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Beta and alpha desynchronizations underlie reconsolidation-mediated episodic memory updating

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ABSTRACT

Reactivation returns a consolidated memory to a plastic state, opening a window for the existing memory to be updated. For episodic memory, learning of competing information upon reactivation either integrates the new information into the reactivated memory or disrupts the reactivated memory directly, but the two effects were found in distinct experimental paradigms and their neural mechanisms are largely unknown. The current study explored the effects and neural mechanisms of episodic memory reactivation using behavioural and MEG techniques. Taking advantage of an independent-cue retrieval procedure, we revealed both the integration and the forgetting effects by a single post-reactivation interference paradigm. However, while the integration effect followed the reconsolidation window, the forgetting effect did not, suggesting only the integration effect being caused by memory reconsolidation. MEG measurements further revealed beta-band power decrease during reactivation and alpha-band power decrease during post-reactivation interference, both of which parametrically predicted the degree of memory integration. But neither the beta nor the alpha desynchronization was related to the forgetting of the original memory. Our results suggest original memory forgetting and new information integration happen in different time periods after memory reactivation, and beta and alpha desynchronizations underlie reconsolidation-mediated episodic memory updating.

1. Introduction

The memory system requires large flexibility as the existing information becomes inaccurate, outdated, or even unwanted continuously. In the past decades, researchers have found that a brief reactivation can destabilize a consolidated memory, making it vulnerable to modifications again (Nader et al., 2000). The destabilization role of reactivation has been established by decreased memory performance, mainly in conditioning and procedural memory, when amnesic treatments, including pharmacological blockade (Duvarci et al., 2005; Nader et al., 2000; Rossato et al., 2007), electroconvulsive shocks (Kroes et al., 2014; Misanin et al., 1968), and new competing information (Chan and LaPaglia, 2013; Diekelmann et al., 2011; Walker et al., 2003) were given upon reactivation. Remarkably, the memory decrease only shows up after a period of at least several hours, during which a reconsolidation process which involves complicated protein synthesis occurs to re-stabilize the reactivated memory. This period of time, also known as the reconsolidation window, supports that the forgetting effect results from disruptions on memory reconsolidation.

In contrast to memory destabilization in other memory formats, forgetting of the reactivated memory is not consistently found in episodic memory. First, post-reactivation interference approaches often failed to disrupt memory (Beckers and Kindt, 2017; Levy et al., 2018). Instead, reactivation sometimes enhanced the supposedly destabilized memory and protected the memory from being disrupted by interference given right after reactivation (Pashler et al., 2013; Potts and Shanks, 2012). Recent failures to replicate the forgetting effect in episodic memory have even cast doubts on whether post-reactivation interference could disrupt episodic memory reconsolidation (Levy et al., 2018). In our recent study, we introduced an additional cue that was independent of the reactivation and post-reactivation interference manipulations to retrieve the reactivated memory. This independent cue succeeded to detect performance decrease of the reactivated episodic memory (Zhu et al., 2016), providing a way to reveal the forgetting

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effect in episodic memory. However, because the memory decrease was observed before the reconsolidation window closed, it is yet unknown whether the effect could be fully attributed to the disruption of memory reconsolidation.

Another effect, memory integration, has been found by post-reactivation interference in episodic memory. In a series of studies, Hupbach et al. presented participants with a set of objects; half of the participants were reminded of the objects 48 h later, and then all participants were presented a second set of objects for relearning. They did not find memory change on the first set of objects. Instead, participants who had been reminded of the first set of objects exhibited misattribution of the second set of objects to be among the first set, showing an integration of the new information into the reactivated memory (Gershman et al., 2013; Hupbach et al., 2007; Hupbach et al., 2009; Hupbach et al., 2008). More than that, consistent with the reconsolidation theory, this integration effect showed up after but not before the reconsolidation window closed, indicating it being caused by memory reconsolidation. Therefore, reactivation rendered new information learned within the reconsolidation window more likely to be incorporated into the existing memory.

Now that both forgetting and integration can be induced by post-reactivation interference in human episodic memory, it is unknown whether the same mechanism underlying the two effects. Meanwhile, there is no evidence that both effects coexist in post-reactivation interference. To answer these questions, the current study applied the post-reactivation interference (R-interference) procedure with the independent cue technique (Zhu et al., 2016) to consolidated word associates and tested its e

later, on Day 4. Target words that had been learnt in the associative learning and R-interference training phases, namely target words from the original word pairs and substitute words from the interference word pairs, were presented to participants in counterbalanced order. Participants judged when each word was learnt, by selecting from the following choices: *Day 1 (learning phase 1)*, *Day 2 (learning phase 2)*, *Day 3 (interference phase)*, and *both Day 1 and Day 2 (learning phase)*. Considering that the studied target words were learnt on both *Day 1* and *Day 2*, only response of *Day 3* was coded as new; the remaining responses were coded as old. No time limit was given during either the recall or the memory source test.

MEG recording. Ongoing brain activity was recorded (sampled at 1000 Hz) using a whole-head MEG Neuromag (VectorView™, Elekta Neuromag Oy, Helsinki, Finland) acquisition system. It consists of 306 sensors arranged in triplets of two planar gradiometers and one magnetometer. Before the recordings, four head position indicator coils attached to the scalp determined the head position with respect to the sensor array. The location of the coils was digitized with respect to three anatomical landmarks (nasion and preauricular points) with a 3D digitizer (Polhemus Isotrak system). A custom-made chin set was used to fix the head. The head position with respect to the device origin was acquired before each block and monitored throughout each recording block to ensure that head movements did not exceed 0.5 cm at any time. Two bad channels were manually detected (noisy, saturated, or with SQUID jumps) and excluded for further analysis. The temporal extension of signal-space separation (tSSS) method was applied during the pre-processing stage of analysis using Elekta Neuromag MaxFilter software to reduce noise from the external environment (Taulu and Simola, 2006). After the MEG session, anatomical MRI images were acquired using a GE MR750 3.0 T system.

MEG preprocessing. Data analysis was performed in Matlab 2016a (MathWorks, Natick, MA) using the Fieldtrip open source Matlab toolbox (Oostenveld et al., 2011) (http://fieldtrip.fcdonders.nl), and custom scripts. The data were bandpass filtered between 1 and 30 Hz offline and were downsampled to 200 Hz before further analysis. The continuously recorded MEG data were divided into epochs of 3 s length (1 s before and 2 s after the cue word presentation) for the reactivation condition and 7 s length (3 s before and 4 s after the substitute word presentation) for the interference condition. Baseline correction was applied by subtracting the average response of the 0.5 s prior to the word presentation from all data points throughout the epoch. Considering that each interference trial followed immediately after a reactivation trial, to avoid contamination, the 2.5–2.0 s prior to the interference trial (i.e. the 0.5 s prior to the reactivation trial) was used as baseline for the interference trial. An independent component analysis (ICA) was performed to remove artefacts including cardiac, eye movements, blinks, and environmental noise.

Spectral analysis. Spectral analysis was performed on single trials and then averaged across trials. The time-frequency representations (TFRs) was estimated for frequencies < 30 Hz, using Morlet wavelet transform (width: 7 cycles; frequency resolution: 1 Hz). TFRs were estimated for each condition, on each channel, and in each subject respectively. We prespecified 8–12 Hz as alpha-band based on a priori expectations (Klimesch, 1999) and 15–25 Hz as beta-band activity based on the congruency effect observed over sensors. Baseline correction for each frequency was applied by subtracting the averaged absolute values of the 0.5 s prior to the word presentation from all data points throughout the epoch.

Sensor-based analysis was performed only on planar gradiometers, considering that planar gradient maxima are located above neural sources which facilitates the interpretation of MEG results (Bastiaansen and Knösche, 2000; Hari and Salmelin, 1997). We computed metrics separately for the horizontal and vertical planar gradients, and combined the two by computing the sum. Cluster-based nonparametric permutation test (Maris and Oostenveld, 2007), which ensures correction for multiple comparisons over time of sensors and frequencies, was used to detect sensor clusters that exhibit significant difference between different conditions.

Source analysis. Source estimates were computed applying a frequency domain adaptive spatial filtering algorithm (dynamic imaging of coherent sources; Gross et al., 2001). This algorithm uses the cross-spectral density (CSD) matrix from the MEG data and the lead field derived from the forward model to construct a spatial filter for a specific location ("voxel"). These spatial filters were estimated on the basis of all trials. After identification of the fiducials, the nasion, and the left and right preauricular points, coregistration with Montreal Neurological Institute (MNI) coordinates was applied. A realistic, single-shell brain model was constructed based on the anatomical MRI. Forward solution for each participant was estimated using a common dipole grid (1 cm^3 grid) in MNI space warped onto each participant's anatomy. To reconstruct the source activity in the significant time and frequency ranges based on sensor-level statistics, we used multitaper frequency transformation to extract beta-band (15–25 Hz) and alpha-band (8–12 Hz) activities. Relative change in power for strong versus control [(strong - control)/control] condition was computed to assess effects of strong reactivation and interference.

Control Experiment 1. Eighteen independent participants (aged 22–29 years, 13 females) were recruited with the same criterion as in the main experiment. Same materials and procedure were used as in the main experiment, with two exceptions. First, the source test, which was given on Day 4 in the main experiment, was given immediately after R-interference, using the same procedure as in the main experiment; second, no recall tests were given.

Control Experiment 2. Nineteen independent participants (aged 23–31 years, 11 females) were recruited with the same criterion as in the main experiment. Because the weak association condition was not included, only word pairs from the strong association conditions (i.e. 40 A-X and 40 B-X pairs) in the main experiment were used. On Day 1, participants studied the 40 A-X pairs to 100% accuracy, using the learning and test-feedback procedure; then they studied the 40 B-X pairs to 100% accuracy, on the same day using the same procedure. On Day 2, the same reactivation procedure as in the main experiment was applied to 20 of the cue As and 20 filler words. However, no interference training was given afterwards. Instead, participants passively viewed a fixation point on the screen for 4 s after reactivation. A recall test was given right after the reactivation training, on both the trained and the independent cues.

3. Results

3.1. New information integrated into the reactivated memory through reconsolidation

Participants learned cue-target word associations in the first two days (e.g. *wisdom - plane; virus - plane*). On Day 3, the original association was reactivated by retrieving the cue item and then disrupted by learning a cue-substitute (e.g. *wisdom - extreme*) association. In addition to this classical reconsolidation disruption procedure, two critical manipulations were applied. First, the reactivation level was manipulated by controlling the strength of the memory. Specifically, strong and weak cue-target associations were formed at the beginning so that presenting the cues individually would induce strong and weak reactivations on the target items respectively. Second, a double-cue/one-target procedure was employed (Zhu et al., 2016), which introduced a second cue to measure the forgetting effect of the reactivated memory. To examine the memory integration effect, we used a source attribution task (Hupbach et al., 2007). The source attribution test was given 24 h after R-interference training (Fig. 1A), in which the original and substitute target items were presented and participants judged when each item was learnt.

We calculated the percentage of the new information (i.e. substitute target items) that was misattributed as the original information (i.e. the

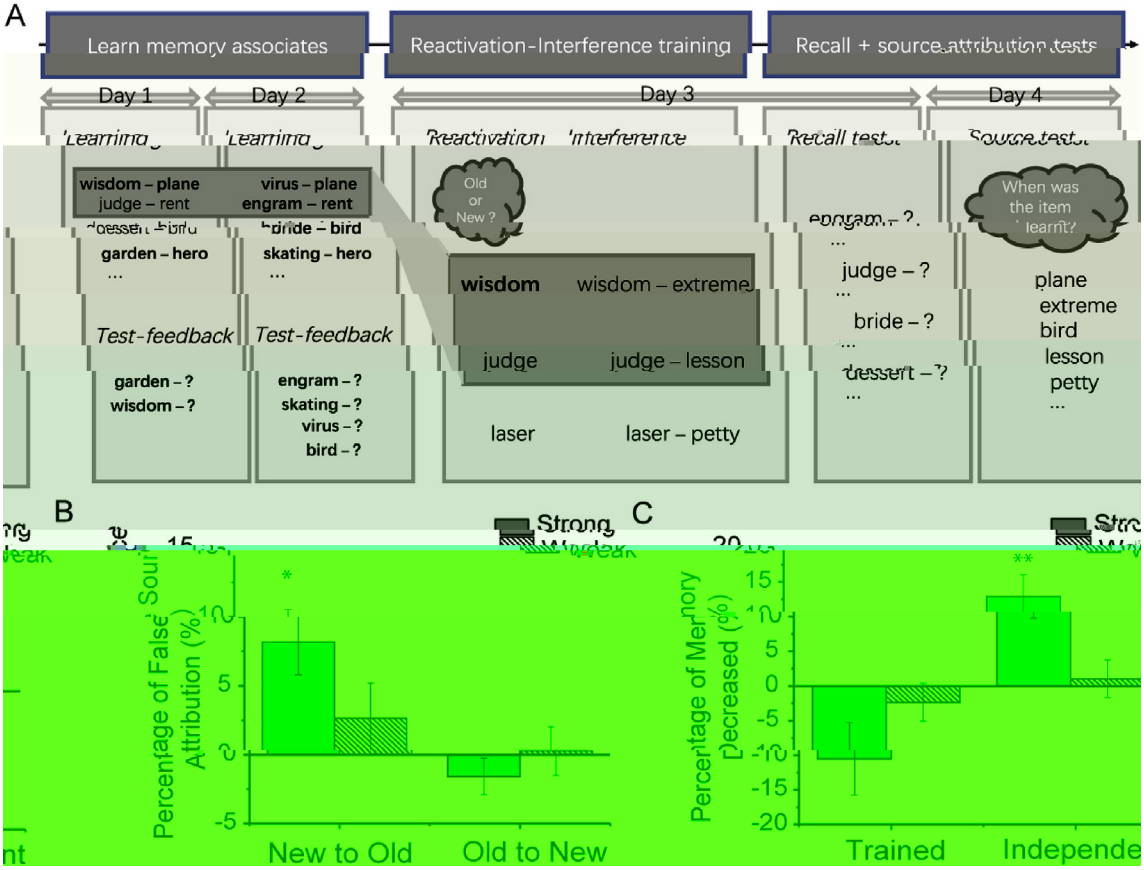

Fig. 1. Experimental procedure and behavioural results. (A) Cue-target word associations (i.e. A-X). were learnt on Day 1. Strong associations (shown in bold font) were formed for half of the pairs through a test-feedback procedure to ensure those associations were fully memorized. On Day 2, a second series of cue-target word associations (i.e. B-X) that shared the same targets with those on Day 1 was learnt and fully memorized. On Day 3, a subset of A-X pairs was first reactivated by a recognition test on the cue words and then interfered by pairing the cue words with substitute words for learning (i.e. A-Y, shown in shaded area). Novel cue words and cue-target associates were included as control. A recall test on all the cue words that have been learnt on Day 1 and Day 2 was given right after the reactivation-interference (R-interference) training. On day 4, a source attribution test was given on both the target words and the substitute words, during which participants reported when they have learnt each word. (B) Percentage of false source attribution. Substitute words under strong association were misrecognized as the original target words, but not vice versa. (C) Percentage of memory impaired relative to the control condition. New learning after strong association caused significant forgetting when tested by independent cues. *$p < .05$, **$p < .01$ (two-tailed t-test); error bars, SEM.

original target items), and the other way around (see Supplementary Table S1 for data in each condition). Results (Fig. 1B) showed that more substitutes (i.e. Y) were misattributed as the original targets (i.e. X) in the strong association condition than in its control condition ($t(18) = 3.45$, $p = .003$, Cohen's $d = 0.79$). But the original targets (i.e. X) were not misrecognized as the interference information (i.e. Y; strong association: $t(18) = -1.19$, $p = .25$, Cohen's $d = 0.27$; weak association: $t(18) = 0.15$, $p = .88$, Cohen's $d = 0.04$). These results replicated previous findings (Hupbach et al., 2007, 2008, 2009). Indeed, reactivation at a certain level causes the newly learnt information to be integrated with the original memory.

A control experiment was performed to examine whether the integration effect was dependent on presentation after the time window of the reconsolidation process (Supplementary Table S1). A procedure consistent with Hupbach et al.'s (Hupbach et al., 2007) control condition was used: this time the source attribution test was given immediately after R-interference, before the reconsolidation process could complete. No more source confusion was found in the strong association than in the weak reactivation ($t(17) = 0.46$, $p = .65$, Cohen's $d = 0.11$) or control ($t(17) = -0.70$, $p = .49$, Cohen's $d = 0.17$) condition. Therefore, the interference information is integrated into the reactivated memory through the reconsolidation process.

3.2. Forgetting of the reactivated memory by interference upon reactivation

Next, we examined whether R-interference could disrupt episodic memory after consolidation (Fig. 1A). As addressed above, to increase the sensitivity of measurements, an independent cue was introduced in addition to the original cue to retrieve each target word (Zhu et al., 2016). That is, each target word was paired with two different cue words for learning but only one cue-target series received further R-interference training. In the final test, both the trained and the untrained cue (i.e. independent cue) were used to retrieve the target item. Thereby, changes on the target item could also be revealed by its independent cue.

Considering that different controls were used for the strong and weak conditions, comparisons were conducted between each experimental condition and its corresponding control separately (see Supplementary Table S2 for data in each condition). We replicated our previous finding (Zhu et al., 2016) that interference under strong association condition caused significant forgetting (Fig. 1C, $t(18) = 4.11$, $p < .001$, Cohen's $d = 0.87$) when examined by an independent cue that did not receive direct reactivation or interference training. However, no memory decrease but a trend of improvement were observed when the target items were tested by the trained cues ($t(18) = -2.01$, $p = .059$, Cohen's $d = 0.52$). The weak association condition instead failed to affect the original memory, as post-reactivation interference

did not cause any memory changes in either the trained- (t (18) = −0.87, p = .39, Cohen's d = 0.13) or the independent-cue (t (18) = 0.39, p > .70, Cohen's d = 0.07) condition. Therefore, the effect is restricted to a certain degree of memory reactivation.

A control experiment with strong association but no interference manipulation was further conducted (Supplementary Table S2). No forgetting was found any more when tested by the independent (t (19) = 1.05, p = .31, Cohen's d = 0.23) or trained (improvement: t (19) = −2.67, p = .02, Cohen's d = 0.60) cue, excluding the possibility that the forgetting effect found in the independent-cue condition was caused by reactivation but not interference upon reactivation. Therefore, through introducing an additional dimension, the independent cue, we verified that post-reactivation interference could disrupt consolidated episodic memory (Zhu et al., 2016).

3.3. No correlation between the integration and forgetting effects

According to the results above, while the integration effect showed up only after a period of reconsolidation, the forgetting effect was detected in immediate test. The different time courses of the two effects suggest that integration and forgetting might rely on different processes. To verify this, we performed two correlation analyses: correlation between the two memory effects and correlation between each effect with the degree of original memory intrusion. Difference between the percentages of source misattribution in the strong association condition with that in its control condition was used to represent the degree of memory integration. Pearson correlation analysis showed no significant correlation between the integration and forgetting effects in either the trained- (r(19) = 0.191, p = .43) or independent-cue (r (19) = −0.207, p = .40) condition. Considering that reactivation might cause intrusion of the original memory, we also asked participants to report spontaneous memory intrusions during interference training (Benoit et al., 2015). The intrusion degrades gradually over time. We measured the degree of intrusion change across the eight training blocks for each participant by calculating its curve slope after linear fitting. Interestingly, correlation was found between the intrusion decrease slope with the forgetting of the original memory (Fig. 2B: r (19) = 0.46, p = .05) but not with the integration of new information (Fig. 2A: r(19) = −0.07, p = .76). These results congruently suggest that the forgetting effect was likely to result from a different process which was also induced by memory reactivation than the reconsolidation process.

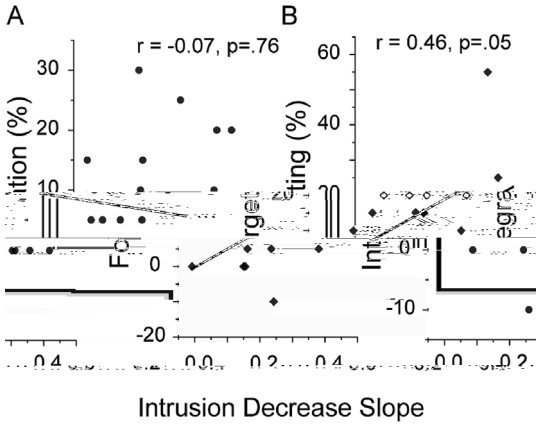

Fig. 2. Correlations between behavioural effects and memory intrusion. The degree of memory intrusion decrease was uncorrelated with the integration effect (A), but was correlated with the forgetting effect (B) across individual participants.

3.4. Beta desynchronization during reactivation predicting integration

To identify the neural activity signalling the integration and forgetting effects, we recorded ongoing brain activity using MEG during the R-interference phase. The spectrogram (1–30 Hz) of the MEG signal between strong reactivation and control (Str vs. Ctr) was compared statistically. Cluster-based permutation test was used to avoid the multiple comparison problem over time points and sensors (Maris and Oostenveld, 2007). We found decreased beta-band (15–25 Hz) power caused by strong memory reactivation over a large cluster of sensors (p = .01, Fig. 3B). This reactivation effect was statistically significant between 0.86 and 1.22 s after presentation of the retrieval cue (Fig. 3 A,C). Reconstructing the source locations of this difference showed decreases of beta-band activity for strong association trials distributed around the left fusiform gyrus (Fig. 3D), which was mainly in the visual word form area (VMFA). No significant difference was found in the alpha band activity. The same procedure was also applied to the comparison between strong and weak reactivation conditions (Str vs. Weak), but did not detect significant clusters.

We then tested the behavioural relevance of this beta-band change. We calculated the mean beta-band difference between the strong and control conditions across the most significant time points (ps < .005, 0.89–1.09 s) within the significant window (0.86–1.22 s), and performed its Pearson correlation with the behavioural integration and forgetting effects respectively (Fig. 3E, black). Significant correlation was found between beta-band decrease and the integration effect across individuals (r(19) = −0.56, p = .01). Yet no correlation was found with the forgetting effect (r(19) = 0.13, p = .60). Fig. 3E (cyan) also showed correlations between the neural and behavioural differences under the Str vs. Weak comparison. Although being nonsignificant, correlations of the Str vs. Weak comparison were in the same directions as those of the Str vs. Ctr comparison. Notably, beta-band decrease (i.e. strong – control/weak) was used for correlation analysis; therefore, the negative correlation means that more beta-band desynchronization is accompanied with more integration of new information into the reactivated memory.

3.5. Alpha desynchronization during interference signalling integration

The same analysis was applied to the interference phase as above. Cluster-based permutation test on the interference phase detected a concentrated cluster of decreased alpha-band (8–12 Hz) power in the strong interference versus the control condition (Str vs. Ctr), with significant differences within 2.02–3.35 s after substitute word onset (cluster-based permutation test, p = .013, Fig. 4 A,C). Differences were present over anterior sensors (Fig. 4B). Reconstructing alpha-band activity revealed condition differences (i.e. stronger alpha-band desynchronization in the strong interference condition) in the left posterior superior temporal gyrus (pSTG) and supramarginal gyrus (SMG) that are related to language processing and the right frontal lobe/middle frontal gyrus (MFG) that is involved in reorienting endogenous attention (Fig. 4D). No significant difference was found in the beta band activity or under the Str vs. Weak comparison.

To test the behavioural relevance of this alpha-band change, we calculated the correlation between the alpha-band change (i.e. strong interference - control) with the behavioural memory integration and forgetting effects. Same to the procedure for beta-band correlations, we calculated the mean alpha difference across the most significant time points (ps < .005, 2.61–2.75 s) within the significant time window (2.02–3.35 s). Pearson correlation across individuals revealed marginal significant negative correlation between alpha-band change and memory integration (r(19) = −0.45, p = .05). Namely, more new information is integrated into the reactivated memory when more alpha desynchronization emerged. Correlation between alpha-band change and memory forgetting (r(19) = 0.28, p = .24) was not significant however. Fig. 4E (cyan) also showed correlations between the neural

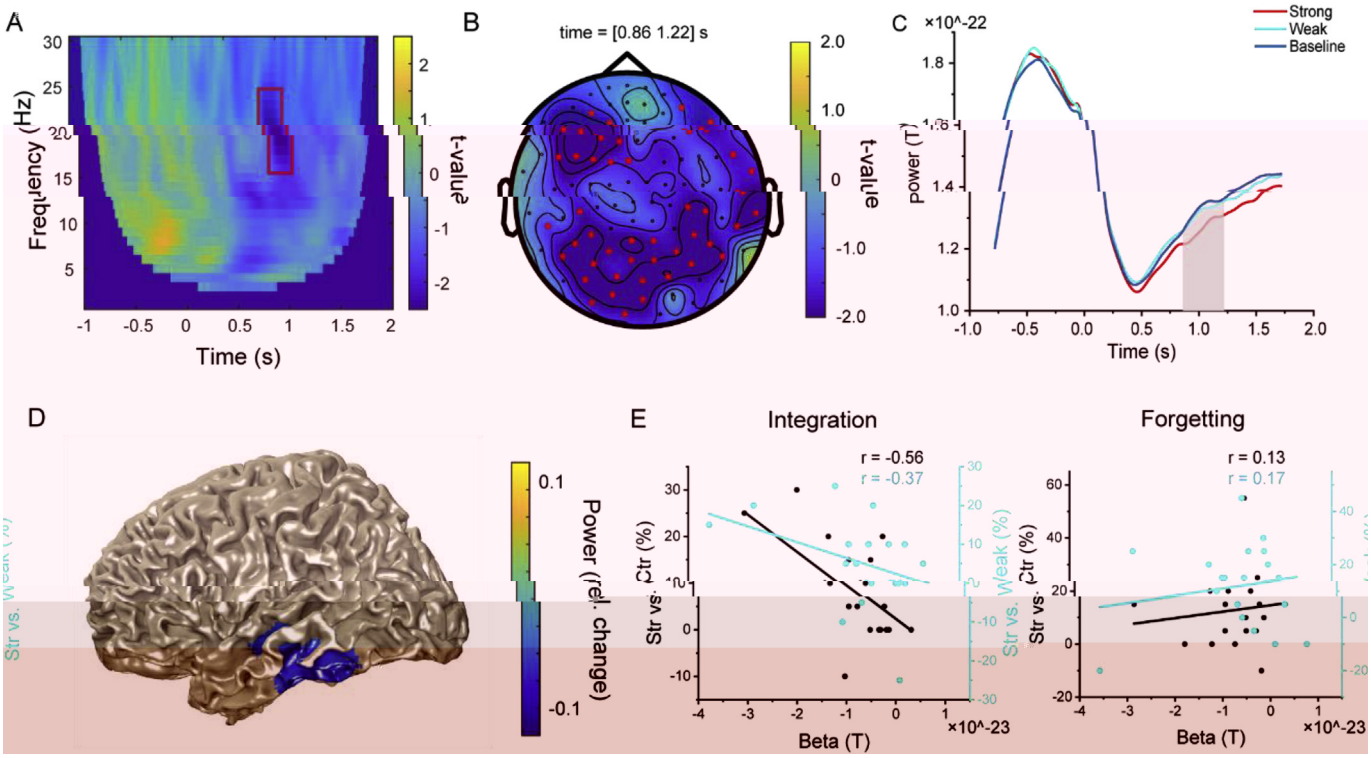

Fig. 3. MEG results for the reactivation phase. (A) Time-frequency plot of between condition differences (t values: Str vs. Ctr) averaged over sensors with a significant effect (see B). Time 0: reactivation onset. The red box shows the time-frequency interval with a significant reactivation effect (0.86–1.22 s after reactivation onset). (B) Topographic distribution of sensors with a significant reactivation effect at beta band (15–25 Hz). The significant sensors are marked in red. (C) Changes over time in beta-band power (15–25 Hz) averaged across significant sensors (see B). The epoch with a significant difference between strong reactivation and control conditions is marked in grey. (D) Cortical distribution of the beta-band reactivation effects, thresholded by 80% of the max value. (E) Correlation between beta-band change and the memory integration (left) and forgetting (right) effects under the Str vs. Ctr comparison (black) and Str vs. Weak comparison (cyan).

and behavioural differences under the Str vs. Weak comparison, which were in the same directions as those under the Str vs. Ctr comparison. Therefore, both alpha- and beta-band desynchronization underlie the memory integration effect during different periods and neither underlie the forgetting effect.

4. Discussion

By combining the post-reactivation interference procedure with the independent-cue retrieval technique, the current study showed that competing information learned upon reactivation updates the memory with the newly learned information and at the same time causes forgetting of the existing memory. However, while the integration effect showed up after the reactivated memory was reconsolidated, the forgetting effect was found before the reconsolidation process could complete. Therefore, only the integration effect is likely to be caused by memory reconsolidation. This was further confirmed by the finding that, although induced by the same manipulation, the two effects were unrelated to each other. MEG recording revealed power decreases of beta activities during reactivation and of alpha activities during post-reactivation interference, both of which were parametrically correlated with the degree of memory integration but were unrelated to the degree of memory forgetting. Taken together, beta and alpha desynchronizations are likely to underlie episodic memory integration mediated by memory reconsolidation.

Introducing interference information upon reactivation integrates the new information into the reactivated memory. This is consistent with Hupbach et al.'s classical finding (Hupbach et al., 2007). Notably, the integration function of memory reconsolidation has only been demonstrated in episodic memory; while in other memory formats, post-reactivation interference mainly causes direct forgetting on the reactivated memory. As has been discussed by Hupbach and colleagues, this might be due to low conflict between the interference information and the reactivated memory in episodic memory (Scully et al., 2017). For instance, in conditioning memory, the interference information (e.g. the conditioned stimulus is no longer paired with electric shocks) is incompatible with the original memory (e.g. the conditioned stimulus is paired with electric shocks). The two memories cannot be integrated with each other, and instead one replaces the other and causes forgetting on the original memory. Instead, in episodic memory, the original and new information is usually compatible. Consequently, it is possible that when the interference information and the original memory is contradictory, forgetting is likely to occur; while when they are compatible, integration is likely to occur. Indeed, such mutual existence of integration and forgetting has been found in other memory manipulations. For example, under the same procedure of retrieving one target from a contextual cue, either forgetting or facilitation can be induced to the non-retrieved targets of the same contextual cue (Jonker et al., 2018). The relative competition between the retrieved and non-retrieved targets determines which effect to occur. These findings suggest great flexibility of the memory system, and whether integration or forgetting happens might rely on the relative competition between the original and the newly-learnt information.

The integration of new information to the existing memory was mediated by memory reconsolidation, as evidenced by the finding that integration only occurred after the reconsolidation window closed. This contrasts with memory integration of other forms. Studies have shown that memory integration occurs widely for overlapping events that were close in temporal, spatial, and conceptual representations, not necessarily under reconsolidation (Morton et al., 2017). Based on neuroimaging and neuropsychological evidence, Preston and colleagues have showed critical involvement of hippocampus and medial

the fusiform gyrus, which was likely to contain VWFA, an area that is related to word recognition (Dehaene and Cohen, 2011) and higher-level processing of word meaning (Devlin et al., 2006). This is in accordance with the task requirement that participants judged whether the cue word on the screen had been studied or not depending on the meaning of the cue word or the cue-target association. In a seminal study, Khader and Rosler (Khader and Rosler, 2011) parametrically modulated the number of retrieved items and found that beta power decreases varied systematically as a function of the number of retrieved items, with higher numbers of retrieved items inducing stronger beta desynchronization. In line with this, the beta desynchronization in the current results might represent successful reactivation of the old memory. We speculate that the successful reactivation triggers memory reconsolidation which causes memory integration. This explains the positive correlation between beta desynchronization and memory integration.

Interference information learning upon reactivation was accompanied with alpha-band activity decrease. Alpha power increase has been attributed to inhibitory activities, while its decrease represents processing of task-related information (Klimesch et al., 2007; Roux and Uhlhaas, 2014). For long-term memory processing, alpha power decrease has been found during successful compared with unsuccessful encoding and retrieval (Hanslmayr et al., 2012; Park et al., 2014). The current results further revealed that alpha power decreased when information conflicting with the reactivated memory was being studied. However, since this phase contained both retrieval of the original memory and encoding of interference information, it was unclear which process the alpha desynchronization reflected. The finding that larger alpha desynchronization predicted more integration of these conflicting information 24 h later gave us some hints. Based on the reconsolidation theory, the memory integration effect, which occurred after but not before reconsolidation window closed, was likely to be mediated by memory reconsolidation. Therefore, the alpha desynchronization might represent successful initiation of memory reconsolidation, or successful destabilization of the consolidated memory which would then initiate memory reconsolidation. Notably, the alpha-power decreases were originated from semantic processing brain areas STG and SMG (Stoeckel et al., 2009). Decreased alpha power in these areas might represent successful retrieval of the cue-target associations from existing memory, successful integration of the interference information into the old memory, or both. Decreased alpha power was also found in MFG, an area that is involved in regulation of attention and initiation of voluntary inhibitions (Corbetta et al., 2008). The decreased alpha activity here might represent higher need of attention regulation due to increased competitions between the reactivated memory and the interference information. It is worth noticing that the present results merely provided correlational evidence that the beta and alpha desynchronizations could reflect processes related to memory reactivation or reconsolidation. The reason why no neural correspondence was found for the forgetting effect is unclear. Considering the critical role of hippocampus in this process (Gershman et al., 2013) and the insensitivity of MEG to subcortical brain areas, further studies could explore the contributions of hippocampus to the forgetting effect using other techniques such as functional magnetic resonance imaging.

The integration and forgetting effects were both detected only on strong cue-target associations. This might be because simply presenting the cue was not able to reactivate the associated memory when the association was too weak. In Scully et al.'s (2017) meta-analysis, reactivation by different procedures does not affect the effect. But the current study suggests that at least a certain degree of reactivation strength is required. This is consistent with Gershman et al.'s finding that the degree of memory reactivation is associated with the amount of memory integration (Gershman et al., 2013). Computational modelling evidence has suggested that, for episodic memory, moderate level of memory activation causes disruptions on the target memory, while strong activation strengthens the target memory (Detre et al., 2013).

One potential explanation for the immediate forgetting effect, which violates the reconsolidation window, is that forgetting is induced by other processes than reconsolidation disruption. This does not contradict with previous findings. While previous studies discovered that interference upon reactivation disrupted reconsolidation, they did not exclude other processes being triggered by reactivation. The existence of immediate memory forgetting implies that the post-reactivation interference procedure does not only disrupt memory reconsolidation (at least for episodic memory) but also triggers some other processes. However, it remains unknown whether and how much the immediate forgetting contributes to the forgetting effect found in remote tests, when reconsolidation is completed. In any case, the time window should be carefully tested before attributing any effect to reconsolidation disruption.

To sum up, the current study reveals that reactivation labializes the consolidated memory and causes new information more likely to be integrated into the existing memory. The finding that the forgetting effect appears even before the reconsolidation process completes calls attention to the theoretical speculation of the effects by post-reactivation interference in episodic memory studies. We also provide the first evidence that reconsolidation-dependent memory integration is predicted by beta- and alpha-band power decrease while reconsolidation-independent memory forgetting is not, implying that beta and alpha desynchronization may underlie episodic memory reconsolidation. Based on this, reactivation and reconsolidation may occur in brain areas that are related to memory information processing and attention attribution. Considering the differences between episodic memory and other memory formats, it is yet unknown whether the effects and neural mechanisms apply to conditioning memory, procedural memory, and so on.

Declaration of interests

The authors declare no competing financial interests.

CRediT authorship contribution statement

Zijian Zhu: Conceptualization, Investigation, Writing - original draft, Project administration. **Yingying Wang:** Conceptualization, Formal analysis, Writing - review & editing. **Jianrong Jia:** Formal analysis, Data curation, Writing - review & editing. **Yanhong Wu:** Conceptualization, Writing - review & editing, Supervision, Funding acquisition.

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Appendix A. Supplementary data

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