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Stress and the medial temporal lobe at rest: Functional connectivity is associated with both memory and cortisol

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ABSTRACT

When acute stress is experienced immediately after memory encoding (i.e., post-encoding stress) it can significantly impact subsequent memory for that event. For example, recent work has suggested that post-encoding stress occurring in a different context from encoding impairs memory. However, the neural processes underlying these effects are poorly understood. We aimed to expand this understanding by conducting an analysis of resting functional connectivity in the period following post-encoding stress that occurred in a different context than encoding, using seed regions in the medial temporal lobes known for their roles in memory. In the current study of 44 males randomized to stress (n = 23) or control (n = 21) groups, we found that stress increased cortisol, impaired recollection of neutral materials, and altered functional connectivity with medial temporal lobe regions. Although stress did not significantly alter hippocampus-amygdala functional connectivity, relative to participants in the control group, participants in the post-encoding stress group showed lower functional connectivity between the hippocampus and a region with a peak in the superior temporal gyrus. Across participants in both groups, functional connectivity between these regions was related to greater increases in cortisol, and it was also inversely related to recollection of neutral materials. In contrast, the stress group showed greater parahippocampal cortex functional connectivity with a region in the left middle temporal gyrus than the control group. Moreover, greater functional connectivity between the parahippocampal cortex and the observed cluster in the middle temporal gyrus was associated with greater cortisol changes from pre- to post-manipulation, but was not related to differences in memory. The results show that post-encoding stress can alter the resting-state functional connectivity between the medial temporal lobe and neocortex, which may help explain how stress impacts memory.

1. Introduction

Many studies have shown that experiencing a stressful event can enhance retention of information learned before the event took place. Research in animal models and humans has motivated the hypothesis that such "post-encoding stress" effects (Cahill et al., 2003; McCullough and Yonelinas, 2013; Shields et al., 2017b) may be related to cellular memory consolidation mechanisms (Cahill et al., 2003; McGaugh, 2000; Schwabe et al., 2012). In particular, actions of stress-induced glucocorticoids in the hippocampus interact with stress- or arousal-induced changes in amygdala activity brought about by actions of norepinephrine to strengthen—or "consolidate"—memory traces (Joëls et al., 2011; McGaugh, 2000). This model has found extensive support in animal work and pharmacological manipulations, which have shown that glucocorticoids and norepinephrine can strengthen emotional memories through effects on the hippocampus and the amygdala (Joëls et al., 2011; Roozendaal et al., 2006; Schwabe et al., 2012). As such, the consolidation model has become the primary theoretical explanation for why post-encoding stress enhances memory.

Despite the success of the consolidation model in explaining post-encoding stress-induced enhancements of memory, it is clear that there are important boundary conditions that can fundamentally alter the effects of stress on memory. For instance, some studies have shown that the post-encoding stress effect is context-dependent (Sazma et al., 2019; Shields et al., 2017b; Trammell and Clore, 2014). That is, post-encoding stress that occurs in a different spatial context than encoding does not benefit memory and may in fact hurt it. For example, in a recent functional magnetic resonance imaging (fMRI) study, we found that

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post-encoding stress that occurred in a different context from the encoding task led to reductions in recollection of studied information (McCullough et al., 2015; Ritchey et al., 2017). The standard cellular consolidation model cannot explain post-encoding stress-induced impairments in memory, suggesting that there may be additional neural mechanisms that affect the relationship between post-encoding stress and memory performance.

Understanding stress-induced differences in whole-brain functional connectivity with brain regions supporting memory may be crucial to understanding the mechanisms underlying post-encoding stress effects on memory. For example, the hippocampus closely interacts with at least two distinct memory networks (Ranganath and Ritchey, 2012; Ritchey et al., 2015a) during memory formation, consolidation, and retrieval, and stress alters functional connectivity between numerous neural networks (Hermans et al., 2014; Quaedflieg et al., 2015; Soares et al., 2013). These findings suggest the possibility that post-encoding stress might alter communication within or between regions important for memory consolidation including, but not limited to, the hippocampus and amygdala (though see de Voogd et al., 2017). Thus, stress-induced differences in whole-brain functional connectivity with medial temporal lobe regions that support memory may be important mechanisms underpinning the effects of post-encoding stress on memory; to date, however, no study has examined this possibility.

Here, we tested how post-encoding stress modulated functional connectivity with memory-related brain regions in the medial temporal lobes relative to a control group. In the current study, we randomly assigned 44 participants to a stress or control task that took place after, and in a different context from, an incidental encoding task. Immediately following the stress or control manipulation, we scanned participants to obtain fMRI data during rest. We hypothesized that the stress manipulation would modulate seed-to-voxel functional connectivity with seeds in medial temporal lobe regions known for their roles in memory and stress effects on memory (i.e., the hippocampus, parahippocampal cortex, perirhinal cortex, and amygdala). Because both the hippocampus and the amygdala have been critically implicated in post-encoding stress effects in prior work (de Voogd et al., 2017; McGaugh, 2015), we expected the stress manipulation to influence whole-brain functional connectivity with both of these regions. Additionally, we expected the stress manipulation to influence functional connectivity with the perirhinal and parahippocampal cortices, as these regions play important roles in memory processes that are influenced by post-encoding stress (Diana et al., 2007; McCullough and Yonelinas, 2013; Sazma et al., 2019). Finally, we hypothesized that stress-induced alterations in functional connectivity with these regions would be associated with both changes in cortisol and memory performance.

2. Method

2.1. Participants

Fifty young adult males participated in this experiment. We included only male participants in this study due to prior work from our lab suggesting that the effects of post-encoding stress on memory are stronger in males (McCullough and Yonelinas, 2013). The behavioral data and the fMRI data from the encoding phase of this experiment have been published previously (McCullough et al., 2015; Ritchey et al., 2017). Incomplete data resulted in exclusion of four participants (one participant failed to return for the memory test, one received an incorrect version of the memory test involving re-presentation of studied items, and two others completed the test outside of the MRI scanner due to technical difficulties). Data from one additional participant was excluded from all analyses due to excessive motion in the MRI scanner. One final participant was excluded due to incidental finding of a structural brain abnormality. Exclusion of those six participants resulted in a final sample of 44 participants (23 stress, 21 control).

Table 1
Participant demographic information by condition.

	Stress Condition			Control Condition		
Variable	*Mean*	(*SD*)	*n*	*Mean*	(*SD*)	*n*
Age	24.1	(2.8)		23.2	(4.2)	
Years of Education	16.5	(2.6)		15.9	(2.4)	
Race/Ethnicity						
Asian			5			5
Hispanic			3			1
Non-Hispanic White			12			12
Other/Biracial			3			3

Note: All study participants were male.

Table 1 lists these participants' demographic characteristics.

One additional participant exhibited excessive motion in the MRI scanner during the pre-manipulation resting state scan but not the post-manipulation resting state scan. In the interest of retaining the largest possible sample size (i.e., $N = 44$), prior to conducting any analyses, we opted to examine the effects of post-encoding stress solely on the post-manipulation resting state functional scan for our primary analyses (because random assignment of participants to stress/control groups allows for causal inference without comparing pre- to post-manipulation). Nonetheless, results (presented within Supplemental Material) were similar when excluding this additional participant (i.e., $N = 43$) and examining Condition by Time interactions on functional connectivity, as well as the relations between connectivity and memory and cortisol changes.

2.2. Materials

The experiment used 312 images used in previous research (McCullough et al., 2015; McCullough and Yonelinas, 2013), selected primarily from the International Affective Picture System (Lang et al., 2008) as well as from an in-house set. Half of the images were emotionally arousing and half were neutral. Images were approximately 315 pixels square. Eight of the images were used as example trials: four were presented before the encoding and recognition tasks, two were presented only before encoding, and two were presented only before retrieval. In the encoding phase, 100 negative images and 100 neutral images were presented in a random order. In the recognition test, the 200 studied images and 104 new images (52 negative) were presented in a random order.

2.3. Procedure

Study procedures are depicted in Fig. 1. Each participant completed two experiment sessions on consecutive days. In the first session, participants provided a baseline saliva sample (approximately 42 min prior to the stressor) before being provided with instructions for the encoding task and subsequently entering the MRI scanner for the task. During encoding, participants viewed a series of sequentially-presented images (see above), which were removed from the screen after they were presented for 1000 ms. After each image was removed, participants were given up to 3000 ms to provide a judgment of "visual complexity" for the picture, using a 1–6 scale. The response window closed after a response was provided, and an intertrial interval that varied from 2000 ms to 8000 ms separated each trial from the next. The "visual complexity" ratings were obtained to ensure participants attended to each image in the incidental encoding paradigm; these data were not analyzed. Prior to the task, participants were provided brief instructions on how to rate the images and were shown examples of images "high" and "low" in visual complexity. Participants were not informed that their memory for the pictures would be tested the following day. Following the encoding task, a 7 min resting-state scan was conducted.

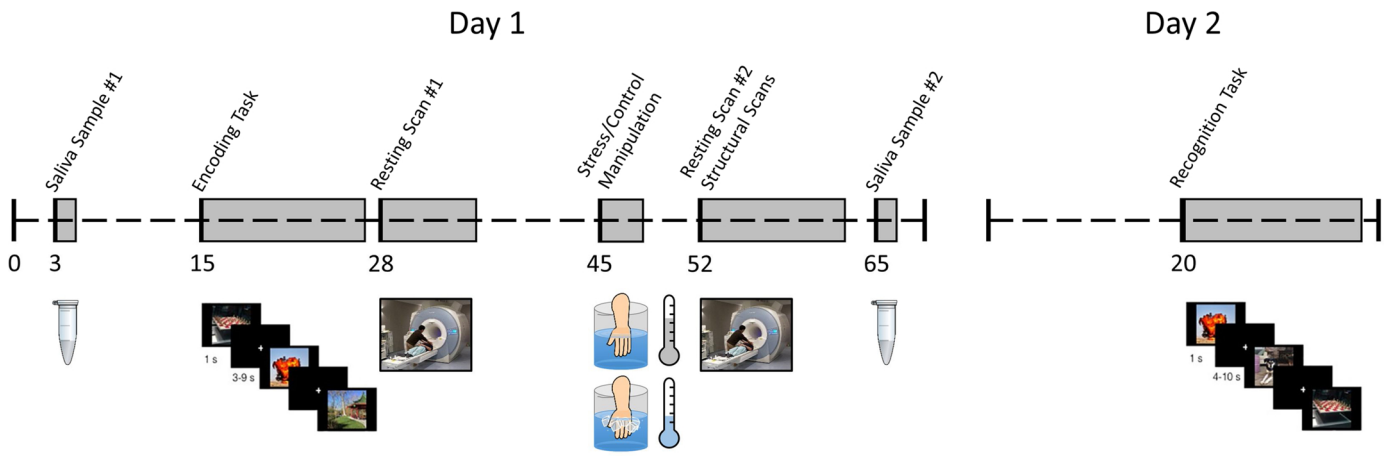

Fig. 1. Illustration of experimental procedure. Numbers listed along the dashed line represent the time the task began relative to the onset of the study (in minutes). Gray boxes illustrate approximately how long each task took to complete. Sections of the experiment containing elements irrelevant to the current study (e.g., filler questionnaires) are omitted from the figure for clarity.

Participants then exited the scanner and completed demographic surveys and personality questionnaires before completing either the cold-pressor test (stress group) or a control task. Participants in the stress group put and held their nondominant arm in ice water (M = 0.06 °C), whereas participants in the control group put and held their nondominant arm in lukewarm water (M = 23.71 °C). Each participant was instructed to keep his arm in the water for 3 min and to refrain from talking during the task. Participants then completed filler questionnaires unrelated to the present study before returning to the MRI scanner for another 7 min resting-state scan followed by a structural scan, then exited the scanner and provided another saliva sample (20 min post-stressor onset).

In the second session, which always began 24 h after the first, after acclimating to the laboratory, participants returned to the MR scanner, were presented with a randomized list of studied and new images, and were asked to rate their memory for each image. Participants rated each picture on a 1–5 or Recollect scale. Participants classified a picture as "Recollected" only if they could provide details regarding the earlier experience (though note that participants were not actually required to describe their recollective experience). Participants classified any picture that was not recollected on a 1–5 scale, where "5" expressed high confidence that the picture was studied (but without recollecting details about the experience), and "1" expressed high confidence that the picture was new.

A dual-process model of recognition memory was fit to the recognition memory data using standard confidence-based receiver operator characteristic (ROC) procedures (Yonelinas, 2002). In this model, participants are assumed to respond "old" to an old item if it is either recollected (R), or if the familiarity of the item exceeds the participant's response criterion (F_o) when the item is not recollected. Mathematically, then, Hits = $R + (1 - R)F_o$. Participants are assumed to respond "new" to a new item whenever the item's familiarity exceeds the participant's response criterion (F_n); mathematically, false alarms = F_n. Familiarity is assumed to be described by signal detection theory, which means that the proportion of old and new items that will be labeled "old" is equal to the proportion of those items that exceed the participant's response criterion at a given level of confidence. Mathematically, $F_o = \Phi(d'/2 - c_i)$ and $F_n = \Phi(-d'/2 - c_i)$, where d' is the distance between the old and new familiarity distributions. These equations can be combined into a single equation for each level of confidence, $p(\text{"old"}|\text{old})_i = R + (1 - R)\Phi(d'/2 - c_i) + p(\text{"old"}|\text{new})_i - \Phi(-d'/2 - c_i)$, and this equation was fit to each participant's observed ROC by minimizing the sum of squared errors, providing estimates of recollection (R) and familiarity (d'). Notably, quantifying recollection and familiarity using the Remember/Know procedure (with "Recollect" responses as Remember, and 4 and 5 responses as "Know") produced virtually identical results.

2.4. Cortisol assays

Saliva was assayed for salivary cortisol at the California National Primate Research Center at UC Davis in two batches using commercially available high-sensitivity ELISAs purchased from Salimetrics. The intra-assay coefficient of variation was 6.01%, and the inter-assay coefficient of variation was 11.30%. The minimum detectable value was 1.3854 nmol/L; one sample from a control participant fell below this threshold, so the minimum detectable value was substituted for that data point. Because of skew, cortisol values were log transformed prior to analyses. Residualized changes in cortisol were calculated by regressing log-transformed post-manipulation cortisol on log-transformed pre-manipulation cortisol. Residualized changes were used instead of simple change scores because residual change scores are more reliable than simple change scores (Cronbach and Furby, 1970). Analyses using simple change scores were similar: The association with hippocampal functional connectivity was stronger than that presented in the results below, whereas the association with parahippocampal functional connectivity was weaker.

2.5. MRI data acquisition and preprocessing

MRI data were acquired using a 3-T Siemens Skyra scanner equipped with a 32-channel head coil. Padding was used to minimize head motion, and earplugs were provided to attenuate acoustic noise from the scanner. At the beginning of each scan session, brief localizer scans were used to set the field of view (20.5 × 21.14 cm) to cover the entire brain except the inferior cerebellum and in some cases the most superior regions. Additionally, to correct for distortions due to magnetic field inhomogeneities, field maps were collected using Siemens field map sequence, with a short TE (4.92 ms) and a long TE (7.38 ms). Resting-state fMRI data were obtained in four runs (two runs during session 1, and two runs during session 2; only session 1 data are considered in this study) with a T2*-weighted gradient echo-planar imaging (EPI) sequence with the following parameters: repetition time, 2000 ms; echo time, 25 ms; flip angle, 90; matrix size, 64 × 66. Each volume consisted of 34 interleaved axial slices oriented parallel to the AC-PC line, with no inter-slice gap and a voxel size of 3.20 × 3.20 × 3.20 mm. Additionally, high-resolution T1-weighted structural images were acquired co-planar with the functional EPIs using a magnetization prepared rapid acquisition gradient echo (MPRAGE) sequence with an in-plane resolution of 1.0 × 1.0 mm^2

(field of view = 25.6 cm, matrix size = 256 × 256, 208 axial slices with 1.0 mm thickness).

Preprocessing of all MRI data was performed using Statistical Parametric Mapping (SPM8) software. Functional EPI data were corrected to account for timing differences in acquisition of adjacent slices using sinc interpolation, re-aligned to the first image using a six-parameter rigid-body transformation, and corrected for inhomogeneities in the magnetic field (i.e., unwarped) using the field map images. The high-resolution structural image was co-registered to the mean unwarped EPI. Normalization parameters were obtained by segmenting the co-registered T1 and applied to the T1 and functional EPIs, in order to normalize to the MNI template. Functional images were re-sliced to a resolution of 3 mm^3, and spatially smoothed with an isotropic 8 mm full-width half-maximum Gaussian kernel. The time series were inspected for sudden motion and rapid changes in global mean signal using the Artifact Detection Tools (ART; http://www.nitrc.org/projects/artifact_detect). Suspect time-points, defined as those marked by greater than 0.5 mm in movement or 1.5% global mean signal change were modeled out using nuisance regressors at the participant level. Additionally, participants were excluded if they exhibited > 3 mm motion in any direction throughout the scan session.

2.6. Functional connectivity analysis

Seed-to-voxel functional connectivity analyses of the post-manipulation resting state scan were conducted using the CONN toolbox v17.f (Whitfield-Gabrieli and Nieto-Castanon, 2012). After preprocessing, a band-pass filter of 0.008 Hz to 0.09 Hz was applied to the images, and motion was then regressed out. Seed-to-voxel analyses were conducted by computing the temporal correlation between the BOLD signals from a given seed to all other voxels in the brain. Physiological (including white matter and cerebrospinal fluid; CSF) and other noise reduction was implemented using a component-based noise reduction method known as the CompCor approach (Behzadi et al., 2007), which provides five principal components for both white matter and CSF to account for variability in their noise across the brain. Whole-brain BOLD signal was not included as a regressor in order to avoid obtaining incorrect anti-correlations (Murphy et al., 2009). The CompCor approach addresses the same concerns as those addressed by regressing out the global signal—in fact, CompCor accounts for 62% of the variance in the global signal (Yeo et al., 2015)—without risk of artificially introducing anticorrelations, thus making anticorrelations interpretable (Whitfield-Gabrieli and Nieto-Castanon, 2012). Moreover, specificity and sensitivity of positive correlations are better using the CompCor approach than the global signal regression method (Chai et al., 2012), and CompCor has been shown to be superior to global signal regression for discerning true group differences in functional connectivity (Saad et al., 2012; Shirer et al., 2015).

BOLD timeseries were first preprocessed and denoised, then spatially normalized to build the time series for each ROI and voxel. In preliminary ROI-to-ROI analyses, the BOLD timeseries for one ROI was correlated with the BOLD timeseries for the second ROI, and the resulting correlation was fisher transformed for analysis. In seed-to-voxel analyses, the BOLD timeseries for the seed was correlated with the timeseries of each voxel in the brain, and the resulting correlation was fisher transformed for analysis. Thresholds for significant functional connectivity of each seed with clusters of voxels were set to the recommended (i.e., CONN toolbox default) FDR-corrected $p < .05$ for cluster/size at $p < .001$ uncorrected for height/peak. Group differences were examined using a GLM controlling for motion and noise; seed-to-voxel comparisons between groups were thresholded at a whole-brain cluster-level FDR-corrected $p < .05$ at height/peak $p < .001$ uncorrected. Correlations presented in all functional connectivity analyses are partial correlations controlling for motion and noise.

We used four regions of interest as seeds for the seed-to-voxel analyses: the hippocampus, amygdala, parahippocampal cortex, and perirhinal cortex. These regions were chosen because they are known to play important roles in role in episodic memory and emotional memory modulation (Akirav and Richter-Levin, 2002; Diana et al., 2007; Eichenbaum et al., 2007; Ranganath et al., 2003; Yonelinas, 2002; Yonelinas and Ritchey, 2015). The amygdala ROI used is the mask provided in the FSL atlas, and is included as a default ROI within CONN toolbox. The ROIs used for the hippocampus, parahippocampal cortex, and perirhinal cortex were developed by Ritchey and colleagues (Ritchey et al., 2015b), and are available from NeuroVault (https://neurovault.org/collections/3731/). ROI masks warped to MNI space were applied to the preprocessed structural data in this study. Because we did not have any a priori hypotheses regarding differentiation of the hippocampus head, body, and tail, we combined those ROIs by averaging the signal across them, thereby making the hippocampus a single ROI for our analyses. Importantly, though, analyses conducted using the corresponding FSL atlas ROIs yielded identical results to those reported here. We did not have any hypotheses regarding laterality, so in all analyses we averaged across hemispheres to examine main effects of functional connectivity with these structures bilaterally. Prior to analysis, we planned to extract functional connectivity values between each of the four seed regions and any significant clusters of voxels throughout the brain, in order to use as predictors of memory and cortisol responses, given the known roles of the seed regions in memory and/or stress responsivity (Dedovic et al., 2009; Schwabe et al., 2012).

2.7. Data analysis

Between-groups functional connectivity analyses, which are described in Section 2.6, were conducted in CONN Toolbox, v.17f; relevant functional connectivity values were extracted from CONN Toolbox for use in other analyses. Extracted values were *z*-to-*r* transformed for reporting magnitudes of correlations. In all other analyses, connectivity values were retained as Fisher *z*-transformed correlations. All other analyses were conducted using R, version 3.5.1. Repeated measures ANOVA was used for analyses examining effects of stress on cortisol. Student's *t*-tests were used to examine the effects of stress on memory. Pearson correlations examined the magnitude of associations between functional connectivity values and both memory and changes in cortisol.

3. Results

3.1. Preliminary analyses

3.1.1. Effects of stress on cortisol

The behavioral results from a larger sample of participants in this study have been described in a previous publication (McCullough et al., 2015); nevertheless, we report effects of our stress manipulation on cortisol within the subset of participants included in this study (i.e., those with usable post-stress resting neuroimaging data). As expected, we found a significant Stress × Time interaction, $F(1, 42) = 9.91$, $p = .003$. Participants in the stress group ($M = 1.89$, $SE = 0.18$) did not differ in log-transformed cortisol from participants in the control group ($M = 1.63$, $SE = 0.19$) at baseline, $t(42) = 0.98$, $p = .333$, $d = 0.30$, whereas post-manipulation, participants in the stress group ($M = 2.37$, $SE = 0.17$) had significantly higher log-transformed cortisol levels than participants in the control group ($M = 1.28$, $SE = 0.18$), $t(42) = 4.35$, $p < .001$, $d = 1.31$.

3.1.2. Behavioral results

We also examined effects of our stress manipulation on memory performance within the subset of participants included in this study. Participants in the stress group showed significantly worse recollection of neutral images ($M = 0.12$, $SE = 0.03$) than participants in the control group ($M = 0.24$, $SE = 0.04$), $t(42) = -2.36$, $p = .023$, $d = -0.71$.

Participants in the stress group did not differ significantly from participants in the control group in recollection of negative images (p = .136, d = -0.45) or in familiarity of neutral or negative images (ps > .842, $|d|s$ < .07).

3.1.3. ROI analyses

Our first resting-state analyses characterized post-manipulation functional connectivity between the hippocampus and amygdala. Participants in the both groups showed strong functional connectivity between the hippocampus and the amygdala (Stress: $r(22)$ = .421, p < .001; Control: $r(20)$ = .415, p < .001). Surprisingly, however, a Z test of difference between fisher-transformed correlations showed that there were no differences between the stress and control groups in functional connectivity between the hippocampus and the amygdala, Z = .02, p = .982.

3.2. Whole-brain functional connectivity using MTL regions as seeds

We hypothesized that whole-brain functional connectivity with the hippocampus, parahippocampal cortex, perirhinal cortex, or amygdala would show stress-induced differences between groups. Complete functional connectivity profiles with these regions are presented in the Supplemental Material.

As hypothesized, we found that the stress and control groups differed in post-manipulation functional connectivity with the hippocampus. In particular, the stress and control group differed in functional connectivity between the hippocampus and a cluster of 269 voxels with a peak in the right superior temporal gyrus (peak coordinates x: 64, y: -14, z: 6), $p_{cluster}$ < .001 (corrected), p_{height} < .001. This cluster included portions of the central opercular cortex, planum temporale, Heschl's gyrus, and postcentral gyrus (Fig. 2a). Follow-up analyses showed that the stress group showed a significant inverse correlation between this cluster of voxels and the hippocampus, $r(22)$ = -.070, p = .006, whereas the control group showed a significant positive correlation, $r(20)$ = .088, p = .001 (see Fig. 2b).

Similarly, we found that the stress and control groups differed in functional connectivity between the parahippocampal cortex and a cluster of 123 voxels in the left middle temporal gyrus (peak coordinates x: -68, y: -44, z: -6), $p_{cluster}$ = .038 (corrected), p_{height} < .001 (Fig. 3a). In particular, the stress group showed a nonsignificant positive correlation between this cluster of voxels and the parahippocampal cortex, $r(22)$ = .036, p = .198, whereas the control group showed a significant inverse correlation, $r(20)$ = -.155, p < .001 (see Fig. 3b). Surprisingly, however, we found no significant differences between the stress and control groups in functional connectivity with the amygdala or perirhinal cortex as seeds.

To ensure that our observed differences were not due to pre-existing differences between the stress and control groups, we examined pre-manipulation resting functional connectivity with all four regions (i.e., connectivity before the stress or control task). As expected, functional connectivity between the seeds and the observed clusters was not significantly different between the groups pre-manipulation, ps > .632.

3.3. Correlations of functional connectivity with memory and cortisol

Our next analyses focused on the relationships between group differences in functional connectivity and both memory and cortisol. First, we extracted post-manipulation connectivity values between the hippocampus and the right temporal lobe region that showed significant manipulation-induced differences in hippocampal connectivity, as well as the connectivity values between the parahippocampal cortex and the left temporal lobe region that showed significant manipulation-induced differences in parahippocampal cortex connectivity. Next, we correlated these connectivity values with memory performance and cortisol change values.

We found that functional connectivity of the hippocampus with the cluster in the right temporal lobe was inversely correlated with changes in cortisol from pre- to post-manipulation (i.e., before and after stress/control), $r(42)$ = -.361, p = .016 (Fig. 4a), and positively associated with recollection estimates for neutral images, $r(42)$ = .349, p = .020 (Fig. 4b). We then conducted moderated regression analyses to examine whether the stress/control groups showed different associations between this hippocampal functional connectivity and recollection of neutral materials or changes in cortisol (i.e., Condition × Functional Connectivity interactions in predicting outcomes). We found that the association between this hippocampal functional connectivity with recollection of neutral images was not significantly different between groups, $t(40)$ = 0.58, p = .567; similarly, the association of hippocampal functional connectivity with changes in cortisol was not significantly different between groups, $t(40)$ = 1.28, p = .207. This functional connectivity of the hippocampus with the cluster in the right temporal lobe was not significantly associated with recollection estimates for negative images, $r(42)$ = .171, p = .267, or familiarity estimates for neutral or negative images, $|r|s$ < .09, ps > .564.

In contrast, functional connectivity of the parahippocampal cortex with the cluster in the left temporal lobe was positively associated with changes in cortisol from pre- to post-manipulation, $r(42)$ = .325, p = .032 (Fig. 5). This association did not differ between the stress and control group, $t(40)$ = 0.17, p = .868. Finally, functional connectivity of the parahippocampal cortex with the left temporal lobe cluster was

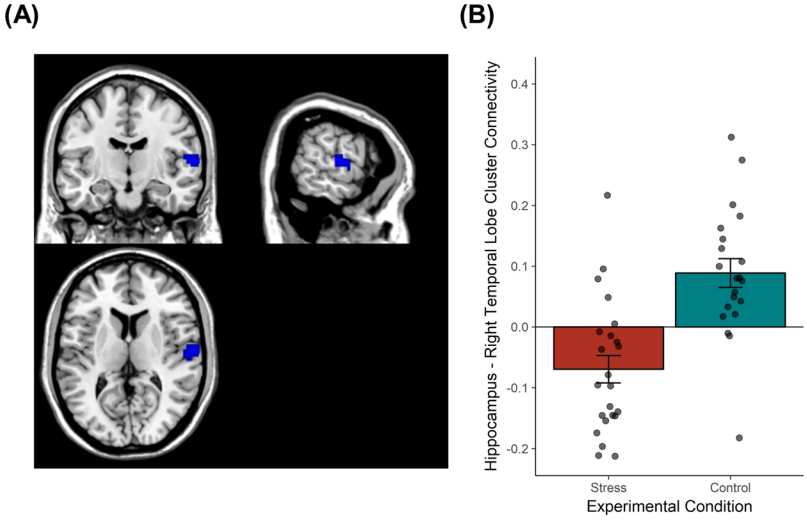

Fig. 2. Stress-induced differences in functional connectivity between the hippocampus and a cluster of voxels in the right temporal lobe with a peak in the superior temporal gyrus (thresholded at a whole-brain cluster-level FDR-corrected p < .05 at height/peak p < .001 uncorrected). Participants in the post-encoding stress group showed significant negative connectivity between the hippocampus and this area, whereas control participants evidenced significant positive connectivity.

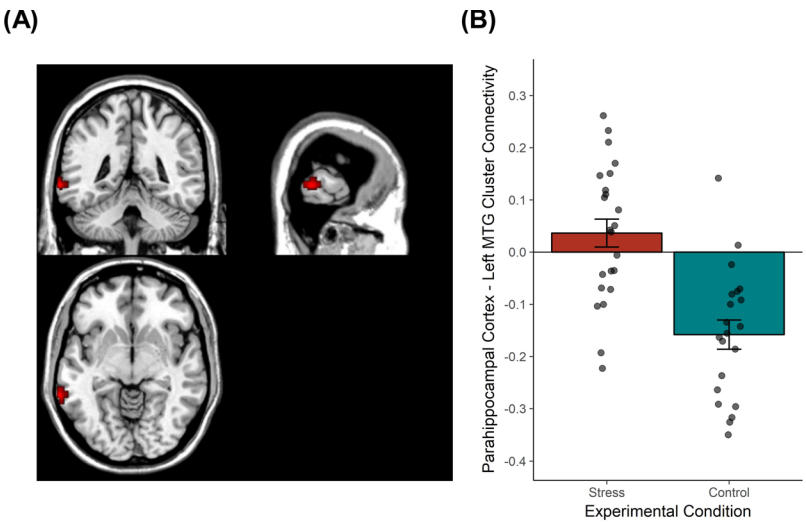

Fig. 3. Stress-induced differences in functional connectivity between the parahippocampal cortex and a cluster of voxels encompassed by the middle temporal gyrus (thresholded at a whole-brain cluster-level FDR-corrected $p < .05$ at height/peak $p < .001$ uncorrected). Participants in the stress group showed no significant connectivity between the parahippocampal cortex and this middle temporal gyrus cluster, whereas control participants evidenced a significant negative correlation (i.e., anticorrelation).

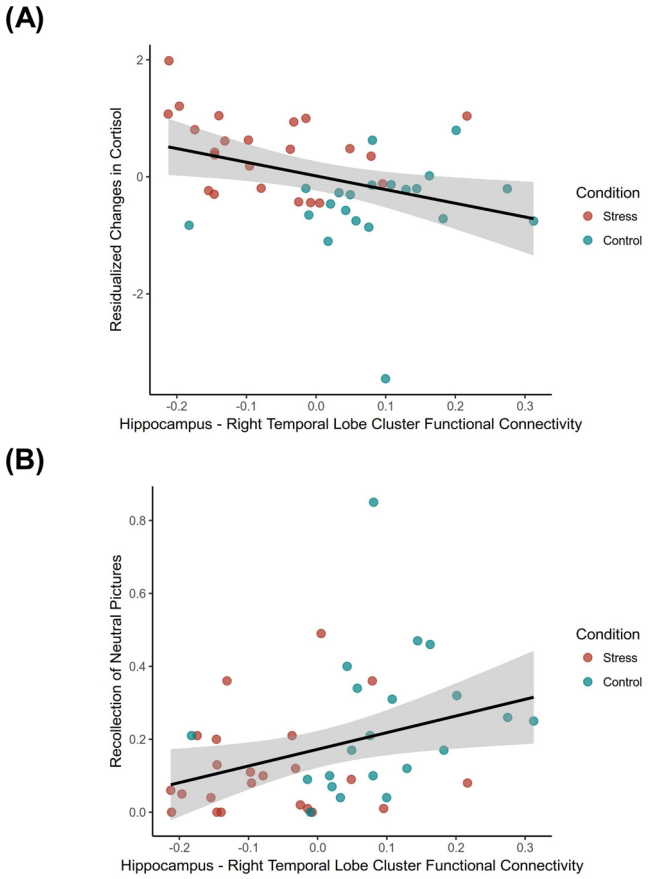

Fig. 4. Association between hippocampus–temporal lobe cluster connectivity and (A) changes in cortisol and (B) recollection of neutral pictures. Across all participants, connectivity between the hippocampus and the right temporal lobe cluster that differed between groups was (A) a significant inverse predictor of changes in cortisol from pre- to post-manipulation, $r = -.361$, $p = .016$, and (B) a significant predictor of recollection of neutral images, $r = .35$, $p = .020$. These associations remained significant after removing the outlying low value in residualized changes in cortisol ($p = .013$) or the outlying high value in recollection ($p = .014$).

not associated with recollection or familiarity of neutral or negative images, $|r|$s $< .19$, $ps > .229$.

Additional analyses are presented within the Supplemental Material, namely, Stress × Time functional connectivity analyses,

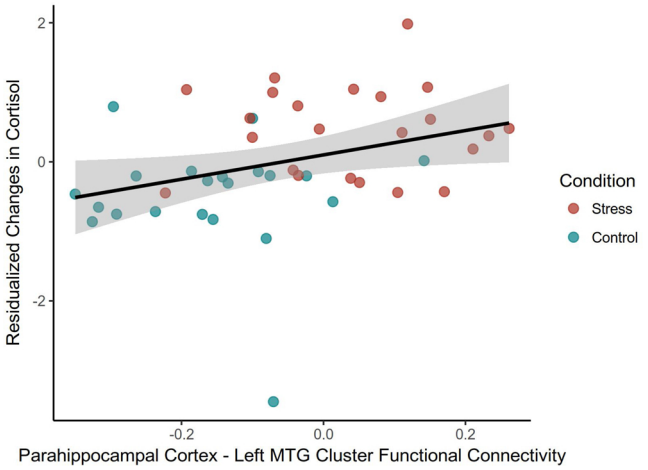

Fig. 5. Association between parahippocampal cortex–middle temporal gyrus cluster connectivity and changes in cortisol. Across all participants, connectivity between the parahippocampal cortex and the left middle temporal gyrus cluster that differed between groups was a significant predictor of changes in cortisol from pre- to post-manipulation, $r = .32$, $p = .032$. Removing the outlying low value in residualized changes in cortisol only strengthened this association, $p = .008$.

Stress × Valence analyses in memory, and functional connectivity associations collapsing valence.

4. Discussion

Stress impairs many cognitive processes (Raio et al., 2013; Sänger et al., 2014; Shields et al., 2017a, 2016a, 2016b), though its effects on memory are nuanced (Henckens et al., 2009; Qin et al., 2012; Shields et al., 2017b; Wolf, 2012; Zoladz et al., 2015). Post-encoding stress generally enhances memory performance (Cahill et al., 2003; Joëls et al., 2011; Preuß and Wolf, 2009; Schwabe et al., 2012; Zoladz et al., 2015), and most theories of stress and memory argue that functional connectivity between the hippocampus and amygdala plays a crucial role in post-encoding stress enhancements of memory (de Voogd et al., 2017; Shields et al., 2017b). However, post-encoding stress that occurs in a different context from learning can impair memory (Sazma et al., 2019; Shields et al., 2017b), and no study has examined the neural basis of this effect. To address this gap, we examined how post-encoding stress that occurred in a different context from learning modulated functional connectivity with medial temporal lobe regions implicated in

theories of stress effects on memory. Our initial analyses showed that stress increased cortisol and impaired recollection relative to a control group, and that stress did not alter hippocampus-amygdala functional connectivity. In our primary analyses, we found that post-encoding stress resulted in lower hippocampal functional connectivity with a region with a peak in the right superior temporal gyrus, and resulted in greater parahippocampal cortex connectivity with a region in the left middle temporal gyrus relative to a control group. Moreover, functional connectivity between the hippocampus and the right superior temporal gyrus was inversely associated with cortisol responses but positively associated with memory performance (i.e., recollection of neutral items), whereas functional connectivity of the parahippocampal cortex with the left middle temporal gyrus was positively associated with cortisol responses but had no associations with memory. Surprisingly, we did not find significant differences in functional connectivity between the stress and control groups using the amygdala or perirhinal cortex as seeds.

The finding that the hippocampus showed a stress-induced difference in functional connectivity to a cluster with a peak in the right superior temporal gyrus suggests that this region plays an important role in the effects of stress on recognition, but its precise role is not yet clear. A number of prior studies have implicated this area in memory-related processes, including in autobiographical memory maintenance/elaboration (Daselaar et al., 2008), intrusive memory recall (Clark et al., 2016), correct source memory judgments (Dobbins et al., 2003), integration of visual and auditory information in recognition memory (Joassin et al., 2011; Plank et al., 2012), and integration of temporal information in facial recognition memory (Lee et al., 2012). Notably, this cluster has also been implicated in feature binding of items within working memory, and negative emotional arousal disrupts this feature binding (Mather et al., 2006), which is broadly consistent with our finding that stress disrupts functional connectivity with this region, leading to worse recollection. Perhaps most importantly, task-evoked functional connectivity between the hippocampus and this region predicts successful memory encoding (Ranganath et al., 2005). Thus, one possibility is that functional connectivity between these regions is critical for normal memory performance, and post-encoding stress in a different context from learning may disrupt this functional connectivity, leading to a decrease in recollection.

The observed association of hippocampal functional connectivity with recollection of neutral images fits with the well-known role of the hippocampus in recollection (Diana et al., 2007), especially recollection of neutral information/materials (Yonelinas and Ritchey, 2015). Because recollection of emotional materials is more amygdala- than hippocampus-dependent (Yonelinas and Ritchey, 2015), the observed association between hippocampal functional connectivity and recollection of neutral images is in agreement with prior literature. In contrast, familiarity is primarily supported by the perirhinal cortex (Diana et al., 2007; Eichenbaum et al., 2007); as such, our lack of observed associations between familiarity and functional connectivity with either the hippocampus or the parahippocampal cortex are in agreement with current knowledge of the neural basis of these memory processes. Although we did not find any clusters showing stress-induced differences in functional connectivity with the perirhinal cortex (and thus no clusters in which to examine correlations with familiarity), we note that the behavioral effects of stress in this experiment were more pronounced for recollection than familiarity (McCullough et al., 2015).

The observed results may be useful in modifying or extending theories of stress and memory, such as the cellular consolidation model (McGaugh, 2015, 2000). At the most basic level, these results show that under some conditions post-encoding stress can exert effects on the brain that are associated with impairments in memory, showing that—in contrast to the foundational predictions of the cellular consolidation model—post-encoding stress is not universally beneficial for memory-related neural function. However, these effects do not rule out effects of cellular consolidation. Indeed, a wealth of cellular consolidation research has demonstrated that glucocorticoids administered or endogenously released shortly after encoding act on hippocampal neurons to enhance long-term potentiation during the post-encoding window (Akirav and Richter-Levin, 2002; McGaugh, 2000; Roozendaal et al., 2010; Schwabe et al., 2012). Therefore, our results suggest that cellular consolidation mechanisms enhanced by post-encoding stress may interact with other effects of stress to influence memory (Andreano and Cahill, 2009; Schwabe et al., 2012).

On a broader level, the idea that post-encoding stress may only enhance memory for information that is relevant to the stressor (e.g., learned within the same context) makes intuitive sense given the presumptive adaptive value of such preferential consolidation (Joëls et al., 2011; Schwabe et al., 2012; Wolf et al., 2016). That is, information related to a stressor is often beneficial for avoiding that stressor in the future, whereas information unrelated to the stressor is presumably only coincidental to the stressor's occurrence, and it would therefore be most adaptive to preferentially forget stress-irrelevant information if competing with stress-relevant information.

The meaning of the correlations between cortisol changes and functional connectivity with the temporal lobe regions is difficult to determine. Because the second resting state scan occurred prior to the second saliva sample, it is possible that both functional connectivity of the parahippocampal cortex with the left middle temporal gyrus and functional connectivity of the hippocampus with the right temporal cluster are causally implicated in cortisol production. For example, perhaps the right temporal cluster integrates the associative information relevant to a stress response and controls whether or not the hippocampus activates the HPA axis (Dedovic et al., 2009). Alternatively, these associations with cortisol may merely reflect the effects of the manipulation on a continuous variable. That is, not only did the stress manipulation influence functional connectivity with these regions, but the more stress an individual experienced, the more this functional connectivity was affected—resulting in a correlation between functional connectivity and cortisol production without one exerting a causal influence on the other. Future work should attempt to determine the role of this functional connectivity in HPA axis activity.

To our knowledge, only one prior study has examined functional connectivity with the hippocampus following post-encoding stress (de Voogd et al., 2017). However, their paradigm differed from ours in a number of ways. Perhaps most importantly, de Voogd et al.'s study was conducted with the encoding task, stress/control manipulation, and fMRI scan in the same context. Additionally, the type of stressor differed between studies, as de Voogd et al. used a stressful movie to induce stress. Further, the materials de Voogd et al. used in their encoding task were not intrinsically arousing or negative; instead, the negative information was associated with faces during their encoding task, and those associations were later retrieved during their memory test. These differences make cross-study comparisons difficult, but some discussion of their results in comparison to ours may be informative.

In contrast to our results, de Voogd et al. (2017) observed the classic post-encoding stress effect on memory, with memory enhanced in the within-subjects stress condition relative to the control condition. Similar to our results, de Voogd et al. observed decreased functional connectivity with the hippocampus after stress relative to control. However, the decreased functional connectivity seen by de Voogd et al. was between the hippocampus and areas very different from those we observed, such as areas in the parietal and occipital lobes. Relative to the control group, participants in our stress induction group evidenced lower functional connectivity between the hippocampus and the superior temporal gyrus, which is a region implicated in successful memory encoding (Ranganath et al., 2005). It is likely that methodological differences (such as different stressors, different stimuli, and changing contexts between learning and stress) contribute to behaviorally relevant differences in functional connectivity with medial temporal lobe regions, potentially contributing to the differences in hippocampal functional connectivity observed between studies.

Despite the strengths of this study, including use of a standardized acute stress manipulation and assessment of neural activity, hormones, and behavior, some limitations should be noted. First, although our experimental manipulation of acute stress allowed us to study the effects of post-encoding stress in a different context from learning, we did not experimentally manipulate context. Thus, it is possible that the effects we observed here are general effects of post-encoding stress that have nothing to do with context. Indeed, post-encoding stress effects on memory may be quadratic in nature (e.g., an inverted-U), which could result in post-encoding stress-induced memory impairments without any context change between encoding and stress. However, the behavioral result of worse memory in the post-encoding stress group argues against this possibility (Sazma et al., 2019; Shields et al., 2017b). Nonetheless, experimentally manipulating both stress and context is an important avenue for future research aimed at understanding the neurobiological basis of context-dependency in post-encoding stress effects on memory. Second, we did not include any women in this study, which limits our generalizability to males alone. Third, because we could not experimentally manipulate functional connectivity itself, associations with memory and cortisol were correlational and causation cannot be inferred. Fourth, because we did not statistically test for differences between associations of functional connectivity with memory for neutral compared to negative items, we cannot conclude that the association of functional connectivity with neutral items is different from than the association of functional connectivity with negative items. Finally, this study was conducted using an undergraduate sample of young adults. Although fairly ethnically diverse, it is important to note that the effects we observed may not generalize to different populations.

In conclusion, we found that post-encoding stress altered functional connectivity with both the hippocampus and the parahippocampal cortex, but we did not find stress-induced differences in functional connectivity with either the amygdala or perirhinal cortex. Moreover, post-stress/control functional connectivity between the hippocampus and the right temporal lobe cluster was significantly associated with recollection of neutral materials and significantly inversely associated with changes in cortisol from pre- to post-stress/control manipulation. Similarly, post-stress/control functional connectivity between the parahippocampal cortex and the left temporal cluster lobe was significantly associated with cortisol changes from pre- to post-manipulation. Thus, post-encoding stress altered functional connectivity with seed regions in the medial temporal lobes that support memory, and these alterations were related both to changes in cortisol from before to after the manipulation and to memory performance.

Conflict of interest statement

The authors declare no conflict of interest in this work.

Author note

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