition compared to daytime wake (Table S2), likely because post-encoding sleep provides both passive protection against daytime interference and active memory consolidation [21,92]. We collapsed sleep data from the two delay conditions because our focus was on spectral bandpower as a trait-like indicator of sleep physiology, given its high test-retest reliability [23,43,114]. This approach, however, could introduce a sleep-related confound, such that any observed differences in sleep and memory might reflect the timing of sleep rather than the trait-like hyperarousal during sleep.

Relatedly, collapsing across conditions may conflate time-of-day effects: participants in the sleep condition completed encoding in the evening, when accumulated fatigue and reduced alertness may have impaired performance, whereas wake participants encoded in the morning, when they were likely more rested and alert [153,155]. Although a prior study using the same Emotional Memory Trade-Off task found no time-of-day differences on immediate memory performance [119], future studies should better control the timing of encoding and recognition sessions to disentangle sleep-related effects from circadian influences.

In addition, it is important to note that temporal precedence is a key assumption of mediation analysis, which would require that sleep precede memory performance and memory precede internalizing symptoms [84,141]. This assumption could not be established in current study because both spectral bandpower and internalizing symptoms reflect relatively stable, trait-like characteristics with limited within-person variability over the study period. Nonetheless, prior research suggests that cross-sectional mediation models can still offer empirical and theoretical value when interpreted as *atemporal* associations rather than implying causal directionality [64,113,154]. Accordingly, in the present study, we have kept all interpretations correlational in nature and avoided causal claims.

5. Conclusions

Our findings indicate that individuals with elevated beta and gamma spectral power during REM sleep tend to show higher levels of mixed general distress, which is linked to increased negative false memories. Similarly, higher beta spectral power during SWS is indirectly connected to mixed general distress through overall memory impairment. These results underscore memory disruption as a potential pathway that links trait-level cortical hyperarousal in sleep microarchitecture, especially increased activity in higher frequency bands, to the comorbidity between depression and anxiety.

CRediT authorship contribution statement

Sanders Kristin: Writing – review & editing, Validation, Supervision, Software, Resources, Project administration, Methodology, Investigation, Formal analysis, Data curation, Conceptualization. **Dan Denis:** Writing – review & editing, Validation, Supervision, Software, Resources, Project administration, Methodology, Investigation, Formal analysis, Data curation, Conceptualization. **Xinran Niu:** Writing – review & editing, Writing – original draft, Visualization, Validation, Supervision, Software, Project administration, Methodology, Investigation, Formal analysis, Data curation, Conceptualization. **Kensinger Elizabeth:** Writing – review & editing, Validation, Supervision, Resources, Methodology, Investigation, Funding acquisition, Conceptualization. **Payne Jessica:** Writing – review & editing, Validation, Supervision, Resources, Methodology, Investigation, Funding acquisition, Conceptualization. **Cunningham Tony:** Writing – review & editing, Validation, Supervision, Resources, Methodology. **Guangjian Zhang:** Writing – review & editing, Visualization, Validation, Supervision, Software, Formal analysis.

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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.

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Appendix A. Supporting information

Supplementary data associated with this article can be found in the online version at doi:10.1016/j.bbr.2025.115932.

Data Availability

Preregistration, data files, and R scripts are available at https://doi.org/10.17605/OSF.IO/8YVC5.

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