Report

Current Biology

Stimulation of the Posterior Cortical-Hippocampal Network Enhances Precision of Memory Recollection

Highlights

- Network-targeted stimulation caused lasting, selective increase in memory precision
- Stimulation reduced evoked oscillatory neural correlates of recollection
- The hippocampal posterior-medial network is causally involved in memory precision

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In Brief

Nilakantan et al. use network-targeted brain stimulation to selectively enhance a component of memory—recollection precision—and modulate its neural correlates. This supports the causal role of the posterior hippocampal network in memory precision and suggests that noninvasive stimulation can enhance reactivation of precise memory details.

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Report

Stimulation of the Posterior Cortical-Hippocampal Network Enhances Precision of Memory Recollection

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SUMMARY

Episodic memory is thought to critically depend on interaction of the hippocampus with distributed brain regions [1–3]. Specific contributions of distinct networks have been hypothesized, with the hippocampal posterior-medial (HPM) network implicated in the recollection of highly precise contextual and spatial information [3–6]. Current evidence for HPM specialization is mostly indirect, derived from correlative measures such as neural activity recordings. Here we tested the causal role of the HPM network in recollection using network-targeted noninvasive brain stimulation in humans, which has previously been shown to increase functional connectivity within the HPM network [7]. Effects of multiple-day electromagnetic stimulation were assessed using an object-location memory task that segregated recollection precision from general recollection success. HPM network-targeted stimulation produced lasting (~24 hr) enhancement of recollection precision, without effects on general success. Canonical neural correlates of recollection [8–10] were also modulated by stimulation. Late-positive evoked potential amplitude and theta-alpha oscillatory power were reduced, suggesting that stimulation can improve memory through enhanced reactivation of detailed visuospatial information at retrieval. The HPM network was thus specifically implicated in the processing of fine-grained memory detail, supporting functional specialization of hippocampal-cortical networks. These findings demonstrate that brain networks can be causally linked to distinct and specific neurocognitive functions and suggest mechanisms for long-lasting changes in memory due to network-targeted stimulation.

RESULTS

Even when recollection of past episodes is successful, the amount of retrieved information can vary [11], providing memory for precise details in some cases (e.g., "the store was on the left, four blocks ahead of the first stoplight") and more general memory in others (e.g., "the store was on the left side of the street"). Several lines of evidence suggest preferential contributions to high-precision memory by the posterior hippocampus [6], including smaller receptive fields for posterior compared to anterior hippocampus ([12]; dorsal versus ventral in the rodent). Indeed, distinct functional large-scale networks of posterior versus anterior hippocampus and surrounding parahippocampal regions [13] have been hypothesized to differentially support precise versus general/gist-based memory, respectively [3–6]. However, there is little direct evidence for the reliance of recollection precision on distributed functional brain networks. To test the hypothesized involvement of the hippocampal posterior-medial (HPM) network in memory precision, we used noninvasive electromagnetic stimulation methods that increase functional connectivity of the HPM network [7] in conjunction with a graded assessment of associative object-location memory (Figure 1A) designed to segregate recollection precision from general success [11]. We predicted that HPM network-targeted stimulation would improve memory precision and modulate established electroencephalogram (EEG) correlates of recollection [8].

There is high anatomical [14] and fMRI connectivity [13, 15] between lateral-parietal cortex and hippocampus. We have previously shown that five daily sessions of repetitive high-frequency transcranial magnetic stimulation (rTMS) delivered to stimulation-accessible parietal cortex locations enhances fMRI connectivity between posterior hippocampus and the associated retrosplenial, parahippocampal, medial-parietal, and lateral-parietal HPM cortical network regions [7]. We identified participant-specific stimulation locations in the lateral-parietal cortex based on high resting state fMRI connectivity with anatomically defined left hippocampal seed locations (Figure 1B; Figure S1), in order to noninvasively target the HPM network.

Sixteen participants completed a 2-week, sham-controlled, counterbalanced paradigm (Figure 1C), involving memory testing ~24 hr before and after HPM network-targeted stimulation (stim) compared to rTMS of the same intensity delivered to the vertex, a location outside of the HPM network (sham) (see the Supplemental Experimental Procedures for further details). At each of the assessments (pre-stim, post-stim, pre-sham, and post-sham), EEG was collected during memory recall of 96 unique object locations (Figure 1A). Pre-stim and pre-sham

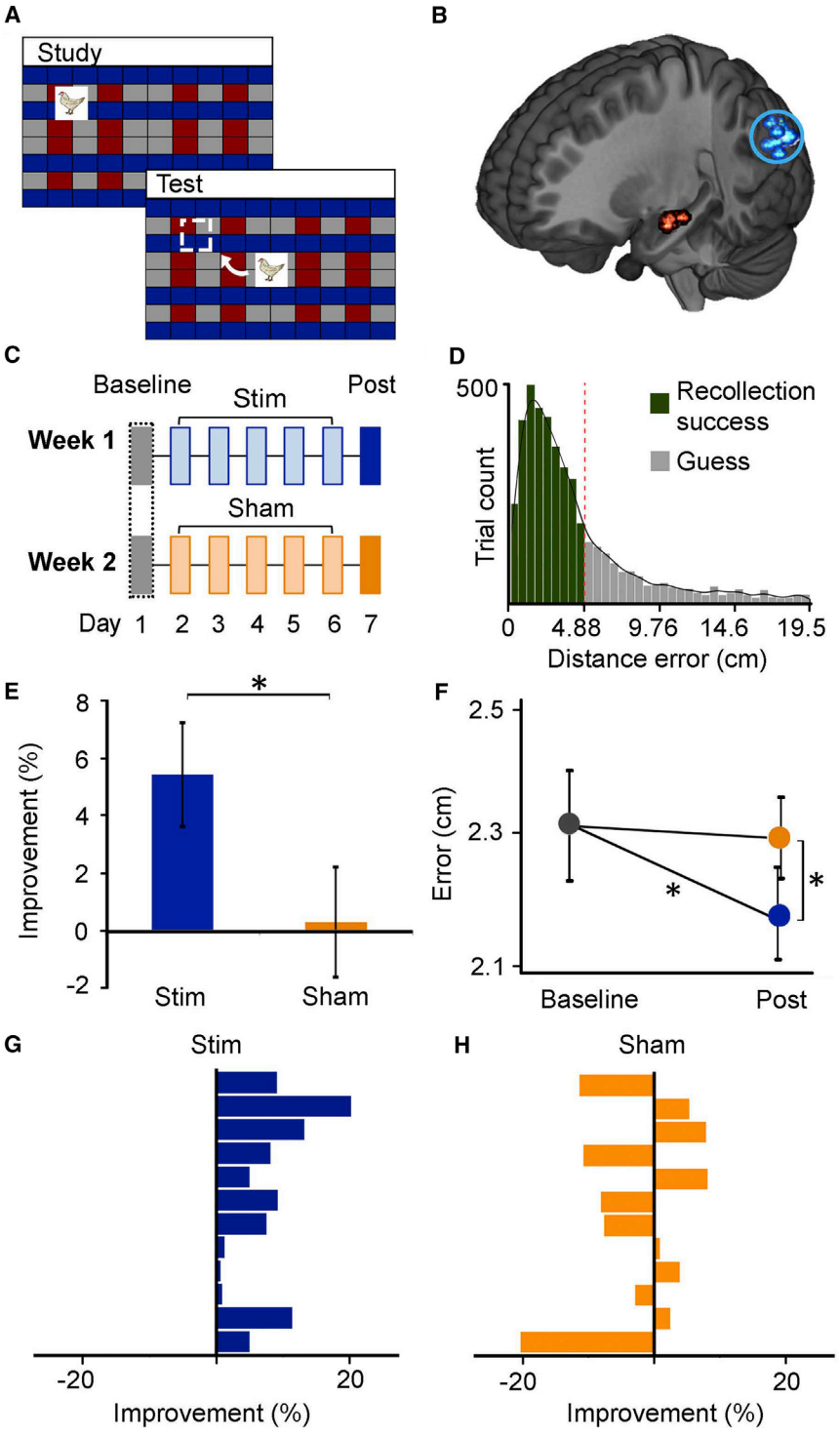

Figure 1. HPM Network-Targeted Stimulation Enhanced Recollection Precision

(A) Participants studied trial-unique objects at randomly assigned locations. Subsequent memory testing involved object cued recall of locations.

(B) fMRI connectivity used anatomically defined hippocampal body seeds (red) to define parietal maximum stimulation locations (blue, circled) in each participant. Each dot indicates the locations for one participant (see Figure S1).

(C) Five daily sessions of stim or sham stimulation followed baseline memory testing. Post-stim and post-sham testing followed the final daily stimulation session by ~24 hr. Stim and sham conditions were administered within subjects in counterbalanced order.

(D) Histogram of distance error for all participants and conditions. Successful recollection (green) and guessing (gray) trials were defined via converging modeling approaches (see the Supplemental Experimental Procedures).

(E) Percentage improvement from baseline was significantly above zero for post-stim, but not post-sham, and significantly greater for post-stim than post-sham.

(F) Distance error for successful recollection was reduced post-stim relative to baseline (but not post-sham relative to baseline) and post-stim relative to post-sham (see Table S1).

(G) Percentage change in precision due to stim for each participant.

(H) Percentage change in precision due to sham for each participant. Error bars indicate SEM. *$p < 0.05$.

data were collapsed into a common baseline. Main analyses included data from only the participants that contributed to EEG comparisons (n = 12), although the same pattern of selective effects on recall precision was found in the entire sample (see below and the Supplemental Experimental Procedures for more details). The Northwestern University Institutional Review Board approved all procedures.

Network-Targeted Stimulation Selectively Improved Recollection Precision

Trials were scored for distance error (difference between recalled and studied locations) and sorted into successful recollection (67.6% of trials, SE = 4.5%) and guess conditions using a two-parameter model that segregates recollection precision from general recollection success [11] (Figure 1D; see the Supplemental Experimental Procedures). We did not hypothesize the effects of stimulation on general recollection success, which was tested using two complementary approaches. First, the proportion of trials categorized as reflecting successful recollection (distance errors less than the 4.88-cm threshold, see the Supplemental Experimental Procedures) did not significantly change post-stim ($t(11) = 1.88$, $p = 0.26$) or post-sham ($t(11) = 0.18$, $p = 0.86$) relative to baseline. Second, the same growth-mixture fitting that was used to define the group-level threshold for recollection success was used to estimate successful recollection for each participant and memory assessment [11] (see the Supplemental Experimental Procedures). Individualized

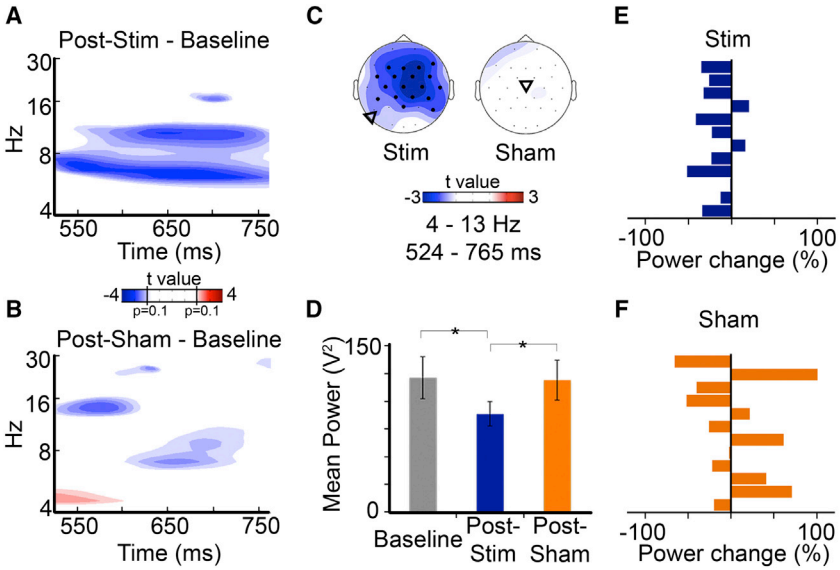

Figure 2. Stimulation Reduced Evoked Oscillatory Correlates of Successful Recollection

(A and B) Plot indicates *t* values for pairwise comparisons of the time × frequency power spectra for (A) post-stim and (B) post-sham versus baseline, averaged across electrodes and latency identified via cluster detection. The significant cluster of reduced power relative to baseline is evident for post-stim, but not post-sham.

(C) Topographical maps of *t* values demonstrate the frontal-central distribution of these effects. Electrodes identified via cluster detection are highlighted by bold markers. Triangles indicate the approximate averaged stimulation locations for each condition. See also Figure S2.

(D) Mean 4- to 13-Hz averaged power for all electrodes was reduced for post-stim relative to both baseline and post-sham.

(E and F) For each participant, mean 4- to 13-Hz power percentage change is shown (E) post-stim and (F) post-sham relative to baseline values. Error bars indicate SEM. *p < 0.05.

successful recollection thresholds did not significantly differ for pre-stim versus post-stim ($t(11) = 0.603$, $p = 0.56$) or for pre-sham versus post-sham ($t(11) = 0.591$, $p = 0.57$). Both methods thus converged to indicate that stimulation did not alter general recollection success.

Precision was defined as the mean distance error for successful recollection trials [11]. In contrast to general success, recollection precision measured post-stim improved relative to baseline ($t(11) = 2.99$, $p = 0.01$; Cohen's $d = 0.86$), but not post-sham relative to baseline ($t(11) = 0.14$, $p = 0.89$) (Figure 1E). The percentage improvement from baseline was significantly greater for post-stim than post-sham ($t(11) = 2.63$, $p = 0.02$; Cohen's $d = 0.76$). Furthermore, raw distance error (Figure 1F) was less for post-stim than for post-sham ($t(11) = 2.68$, $p = 0.02$, Cohen's $d = 0.77$). Thus, HPM network-targeted stimulation (and not sham) improved recollection precision. Recollection precision improvements were highly consistent across subjects due to stim (12/12 improved; Figure 1G; sign test $p < 0.0005$) but were at chance due to sham (6/12 improved; Figure 1H; sign test $p = 1.0$). Similar effects of stimulation were identified when individual pre-stim and pre-sham values were used rather than the common baseline (see the Supplemental Experimental Procedures), confirming that stimulation improved precision irrespective of the choice of baseline. Precision improvements were also highly consistent in the entire N = 16 sample. Percentage improvement values (post versus pre) were significantly greater for stim than for sham ($t(15) = 2.89$, $p = 0.01$; Cohen's $d = 0.72$), and precision improvements due to stim were highly consistent across subjects (15/16 improved; sign test $p = 0.0005$) but were at chance due to sham (7/16 improved; sign test $p = 0.80$) (see the Supplemental Experimental Procedures for additional full-sample analysis details).

To test the possibility that the relative difference for stim versus sham effects was due to impairments for sham rather than improvements for stim (as rTMS intensity for stim and sham was matched but with delivery to different locations), we also performed a separate control experiment in which zero-intensity stimulation was delivered to the HPM parietal target in an additional group of subjects (N = 12; see the Supplemental Experimental Procedures). Precision memory was not reliably improved in this additional control condition. There was no significant change in raw error post-control versus pre-control ($t(11) = 1.57$, $p = 0.145$). Furthermore, improvements were not consistent across subjects (7/12 participants improved; sign test $p = 0.774$; see the Supplemental Experimental Procedures for more details). Precision improvements were therefore selective for stim and did not occur for either of the control conditions.

Network-Targeted Stimulation Reduced Evoked EEG Correlates of Recollection

Theta-alpha frequency oscillatory activity and late-positive event-related potentials (ERPs) are stimulus-evoked neural correlates of recollection [9, 10]. We hypothesized that stimulation would modulate these neural signals of memory retrieval [16], providing neural correlates of the corresponding recollection precision improvement [17]. Many manipulations that increase the subjective experience of recollection and memory response accuracy correspond to increases in low-frequency oscillatory power and event-related late-positive ERP amplitude. However, reductions in oscillations and ERPs have also been associated with memory retrieval, specifically for sensory reactivation [18, 19] and increased flexibility for high-resolution information storage [20]. The nature of effects on EEG/ERP correlates of memory retrieval (i.e., enhancements or reductions) therefore can provide mechanistic insights regarding precision improvement due to stim.

Based on fMRI-EEG evidence linking 4- to 13-Hz (theta-alpha) oscillatory EEG activity to fMRI connectivity of the retrosplenial cortex and hippocampus during recollection [8], we first tested the effect of stimulation on evoked oscillatory EEG power using this a priori frequency band of interest. We compared 4- to 13-Hz evoked oscillations for successfully recollected trials among post-stim, post-sham, and baseline conditions. Cluster-based non-parametric simulation testing yielded significant fronto-parietally distributed (Figure S2A) power reduction from 524 to 765 ms for post-stim relative to baseline (Figures 2A–2C; cluster-corrected $p = 0.03$). The same test for post-sham relative

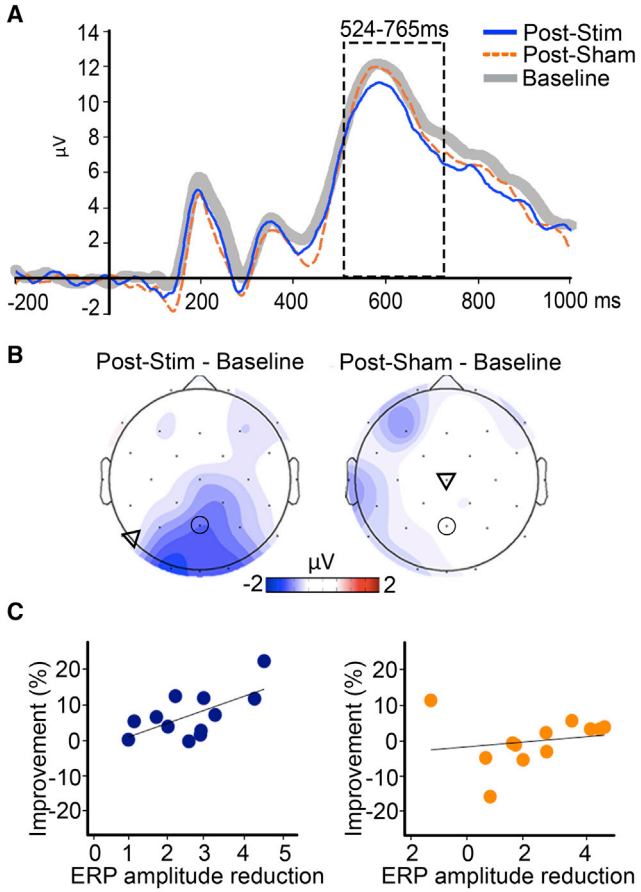

Figure 3. Stimulation Reduced ERP Correlates of Successful Recollection

(A) ERPs for post-stim (blue), post-sham (dashed orange), and baseline (thick gray) for one representative electrode (Pz). See also Figure S3.

(B) Topographical plot of the amplitude reduction relative to baseline shows the posterior distribution characteristic of the parietal memory effect (the circled electrode is Pz). Triangles indicate the approximate averaged stimulation locations for each condition.

(C) Relative to baseline, a greater reduction in ERP amplitude (baseline − post) was associated with a greater percentage improvement in recall precision for stim (blue), but not sham (orange), tested using robust correlation.

to baseline identified no significant power differences ($p > 0.3$). The 4- to 13-Hz power averaged for all electrodes for the 524- to 765-ms period (Figure 2D) was significantly less post-stim compared to baseline ($t(11) = 3.00$, $p = 0.01$; Cohen's $d = 0.87$) and compared to post-sham ($t(11) = 2.24$, $p = 0.05$; Cohen's $d = 0.65$), whereas the post-sham versus baseline difference was not significant ($t(11) = 0.14$, $p = 0.88$). These reductions of theta-alpha power were consistent across subjects due to stim (reductions in 10/12 participants; sign test $p = 0.039$; Figure 2E), but not due to sham (reductions in 7/12 participants; sign test $p = 0.774$; Figure 2F). Further, an independent cluster-based non-parametric test for frequency, across an a priori recollection latency interval [10], identified a significant 6- to 11.5-Hz power reduction for post-stim versus baseline (Figure S2B) and no significant differences for post-sham versus baseline, consistent with the a priori frequency band used for primary analyses. Inter-trial theta-alpha phase coherence was reduced post-stim relative to baseline and post-sham late in the epoch (Figure S2C), suggesting that sustained stimulus-evoked processing was reduced post-stim. Thus, stimulation-induced recollection precision improvement was associated with corresponding reductions in theta-alpha oscillatory activity.

We next tested the effect of stimulation on ERP correlates of successful recollection. Comparison of recollection success ERPs for both post-sham and post-stim yielded prototypical late-onset positive increases in amplitudes for successful recollected trials relative to guesses at parietal and occipital electrodes (known as the "parietal memory effect" [10]; Figure S3). This indicates reliable ERP correlates of successful memory irrespective of stimulation condition. For the 524- to 765-ms latency interval of interest derived from cluster-based permutation testing, mean ERP amplitude for parietal-occipital electrodes was reduced post-stim relative to baseline ($t(11) = 3.31$, $p < 0.01$; Cohen's $d = 0.96$), whereas post-sham amplitudes did not differ from baseline ($t(11) = 0.86$, $p = 0.41$) (Figures 3A and 3B). Further, correlation analyses using robust fitting to guard against outlier influences indicated that greater post-stim versus baseline amplitude reduction was associated with greater recollection precision improvement (robust-$r = 0.659$, $p = 0.02$) (Figure 3C). This relationship was not significant for post-sham (robust-$r = 0.023$, $p = 0.94$). Stimulation thus reduced amplitudes of ERP correlates of recollection and these reductions corresponded to recollection precision improvements. As was the case for effects on recollection precision, theta-alpha power and mean ERP amplitudes were not significantly changed due to zero-intensity control stimulation in the additional control group (see the Supplemental Experimental Procedures).

DISCUSSION

Stimulation targeted to the HPM network improved precision, but not general success, of associative object-location memory. Notably, lesion-deficit and fMRI studies have implicated the human hippocampus [21–24] as well as parietal cortex [25, 26] in memory for precise details, suggesting that stimulation affected these HPM network locations. Indeed, changes in fMRI connectivity caused by the same stimulation parameters used here enhanced fMRI connectivity within the HPM network, particularly for hippocampus and medial aspects of parietal, occipital, and retrosplenial cortex [7]. Interestingly, just as fMRI connectivity enhancements with hippocampus were greater for medial regions than the lateral-parietal regions that were stimulated [7], the changes in EEG/ERP correlates of recollection reported here occurred with medial distributions that were distal to the stimulation location (Figures 2 and 3). Collectively, this supports the interpretation that there were network-level effects of stimulation reflecting HPM network involvement in memory precision.

In a previous study, HPM-network-targeted stimulation improved paired-associate cued recall, also measured ~24 hr after the final stimulation session [7]. Cued recall is an "all-or-nothing" memory measure and so differentiation of recollection precision from success was not possible. Relative to the previous cued recall testing, the current memory test involved substantially larger memory demands using a very different format (~100 random objects at precise locations within a redundant

grid display versus ~20 face-word pairings in the previous study). Nonetheless, it is noteworthy that the effect sizes for stimulation on precision reported here (in a range typically classified as large) are similar to those obtained previously for cued recall [7]. The current findings provide novel information on the network basis of memory, because they demonstrate the link between a highly specific aspect of memory, recollection precision, and the HPM network. Isolation of stimulation effects on precision from other co-occurring memory processes, such as memory success within the same task, is especially crucial for validating stimulation effects on memory and network-level processing, as condition-selective effects help mitigate influences from potential nonspecific factors, such as history, practice, and placebo effects.

Stimulation targeted to the HPM network reduced the amplitude of EEG correlates of recollection precision. This reduction is consistent with the hypothesis that successful retrieval of visual details corresponds to rapid memory reactivation [19, 27] and aligns with mounting evidence that reduced theta power correlates with better item-context memory [20]. One possibility is that stimulation promotes asynchronous activity within the medial temporal lobe and the neo-cortex, which produces the flexibility for higher resolution information storage and retrieval [27–31]. Increased EEG/ERP power and amplitude have been related to improved memory in many studies [9, 10, 17], and EEG/ERP oscillatory enhancements versus reductions may represent a neural distinction between general/semantic memory success and visuospatial memory precision. Memory for general information can benefit from verbal-semantic mnemonic strategies associated with the anterior hippocampal network, with heightened verbalization of recollection content during retrieval related to EEG/ERP increases. In contrast, the HPM network may support memory for precise perceptual details [3, 5] that does not benefit substantially from semantic strategies. This latter type of memory might be indicated by EEG/ERP decreases, as we observed due to stimulation in conjunction with enhanced memory for these perceptual details.

Evoked activity reductions may also indicate efficient processing. For example, evoked activity reductions can occur in conjunction with enhanced fMRI connectivity in experiments on priming [32], which is thought to reflect heightened processing efficiency [33]. This pattern is consistent with our findings, in which stimulation enhanced HPM network fMRI connectivity [7] and reduced recollection-related evoked activity, specifically at frequencies characteristic of HPM network communication [34]. Although effects on memory precision were robust in the entire sample, our EEG/ERP subsample was relatively small. Nonetheless, several design features enhance confidence in our reported neural findings, including strong a priori hypotheses on the particular neural signals that would be affected by stimulation, effects that significantly outlasted the stimulation sessions, as well as matched-intensity control (sham) stimulation in addition to a separate zero-intensity, site-specific control group.

To summarize, our findings causally support hypothesized functional specialization of large-scale hippocampal-cortical networks. Although stronger tests of network functional subdivision could utilize control stimulation sites at varying levels of distance from the targeted network, our findings of stimulation-induced changes for a specific aspect of memory, recollection precision, extend previous lesion-based causal evidence for large-scale brain network involvement in cognition. That is, lesion-based accounts have necessarily focused on broad cognitive constructs, providing evidence for large-scale networks involved in language, attention, memory, and visuospatial processing [35], whereas the current findings demonstrate a causal role for a stimulation-responsive brain network and a highly specific neurocognitive construct. Finally, mechanisms by which brain stimulation affects cognition remain mysterious. There have been few demonstrations of stimulation effects on highly specific neural markers of well-defined cognitive processes [36], but in those studies the effects did not outlast the period when stimulation was applied. The recollection precision improvements reported here outlasted the period of stimulation by ~24 hr, consistent with our previous report of improvements lasting up to ~2 weeks after stimulation [37]. These long-lasting stimulation-induced changes included neural markers of detailed memory content reactivation during recollection, thereby advancing the understanding of how noninvasive brain stimulation could alter network function. Generation of long-lasting improvement in memory ability (rather than improved retention of specific material) has implications for the development of treatments for the many disorders related to hippocampal-cortical network dysfunction [15].

SUPPLEMENTAL INFORMATION

Supplemental Information includes Supplemental Experimental Procedures, three figures, and one table and can be found with this article online at http://dx.doi.org/10.1016/j.cub.2016.12.042.

AUTHOR CONTRIBUTIONS

D.J.B. and J.L.V. designed the study. A.S.N., D.J.B., and E.P.G. collected the data. S.A.V. monitored stimulation parameter and participant safety. A.S.N., D.J.B., and J.L.V. performed the analyses. All authors discussed the results and wrote the manuscript.

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