Stress Alters the Neural Context for Building New Memories

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Abstract

■ Stressful events affect mnemonic processing, in particular for emotionally arousing events. Previous research on the mechanisms underlying stress effects on human memory focused on stress-induced changes in the neural activity elicited by a stimulus. We tested an alternative mechanism and hypothesized that stress may already alter the neural context for successful memory formation, reflected in the neural activity preceding a stimulus. Therefore, 69 participants underwent a stress or control procedure before encoding neutral and negative pictures. During encoding, we recorded high-density EEG and analyzed—based on multivariate searchlight analyses—oscillatory activity and cross-frequency coupling patterns before stimulus onset that were predictive of memory tested 24 hr later. Prestimulus theta predicted subsequent memory in controls but not in stressed participants. Instead, prestimulus gamma predicted successful memory formation after stress, specifically for emotional material. Likewise, stress altered the patterns of prestimulus theta–beta and theta–gamma phase–amplitude coupling predictive of subsequent memory, again depending on the emotionality of the presented material. Our data suggest that stress changes the neural context for building new memories, tuning this neural context specifically to the encoding of emotionally salient events. These findings point to a yet unknown mechanism through which stressful events may change (emotional) memory formation. ■

INTRODUCTION

Stress has a major impact on our memory (Schwabe, Joëls, Roozendaal, Wolf, & Oitzl, 2012; Joëls, Fernandez, & Roozendaal, 2011; Roozendaal, McEwen, & Chattarji, 2009). Through the action of glucocorticoids and catecholamines—both released during stressful episodes—on prefrontal and medial temporal lobe areas, stress may enhance memory formation but impair memory retrieval (Roozendaal & McGaugh, 2011; Buchanan, Tranel, & Adolphs, 2006; Roozendaal, Hahn, Nathan, de Quervain, & McGaugh, 2004; Cahill, Gorski, & Le, 2003; de Quervain, Roozendaal, & McGaugh, 1998; McGaugh, Cahill, & Roozendaal, 1996). Although the direction of the stress effects depends on the affected memory stage, emotionally arousing information appears to be generally more sensitive to both the enhancing and impairing effects of stress than neutral information (Goldfarb, Tompary, Davachi, & Phelps, 2019; Buchanan et al., 2006; Cahill et al., 2003), presumably because of an interaction of glucocorticoid hormones with arousal-related noradrenergic activation (Roozendaal & McGaugh, 2011; Roozendaal, Okuda, de Quervain, & McGaugh, 2006). The preferential processing of emotional information under stress may aid coping with the ongoing stressor and facilitate the preparation for similar future events. On the other hand, such a prioritization may contribute to the aberrant emotional memory that is prominent in several stress-related disorders, including depression, addiction, and posttraumatic stress disorder (de Quervain, Schwabe, & Roozendaal, 2017; Pitman et al., 2012; Hyman, 2005; Dalgleish & Watts, 1990).

Given the far-reaching implications of stress effects on (emotional) memory, a number of studies aimed at elucidating the neural mechanisms underlying stress-induced changes in memory (Schwabe et al., 2012; Roozendaal et al., 2009; Joëls, Pu, Wiegert, Oitzl, & Krugers, 2006). Human studies employed, in particular, fMRI and EEG to examine how (and where) stress changes the neural activity elicited by a stimulus during encoding (Wirz, Wacker, Felten, Reuter, & Schwabe, 2017; Quaedflieg, Schwabe, Meyer, & Smeets, 2013; Schwabe & Wolf, 2012; Weymar, Schwabe, Löw, & Hamm, 2012; Qin, Hermans, van Marle, Luo, & Fernández, 2009). Although these analyses of event-related activity provided valuable insights into the neural underpinnings of stress effects on memory, there is evidence that event-related activity is not the sole determinant of successful memory but that, in particular, memory encoding is also critically influenced by the neural activity preceding a stimulus (Cohen et al., 2015; Sweeney-Reed et al., 2015; Fell et al., 2011; Otten, Quayle, Akram, Ditewig, & Rugg, 2006). For instance, increases in oscillatory theta (4–8 Hz), beta (13–30 Hz), or gamma (>30 Hz) power shortly before stimulus presentation were linked to successful later remembering (Fell et al., 2011; Guderian, Schott, Richardson-Klavehn, & Düzel, 2009). Prestimulus activity may provide a "neural context" for information processing, reflecting the adoption of a specific "task set" (Otten et al., 2006) or readiness to form new memories. Although it has recently been suggested that stress may induce a large-scale network

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reconfiguration leading to the prioritized processing of emotionally salient information (Hermans, Henckens, Joëls, & Fernández, 2014; Hermans et al., 2011), it not been tested yet whether stress may alter the neural context for successful encoding, as reflected in prestimulus activity. If so, this would represent a yet unknown mechanism through which stress changes memory formation.

Therefore, we examined here whether acute stress may modulate the neural context for successful memory formation by altering the stimulus-preceding activity relevant for memory and whether stress may further tune this neural context specifically toward the encoding of emotionally arousing information. To this end, we exposed healthy individuals to a stress or control manipulation before they encoded emotionally neutral and negative pictures. During encoding, we recorded high-density EEG. To investigate stimulus-preceding neural activity related to successful memory formation, we combined a multivariate-pattern-analysis-based searchlight analysis focusing on oscillatory power and cross-frequency coupling before stimulus onset with a subsequent memory analysis separating prestimulus activity patterns associated with subsequently remembered and forgotten items.

METHODS

Participants and Design

Seventy-three healthy volunteers participated in this experiment. Exclusion criteria were checked in a standardized interview before participation and comprised any current or chronic mental or physical disorders, medication intake, smoking, or drug abuse. Furthermore, women taking hormonal contraceptives were excluded from participation. In addition, participants were asked to refrain from food, caffeine, and physical activity for 2 hr before testing. Four participants had to be excluded from analysis because of medication intake shortly before participation, thus leaving a final sample of 69 participants (30 men, 39 women; age: mean = 26.3 years, range = 18–38 years). All participants gave written informed consent before entering the study, which was approved by the local ethics committee. In a between-participant design, participants were randomly assigned to a stress or control group. To control for the diurnal rhythm of cortisol, all testing took place in the morning between 8 a.m. and 12 p.m.

Stress and Control Manipulation

Participants in the stress condition underwent the socially evaluated cold pressor test (SECPT; Schwabe, Haddad, & Schächinger, 2008), a standardized stress protocol known to elicit both subjective and physiological stress responses (Schwabe & Schächinger, 2018). In brief, participants were requested to immerse their left hand, including the wrist, for 3 min into ice water (0–2°C), while being videotaped and evaluated by a rather cold and nonreinforcing experimenter dressed in a white lab coat. In the control condition, participants were asked to immerse their left hand, including the wrist, for 3 min into warm water (35–37°C), without being videotaped or evaluated.

To assess the effectiveness of the stress manipulation, subjective stress ratings, blood pressure measurements, and saliva samples were taken at several time points before and after the experimental manipulation. Participants rated the stressfulness, unpleasantness, and painfulness of the treatment immediately after the SECPT or control manipulation on a scale from 0 (*not at all*) to 100 (*very much*). Blood pressure was measured using an OMRON M400 device (OMRON, Inc.) 5 min before the SECPT or control manipulation and during the SECPT or control manipulation as well as 5, 20, and 60 min after the SECPT or control manipulation. Finally, saliva samples were collected using Salivette collection devices (Sarstedt) 5 min before the SECPT or control manipulation as well as 5, 20, 40, and 60 min thereafter. Saliva samples were stored at −20°C until the end of testing. At the end of data collection, we determined the free fraction of cortisol from the saliva samples using a commercially available luminescence assay (IBL).

Incidental Encoding Task

About 20 min after the SECPT or control manipulation, participants encoded 200 pictures (size: 512 × 384 pixels) while EEG was recorded. These pictures were randomly selected from a pool of 400 pictures taken from the International Affective Picture System (Lang, Bradley, & Cuthbert, 2008); the remaining pictures were used as lures during recognition testing (see below). Half of the encoded pictures were emotionally neutral, and half were emotionally negative, with pictures from these categories being presented in randomized order. Each picture was shown for 2 sec at the center of a computer screen, and participants were instructed to rate its valence and arousal on a scale from 0 (*negative/not at all arousing*) to 4 (*neutral/very arousing*). Between trials, there was an interval of 3–6 sec. In retrospect, the valence ratings confirmed that the negative pictures were experienced as significantly more negative than the neutral pictures, $F(1, 67) = 422$, $p < .001$, $\omega^2 = .761$. Moreover, negative pictures were associated with higher arousal than neutral ones, $F(1, 67) = 1258$, $p < .001$, $\omega^2 = .862$. There were no differences between the stress and control groups with respect to the valence, $F(1, 67) = 0.92$, $p = .341$, $\omega^2 = .000$, and arousal ratings, $F(1, 67) = 0.52$, $p = .472$, $\omega^2 = .000$. The encoding task took about 25 min.

Recognition Test

In the recognition test, 24 hr after encoding, all pictures shown during the encoding task and 200 new pictures (100 negative and 100 neutral) were presented randomly one after another. Each picture was presented for 2 sec,

and participants had to indicate via button presses whether they had seen the shown picture during encoding ("old") or not ("new"). In addition, participants were asked to indicate the confidence of their response on a scale from 0 (*very uncertain*) to 4 (*very certain*). Between recognition trials, there was a variable interval of 3–6 sec. The recognition task lasted about 50 min in total. To make sure that the stress and control groups did not differ in their arousal state during the recognition test, blood pressure measurements and a saliva sample were taken before recognition testing. In addition, participants completed a German mood questionnaire (Multidimensional Mood Scale; Steyer, Schwenkmezger, Notz, & Eid, 1994) that measures subjective feeling on three dimensions (elevated vs. depressed mood, wakefulness vs. sleepiness, and calmness vs. restlessness) before the recognition task.

Control Variables

To control for potential group differences in depressive mood, chronic stress, and anxiety, participants completed the Beck Depression Inventory (Beck, Ward, Mendelson, Mock, & Erbaugh, 1961), the Trier Inventory for the Assessment of Chronic Stress (Schulz, Schlotz, & Becker, 2004), and the State-Trait Anxiety Inventory (Spielberger, Gorsuch, & Lushene, 1970) at the end of the experiment.

Analysis of Behavioral and Physiological Data

The recognition data (hits, false alarms, d') as well as the subjective and physiological stress responses were analyzed by means of mixed-design ANOVAs, paired t tests, and t tests for independent samples. All reported p values are two-tailed and were Bonferroni corrected when indicated. All major statistical analyses were calculated using SPSS 22 (IBM SPSS Statistics); additional analyses were performed in MATLAB (The MathWorks). Because of technical failure of the device for blood pressure measurement, blood pressure data were missing for one participant before and after the SECPT, for two participants during the SECPT, and for six participants on Day 2. Furthermore, salivary cortisol concentrations were too low to be detected for two participants on Days 1 and 2.

EEG Data Acquisition

During the encoding session on Experimental Day 1, participants were seated approximately 80 cm from the monitor in an electrically shielded and sound-attenuated cabin. EEG was recorded using a 128-channel BioSemi ActiveTwo system (BioSemi) organized according to the 10–5 system digitized at 2024 Hz. Additional electrodes were placed at the left and right mastoids, approximately 1 cm above and below the orbital ridge of each eye and at the outer canthi of the eyes. The EEG data were online referenced to the BioSemi CMS-DRL (common mode sense-driven right leg) reference and rereferenced offline to a common average. Electrode impedances were kept below 30 kΩ. EEG and EOG were amplified with a low cutoff frequency of 0.53 Hz (= 0.3-sec time constant) and resampled to 256 Hz.

EEG Data Analysis

Preprocessing

EEG data from the encoding task were analyzed offline using FieldTrip (Oostenveld, Fries, Maris, & Schoffelen, 2011; www.ru.nl/neuroimaging/fieldtrip) and EEGLAB (Delorme & Makeig, 2004; sccn.ucsd.edu/eeglab/index.html) as well as custom scripts implemented and processed in MATLAB (The MathWorks). An independent component analysis (Makeig, Bell, Jung, & Sejnowski, 1996) was used to correct for eye and eyelid movements. Components that indicated ocular activity were removed (on average, 1.2 components per participant). Because the control for power line noise during data acquisition often cannot reduce the power line interference completely (Nottage & Horder, 2015), we applied spectrum interpolation to remove potential power line noise from EEG data (Leske & Dalal, 2019). More specifically, the time domain signal was transformed into the frequency domain using a discrete Fourier transformation. Then, the line noise component was removed in the amplitude spectrum by interpolating the curve at the interference frequency of 50 Hz according to the neighboring Fourier coefficients of 47 and 53 Hz ($\Delta = \pm 3$ Hz). Finally, data were transformed back into the time domain utilizing an inverse Fourier transformation. Furthermore, epochs from −2000 msec until stimulus onset were extracted. In the face of a lack of previous studies on stress and prestimulus activity, this epoch duration was chosen in line with previous prestimulus studies on prestimulus activity and memory (Scholz, Schneider, & Rose, 2017; Schneider & Rose, 2016). Trials containing large EMG bursts (as a sign of muscular artifacts) or spikes (as an indicator of badly connected electrodes) were detected by visual inspection of the data and excluded from the analysis. Finally, data of all trials for each participant and each condition were averaged. Because we focused on the influence of prestimulus activity on subsequent encoding, no baseline correction was carried out on the data (Schneider & Rose, 2016). Data of 24 participants had to be excluded from the EEG analysis because of technical failure during the EEG, leaving a sample of 52 participants (24 in the stress group, 28 in the control group) for these analyses. However, the stress response, memory performance, and demographics for the 52 participants included in the EEG analysis were comparable to those of the entire sample (Tables S1–S3 in the supplemental materials). In our EEG analyses, we did not apply any criteria for a minimal number of misses. In addition, however, we computed all analyses again considering only the participants who had at least 10% misses. This additional analysis, however, left our findings largely unchanged, except for the effects in high gamma, which were not significant in the reduced sample (Table S4 in the supplemental materials).

Phase and Amplitude Extraction

For phase and amplitude extraction, the EEG data were first band-pass filtered into the relevant frequency bands (frequencies of interest: theta [4–8 Hz], alpha [9–12 Hz], low beta [13–17 Hz], high beta [18–30 Hz], low gamma [30–45 Hz], and high gamma [55–70 Hz]). Using the Hilbert transformation, the band-pass-filtered time series was then converted into a complex, analytic signal. Finally, instantaneous phase $\Phi(t)$ and the amplitude $A(t)$ were extracted from this complex-valued analytical signal taking argument or modus (Dvorak & Fenton, 2014). For spectral power (μV^2) analyses, we used z-transformed mean power to control for differences in EEG power spectra among the participants (i.e., for within-participant variability).

Searchlight Analysis: Spectral Power

To identify frequency-specific topographical regions implicated in later memory performance, a searchlight approach was implemented (Kriegeskorte, Goebel, & Bandettini, 2006). This approach was implemented with the help of the MVPA-Light Toolbox (github.com/treder/MVPA-Light). For the searchlight analysis, a loop was implemented to classify single-trial data of every electrode for an averaged time window of 500 msec. To decode the class information (memory performance) in the searchlight, a linear support vector machine (SVM) pattern classifier was trained to distinguish between the two classes of stimuli (remembered/forgotten). An SVM was chosen because of its advantages compared to other classifiers in dealing with many features (Murphy et al., 2011) such as those resulting from EEG recordings. The proportion of the remembered and forgotten classes varied depending on the performance of each participant, thus partly resulting in an imbalanced data set. This bias in the training data set can influence many machine learning algorithms—such as it is the case for the utilized SVM—toward the majority class. Different techniques have been suggested to control for this issue (e.g., Noh, Herzmann, Curran, & de Sa, 2014). Here, we used a balanced cross-validation approach. Therefore, we randomly resampled the training data set. Trials from the minority class were randomly duplicated and added to the training data set producing a new balanced training data set. Herawan, Deris, and Abawajy (2014) recommended this oversampling technique for classification in imbalanced datasets and suggested that this approach yields results comparable to other methods, such as undersampling. The classifier's ability to generalize the distinction between the two classes to new data was assessed using a 10-fold stratified cross-validation (Jamalabadi, Alizadeh, Schönauer, Leibold, & Gais, 2016), which reflects the best method for model validation based on a comparison of different number of folds and bootstrap methods (Kohavi, 1995). Thereby, one tenth of each class is randomly selected and left out from classifier training procedure. The left-out set was tested using the classifier trained from all trials in the cross-validation set for each fold. If the classifier's performance after cross-validation is significantly above chance, it indicates that the EEG patterns contain class-specific information and that the class can be reliably decoded from the EEG data (Murphy et al., 2011). The chance level in a simple two-class paradigm is not exactly 50% but 50% with a confidence interval at a certain alpha level. Therefore, we calculated this interval utilizing the Wald interval with adjustments for a small sample size (Müller-Putz, Scherer, Brunner, Leeb, & Pfurtscheller, 2008; Agresti & Caffo, 2000). Choosing a highly conservative estimation based on the Wald interval for further inference statistical testing, we pooled electrodes with a classification accuracy ≥ 0.6 into global clusters.

Searchlight Analysis: Phase–Amplitude Coupling

The relationship between the phase and the amplitude time series was examined using circular statistics by calculating the mean vector of the complex composite signal: $z(t) = A(t)\exp[i\Phi(t)]$ (Canolty & Knight, 2010). Because we assumed, based on previous findings, that the phase of a slow frequency would modify the amplitude of a higher frequency (Canolty & Knight, 2010), we focused on the coupling between the theta phase and higher frequencies (low beta, high beta, low gamma, high gamma). To identify topographical prestimulus coupling–SME cluster, the average phase of the three frontal, predefined clusters were determined (frontal-left, frontal-midline, and frontal-right) using the complex signal. Thereafter, the phase–amplitude coupling (PAC) between these predefined frontal clusters and every other electrode for the time range of −2000 msec relative to the stimulus onset was determined. Next, the prestimulus PAC was averaged for a time window of 500 msec, resulting in four PAC indices. To create a standardized Z value of each observed PAC, we applied permutation testing. This involved a temporal shift of the phase time series by a random temporal offset without changing the power time series (Cohen, 2014). The PAC value is then computed according to the former formula, generating one PAC value under the null hypothesis. This procedure was repeated using 1000 permutations. Then, we compared the appropriate observed PAC to the distribution of PAC values under the null hypothesis by subtracting the mean and dividing by the standard deviation, generating a standardized Z value of PAC. Therefore, negative PAC values, for example, indicate lower coupling strength than it would be expected based on a random distribution. To identify couplings linked to subsequent memory performance, the searchlight method was applied also using a 10-fold cross-validation (see above). Coupling strength based on standardized Z value of PAC for each electrode relative to the predefined cluster served as input. Clusters for which coupling exhibited classification accuracy ≥ 0.6 were subjected to univariate testing.

RESULTS

Successful Stress Manipulation

Significant subjective and physiological changes in response to the SECPT confirmed the successful stress induction. Compared to participants in the control group, participants exposed to the SECPT experienced the treatment as significantly more stressful, painful, difficult, and/or unpleasant than those in the control condition, all ts(67) < −6.2, all ps < .001, all Cohen's ds < −1.497 (Table 1). At the physiological level, exposure to the SECPT elicited significant increases in both diastolic and systolic blood pressure (Group × Time Point of Measurement interaction, both Fs(5, 320) > 27.36, both ps < .001, both ω^2 > .099). As shown in Table 1, groups had comparable blood pressure before and after the SECPT and control manipulation, respectively (all ts(66) < 1.51, all ps > .135, all Cohen's ds < 0.357), although participants in the stress condition had significantly higher blood pressure than those in the control condition during the hand immersion (both ts(65) > 5.54, both ps < .001, both Cohen's ds > 1.36; see Table 1). Finally, salivary cortisol concentrations increased in response to the SECPT but not after the control manipulation (Group × Time Point of Measurement interaction, $F(4, 256) = 9.93$, $p < .001$, $\omega^2 = .055$). As shown in Figure 1, participants of the stress and control groups had comparable cortisol concentrations at baseline and immediately after the SECPT, all ts(65) < 0.25, all ps > .807, all Cohen's ds < 0.060, whereas cortisol levels were significantly higher in the stress group than in the control group 20 and 40 min after the SECPT, both ts(65) > 2.89, both ps < .005, both Cohen's ds > 0.715. Salivary cortisol increased about 100% relative to baseline in the stress group and peaked when the encoding task started (see Figure 1). At 60 min after the treatment, stress-induced cortisol concentrations returned to the level of the control group, $t(65) = 0.35$, $p = .729$, Cohen's $d = 0.086$.

Emotional Memory Enhancement

At recognition testing 24 hr after encoding, groups did not differ in Multidimensional Mood Scale scores (all Fs(1, 67) < 0.73, all ps > .395, all ω^2 < .000), blood pressure (both ts (61) < 0.62, both ps > .54, both Cohen's ds < 0.156), or salivary cortisol, $t(65) = 1.74$, $p = .087$, Cohen's d = 0.425. As expected, recognition performance was overall significantly better for negative than for neutral pictures as reflected in a higher d', $F(1, 67) = 41.08$, $p < .001$, $\omega^2 = .083$

Table 1. Subjective Stress Ratings and Blood Pressure Before, During, and After the SECPT or Control Manipulation

		Control	*Stress*
Subjective assessments	Difficulty	12.1 (4.0)	59.3* (5.5)
	Stressfulness	12.1 (3.5)	54.6* (5.3)
	Painfulness	9.4 (3.9)	63.7* (5.9)
	Unpleasantness	16.2 (4.4)	62.1* (6.0)
Systolic blood pressure (mmHg)	Before hand immersion	109.7 (1.6)	109.3 (2.0)
	During hand immersion	110.4 (1.5)	128.8* (3.1)
	5 min after hand immersion	107.2 (1.8)	111.5 (2.3)
	20 min after hand immersion	108.4 (1.3)	106.8 (2.0)
	40 min after hand immersion	109.2 (1.2)	108.6 (2.0)
	60 min after hand immersion	110.1 (1.2)	109.3 (2.1)
	24 hr after hand immersion	112.4 (1.9)	110.5 (2.3)
Diastolic blood pressure (mmHg)	Before hand immersion	73.6 (1.3)	76.6 (2.1)
	During hand immersion	74.6 (1.3)	91.9* (2.2)
	5 min after hand immersion	73.7 (1.1)	76.5 (1.5)
	20 min after hand immersion	75.3 (1.3)	75.1 (1.3)
	40 min after hand immersion	75.1 (1.1)	75.1 (1.2)
	60 min after hand immersion	75.8 (1.2)	76.4 (1.5)
	24 hr after hand immersion	70 (1.7)	70.1 (1.1)

Data represent means. *SEM*s are given in parentheses.

* $p < .01$ (stress vs. control).

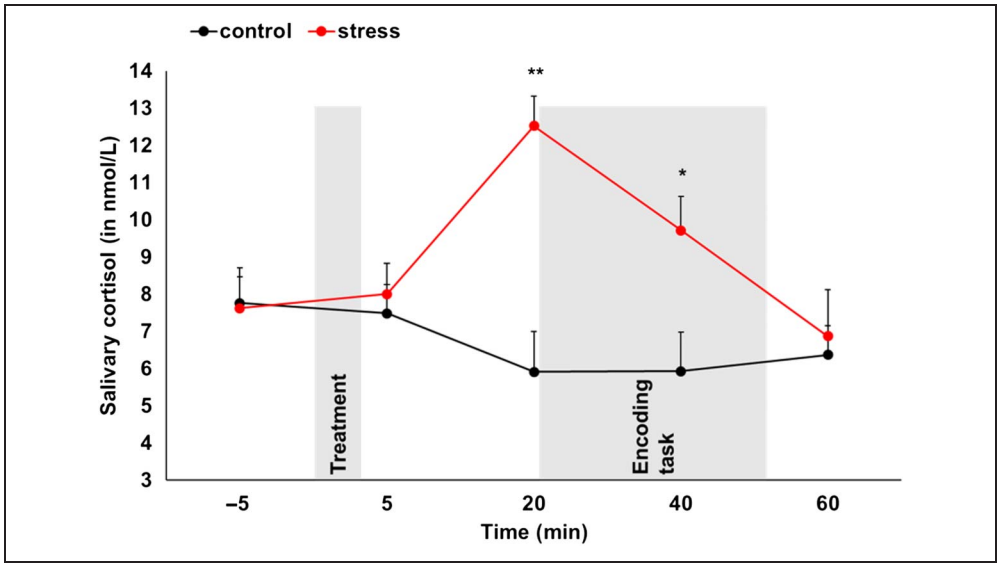

Figure 1. Salivary cortisol response (in nanomoles per liter) to the stress and control manipulation. The gray bars denote the timing and duration of the treatment (SECPT vs. control manipulation) and the encoding task, respectively. Note that the learning task was presented during the high-cortisol period of the stress group. Data represent means ± *SEM*. *$p < .05$ (corrected) and **$p < .001$, respectively, indicate a significant group difference.

(Table 2). The stress and control groups, however, did not differ in their overall memory performance, $F(1, 67) = 0.21$, $p = .650$, $\omega^2 = .000$, or the emotional memory enhancement (Valence × Group interaction, $F(1, 67) = 0.1$, $p = .750$, $\omega^2 = .000$). In addition, memory performance did not correlate with heart rate immediately before encoding, neither in the stress group ($r = .14$, $p = .445$) nor in the control group ($r = .08$, $p = .64$). Because previous studies showed that a stress-induced enhancement of memory performance may depend on the subjective arousal elicited by an individual stimulus (Goldfarb et al., 2019), we additionally analyzed stress effects considering stimulus-specific subjective valence and arousal ratings, respectively. Similar to d' based on a priori categories in our initial analysis, recognition performance was overall significantly better for negative than for neutral pictures, $F(1, 65) = 59.81$, $p < .001$, $\omega^2 = .158$. The stress and control groups still did not differ in memory performance, $F(1, 65) = 2.19$, $p = .144$, $\omega^2 = .017$, or the emotional memory enhancement (Valence × Group interaction, $F(1, 65) = 3.09$, $p = .083$, $\omega^2 = .011$). Considering subjective arousal ratings, recognition performance was significantly higher for high-arousal compared to low-arousal pictures, $F(1, 66) = 22.11$, $p < .001$, $\omega^2 = .098$. However, the stress and control groups still did not differ in memory performance, $F(1, 66) = 0.42$, $p = .521$, $\omega^2 = .000$, or the emotional memory enhancement (Valence × Group interaction, $F(1, 66) = 0.95$, $p = .334$, $\omega^2 = .000$). Furthermore, confidence ratings were higher for remembered ratings than for forgotten pictures, $F(1, 67) = 342$, $p < .001$, $\omega^2 = .617$. Confidence ratings did not differ between stimuli valence, $F(1, 67) = 2.87$, $p = .095$, $\omega^2 = .005$ (Valence × Memory interaction, $F(1, 67) = 2.34$, $p = .131$, $\omega^2 = .004$) or groups, $F(1, 67) = 0.00$, $p = .969$, $\omega^2 = .000$ (Memory × Group interaction, $F(1, 67) = 0.04$, $p = .842$, $\omega^2 = .000$; Valence × Group interaction, $F(1, 67) = 0.14$, $p = .712$, $\omega^2 = .000$; Valence × Memory × Group interaction, $F(1, 67) = 0.17$, $p = .685$, $\omega^2 = .000$).

Stress Alters the Neurophysiological Readiness to Form New Memories

Although memory performance was overall comparable in the stress and control groups, we leveraged EEG and multivariate pattern analysis to analyze whether stress alters the neural context for forming new memories, reflected in the neural activity before a stimulus is presented, and thus the mechanism through which memories are built. In a first step, we focused on spectral power and performed a searchlight analysis to detect frequency bands, present before stimulus onset, which can distinguish subsequently remembered and forgotten items.

This analysis showed that theta activity immediately before stimulus onset was significantly lower for subsequently remembered versus forgotten items, $F(1, 50) = 12.32$, $p <$

Table 2. Recognition Performance

	Control		*Stress*	
	Neutral	*Negative*	*Neutral*	*Negative*
d'	2.12 (0.13)	2.55 (0.12)	2.25 (0.11)	2.65 (0.11)
Hit rate (%)	0.82 (0.02)	0.90 (0.01)	0.84 (0.01)	0.91 (0.01)
False alarm rate (%)	0.15 (0.12)	0.15 (0.10)	0.16 (0.12)	0.15 (0.09)

Data represent means. *SEM*s are given in parentheses.

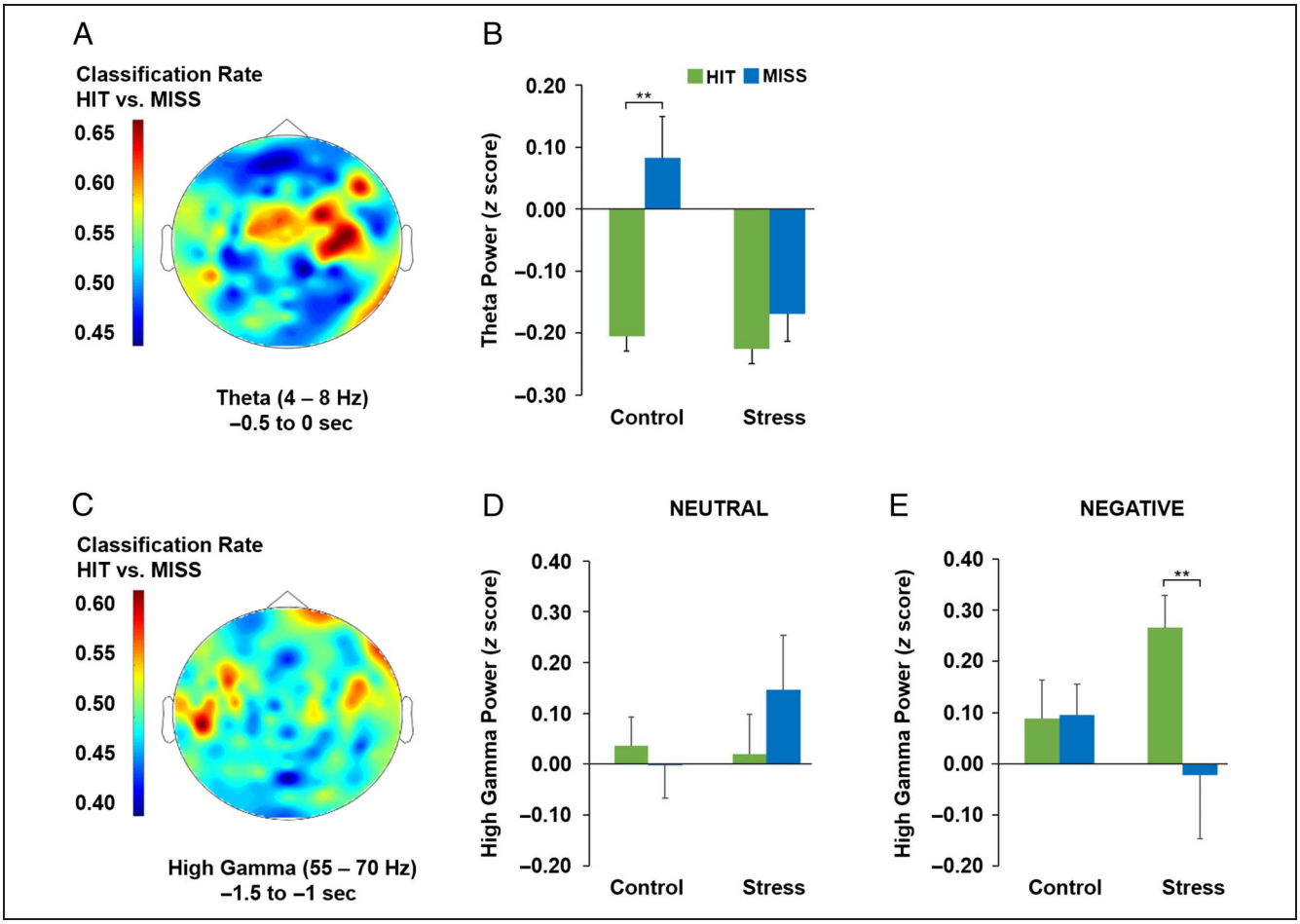

Figure 2. Subsequent memory effect of prestimulus power for each group. (A) Topographic distribution of theta power based on searchlight clustering. (B) Mean theta power based on searchlight clustering (averaged across electrodes with accuracy > 0.6) within an interval between −0.5 and 0 sec relative to stimulus onset as a function of recognition (HIT vs. MISS) and experimental group (stress vs. control). (C) Topographic distribution of high gamma power based on searchlight clustering. (D) Mean high gamma power based on searchlight clustering (averaged across electrodes with accuracy > 0.6) within an interval between −1.5 and −1 sec relative to stimulus onset as a function of recognition (HIT vs. MISS) and experimental group (stress vs. control) for neutral pictures. (E) Mean high gamma power based on searchlight clustering (averaged across electrodes with accuracy > 0.6) within an interval between −1.5 and −1 sec relative to stimulus onset as a function of recognition (HIT vs. MISS) and experimental group (stress vs. control) for negative pictures. Data represent means ± *SEM*. *$p < .05$ and **$p < .001$.

.001, $\omega^2 = .106$. The topographical distribution of this cluster covered right central–parietal regions (see Figure 2A). Interestingly, however, the role of prestimulus theta in memory formation differed between the stress and control groups (Group × Memory interaction, $F(1, 50) = 4.05, p = .05$, $\omega^2 = .031$; see Figure 2B): Prestimulus theta activity was linked to subsequent memory performance in the control group, $t(27) = 3.74, p < .001$, Cohen's $d = 0.707$, but not in the stress group, $t(23) = 1.15, p = .263$, Cohen's $d = 0.234$.

In sharp contrast to prestimulus theta, high gamma was linked to subsequent remembering (vs. forgetting) exclusively in stressed participants and specifically for emotionally arousing material (Valence × Memory × Group interaction, $F(1, 50) = 5.86, p = .002, \omega^2 = .042$). As shown in Figure 2C–E, high gamma activity over the left temporal lobe was significantly higher for subsequently remembered (vs. forgotten) negative pictures, $t(23) = 4.12, p < .001$, Cohen's $d = 0.86$, but not neutral pictures, $t(23) = 0.89$, $p = .385$, Cohen's $d = 0.181$, in the stress group, whereas no such association was observed in nonstressed controls (negative pictures: $t(27) = 0.07, p = .946$, Cohen's $d = 0.013$; neutral pictures: $t(27) = 0.46, p = .648$, Cohen's $d = 0.087$). Interestingly, the difference in the effect of this high-gamma-activity cluster on memory performance between negative and neutral pictures tended to be higher in individuals showing the highest cortisol peak concentration ($r = .26, p = .066$). In a similar vein, this cluster showed a significant coupling with theta phase for negative stimuli in stressed participants, which was also significantly correlated with cortisol ($r = .43, p = .043$).

In a next step, we focused on PAC, a measure of interactions between oscillations in different frequency bands that may reflect the crosstalk between brain areas (Engel, Fries, & Singer, 2001). We therefore analyzed, again using a searchlight approach, which PACs before stimulus onset were predictive for later memory. This analysis revealed that the coupling between theta and high beta was higher

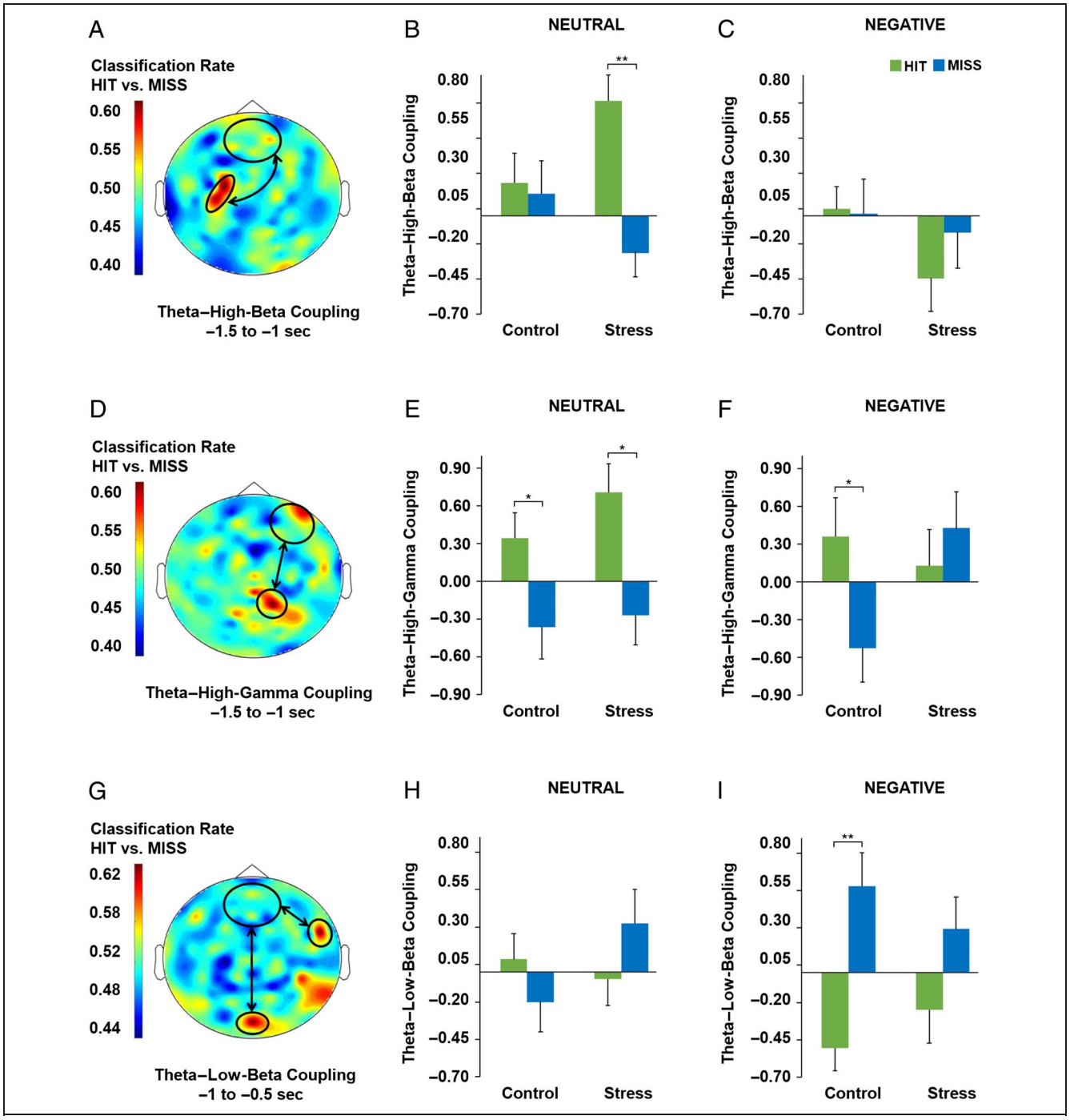

Figure 3. Subsequent memory effect of prestimulus PAC for each group. (A) Topographic distribution of theta–high-beta coupling based on searchlight clustering. (B) Mean theta–high-beta coupling based on searchlight clustering (averaged across electrodes with accuracy > 0.6) within an interval between −1.5 and −1 sec relative to stimulus onset as a function of recognition (HIT vs. MISS) and experimental group (stress vs. control) for neutral pictures. (C) Mean theta–high-beta coupling based on searchlight clustering (averaged across electrodes with accuracy > 0.6) within an interval between −1.5 and −1 sec relative to stimulus onset as a function of recognition (HIT vs. MISS) and experimental group (stress vs. control) for negative pictures. (D) Topographic distribution of theta–high-gamma coupling based on searchlight clustering. (E) Mean theta–high-gamma coupling based on searchlight clustering (averaged across electrodes with accuracy > 0.6) within an interval between −1.5 and −1 sec relative to stimulus onset as a function of recognition (HIT vs. MISS) and experimental group (stress vs. control) for neutral pictures. (F) Mean theta–high-gamma coupling based on searchlight clustering (averaged across electrodes with accuracy > 0.6) within an interval between −1.5 and −1 sec relative to stimulus onset as a function of recognition (HIT vs. MISS) and experimental group (stress vs. control) for negative pictures. (G) Topographic distribution of theta–low-beta coupling based on searchlight clustering. (H) Mean theta–low-beta coupling based on searchlight clustering (averaged across electrodes with accuracy > 0.6) within an interval between −1 and −0.5 sec relative to stimulus onset as a function of recognition (HIT vs. MISS) and experimental group (stress vs. control) for neutral pictures. (I) Mean theta–low-beta coupling based on searchlight clustering (averaged across electrodes with accuracy > 0.6) within an interval between −1 and −0.5 sec relative to stimulus onset as a function of recognition (HIT vs. MISS) and experimental group (stress vs. control) for negative pictures. Data represent means ± *SEM*. *$p < .05$ and **$p < .001$.

for subsequently remembered neutral pictures, $t(23) = 3.7$, $p < .001$, Cohen's $d = 0.756$, but not for negative pictures, $t(23) = 0.97$, $p = .343$, Cohen's $d = 0.198$, in stressed participants, whereas there was no association between theta–high-beta coupling and subsequent memory in control participants (neutral and negative, both $ts(27) < 0.26$, both $ps > .799$, both Cohen's $ds < 0.049$; Valence × Memory × Group interaction, $F(1, 50) = 4.72$, $p < .035$, $\omega^2 = .038$; see Figure 3A–C).

Although this finding pointed to a prestimulus activity pattern specific for subsequently remembered neutral material in stressed participants, we further obtained a significant role of the PAC between frontal-right theta and parietal-right high-gamma frequency that appeared to be relevant for memory formation in nonstressed controls as well as for neutral material after stress (Valence × Group × Memory interaction, $F(1, 50) = 4.43$, $p = .04$, $\omega^2 = .028$; see Figure 3D–F). More specifically, this theta–high-gamma coupling was linked to subsequent memory for neutral, $t(23) = 3.09$, $p = .005$, Cohen's $d = 0.630$, but not negative pictures, $t(23) = 0.89$, $p = .385$, Cohen's $d = 0.181$, in stressed participants, whereas it was equally relevant for subsequent remembering of neutral and negative pictures in control participants (neutral pictures: $t(27) = 2.16$, $p = .039$, Cohen's $d = 0.409$; negative pictures: $t(27) = 2.39$, $p = .024$, Cohen's $d = 0.451$).

In addition to these findings showing prestimulus activity and connectivity patterns that were specific to successful memory formation for neutral or negative material in stress participants, there was one pattern of cross-frequency coupling that was specific to emotional memory formation in nonstressed controls: In controls, theta–low-beta coupling between frontal-midline on the one hand and frontal-right and occipital regions on the other hand was specifically linked to subsequent memory for negative pictures, $t(27) = 4.02$, $p < .001$, Cohen's $d = 0.760$, but not for neutral pictures, $t(27) = 1.17$, $p < .253$, Cohen's $d = 0.221$. In stressed participants, there was no such link (neutral and negative, both $ts(23) < 1.84$, both $ps > .078$, both Cohen's $ds < 0.243$; Valence × Memory × Group interaction, $F(1, 50) = 7.28$, $p = .01$, $\omega^2 = .043$; see Figure 3G–I).

The reported prestimulus effects do not necessarily rule out the possibility that the stressor also affects more sustained neural activity that was not captured in the analysis of prestimulus spectral power and PAC. To clarify this aspect, we performed additional analyses for the time window during stimulus presentation (0–2000 msec relative to stimulus onset; analyses were carried out in the same manner as the prestimulus analyses). There were stress effects on the SME within high and low beta power (Figure S1A–F in the supplemental materials) but no effects of PAC during the poststimulus interval (see supplemental materials).

Given evidence that the processing of high-arousing emotional compared to low-arousing neutral stimuli may affect information processing in trials after their presentation (Wirkner, Ventura-Bort, Schulz, Hamm, & Weymar, 2018; Flaisch, Stockburger, & Schupp, 2008; Kunde & Mauer, 2008), we reanalyzed the prestimulus spectral power and PAC with respect to the valence of the preceding picture. These analyses suggested that the emotional content of the previous picture did not affect prestimulus spectral power (theta spectral power within the time window of −500 msec to stimulus onset: effects of previous valence: $F(1, 50) = 0.11$, $p = .743$, $\omega^2 < .000$; Previous

Table 3. Control Variables

		Control	*Stress*
BDI		6.40 (1.14)	6.64 (0.81)
STAI scales	State anxiety	38.20 (1.64)	36.93 (1.42)
	Trait anxiety	35.76 (1.52)	37.82 (1.86)
TICS scales	Work overload	10.00 (1.05)	12.20 (1.26)
	Social overload	5.91 (0.76)	7.76 (0.86)
	Performance pressure	12.16 (1.22)	13.77 (1.01)
	Work discontent	11.00 (1.03)	11.20 (1.10)
	Excessive workload	5.10 (0.65)	6.13 (0.84)
	Lack of social recognition	3.94 (0.56)	4.00 (0.55)
	Social tension	5.13 (0.77)	5.06 (0.69)
	Social isolation	7.24 (0.78)	6.89 (0.74)
	Chronic worrying	4.48 (0.63)	6.03 (0.60)
	TICS screening scale	11.66 (1.33)	15.33 (1.66)

Data represent means. *SEM*s are given in parentheses. BDI = Beck Depression Inventory; STAI = State-Trait Anxiety Inventory; TICS = Trier Inventory of Chronic Stress.

Valence × Group interaction, $F(1, 50) = 0.21$, $p = .649$, $\omega^2 < .000$; high gamma spectral power within the time window of −1500 to −1000 msec relative to stimulus onset: effects of previous valence: $F(1, 50) = 0.19$, $p = .665$, $\omega^2 < .000$; Previous Valence × Target Valence interaction, $F(1, 50) = 0.24$, $p = .623$, $\omega^2 < .000$; Previous Valence × Target Valence × Group interaction, $F(1, 50) = 0.62$, $p = .436$, $\omega^2 < .000$) or PAC (effects of previous valence: all Fs$(1, 50) < 3.7$, all ps $> .061$, all $\omega^2 < .035$; Previous Valence × Target Valence interaction, all Fs$(1, 50) < 1.28$, all ps $> .263$, all $\omega^2 < .004$; Previous Valence × Target Valence × Group interaction, all Fs$(1, 50) < 3.73$, all ps $> .06$, all $\omega^2 < .03$).

Control Variables

As shown in Table 3, groups did not differ in subjectively reported chronic stress levels, depressive mood, state, or trait anxiety, all ts$(67) < 0.57$, all ps $> .114$, all Cohen's ds < 0.143.

DISCUSSION

Previous research showed that stress may facilitate memory formation, specifically for emotionally arousing material (Buchanan et al., 2006; Roozendaal et al., 2006; Cahill et al., 2003), and linked this memory enhancement to an interaction of noradrenaline and glucocorticoids in the basolateral amygdala, which then modulates mnemonic processing in other areas, such as the hippocampus (Roozendaal & McGaugh, 2011). Whereas previous research (in particular, in humans) focused primarily on stress-induced changes in stimulus-related activity, we tested here an alternative mechanism: whether stress may alter the neural context for memory encoding, reflected in the activity preceding a stimulus. Our findings show that stress before encoding indeed changed the prestimulus activity predictive for remembering 24 hr later. Specifically, whereas prestimulus theta activity predicted memory formation, irrespective of the emotionality of the material, in nonstressed controls but not in stressed participants, prestimulus high gamma was predictive for subsequent memory, selectively for emotionally arousing material, after stress. Frontal theta–high-gamma coupling, in turn, was predictive for subsequent overall memory in nonstressed controls and for neutral memory in stressed individuals but selectively not for emotional stimuli after stress. Together, the present findings provide, to the best of our knowledge, the first evidence that stress might alter the neural context or readiness for memory formation, in a manner that is, to some degree, specific to emotionally arousing information.

It is by now well established that the neural activity preceding a stimulus may determine whether this stimulus is later remembered (Sweeney-Reed et al., 2015; Fell et al., 2011; Otten et al., 2006). In line with earlier reports (Burke et al., 2013; Staudigl & Hanslmayr, 2013), we observed here in nonstressed controls that a reduction in activity in the theta band (over central and temporal cortical regions) is linked to enhanced subsequent memory (but see also Schneider & Rose, 2016, and Fellner, Bäuml, & Hanslmayr, 2013, for enhancement in theta band related to better memory performance), possibly indexing enhanced hippocampal theta activity resulting from a synchronizing loop between the cortex and hippocampus (Lega, Jacobs, & Kahana, 2012). Although the exact functional meaning of frequency bands is still debated (Klimesch, Schack, & Sauseng, 2005), cortical theta might further point to attenuated activity in the "default mode network," shaping the neural context toward external stimulus processing through less interference from internal monitoring processes (Fair et al., 2008). In addition to theta, prestimulus theta–high-gamma coupling over frontal–parietal areas predicted subsequent remembering. Theta–gamma coupling has so far been mainly implicated in working memory, specifically in the maintenance of information and sequential memory organization (Lisman, 2005), which may however promote long-term memory formation as well.

Whereas both prestimulus theta activity and theta–high-gamma coupling were linked to subsequent remembering irrespective of the emotionality of the encoded material, theta–low-beta coupling preceding a stimulus predicted later memory specifically for emotionally arousing stimuli in nonstressed controls. Thus, reduced theta–low-beta coupling appears to represent a neural state that facilitates emotional memory formation under no-stress conditions, thus contributing to the well-known memory advantage for emotional relative to neutral stimuli (Dolcos et al., 2017, 2020; Kensinger, 2007; McGaugh et al., 1996; Cahill, Prins, Weber, & McGaugh, 1994; Christianson, 1992) that we observed also in the present experiment. Our data point to an involvement of frontal regions in this effect, in line with the idea that reduced theta–low-beta coupling between frontal and posterior regions might reflect an inhibitory mechanism enhancing the processing of relevant stimulus features by reducing interference from other (visual) sensory input. This view is further in line with previous findings suggesting that reduced frontoparietal theta–beta ratio is associated with better orienting network functioning (Morillas-Romero, Tortella-Feliu, Bornas, & Putman, 2015).

Stress before encoding markedly changed the neural context relevant for memory formation. Neither prestimulus theta nor frontal theta–low-beta coupling was relevant for subsequent memory in stressed participants, which is in sharp contrast to the pattern observed in the control group. Instead, high gamma activity preceding a stimulus was predictive for subsequent memory in stressed individuals. Given that gamma oscillations have been linked to cortical activation and attentional processing before (Herrmann, Strüber, Helfrich, & Engel, 2016), this finding might point to a switch in encoding mechanisms, and perhaps the attentional focus, after stress. In line with this idea, high gamma was selectively involved in memory formation for emotionally salient material under stress. For neutral

stimuli, prestimulus high gamma was irrelevant but became relevant only when followed by emotionally arousing material. Conversely, theta–high-gamma coupling before a stimulus was relevant for overall memory in controls and memory for neutral stimuli in stressed individuals but not for emotional material in the stress group. These data suggest that stress tuned the neural encoding context specifically toward processing emotionally arousing material, which would be generally in line with a proposed stress-induced bias of large-scale networks toward processing emotionally salient information (Hermans et al., 2011). Furthermore, the finding that the level of prestimulus gamma that was implicated in emotional memory formation under stress tended to be higher in individuals with a strong cortisol response to the stressor dovetails with extensive evidence showing an interaction of glucocorticoids and noradrenergic arousal in memory formation under stress (Roozendaal & McGaugh, 2011; Roozendaal et al., 2006).

Interestingly, the prestimulus high gamma increase for subsequently remembered stimuli after stress was most pronounced over temporal sites known to play a key role in memory formation (Hannula & Ranganath, 2008; Davachi, Mitchell, & Wagner, 2003; Squire & Zola-Morgan, 1991), albeit the low spatial resolution of EEG makes the mapping to specific brain areas difficult. The absence of a subsequent memory effect for emotional material in theta–high-gamma coupling over frontal sites after stress might be taken as evidence for a reduction in relevant top–down processing, known to rely on prefrontal areas (Márton, Fukushima, Camalier, Schultz, & Averbeck, 2019). A potential bias from prefrontal top–down control to more bottom–up processing would again be in line with a stress-induced network reconfiguration in favor of a salience network and at the expense of an executive control network (Hermans et al., 2011, 2014) as well as with findings showing that stress biases mnemonic processing from "cognitive" toward more rigid systems (Schwabe, 2017; Vogel, Fernández, Joëls, & Schwabe, 2016).

Although these stress-induced changes in memory-related prestimulus activity appeared to tune encoding toward emotionally relevant material, frontocentral theta–high-beta coupling before stimulus onset was specific to subsequent memory for neutral material under stress. It is tempting to speculate that this neural state may be required for overcoming the emotional encoding bias under stress. At this point, it is, however, important to note that stress did not change memory performance in this study. Earlier studies in which stress was induced before encoding reported very heterogeneous results, with some studies reporting enhancing effects on memory (Goldfarb et al., 2019; Schwabe, Bohringer, Chatterjee, & Schächinger, 2008), whereas others obtained impairing effects (Zoladz et al., 2011; Elzinga, Bakker, & Bremner, 2005) or, as in this study, no effect (Shields, Sazma, McCullough, & Yonelinas, 2017; Weymar et al., 2012). This heterogeneity may be owing to the fact that stress before encoding affects several distinct processes, with some directly related to not only encoding but also consolidation processes. Moreover, the temporal proximity between stressor and encoding is thought to be critical for stress effects on memory (Joëls et al., 2006, 2011); thus, across an encoding task of about 30 min, distinct effects might develop that are difficult to disentangle later on. Recent research also demonstrates that stress effects are modulated by arousal responses during encoding (Segal et al., 2014; Bryant, McGrath, & Felmingham, 2013). Here, we did not collect marker for arousal during stimulus-specific encoding, making it difficult to directly address this question. Although we did not find any association of stress effects on subsequent memory with heart rate shortly before encoding, future studies could use continuous heart rate or skin conductance measurements to address the contribution of arousal during encoding on memory. Importantly, however, encoding under stress is highly relevant in everyday life, not only in clinical contexts but, for instance, also in educational settings, and studying stress effects on encoding and the mechanisms involved in these effects is highly relevant. Our findings yield novel insights into the mechanisms that are critical for memory formation under stress. These encoding differences may translate into differential memory performance depending on the specific demands of the test situation and its relation to the encoding context.

Although acute stress affected the neural prestimulus state associated with subsequent recognition performance, this does not necessarily imply that the stress effects are specific to the prestimulus period. In our analysis, we focused on the time interval before stimulus onset because we did not aim to examine stress-induced subsequent memory effects per se but to unravel whether stress may alter the neural context for successful encoding, as reflected in prestimulus activity. However, to assess whether stress might also affect sustained activity, we conducted additional analyses for the window during stimulus presentation. The stress effects on spectral power during stimulus presentation differed from the prestimulus activity in (i) the crucial frequency (theta and high gamma within the prestimulus time window vs. low and high beta within the poststimulus time window) and (ii) the topography (central parietal and left temporal for the prestimulus time window vs. right temporal clusters for the poststimulus time window). In addition, post hoc analysis of PAC during the poststimulus interval revealed that there were no effects of stress on subsequent memory at all. Taken together, spectral power and PAC data argue clearly in favor of the view that the reported stress effects are specific to the prestimulus time window. Although these data argue against more general stress effects on sustained activity, it remains unclear how long the stress effects on the neural context for encoding that we suggest here last. There is evidence that stress may exert effects on encoding long after the actual stress exposure (Schwabe, 2017; Tambini, Rimmele, Phelps, & Davachi, 2017; Hermans et al., 2011, 2014; Buchanan & Lovallo, 2001), although the nature of these effects may change depending on the mode of stress hormone action (Hermans et al., 2014; van Ast, Cornelisse, Meeter, Joëls, & Kindt, 2013).

Thus, it may well be that the effects reported here may last for several hours of the stressful event.

In previous prestimulus studies, a preceding cue predicted the upcoming stimulus (Scholz et al., 2017; Schneider & Rose, 2016; Otten et al., 2006). In contrast, we focused here on stress effects on prestimulus activity without displaying such a cue. We reasoned that a stressful event, which may be conceptualized as motivational stimulus, induced—through the hormones and neurotransmitters released in response to stress—a physiological state characterized for an enhanced focus of information processing for emotionally relevant material. Our data support this idea and therefore suggest that prestimulus activity may not only be altered by cues immediately preceding a stimulus but also by more long-lasting states triggered by a potent motivational stimulus, such as a stressful event.

We provide here the first evidence that acute stress modulates the neural context for successful memory formation by altering the stimulus-preceding activity relevant for memory. Nevertheless, it is to be noted that, because of largely missing data on a stress-induced modulation of prestimulus activity related to subsequent memory effects and methodological constraints inherent to the searchlight approach, the prestimulus duration of 2000 msec was subdivided into four time bins, each consisting of consecutive 500 msec. Moreover, because stress-induced changes in neural activity were obtained at different time points, with different temporal proximity to the stimulus, another challenge for future research is to investigate whether these changes at different time points before stimulus onset have distinct functional implications or whether these reflect distinct cognitive processes.

Furthermore, contamination of the gamma band of the human scalp EEG by nonneural signals is a major and long-standing concern (Whitham et al., 2007). Different sources of contamination were discussed (Nottage & Horder, 2015). To address potential contamination by power line noise, we kept impedance low and balanced across electrodes. In addition, EEG recording was carried out in an electrically shielded laboratory, and active electrodes having an amplifier incorporated into each electrode (BioSemi) were used. Because power line noise during data acquisition often cannot reduce the power line interference completely (Nottage & Horder, 2015), we also applied spectrum interpolation to remove potential power line noise from EEG data (Leske & Dalal, 2019). Furthermore, we removed muscular artifacts to correct for blinks and eye movements via independent component analysis.

Finally, it is to be noted that recognition tests are typically associated with a rather higher hit rate. This was also the case in this study. Our EEG analyses were thus based on a relative imbalance of hits and misses. Although additional analyses that included only participants with a minimum number of misses resulted largely in a comparable pattern of results as the analysis of the full sample, effects of high gamma power on subsequent memory could not be identified consistently. Therefore, a more balanced number of hits and misses may be beneficial, proving the reliability of our findings. Future studies might achieve this by extending the interval between encoding and memory test.

In summary, the present findings indicate that acute stress changes the neural context for building new memories. In particular, stress appeared to alter specifically the neural context for encoding emotionally arousing material. Such changes in the neural activity preceding a stimulus might represent a so far unknown mechanism through which stress tunes the organism toward the preferential encoding of emotionally salient cues, thus fostering adaptation to similar future events on the one hand but contributing to the aberrant emotional memory characteristic for stress-related psychopathologies on the other hand.

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REFERENCES

Agresti, A., & Caffo, B. (2000). Simple and effective confidence intervals for proportions and differences of proportions result from adding two successes and two failures. *American Statistician*, *54*, 280–288. **DOI:** https://doi.org/10.2307/2685779, https://doi.org/10.1080/00031305.2000.10474560

Beck, A. T., Ward, C. H., Mendelson, M., Mock, J., & Erbaugh, J. (1961). An inventory for measuring depression. *Archives of General Psychiatry*, *4*, 561–571. **DOI:** https://doi.org/10.1001/archpsyc.1961.01710120031004, **PMID:** 13688369

Bryant, R. A., McGrath, C., & Felmingham, K. L. (2013). The roles of noradrenergic and glucocorticoid activation in the development of intrusive memories. *PLoS One*, *8*, e62675. **DOI:** https://doi.org/10.1371/journal.pone.0062675, **PMID:** 23658640, **PMCID:** PMC3639204

Buchanan, T. W., & Lovallo, W. R. (2001). Enhanced memory for emotional material following stress-level cortisol treatment in humans. *Psychoneuroendocrinology*, *26*, 307–317. **DOI:** https://doi.org/10.1016/S0306-4530(00)00058-5

Buchanan, T. W., Tranel, D., & Adolphs, R. (2006). Impaired memory retrieval correlates with individual differences in cortisol response but not autonomic response. *Learning & Memory*, *13*, 382–387. **DOI:** https://doi.org/10.1101/lm.206306, **PMID:** 16741288, **PMCID:** PMC1475821

Burke, J. F., Zaghloul, K. A., Jacobs, J., Williams, R. B., Sperling, M. R., Sharan, A. D., et al. (2013). Synchronous and asynchronous theta and gamma activity during episodic memory formation. *Journal of Neuroscience*, *33*, 292–304. **DOI:** https://doi.org/10.1523/JNEUROSCI.2057-12.2013, **PMID:** 23283342, **PMCID:** PMC3711714

Cahill, L., Gorski, L., & Le, K. (2003). Enhanced human memory consolidation with post-learning stress: Interaction with the degree of arousal at encoding. *Learning & Memory*, *10*, 270–274. **DOI:** https://doi.org/10.1101/lm.62403, **PMID:** 12888545, **PMCID:** PMC202317

Cahill, L., Prins, B., Weber, M., & McGaugh, J. L. (1994). β-adrenergic activation and memory for emotional events. *Nature*, *371*, 702–704. **DOI:** https://doi.org/10.1038/371702a0, **PMID:** 7935815

Canolty, R. T., & Knight, R. T. (2010). The functional role of cross-frequency coupling. *Trends in Cognitive Sciences*, *14*, 506–515. **DOI:** https://doi.org/10.1016/j.tics.2010.09.001, **PMID:** 20932795, **PMCID:** PMC3359652

Christianson, S.-Å. (Ed.) (1992). *The handbook of emotion and memory: Research and theory*. Hillsdale, NJ: Erlbaum.

Cohen, M. X. (2014). *Analyzing neural time series data: Theory and practice*. Cambridge, MA: MIT Press. **DOI:** https://doi.org/10.7551/mitpress/9609.001.0001

Cohen, N., Pell, L., Edelson, M. G., Ben-Yakov, A., Pine, A., & Dudai, Y. (2015). Peri-encoding predictors of memory encoding and consolidation. *Neuroscience & Biobehavioral Reviews*, *50*, 128–142. **DOI:** https://doi.org/10.1016/j.neubiorev.2014.11.002, **PMID:** 25446944

Dalgleish, T., & Watts, F. N. (1990). Biases of attention and memory in disorders of anxiety and depression. *Clinical Psychology Review*, *10*, 589–604. **DOI:** https://doi.org/10.1016/0272-7358(90)90098-U

Davachi, L., Mitchell, J. P., & Wagner, A. D. (2003). Multiple routes to memory: Distinct medial temporal lobe processes build item and source memories. *Proceedings of the National Academy of Sciences, U.S.A.*, *100*, 2157–2162. **DOI:** https://doi.org/10.1073/pnas.0337195100, **PMID:** 12578977, **PMCID:** PMC149975

Delorme, A., & Makeig, S. (2004). EEGLAB: An open source toolbox for analysis of single-trial EEG dynamics including independent component analysis. *Journal of Neuroscience Methods*, *134*, 9–21. **DOI:** https://doi.org/10.1016/j.jneumeth.2003.10.009, **PMID:** 15102499

de Quervain, D. J.-F., Roozendaal, B., & McGaugh, J. L. (1998). Stress and glucocorticoids impair retrieval of long-term spatial memory. *Nature*, *394*, 787–790. **DOI:** https://doi.org/10.1038/29542, **PMID:** 9723618

de Quervain, D. J.-F., Schwabe, L., & Roozendaal, B. (2017). Stress, glucocorticoids and memory: Implications for treating fear-related disorders. *Nature Reviews Neuroscience*, *18*, 7–19. **DOI:** https://doi.org/10.1038/nrn.2016.155, **PMID:** 27881856

Dolcos, F., Katsumi, Y., Moore, M., Berggren, N., de Gelder, B., Derakshan, N., et al. (2020). Neural correlates of emotion–attention interactions: From perception, learning, and memory to social cognition, individual differences, and training interventions. *Neuroscience & Biobehavioral Reviews*, *108*, 559–601. **DOI:** https://doi.org/10.1016/j.neubiorev.2019.08.017, **PMID:** 31446010

Dolcos, F., Katsumi, Y., Weymar, M., Moore, M., Tsukiura, T., & Dolcos, S. (2017). Emerging directions in emotional episodic memory. *Frontiers in Psychology*, *8*, 1867. **DOI:** https://doi.org/10.3389/fpsyg.2017.01867, **PMID:** 29255432, **PMCID:** PMC5723010

Dvorak, D., & Fenton, A. A. (2014). Toward a proper estimation of phase–amplitude coupling in neural oscillations. *Journal of Neuroscience Methods*, *225*, 42–56. **DOI:** https://doi.org/10.1016/j.jneumeth.2014.01.002, **PMID:** 24447842, **PMCID:** PMC3955271

Elzinga, B. M., Bakker, A., & Bremner, J. D. (2005). Stress-induced cortisol elevations are associated with impaired delayed, but not immediate recall. *Psychiatric Research*, *134*, 211–223. **DOI:** https://doi.org/10.1016/j.psychres.2004.11.007, **PMID:** 15892980

Engel, A. K., Fries, P., & Singer, W. (2001). Dynamic predictions: Oscillations and synchrony in top–down processing. *Nature Reviews Neuroscience*, *2*, 704–716. **DOI:** https://doi.org/10.1038/35094565, **PMID:** 11584308

Fair, D. A., Cohen, A. L., Dosenbach, N. U. F., Church, J. A., Miezin, F. M., Barch, D. M., et al. (2008). The maturing architecture of the brain's default network. *Proceedings of the National Academy of Sciences, U.S.A.*, *105*, 4028–4032. **DOI:** https://doi.org/10.1073/pnas.0800376105, **PMID:** 18322013, **PMCID:** PMC2268790

Fell, J., Ludowig, E., Staresina, B. P., Wagner, T., Kranz, T., Elger, C. E., et al. (2011). Medial temporal theta/alpha power enhancement precedes successful memory encoding: Evidence based on intracranial EEG. *Journal of Neuroscience*, *31*, 5392–5397. **DOI:** https://doi.org/10.1523/JNEUROSCI.3668-10.2011, **PMID:** 21471374, **PMCID:** PMC6622710

Fellner, M.-C., Bäuml, K.-H. T., & Hanslmayr, S. (2013). Brain oscillatory subsequent memory effects differ in power and long-range synchronization between semantic and survival processing. *Neuroimage*, *79*, 361–370. **DOI:** https://doi.org/10.1016/j.neuroimage.2013.04.121, **PMID:** 23664950

Flaisch, T., Stockburger, J., & Schupp, H. T. (2008). Affective prime and target picture processing: An ERP analysis of early and late interference effects. *Brain Topography*, *20*, 183–191. **DOI:** https://doi.org/10.1007/s10548-008-0045-6, **PMID:** 18335309

Goldfarb, E. V., Tompary, A., Davachi, L., & Phelps, E. A. (2019). Acute stress throughout the memory cycle: Diverging effects on associative and item memory. *Journal of Experimental Psychology: General*, *148*, 13–29. **DOI:** https://doi.org/10.1037/xge0000472, **PMID:** 30221962, **PMCID:** PMC6312485

Guderian, S., Schott, B. H., Richardson-Klavehn, A., & Düzel, E. (2009). Medial temporal theta state before an event predicts episodic encoding success in humans. *Proceedings of the National Academy of Sciences, U.S.A.*, *106*, 5365–5370. **DOI:** https://doi.org/10.1073/pnas.0900289106, **PMID:** 19289818, **PMCID:** PMC2663999

Hannula, D. E., & Ranganath, C. (2008). Medial temporal lobe activity predicts successful relational memory binding. *Journal of Neuroscience*, *28*, 116–124. **DOI:** https://doi.org/10.1523/JNEUROSCI.3086-07.2008, **PMID:** 18171929, **PMCID:** PMC2748793

Herawan, T., Deris, M. M., & Abawajy, J. H. (Eds.) (2014). Lecture notes in electrical engineering. In *Proceedings of the First International Conference on Advanced Data and Information Engineering (DaEng-2013)*. Piscataway, NJ: IEEE. **DOI:** https://doi.org/10.1007/978-981-4585-18-7

Hermans, E. J., Henckens, M. J. A. G., Joëls, M., & Fernández, G. (2014). Dynamic adaptation of large-scale brain networks in response to acute stressors. *Trends in Neurosciences*, *37*, 304–314. **DOI:** https://doi.org/10.1016/j.tins.2014.03.006, **PMID:** 24766931

Hermans, E. J., van Marle, H. J. F., Ossewaarde, L., Henckens, M. J. A. G., Qin, S., van Kesteren, M. T. R., et al. (2011). Stress-related noradrenergic activity prompts large-scale neural network reconfiguration. *Science*, *334*, 1151–1153. **DOI:** https://doi.org/10.1126/science.1209603, **PMID:** 22116887

Herrmann, C. S., Strüber, D., Helfrich, R. F., & Engel, A. K. (2016). EEG oscillations: From correlation to causality. *International Journal of Psychophysiology*, *103*, 12–21. **DOI:** https://doi.org/10.1016/j.ijpsycho.2015.02.003, **PMID:** 25659527

Hyman, S. E. (2005). Addiction: A disease of learning and memory. *American Journal of Psychiatry*, *162*, 1414–1422. **DOI:** https://doi.org/10.1176/appi.ajp.162.8.1414, **PMID:** 16055762

Jamalabadi, H., Alizadeh, S., Schönauer, M., Leibold, C., & Gais, S. (2016). Classification based hypothesis testing in neuroscience: Below-chance level classification rates and overlooked statistical properties of linear parametric classifiers. *Human Brain Mapping*, *37*, 1842–1855. **DOI:** https://doi.org/10.1002/hbm.23140, **PMID:** 27015748, **PMCID:** PMC6867424

Joëls, M., Fernandez, G., & Roozendaal, B. (2011). Stress and emotional memory: A matter of timing. *Trends in Cognitive Sciences*, *15*, 280–288. **DOI:** https://doi.org/10.1016/j.tics.2011.04.004, **PMID:** 21571575

Joëls, M., Pu, Z., Wiegert, O., Oitzl, M. S., & Krugers, H. J. (2006). Learning under stress: How does it work? *Trends in Cognitive Sciences*, *10*, 152–158. **DOI:** https://doi.org/10.1016/j.tics.2006.02.002, **PMID:** 16513410

Kensinger, E. A. (2007). Negative emotion enhances memory accuracy: Behavioral and Neuroimaging evidence. *Current Directions in Psychological Science*, *16*, 213–218. **DOI:** https://doi.org/10.1111/j.1467-8721.2007.00506.x

Klimesch, W., Schack, B., & Sauseng, P. (2005). The functional significance of theta and upper alpha oscillations. *Experimental Psychology*, *52*, 99–108. **DOI:** https://doi.org/10.1027/1618-3169.52.2.99, **PMID:** 15850157

Kohavi, R. (1995). A study of cross-validation and bootstrap for accuracy estimation and model selection. In *Proceedings of the 14th International Joint Conference on Articial Intelligence* (pp. 1137–1143). San Francisco: Morgan Kaufmann Publishers.

Kriegeskorte, N., Goebel, R., & Bandettini, P. (2006). Information-based functional brain mapping. *Proceedings of the National Academy of Sciences, U.S.A.*, *103*, 3863–3868. **DOI:** https://doi.org/10.1073/pnas.0600244103, **PMID:** 16537458, **PMCID:** PMC1383651

Kunde, W., & Mauer, N. (2008). Sequential modulations of valence processing in the emotional Stroop task. *Experimental Psychology*, *55*, 151–156. **DOI:** https://doi.org/10.1027/1618-3169.55.3.151, **PMID:** 18549161

Lang, P. J., Bradley, M. M., & Cuthbert, B. N. (2008). *International Affective Picture System (IAPS): Affective ratings of pictures and instruction manual*. Technical Report A-8. Gainesville: University of Florida.

Lega, B. C., Jacobs, J., & Kahana, M. (2012). Human hippocampal theta oscillations and the formation of episodic memories. *Hippocampus*, *22*, 748–761. **DOI:** https://doi.org/10.1002/hipo.20937, **PMID:** 21538660

Leske, S., & Dalal, S. S. (2019). Reducing power line noise in EEG and MEG data via spectrum interpolation. *Neuroimage*, *189*, 763–776. **DOI:** https://doi.org/10.1016/j.neuroimage.2019.01.026, **PMID:** 30639330, **PMCID:** PMC6456018

Lisman, J. (2005). The theta/gamma discrete phase code occuring during the hippocampal phase precession may be a more general brain coding scheme. *Hippocampus*, *15*, 913–922. **DOI:** https://doi.org/10.1002/hipo.20121, **PMID:** 16161035

Makeig, S., Bell, A. J., Jung, T.-P., & Sejnowski, T. J. (1996). Advances in neural information processing systems. In D. Touretzky, M. Mozer, & M. Hasselmo (Eds.), *Independent component analysis of electroencephalographic data* (pp. 145–151). Cambridge, MA: MIT Press.

Márton, C. D., Fukushima, M., Camalier, C. R., Schultz, S. R., & Averbeck, B. B. (2019). Distinct patterns for top–down and bottom–up information processing via cross-frequency coupling in macaque auditory cortex. *eNeuro*, *6*, ENEURO.0467-18.2019. **DOI:** https://doi.org/10.1523/ENEURO.0467-18.2019, **PMID:** 31088914, **PMCID:** PMC6520641

McGaugh, J. L., Cahill, L., & Roozendaal, B. (1996). Involvement of the amygdala in memory storage: Interaction with other brain systems. *Proceedings of the National Academy of Sciences, U.S.A.*, *93*, 13508–13514. **DOI:** https://doi.org/10.1073/pnas.93.24.13508, **PMID:** 8942964, **PMCID:** PMC33638

Morillas-Romero, A., Tortella-Feliu, M., Bornas, X., & Putman, P. (2015). Spontaneous EEG theta/beta ratio and delta–beta coupling in relation to attentional network functioning and self-reported attentional control. *Cognitive, Affective & Behavioral Neuroscience*, *15*, 598–606. **DOI:** https://doi.org/10.3758/s13415-015-0351-x, **PMID:** 25860658

Müller-Putz, G. R., Scherer, R., Brunner, C., Leeb, R., & Pfurtscheller, G. (2008). Better than random? A closer look on BCI results. *International Journal of Bioelectromagnetism*, *10*, 52–55.

Murphy, B., Poesio, M., Bovolo, F., Bruzzone, L., Dalponte, M., & Lakany, H. (2011). EEG decoding of semantic category reveals distributed representations for single concepts. *Brain and Language*, *117*, 12–22. **DOI:** https://doi.org/10.1016/j.bandl.2010.09.013, **PMID:** 21300399

Noh, E., Herzmann, G., Curran, T., & de Sa, V. R. (2014). Using single-trial EEG to predict and analyze subsequent memory. *Neuroimage*, *84*, 712–723. **DOI:** https://doi.org/10.1016/j.neuroimage.2013.09.028, **PMID:** 24064073, **PMCID:** PMC3874086

Nottage, J. F., & Horder, J. (2015). State-of-the-art analysis of high-frequency (gamma range) electroencephalography in humans. *Neuropsychobiology*, *72*, 219–228. **DOI:** https://doi.org/10.1159/000382023, **PMID:** 26900860

Oostenveld, R., Fries, P., Maris, E., & Schoffelen, J.-M. (2011). FieldTrip: Open source software for advanced analysis of MEG, EEG, and invasive electrophysiological data. *Computational Intelligence and Neuroscience*, *2011*, 156869. **DOI:** https://doi.org/10.1155/2011/156869, **PMID:** 21253357, **PMCID:** PMC3021840

Otten, L. J., Quayle, A. H., Akram, S., Ditewig, T. A., & Rugg, M. D. (2006). Brain activity before an event predicts later recollection. *Nature Neuroscience*, *9*, 489–491. **DOI:** https://doi.org/10.1038/nn1663, **PMID:** 16501566

Pitman, R. K., Rasmusson, A. M., Koenen, K. C., Shin, L. M., Orr, S. P., Gilbertson, M. W., et al. (2012). Biological studies of post-traumatic stress disorder. *Nature Reviews Neuroscience*, *13*, 769–787. **DOI:** https://doi.org/10.1038/nrn3339, **PMID:** 23047775, **PMCID:** PMC4951157

Qin, S., Hermans, E. J., van Marle, H. J. F., Luo, J., & Fernández, G. (2009). Acute psychological stress reduces working memory-related activity in the dorsolateral prefrontal cortex. *Biological Psychiatry*, *66*, 25–32. **DOI:** https://doi.org/10.1016/j.biopsych.2009.03.006, **PMID:** 19403118

Quaedflieg, C. W. E. M., Schwabe, L., Meyer, T., & Smeets, T. (2013). Time dependent effects of stress prior to encoding on event-related potentials and 24 h delayed retrieval. *Psychoneuroendocrinology*, *38*, 3057–3069. **DOI:** https://doi.org/10.1016/j.psyneuen.2013.09.002, **PMID:** 24074803

Roozendaal, B., Hahn, E. L., Nathan, S. V., de Quervain, D. J.-F., & McGaugh, J. L. (2004). Glucocorticoid effects on memory retrieval require concurrent noradrenergic activity in the hippocampus and basolateral amygdala. *Journal of Neuroscience*, *24*, 8161–8169. **DOI:** https://doi.org/10.1523/JNEUROSCI.2574-04.2004, **PMID:** 15371517, **PMCID:** PMC6729799

Roozendaal, B., McEwen, B. S., & Chattarji, S. (2009). Stress, memory and the amygdala. *Nature Reviews Neuroscience*, *10*, 423–433. **DOI:** https://doi.org/10.1038/nrn2651, **PMID:** 19469026

Roozendaal, B., & McGaugh, J. L. (2011). Memory modulation. *Behavioral Neuroscience*, *125*, 797–824. **DOI:** https://doi.org/10.1037/a0026187, **PMID:** 22122145, **PMCID:** PMC3236701

Roozendaal, B., Okuda, S., de Quervain, D. J.-F., & McGaugh, J. L. (2006). Glucocorticoids interact with emotion-induced noradrenergic activation in influencing different memory functions. *Neuroscience*, *138*, 901–910. **DOI:** https://doi.org/10.1016/j.neuroscience.2005.07.049, **PMID:** 16310958

Schneider, S. L., & Rose, M. (2016). Intention to encode boosts memory-related pre-stimulus EEG beta power. *Neuroimage*, *125*, 978–987. **DOI:** https://doi.org/10.1016/j.neuroimage.2015.11.024, **PMID:** 26584862

Scholz, S., Schneider, S. L., & Rose, M. (2017). Differential effects of ongoing EEG beta and theta power on memory formation. *PLoS One*, *12*, e0171913. **DOI:** https://doi.org/10.1371/journal.pone.0171913, **PMID:** 28192459, **PMCID:** PMC5305097

Schulz, P., Schlotz, W., & Becker, P. (2004). *Trierer Inventar Zum Chronischen Stress (TICS)*. Göttingen, Germany: Hogrefe.
Schwabe, L. (2017). Memory under stress: From single systems to network changes. *European Journal of Neuroscience*, *45*, 478–489. **DOI:** https://doi.org/10.1111/ejn.13478, **PMID:** 27862513
Schwabe, L., Bohringer, A., Chatterjee, M., & Schächinger, H. (2008). Effects of pre-learning stress on memory for neutral, positive and negative words: Different roles of cortisol and autonomic arousal. *Neurobiology of Learning and Memory*, *90*, 44–53. **DOI:** https://doi.org/10.1016/j.nlm.2008.02.002, **PMID:** 18334304
Schwabe, L., Haddad, L., & Schächinger, H. (2008). HPA axis activation by a socially evaluated cold-pressor test. *Psychoneuroendocrinology*, *33*, 890–895. **DOI:** https://doi.org/10.1016/j.psyneuen.2008.03.001, **PMID:** 18403130
Schwabe, L., Joëls, M., Roozendaal, B., Wolf, O. T., & Oitzl, M. S. (2012). Stress effects on memory: An update and integration. *Neuroscience & Biobehavioral Reviews*, *36*, 1740–1749. **DOI:** https://doi.org/10.1016/j.neubiorev.2011.07.002, **PMID:** 21771612
Schwabe, L., & Schächinger, H. (2018). Ten years of research with the socially evaluated cold pressor test: Data from the past and guidelines for the future. *Psychoneuroendocrinology*, *92*, 155–161. **DOI:** https://doi.org/10.1016/j.psyneuen.2018.03.010, **PMID:** 29573884
Schwabe, L., & Wolf, O. T. (2012). Stress modulates the engagement of multiple memory systems in classification learning. *Journal of Neuroscience*, *32*, 11042–11049. **DOI:** https://doi.org/10.1523/JNEUROSCI.1484-12.2012, **PMID:** 22875937, **PMCID:** PMC6621021
Segal, S. K., Simon, R., McFarlin, S., Alkire, M., Desai, A., & Cahill, L. F. (2014). Glucocorticoids interact with noradrenergic activation at encoding to enhance long-term memory for emotional material in women. *Neuroscience*, *277*, 267–272. **DOI:** https://doi.org/10.1016/j.neuroscience.2014.06.059, **PMID:** 25010401
Shields, G. S., Sazma, M. A., McCullough, A. M., & Yonelinas, A. P. (2017). The effects of acute stress on episodic memory: A meta-analysis and integrative review. *Psychological Bulletin*, *143*, 636–675. **DOI:** https://doi.org/10.1037/bul0000100, **PMID:** 28368148, **PMCID:** PMC5436944
Spielberger, C. D., Gorsuch, R. L., & Lushene, R. E. (1970). *Manual for the state-trait anxiety inventory*. Palo Alto, CA: Consulting Psychologists Press.
Squire, L. R., & Zola-Morgan, S. (1991). The medial temporal lobe memory system. *Science*, *253*, 1380–1386. **DOI:** https://doi.org/10.1126/science.1896849, **PMID:** 1896849
Staudigl, T., & Hanslmayr, S. (2013). Theta oscillations at encoding mediate the context-dependent nature of human episodic memory. *Current Biology*, *23*, 1101–1106. **DOI:** https://doi.org/10.1016/j.cub.2013.04.074, **PMID:** 23746635
Steyer, R., Schwenkmezger, P., Notz, P., & Eid, M. (1994). Testtheoretische analysen des Mehrdimensionalen Befindlichkeitsfragebogen (MDBF) [Theoretical analysis of a multidimensional mood questionnaire (MDBF)]. *Diagnostica*, *40*, 320–328.
Sweeney-Reed, C. M., Zaehle, T., Voges, J., Schmitt, F. C., Buentjen, L., Kopitzki, K., et al. (2015). Thalamic theta phase alignment predicts human memory formation and anterior thalamic cross-frequency coupling. *eLife*, *4*, e07578. **DOI:** https://doi.org/10.7554/eLife.07578, **PMID:** 25993559, **PMCID:** PMC4459033
Tambini, A., Rimmele, U., Phelps, E. A., & Davachi, L. (2017). Emotional brain states carry over and enhance future memory formation. *Nature Neuroscience*, *20*, 271–278. **DOI:** https://doi.org/10.1038/nn.4468, **PMID:** 28024158
van Ast, V. A., Cornelisse, S., Meeter, M., Joëls, M., & Kindt, M. (2013). Time-dependent effects of cortisol on the contextualization of emotional memories. *Biological Psychiatry*, *74*, 809–816. **DOI:** https://doi.org/10.1016/j.biopsych.2013.06.022, **PMID:** 23972529
Vogel, S., Fernández, G., Joëls, M., & Schwabe, L. (2016). Cognitive adaptation under stress: A case for the mineralocorticoid receptor. *Trends in Cognitive Sciences*, *20*, 192–203. **DOI:** https://doi.org/10.1016/j.tics.2015.12.003, **PMID:** 26803208
Weymar, M., Schwabe, L., Löw, A., & Hamm, A. O. (2012). Stress sensitizes the brain: Increased processing of unpleasant pictures after exposure to acute stress. *Journal of Cognitive Neuroscience*, *24*, 1511–1518. **DOI:** https://doi.org/10.1162/jocn_a_00174, **PMID:** 22185496
Whitham, E. M., Pope, K. J., Fitzgibbon, S. P., Lewis, T., Clark, C. R., Loveless, S., et al. (2007). Scalp electrical recording during paralysis: Quantitative evidence that EEG frequencies above 20 Hz are contaminated by EMG. *Clinical Neurophysiology*, *118*, 1877–1888. **DOI:** https://doi.org/10.1016/j.clinph.2007.04.027, **PMID:** 17574912
Wirkner, J., Ventura-Bort, C., Schulz, P., Hamm, A. O., & Weymar, M. (2018). Event-related potentials of emotional and neutral memories: The role of encoding position and delayed testing. *Psychophysiology*, *55*, e13069. **DOI:** https://doi.org/10.1111/psyp.13069, **PMID:** 29457220
Wirz, L., Wacker, J., Felten, A., Reuter, M., & Schwabe, L. (2017). A deletion variant of the α2b-adrenoceptor modulates the stress-induced shift from "cognitive" to "habit" memory. *Journal of Neuroscience*, *37*, 2149–2160. **DOI:** https://doi.org/10.1523/JNEUROSCI.3507-16.2017, **PMID:** 28115477, **PMCID:** PMC6705683
Zoladz, P. R., Clark, B., Warnecke, A., Smith, L., Tabar, J., & Talbot, J. N. (2011). Pre-learning stress differentially affects long-term memory for emotional words, depending on temporal proximity to the learning experience. *Physiology & Behavior*, *103*, 467–476. **DOI:** https://doi.org/10.1016/j.physbeh.2011.01.016, **PMID:** 21262248