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Human neocortical oscillations exhibit theta phase differences between encoding and retrieval

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We analyzed intracranial brain activity recorded from human participants during the performance of a working-memory task. We show that 6–13 Hz activity exhibits consistent phase across trials following experimental stimuli, and that this phase significantly differs between study and test stimuli. These findings suggest that oscillatory phase reflects the encoding-retrieval state of neural networks, supporting predictions of recent models of memory. [Hasselmo, M.E., Wyble, B.P., and Bodelon, C., 2002. A proposed function for hippocampal theta rhythm: Separate phases of encoding and retrieval enhance reversal of prior learning. Neural Comput. 14, 793-817; Judge, S.J., Hasselmo, M.E., 2004. Theta rhythmic stimulation of stratum lacunosum-moleculare in rat hippocampus contributes to associative LTP at a phase offset in stratum radiatum. J. Neurophys. 92, 1516-1624].

Introduction

Rhythmic activity is a prominent feature of neuronal networks, appearing at numerous frequencies and exhibiting a variety of behavioral correlates in both animals and humans (Kahana, 2006; Varela et al., 2001; Buzsáki and Draguhn, 2004). In the rat hippocampus, where oscillations have been studied in exquisite detail, a 4–12 Hz oscillation known as the theta rhythm appears prominently during locomotion and orienting (Bland, 1986; O'Keefe and Recce, 1993; Skaggs et al., 1996; Shin and Talnov, 2001), and has been shown to modulate both synaptic plasticity (Hölscher et al., 1997) and memory function (Landfield, 1977; Winson, 1978; Macrides et al., 1982; Givens and Olton, 1990).

An intriguing feature of the hippocampal theta rhythm is its ability to organize the firing of individual pyramidal cells, which fire at specific phases during the ongoing oscillation. This has been shown both for hippocampal cells that encode spatial location (so-called place cells; O'Keefe and Recce, 1993; Skaggs et al., 1996), as well as for cells that represent other types of information (Harris et al., 2002). Jensen and Lisman (2000) found that theta phase information can be used in concert with place-cell firing rates to reconstruct the position of a rat with better accuracy than firing rate information alone. Similarly, Mehta et al. (2002) have shown how the phase dependence of place cell activity provides a mechanism for temporal coding in the hippocampus and may underlie sequence learning in neural networks.

Recent computational models of hippocampus and surrounding medial temporal lobe structures have hypothesized that oscillations serve to organize the processes of encoding and retrieval (Hasselmo et al., 2002; Judge and Hasselmo, 2004) or the way in which multiple items are held in working memory (Jensen and Lisman, 1998). These models rely on the ability of neural networks to reset their oscillations when they are recruited to process information. In rats, Givens (1996) demonstrated stimulus-induced reset of ongoing oscillations in response to the demands of a working-memory task.

To determine whether similar reset could be observed in human cortex, Rizzuto et al. (2003) had epileptic patients undergoing intracranial EEG (iEEG) monitoring for seizure localization perform a recognition-memory task. They found that 6–13 Hz oscillations exhibited an almost instantaneous stopping and restarting following both encoding and retrieval of consonants. The consistent phase of the oscillatory activity following stimulus presentation suggested that some stimulus-evoked cognitive process resets the phase of ongoing oscillations. Measuring the trial-to-trial variability in the timing of phase reset, they found that the presentation of list items at study often elicited phase reset with less temporal precision than did the presentation of those same items at test. That is, the reset to list items often exhibited greater temporal variance than did the reset to test probes. These effects were widespread throughout human

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Table 1
Participant demographics and performance

Participant	Age	Gender	Electrode placement	Resection	# Electrodes	# Excluded	Accuracy (%)	Mean reaction time (ms)
1	17	M	subdural	L anterior temporal	80	20	98	1168
2	12	F	subdural	R sensory-motor transection	104	12	94	970
3	14	M	depth	no resection	19	2	99	1240
4	21	M	depth/subdural	R temporal	76	17	85	1232
5	18	M	subdural	anterior temporal	64	16	96	655
6	15	M	subdural	R anterior temporal	128	31	92	1158
7	9	M	subdural	R temporal	80	15	92	1104
8	42	F	depth/subdural	R temporal	62	30	90	766
9	41	M	subdural	R temporal	64	12	97	954
10	38	M	subdural	R temporal	94	4	98	1483

Age, gender, electrode placement, resection, number of electrodes, accuracy (percent correct), and mean reaction time (correct responses only) for each participant.

cortex, and exhibited specificity for either the encoding or the retrieval phase of the task in different brain regions. Importantly, Rizzuto et al. (2003) found that reset of oscillations was not generally accompanied by increases in oscillatory power, as would be expected if they resulted from an evoked component in the EEG (Yeung et al., 2004). Fernández and colleagues have recently shown that phase reset occurs during continuous recognition as well (Mormann et al., 2005).

The finding of phase reset during the performance of a recognition-memory task highlights the importance of oscillatory phase in human cognitive function. Although list items and test probes both elicited phase reset, it is still unknown whether the resulting phase (after reset) is the same or different in these two cases. In the current study, we reanalyzed the data from Rizzuto et al. (2003), examining the mean phase of oscillatory reset during performance of a short-term item-recognition task (Sternberg, 1966). Specifically, we examined differences in mean phase angle between study items and test probes at a wide range of frequencies and found significant differences at many locations.

Methods

Participants

We tested ten participants who had been implanted with cortical surface (subdural) and/or bilateral depth electrodes. The clinical team determined the placement of these electrodes to best localize epileptogenic regions. Across all patients, we recorded from 765 electrodes; 159 of these (a) lay in the epileptic focus, (b) overlay regions of radiographically evident structural brain damage, or (c) exhibited epileptiform EEG (i.e., spikes and/or sharp waves, as determined by the clinical team). We restricted our analyses to the remaining 606 electrodes. All participants had normal range

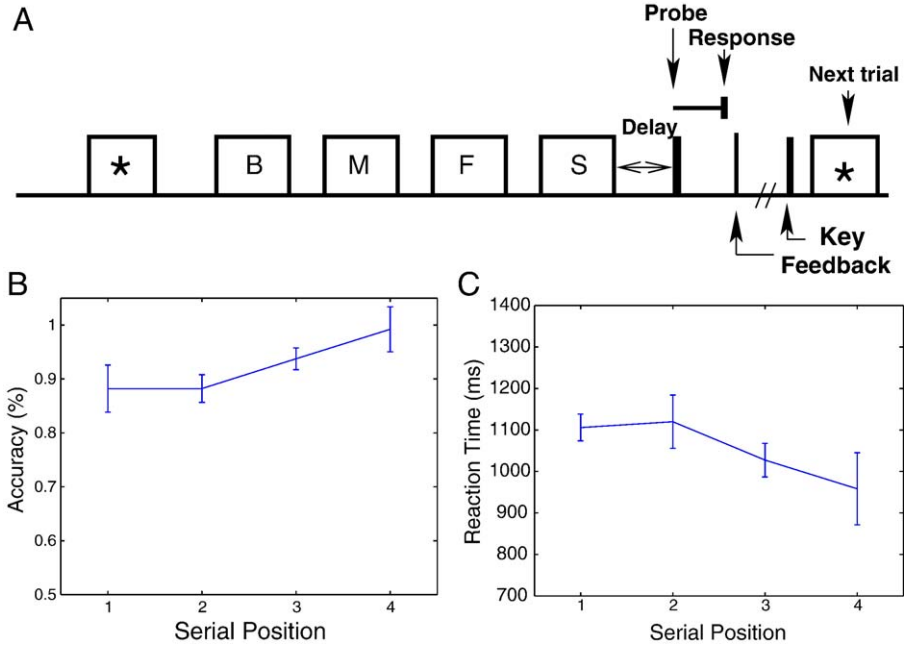

Fig. 1. The Sternberg item-recognition task and behavioral measures. (A) Illustration of the experimental design. The orienting stimulus (asterisk) appears first, followed by the four list items and a delay. After the delay, a test probe is presented and the participant makes a response indicating whether or not the test probe was in the preceding list. (B) Mean accuracy for targets testing each serial position. (C) Reaction time of correct responses to targets at each serial position. Error bars denote 95% within-participant confidence intervals calculated according to Loftus and Masson (1994).

personality and intelligence and were able to perform the task within normal limits (see Table 1 and Fig. 1). Our research protocol was approved by the institutional review boards at Children's Hospital, Boston, at Brigham and Women's Hospital, Boston, and at Universitätsklinikum, Freiburg, and informed consent was obtained from the participants and/or their guardians.

Procedure

Fig. 1A illustrates the behavioral task. Participants pressed a key to initiate each trial. An orienting stimulus (an asterisk) was then displayed in the center of the computer screen and remained visible for 1 s. Following a variable delay of 200 ± 75 ms, uniformly distributed, four consonants were sequentially displayed (study items). The temporal jitter was introduced to ensure that each stimulus arrived at a random phase with respect to ongoing oscillations, thus helping to maintain a uniform prestimulus phase distribution for all conditions. Each study item was displayed for 700 ms, followed by a variable delay of 275 ± 75 ms. Study items were randomly selected, subject to the constraint that a particular study item not repeat within three successive lists. The last (fourth) study item was followed by a retention interval of 500 ± 75 ms and the presentation of the test probe. The participant was instructed to indicate as quickly and accurately as possible whether the probe item either was in the preceding list (a target) or was not in the preceding list (a lure) by pressing the right control key to indicate a target item and the left control key to indicate a lure. Targets and lures occurred with equal probability, and target items were drawn equally from each of the list positions. Because there were so few error trials during this task (<5%), we restricted our analyses to correct trials.

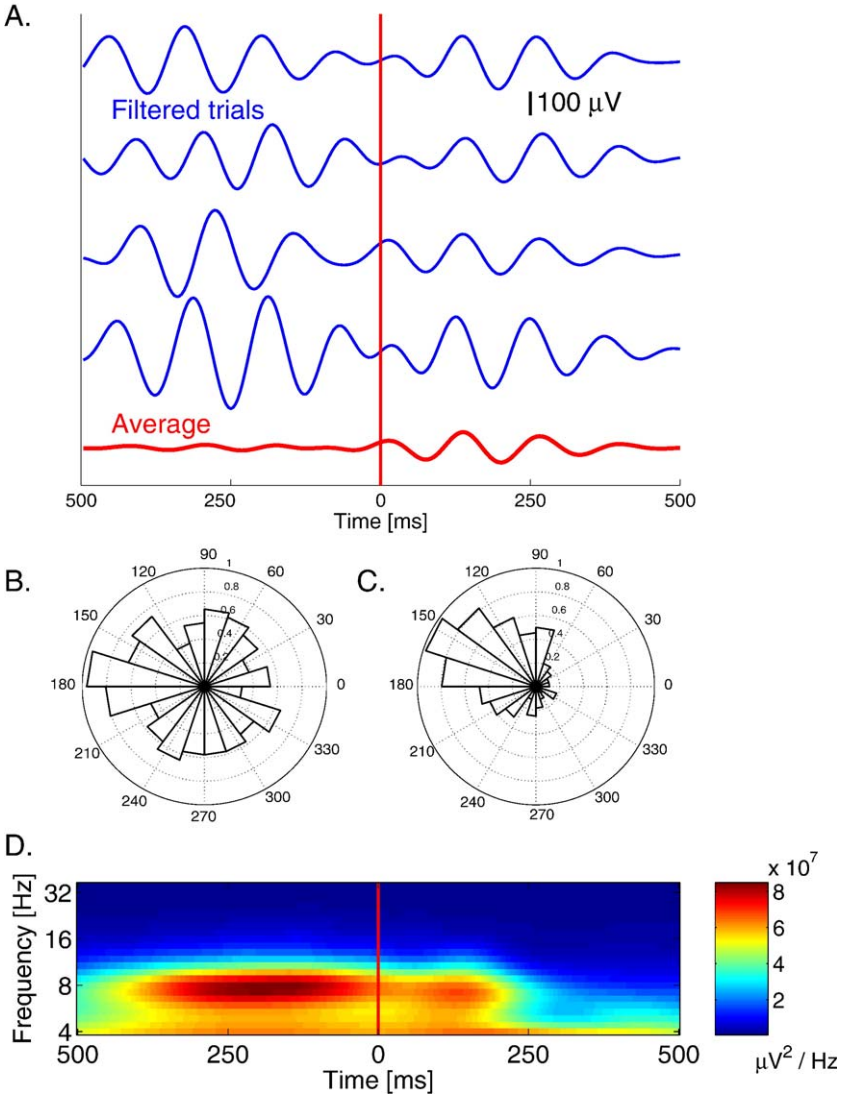

Fig. 2. Test probes reset 8 Hz phase. (A) Filtered single trials (blue lines) and average activity (red line) illustrating phase reset to the test probes at the level of single trials for the 1-s interval surrounding probe presentation. Average calculated over all 304 trials completed. Probe onset occurs at time $t = 0$. Electrode located in the left inferior temporal lobe of Participant 5. [Talairach coordinates: (L–R, A–P, I–S) (−33, −56, −17) mm.] (B) 8 Hz phase distribution 250 ms prior to the presentation of the test probe, calculated across trials. (C) 8 Hz phase distribution 250 ms after the probe has been presented. (D) Time–frequency spectrogram showing average power (calculated across all unfiltered trials) at each frequency for the 1-s interval surrounding probe presentation.

iEEG recordings

The iEEG signal was recorded from platinum electrodes (3 mm diameter) with an inter-electrode spacing of 1 cm (for subdural electrodes) or 8 mm (for depth electrodes). The signal was amplified, sampled at 256 Hz (Children's Hospital, Bio-Logic Corp. apparatus, Participants 1–7; Universität Freiburg, DeltaMed SA apparatus, Participant 8) or at 200 Hz (Brigham and Women's Hospital, Nicolet Biomedical Inc. apparatus, Participants 9 and 10), and band-pass filtered (Bio-Logic Corp., 0.3–70 Hz; Nicolet Biomedical Inc., 0.5–60 Hz; DeltaMed SA, 0.015–120 Hz). For all participants, the locations of the electrodes were determined using coregistered postoperative CTs and preoperative MRIs by an indirect stereotactic technique.

Analysis of phase reset

The analysis of phase reset follows Rizzuto et al. (2003). Briefly, iEEG records were first transformed into the frequency domain using a five-cycle Morlet wavelet. The phase of activity at each frequency and time point was calculated, and the distribution of phase (across trials) was tested against the null hypothesis of uniformity using the Rayleigh statistic (Beran, 1968, 1969). A condition was deemed to elicit significant phase reset at a given electrode/frequency if it exceeded a significance threshold ($P < 0.001$) throughout a 2-period duration threshold.

Analysis of mean phase

Mean phase differences were assessed using the summary statistic developed by Watson (1983),

$$Y_r = 2\left[\frac{N - \sqrt{\left(\sum_{i=1}^{r} n_i \cos\hat{\mu}_i\right)^2 + \left(\sum_{i=1}^{r} n_i \sin\hat{\mu}_i\right)^2}}{\sum_{i=1}^{r} n_i \hat{\delta}_i / N}\right] \quad (1)$$

where r represents the number of conditions being compared (two, in all the cases we consider here), and μ_i, δ_i, n_i and N represent the estimated mean, estimated circular dispersion of the mean (analogous to the standard deviation), number of trials for the ith condition, and total trials over all conditions, respectively. This equation applies to the case of all distributions having approximately equal variability ($\delta_{\max}/\delta_{\min} \leq 4$). When this constraint is not met, the equation above is modified as follows (see Fisher, 1993),

$$Y_r = 2\left[\sum_{i=1}^{r} 1/\hat{\sigma}_i^2 - \sqrt{\sum_{i=1}^{r} (\cos\hat{\mu}_i)/\hat{\sigma}_i^2 + \sum_{i=1}^{r} (\sin\hat{\mu}_i)/\hat{\sigma}_i^2}\right] \quad (2)$$

where σ_i represents the circular standard error of the mean. Y_r is χ^2_{r-1} distributed and the appropriate χ^2 distribution was consulted to assess statistical significance.

Following Rizzuto et al. (2003), we adopt a P value and a duration threshold for assessing statistical significance. Two conditions were deemed to be significantly different at a given electrode/frequency if they exceeded a significance threshold ($P < 0.001$) throughout a 1.5-period duration threshold.

Results

All participants completed the task with high accuracy, and with response times that were slightly slower than normal participants (see Table 1; Hwang et al., 2005; Zhang et al., 2003; Manoach et al., 2003). Fig. 1 plots the accuracy (panel A) and response time (panel B) as a function of the test probe's position in the list. As expected, accuracy increases and reaction time decreases for more recently presented test probes.

As has been reported previously (Rizzuto et al., 2003), many brain locations exhibited a phase reset of oscillatory activity during performance of this task. That is, oscillations of different frequencies undergo a nearly instantaneous stopping and restarting at many cortical locations.

Fig. 2A plots several trials of representative activity (filtered in the 6–10 Hz band) located in the left inferior temporal lobe. Before presentation of the test probe, the oscillations do not show a consistent phase across trials. This can be seen in the fact that the peaks and troughs do not line up from trial to trial. However, after presentation of the test probe (indicated by the red line), the oscillations become synchronized and the peaks and troughs line up

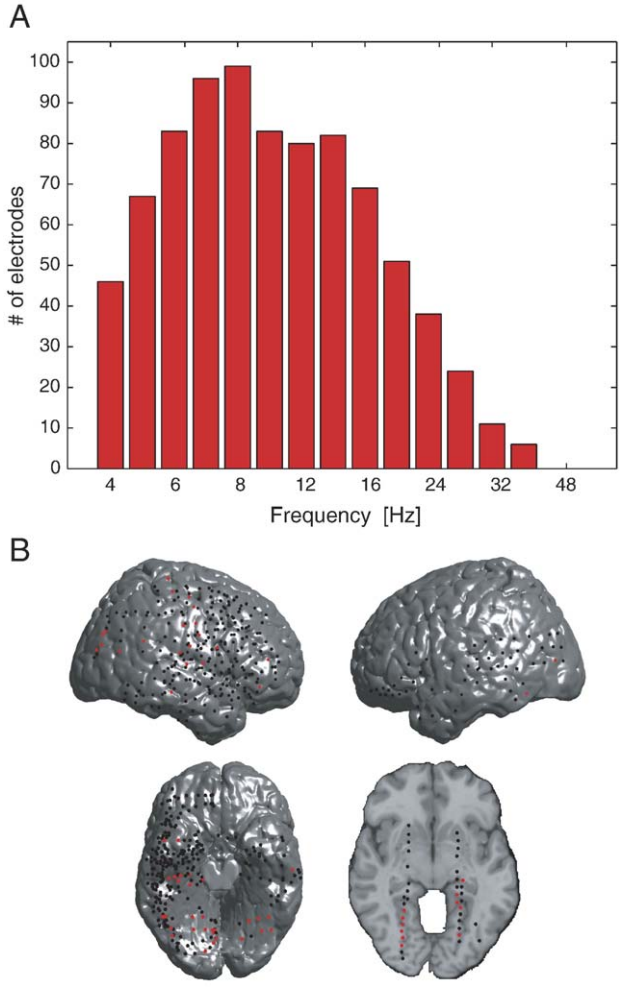

Fig. 3. Test probes elicit unimodality at many frequencies and brain locations. (A) Histogram of the number of electrodes exhibiting phase reset at each frequency. (B) Topographic maps showing the location of all electrodes exhibiting unimodality in the 6- to 13-Hz frequency band (red circles).

across trials. To quantitatively examine this effect, we analyzed the 8 Hz phase distributions directly. Fig. 2B plots the phase distribution from a window centered 250 ms prior to presentation of the probe. This distribution is not significantly different from a uniform distribution (Rayleigh test, $\overline{R} = 0.02$, $P = 0.83$). However, 250 ms after the presentation of the probe the phase distribution becomes highly concentrated around a particular phase (Fig. 2C), and the possibility that this distribution is uniform can be rejected ($\overline{R} = 0.64$, $P < 0.001$). Fig. 2D plots the spectral power as a function of frequency and time, showing that 8 Hz oscillations are present both before and after presentation of the test stimulus.

Fig. 3A plots a histogram of the number of electrodes exhibiting phase reset (see Methods) between 4 and 45 Hz. The majority of electrodes exhibit reset between 6 and 13 Hz, and we therefore focus on this band for the remainder of our analyses. Fig. 3B plots the locations of all electrodes exhibiting phase reset to test probes in this frequency band.

Although the data portrayed in Fig. 2 demonstrate the reset of brain oscillations, they do not address the theoretically important question of whether study and test items reset brain oscillations to different phase angles. To assess oscillatory phase differences between study and test, we compared phase estimates taken after

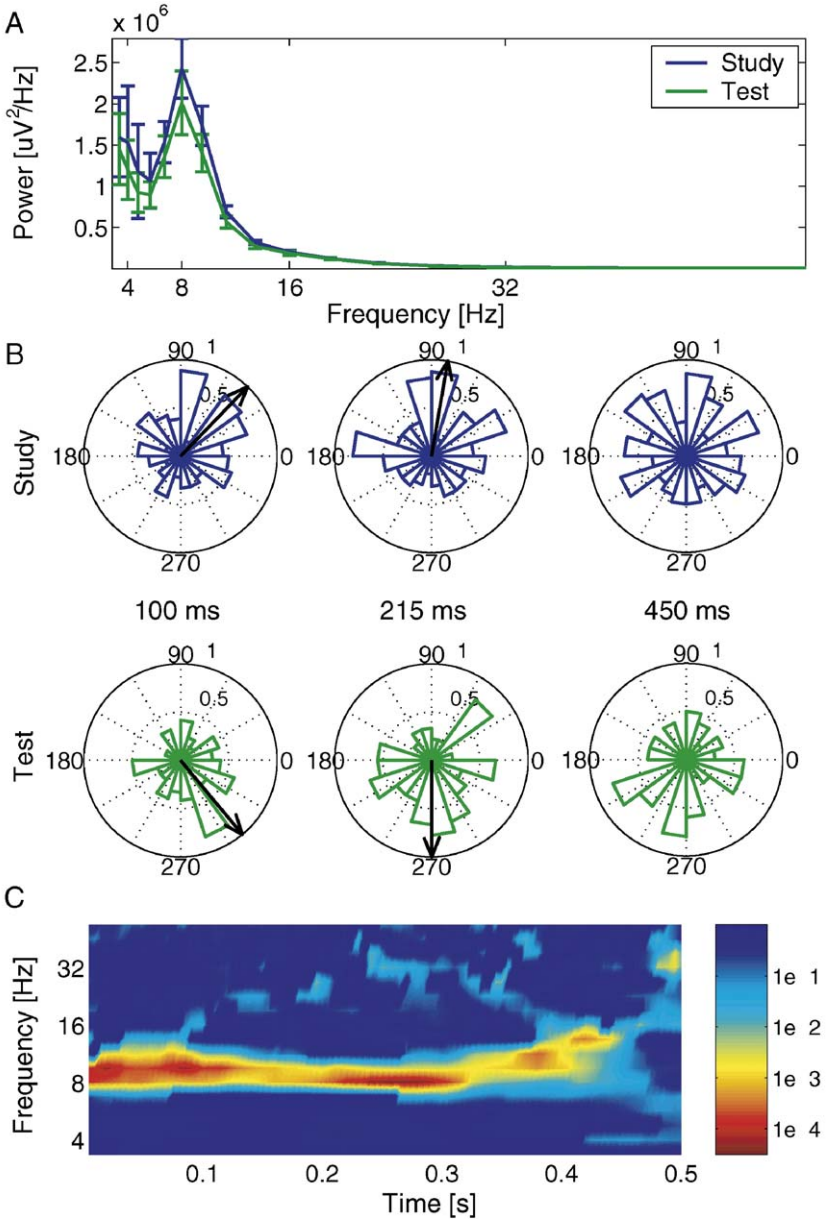

Fig. 4. Phase differences exist between oscillatory activity at study and test. (A) Mean spectral power for the 500 ms interval following study items (list item 4, in blue) and test items (correctly rejected lures, in green) for an electrode in the right frontal lobe of Participant 10. Talairach coordinates: (L–R, A–P, I–S) = (+42 +6 +17). Error bars denote SEM. (B) Polar plots of the 8 Hz phase distributions (aggregated across trials) for study (blue) and test (green) at three time points poststimulus for the same electrode. Black arrows indicate the mean phase of each distribution and are plotted only when significant unimodality exists to estimate mean phase. (C) Plot showing the significance of the difference between mean phase estimates at each frequency and time point. Time $t = 0$ represents the onset of the stimuli.

presentation of the fourth list item (the last study item seen by the participant) with those taken after presentation of correctly rejected lures. This comparison between study items and lures controlled for the matching process that occurs when a participant correctly recognizes a test probe (i.e., responds *yes* to a target) because neither the study item nor the lure item had been seen within the current trial. The conditions being compared differed in that the participants were asked to study the fourth item, but in the case of correctly rejected lures they were asked to make a judgment regarding the item's membership in the preceding list.

Of the 606 electrodes studied, 114 (18%) exhibited significant differences at one or more frequencies. Fig. 4A plots the mean spectral power in a 500 ms interval following study and test stimuli for a representative electrode in the right frontal lobe of Participant 10. This figure shows a spectral peak at 8 Hz both during presentation of the study items and during presentation of the test stimuli. Fig. 4B shows the actual 8 Hz phase distributions for each condition at three specific time points poststimulus. As early as 100 ms poststimulus the distributions have significantly different mean phase angles from one another ($P < 0.001$). This mean phase difference is also present at 215 ms poststimulus ($P < 0.001$), but is finally gone by the end of the 500 ms interval ($P > 0.3$). At 215 ms poststimulus the means of the distributions are almost 180° out of phase with one another. To visualize mean phase differences, Fig. 4C plots the P value signifying the difference in mean phase between study and test items for a wide range of frequencies in the 1/2 s interval following item presentation. This figure shows that differences in mean phase are restricted to the 8 Hz band and reach their peak between 200 and 300 ms poststimulus.

To display the frequency distribution of this effect, Fig. 5A plots the number of electrodes that exhibit significant mean phase differences between study and test. Interestingly, the majority of the significant electrodes exhibit differences in the 6- to 13-Hz frequency band. Most of the electrodes exhibiting significant mean phase differences between study and test items in this frequency range were found in medial temporal and inferotemporal regions (see Fig. 5B).

One may ask whether our findings of oscillatory phase differences between study and test items reflect a difference between successively processed stimuli rather than a difference between encoding and retrieval processes. We tested this alternative hypothesis by comparing 6–13 Hz mean phase differences between study items in list positions 3 and 4. Of the 606 electrodes tested, only six electrodes (<1%) exhibited differences between the study item in list position 3 and that of list position 4. In comparison, 64 electrodes (>10%) exhibited differences between list item 4 and lures in the same frequency band. This difference was statistically significant ($\chi^2 = 50.9$, $df = 1$, $P < 0.001$).

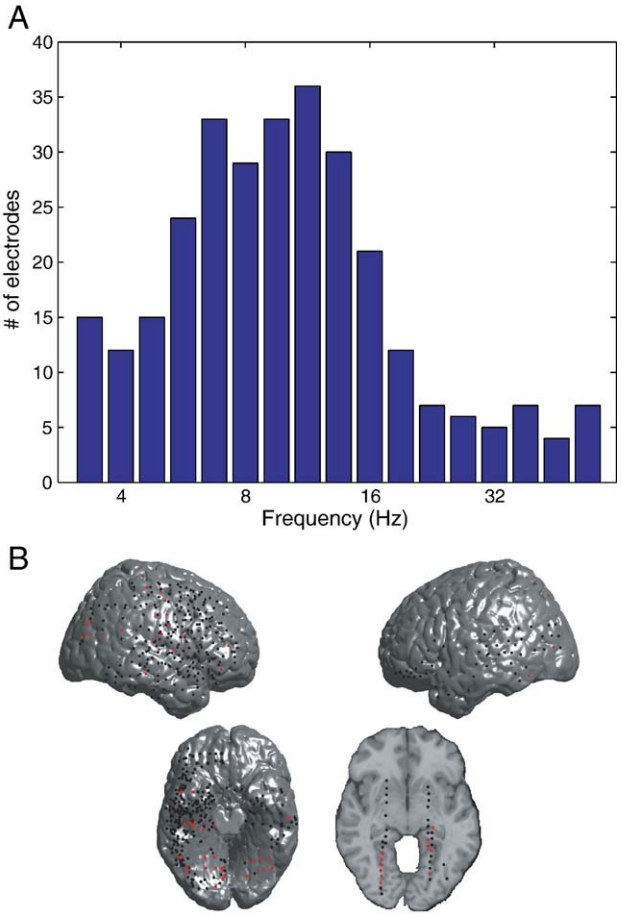

Fig. 5. Many electrodes and brain locations exhibit study-test differences. (A) The number of electrodes exhibiting mean phase differences at each frequency. (B) Topographic maps showing the locations of all electrodes exhibiting study-test differences in the 6–13 Hz band (red circles).

Discussion

Oscillations have been hypothesized to serve as timing mechanisms for neural activity, organizing different neural processes in different phases of an ongoing rhythm. In particular, phase-coding of neural activity may be used to separate the processes underlying memory encoding and retrieval (Hasselmo et al., 2002; Judge and Hasselmo, 2004). This view is supported by experimental evidence from rodents showing that the phase of theta oscillations controls whether cellular stimulation will result in long-term potentiation (LTP) or long-term depotentiation (LTD) of the synapse (Pavlides et al., 1988; Huerta and Lisman, 1993; Hölscher et al., 1997). Inasmuch as LTP and LTD serve as learning and memory mechanisms, these findings suggest that the phase of theta reset may set the state of neural networks involved in learning and memory processing.

We set out to test the hypothesis that encoding and retrieval have distinct oscillatory phases by measuring the phase of oscillatory activity surrounding the encoding or retrieval of symbolic stimuli (consonants) while patients with implanted electrodes performed a working-memory task. Recording from 606 electrodes across ten patients, we found reset of oscillatory activity in the 6–13 Hz frequency band in the interval immediately following presentation of study items and test probes (cf. Rizzuto et al., 2003). Of critical interest was whether the phase following stimulus presentation differed consistently between study and test items.

At 114 out of 606 recordings sites (18%), we found that oscillations had consistent phases across trials, but that these phases were significantly different following study items and test probes. At some sites, the phase differences were as large as 180° (Fig. 4B, middle panel); that is, the latency of the peak of oscillatory activity following study items was the same as the latency of the trough of oscillatory activity following memory probes.

To control for the effect of stimulus repetition, we limited our comparison to that between study items and novel test probes

(lures) that were correctly rejected. This comparison ensured that neither item had been seen before in the current list, and that the observed differences between the two stimulus classes were not likely to be the result of a simple repetition effect (as would be the case for correctly recognized targets). Instead, observed differences were most likely due to differences in memory processing between the two stimulus types. Additionally, we were able to reject the alternative hypothesis that the differences in oscillatory phase following study and test items reflect a difference between successively processed stimuli, rather than a difference between encoding and retrieval processes.

Although it would be interesting to know whether the mean phase of oscillatory activity was constant across subjects and recording sites, following either study or test items, this question is not well posed in our human intracranial data set, where electrodes are implanted in widely varying brain regions. It is also particularly difficult to justify a single time point as being representative, especially when different sites reset at different times.

In summary, the phase of 6–13 Hz oscillations distinguished study and test stimuli, thus providing further evidence that oscillatory activity plays an important role in memory processing. More important, our findings corroborate Hasselmo's memory model (Hasselmo et al., 2002; Judge and Hasselmo, 2004), in which encoding and retrieval operate under distinct phases of the ongoing theta activity in human temporal lobe.

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